16th World Intensive and Critical Care Congress (WICC 2023)

POSTER PRESENTATIONS

Metabolism - Endocrinology

Abstract:0011

A CASE OF DIABETIC KETOACIDOSIS ASSOCIATED WITH GUILLAIN-BARRÉ SYNDROME

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Introduction: Diabetic ketoacidosis is a serious acute complication of diabetes mellitus, which is life-threatening. Guillain-Barré syndrome is an acute immune-mediated peripheral neuropathy. Clinical features of GBS are characterized by progressive limb weakness and absent or depressed deep tendon reflexes.

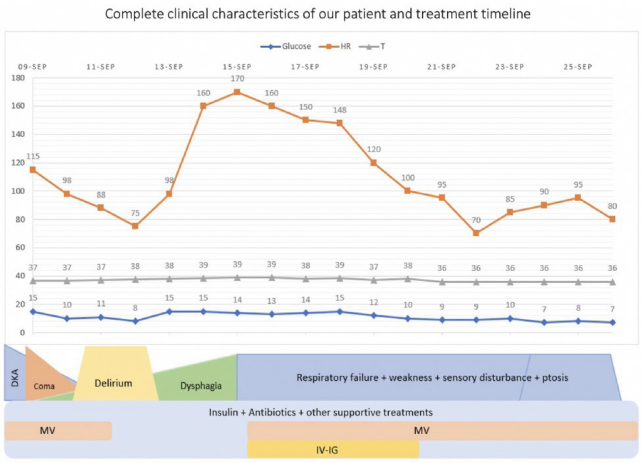
Case: We report a case of GBS-AMSAN type which became clinically severe and involved acute respiratory failure and autonomic dysfunction that developed in a young female with onset of diabetes type 1, DKA. GBS-AMSAN type is considered to be a rare variant and one that usually has a more serious clinical course and slower recovery than the classic demyelinating form of GBS. A 24-year-old woman was admitted to the hospital in a comatose state, after suffering from fatigue and shortness of breath for 2-3 days. DKA was diagnosed on the basis of an increased blood glucose level, severe metabolic acidosis, and urinary ketone bodies, also on blood tests there were inflammatory changes, and electrolyte imbalance, on urine test was seen blood, leukocytes, bacteria suggesting urinary tract infection and autoantibodies were not detectable.

Our diagnosis was urinary tract infection, diabetes mellitus type 1 with ketoacidosis coma, depending on it used combined antibiotics, insulin, and artificial ventilation for repairing metabolic disturbance. Despite improvement of mental status and recovery from the critical state of ketoacidosis, dysphagia and muscle weaknesses had developed, which is worsened with respiratory failure. Autonomic dysfunction was manifested by increased heart rate, mild hypertension, decreased intestinal motility, and bladder dysfunction. The diagnosis of Guillain-Barré syndrome was made based on the nerve conduction study and cerebrospinal fluid analysis. In electromyoneurography, we found changes of GBS-AMSAN type, CSF chemistry – a combination of elevated protein levels and normal white blood cell count, which is termed cytoalbuminologic dissociation. Once GBS is diagnosed we started IV-IG treatment. No complications observed during immunoglobulin treatment, no obvious improvement was observed in the first days, and on the 7th day, increased muscle strength, and complaints connected with eyes were completely gone, muscle strength increased, and limb mobility was improved. During this period, heart rate decreased, arterial pressure stabilized, and fever was not observed. Although the association of GBS with DKA could not be completely excluded, we considered that DKA triggered the development of GBS in this case.

Conclusion: In cases of new onset DKA, patients should be evaluated carefully for GBS if there are clinical symptoms such as dysphagia, muscle strength loss, autonomic findings, and pain along with a previously known diabetic polyneuropathy.

Keywords: Diabetes type 1, Diabetic ketoacidosis, Guillain-Barré syndrome

Complete clinical characteristics of our patient and treatment timeline



Neuro-intensive care

Abstract:0021

ISCHEMIC STROKE FOLLOWING THORACIC POSTERIOR INSTRUMENTATION FOR PATIENT WITH ANKYLOSING SPONDYLITIS: CASE REPORT

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Case: A 52-year-old male patient with a known history of ankylosing spondylitis underwent thoracic posterior instrumentation operation due to kyphosis. On the development of weakness in the left arm and left leg of the patient 12 hours after the operation. On neurological examination; the patient opened his eyes slightly with a painful stimulus. He developed left facial paresis, left hemiplegia, motor and sensory aphasia. The National Institutes of Health Stroke Scale (NIHSS) was found 25 in the evaluation. The patient with a glasgow coma score of 8 was endotracheal intubated. No pathology was observed an urgent BT and cranial and cervical BT angiography examinations. No great vessel occlusion was observed. The patient was not considered suitable for intravenous thrombolytic therapy due to spinal surgery performed approximately 12 hours ago. In the diffusion MRI of the patient, there were diffuse hyperintense areas prominent in the right frontoparietal region, left frontoparietal region, and the left occipital region. Antiaggregant therapy was given to the patient who was not suitable for intravenous thrombolytic therapy and mechanical thrombectomy. After approximately 15 days of intensive care hospitalization, the patient was extubated and taken to the service. No cardioembolic etiology or any genetic disease was found in the studies conducted for the etiology of ischemic stroke. Ischemic stroke due to hemodynamic changes that may occur during the operation was considered. The patient continued antiaggregant therapy and physical therapy. In the neurological examination of the patient in the first month of the event, the comprehension was normal, speech was dysarthric, the left upper and lower extremity muscle strength was 4/5, and he was mobilized. The 1st month modified ranking score is 3.

Keywords: Ischemic stroke, ankylosing spondylitis, spine surgery

Metabolism - Endocrinology

Abstract:0024

THYROID STORM ASSOCIATED WITH COVID-19, COMPLICATED WITH DIABETIC KETOACIDOSIS: A CASE REPORT

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Introduction: Thyroid storm (TS) is a rare life-threatening health condition associated with undertreated hyperthyroidism. During thyroid storm, individual vitals can soar to dangerously high levels and without prompt, aggressive management, thyroid storm can be fatal. Diabetic ketoacidosis (DKA) is a trigger for thyroid crisis. However, TS and DKA rarely occur simultaneously. Here we are presenting a case of thyroid storm having Covid-19 at admission, complicated with Diabetic ketoacidosis (DKA).

Case: A 72-year female with a history of Diabetes mellitus (DM) was bought to the emergency room with symptoms of recurrent DKA. She was covid-19 positive at the time of admission but with minimal upper respiratory symptoms. DKA protocol was started according to our hospital policy, but she developed tachycardia, high fever, and a disturbing level of consciousness. After laboratory investigations revealed disturbing thyroid functions. So DKA with TS was diagnosed. Anti thyroid medication, inorganic iodine, and corticosteroids were then started as a treatment for TS, and b-blockers were administered to manage tachycardia. With these treatments, the patient's health improved, and she recovered.

Conclusions: In severe cases of recurrent DKA, the presence of TS should be considered, and early treatment should be initiated before the patient's condition worsens.

Keywords: Thyroid storm, Diabetic ketoacidosis, Covid-19, thyroid medications.

Neuro-intensive care

Abstract:0030

A CASE REPORT OF STIFF PERSON SYNDROME PRESENTING WITH QUADRIPLEGIA IN EMERGENCY MEDICINE DEPARTMENT

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Introduction: Stiff person syndrome (SPS) or Moersch-Woltmann syndrome is characterized by axial muscle rigidity, progressive stiffness, and spontaneous, reflex or action-induced painful spasms of the paraspinal, abdominal and occasionally proximal leg muscles associated with exaggerated lumbar lordosis.[1] Electrophysiological studies show continuous motor unit activity with abnormal exteroceptive reflexes with a normal interference pattern during spasms. Antiglutamic acid decarboxylase (GAD) antibodies in both serum and cerebral spinal fluid (CSF) with additional evidence of autoimmune disease are features of this syndrome.[1] SPS affects twice as many females as males. It is frequently associated with other autoimmune diseases such as t1DM, thyroiditis, vitiligo, and pernicious anemia.

Case: A 59 year old female from Western Indian origin who was a K/C/O atypical parkinsonism since last 3 months, on Rx, presented to the ED of a regional level institute with c/o b/l leg pain and difficulty in walking since a week, she was admitted, o/e she had hypertonia, hyperreflexia, MRI spine s/o multilevel desiccation, NCV s/o mixed myogenic and neurogenic changes; she suddenly developed c/o all four limb weakness and SOB at rest f/b altered sensorium, so she was intubated and shifted to ICU. Her routine blood investigations were unremarkable but Anti-GAD and c-ANCA was positive hence was diagnosed with SPS. She was initially treated with infusion of Diazepam, and clonazepam and baclofen but her condition was not improving so she was given 5 divided doses of IVIg a/c to her weight and drastic improvement was seen in her condition. Eventually, she was weaned off of ventilator and shifted to ward after 21days' stay of ICU.

Conclusion: Stiff person (or stiff man) syndrome is also known as Moersch-Woltmann syndrome. It has no genetic predisposition. There is insidious onset and it affects middle-age persons. It is characterized by persistent and intense spasms, particularly of the proximal lower limbs and lumbar paraspinal muscles with exaggerated lumbar lordosis. Stiffness is aggravated by noise, sensory stimulus and any movement. Stiffness disappears during sleep, with proximal nerve block, general anaesthesia and use of benzodiazepine. Antibodies against GAD are found in 65-70% cases. GAD is synthesizing enzyme for GABA (γ-aminobutyric acid) so antibodies against it results in decreased synthesis of GABA. The imbalance between the spinal inhibitory and excitatory input to alpha motor neurons results in continuous stimulation of motor neurons and stiffness of muscles. It may be confused with tetanus or strychnine poisoning or parkinsonism.

Keywords: Stiff Person Syndrome, Spasms, Stiffness, IVIG

Cardiovascular - Other

Abstract:0044

A SERIES OF COMPLICATIONS RELATED TO AN INADVERTENT ARTERIAL CANNULATION DURING CENTRAL VENOUS CATHETER PLACEMENT

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Introduction: Even with the use of real-time ultrasound, inadvertent arterial canulation remains a complication related to central venous catheter (CVC) insertion. We report a patient with a series of complications related to this unintended event.

Case: An 80-year-old female with a history of hypertension and hyperlipidemia presented with acute diverticulitis. CT scan showed colonic perforation with free fluid in the upper abdomen for which an urgent laparotomy was performed. During the surgery, she developed hypotension and required vasopressors, thus a CVC was inserted under real-time ultrasound guidance. The guidewire was noted to be in the right internal jugular vein and catheter placement proceeded. However, an arterial waveform was observed upon connection to the pressure transducer and the arterial placement was confirmed with blood gas analysis. The catheter was left in-situ after consultation with the vascular surgeon. CT angiogram showed through-and-through puncture of the right internal jugular vein to enter the right subclavian artery just anterior to the vertebral artery, the tip of the catheter was seen at the aortic arch.

Catheter removal was performed in the radiological suite. A percutaneous right subclavian artery covered stent was placed after balloon tamponade at the exit site was not successful.

Immediately after the procedure, the patient developed left sided weakness and slurred speech. CT brain performed showed acute infarct in the right MCA territory likely from an embolic event. It was possibly caused by the arterial manipulation during the stenting. She was thus brought back to the radiological suite for an emergent

endovascular thrombectomy. Procedure was successful with the establishment of TICI 3 perfusion.

Post procedure, patient's neurological status improved from NIHSS of 21 to 13. However, her renal function acutely deteriorated, likely contributed by contrast administration. Her creatinine level rose from 108 umol/L to 283 umol/L with oliguria. She was treated with aggressive hydration as well as blood pressure augmentation. Eventually, after 2 days of oliguria, her renal function started to recover and urine output returned to normal.

After a long course of stay in the ICU, she was weaned off all support and was eventually sent to the ward for further rehabilitation.

Conclusion: Even though uncommon, arterial canulation during CVC placement can lead to unintended complications that may lead to additional procedures and morbidity for vulnerable patients.

Reference

Mechanical complications after central venous catheterization in the ultrasound-guided era: a prospective multicenter cohort study. Adrian M, O Borgquist, Kröger et al. BJA 2022:129(6): 843-50.

Keywords: complications, central venous catheter, arterial cannulation

Perioperative critical care

Abstract:0049

A CASE REPORT OF ACUTE ARTERIAL EMBOLISM AFTER REPAIR OF GASTRIC ULCER PERFORATION

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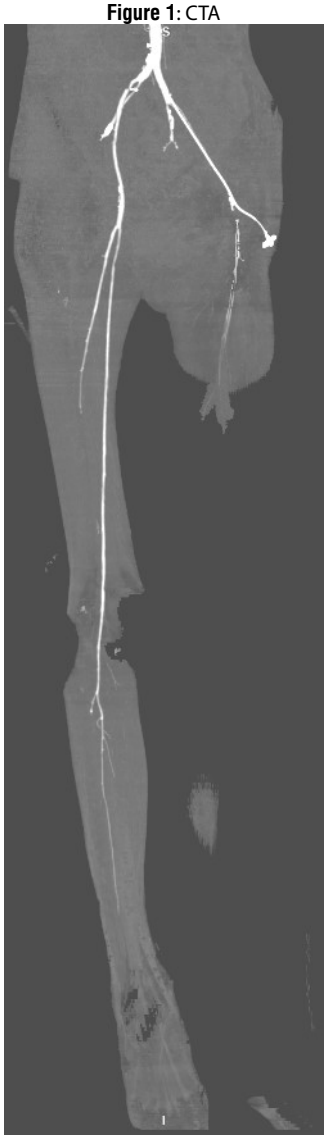
Case: A 64-year-old male presented with pale and cool skin on his left lower limb after repair of gastric ulcer perforation, local bluish and poor movement on his left lower leg. The pulse above the puncture point of the left femoral artery was ++; the pulse below the puncture point was not touched; the pulse of the left popliteal artery, posterior tibial artery and dorsal foot artery was not touched. Lower extremity arterial CTA: Multiple low-density foci and calcification foci were observed in the artery wall of both lower limbs, the wall was uneven, the lumen was narrow to varying degrees, the proximal lumen of the left superficial femoral artery was medium-severe stenosis, the distal lumen was blocked, the right superficial femoral artery segment was also significantly narrow, the left popliteal artery and the left anterior tibial artery, posterior tibial artery and peroneal artery segment were blocked. Multiple stenosis and occlusion of anterior tibial artery, posterior tibial artery and peroneal artery segments of the right calf. Femoral artery thrombectomy + femoral intimal exfoliation + repair was performed immediately.

Intraoperative Findings: there was no pulsation in the proximal, deep and superficial femoral arteries of the common femoral artery, local hematoma formation was observed in the common femoral artery, and a large number of dark red thrombus and the head of the mechanical thrombus were removed from the common femoral artery. Postoperative pulse of left femoral artery ++, pulse of left popliteal artery +, left dorsal pedis artery and posterior tibial artery were not touched. The skin colour temperature above the left knee was better than before, and the skin temperature below the knee was better than before. The skin colour was mottled with local necrotic spots and high tension. Postoperative sensorimotor restriction was considered for the left lower leg osteofascial compartment syndrome, and muscle fascial incision decompression plus wound closed negative pressure drainage was performed. The patient still had pale skin of the left lower limb accompanied by patellar cyanosis, obvious extremities, low skin temperature, grade 1-2 muscle strength, and dorsal foot artery beat was not touched. The left lower limb necrosis was diagnosed and amputation was performed. The patient finally recovered and was discharged.

Keywords: Acute arterial embolism, Perioperative period, Osteofascial compartment syndrome.

Figure 1: CTA



Multiple low-density lesions and calcifications were found in the arterial wall of both lower extremities. The arterial wall was uneven, and the lumen was narrowed to varying degrees.

Cardiovascular - Other

Abstract:0057

THE WIDOW MAKER - AN IMPORTANT PATTERN TO RECOGNISE ON THE 12-LEAD ECG

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Introduction: ECG interpretation is an essential skill for all emergency and critical care practitioners. The so-called widow maker pattern on the ECG, consisting of widespread ST segment depressions and

ST elevation in lead aVR is important to recognize. This pattern may signify major cardiovascular disease or other serious pathologies, or it may be indicative of more benign conditions. It is mandatory that every practitioner working with critically ill patients understand this ECG pattern and its meaning.

Case: A 50 y/o male patient c/o chest pain and SOB in his GP's room. The GP performed a 12 lead ECG suggestive of myocardial ischemia (widespread ST depressions). The GP then consulted a cardiologist who arranged direct admission to a coronary unit. An angiogram was arranged for the next day. Angiography revealed open coronary arteries but the patient deteriorated post angiography. The patient was pushed into the ED hypoxic with a low saturation despite application of a partial rebreather oxygen mask. Saturation was 66% and the patient was hemodynamically unstable - BP 60/30; HR 60/min; RR = 28. On follow up the patient's ECG showed an extensive RBBB pattern and on the point of care ultrasound observed a massively dilated RV. CPR with fibrinolytic therapy for massive pulmonary embolism was unsuccessful.

In this case, the ECG (see attached figure) displayed a widow maker pattern and additional features of a pulmonary embolus including a P-pulmonale, right axis deviation (RAD), prominent R-waves in aVR, V1 and V2, T-wave inversions in V1-V4 (right heart strain pattern) and an S1T3 pattern. When a widow maker pattern is present on the ECG the following conditions must always be ruled out:

Worrisome diagnoses:

LMCA occlusion (true widow maker)/ Acute proximal LAD occlusion / triple vessel occlusion

Thoracic aortic dissection

Massive pulmonary embolism

Massive gastro intestinal hemorrhage

Less worrisome diagnoses:

LBBB (expected discordant STE)

Left ventricular hypertrophy (LVH) with strain pattern and severe HT (resolves with BP lowering)

Severe atrial tachydysrhythmias (resolves with rate control)

Conclusions: In the above case, the ECG features (see figure attached) clearly demonstrated a widow maker pattern and features suggestive of a pulmonary embolus. This diagnosis could have been considered earlier in the management of this patient, had this pattern been fully understood by the treating clinicians.

Keywords: Left main coronary artery (LMCA), Left anterior descending artery (LAD), Tipple vessel disease, Massive pulmonary embolism

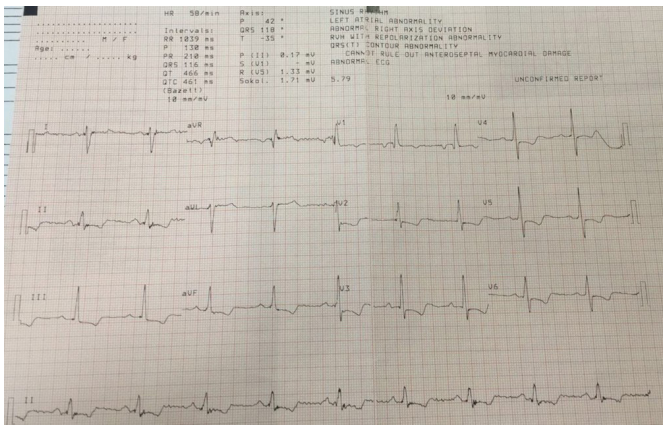

Figure 1. Widow maker ECG
ECG with widow maker pattern and additional features suggestive of a pulmonary embolism

Neuro-intensive care

Abstract:0060

AN UNUSUAL PRESENTATION OF SEROTONIN WITHDRAWAL SYNDROME – CLINICAL APPROACH IN DIAGNOSIS

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Introduction: Serotonin withdrawal syndrome, also known as serotonin discontinuation syndrome or SSRI withdrawal syndrome, is a constellation of symptoms that can occur when a person stops taking selective serotonin reuptake inhibitors (SSRIs) or other medications that increase serotonin levels in the brain. Depression is a common entity in today's world of virtual life we live in, it is estimated that 1 in 20 persons has depression 10. With increasing prevalence in general community, it is important to mention here the number of patients admitted to ICU for any other reasons who can have past medical history of depression and on some or other type of antidepressants. Hence a careful medical history with a complete medication history must be documented. This article aims to provide a general understanding of this condition, describing how we have come across a patient in an unusual presentation post recovery from his status epilepticus masking diagnosis as restless and agitation secondary to anoxic brain injury overlooking the current medications he was taking, delayed in diagnosis leading to prolonged ICU stay, outlining how we have managed the case and discharged the patient.

Case: Taxi driver was brought by ambulance in an unconscious state, with an unknown past medical history, treated as status epilepticus >1 month in ICU difficulty to wean due to recurrent restlessness requiring high sedations, and tracheostomy and PEG tube were performed. Readmitted to ICU with worsening GCS, increasing restlessness agitations. Consulted for a second opinion from another Neurologist. He was treated with Levetiracetam 1.5g BD/ Lamotrigine 400 BD/ Perampanel8 mg OD/ Clonazepam 4 mg TDS /Diazepam 10 TDS / Olanzapine 5mg BD/ Clonidine 150mcg TDS/Precedex infusion/Seroquel 200 BD/Gabapentin 400 TDS/ Chlorpheniramine 4 mg HS. In physical examination, eyes were closed and all limbs were moved restlessly. Upon careful clinical examination it showed on maximum restrained gentleman who was hyper-restless diaphoretic moving all limbs erratically with superimposed myoclonic jerks not fixing. Gaze was not redirectable, he did not follow any commands and, pupils were not focused equally and fully dilated. Reviewed medication all signs and history were carefully done came to a clinical diagnosis of serotonin withdrawal syndrome. Following this he was able to get discharged from hospital within 1 month of starting his old medication SSRI.

Conclusion: Serotonin withdrawal syndrome is now interchangeable with serotonin discontinuation syndrome as part of antidepressant discontinuation syndrome (ADDS). The very early stage would have prevented the patient from slipping into serotonin discontinuation syndrome.

Keywords: Serotonin discontinuation syndrome, ICU delirium, anoxic brain injury

Gastrointestinal system and Liver

Abstract:0071

TOXIC EPIDERMAL NECROLYSIS WITH SEVERE LIVER FAILURE

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Introduction: Toxic epidermal necrolysis (TEN) is life treating skin reaction characterized by high fever, epidermal detachment, and mucositis. TEN occasionally affects various organs, leading to permanent damage. Although liver dysfunction is a relatively common complication of TEN, severe acute liver dysfunction requiring liver transplantation is rare. This case report aims to present TEN complicated by severe and rapidly progressive liver dysfunction, specifically, acute liver failure (ALF) requiring liver transplantation (1).

Case: A 23-year-old male patient was admitted to the emergency department with extensive erythematous lesions (extensive confluent erythema, multi-side mucositis) and icterus. He had no comorbidities or drug use before. According to patient's history, three days after an insect bite, erythema and diarrhea began. He was referred to our hospital due to high liver enzymes and hyperbilirubinemia. Lesions were widely present on the anterior aspect of his trunk, arms and face. The lesional skin was tender to the touch. Nikolsy's sign, in which lateral pressure causes lesional detachable epidermis to slide over the dermis, was positive. His vital signs were normal except for sinus bradycardia. He had severe liver function test abnormalities (Figure 1), his liver ultrasonography was normal with normal gallbladder dimensions, but the wall was diffusely thick and edematous. The results of other laboratory tests for differential diagnosis were normal. Pulse steroid (Methylprednisolone 250 mg/daily and immune globulin iv 3 g/kg) treatments were immediately started and given for three days. As the lesions and liver function tests slightly regressed after those treatments, cyclosporin therapy was started. In the course of care, the patient's lesions regressed further (figure 1) but despite all hepatoprotective approaches and therapies, liver failure persisted. After clinical improvement, the patient was transferred to transplantation unit for liver transplantation.

Conclusions: In this case, TEN may be triggered due to an unknown insect bite. Although the liver dysfunction associated with TEN does not generally influence the mortality; liver dysfunction of this patient was quite severe. However, there are rare TEN cases of severe liver dysfunction requiring liver transplantation (2). Therefore, it is important to evaluate the liver functions of TEN patients continuously to consult a gastroenterologist on time.

References

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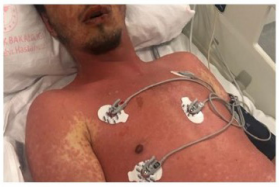
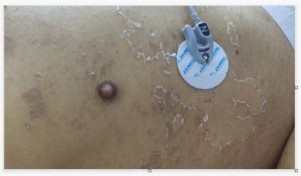
Keywords: toxic epidermal necrolysis, liver failure, mucositis



Before treatment



After treatment

Table 1. Liver function tests of the patient

	ALT U/L	AST U/L	ALP U/L	GGTU/L	DB mg/dL *	IBmg/dL **	INR	ALBg/L ***
DAY 1	1769	1632	159	81	5.49	1.0	1.93	35
DAY 7	75	60	162	70	11.2	2.74	1.48	18
DAY14	103	106	267	99	14.9	4.94	1.38	26

*DIRECT BILIRUBIN, **INDIRECT BILIRUBIN, ***ALBUMIN

Figure 1. The patient's lesions and liver function test results

Brain death and organ donation

Abstract:0075

HEART TRANSPLANTS FROM CONTROLLED DONATION AFTER THE CIRCULATORY DETERMINATION OF DEATH (CDD): OUR INITIAL EXPERIENCE

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Introduction: Heart transplantation from controlled donation after the circulatory determination of death (CDD) could improve the availability of heart for transplantation, reducing the waiting list. At Hospital Virgen de la Arrixaca in Murcia, we have performed 13 transplants from CDD between January 2020 and March 2023. Our aim with this study was to review the evolution of the patients receiving hearts from CDD, including the need for extracorporeal or vasoactive support after surgery and mortality during their hospitalization.

Cases: We included 13 patients, 69% (9/13) of them were men, who received heart transplantation after CDD between January 2020 and March 2023. The extraction surgery was performed after CDD at the operating room, with cardiac resuscitation after supraaortic vessels were clamped and the thoracoabdominal normothermic regional perfusion (TA-NRP) was started, recovering sinus rhythm after a few minutes of TA-NRP. After surgery, we registered the implant function and the need for extracorporeal and vasoactive support on arrival, at 24 hours, and in the first two weeks in the Intensive Care Unit (ICU). We also registered the total ICU stay, complications and mortality during ICU and hospitalization stay. After that, we used SPSS software to analyze and study the results.

The average stay at ICU was 8,31 (SD 7,73) days with a median of 5 days. The left ventricular function (evaluated with LVEF) was normal at the arrival in 76% (10/13) patients, becoming normal in 92% (12/13) in the first week and in all of them in less than two weeks. Only one of them required extracorporeal support at the arrival at ICU during less than a week because of a primary disfunction of the graft, with favorable evolution after it. We also rated the right ventricular function according to the need of dobutamine during their

stay in ICU. 92% (12/13) patients required dobutamine at arrival at ICU, being able to withdraw it in less than a week in 84% (11/13). 23% (3) of them required surgical reintervention, another 23% (3) of them required re-entry in ICU. There were 2 deaths (15%) during the hospitalization stay, one of them because of pericardial effusion and the other one because of an interstitial pneumonia.

Conclusion: The patient's evolution from heart transplantation after CDD is favorable, most of them with normal biventricular function in the first week after surgery. Thus, it can be considered as an optimal way to improve the availability of donors for heart transplantation.

Keywords: Transplants, Heart, Circulatory determination of death.

Respiratory - Other

Abstract:0092

SEVERE CHRONIC OBSTRUCTIVE PULMONARY DISEASE EXACERBATIONS IN ICU IN PATIENT WITH SCOLIOSIS (A CLINICAL CASE)

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Introduction: Chronic obstructive pulmonary disease (COPD) remains one of the major causes of mortality worldwide. Exacerbations are often the cause of mortality in patients. The ICU management must be individualized for each patient and guided by symptoms, exacerbations, pulmonary functions and comorbidities.

Case: A retrospective analysis of a clinical case of a patient hospitalized in ICU. A 56 year old male, diagnosed with acute exacerbations COPD was admitted to the ICU for treatment. APACHE score was 21 points, SOFA score was 11 points. His respiratory frequency >28 breaths/min, acute respiratory failure with severe hypoxia, persistent respiratory acidosis, (arterial blood gas analysis: ph=7.36, PCO2=77,2 mmHg, PO2=86,2 mmHg, HCO=42,8 mmol/L, BE=14 mmol/L, ctCO2=45,2 mmol/L, Hct=38%, tHb=12,8g/L, PaO2/FIO2=1.91) WBC>26.0x10^9/L. procalcitonin level 40,7 ng/ml, altered mental status, hemodynamic instability (hypotension).

Intensive treatment was initiated: early evaluation of severity, ventilatory support (PEEP↑, Vt↓, PΔ), application of tracheostomy in prolonged mechanical ventilation, the guidance of antibacterial therapy using procalcitonin/microbiological cultures, the screening of nutritional condition of patient (nutritional risk screening 2002).

The patient recovered after 61 days of mechanical ventilation and on the 64th day was able to be discharged from ICU.

Conclusions: The factors which suggest a poorer prognosis include low score Glasgow Coma Scale, or multi-organ impairment indicated by high APACHE II score. For these reasons, placement in ICU with mechanical ventilation can improve mortality outcomes. So, this case represent the importance of early diagnosis, protective ventilation, the alveolar recrutation and effective treatment care of critical patient. This patient experienced clinical and oxygenation improvement with medical therapy, mechanical ventilation. Management in ICU had positive role over prognosis, with patient`s survival.

Keywords: chronic obstructive pulmonary disease, bronchoscopy, tracheostomy, protective ventilation,

Respiratory - Other

Abstract:0097

SUCCESSFUL VENOVENOUS ECMO ADMINISTRATION IN TYPE I RESPIRATORY FAILURE OF UNKNOWN CAUSE

Rabia Sarı Küçük, Duran Karakuş, Namigar Turgut

University of Health Sciences, Prof Dr Cemil Taşcıoğlu City Hospital, Depatment of Intensive Care, Istanbul, Turkey

Introduction: Extracorporeal membrane oxygenation (ECMO) is a treatment method which provides lung (venovenous) and/or cardiac (venoarterial) support in patients who don't respond adequately despite supportive treatments.

Case: A37-year-old male patient with BMI 43 was brought to the emergency room unconscious after drinking alcohol. Patient's Glascow Coma Scale was 7 and sPO2 85, pH 7.18, PCO2 72 mmHg, pO2 53.5 mmHg, HCO3 26.5 in arterial blood gas (ABG) under 8 lt/min oxygen via face mask. He was intubated orotracheally. Cardiac arrest developed during intubation. After five minutes of cardiopulmonary resuscitation, spontaneous circulation was achieved and the patient was taken to the intensive care unit. Mechanical ventilation was started with a fractioned of inspired oxygen (FiO2) 80% and PEEP 8 cmH2O with SIMV, and pH 7.23, PCO2 64.9 mmHg, pO2 113.7mmHg, HCO3 26 on ABG. PEEP titration was applied. Since the static compliance of the patient was found ≥50 cmH2O and P/F (pO2/ FiO2) was decreased with PEEP of ≥10 cmH2O, the PEEP was kept at 10 cm H2O. Neuromuscular blocker infusion and steroids were started. The patient couldn't be placed in prone position due to obesity. Intracranial pathology, lung infiltration or pulmonary embolism were not detected in the radiological studies. The patient's P/F was 88 with 80% FiO2 on the 3rd day of hospitalization. FiO2 increased to 100%. Since the P/F was ≤70 under 100% FiO2 on the 4th day of hospitalization, VVECMO was started. Respiratory viruses were negative in the viral panel. There was no positivity in tracheal aspirate cultures. Patient was followed up for 14 days without any decrease in support. Weaned from ECMO after P/F 120 was detected on day 14. Tracheostomy was performed on the 24th day of hospitalization. He was consciously taken to pressure support ventilation on the 27th day. He was followed up with spontaneous breathing on the 30th day and the tracheostomy was closed. He was discharged from the intensive care unit conscious and mobilized.

Conclusion: Although our patient was severely hypoxic, diffuse bilateral opacity was not detected and vessels were clear in radiological lung imaging. Compliance was normal. Cardiac function and pulmonary artery pressure were normal in the echocardiography. Chemical pneumonitis secondary to ethyl alcohol aspiration can be suspected when considering the loss of consciousness, cardiac arrest and intubation. Despite the fact that the etiology is unknown, our patient underwent an early and simple ECMO application and received supportive care before being discharged.

Keywords: Ethyl alcohol aspiration, extracorporeal membrane oxygenation, type I respiratory failure

Gastrointestinal system and Liver

Abstract:0124

A SEVERE CASE OF MIXED SHOCK AND MULTIPLE ORGAN FAILURE AFTER ELECTIVE LIVER TRANSPLANTATION

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Introduction: Liver transplant is one of the therapeutic tools that improve mortality in patients with terminal liver disease. This surgery has high morbidity and mortality, among other complications related to liver failure such as vascular problems, primary dysfunction of the graft and acute rejection.

Case: A 64-year-old female with a history of multifocal liver hemangioendothelioma was admitted for an elective liver transplant with a live donor (her daughter). During surgery her hepatic artery got obstructed five times due to thrombosis, she required massive fluid administration and was started on vasopressors (Norepinephrine). After 14 hours of surgery, she was transferred to the intensive care unit (ICU), at the time of admission she was still receiving a norepinephrine drip (0.11 ug/kg/min), on examination her capillary refill time was 6 seconds, and her laboratory was notable for a lactate > 20 mmol/lt, pH 7.2 and bicarbonate 12.5 mEq/lt. She was reanimated with ringer lactate and albumin 5% according to a preload dependency strategy, trying to avoid raising the norepinephrine dose to prevent a new occlusion of the hepatic artery. Patient remained vasoplegic despite aggressive resuscitation according to Swan Ganz catheter, so vasopressin was added, and she was started on high volume hemofiltration (HVHF), at 70 ml/kg/hr of ideal body weight, pre filter, in an attempt to stop the inflammatory cascade. There was a notorious decrease in norepinephrine dose requirements along with lower lactate levels, but patient presented with worsening cardiac function. Initially, milrinone was started but promptly changed to adrenaline due to the predominant vasoplegic state. After 22 hours of being admitted to the ICU, most of her clinical and analytical trends improved. Nevertheless, she went into septic shock due to a catheter-related bloodstream infection, norepinephrine was administered up to 0.8 ug/kg/min, which led to an acute liver failure and the need for a second liver transplant.

A second liver transplant was performed successfully, and after 4 months of multidisciplinary rehabilitation, she was discharged to her home.

Conclusion: We are showing a case of severe liver dysfunction after an elective liver transplant due to a vascular complication. The patient was refractory to most therapies, showing no improvement with modulation therapy (HVHF). There is no gold standard therapy for this type of complication, but invasive hemodynamic monitoring was useful to lead therapy.

Keywords: liver transplant, liver failure, shock, high volume hemofiltration.

Neuro-intensive care

Abstract:0130

ACUTE DISSEMINATED ENCEPHALOMYELITIS SECONDARY TO *ENTEROCOCCUS FAECALIS*. A COMBINATION NOT DESCRIBED IN THE LITERATURE

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Introduction: Post-infectious acute disseminated encephalomyelitis (ADEM) is a demyelinating disease of the central nervous system (CNS), mostly secondary to viruses, although post-vaccination, bacterial and parasitic etiologies have also been described. It commonly occurs in children under 10 years of age and young adults, with an estimated prevalence of 0.3 to 0.8 per 100,000 inhabitants, manifesting with multifocal neurological symptoms and progressive encephalopathy during the first three months of the disease, associated with evidence of focal demyelination in neuroimaging.

In the last 30 years, *Enterococcus* have become the third most frequent cause of nosocomial infectious agents but CNS involvement by enterococcal infection is infrequent, representing between 0.3% to 4% of bacterial causes. In the reported cases of enterococcal infection of the CNS in adults, the mean age of the spontaneous events is 57 years, with a prevalence of *Enterococcus faecalis* infections over faecium in a 3:1 ratio. Most patients present with fever (85%), meningeal signs (44%), altered state of consciousness (44%), headache (36%) and seizures (20%), with neurological sequelae in up to 23% and mortality associated with Enterococcus CNS infections at 26%.

Given the low incidence of these two pathologies, as well as the absence in the current medical literature of a description of their coexistence, it was decided to report the present clinical case.

Case: A 42-year-old man, chronic renal patient on peritoneal dialysis, was consulted for headache and compromised consciousness. He presented with fever and positive cerebrospinal fluid culture for Enterococcus faecalis, initiating ampicillin plus linezolid. Given the poor clinical evolution, requiring ICU and prolonged mechanical ventilation, an MRI complemented the study, showing a focus on cerebritis which was biopsied reporting a result compatible with acute disseminated encephalomyelitis (ADEM), so steroid therapy was started, and the patient responded favourably.

Conclusion: Nonspecific infectious symptoms associated with consciousness compromise is the classic presentation described of spontaneous meningitis due to Enterococcus in adults. Therefore, we suggest that in patients with risk factors for Enterococcus infections, active suspicion and management should be maintained according to the diagnostic possibility.

The identification of ADEM is central to the prognosis of these patients, who without opportune treatment have a rate of neurological sequelae up to 45% and death between 8% and 25%. In this patient, recovery occurred after day 5 of corticosteroids therapy, which is the mainstay of treatment of this pathology.

Keywords: Acute Disseminated Encephalomyelitis, Bacterial Meningitis, Enterococcus, Encephalitis

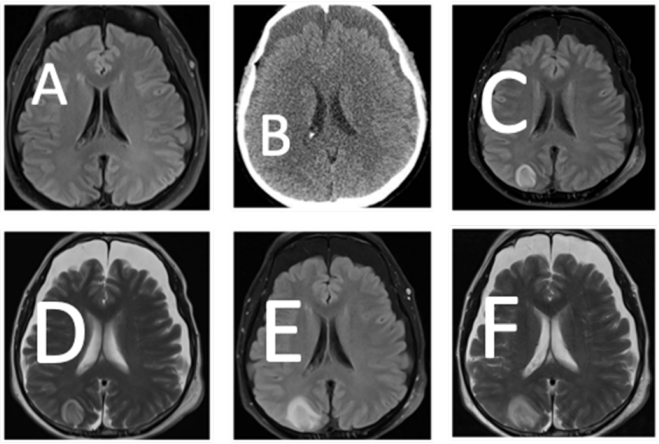

Figure 1. Brain Images, evolutive changes of ADEM.
A) admission MRI without lessions B) Day 6 CT.without lessions C / D) Day 17 MRI. Right occipital occipital corticosubcortical lesion with high T2/FLAIR signal, low T1 signal, without water diffusion restriction except for the adjacent parenchyma showing a laminar diffusion restriction band (measuring approx. 20x22mm in axial plane). E / F) Day 21 MRI: Increased size of right occipital corticosubcortical lesion compared to day 17 study, in front of an area of cerebritis, also showing discrete increase of signal in diffusion with less signal in ADC maps. Discrete increase of the local mass effect.

Hematology and Oncology

Abstract:0132

MACROAGGLUTINATION AS CAUSE OF DEATH. WHEN VIRCHOW'S PERFECT STORM STRIKES

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Introduction: Since Rudolf Virchow in 1856, the coexistence of blood stasis, endothelial damage and hypercoagulability are the axes of analysis of all coagulation phenomena. Causes of these three elements have been progressively added in the literature, but little remains to be done when the coexistence of causes becomes an unmanageable procoagulant avalanche. The predisposing factors are particularly frequent in intensive care patients.

Epidemiologically, thromboembolic disease has an incidence up to 10% in ICU patients and 45% in patients on extracorporeal support (ECMO). SARS COV2-related disease (COVID) was associated with an increased incidence of thromboembolic disease in hospitalized patients of 11%.

As the maximum representation of hypercoagulability macro hemagglutination has been neglected in clinical reports, it is our aim to reconsider it.

Case: A 65-year-old woman with diffuse large B-cell lymphoma undergoing active chemotherapy was admitted to the hospital due to respiratory failure caused by COVID pneumonia requiring orotracheal intubation, invasive mechanical ventilation, and prone position. Chest CT angiography was performed, confirming multilobar pulmonary thromboembolism. Anticoagulation was initiated, and fibro bronchoscopy revealed invasive pulmonary aspergillosis.

Despite these interventions, the patient developed progressive multiorgan failure, and on the twelfth day of hospitalization, the decision was made to connect her to veno-venous ECMO while maintaining anticoagulation within the appropriate range. The hematology unit reported progressive red blood cell agglutination phenomena at the microscopic and macroscopic level.

On the eighteenth day of hospitalization, the patient developed a radial artery thrombus, despite appropriate anticoagulation, requiring surgical management. Additionally, the patient presented with persistent increases in free haemoglobin up to 30 mg/dl.

The patient experienced new thromboembolic phenomena in the contralateral extremity and new hepatic and hemodynamic dysfunctions. A CT scan of the chest, abdomen, and pelvis was performed, revealing new multisystemic arterial and venous thromboembolism in splenic, hepatic, renal, and pulmonary territories.

Conclusion: The macro hemagglutination was the pre-warning of the potential for a complicated clinical course, with the convergence of multiple factors contributing to the patient's condition. These included the procoagulant state of an onco-hematological disease, the disease-binding effect of COVID-19, endothelial disruption leading to sepsis-induced inflammation, and hemolysis from mechanical devices with the release of haemoglobin, among others.

These factors suggested a loss of the normal balance between pro- and anticoagulant effects that are typically maintained in healthy individuals, leading to a disruption of hemostatic autoregulation. This disruption triggered a cascade of cross-reactions resulting in generalized thrombosis, which ultimately led to irreversible and untreatable systemic deterioration.

Keywords: Erythrocyte Aggregation, Embolism and Thrombosis, Extracorporeal Membrane Oxygenation, Lymphoma

Perioperative critical care

Abstract:0133

HEMORRHAGIC SHOCK FOLLOWING COLONOSCOPY: WHEN THE SPLEEN HIDES THE ANSWER

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Introduction: Post-colonoscopy shock is a rare complication that typically arises from post-procedure bacterial translocation, intestinal perforation, or bleeding colonic injury. These conditions are usually considered diagnostic alternatives during the post-procedure management of patients.

Among the infrequent causes of post-colonoscopy shock, splenic lesions without intestinal perforation are a complication that can go unnoticed until phases of clinical instability. This complication requires surgery in 75% of patients and has a mortality rate of 20%, according to nearly 100 cases published in the literature.

We present the clinical case of a patient with rapidly evolving shock, intensive care management, and emergency splenectomy due to splenic rupture after colonoscopy.

Case: A 64-year-old woman, who was being studied for chronic diarrhea due to suspected inflammatory bowel disease-like syndrome after immunotherapy, was admitted for study. Three hours after

colonoscopy, she experienced intense abdominal pain, tachycardia, and progressive hypotension with partial response to volume, and was admitted to the intensive care unit. She rapidly progressed to shock associated with anemia. Abdomen and pelvis CT angiography revealed splenic rupture with active arterial bleeding, which was treated with emergency splenectomy. The focus of bleeding was resolved, and the patient's condition subsequently stabilized.

Conclusion: Hemorrhagic shock due to splenic lesion after colonoscopy is an infrequent entity with high lethality, given the low clinical suspicion with which it is pursued in patients with signs of complications after the procedure. This can reduce the possibility of less invasive management than surgery, such as control by interventional radiology.

Of the nearly 100 cases of this complication published in the literature, a predominantly female population is described, between 50 and 70 years of age, with the presence of collagenopathies as an associated clinical history, like the case described for our patient. Therefore, this group should be given greater suspicion in the presence of abdominal pain, tachycardia, and hypotension after the procedure.

Keywords: Shock, Hemorrhage, Colonoscopy, Splenic Rupture

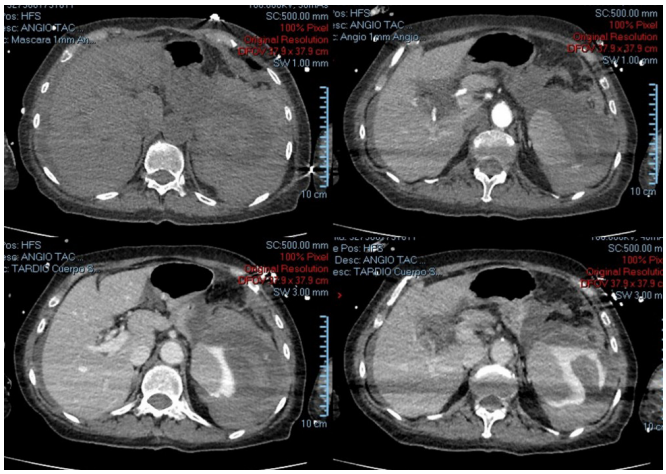

Figure 1. Splenic rupture
Computed tomography of the abdomen with splenic rupture and active bleeding.

Critical care pharmacy and drug monitoring

Abstract:0137

USE OF MIDODRINE FOR TREATMENT OF HYPOTENSION: CASE REPORT

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Introduction: Patients admitted to intensive care unit (ICU) often require intravenous vasoactive drugs to maintain normotension or other clinically indicated blood pressure targets. In hypotensive patients without impairment of tissue oxygenation, there is a need for the use of oral agents that could facilitate weaning from intravenous vasopressors and assist in earlier discharge.

Midodrine, an oral α1-adrenergic agonist, received accelerated approval by the U.S. Food and Drug Administration (FDA) in 1996 for the treatment of symptomatic orthostatic hypotension. In this case report, the use of midodrine in the treatment of hypotension in a patient hospitalized in the ICU was evaluated.

Case: A 54-year-old woman with a known diagnosis of schizophrenia is admitted to ICU of a university hospital with confusion and suspicion of intoxication. Because the patient had hypotensive values, vasopressor (norepinephrine) support was started. Vasopressor requirement varied between 0.01-0.5 mcg/kg/min throughout the patient's hospitalization. Midodrine was recommended by the clinical pharmacist to the patient due to the increase in the patient's need for norepinephrine and given to the patient as 2*2.5 mg/day. While midodrine was given, 0.1 mcg/kg/min of norepinephrine was continued to be given to the patient. 4 days after starting midodrine, the patient's norepinephrine requirement decreased to 0.03 mcg/kg/min. On the eighth day, the patient's norepinephrine was stopped and midodrine was increased to 2*5 mg/day. During this period, the patient did not need vasopressor therapy. On the fourteenth day, the dose of midodrine was reduced to 2*2.5 mg/day because the patient's blood pressure was within the normal range and the patient was transferred to ward.

Conclusions: As a result, there is insufficient and low-quality evidence to support the use of midodrine in the treatment of orthostatic hypotension. The use of midodrine should be considered in the treatment of patients who are vasopressor unresponsive and have orthostatic hypotension according to the clinical experience of the physicians.

Keywords: Midodrine, hypotension, vasopressors

Infections and antimicrobials

Abstract:0139

INCREASE OF SERUM CREATINE KINASE AND MYOGLOBIN LEVELS DUE TO CEFOPERAZONE/SULBACTAM TREATMENT IN THE MULTIDRUG-RESISTANT *ACINETOBACTER BAUMANNII* LUNG INFECTION: A CASE REPORT

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Introduction: *Acinetobacter baumannii*, which has developed resistance to many conventional treatments, is a major cause of nosocomial infection, particularly in critically ill patients. Carbapenems, polymyxins, tetracyclines, aminoglycosides, fluoroquinolones and combination therapies containing the sulbactam have been used to treat *A. baumannii* infection. In this case report, the increase of serum creatine kinase and myoglobin levels due to cefoperazone/sulbactam treatment in a woman patient with nosocomial pneumonia led to *A. baumannii* is presented.

Case: A 74-year-old female patient with chronic obstructive pulmonary disease, type 2 diabetes and hypertension was admitted due to dyspnea, blood gas disturbance and confusion to the Anesthesiology Intensive Care Unit at Hacettepe University Hospital. Piperacillin-tazobactam and doxycycline treatment were started in terms of bacterial infection due to the increased oxygen demand and acute phase reactants in the follow-up. Due to increased infiltration on chest X-ray and multidrug-resistant *A. baumannii* growth in deep tracheal

aspiration culture despite empirical antimicrobial therapy, the current treatment was discontinued and a combined treatment regimen with colistin (loading dose: 300 mg; maintenance dose: 70 mg every 12 hours) and cefoperazone/sulbactam (cefoperazone 1 gr / sulbactam 1 gr: every 12 hours) was started (Glomerular filtration rate 7.33 mL/dk/1.73m2). On the 3rd day of colistin and cefoperazone/sulbactam treatment, an increase in serum creatine kinase and myoglobin levels was observed. Then, the cefoperazone/sulbactam treatment was discontinued and ampicillin/sulbactam (ampicilin 2 gr / sulbactam 1 gr: every 24 hours) treatment was started. With the discontinuation of cefoperazone/sulbactam treatment, the patient's creatine kinase and myoglobin levels returned to normal (Figure1). In addition, patient's concurrent drug treatment with salbutamol, budesonide, ipratropium bromide, metoprolol, furosemide, pantoprazole, dexamethasone, midazolam, fentanyl, acetyl salicylic acid, clopidogrel and heparin was found not to be associated with the adverse event, after an extensive review and assessment by a clinical pharmacist.

Conclusion: In our case, the increase in serum creatine kinase and myoglobin levels was associated with cefoperazone, which was included in the cefoperazone/sulbactam combination therapy. Although the patient's *A. baumannii* infection treatment was continued with ampicillin/sulbactam, there was no increase in serum creatine kinase and myoglobin levels. This information confirms our hypothesis. The possible mechanism of this observed adverse effect is unknown, as no case reports or studies have been published on this subject before. It is important to monitor patients receiving high dose cefoperazone/sulbactam therapy in terms of serum creatine kinase and myoglobin levels, and more studies are needed to evaluate this relationship.

Keywords: Acinetobacter baumannii, cefoperazone/sulbactam, creatine kinase, myoglobin

Cardiovascular - Cardiovascular dynamics

Abstract:0142

"TOO CLOSE FOR COMFORT" – CARDIAC TAMPONADE FROM RETROSTERNAL HEMATOMA – A RARE COMPLICATION OF CHEST COMPRESSIONS

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Introduction: Cardiac tamponade is a form of obstructive shock with high mortality rates (1). It is typically from the accumulation of fluid in the pericardial space but can also be caused by extrinsic compression. Chest compressions during cardiopulmonary resuscitation (CPR) are frequently associated with complications such as thoracic skeletal injuries (2) – and a retrosternal hematoma is a less known but potentially lethal complication.

We describe a patient with out-of-hospital-cardiac-arrest (OOHCA) whose clinical course was complicated by retrosternal hematoma causing cardiac tamponade.

Case: Our patient is a 71-year-old male with a history of diabetes and ischemic cardiomyopathy with ejection fraction (EF) of 35%. He was brought into the ER with witnessed OOHCA. CPR was commenced by the ambulance crew and return of spontaneous circulation (ROSC) was achieved 10 minutes in. Post-ROSC electrocardiogram showed possible inferoposterior ST-elevations and a point-of-care cardiac ultrasound (POCUS) demonstrated EF of 20% and global hypokinesia. He was brought for coronary angiography which showed triple vessel disease but no culprit lesion and the current impression at this point was cardiac arrest of likely arrhythmic etiology. Hemodynamics at this point were stable – with a blood pressure (BP) of 140/80mmHg and heart rate (HR) of 80.

4 hours later, there was abrupt deterioration of his hemodynamics with BP 80/55mmHg and HR 100, requiring support with noradrenaline and vasopressin. A repeat POCUS at this point demonstrated a lentiform dense mass with extra-pericardial compression of the right ventricle.

An urgent CT (computed tomography) confirmed the presence of a retrosternal hematoma and bleeding from the right internal mammary artery. Surgical evacuation of the hematoma was performed – and there was immediate improvement of hemodynamics post-operatively. The patient was subsequently weaned off vasopressor/inotropic support, and successfully extubated 2 days post-op, with good neurological recovery.

Conclusions: There exist multiple etiologies for unexplained hypotension in the post OOHCA patient – and our case demonstrates that cardiac tamponade from retrosternal hematoma due to chest compressions is a potential rare cause of unexplained hypotension. Timely bedside focused echocardiography and cross-sectional imaging allows rapid diagnosis and management of such patients.

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Keywords: hypotension, echocardiogram, tamponade

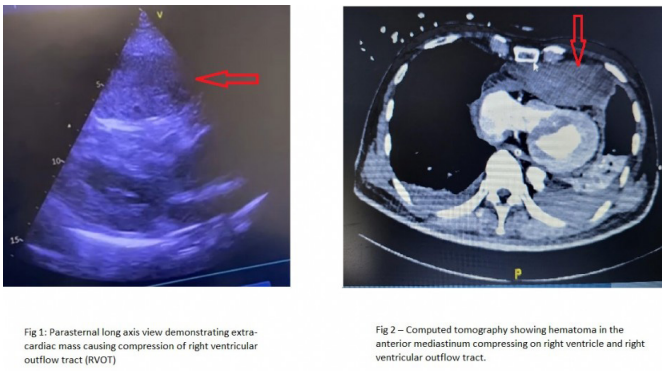

Fig 1: Parasternal long axis view demonstrating extra-cardiac mass causing compression of right ventricular outflow tract (RVOT)

Fig 2 – Computed tomography showing hematoma in the anterior mediastinum compressing on right ventricle and right ventricular outflow tract.

Figure 1. Ultrasound and CT images of retrosternal hematoma

Critical care pharmacy and drug monitoring

Abstract:0144

POLYMYXIN B-ASSOCIATED NEUROTOXICITY: A BRIEF CASE REPORT

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Introduction: Polymyxin B and colistin (colistimethate), as a final line of defense against extensively drug-resistant pathogens. Administration of intravenous (IV) polymyxin B and colistimethate can potentially induce nephrotoxicity, neurotoxicity, and allergic reactions in patients. Patients may experience a different set of symptoms

such as paresthesia (tingling or pricking sensation), diplopia (double vision), weakness and respiratory failure. In this brief case report, we documented a case of polymyxin B-associated neurotoxicity.

Case: A 24-year-old female without a chronic disorder was trapped under rubble for 9 hours during the earthquake that occurred in Hatay-Turkiye on February 6th, 2023. The patient was referred to our university hospital from an external medical facility and admitted to intensive care unit of the internal medicine department. Accordingly, meropenem treatment was started due to the use of antibiotics from an external center, unknown culture growths and a preliminary diagnosis of intraabdominal infection. Acinetobacter baumani was observed in the aerobic culture obtained from the peritoneum. Therefore, tigecycline (daily 200 mg IV loading dosage followed by 100 mg twice daily maintenance dose were planned) and polymyxin B (daily 25000 IU/kg IV loading dosage followed by 15.000 IU/kg twice daily maintenance dose was planned) were added to the treatment of the patient. Concomitant medications were heparin, tigecycline, meropenem, pantoprazole and paracetamol while initiation of polymyxin B. According to Lexicomp software system, no significant drug-drug interactions were identified between medications.

The patient had no hepatic impairments. However, a creatinine value of 5.29 mg/dL and a glomerular filtration rate of 10.44 ml/min/1.73m2 were documented on the first day of the medication.

Conclusion: Following the administration of polymyxin B infusion, the patient reported numbness and tingling in her hands, face, and head. The second dose of polymyxin B was not administered, and colistimethate treatment was initiated while all concomitant medications were continued. Colistimethate was initially dosed at 300 mg, followed by 65 mg twice daily, and plus 40 mg on the days the patient received hemodialysis. After the switch to colistimethate, the patient reported a simultaneous decrease in numbness and tingling symptoms, as documented by the patient. Also, the total score on the Naranjo Scale was 7 and classified as "possible".

In conclusion, healthcare professionals should be aware of the occurrence of neurotoxic symptoms and risk factors in patients undergoing treatment with polymyxin B. Immediate discontinuation of treatment upon recognition of such symptoms is critical to avoid further neurological damage.

Keywords: polymyxin B, neurotoxicity, paresthesia

Sepsis - Management

Abstract:0160

LEPTOSPIROSIS PRESENTING WITH MULTIPLE ORGAN FAILURE: A DIAGNOSTIC CHALLENGE

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Introduction: Leptospirosis is a zoonotic infection caused by spirochetes of the genus Leptospira. While 90% of the patients have non-icteric asymptomatic form, approximately 5-10% develop multiple organ failure, and mortality is high. Leptospirosis is rarely suspected due to the non-specific clinical presentation. Here we report an uncommon case of multiple organ failure caused by leptospirosis in a 67-year-old man who was successfully treated with antibiotics and intravenous immunoglobulins (IVIG).

Case: A 67-year-old male presented with a history of nausea, vomiting and abdominal pain. He also had a history of decrease in urine output and darkening of the urine colour for two days. He had no significant medical history.

On admission, the patient was conscious with a blood pressure of 120/75 mm/Hg, heart rate of 118/min, body temperature of 37,5° C, respiratory rate of 18/min, oxygen saturation of 98% on ambient air. Lung auscultation revealed crepitant rales and he had pretibial edema. Due to acute renal failure, thrombocytopenia and high acute phase reactants in laboratory results, the patient was hospitalized in the internal medicine intensive care unit with a pre-diagnosis of sepsis and multiple organ failure. Laboratory findings on admission and follow-up are shown in Table 1. During follow-up, the day after admission; with worsening of consciousness and hypotension, the patient developed septic shock. Deterioration of the patient's condition also observed in laboratory results (Table 1). He was treated with wide spectrum antibiotics, positive inotropic agents and renal replacement therapy for treatment of worsening renal functions and anuria. There was no positive blood and urine cultures. Viral markers were negative. Leptospira, rickettsia and Coxiella serologies were also investigated because of fever, renal failure, jaundice and severe thrombocytopenia. Leptospira interrogans PCR resulted positive. Doxycycline and IVIG were added to the treatment. Clinical and laboratory response was obtained on 3rd day of the treatment. At the end of 8 days, the acute kidney injury resolved, clinical and laboratory parameters improved, and he had complete recovery on the 15th day. After recovery, he mentioned a history of contact with a stray dog.

Conclusion: We describe a rare and unusual presentation of leptospirosis. Zoonoses, as well as leptospirosis, are rarely involved, and thus, widely ignored in the etiology of sepsis and multiple organ failure. In our country, mortality of leptospirosis has been reported to reach 10-17% in severe cases. It is important to keep in mind that initial presentation of leptospirosis could be as multiple organ failure.

Keywords: Leptospirosis, sepsis, septic shock, multiple organ failure

Table 1. Laboratory findings of the patient

	On admission	At the beginning of treatment	On the first day of treatment	On the 3rd day of the treatment
AST/ALT (U/L)	37/25	298/104	227/176	23/42
T.bil/D.bil (mg/dL)	0.5/0.32	1.31/1.08	1.64/1.01	1.46/0.73
Urea/Creatinine (mg/dL)	111/3.23	160/4.47	222/4.25	233/2.28
LDH (U/L)	276	551	677	311
CK (U/L)	90	290	230	120
CRP (mg/L)	349	443	309	49
Procalcitonin (µg/L)	18.3	>100	95	6
WBC /neutrophil (10^3/µL)	5900/5500	25900/24000	11600/9000	18000/15000
Hemoglobin (g/dL)	14.1	13	14	13
Platelet (10^3/µL)	48	38	39	51
INR	1.06	1.3	1.2	1.16

Cardiovascular - Other

Abstract:0161

ANTISYNTHETASE SYNDROME: A MISDIAGNOSED CASE PRESENTING WITH MYOCARDITIS IN INTENSIVE CARE UNIT

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Introduction: Antisynthetase Syndrome (ASS) is an autoimmune disease characterized by the triad of arthritis, myositis and interstitial lung disease (ILD). The diagnosis can commonly be confused with Rheumatoid Arthritis (RA). ASS associated myocarditis has a prevalence of 3-4%. Here we report a case with a pre-diagnosis of RA and subsequently diagnosed as ASS complicated by myocarditis.

Case: A 49-year-old woman who had been previously diagnosed with RA presented to emergency department with shortness of breath. In her past clinical history, the patient reported RA treated with methylprednisolone and methotrexate. 3 months after the diagnosis, she presented with shortness of breath, edema in her lower extremities, weakness in proximal muscles and dysphagia. The patient was admitted to internal medicine intensive care unit with suspected myocarditis due to bilateral pleural effusion, increased bilateral interstitial markings on her chest X-ray and troponin T level of 800 ng/L. On admission, the patient was dyspneic, hypotensive, and tachycardic, and muscle strength was 2/5 in both lower extremities and 3/5 in both upper extremities. Laboratory results revealed elevated levels of CK, troponin T, NT-proBNP, ferritin and CRP (Table 1). Anti-CCP titer was negative. ANA titer was 1/1280, granular pattern. Anti-Jo 1 test, which was requested due to proximal muscle weakness and elevated levels of CK revealed a positive result, which was also measured by ELISA showed >200 RU/mL. When her previous records were analyzed, it was seen that elevated levels of CK were also present during the period when she was diagnosed with RA. Pulse steroid and cyclophosphamide treatment were started with the diagnosis of ASS associated myocarditis due to EF being 56% and elevated levels of CRP, ferritin, NT-proBNP and troponin. Further tests concerning any possible malignancies and viral etiologies revealed negative results. She was discharged on the 15th day of treatment with clinical improvement.

Conclusion: Myocarditis is a rare however, potentially life-threatening manifestation of some autoimmune diseases. Diagnosis is usually made based on clinical presentation and non-invasive imaging findings, classically echocardiography. Glucocorticosteroids and immunosuppressive agents remain the cornerstone of the treatment. This case report implies compelling considerations about inflammatory cardiac involvement in an autoimmune disease.

Keywords: Antisynthetase Syndrome, Myocarditis, Rheumatoid Arthritis

Table 1. Laboratory Findings of the Patient

	August, 2021	September, 2021	October,2021	Before Treatment	15th Day of Treatment	Reference Range
Creatine Kinase	2197 IU/L	786 IU/L	801 U/L	306 IU/L	120 IU/L	34-145 IU/L
Troponin T	-	806 ng/L	820 ng/L	487 ng/L	269 ng/L	<14 ng/L
NT-ProBNP	-	-	892 ng/L	5360 ng/L	1009 ng/L	<125 ng/L
CRP	15.8 mg/L	190 mg/L	15.5 mg/L	16 mg/L	3.56 mg/L	0-5 mg/L
Ferritin	-	-	1016.2 µg/L	693 µg/L	430 µg/L	13-150 µg/L

Cardiovascular - Cardiac arrest

Abstract:0182

PHAECHROMOCYTOMA/PARAGANGLIOMA INDUCED CATECHOLAMINE CRISIS PRESENTING AS STEMI

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Introduction: Phaechromocytoma/paraganglioma (PPGL) - induced catecholamine crisis is a rare but potentially fatal endocrine emergency. The cardiovascular system is often involved, usually mediated by hypersecretion of catecholamines leading to direct toxicity to the cardiomyocytes and significant reduction in myocardial blood flow through coronary vasoconstriction (1). We reported a case of PPGL-induced catecholamine crisis leading to ST- segment elevation myocardial infarction (STEMI) complicated by cardiac arrest and the post-cardiac arrest care.

Case: A 57-year-old Chinese male with background history of hypertension and hyperlipidaemia came for elective surgical resection of functioning left paraganglioma confirmed with raised 24-hour urine catecholamines/metanephrines level and increased tracer uptake on positron emission tomography–computed tomography (PET-CT). Patient was on endocrine follow-up for pre-operative medical therapy optimization and was started on phenoxybenzamine, atenolol and nifedipine. His pre-operative transthoracic echocardiogram (TTE) was normal.

30 minutes after uneventful induction with general anaesthesia (GA) and intubation, his blood pressure declined rapidly and he suffered cardiac arrest with initial non-shockable rhythm. Immediate cardiopulmonary resuscitation effort was commenced, and he had return of spontaneous circulation (ROSC) after a total low-flow time of 20 minutes. His electrocardiogram (ECG) post-ROSC (Figure 1A) showed inferior STEMI changes. Patient underwent emergency coronary angiogram which revealed no obstructive coronaries (Figure 1B,1C). Left ventriculogram showed normal left ventricular systolic function with no cardiac or aortic structure abnormalities. The cause of cardiac arrest was attributed to PPGL-induced catecholamine crisis leading to severe coronary vasoconstriction possibly related to GA induction. He was transferred to surgical intensive care unit for further management including targeted temperature management (TTM).

TTM was commenced immediately via intravascular cooling device with targeted hypothermia of 33 degree Celsius (°C). Subsequent ICU management follows standard PCAS protocol (2) which includes active weaning of vasopressors to aim for mean arterial pressure of 80 to 85 mmHg to improve organ perfusion while maintaining good cerebral blood flow by aiming for cerebral regional O2 saturation >50%, targeting normocarbia due to raised intracranial pressure shown by increased optic nerve sheath diameter >5 mm bilaterally. Patient had gradual rewarming to 37 °C after 24-hour cooling and achieved good neurological recovery in Cerebral Performance Category 1. He was discharged well with outpatient endocrinology and surgical follow up for further review.

Conclusion: We described a rare and unusual presentation of STEMI possibly from coronary vasospasm precipitated by PPGL-induced catecholamine crisis following GA induction. This case also illustrated the importance of comprehensive post-resuscitation ICU care to achieve favourable outcome.

Keywords: paraganglioma/phaechromocytoma crisis, STEMI, cardiac arrest, TTM

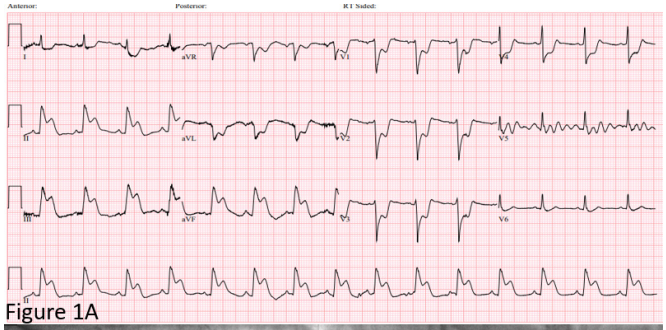
Figure 1A

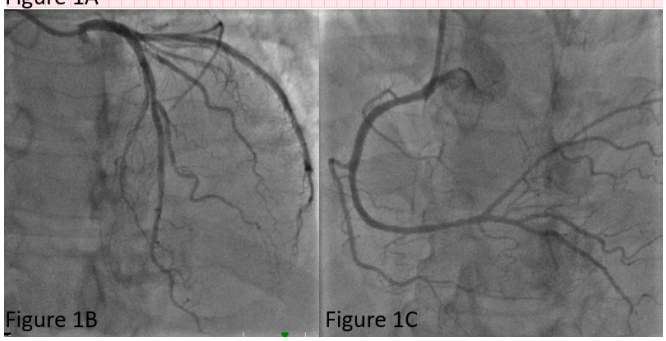
Figure 1B Figure 1C

Figure 1. ECG and Coronary angiogram

A. showed ST-segment elevation in leads II, III, aVF with reciprocal ST -depression changes seen in leads I, aVL, V1-V4. **B** and **C** showed no significant obstruction in the left and right coronary arteries.

Critical care pharmacy and drug monitoring

Abstract:0188

PYRIDOSTIGMINE ASSOCIATED DIARRHEA IN A MYASTHENIC PATIENT: A CASE REPORT

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Introduction: Pyridostigmine is an acetylcholinesterase inhibitor that is included in the initial therapy for most patients with myasthenia gravis. (1). The side effects of pyridostigmine are mostly due to its cholinergic properties. Diarrhea, abdominal cramps, and muscle twitching are the most common side effects requiring discontinuation of pyridostigmine therapy. (2). In this case study, the management of diarrhea in a patient receiving pyridostigmine for the treatment of myasthenia gravis is presented.

Case: A 50-year-old female patient, who has been followed up with myasthenia gravis for 7 years, was admitted to the anesthesiology and reanimation unit with the diagnosis of myasthenic crisis. Continuing the patient on 60 mg of pyridostigmine every 6 hours and 50 mg of azathiopurine every 12 hours, which she used at home; in addition, starting 16 mg of methylprednisolone once a day and applying plasmapheresis was recommended by the neurology department. The patient was consulted to infectious diseases because of diarrhea that started on the 4th day of hospitalization and increased in frequency (9 times a day). Infectious diseases experts recommended that stool culture be studied from the patient. The stool culture studied was Clostridium difficile Polymerase Chain Reaction (PCR), which was negative, and stool direct microscopy: At 40 times magnification, no erythrocytes/leukocytes were seen. The clinical pharmacist in the unit evaluated the patients' therapy whether the patient's diarrhea was caused by a drug or not. A review of the literature suggested that diarrhea could be a side effect of pyridostigmine. At the same time, it was shared with the physician in charge that the patient might have gastrointestinal system discomfort caused by the enteral nutrition product she was taking, and some suggestions were presented. The recommendations made to the physician, the implementation of the recommendations, the relevant explanations, and the change in the frequency of diarrhea are given in Table 1.

Conclusions: There are various methods for treating acetylcholinesterase inhibitor-induced diarrhea. Some of these include giving the medicine with food and adding anticholinergic and antidiarrheal medicines to the treatment plan. Apart from this, one of the most common side effects of enteral formulas is diarrhea. In this case, the content of the food should be questioned, and an alternative product should be used if necessary. It is thought that clinical pharmacists who can identify and manage the side effects of drugs and provide consulting on nutrition should be included in the healthcare team.

Keywords: anticholinergic drugs, diarrhea, pyridostigmine, myasthenia gravis, nutrition

Tablo 1 The recommendations made to the physician, the implementation of the recommendations, the relevant explanations, and the change in the frequency of diarrhea

Suggestions	Application situations	Explanations	Diarrhea frequency
It was said that the enteral product that the patient was taking could cause diarrhea and that gastrointestinal system disorders could be resolved if a product containing less soluble fiber was used.	The responsible doctor swapped out the enteral product the patient was taking for another enteral product with less soluble fiber.	It has various effects according to the types of fibers in enteral foods. Products containing soluble fiber can cause diarrhea, while products containing insoluble fiber can cause constipation.	Daily 9→ Daily 6-7
It was said that the drug pyridostigmine should be given with meals.	While the patient was constantly fed, the ward physician changed the feeding regimen to 300 cc of food every 4 hours and said that pyridostigmine should be given with meals.	If taken with meals, gastrointestinal side effects caused by pyridostigmine may decrease.	Daily 6-7→ Daily 4-5
It was said that diarrhea may be related to pyridostigmine, so atropine drops should be prescribed in consultation with the neurology service.	The service physician said that a consultation with neurology should be made. Neurology recommended 1 drop of atropine before each pyridostigmine.	Anticholinergic drugs such as atropine may be helpful in pyridostigmine-related diarrhea.	Daily 5→ Daily 3-4
Since diarrhea continued despite a decrease in frequency, a drug containing the antidiarrheal diphenoxylate and atropine combination was recommended.	The service physician added the recommended drug to the treatment.	Antidiarrheal drugs such as loperamide or diphenoxylate plus atropine may be useful in the treatment of diarrhea that continues after atropine.	Daily 3-4→ Daily 2-3
It was said that due to the ongoing diarrhea, the diphenoxylate and atropine dose should be adjusted twice with 6-hour intervals instead of once with 6-hour intervals, and that the dose of atropine drops should be reduced on the grounds that it may cause an increase in anticholinergic side effects.	The responsible physician increased the dose of the antidiarrheal drug from once with a 6-hour interval to twice with an interval of 6 hours and requested consultation from the neurology service for atropine drops. Neurology recommended that the atropine drop should be dropped to 1 drop at 12-hour intervals instead of 1 drop every 6 hours, and it should be used for a few days and then stopped.	The antidiarrheal diphenoxylate and atropine combination can be used as 8 tablets per day if necessary.	Daily 2-3→ Daily 1-2
It was said that the antidiarrheal drug should be stopped because the diarrhea had resolved.	The responsible physician stopped the drug because the drug containing the diphenoxylate and atropine combination, which was taken twice at 6-hour intervals for approximately 7 days, had been sufficiently taken and the diarrhea was no longer present.	Antidiarrheal drugs should be stopped after the diarrhea has resolved.	Diarrhea was treated

Physiotherapy

Abstract:0193

EARLY REHABILITATION PREVENTS ICU-ACQUIRED WEAKNESS IN PATIENT WITH SEVERE TRAUMATIC BRAIN INJURY (TBI): AN ALGERIAN CASE REPORT

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Introduction: ICU patients frequently develop neuropathy and/or myopathy referred to as ICU-acquired-weakness. Although pathophysiology's complex structural and functional changes are still not fully known, this condition frequently affects limb and respiratory muscles. Early rehabilitation may reduce the incidence of this disorder in critically ill patients.

Case: We present a case of a 24-year-old Algerian male who was a victim of a road accident, that caused a severe brain injury; loss of consciousness with Glasgow inferior to 8 before the first 24 hours, diffuse cerebral contusion, and coma for 10 days. He benefited from early rehabilitation during his 2 months of hospitalization. 2 months after the accident, the patient was referred to our rehabilitation center with flasque tetra paralysis. He carried out daily physical, occupational, and speech therapy. A significant functional improvement resulted from the early rehabilitation.

Conclusion: The current case report describes the importance of early rehabilitation in ICU patients to prevent ICU-Acquird weakness.

Keywords: ICU-Acquired weakness, critically ill patients, early rehabilitation

Infections and antimicrobials

Abstract:0197

THE USE OF COUPLED PLASMA FILTRATION AND ADSORPTION (CPFA) IN COLD AGGLUTINATION DISEASE WITH SEVERE HEMOLYSIS; EFFECTIVE BILIRUBINE DETOXIFICATION

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Introduction: The CPFA system consists of plasma separation followed by plasma adsorption through a single hydrophobic styrene resin cartridge. It is an option in intensive care patients such as acute kidney injury, septic shock, and hepatic malnutrition, with its ability to remove cytokines and inflammatory mediators without severe albumin loss. CPFA was used as an extracorporeal treatment method in a patient with severe hyperbilirubinemia associated with severe anemia and it's presented with literature.

Case: A 60-year-old male patient with no history of comorbidities was admitted to a hospital for jaundice and hematuria. The patient was admitted to our intensive care unit with the preliminary diagnoses of severe anemia and hyperbilirubinemia, respiratory and liver failure. On physical examination, the patient was nonoriented, noncooperative, tachypneic, and sclera, skin and mucous membranes were icteric. He was intubated and started mechanical ventilator support. Total/direct bilirubin: 66.83/ 36.89 mg/dL, ALT:154 U/L, AST:243 U/L, GGT:207U/L, alkaline phosphatase:162U/L, CK:793 U/L, LDH: 2435 U/L, hemoglobin:2.3 g/dL, hematocrit:2.3%, MCV:121.1 fL, MCH:121.1 pg, MCHC:100 g/dL, platelet:141x10^3/μL, total iron:611 μg/ dL, iron binding capacity:<55 μg/dL, B12:502 ng/L, folate:7.38 μg/L, ferritin;582.7 μg/L, CRP:123.9 mg/L, procalcitonin:0,7 μg/L, fibrinogen:373.87mg/dL, INR:1.45, PT:16.66sec, d-dimer:11.82 mg/L, indirect coombs:(+), direct coombs (polyspecific): (+4), cold agglutinins: (+) were seen. EBV VCA IgM (-), respiratory viral panel (-) was monitored. The blood group of the patient could not be determined. Agglutination was observed in the peripheral smear. The patient was evaluated for hemolytic anemia caused by cold antibodies secondary to infection. Appropriate blood and blood product replacement was performed. Steroid treatment was started, and blood hemoglobin could be increased up to 5g/dL with replacements. Three sessions of plasma separation were performed with CPFA and direct/total bilirubin could be reduced to 37.92/20.69mg/dL. Sudden tachyarrhythmia developed while he was in clinical and laboratory improvement and the patient died on the 2nd day.

Conclusion: Hemolytic anemias induced by cold antibodies can occur as idiopathic and secondary (infection, lymphoproliferative diseases, non-lymphoid malignancies). Hemolytic anemia usually occurs in the second and third week of acute infection. The direct Coombs test is positive for complement. In severe forms, corticosteroid therapy or plasmapheresis can be applied. CPFA is a detoxification system that combines a plasma adsorption circuit with continuous renal replacement therapy and can play an active role in eliminating the toxic effects of severe hyperbilirubinemia as a result of hemolysis. Bilirubin, which represents the main marker for predicting liver function, is significantly reduced by CPFA. It can buy time until the primary disease is treated.

Keywords: Cold antibody-induced hemolytic anemia, Hyperbilirubinemia, CPFA device

Cardiovascular - Other

Abstract:0202

AN UNEXPECTED COMPLICATION OF MASSIVE CEREBRAL ARTERIAL AIR EMBOLISM WHILST ON CARDIOPULMONARY BYPASS

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Introduction: Cardiopulmonary bypass (CPB) is a form of extracorporeal circulation during cardiac surgery where the patient's blood is diverted away from the heart and lungs to outside of the body. The CPB machine is operated by a perfusionist, whose role may be within the scope of a cardiac anaesthetist or other professionals who is trained in this field of expertise and registered with the Australian and New Zealand College of Perfusionists (ANZCP). The ANZCP has published set standards and regulations on the practice of perfusion, and a key component of safe practice is the performance of safety checklists prior to each procedure to minimize complications [1].

Case: This case report explores the scenario of a patient who undergoes an elective aortic root replacement and is placed on cardiopulmonary bypass. An unexpected complication arises after hearing the sound of ectopic beats from the cardiac monitor and seeing ECG changes of ST elevation consistent with air in the coronary arterial system. Air could be seen in the descending aorta through the use of a transoesophageal echo. It was assumed that the patient had entrained air to the cerebral system, whereby after stabilizing and ultimately transferring the patient to intensive care, there was an investigation of the CPB machine to show that the suction system of

the pump had been placed incorrectly; instead of suctioning air from the left ventricle, it was actually pumping air forwards.

Conclusion: Air embolism is one of the most common complications that can occur during CPB. Literature has described management through immediate cessation of the pump and assuming the deep Trendelenburg position, retrograde cerebral perfusion, initiating hypothermia, administering deep barbiturate anaesthesia, applying 100% oxygen ventilation, steroids, and massaging air to allow escape from the coronary arteries [2]. In the case of reversed placement of the suction and vent tubing, the ANZCP guidelines state that the tubing should be visually inspected and tubing must always be verified to be attached correctly by the perfusionist [1]. The direction of the tubing and pump heads should be marked with arrows and labelled. To prevent the incorrect placement of tubing, certain CPB manufacturers such as Sorin and Maquet have incorporated mechanical coding inserts to the specific tubes to ensure the right tube is placed into the right system [1]. In this particular case scenario, the patient unfortunately suffered the sequelae of the cerebral air embolism and was not able to return to previous independent function.

Keywords: Cerebral arterial air embolism, cardiopulmonary bypass, air embolism

Cardiovascular - Cardiac arrest

Abstract:0204

SEVERE ACCIDENTAL HYPOTHERMIC CARDIAC ARREST CAUSED BY EXTRACORPOREAL MEMBRANE OXYGENATION HEAT EXCHANGER ALARM FAILURE: A RARE CASE REPORT

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Introduction: Severe accidental hypothermia, an unintentional drop in body temperature, is a rare life-threatening emergency, corresponding with high mortality risk, particularly in cases leading to cardiac arrest. The use of extracorporeal membrane oxygenation (ECMO) has been successfully utilized to treat cardiac arrest caused by hypothermia. Nonetheless, severe accidental hypothermic cardiac arrest caused by ECMO heat exchanger alarm malfunction is ultimately unusual. Fast recognition of such tragic events is undoubtedly a life-saving step affecting the patient's survival.

Case: We report a case of a 28-year-old female patient with past medical history of six-year systemic lupus erythematosus complicated with renal failure and COVID-19 infection who was diagnosed with pneumonia worsened by acute respiratory distress syndrome (ARDS). Her hemodynamic status was normal. She was initially treated with broad-spectrum antibiotics (Colistin, Ceftazidime/Avibactam) and mechanical ventilator according to ARDSNet protocol, which was then converted to venovenous ECMO (V-V ECMO) because of unresponded respiratory status to maximal ventilator support on the first day. On the second day, the patient suddenly experienced bradycardia, ventricular fibrillation and unexpected cardiac arrest. She was resuscitated based on advanced cardiac life support but not respond after fifteen minutes, a venoarterial ECMO (V-A ECMO) was inserted using a 16.5Fr cannula (Terumo) into the right femoral artery. Within thirty minutes, the patient achieved return of spontaneous circulation with normotension without vasopressor use and her body temperature was 22^{0}C. We unintentionally discovered water accumulation on the ECMO oxygenator and coldness upon palpation as well as on the ECMO heat exchanger monitor screen (Terumo), the temperature was 22^{0}C, and no alarm was triggered. Thinking of cardiac arrest caused by hypothermia, we immediately replaced new reheat system and activated the active internal rewarming. The patient's temperature stabilized at around 36^{0}C after four hours. The ECMO mode was changed back to V-V ECMO with the prior jugular vein cannula, and the femoral artery cannula was removed. Twelve hours later, she regained alert and responsive status. The patient displayed an improved clinically normal condition. The subsequent progression was uneventful. She was transferred to the pulmonology department for further treatment after one month in the intensive care unit and discharged after a week.

Conclusion: Our case emphasizes the significance of early detection of accidental hypothermia during the treatment of ECMO which may lead to cardiac arrest. Accidental hypothermic cardiac arrest is a reversible state, quick diagnosis and appropriate care, especially with V-A ECMO, allows for an increase in likelihood of positive neurologic outcomes.

Keywords: extracorporeal membrane oxygenation, hypothermia, cardiac arrest, extracorporeal life support

Cardiovascular - Other

Abstract:0227

PERSISTENT LEFT SIDED SUPERIOR VENA CAVA: AN UNUSUAL PATH OF LEFT INTERNAL JUGULAR CENTRAL LINE, A CASE REPORT

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Introduction: Central venous cannulation is a commonly performed procedure in intensive care units and perioperative settings. Incidence of central venous catheter malposition is rarely due to anatomical variation and it is often associated with fatal complications. One such variation is the persistent left-sided superior vena cava (PLSVC).

Case: We report a case who underwent central venous catheter placement via the left internal jugular vein, and a subsequent chest x-ray and contrast fluoroscopy revealed the malposition of the central venous catheter and presence of persistent left-sided superior vena cava (PLSVC). Persistent left superior vena cava (PLSVC) is a thoracic venous anomaly which is found in 0.3-0.5% of general population. Although PLSVC is often asymptomatic, it can lead to significant problems, such as arrhythmias and cyanosis, and can cause serious complications during vascular interventional procedures.

Conclusions: The case highlights the importance of considering anatomical variations in central vessels while performing central venous cannulation to improve the safety of the procedure and avoid complications. It also emphasizes the need for further imaging modalities to confirm the position of the catheter in such cases.

Keywords: central venous catheter, catheter malposition, persistent left sided superior vena cava, internal jugular venous access

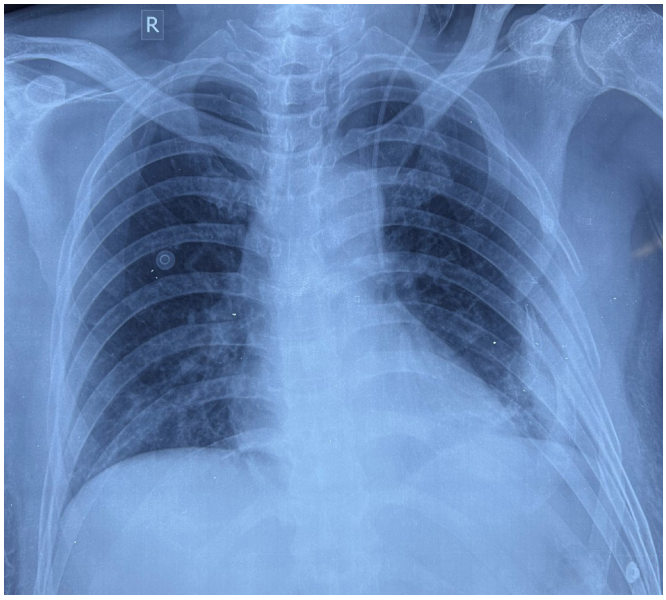

Figure 1. Post central venous catheter insertion chest x-ray showing the catheter course in left side of mediastinum

Tip of central venous catheter is seen in left side of mediastinum which is an unusal course

Respiratory - ARDS and acute respiratory failure

Abstract:0230

THE CRUCIAL ROLE OF CO_2 MANAGEMENT DURING EXTRACORPOREAL MEMBRANE OXYGENATION IN STATUS ASTHMATICUS - FATALITY IN NEAR FATAL ASTHMA

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Introduction: ECMO is a life-salvaging measure in a case of refractory status asthmaticus and has been associated with increased risk of cerebral bleeding. This case reports highlights possible reason behind the fatal complication and also lessons learnt from the experience.

Case: A 34-year-old female was admitted with status asthmaticus refractory to medical therapy including bronchodilators, steroids, beta-agonists, general and inhaled anesthetics. In view of severe hypoxia and hypercarbia, a decision to initiate VV ECMO was made and since the patient was unstable ECMO was initiated on site and retrieved. Prior to ECMO neurological assessment could not be done because of deep sedation and paralysis. Her pCO_2 was 128 mm hg at the time of VV ECMO initiation and was reduced to 70 mm hg. Minimal heparin was used during initiation and ACT was maintained within limits. There was improvement in hypoxia and hypercarbia but she remained in poor neurological state. CT showed multiple hemorrhagic areas in bilateral cerebral hemispheres and few infarcts in the brain. On Day 2 of ECMO she had no brainstem reflexes. CT angiogram showed absent cerebral circulation suggestive of brain death and ECMO was used as the bridge to organ donation.

Conclusion: This report highlights the high risk of bleeding complications associated with ECMO in status asthmaticus. There is evidence which shows hemorrhages and infarct to be common complication. Bleeding can be a result of prolonged refractory hypercarbia increasing cerebral blood flow and blood pressure prior to ECMO initiation. It can also be attributed to sudden decrease in pco2 levels after initiation of ECMO compounded by anticoagulation. Brain imaging prior to ECMO initiation would have been a decision maker with regard to avoidance of anticoagulation during ECMO however it could not be done in this case due to clinical instability. In clinical practice, there is a need to have a high index of suspicion and preventive measures like avoiding sudden and large changes in pco2 with gradual increase in ECMO flow and sweep gas. Anticoagulation and blood pressure management needs to be more stringent in status asthmaticus along with early ECMO initiation to avoid deleterious effects of prolonged hypercarbia. This report also demonstrates the use of ECMO as a bridge to organ donation.

Keywords: ECMO, Asthma, Pco2, intracranial bleed, organ donation

Metabolism - Other

Abstract:0249

TOXIC ALCOHOLS ARE STILL PROBLEMATIC: AN EXEMPLIFY CASE SERIES FROM TURKEY

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Introduction: Accidental or intentional ingestion of substances containing toxic alcohol can result in death. In our country, illegally produced alcohols bring along intoxication.

Cases: Case 1: A 35-year-old male patient was administered to the hospital with vision loss after 2 hours of homemade alcohol ingestion. In the first examination, he was conscious, cooperative, oriented, IR:+/+ and GKS was 15, blood pressure (BP) 145/90 mmHg, pulse (P) 114/min, and respiratory rate 22/min. ABG: pH: 7.16, PCO2:14.5 mmHg, PO2:58 mmHg, HCO3:5.1 lactate: 7.8 mmol/L, anion gap(AG): 31 mmol/L. Ethyl alcohol treatment and sodium bicarbonate was administered and CRRT treatment was started. Patient was intubated due to loss of consciousness. With in 10 hours patient deteriorated and died.

Case 2: A 30-year-old male patient who was referred to our intensive care unit by another hospital with metabolic acidosis and loss of consciousness. Cologne has been taken by patient. At ICU admission he was intubated and sedated, IR: +/+, BP: 130/700 mmHg, P: 85/min. ABG was pH:6.97, PCO2:19.2 mmHg, PO2:212.7mmHg, HCO3: 4.3mmol/L, AG:25mmol/L, lactate: 2.3mmol/L. Ethyl alcohol treatment was given for 5 days and CRRT was applied for 3 days. In the follow metabolic acidosis improved and successful weaning was performed on fifth day. He was discharged to hospital ward on tenth day.

Case 3: A 54-year-old male patient had recourse to a hospital with loss of vision and decreased awareness. It was learned that he made alcohol at home and drank it. There urgent dialysis and ethyl alcohol was administered and levetiracetam was given to the patient who developed epileptic seizures. After ICU admission he was intubated and sedated, IR: +/+, BP: 180/110 mmHg, P: 105/min. ABG was pH:7.48, PCO2:34 mmHg, PO2:108mmHg, HCO3: 24mmol/L, AG:20mmol/L, lactate: 4.18 mmol/L, methanol:99.7 mg/dl. Ethyl alcohol treatment was given for 3 days and hemodialysis was applied. Anisocoria rred therefore cranial CT scan was performed. Intracranial bleeding in temporoparietal lobe was diagnosed. A decompressive craniectomy was done. On 3rd day methanol levels decreased to 0.4 mg/dl. Patient remained in coma. He has died from septic shock on the fiftieth day.

Conclusion: Toxic alcohol effects are usually manifest a few hours after consumption. Visual impairment, gastrointestinal disorders severe metabolic acidosis, hypotension, confusion, and seizures can

occur. In our case series, two of patients have died despite adequate treatment. Critical care physicians need to be familiar with these conditions as well as each toxic alcohol so that an exposure can be identified promptly and treated appropriately.

Keywords: toxic alcohol, methanol, intracerebral hemorrhage

Neuro-intensive care

Abstract:0253

A COVID-19 PATIENT WITH NECROTIZING INFLAMMATORY MYOPATHY AND RHABDOMYOLYSIS: A CASE REPORT

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Introduction: COVID-19 is a multisystem disease that presents with diverse manifestations involving the lungs, kidneys, musculoskeletal and also other systems. Muscle weakness, myalgia, and rhabdomyolysis are symptoms associated with COVID-19. We want to present a COVID-19 patient with necrotizing inflammatory myopathy and rhabdomyolysis.

Case: 21-years-old, male, who had upper respiratory tract symptoms about 5 days ago, presented to the emergency department with complaints of widespread body pain, muscle weakness and breathlessness.

Physical examination: BP:150/100 mmHg, pulse:101 beat/minute, BT: 37.8°C, O2 sat: %97 mild rales in the lower basal areas of the lung, mild tachycardia, pretibial edema was absent.

Upper extremity muscle strength examination was right 3/5, left 0/5 and both lower extremities were 0-1/5.

Laboratory results: cre: 0.73 alb:4.3 alt:1930 ast:6590 alp:74 ggt:27 ld:10510, ck:352820 ck-mb:280000 trop:25, crp:13,8 pct:0,59, wbc:23000 hgb:19,9 ph:7,31 paco2:66mmhg.

Thorax CT showed slight ground glass densities in the subpleural areas at posterobasal levels of the lower lobes of both lungs. It was evaluated as suspicious for Covid-19 disease.

The patient was transferred to the ICU due to the development of severe breathlessness and carbon dioxide retention. The patient was intubated. Covid-19 result was positive he was transferred to the covid ICU. Covid-19 associated polymyositis and inflammatory polymyopathy were suspected with the development of severe breathlessness, muscle weakness and rhabdomyolysis. Muscle biopsy was done and striated muscle biopsy showing few necrotic muscle fibers were evaluated in the biopsy.

Covid-19 related necrotizing inflammatory myopathy and rhabdomyolysis were considered in the patient. Plasmapheresis and IVIG treatment were performed for 7/5 days. Pulse steroid for 3 days and then maintenance treatment was started. Imuran and mycophenolate mofetil were also added to the treatment. Tracheostomy and PEG were performed. Protein-rich enteral nutrition solution, fluid replacement, antibiotics, vitamin D, B, carnitine, baclofen treatment were started. The patient was included in an exercise program. Weaning was achieved after his vital signs stabilized. Tracheostomy and PEG were removed after 3 days of follow-up with fenestrated cannula in room air. The patient, who was GCS 15, orally fed, was able to walk, and whose vital signs were stable, was transferred to the general internal medicine ward. The patient, whose general health improved, was discharged with full recovery.

Conclusion: Covid-19 may present with acute respiratory failure as well as upper and lower extremity muscle weakness and rhabdomyolysis.

Keywords: Covid-19, acute respiratory failure, necrotizing inflammatory myopathy, rhabdomyolysis, proximal and distal muscle weakness

Kidney - Renal replacement therapy

Abstract:0260

CYTOKINE FILTER TREATMENT IN OCRELIZUMAB TOXICITY CASE

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Introduction: Ocrelizumab is a recombinant human anti-CD20 monoclonal antibody designed to optimize B cell depletion. The most common side effects of ocrelizumab therapy are infusion reactions, upper and lower respiratory tract infections, and skin infections. Ocrelizumab is contraindicated in patients with active hepatitis B virus infection. In our case, the hyperinflammation due to ocrelizumab and the use of a cytokine filter will be presented.

Case: A 56-year-old male patient with a diagnosis of secondary progressive multiple sclerosis was admitted to the emergency department with complaints of increasing weakness and abdominal pain for about 2 weeks. In the examinations, elevated liver and kidney function tests were detected. His history was unremarkable except for the ocrelizumab he had taken 1 month ago. The patient, whose general condition was poor, was admitted to the intensive care unit with the diagnosis of multiple organ dysfunction. The patient was taken to hemodialysis because he had acidosis and anuria during his hospitalization. N-acetyl cysteine infusion was started. Hepatitis markers were negative. There was no improvement in creatinine levels in the subsequent control blood. Laboratory values; ferritin: 2309, uric acid: 15.8, urea: 125, creatinine: 1.99, AST: 9191, ALT: 3394 LDH: 5813. Considering the hyperinflammation and cytokine-induced organ dysfunction, the patient underwent continuous renal replacement therapy (CRRT) with a cytokine filter. Significant improvement was observed in the 24-hour laboratory of the patient, and the treatment was continued for 72 hours. Post-treatment values; creatinine: 1.59, ferritin: 482, uric acid: 5.2, AST: 1845, ALT: 1713, LDH: 880. During the follow-up, the patient's AST value decreased to 123 and ALT to 434. The patient, who was considered to be transferred to the ward on the 5th day of his hospitalization, was intubated with sudden onset of tachypnea and tachycardia. He had elevated cardiac enzymes since his admission. On the 8th day of his hospitalization, the patient was declared dead with sudden cardiac arrest. Myocarditis was considered as the cause of death.

Conclusion: Ocrelizumab is one of the new molecules used for many autoimmune diseases. Due to the drug related immunosuppression, patients are predisposed to infective manifestations. In this case, dysregulated immune response due to this condition and multiple organ dysfunction due to cytokine storm were observed. A significant improvement was observed in laboratory values by applying cytokine filter. It should be kept in mind that this drug, which is a new molecule, can use cytokine filter for its toxicity.

Keywords: Cytokin Filtre, Ocrelizumab, MODS

Respiratory - Other

Abstract:0262

A CASE OF CRUSH INJURY WITH MASSIVE PULMONARY EMBOLISM

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Introduction: Crush injury is the result of physical trauma from prolonged compression of the torso, limb(s), or other parts of the body. The resultant injury to the soft tissues, muscles, and nerves can be due to the primary direct effect of the trauma or ischemia related to compression. Systemic manifestations resulting from crush injury, which is defined as crush syndrome, can result in organ dysfunction or death. In this case, pulmonary embolism accompanying traumatic crush injury will be described.

Case: A 57-year-old male patient applied to our hospital from the earthquake zone with his own means. The patient, who was under the rubble for about an hour, complained of shortness of breath and pain in the leg. He arrived immobile during a 12-hour car journey. The patient, who was found to have a right tibial fracture and a fracture at the base of the skull, was consulted to the necessary departments when he came to our hospital. He was followed up for compartment syndrome. On thorax CT, extensive filling defects consistent with embolism were observed in the right main pulmonary artery, lobar and segmental branches. The patient with respiratory distress was taken to the intensive care unit. Due to pulmonary embolism, a high risk was given for the operation, and it was decided to operate after the acute period. High dose low molecular weight heparin therapy was given. He was followed closely in terms of Crush syndrome and necessary hydration was provided. He was referred to the orthopedic department after his general condition recovered. About 18 days after the trauma, the patient had sudden respiratory distress and then respiratory arrest in the ward. It was thought that death occurred due to massive pulmonary embolism.

Conclusion: Trauma is an emergency that requires a multidisciplinary approach. Although Crush syndrome is one of the most known complications, other emergencies that this situation may cause should also be considered. Although necessary precautions were taken in terms of crush syndrome and compartment syndrome in our case, an embolism that did not improve despite treatment led to death. Specialized approaches and versatility should be mandatory in trauma patients.

Keywords: crush injury, pulmonary embolism, respiratory failure

Hematology and Oncology

Abstract:0263

HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS IN INFLAMMATORY BOWEL DISEASE CASE

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Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a rare, severe systemic syndrome characterized by a rapidly progressive inflammatory response associated with the release of pro-inflammatory cytokines and the activation of the immune system. Macrophage activation syndrome is a form of secondary HLH that occurs in patients with autoimmune diseases. In this case, we will describe the macrophage activation syndrome that developed in a patient followed up with ulcerative colitis.

Case: A 38-year-old male patient presented with fever, chills and malaise for the last three or four days. Pancytopenia was found in the examinations of the patient with known rheumatoid arthritis and ulcerative colitis diagnoses. Among the drugs he used were mesalamine, azathiopurine and adalimumab. The patient was admitted to the hematology ward to determine the etiology. He was consulted on infectious diseases in terms of sepsis. Bone marrow aspiration was performed and it was compatible with the macrophage activation syndrome. The patient whose general condition deteriorated was taken to the intensive care unit. Pulse steroid therapy and etoposide therapy were applied to the patient. Hemodialysis with cytokine filter was started with plasmapheresis. During the follow-up, he developed melena and severe oral bleeding. There was no improvement in cytokine levels or liver and kidney function tests. The patient died on the tenth day of his intensive care admission.

Conclusion: HLH is a rare and life-threatening syndrome which can be confused with sepsis. If not treated quickly, it can lead to death shortly. Specific treatment must be initiated as soon as possible. It is generally used an association of high-dose steroids, cyclosporine A and etoposide, in an attempt to control inflammation. If suspected, examination and treatment should be done as soon as possible.

Keywords: Inflammatory Bowel Disease, Hemophagocytic Lymphohistiocytosis, pansitopenia

Respiratory - Airway management

Abstract:0267

A CASE REPORT OF PNEUMOMEDIASTINUM, PNEUMOTHORAX, PNEUMOPERITINIUM AND PNEUMOPERICARDIUM AFTER INTUBATION, FOLLOWED IN THE INTENSIVE CARE UNIT

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Introduction: In the intensive care unit, endotracheal intubation(ETT) is performed with many different indications and various complications can be seen. It is the most common laryngeal injury among mechanical, and hemodynamic complications. Not infrequently, pneumothorax, pneumomediastinum, pneumoperitoneum and subcutaneous emphysema can also be seen. Iatrogenic pneumothorax is more common after lung procedures and central venous catheterization. Pneumomediastinum and pneumoperitoneum can be found alone or with pneumothorax on chest x-rays. Pneumomediastinum and pneumoperitoneum due to pulmonary trauma are usually self-limited and managed with decreasing ventilation pressures, monitorization and supportive measures. Subcutaneous emphysema usually presents as a sudden, painless swelling of soft tissue, mostly involving the upper chest, neck, and face. In this case, pneumothorax, pneumomediastinum, pneumopericardium, pneumoperitoneum developed after ETT will be presented.

Case: A 79-year-old female patient with known stories of DM, HT, AF, HF was admitted to ICU due to LCHF. While following with non-invasive mechanical ventilation, the patient observed desaturated and tachypneic, and then she was intubated electively. Thoracic CT was performed for etiology research in the patient with

subcutaneous emphysema and refractory hypoxemia after intubation. Thorax CT showed that apical, mediastinal and pericardial air, which is common through the thorax wall from the neck level, including the examination (figure 1). In the follow-up of the patient, distension in the abdomen and extensive abdominal sensitivity developed. Performed abdomen CT showed diffuse air trapping under the skin in the peritoneal and retroperitoneal region with distributional location in the mesothelial surface which was reached 6 cm in the deepest place, distributed in millimetric dimensions (figure 2). The patient was immediately consulted in chest surgery and general surgery clinics for intervention. Emergency surgery was not considered due to solid organ injury, and no rupture in the esophagus or trachea. Three bilateral branules installed in the subcutaneous tissue for subcutaneous emphysema. Peep: 2-3 cmH_2O; FiO_2: between 40-60% titrated as PaO_2>60 mmHg, SaO_2>90%. The patient, whose pneumothorax, pneumomediastinum, and subcutaneous emphysemas were regressed in the follow-up with daily lung x-ray. The patient without hypoxemia and hypercapnia, was extubated with a 4-day weaning trial.

Keywords: Intubation, Pneumothorax, Pneumomediastinum, Pneumoperitoneum, Subcutaneous Emphysema

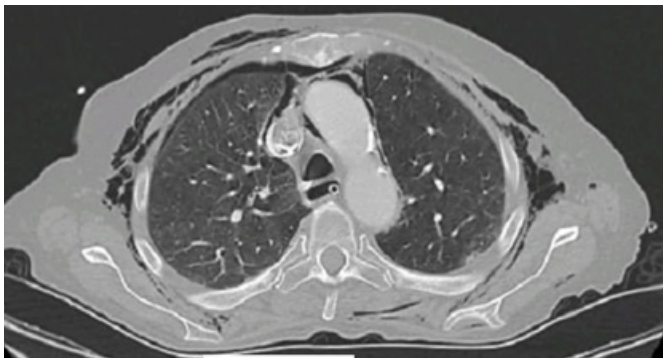

Figure 1. Thoracic CT Image
Pneumomediastinum, pneumothorax, pneumopericardium

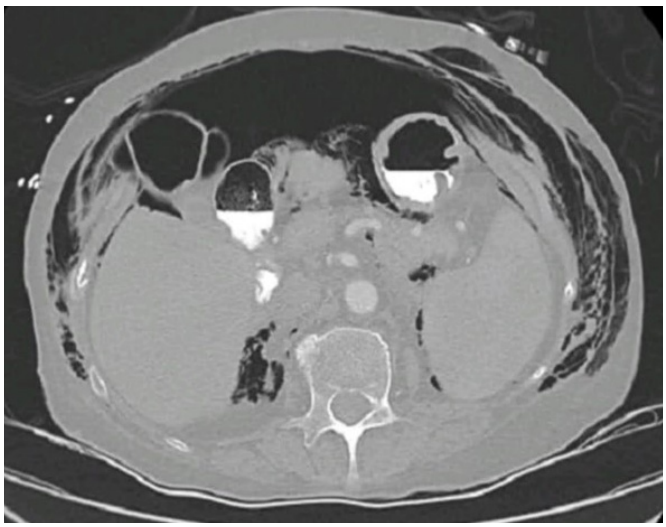

Figure 2. Abdomen CT Image
Pneumoperitoneum

Respiratory - Airway management

Abstract:0273

TRACHEOESOPHAGEAL FISTULA DEVELOPMENT IN A NEAR HANGING PATIENT: A CASE REPORT

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Introduction: Fistula formation between the trachea and esophagus is a difficult to treat condition. Trauma, mediastinal granulomatous infections, immunodeficiencies and iatrogenic trauma are the most common causes of non-malignant tracheoesophageal fistula (TEF) in the last 30 years (1). The term "near-hanging" refers to patients who survive a hanging injury long enough to reach the hospital. This case describes the development of tracheoesophageal fistula in a near-hanging patient.

Case: A 41-year-old male patient with no known history of chronic disease, who developed cardiac arrest after a suicide attempt with hanging and whose circulation returned with 15 minutes of cardiopulmonary resuscitation, was transferred from another hospital. It was learned that patient was intubated uneventfully with a 7.5 mm endotracheal tube on arrival. The patient was evaluated as hypoxic encephalopathy. Current scanning was done on patient's arrival. In computerized tomography scan, any sign of TEF wasn't observed. Patient was inserted nasogastric tube for feeding. TEF was considered in patient, because the bag that nasogastric tube attached filled with air. In diagnostic fiberoptic bronchoscopy (FOB), proceeding from trachea entrance, areas where necrosis is likely to develop in posterior trachea and both lateral areas, and a fistulous necrosis area on the posterior wall were observed. Air and methylene blue came from the fistula area. Esophageal stenting was recommended. In endoscopy, TEF was observed at 20.cm from incisors. It wasn't considered suitable for stenting due to the proximal position of TEF. Tracheostomy was not suitable due to fistula localization. Patient was followed with parenteral nutrition throughout the hospitalization.

Conclusion: TEF should be considered if breathing sound comes from patient's mouth, if the bag which nasogastric tube is attached filled with air, if the food and secretions come from trachea or around tracheostomy cannula while aspirating patient (2). The diagnosis can be confirmed by fiberoptic bronchoscopy or endoscopy. Administration of methylene blue from esophagus during bronchoscopy can be diagnostic even for small fistulas. In conclusion, tracheoesophageal fistula should be kept in mind in patients with neck trauma such as hanging, long-term intensive care hospitalization and intubation history.

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Keywords: hanging, tracheoesophageal fistula, nasogastric tube

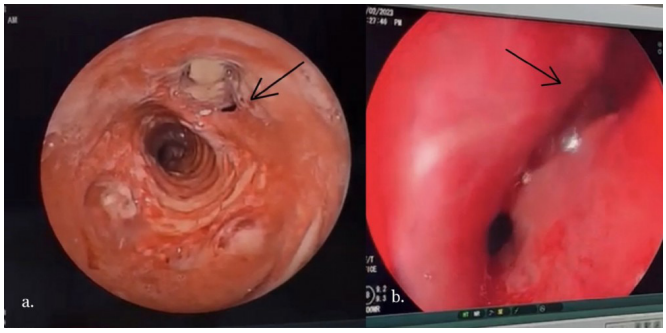

Figure 1a. TEF area and other necrotic areas in bronchoscopy,1.b:TEF area in endoscopy

Infections and antimicrobials

Abstract:0275

A CASE OF SEVERE COMPLICATED MALARIA TREATED WITH ARTEMETHER / LUMEFANTRINE

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Introduction: Malaria is a major public health problem in developing countries because of its high rates of morbidity and mortality. Severe malaria is defined by clinical or laboratory evidence of vital organ dysfunction. Plasmodium falciparum (P. falciparum) is the major cause of severe malaria, which manifests as multiple organ dysfunction with high parasitemia counts that is characterized by coma, stupor, and severe metabolic acidosis. Here, we present a serious case of complicated malaria, which is very rare in our country.

Case: A previously healthy 50-year-old man with no significant past medical history is admitted to our intensive care unit with a severe neurological, renal, and respiratory complicated form of malaria due to P. falciparum. He has been working as a worker for only two months in Benin located in the west of Africa, he had no previous malaria prophylaxis. First, he applied to a local hospital in Benin with complaints of fever, sweating, meaningless speech, and altered mental status. Trophozoites and gametocytes were detected in the thin spread and thick drop preparations. He was intubated because of acute respiratory failure and received hemodialysis due to acute kidney failure. He had received iv artesunate in Benin before he was transferred to our intensive care unit by plane. He had once hemodialysis treatment, 3 package of erythrocyte suspension, fluid replacement therapy, and deep vein thrombosis prophylaxis in our unit. Ring forms characteristic of P. falciparum were detected in thin-spread and thick-drop preparations 5 days after the first artesunate treatment. Artemether /Lumefantrine was given for three days again. He had been successfully extubated after seven days of intensive care follow-up.

Conclusion: Severe falciparum malaria is one of the life-threatening and emergency diseases. Early diagnosis and treatment according to the parasite load in blood is essential (1). Intravenous artesunate is recommended for the treatment of severe malaria. Artemisinin-based Combination Therapies (ACTs) has been used to treat P. falciparum malaria for nearly nine years because parasites became resistant to historical antimalarial drugs and artemisinin monotherapy. Artemisinin is administered in combination with a second, long-acting antimalarial to achieve successful treatment and protect against the development of drug resistance (2).

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Keywords: Malaria, Pfalciparum, Artesunate, Artemether/lumefantrine

Respiratory - Other

Abstract:0281

COEXISTENCE OF NECROTIZING PNEUMONIA AND BRAIN ABSCESS-A CASE REPORT

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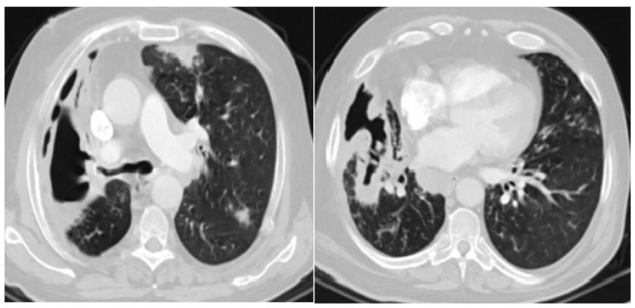
Introduction: Necrotizing pneumonia is a serious infection that disrupts the normal function of the lungs by causing death of lung tissue. It is usually associated with bacterial infections and can rarely spread to other organs in some cases. We aimed to present a case of brain abscess secondary to necrotizing pneumonia.

Case: A 64-year-old male patient was admitted to the intensive care unit after a seizure while being followed up in the chest diseases clinic for 7 days due to necrotizing pneumonia. He had a history of tuberculosis 1 year ago and chest radiograph taken after tuberculosis treatment showed a sequel cavity appearance in the right upper zone. On admission; on physical examination, the patient was unconscious, intubated, sedated (Glaskow coma scale: 3), pulse 130/min, TA: 150/90 mmHg, SpO2: 88%, temperature: 37.2 C°, and decreased respiratory sounds in the right lung. Thorax CT showed cavitary areas with air fluid levels in the upper and middle lobes of the right lung. Ceftazidime+avibactam treatment was started for the patient who had sputum ARB (-) and pseudomonas was grown in the culture. During the follow-up period, the patient had another seizure and was intervened. Electroencephalogram was normal, but neurosurgery clinic was consulted due to the presence of a well-demarcated lesion in the left parietal and right frontal regions on diffusion MRI. Contrast-enhanced brain MRI, which was ordered for differential diagnosis, showed lesions in both frontal lobes, measuring 1.5 cm in the middle frontal gyrus on the right and 22 mm in the left frontal gyrus, with diffusion restriction in the center, annular contrast, and edema around them. Levetiracetam and dexamethasone were started. The patient was operated by neurosurgery and the pathology result was abscess. Antibiotherapy was completed as meropenem, vancomycin and ornidazole for 14 days in accordance with the infectious disease recommendation. The patient was discharged with cure on the 20th day.

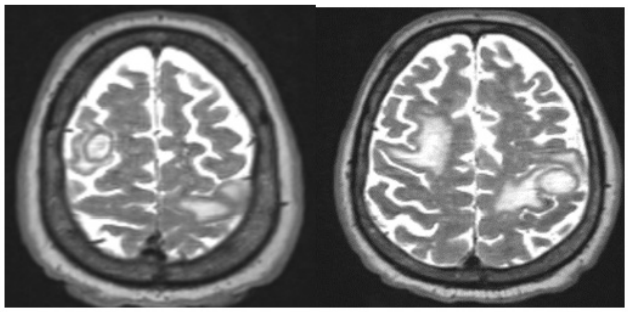
Conclusion: The association of brain abscess and necrotizing pneumonia is a serious and potentially life-threatening condition. Early diagnosis and treatment can increase the chances of recovery and help prevent serious complications. Advanced imaging modalities should be considered in patients with seizures. Abscess should be treated with appropriate antibiotherapy and surgical drainage.

Keywords: antibiotherapy, brain abscess, necrotizing pneumonia, seizure

Radiological Images



Picture 1: Patient's Thoracic CT



Picture 2: Contrast-enhanced Brain MRI of the Patient

Critical care pharmacy and drug monitoring

Abstract:0287

ACUTE PANCREATITIS DUE TO A SUICIDE ATTEMPT WITH OLANZAPINE

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Introduction: Acute pancreatitis (AP) is a reversible inflammatory process of pancreatic tissue. Gallstones, alcohol, hyperlipidemia, hypercalcemia, trauma, infection and drugs are the etiologies. Antipsychotic drugs have been reported to cause AP. It can range from an asymptomatic hyperamylasemia to fatal necrohemorrhagic pancreatitis. In this case, the AP associated with the use of olanzapine for suicidal purpose is presented.

Case: An 18-year-old female patient was referred to our clinic with the preliminary diagnosis of status epilepticus and encephalitis. When she was admitted, she was unconscious, intubated, GCS: 6, light reflexes were normal, neck stiffness, Kernig and Brudzinski signs were negative. Brain MRI, EEG, Brain CT imaging and LP findings were normal. The drug panel was negative in blood. Benzodiazepine was positive in the urine. However, midazolam was administered to the patient before intubation in the center she came to, suspended us from thinking about intoxication. Supportive treatment was started. During the follow-up, the patient regained consciousness on the 2nd day and was extubated. When she was fully consciousness she stated that she used olanzapine (140 mg) for suicidal purposes. A significant increase was observed in amylase and lipase values of the patient with abdominal pain on the 3rd day of his application. (Amylase 118 U/L- 297U/L Lipase 21 U/L- 292 U/L). Pancreatic parenchyma was slightly heterogeneous and increased contamination in abdominal USG. At that time Crp:114 mg/L and WBC:16.17. The patient's triglyceride and glucose levels remained normal. When the patient's clinic and current imaging results were evaluated together, the diagnosis was AP. Abdominal pain regressed after 4 days of supportive treatment, and laboratory findings returned to normal, and she was discharged with full recovery.

Conclusion: Findings related to olanzapine poisoning are; confusion, lethargy, coma, tachycardia, anticholinergic syndrome, myoclonus. Olanzapine-induced pancreatitis has been reported to occur at least 6 months after the start of treatment. Imaging findings in olanzapine-induced AP are acute edematous pancreatitis, hemorrhagic pancreatitis, and necrotizing pancreatitis. Olanzapine can cause AP with hypertriglyceridemia. Also, normal triglyceride levels can be seen. In our patient, pancreatitis developed after 3 days due to acute use of the drug at high doses. The patient's clinical course was mild, with normal triglyceride and enzyme levels. All examinations should be done in detail in patients who come in coma and have suspected intoxication. Pancreatitis should be considered in patients who are known to take antipsychotics and have GIS symptoms. Otherwise, mortality is inevitable.

Keywords: ntoxication, Olanzapine, Pancreatitis, Suicide, Coma

Respiratory - ARDS and acute respiratory failure

Abstract:0291

A PATIENT WITH CARBAMAZEPINE INDUCED DRUG REACTION WITH EOSINOPHILIA AND SYSTEMIC SYMPTOMS SYNDROME

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Introduction: Drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome is severe adverse drug reaction characterized by fever, skin rash, eosinophilia, lymphadenopathy and visceral organ involvement. It is an idiosyncratic reaction to a drug with a prolonged latency period[1]. Visceral involvement is the major cause of morbidity and mortality in this syndrome. Anticonvulsants are the leading cause of DRESS syndrome[2].

Case: An 18-year-old female with epilepsy was admitted to emergency department due to nausea, vomiting and rash for two weeks. On the initial examination, she had submandibular, cervical, axillary lymphadenopathy and diffuse, erythematous skin rash. In the laboratory examination, elevated liver enzymes, hypofibrinogenemia, and eosinophilia were detected. Abdominal ultrasound was revealed findings suggestive of acute liver injury. It was learned that epilepsy treatment was changed to carbamazepine 1 month ago due to the continuation of her seizures. The patient was diagnosed with DRESS syndrome induced by carbamazepine. Carbamazepine was withdrawn immediately, levetiracetam and lacosamide treatment were started. Glucocorticoid therapy was started to treat systemic symptoms. She was treated with amikacin and moxifloxacin due to urinary system infection and pneumonia. In the follow-up, thorax computerized tomography (CT) was taken due to the development of oxygen demand and tachypnea during the hospitalization of the patient. In her CT scan there was bilateral pleural effusion, ground-glass densities and consolidations were evaluated in favor of lymphangitic carcinomatosis in the foreground. Echocardiography was normal. The patient was transferred to intensive care unit due to type-1 respiratory failure. Non-invasive mechanical ventilation and high flow oxygen therapy were applied. Positron Emission Tomography (PET)-CT was

performed to patient for malignancy screening. PET-CT revealed increased FDG uptake in lymph nodes, spleen and bones. Axillary lymph node biopsy and bone marrow biopsy were performed to exclude malignancy. Lymph node biopsy was reported as atypical lymphoid proliferation. Bone marrow biopsy was reported as normocellular bone marrow. Viral serology was negative. With corticosteroid treatment, liver enzymes decreased, rashes disappeared, eosinophilia improved and lymph node sizes decreased. The patient's clinic improved and her need for oxygen disappeared. The patient was discharged with recovery.

Conclusion: Lymphadenopathy, pleural effussion and pulmonary infiltrates that can be mimicked malignancy can be seen in DRESS syndrome. Pathological examination and advanced imaging methods are needed to make the differential diagnosis. In this case, it was shown that atypical lymphoid proliferation, pleural effussion and pulmonary infiltrations were associated with DRESS syndrome. Discontinuation of the causative drug and corticosteroid therapy are the basic principles in treatment.

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Keywords: lymphadenopathy, skin rash, DRESS syndrome, carbamazepine

Table 1. Initial Laboratory Results at The Patient's Admission

Albumin (Kan) - 2,88 g/dL	NÖTROFİL# - 7,5 x10^3/µL
ALT - 288 U/L	EOZİNOFİL# - 1,69 x10^3/µ
AST - 319 U/L	TROMBOSİT - 372 x10^3/µL
ALP - 172 U/L	Anti HAV IgM - 0,09 Negatif
GGT - 599 U/L	Anti-HAV Total - 0,07 Negatif mIU/mL
Bilirubin, total - 5,27 mg/dL	HBs Ag - Negatif
Bilirubin, direkt - 3,729 mg/dL	Anti HBs - 19,61 Pozitif mIU/mL
Bilirubin, indirekt - 1,54 mg/dL	Anti HBc IgM - 0,14 Negatif
HEMOGLOBİN - 12,2 gr/dL	Anti HBc Total - 0,07 Negatif
LÖKOSİT - 16,1 x10^3/µL	Anti HIV - NON-REAKTİF
LENFOSİT# - 4,300 x10^3/µL	Anti HCV - Negatif
MONOSİT# - 2,33 x10^3/µL	Protrombin Zamanı (INR) - 1,47 INR

Paediatrics

Abstract:0295

GENERALISED TETANUS IN A PRETEEN CHILD WITH AUTONOMIC INSTABILITY: A PREVENTABLE EVENT

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Introduction: Tetanus is a largely preventable, infectious neurologic disease contracted through exposure of cuts or wounds to the spores of the bacterium, Clostridium tetani, which live in soil, dust etc.

Case: A 12-year-old male was admitted into the intensive care unit from the children emergency department for post cardiac arrest care with a diagnosis of severe tetanus and sepsis. He presented with a 7-day history of fall from a bike, left sided facial injury and generalized body pains, a 2-day history of facial deviation, neck stiffness, teeth clenching, photophobia and few hours history of fever. Patient was not immunized at birth based on parental belief. Upon admission a diagnosis of tetanus with facial nerve palsy was made, administration of anti-tetanus serum (ATS) 0.5mg and tetanus toxoid(immunoglobulin) 5000IU IM and IV after a test dose of 160 IU, intravenous antibiotics (Ceftriaxone and metronidazole) with wound debridement commenced. Developed spasms while on admission and commenced on phenobarbitone and chlorpromazine, with associated autonomic instability, and on the 4th day of admission, he had a cardiac arrest, was successfully resuscitated, and transferred to the ICU.

On admission in the ICU, he was intubated and mechanically ventilated on volume controlled mode, placed on atracurium for muscle paralysis, midazolam infusion for sedation, magnesium sulphate for when hypertensive, iv lidocaine for arrythmias, fentanyl infusion for analgesia, omeprazole for ulcer prophylaxis, enoxaparin for DVT prophylaxis and NG feeding, antibiotics were changed from ceftriaxone to ciprofloxacin on micro culture and advice while continuing metronidazole.

He had SVTs and propranolol prescribed, developed hypotension, MgS04 discontinued and placed on adrenaline infusion. Spasms continued and muscle relaxant changed to pancuronium due to persistent hypotension with adjustment of sedation. There was continued family update about the prognosis.

On day 4 in ICU, patient was observed to be desaturating with poor air entry, had emergency reintubation, and endotracheal tube was observed to be blocked with mucus plug. It was a difficult intubation due to swollen tongue and had a cardiac arrest at reintubation and was successfully resuscitated.

By day 5 in the ICU,16 days after injury, antibiotics was changed to amikacin and planned to consider intrathecal tetanus immunoglobulin. He subsequently developed severe hypotension later at night despite being on adrenaline infusion and had a cardiac arrest for which resuscitation proved abortive.

Conclusion: Tetanus in unvaccinated children carries a high fatality rate. The need for continued immunization advocacy and booster doses as recommended is paramount in low resource countries.

Keywords: generalised tetanus, unvaccinated, autonomic instability,preteen

Hematology and Oncology

Abstract:0297

HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS IN AN ADULT PATIENT

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Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a life-threatening condition resulting from excessive activation and proliferation of macrophages accompanying systemic inflammatory disorders. HLH can develop due to metabolic diseases, immune deficiencies, collagen tissue diseases, malignancies as well as infections. In this case report, we present a patient who was admitted to our intensive care unit due to sepsis, followed by HLH and her clinical worsening rapidly.

Case: A 29-year-old female without any known additional disease was admitted to the hospital with complaints of recurrent fever, weakness and vomiting, hospitalized in terms of fever etiology. Lung tomography was normal. She was operated on the abdomen with the suspicion of an abscess or cyst but there was no abscess. Antibiotic therapy was extended. She was transferred to our intensive care unit with the preliminary diagnosis of sepsis. Her glasgow coma scale:15, temperature:38.5, pulse:123, blood pressure:130/80. In physical examination, lung sounds were deep, there was hepatosplenomegaly. Pleural fluid was observed in the control tomography. There was no obvious intra-abdominal infection focus and hepatosplenomegaly is present, with no sign of infection in the pleural fluid sampling. Echocardiography was normal. Infection markers and autoimmune markers were negative. C3 level was normal, and C4 level was low. There were high white blood cells, low hemoglobin and platelet levels. In peripheral smear, neutrophil count was significantly high with toxic granulation in neutrophils. Immunoglobulins were normal. Ferritin remained high (>65.000). Genetic disorders not found. On the 3rd day of the follow-up, ARDS-related respiratory failure developed, she was intubated. Bone marrow biopsy was performed when bi-cytopenia deepened. Hematological malignancy was not found, histiocyte and lymphoplasmocytic cells were increased. Fever, hepatosplenomegaly, bi-cytopenia, hypertriglyceridemia, hypofibrinogenemia and the patient was evaluated as HLH. Cytokine filter treatment was performed. After the treatment regimen containing etoposide and cyclosporine was started, neutropenia developed. She went into septic shock, and died on the 38th day of hospitalization.

Conclusion: HLH can occur with primary and secondary causes and usually fatal. Although the main treatment in secondary HLH is directed towards the underlying disease, initiation of a chemotherapy protocol as in primary HLH may be life-saving if the clinical findings worsen. In our case, a picture of HLH occurred due to the trigger of a possible infectious agent, HLH chemotherapy was started due to the clinical progress despite supportive treatments including antibiotics, but the patient died rapidly.

Keywords: Hemophagocytosis, Immunosuppression, Cytopenia, Hepatosplenomegaly, Hyperferritinemia

Respiratory - Other

Abstract:0299

RESPIRATORY FAILURE IN OSLER-WEBER-RENDU SYNDROME CASE

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Introduction: Hereditary hemorrhagic telangiectasia (HHT; also called Osler-Weber-Rendu syndrome -OWR) is a vascular disorder inherited as an autosomal dominant trait, with a variety of clinical manifestations. The most common problems are epistaxis, gastrointestinal bleeding, and iron deficiency anemia, along with characteristic mucocutaneous telangiectasia. In addition, arteriovenous malformations (AVMs) frequently affect the pulmonary, hepatic, and/or cerebral circulations. In this case, a patient with a diagnosis of OWR who progressed with hypoxia that did not improve will be presented.

Case: A 42-year-old patient diagnosed with OWR syndrome was admitted to the emergency department due to respiratory distress for days. She was admitted to the intensive care unit because of her significant hypoxia. She had a history of intervention due to multiple pulmonary AVM and a cerebral ischemic event. Thoracic tomography revealed nodular appearances consistent with multiple AVMs and lobar pneumonia in the lung. Antibiotic therapy was started with recommendations for infectious diseases. In the arterial blood gas at the beginning of the treatment, the partial oxygen pressure of the patient was 59.2 and the p/f ratio was 159. The patient's infective status gradually regressed, and a significant regression was observed in pneumonic involvement in the radiographs. However, there was no significant improvement in the patient's oxygenation. It was thought to be due to arteriovenous shunts in the lung. The patient was consulted by the interventional radiology department, and multiple coils and embolization were performed. After the procedure, oxygenation was increased to normal levels.

Conclusion: In OWR patients, pulmonary arterial blood passing through these right-to-left shunts cannot be oxygenated, leading to hypoxemia. In our patient, respiratory failure deepened by pneumonia due to these were considered. Some patients are asymptomatic and these fistulas are found by chance. It should definitely be considered in OWR patients with hypoxia.

Keywords: Osler Weber Rendu Syndrome, Pulmonary shunt, Hypoksia

Respiratory - Other

Abstract:0300

A CASE OF TRACHEAL MALACIA IN A PATIENT WITH GOITER

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Introduction: Tracheomalacia (TM) refers to diffuse or segmental tracheal weakness. Chronic compression of the trachea can cause TM. This is most commonly due to a benign mediastinal goiter. In our case, tracheal malaise due to compression of the large thyroid gland will be presented.

Case: A 73-year-old female patient presented to the emergency department with severe shortness of breath for the past week. He had been describing shortness of breath for the past year. He had a history of Parkinson's disease, heart failure, and hypertension. The patient's arrival saturation was measured as 52%. It rose to the level of 92% with 4 lt/min oxygen therapy. The patient was admitted to the intensive care unit. In the ultrasonography of the patient, the size of the right lobe of the thyroid gland was measured as 40x40x57 cm, and the size of the left lobe was 29x34x58 cm. Significant narrowing of the trachea was observed at the level of the thyroid gland in the tomography. It was measured as 2.5 mm at its narrowest point. The operation was planned by the thoracic surgeon and followed up with a tracheal stent and tracheostomy. The patient, whose general condition and saturation improved significantly, was discharged.

Conclusion: TM can be asymptomatic, especially if the airway narrowing is mild. However, symptoms and signs frequently develop as the severity of airway narrowing progresses, if the patient becomes challenged, or in certain clinical situations. In this case, we wanted to show that although it is rare in the etiology of hypoxia, malaise due to thyroid gland diseases should also be kept in mind.

Keywords: Goiter, Tracheal Stent, Tracheal Malasia

Respiratory - ARDS and acute respiratory failure

Abstract:0303

PLASMODIUM FALCIPARUM MALARIA IN AN EXCHANGE STUDENT PATIENT: A CASE REPORT

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Introduction: Malaria is transmitted to humans by the bite of female Anopheles mosquitoes infected with the plasmodium parasite and is an endemic disease in tropical countries, especially sub-Saharan Africa. There are four species of plasmodium: P.vivax, P.ovale, P.falciparum and P.malaria that cause infection in humans. P.vivax is the most frequently detected species in our country and in the world. The disease caused by P. falciparum is more severe than other species, the risk of case mortality is high, and P. falciparum malaria left untreated is always fatal. Early diagnosis and appropriate treatment are the most important factors in reducing complications and preventing deaths.

Case: A 21 year-old, female exchange student from Cameroon was admitted to our hospital's emergency department with high fever and fatigue. The patient was admitted to intensive care unit, due to acute respiratory failure with PaO_2/FiO_2 ratio 166.2, SOFA score 6. Differential diagnoses included sepsis, hemolytic uremic syndrome, malaria. Physical examination was normal except for hepatosplenomegaly via deep palpation. Vital signs with oxygen mask showed blood pressure 105/ 47 mmHg, 129 beats/minute- sinus tachycardia, respiratory rate 28/min, SpO_2 95% and body temperature 38,4 degrees celsius Laboratory results showed that decreased glomerular filtration rate with 57 ml/min/ 1.73m2, increased AST level to 60 IU/L, severe anemia with Hb level of 6.4 g/L, thrombocytopenia (28 10^3/ul, Pct: 0.03%), and high CRP 313.4 mg/L. Wide spectrum antibiotic as piperacillin-tazobactam was administered. Ministery of Health laboratory for malaria contacted our unit to inform us about positive results for Plasmodium Falciparum in both peripheral blood smear and rapid diagnostic testing. Treatment regimen included Artesunate, Artemether and Lumefantrine. During follow up, arterial blood gas showed PaO_2/FiO_2 ratio 100 and Rox index: 2.9 and we proceeded to intubate, the patient was placed on a prone position. Lung protective mechanical ventilation strategies was applied (Peep: 10, Pplato:25, Static Compliance:20). Continuous renal replacement with blood purification therapy was administered who had KDIGO Acute Kidney Injury stage 1 for three days. Patient was successfully extubated on the 10th day. On the 12th day the patient was discharged to the infectious disease ward after successful treatment.

Conclusion: Travel history should be taken thoroughly in patients presenting with acute respiratory failure, anaemia and fever. In addition, Malaria should always be taken into consideration during differential diagnosis.

Keywords: Plasmodium Falciparum, Malaria, ARDS

Hematology and Oncology

Abstract:0304

ACQUIRED THROMBOTIC THROMBOCYTOPENIC PURPURA WITH PANCREATITIS AT DISEASE ONSET IN SYSTEMIC LUPUS ERYTHEMATOSUS: SUCCESSFUL MANAGEMENT OF A RARE PRESENTATION

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Introduction: Systemic lupus erythematosus (SLE) is a complex autoimmune disease. Any type of organ -involvement could be found in patients with SLE. Acute pancreatitis as the initial manifestation of SLE has been rarely reported. Mechanisms of pancreatitis in SLE patients include immune complexes precipitation in the pancreatic vessels, pancreatic micro thrombosis, occlusion of arteries and antiphospholipid syndrome or drugs such as tacrolimus, azathiopurine. Thrombotic thrombocytopenic purpura (TTP) in patients with SLE is extremely rare. TTP as the first manifestation of SLE is even rarer. Here, we report a patient presented with acute pancreatitis and diagnosed with SLE related TTP; successfully treated with plasmapheresis, corticosteroids and cyclophosphamide.

Case: A 57-year-old women without any history of chronic disease or drug consumption referred to emergency department with fever, stomach ache and confusion. She had a tenderness of right upper quadrant and epigastric regions. In laboratory data, blood urea nitrogen 119 mg/dL, lipase 656 U/L, creatinine 2,06 mg/dL and platelet count was 26.000 /uL. On CT scan, the severity of pancreatitis was detected mild. There were no gall bladder stones and history of smoking or alcohol consumption. Calcium and triglyceride values were also in normal ranges. Lactate dehydrogenase enzyme was 1095 U/L. Haptoglobuline level was found low and there were more than 3-4% schistocytes observed in peripheral smear. Plasma exchange and corticosteroid treatment was started urgently for the treatment of thrombotic microangiopathy. Complement C3 and C4 levels were low. Antinuclear antibody (ANA) was 1/1280 titer. In follow-up, patient's platelet count increased and also her confusion and stomach ache improved. A disintegrin and metalloproteinase with thrombospondin type 1 motifs, member 13 (ADAMTs-13) activity was detected %0,25, antigen level was in normal ranges but the inhibitor level was determined high. She had proteinuria (3g/day) and hematuria on microscopic assay. The diagnosis of SLE based on the Systemic Lupus International Collaborating Clinics (SLICC) criteria with high ANA concentration, low complement, proteinuria and neurologic involvement was established. Cyclophosphamide was given as maintenance therapy after plasmapheresis due to TTP associated with SLE.

Conclusion: Pancreatitis is a rare but severe involvement in SLE. The precise etiopathogenesis is not known and most probably multiple mechanisms are involved. TTP was the heralding manifestation in our patient which is a life-threatening disease and only presents in 0.5-2% of SLE patients. In our patient, it took a high index of suspicion spurred by the emergence of low complement assay, an immunologic finding, proteinuria and neurologic involvement. Although neurologic findings can be also a finding of TTP, low complement assay and high ANA titers are not expected findings in TTP.

Awareness should be given to the possible autoimmune diseases in acute pancreatitis.

Keywords: TTP, pancreatitis, thrombotic microangyopathy, systemic lupus erythematosus

Values \ Treatment	Plex D1	Plex D2	Plex D3	Plex D4	Plex D5	Plex D6
				750 MP	750 MP	750 MP
Urea/Creatinin (mg/dL)	219/2,0	103/1,3	106/1,0	73/0,8	56/0,7	62/0,7	59/0,7
T.bil/D.bil (mg/dL)	2,1/1,4	5,7/4,8	3,1/2,1	4,2/3,0	2,0/1,2	1,7/1,08	1,4/0,7
LDH (U/L)	1095	858	477	572	474		372
INR/Fibrinogen (mg/dL)	1,3/6,7	1,2/389	1,3/291	1,2/243	1,2/200	1,1/192	1,2/154
Haptoglobin (mg/dL)	<10	<10	<10	<10			
Reticulocytes %	1,3	1,4	1,2	2,1			
Hemoglobin (gr/dL)	16,3	13,6	9,7	9,3	9,2	8,7	8,6
Platelet (10^3/µL)	26,000	6,000	19,000	10,000	55,000	117,000	166,000

Figure 1. Laboratory results and treatment data

Respiratory - Other

Abstract:0315

EXTUBATION FAILURE IN A PATIENT WITH GLUTARIC ACIDURIA TYPE 1

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Introduction: Glutaric aciduria type 1 (GAT1) is an inherited neurometabolic disorder of lysine and tryptophan metabolism. Striatal degeneration and dystonic movement disorders are generally seen in patients with GAT1. Those patients are prone to pulmonary aspiration due to swallowing problems. [1,2]

Case: A 21-year-old female patient with GAT1 was admitted to the Medical Intensive Care Unit (MICU) after she had been intubated in the ward on the 5th day of pneumonia treatment. She had sialorrhea, dystonia and scoliosis. Rhonchi were detected bilaterally in auscultation. Ipratropium bromide and salbutamol treatment were started. The choice of antibiotic was changed to piperacillin-tazobactam from moxifloxacin. Invasive mechanical ventilation was continued via endotracheal tube. A low protein diet, restricted in lysine and tryptophan, was given to the patient and carnitine treatment was started. On the second day of the MICU admission, rapid shallow breathing index (RSBI) was 76 breaths/minute and negative inspiratory force (NIF) was -34cmH2O. The fraction of inspired oxygen was 25%; therefore, patient was extubated after a successful spontaneous breathing trial (SBT). However, she was reentubated 8 hours after extubation to prevent aspiration due to sialorrhea and difficulty in swallowing. We suggested performing a tracheostomy but parents of the patient didn't give consent for the procedure. The choice of antibiotic was changed to meropenem due to deterioration of the clinical and respiratory status during the follow-up of the patient. However, on the 9th day in the MICU, her clinical and respiratory status improved. She was ready to wean according to the weaning parameters. She was extubated after a successful SBT. Nevertheless, she was reentubated on the 11th day in the MICU, because of difficulty in swallowing, sialorrhea, dyspnea and increased need of oxygen. We informed the parents of the patient. Tracheostomy was planned after they gave the consent.

Conclusion: Early tracheostomy and percutaneous gastrostomy procedures should be considered in patients with GAT1 when there is high risk of aspiration due to swallowing problems.

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Keywords: glutaric aciduria type 1, ventilator weaning, swallowing disorder, tracheostomy

Sepsis - Management

Abstract:0316

SUCCESSFUL MANAGEMENT OF A PREGNANT PATIENT WITH SEPTIC SHOCK DUE TO ACALCULOUS CHOLECYSTITIS: A CASE REPORT

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Introduction: Sepsis is a life-threatening organ dysfunction that results from the body's response to infection. It requires prompt recognition, appropriate antibiotics, careful hemodynamic support, and control of the source of infection. In this case, we present a pregnant patient with septic shock due to acalculous cholecystitis.

Case: A 43-year-old female patient, 21 weeks pregnant, applied to another hospital with complaints of headache, chills and shivering. Pancytopenia was detected. Brucella was diagnosed and treated. As her complaints continued, she was admitted to the infectious diseases clinic of the Dicle University Hospital.

Laboratory parameters were wbc: 5 plt:28 t.bil:5 ast: 170 ldh: 808 cre:1.33 crp: 328. Obstetrics clinic did not consider emergency obstetric and gynecological intervention. Internal medicine clinic: According to the abdominal USG report, sepsis and septic shock due to acalculous cholecystitis were considered.

22.02.2023. The patient was transferred to the general surgery intensive care unit. (BP: 70 /25 mmHg, Pulse: 120 BT: 37.6). Hematology clinic consultation: In the examination of the peripheral smear, it was primarily thought to be secondary to septic shock. It was noted that she could be operated after 1 unit of apheresis platelet was given. Obstetrics and gynecology consultation: After medical abortion, she was transferred to the Internal Medicine intensive care unit.

23.02.2023: Rose Bengal and capture brucella were negative. Her general condition is poor, she is hypotensive, and was started on steradine. 2 units of apheresis platelets were givenO2 support was given with HFNO because her tachypnea and PaO2 were low. Sofa score was 14 points, septic shock and multi-organ failure were considered in the patient. Meropenem and Vancomycin 2*1 gr were started. Pneumatic compression was applied.

24.02.2023: Fibrinogen:1.52 (2-4), d-dimer:8.78(0-0.5) thrombocytopenia and schistocyte were detected in peripheral smear. Disseminated intravascular coagulation was considered in the patient. Haemocomplettan was applied. The tachypnea continued and the PaO2 decreased. The patient was electively intubated.

25.02.2023: 1 unit of ES and 1 unit of PLT was applied.

26.02.2023: Steradine is administered. plt: 73,000/ml, intubated.

27.02.2023: 0.03 mcg/kg/min steradine is administered. Hgb:7.5 gr/dl, 2 units of ES were applied. Weaning protocol started. The patient was extubated.

28.02.2023: Clinically stable, breathing in room air, O2 saturation: 95-98%, fibrinogen: 2.18, d-dimer: 0.05, thrombocyte:189.000, the patient was transferred to infectious diseases with cure.

Conclusion: Early management of sepsis and septic shock is crucial for patients' prognosis. Early diagnosis and early treatment of sepsis saves lives.

Keywords: sepsis, septic shock, dissemine ıntravascular coagulation, multiorgan failure, early diagnosis and treatment

Metabolism - Nutrition

Abstract:0318

UNUSUAL CAUSE OF CARDIAC ARREST: REFEEDING SYNDROME

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Introduction: Refeeding syndrome is an exaggerated physiological response to glucose intake after prolonged starvation. Refeeding activates anabolic processes that cause intracellular shift of glucose, water and electrolytes. Due to electrolyte disorders arryhtmia, lung edema, acute heart failure and hemodynamic collapse may occur (1). Hereby, we aimed to present a patient who experienced cardiac arrest due to refeeding syndrome.

Case: A 28 year-old male patient with a history of Type 1 diabetes and alcohol addiction presented with nausea, vomiting and reduction in oral intake for 10 days. Patient was on insulin aspart and risperidone treatment. In emergency room physical examination was normal but transaminase levels were high (AST:129U/L) ALT:83U/L). The patient was admitted to gastroenterology ward and given antiemetic medication. On the 2nd day, symptomes disappeared and the patient started oral intake. Although transaminase levels were decreasing, on the 5th day the patient suddenly collapsed and experienced cardiac arrest. The patient was intubated and ROSC was observed after CPR lasting 25 minutes. The patient was admitted to our medical ICU and detailed laboratory examinations revealed a serum phosphorus level of 0.69 mg/dL and a potassium level of 3.09 mg/dL. Corrected QTc was 600 ms (Table). It was noticed that serum phosphorus level monitoring was not performed during the follow-up on ward. The patient who started oral intake after 15 days was diagnosed with refeeding syndrome causing hypophosphatemia, prolonged QTc and cardiac arrest. Intravenous thiamine, electrolyte and hydration were administered. After serum phosphorus and potassium levels reached normal ranges, enteral nutrition was started gradually. Normal QTc was observed after 48 hours. Patient was extubated on the 11st day of ICU admission. The patient who did not have neurological dysfunction and had normal performance status was discharged on the 15th day.

Conclusion: Refeeding syndrome should be kept in mind for hospitalized patients who experienced prolonged starvation. Serum electrolyte levels should be monitored closely and nutrition therapy should be administered gradually with thiamine replacement.

Keywords: Refeeding syndrome, intensive care, critical care, hypophosphatemia, QT Interval

Reference

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Table 1. Daily Laboratory Parameters

Day	1	2	3	4	5 (cardiac arrest)	6	7	8	9
Lactate (mmol/L)	2.0	3.0	3.1	5.4	9.5	1.7	2.0	1.9	1.3
Sodium (mmol/L)	132	132	131	136	141	142	139	136	136
Potassium (mmol/L)	3.66	3.04	3.84	3.47	3.23	3.09	3.55	3.48	3.72
Phosphorus (mg/ dL)	-	-	-	-	-	0.69	1.81	2.34	2.64
Magnesium (mg/ dL)	-	-	-	-	-	3.13	2.22	2.02	1.95

Perioperative critical care

Abstract:0319

INTRAOPERATIVE AND ICU MANAGEMENT OF NEAR-FATAL ANAPHYLACTOID REACTION DURING REGIONAL ANESTHESIA

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Introduction: Anaphylactic reactions during anesthesia incidence rate ranged between 1 in 4000 to 1 in 25000 cases. Regional anesthesia has been associated with lower incidence rate. However, the occurrence of such a case may lead to catastrophic events. In this paper, we report a case of an anaphylactoid reaction occurring during spinal anesthesia, its intraoperative management as well as ICU management.

Case: A 19-year-old Asian male was admitted to the hospital with varicocele and was planned for surgery under spinal anesthesia. His physical status was ASA I. No history of allergies or asthma were reported, thus no screening for sensitivity to any medication performed. On the day of surgery, an IV line was established, and 2 grams of cefazolin were administered. Spinal anesthesia was performed at the level of the L3-L4 intervertebral space using 2,5 mL Bupivacaine 0.5% Heavy. At the end of the surgery patient complained of burning sensation, coughing and shortness of breath. Expiratory wheezing was present, followed by hypotension, bradiarrhythmia and asystole. Cardiopulmonary resuscitation was performed and an endotracheal tube was placed. Intravenous push of two milligrams of epinephrine were administered, followed by 1 mg epinephrine every three minutes. Intravenous dexamethasone and diphenhydramine were also given during the CPR. His circulation returned after 8 minutes. Then he was put on mechanical ventilation, inotropic and vasopressor support. Once stabilized, he was immediately transferred to the ICU. During the ICU stay, morphine was administered as analgesia, while sedation is maintained with midazolam. Sedation levels were evaluated every 24 hours. Paracetamol and phenobarbital were administered to prevent fever and seizures. Hematologic study suggests an acute hypersensitivity reaction. The corticosteroid was continued and tapered down after 3 days. On the third day of ICU stay, he returned cooperative and spontaneously breathing. No events of seizure or fever were documented. After recovery, he refused the tests to determine the allergic cause and was then discharged.

Conclusion: Perioperative anaphylactoid reaction may occur during regional anesthesia despite its low incidence rate. It may be triggered by hypersensitivity or other causes. Cardiac arrest may follow

anaphylactoid reaction, and poses serious risks to the patient. Rapid recognition, prompt intraoperative intervention, and proper post resuscitation management is essential for survival. A standardized algorithm possibly required to achieve better outcomes.

Keywords: Anaphylactoid reaction, Cardiac arrest, Regional anesthesia

Respiratory - Other

Abstract:0321

AN INTERESTING CASE OF OVERLAP SYNDROME THAT HAD OVERLAPPED WITH ACUTE INFLUENZA INFECTION AND FUNGAL HEALTH CARE ASSOCIATED PNEUMONIA

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Introduction: Overlap syndrome is the coexistence of obstructive sleep apnea (OSA) in patients with Chronic Obstructive Pulmonary Disease (COPD). It is an important cause of morbidity and mortality among ICU patients albeit often overlooked. This case demonstrates the need for clinicians to clinically evaluate patients with OSA or COPD for the occurrence of OS and provide effective treatment options for both for optimal outcomes.

Case: A 67-year-old female, a diabetic and hypertensive patient was admitted in a tertiary care center with history of fever, cough with expectoration and drowsiness. She was promptly intubated and was started on empirical broad-spectrum antibiotics. Her throat swab was positive for Influenza A and oseltamivir was promptly initiated. She was successfully extubated after 4 days but continued to be on oxygen support. Her respiratory cultures had arrived which had yielded Candida Tropicalis and Aspergillus. She was started on oral voriconazole and was discharged with same along with home oxygen after a total of 8 days hospital stay.

At home, the patient continued to receive oxygen continuously, however her mentation continued to be less than satisfactory. However, she became completely unresponsive and tachypneic within a week following discharge and was rushed to our center. She was started on NIV, empirical antibiotics and inhaled bronchodilators. Intubation was deferred in view of strong airway reflexes despite apparently poor mentation. Her sensorium improved; hence she was weaned off to intermittent NIV. While on oxygen episodes of hypoxemia was evident on monitor, hence NIV was continued at night. After stabilizing, the patient underwent a level 2 polysomnography, which proved severe Obstructive Sleep Apnea. A pulmonary function test was also performed which had proven severe obstruction. The patient is conscious and oriented with no daytime drowsiness or somnolence, saturating normally at room air.

Conclusion: Prompt diagnosis of Obstructive Sleep Apnea and management of same improves outcomes in all cases of respiratory failure and a high index of suspicion is necessary for same.

Keywords: Obstructive Sleep Apnea, Overlap Syndrome, Obstructive Airway Disease

Hematology and Oncology

Abstract:0326

A RARE CASE OF HYPEREOSINOPHILIC SYNDROME

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Introduction: Eosinophilic lung diseases are a diverse group of pulmonary disorders associated with peripheral or tissue eosinophilia. Among these diseases, the hypereosinophilic syndromes (HES) are a group of disorders marked by the sustained overproduction of eosinophils, in which eosinophilic infiltration and mediator release cause damage to multiple organs. The most common targets are the skin, lungs, digestive tract, heart, blood and nervous system. Untreated, HES can become life-threatening. Hypereosinophilia (HE) is defined as an absolute eosinophil count of over 1500 cells/microL in the peripheral blood on two examinations separated in time by at least one month and/or pathologic confirmation of tissue HE.

Case: A 40-year-old male patient was admitted to our clinic with progressive shortness of breath and cough. He also complained about severe chest pain during the exertion for the last few days. He was treated for Crohn disease for 8 years with azathioprine. He informed that he had been diagnosed with eosinophilic polyangiitis disease 3 months priorly and had been taking methylprednisolone during this period of time. At the admission, his blood eosinophil level was 17000 with over 30000 leukocytosis. Chest computed tomography showed ground glass lung infiltrations in both lung field. His oxygen level was measured normal at room air. Other organ function tests were within normal limits except troponin which was detected 1230ng/L (normal limit is 50ng/L). He immediately consulted with cardiologist and heart function test were detected normal. With a preliminary diagnosis of cardiopulmonary involvement of possible HES he was transferred to hematology department. He was started high dose methylprednisolone treatment due to eosinophilic heart damage. Subsequently, methylprednisolone treatment was replaced with hydroxyurea and imatinib. However, his lung involvement progressed and he was hypoxemic in the room air. Blood HE was also progressed. He started high dose methylprednisolone again and hydroxyurea, imatinib treatments were discontinued. After this treatment strategy, he becomes normoxemic at the room air.

Conclusion: This is very rare and severe case of young patient with HES who still been investigated for optimal treatment for HE. We presented this case to be aware of this unique condition.

Keywords: Hypereosinophilic syndrome, Hematologic malinancy, ANCA

Kidney - Acute kidney injury

Abstract:0328

UNIQUE CASE OF VENLAFAXINE INDUCED RHABDOMYOLYSIS

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Introduction: Venlafaxine is a serotonin and norepinephrine neuronal reuptake inhibitor. There are reported cases of venlafaxine toxicity at overdose levels including central nervous system depression, seizures, and cardiovascular toxicity. Here we introduce a venlafaxine induced rhabdomyolysis patient who took over 10000 mg of venlafaxine.

Case: A 22-year-old female patient with previously diagnosed severe depression, currently on medication of venlafaxine 150 mg, olanzapine 2,5 mg and mirtazapine 15mg was brought to the emergency department by her friend who found her confused status with an empty bottle of venlafaxine. During the admission her GKS-15 and pupils were mydriatic. She admitted that she had taken 65-70 pieces of 150 mg venlafaxine. She has never smoked and her medical records were unremarkable. Subsequently, she had two separate generalized tonic-clonic seizures that lasted a few seconds. To protect her airway and prevent further seizures, she was intubated and sedated. She was transferred to our hospital in an intubated state. Routine blood tests showed slightly increased liver and kidney function tests. Creatinine kinase level was detected at a very high level of 15000 U/L. Transthoracic echocardiogram revealed mitral valvular prolapsus with normal ejection fraction. Chest X-ray on the admission day was normal. The following day her organ function tests worsened and lung consolidations appeared in both lung fields, compatible with possible aspiration and pulmonary edema. Nevertheless, she could be extubated with success. Liver and kidney function tests deteriorated. She underwent hemodialysis with ultrafiltration twice due to rhabdomyolysis induced acute kidney failure. Eventually, on the 5th day of admission, she was discharged with good health.

Conclusion: There are reported cases of venlafaxine induced rhabdomyolysis in the literature. Our case is unique because despite her intoxication with such a high dose of venlafaxine, she was able to be discharged to her home on the 5th day of her intensive care unit admission without any organ damage. Our case indicates that rapid and optimal intervention could prevent serious complications that could have developed.

Keywords: rhabdomyolysis, venlafaxine, acute kidney injury, suicidal patient, tonic clonic seizure

Neuro-intensive care

Abstract:0331

OSMOTIC DEMYELINATION SYNDROME WITH RARE CORTICAL INVOLVEMENT: A CHALLENGING CASE IN A PATIENT WITH ALCOHOL CONSUMPTION AND CENTRAL DIABETES INSIPIDUS

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Introduction: Osmotic Demyelination Syndrome (ODS) is a rare clinical entity characterized by myelin loss in various brain regions due to abrupt osmolar changes, primarily associated with inadequate correction of hyponatremia. Uncommon cases of cortical involvement affecting the grey matter have been reported, with progressive neurological symptoms and deterioration of consciousness.

Case: We present the case of a 49-year-old man with a history of chronic alcohol consumption and central diabetes insipidus, who developed ODS with involvement of the bilateral primary motor cortex, basal ganglia, and pons. The patient experienced rapid deterioration of consciousness and eventually fell into a coma. Serial magnetic resonance imaging revealed extensive and progressive involvement.

This case illustrates a rare presentation of ODS with cerebral cortical involvement in a patient with known risk factors, such as alcohol consumption. The rapid correction of sodium in this patient, from 102 mEq/L to 126 mEq/L in 18 hours, is a significant factor in the development of ODS. Special attention must be given to the rate of sodium correction in patients with risk factors. Furthermore, the diagnosis of ODS can be challenging due to the lack of specific findings on neuroimaging tests in the early stages of the disease.

Conclusions: The management of patients with ODS can be challenging, especially in those with complex medical histories. It is essential to continue researching and improving management and treatment strategies for patients with ODS, particularly in those with risk factors and atypical presentations.

Keywords: Osmotic Demyelination Syndrome, Hyponatremia, Central Diabetes Insipidus, Alcohol-Related Disorders.

Trauma

Abstract:0344

SYSTEMIC CAPILLARY LEAK SYNDROME RECURRENT AFTER SARS-COV-2 INFECTION: A SCOPING REVIEW IN RELATION TO A CLINICAL CASE

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Introduction: Systemic capillary leak syndrome (SCLS), also called Clarkson's disease, is a severe, rare disease characterized by hypovolemic shock secondary to capillary leak which occurs in the context of monoclonal gammopathy (1). We conducted a scoping review and reported the case of a patient who developed this disease recurrently, in the context of syndrome coronavirus 2 (SARS-CoV-2) infection.

Case: Male whose first episode (debut) was developed in 2021 when he was 38 years old. He had received the second dose of the BNT162b2 vaccine six days before the admission and had acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection confirmed by polymerase chain reaction test (PCR). In the emergency room, hypotension, sinus tachycardia, and poor peripheral perfusion were observed. He presented with a capillary refill greater than 5 sec and coldness of the lower limbs. He was admitted to the intensive care unit (ICU) with a Simplified Acute Physiology Score (SAPS) II of 61 points and a Sequential Organ Failure Assessment (SOFA) score of 7 points. Our patient required respiratory, hemodynamic, renal support and developed rhabdomyolysis, but not compartment syndrome. Moreover, had a high hemoconcentration with a hemoglobin (Hb) of 24 g/dl, which required significant intravenous fluid therapy. The patient had hypogammaglobulinemia and clonality in IgG and IgA kappa. Subsequently, he showed progressive improvement with gradual deescalation of life support measures and was discharged from the intensive care unit (ICU) on day 9. The second episode occurred in 2023 when the patient was 39 years old. He had a positive PCR for SARS-CoV-2 with no respiratory symptoms. The patient presented with criteria compatible with hypovolemic shock, increased hematocrit, dehydration, and hypoalbuminemia, with a SAPS II of 29 and SOFA score of 4. He presented hemoconcentration with Hb of 22 g/dl that improved after expansion with crystalloids. In this episode, the patient did not require respiratory or renal support and was discharged on day 3 of his admission to the ICU.

It is believed that SCLS is due to an exaggerated response to inflammatory changes. In the first episode, the origin was not clearly determined, since the patient's vaccination intercurred with acute SARS-CoV-2 infection; in the second episode, the only trigger was acute SARS-CoV-2 infection. Table 1 summarizes the main findings of our case and those published.

Conclusion: SCLS is a rare disease but has severe clinical implications. Recurrent cases are exceptional but lead to increased morbidity, the therapeutic management of which remains uncertain.

Keywords: Systemic capillary leak, Rhabdomyolysis, SARS-CoV2.

Table 1. Characteristics of systemic capillary leak syndrome (SCLS) cases with SARS-CoV-2 infection.

Article/current report	Age	Gender	Previous SCLS	Gammopathy	Trigger	Hemoglobin (g/dL)	Albumin (g/dL)	Creatinine (mg/dL)	Lactate (mmol/L)	IVIg	Evolution and Outcomes
Current report: episode 1 (2021)	38	M	No	IgG Kappa IgA Kappa	PCR SARS-CoV-2 + BNT162b2 vaccine (Pfizer)	23.6	1.7	3.8	4.0	No	Rhabdomyolysis. IMV and CRRT. Recovery.
Current report: episode 2 (2023)	39	M	Yes	IgG Kappa IgA Kappa	PCR SARS-CoV-2 +	22	2.3	1,99	6.11	No	Rhabdomyolysis. Recovery.
Choi (2021)	38	M	No	Multiple myeloma	Ad26.COV2-S vaccine (Janssen)	22.7	3.3	2.0	5.4	No	Death
Robichaud (2021)	66	M	Yes	MGUS IgG Kappa	ChAdOx1nCOV-19 vaccine (AstraZeneca)	22.4	2.8	1.33	3.8	No	Intravenous fluids Recovery
Matheny (2021)	68	F	Yes	MGUS	Ad26.COV2-S vaccine (Janssen)	20.1	1.1	2.59	10.9	Yes	Rhabdomyolysis. CS with fasciotomy. IMV and CRRT. Death.
	46	F	Yes	IgG Kappa	mRNA-1273 vaccine (Moderna)	21.3	2.0	1.5	7.7	Yes	Vassopresors Recovery
	36	M	No	No	BNT162b2 vaccine (Pfizer)	20.1	2,3	2.4	10.9	No	Status epilepticus; CPA; DIC. IMV Recovery
Pineton de Chambrun (2020)	45	F	Yes	IgG Kappa	PCR SARS-CoV-2 +	19.9	ND	ND	ND	Monthly	CS IMV. Death
Lacout (2020)	38	M	Yes	IgG Kappa	PCR SARS-CoV-2 +	24.9	1.6	1.93	5.7	No	IMV Recovery
Case (2020)	63	M	No	ND	PCR SARS-CoV-2 +	21.6	< 1.5	2.35	4.9	No	Rhabdomyolysis. CS with fasciotomy. IMV and CRRT. Death.
Cheung (2021)	59	F	No	ND	PCR SARS-CoV-2 +	>25	2.4	ND	3.7	Yes	Rhabdomyolysis. IMV and CRRT. Death.
	36	M	Yes	IgG lambda	PCR SARS-CoV-2 +	17.1	ND	ND	9.2	No	CPA at admission Death
Beber (2021)	55	F	No	ND	PCR SARS-CoV-2 +	21	3.5	1.4	5.6	Yes	Rhabdomyolysis. IMV and CRRT. Death.

ND, not detailed; MGUS, monoclonal gammopathy of undetermined significance; M, male; F, female; IVIg, intravenous inmunoglobulin; IMV, invasive mechanical ventilation; NIMV, non invasive mechanical ventilation; CRRT, continuous renal replacement therapy; CPA, cardiopulmonary arrest; CS, compartment syndrome; DIC, disseminated intravascular coagulation.

Infections and antimicrobials

Abstract:0349

FLACCID QUADRIPARESIS CAUSED BY HERPES SIMPLEX VIRUS 6 ASSOCIATED WITH H1N1 INFLUENZA A INFECTION

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Introduction: HHV6 is known as the causative agent of focal encephalopathy in adults, as a trigger for myelopathies, a key factor in the pathogenesis of multiple sclerosis, and the cause of severe encephalitis in immunocompromised patients. As a result of the activation of latent HHV6 neuroinfection, severe forms of neurological deficits can remain.

Case: We report a 43-year-old male patient who was admitted to the intensive care unit due to the development of ARDS caused by the H1N1 Influenza A infection. Due to the rapid radiographic progression, the development of ARDS with the following development of respiratory failure, the patient was intubated and mechanical ventilation was initiated. As high fever and flaccid quadriparesis remained after stabilization of the respiratory status and weaning the patient off MV, the lumbal puncture was performed and DNA positive Human herpesvirus 6 was isolated from the meningitis-encephalitis panel. Treatment with ganciclovir resulted in a partial therapeutic response.

Conclusion: We have presented a case of flaccid quadriparesis caused by HHV6 in the presence of a documented H1N1 Influenza A infection. This virus has not previously been presented as a precursor to a HHV6 infection leading to severe neurological deficits in previously healthy individuals. Other risk factors have been predominantly shown as the cause of neuromuscular deficit in critically ill patients, but this case proves that less frequent factors such as an HHV6 infection cannot be ignored. Treatment with antiviral medication should be initiated immediately after the diagnosis of HHV6-induced encephalitis is established.

Keywords: flaccid quadriparesis, human herpes virus 6, Influenza A, ARDS

Sepsis - Other

Abstract:0352

DIFFERENT CAUSES FOR SHOCK IN THE SAME PATIENT DURING A SINGLE HOSPITALIZATION: A PECULIAR CASE REPORT

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Introduction: Shock is the clinical expression of circulatory failure that results in inadequate cellular oxygen utilization. Shock is a common condition in critical care, affecting about one third of patients.

Clinical findings of different types of shock can easily overlap each other due to which etiological diagnosis remains uncertain. Clinical examination and laboratory investigations often consume time, which is often not suitable for critically ill patients.

Case: A 50-year-old man presented to the emergency department complaining of shortness of breath and chest pain. The associated cough was productive of yellow sputum without hemoptysis. The patient denied a previous history of disease. Upon admission to intensive care, he presented with respiratory failure, requiring mechanical ventilation. Tissue hypoperfusion was quickly evidenced, and, with the help of Point-of-Care ultrasound (POCUS), it was classified as a distributive shock of pulmonary origin. Also, with the help of POCUS, pleural effusion was verified, which in thoracentesis turned out to be empyema, for which pleural drainage was installed and empirical antibiotic coverage was started.

An image compatible with thrombus in the left ventricle was verified, but with preserved cardiac output. On the second day of hospitalization, he presented worsening tissue perfusion parameters, with alterations in the ECG, in motility (localized due to cardiac POCUS) in addition to variation in Troponin I, he was classified as a myocardial infarction of probable embolic origin. Along with compromised cardiac output, it was classified as cardiogenic shock and full anticoagulation was started.

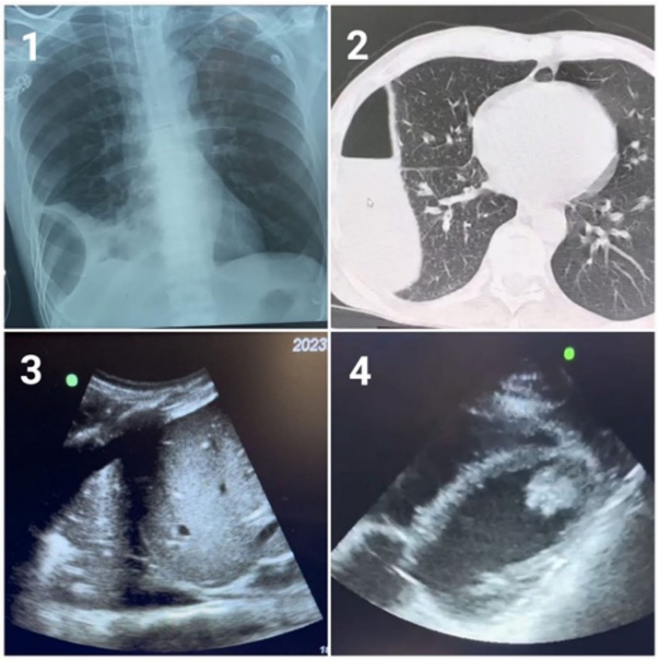
On the 5th day after symptom recovery, the patient was extubated and the pleural drain was removed, but replaced due to the progressive increase in subcutaneous emphysema. With the repositioning of the tube, he developed muscle laceration and hemothorax. There was a drop in Hb to 4.5g/dL and evidence of tissue hypoperfusion, being classified as hypovolemic shock.

He required mechanical ventilation, hemodynamic support, packed red blood cell transfusions and volume resuscitation. On the 8th day, the patient again presented a sudden drop in hemoglobin in addition to melena, requiring vasopressor drugs to improve tissue perfusion and more transfusions. On the 11th day, he was again extubated after hemodynamic compensation.

Conclusions: Delivering early diagnosis of shock in resource-limited settings is challenging, especially with limited availability of point-of-care laboratory diagnostic facilities. There is a growing urgency to provide point-of-care diagnosis and organized treatment, especially for time-sensitive critical conditions like shock.

Keywords: Shock, Point-of-care-ultrasound, Empyema, Intraventricular thrombus

Images on admission to ICU. Pleural cavity and heart



Figuri 1. 1. Chest X-Ray 2. Noncontrast tomography of the lung. 3. Pulmonary POCUS showing complex effusion 4. Cardiac POCUS with image of left ventricular thrombus

Neuro-intensive care

Abstract:0359

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES) IN PREGNANCY: TWO CASES

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Introduction: Posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological entity with acute neurological symptoms including headache, seizures, visual disturbances, and other focal neurological deficits. Classically, two main theories for the pathogenesis of PRES have been proposed. The first is severe hypertension that leads to disruption of the brain autoregulation system, consequently resulting in endothelial edema or injury. The second is endothelial dysfunction caused by circulating endogenous or exogenous toxins. PRES is often associated with hypertensive encephalopathy, preeclampsia, eclampsia, renal failure, immunosuppressive therapy or chemotherapy. Patients with severe manifestation of PRES may get admitted to the intensive care unit (ICU). We describe two cases of PRES syndrome during pregnancy.

Case(s): 1) A 24-year-old woman at 28 weeks of gestation presented with seizures. Her blood pressure was 170/100 mmHg. There was no history of hypertension or other diseases. She was admitted to the ICU after delivery of the fetus via cesarean section. Her symptoms improved with aggressive blood pressure control. T2 weighted brain magnetic resonance imaging (MRI) revealed hyperintensity in the cortical and subcortical bilateral occipital-frontoparietal areas.

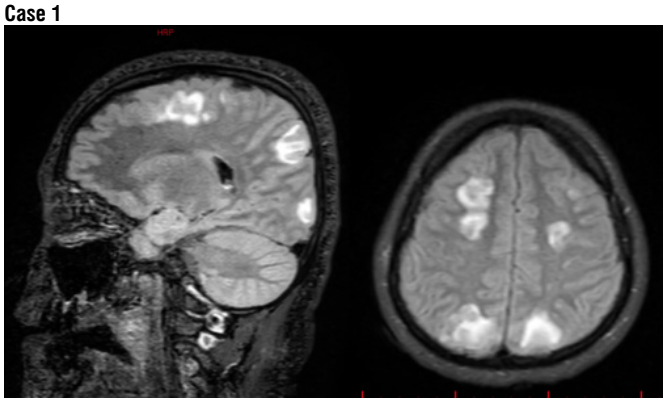
2) A 20-year-old woman at 31 weeks of gestation was admitted to the emergency department due to spontaneous labour. Her blood pressure was 160/80 mmHg. She had epileptic seizures and syncope while being taken to the delivery room. She was admitted to the ICU after delivery of the fetus via cesarean section. Blood pressure was controlled with nicardipine infusion. MRI revealed bilateral frontal-parietal-occipital cortical and subcortical hyperintensities consistent with PRES.

Both patients were weaned from mechanical ventilation on days 2 and 3, respectively and then discharged from the ICU.

Conclusion: Both of these cases highlight the importance of early recognition and treatment of PRES in pregnancy. Prompt delivery of the fetus may be necessary in severe cases. Aggressive blood pressure control is also essential in the management of PRES in pregnancy. It is important to note that PRES can also occur in the postpartum period, and clinicians should be aware of this possibility in patients presenting with neurological symptoms in the weeks following delivery.

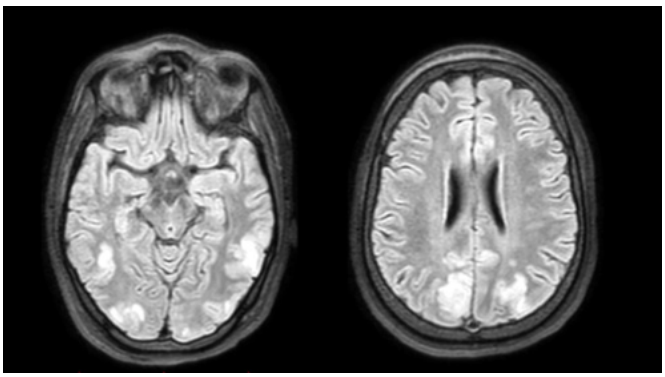
Keywords: ICU, pregnancy, PRES syndrome

Case 1



T2 MRI

Case 2



T2 MRI

Infections and antimicrobials

Abstract:0363

TIGECYCLINE INDUCED HYPERBILIRUBINEMIA AND COAGULATION ABNORMALITIES- A CASE REPORT

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Introduction: Tigecycline, a glycylcycline antibiotic, is approved by the Food and Drug Administration for the treatment of complicated skin and skin structure infections, community-acquired bacterial pneumonia, and complicated intraabdominal infections (1). In this case report we aim to draw attention to tigecycline-induced rare side effects: hyperbilirubinemia, thrombocytopenia and hypofibrinogemia.

Case: An 89-year-old woman who had undergone surgery for a right femoral head fracture 1 month ago was brought to the emergency department with complaints of progressive decrease in oral intake and deterioration in general condition that started after the operation. She had history of hypertension, atrial fibrillation, heart failure, and deep vein trombosis (4 months ago). The patient was admitted to the internal medicine palliative clinic.

Since the patient had new onset fever, teicoplanin 400 mg and meropenem 400 mg were administered after loading dose with the suspicion of osteomyelitis. In the follow-up, Candida albicans growth was observed in the blood culture. The patient was transferred from the palliative clinic to our medical ICU with an initial diagnosis of septic shock. Anidulafungin 100 mg was added to antibiotic regimen after loading dose.

Teicoplanin was discontinued on the 14th day because there was no gram-positive growth in the cultures of the patient whose general condition improved. Meropenem was continued as monotherapy. Acinetobacter baumannii growth was observed in the wound culture taken from the right femoral head fracture operation suture line. Tigecycline was administered intravenously at 100 mg for the first dose and was given at 50 mg maintenance dose every 12 hours.

On the 4th day of tigecycline use, hypofibrinogenemia, elevated D-dimer levels and INR and on the 6th day increase in bilirubin, ALT, AST, ALP, GGT were observed (Table). The coagulation panel returned to normal range after cryoprecipitate treatment. Abdominal ultrasonography and MRCP revealed no pathology in the extrahepatic bile ducts. The increase in transaminase and bilirubin of the patient was evaluated as a side effect of tigecycline. Tigecycline was

discontinued on day 14. Fibrinogen, bilirubin and transaminases improved, and baseline levels were restored (normalized within 7 days). The patient was transferred to the palliative clinic for further treatment.

Conclusion: Tigecycline has been reported to cause hematologic abnormalities. Although the hepatotoxic effect of tigecycline is known, the cases reporting increased bilirubin levels are few in number. Clinicians should be alert to the development of hepatic and coagulation dysfunction.

Reference

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Keywords: Tigecycline, side effects, hyperbilirubinemia, coagulopathy, intensive care

Table 1. Daily Laboratory Parameters

ICU day	Day 3	Day 4 (initiation of tigecycline)	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10	Day 11	Day 12	Day 13	Day 14 (tigecycline stopped.	Day 15	Day 16	Day 17	Day 18
ALT (IU/L)	5	18	26	134	115	68	73	73	65	60	55	63	48	45	39	34
AST (IU/L)	18	32	73	362	186	84	113	104	88	84	70	99	68	55	44	55
GGT (U/L)	23	39	51	137	117	76	76	74	55	50	47	50	37	37	32	31
ALP (IU/L)	183	210	310	901	914	711	790	894	694	730	738	780	626	591	558	527
Direct bilirubin (mg/dL)	0.4	0.3	0.5	1.4	1.6	2.6	2.9	4.8	4.8	5.8	6.6	7.7	6.2	5.7	4.6	4.3
Total bilirubin (mg/dL)	0.8	0.7	0.8	2.4	3.0	4.3	5.4	5.9	5.8	7.0	9.1	9.6	10.1	9.2	7.1	5.5
INR	1.3	1.8	1.4	1.3	1.4	1.3	1.4	1.4	1.3	1.4	2.4	1.6	1.4	1.4	1.5	1.3
Fibrinogen (mg/dL)	144	65	106	168	155	207	195	210	211	174	51	220	213	186	195	167
D-dimer (ng/mL)	1.4	3.2	3.1	5.4	5.5	3.2	2.3	1.9	4.8	1.6	2.5	2.2	1.9	2.5	1.8	2.1

Metabolism - Endocrinology

Abstract:0369

A RARE CAUSE OF METABOLIC ACIDOSIS: NON-DIABETIC KETOACIDOSIS

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Introduction: Ketoacidosis is a common cause of raised anion gap metabolic acidosis. The most common cause is diabetic ketoacidosis, though starvation ketoacidosis and alcoholic ketoacidosis are not uncommon. Non-diabetic ketoacidosis states are frequently overlooked due to relative unawareness among the clinicians leading to misdiagnosis (1). Hereby, we aimed to present a non-diabetic ketoacidosis case followed in our medical ICU.

Case: A 47-year-old male patient came to the emergency room complaining of abdominal pain, nausea, vomiting for one day. He had a past medical history of rickets and gastroesophageal reflux. He takes omeprazole occasionally but no other medications. Detailed anamnesis revealed that the patient had suffered from nausea and abdominal pain for 10 years. Therefore, the patient underwent gastroscopy one day before emergency room admission. The patient had tachypnea (30/min) and tachycardia (132/min) and he did not need oxygen therapy. Dehydration signs were observed on physical examination and capillary refill time was 4.5 seconds. Glasgow Coma Scale score was 15. Laboratory examination revealed raised anion gap metabolic acidosis (ph:6.98, pCO2: 27.2, pO2: 27.8, lactate:2.13, anion gap:23.2, HCO3:6.4), hyperglycemia (275 mg/dL), leukocytosis (27760/mcL), ketonuria (3+) and glucosuria (3+). Serum electrolytes levels, liver and kidney function tests were normal. He was admitted to the medical intensive care unit with a diagnosis of ketoacidosis due to possible new onset diabetes. Intravenous hydration, insulin therapy and sodium bicarbonate were administered. There were not infection signs on chest X-ray, urine culture and blood culture. During follow-up laboratory examination improved. To reveal new onset diabetes detailed laboratory analysis was performed. Hemoglobin A1c and c-peptite levels were normal (5.8% and 1.77 ng/mL, respectively) and diabetic autoantibodies were negative. Patient was diagnosed with non-diabetic ketoacidosis. When the patient was asked for starvation and alcohol history to enlighten etiology of non-diabetic ketoacidosis, he described a 60 hours lasting starvation for gastroscopy. The patient was discharged on the 5th day of follow-up.

Conclusion: Diabetic ketoacidosis is a common cause of metabolic acidosis. However, ketoacidosis is not always accompanied with high blood glucose and diabetes. Starvation and alcoholism may be the cause of non-diabetic ketoacidosis. Clinicians should keep in mind non-diabetic ketoacidosis in patients with metabolic acidosis and non-diabetics.

Reference

1. Bashir B et al. Non-diabetic ketoacidosis: a case series and literature review. Postgrad Med J.2021

Keywords: metabolic acidosis, non-diabetic ketoacidosis, intensive care, critical care, starvation

Respiratory - Mechanical ventilation

Abstract:0371

DIFFERENTIAL LUNG VENTILATION FOR REFRACTORY HYPOXEMIA MANAGEMENT UNDER VIDEO-ASSISTED THORACOSCOPIC SURGERY VATS

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Introduction: In thoracic and pulmonary surgery, one lung ventilation in the lateral decubitus position produces changes in the ventilation/perfusion ratio in the upper (non-dependent) as well as at the lower (dependent) lung (1). A physiological compensatory protective reflex like hypoxic pulmonary vasoconstriction (HPV) is activated, nevertheless even using recruitment maneuvers hypoxemia may still occur (2).

Case: A 47-year-old, 77 kg male patient was scheduled for an apical left lower lobe lung tumor resection under video-assisted thoracoscopy surgery (VATS) with normal preoperatory tests.

Monitoring included ECG, invasive arterial blood pressure (IABP), pulse oximetry (SpO2), capnography (ETCO2), bispectral analysis (BIS), regional cerebral oxygen saturation (rSO2) (INVOSTM 5100C; Covidien/Medtronic Inc., USA) and diuresis. Antibiotic prophylaxis and pre-emptive analgesia were administered.

Anesthesia was based on propofol TCI, rocuronium and remifentanil with a BIS target lower than 45. A peripheral vein with a #18 Fr and an ultrasound-guided central venous catheter with left internal jugular approach were placed as venous access. The intubation-positioning of a Robertshaw 37 Fr right double lumen tube was achieved with a video laryngoscope C-MACTM and flexible video endoscope (Karl Storz, Tutlingen, Germany). Clamping tests were also applied before and after changing patient position. The controlled ventilation parameters were adjusted by capnography: TV: 500 ml, RF: 12, I:E: 1:2, PEEP: 8 cmH2O, FiO2: 0.7.

After 45 minutes, during the tumor detachment (8x4x3cm) hypoxemia and hypercarbia developed. Verifying the position and patency of the tracheal tube and increasing FiO2 to 1.0, recruitment maneuvers were initiated. At minute 90 with hypoxemia (SpO2 <83%, PaO2 58 mmHg) and hypercapnia, differential ventilation was started. The parameters were TV: 85 ml, PEEP: 5 cmH2O, RF: 10, FiO2:0.7 (non-dependent lung) and TV: 450 ml, I:E: 1:2, PEEP: 8 cmH2O, RF: 12 /minute and FiO2:1.0 (dependent lung), Peak airway pressures were 12 and 25 cmH2O, respectively. Subsequently, oximetry improved to 98-100%.

The patient, transferred to the ICU after 290 minutes, hemodynamically stable, with adequate arterial blood gases, 700 ml blood loss and 280 ml diuresis underwent an uncomplicated postoperative period, being discharged five days after surgery.

Conclusion: Differential lung ventilation is a good option to improve oxygenation and gas exchange in one lung anesthesia for VATS, with minor influence at the surgical field and hemodynamics.

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Keywords: Ventilation, hypoxemia, VATS.

Cardiovascular - Other

Abstract:0372

SIMULTANEOUS SURGERY FOR ADVANCED RENAL CELL CARCINOMA AND CONCOMITANT DOUBLE VALVE AND CORONARY ARTERY DISEASE: A RARE CASE REPORT

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Introduction: Cardiovascular and neoplastic diseases are the main causes of death in Europe. Renal cell carcinoma (RCC) is a kidney cancer that is formed in the proximal convoluted tubule. It represents around 3% of all cancers, and are responsible for 80% to 90% of all primary renal neoplasms. The number of patients who have both cardiac disease and cancer has been rising as the proportion of elderly in the general population increases. Surgical procedures for cardiac disease and renal tumors can be performed in stages or simultaneously (at the same time).

Case: A 74-year-old patient with severe aortic valve stenosis, severe mitral valve regurgitation and coronary artery disease was diagnosed with malignant renal cell carcinoma in advanced stage with intravascular extension as tumor thrombus into the inferior vena cava and right atrium. A multidisciplinary team approach consisting of cardiac surgeons, urologists and anesthesiologists has successfully performed a simultaneous curative surgery consisting of aortic valve replacement, mitral valve repair and double coronary artery bypass, and open radical nephrectomy, lateral cavotomy and thrombectomy under cardiopulmonary bypass. The procedure was rendered as very high-risk procedure. In the postoperative period, due to oligo-anuria the patient was placed on continuous veno-venous hemodiafiltration, with subsequent resolution of left kidney function and satisfactory diuresis. Antibiotic therapy was administered including piperacillin/tazobactam, linezolide and antifungal medication fluconazole, adjusted according to eGFR. Catecholamine and vasopressor support were gradually reduced in the postoperative period and discontinued. The patient also suffered from severe form of chronic respiratory failure, after extubation he was placed on non-invasive mechanical ventilation and intensive physical and respiratory therapy was performed. The pathohistological finding revealed Clear renal cell carcinoma in the IV stage, pTNM= pT4 G3 (WHO/ISUP) pNx pM1 L1 V1 R1. Postoperative echocardiography showed good function of the implanted aortic valve and annuloplasty of the mitral valve without residual regurgitation of paravalvular leak. The patient was discharged on the 15th postoperative day in good stable condition.

Conclusion: Patients with renal tumors in advanced stage and cardiac disease can be treated successfully with surgery and benefit from early intervention by a simultaneous approach. This is a rare case report in which extensive cardiac surgery procedure and urologic procedure were performed. The multidisciplinary approach is the key factor for successful outcomes.

Keywords: cardiac disease, cardiac surgery, renal cell carcinoma, simultaneous surgery

Cardiovascular - Cardiovascular dynamics

Abstract:0374

A RARE CASE OF SARS-COV-2 RELATED AMAN THAT IS COMPLICATED WITH SEVERE MYOCARDITIS

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Introduction: Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) causes a coronavirus disease 2019 (COVID-19), responsible for the current pandemic that has caused over 6,8 million deaths globally. Guillain-Barré syndrome (GBS) is an autoimmune inflammatory peripheral neuron disease. Acute Motor Axonal Neuropathy (AMAN) is characterized by a rapidly progressive symmetrical paralysis without sensory loss. This disorder is found to a greater degree, making up nearly half of all GBS cases. In contrast to classic GBS, this variant manifests with more severe muscle weakness

and a higher incidence of respiratory failure. We report a rare case of fulminant SARS-CoV-2 related AMAN that is complicated with severe myocarditis treated successfully with extracorporeal membrane oxygenation (ECMO) and plasmapheresis.

Case: A 36-year-old woman presented to our clinic with loss of consciousness caused by cardiogenic shock due to severe myocarditis. Her blood pressure (BP) was 90/50 mmHg and she was 130/min tachycardic at the admission. Other vitals were within normal limits with 97% oxygen saturation at the room temperature. She complained about difficulty walking, muscle weakness and high fever for two days priorly otherwise her medical records were unremarkable. Chest computed tomography (CT) showed COVID-19 pneumonia and pericardial effusion. She underwent echocardiography and ejection fraction (EF) of %20-25 and 17 mm pericardial effusion with no tamponade sign were detected. Cardiac markers were high with troponin l- 2345 ng/dL, creatinine kinase-440 IU/L and B-Natriuretic peptide-6838 pg/mL. Vasopressor treatments were started to maintain the optimal BP. During the follow up she complained of severe abdominal pain and ST elevation was detected in an electrocardiogram. With a preliminary diagnosis of pulmonary and mesenteric embolus she underwent CT. No embolus was detected. Subsequently, her BP status deteriorated and a decision was made to commence veno-arterial (VA)-ECMO for cardiac failure. On the 8 th day of VA-ECMO support her BP was normal without vasopressor support and ECMO was discontinued. EF was detected as normal on echocardiography. After nine days of mechanical ventilator support she was extubated. Eventually, she complained about difficulty moving all four extremities. Electromyography (EMG) revealed axonal neuropathy compatible with AMAN. Her muscle weakness fully recovered by ten days of plasmapheresis and continuous physical therapy.

Conclusion: This is the first case of COVID-19-induced AMAN that caused myocarditis, supported by ECMO and plasmapheresis and fully recovered with normal cardiac and neurological functions. We reported this case to be aware of this rare life threatening condition.

Keywords: Covid-19, ECMO, GBS, Plasmapheresis, Myocarditis

Gastrointestinal system and Liver

Abstract:0375

EFFECTS OF N-ACETYL CYSTEINE AGAINST DICLOFENAC SODIUM INDUCED LIVER TOXICITY

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Introduction: Diclofenac sodium, one of the non-steroidal anti-inflammatory drugs (NSAIDs), may cause gastrointestinal, renal, and hepatic damage. The frequency of liver damage is not known exactly, it is known to cause idiosyncratic hepatotoxicity with significant morbidity and mortality, especially when used long-term and at high doses. Oxidative stress and mitochondrial damage are associated with diclofenac toxicity, which may be alone or in combination (1,2). N-acetylcysteine is used for the treatment of acetaminophen toxicity, but it may be beneficial in other forms of drug-induced acute liver injury and failure. We report a case of diclofenac-induced idiosyncratic hepatitis, which recovered significantly based on clinical and laboratory, in which N-acetylcysteine (NAC) was used in supportive treatment.

Case: A previously healthy 24-year-old male patient was admitted to the intensive care unit with abdominal pain and nausea for 2 days. The patient who did not have any habits was using NSAIDs irregularly due to migraine pain and had a history of using a total of 20 diclofenac sodium tablets (Dolorex® 50 mg tablet) in the last week, a maximum of 6 tablets in one day. The skin and sclera showed visible jaundice. Laboratory tests revealed AST:3457U/L, ALT:4570 U/L, total bilirubin 6 mg/dL, direct bilirubin 4.4 mg/dL, INR:1.6. Abdominal ultrasonography was normal except for grade 1 steatosis in the liver. After differential diagnosis, diclofenac sodium-associated idiosyncratic hepatitis was considered. NAC (at the dose of paracetamol intoxication treatment) infusion was administered and the patient was transferred to the internal medicine service after the liver function tests regressed, after 10 days of intensive care follow-up.

Conclusions: NAC, which is used as an antidote for paracetamol intoxication, has anti-inflammatory, antioxidant, inotropic, and vasodilation effects and improves microvascular circulation in vital organs in addition to repleting glutathione reserves. It has been shown that NAC has a protective effect on diclofenac-induced hepatotoxicity in rats (2). However, the use of NAC in cases of diclofenac-related hepatotoxicity in humans in the literature is not available to our knowledge. By presenting this case, we wanted to remind clinicians that NAC can be an inexpensive, easily accessible supportive treatment for diclofenac-induced hepatotoxicity.

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Keywords: Diclofenac sodium, idiosyncratic hepatotoxicity, N-acetylcysteine

Critical care outcomes and scoring systems

Abstract:0378

THE MARFAN SYNDROME DILEMMA: A RARE PRESENTATION OF ACUTE CORONARY SYNDROME IN A YOUNG MALE WITH COVID-19 DISEASE

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Introduction: Marfan syndrome is a rare genetic connective tissue disorder caused by a mutation in the Fibrillin-1 (FBN1) gene[1]. Some complications in the heart include aneurysmatic dissection of the aorta and acute coronary syndrome (ACS). COVID-19 is a novel disease due to the severe acute respiratory syndrome coronavirus (SARS-CoV-2) which predominantly affects the respiratory system with multi-organ complications. ACS has been reported in patients with this novel virus[2]. The dilemma in this patient diagnosed with Marfan syndrome was treating the ACS, as a complication of COVID-19. We report a rare case of ACS in a young adult, newly diagnosed with Marfan syndrome and COVID-19.

Case: A 19-year-old male presented at our COVID-19 Treatment Centre in Lagos, Nigeria, with a 5-day history of generalized body weakness, lethargy and a 2-day history of loss of taste and smell, shortness of breath and retrosternal chest pain which radiated to the left arm. He is a known social smoker with no previous history of hospitalization. He had myopia and a body mass index of 23 kg/m2. General examination revealed flaring of the alae nasi, a long face with a high-arched palate, long thin fingers and minimal pectus cavinatum. Respiratory rate was 30/min, with decreased air entry on the left chest on auscultation, a pulse rate of 90/min, Blood Pressure of 120/70 mmHg and heart sounds were normal. Pulse oximetry was 92% on room air. Blood was taken for full blood count and D-Dimer test while a nasal and throat swab was taken for the Polymerase Chain Reaction (PCR) test but a COVID-19 Rapid (Antigen) Test was

positive. An initial electrocardiogram showed sinus tachycardia with elevated ST segment wave.

The patient was placed on 8 L/min of oxygen via a non-rebreathing face mask. Sub-lingual nitroglycerin and aspirin 325mg were administered with other adjunct COVID-19 medications. The D-Dimer was 420ng/mL, PCR positive and the Chest X-Ray showed a ground-glass appearance on the left lung lower zone. Clopidogrel was added to his medication. He became COVID-19 negative and was discharged on the 15th day of admission for further management by the cardiologists.

Conclusions: The mechanism leading to ACS in COVID-19 infection is not fully understood but there is a strong correlation on the possibility of COVID-19-induced myocardial injury. Our case highlights the successful treatment with anti-coagulants and fibrinolytic agents of a Marfan syndrome patient with COVID-19 infection complicated by ACS in a resource-limited clime.

Keywords: Marfan syndrome, COVID-19, acute coronary syndrome

Infections and antimicrobials

Abstract:0385

AN UNUSUAL SUSPECT: CANDIDA DUBLINIENSIS CAUSED PERICARDITIS

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Introduction: Candida pericarditis is a very rare invasive fungal infection and causes fungemia. Addition to clinical suspicion deterioration of clinic status despite long-term broad-spectrum antibiotic therapy, pericardial fluid seen in echocardiography, positivity of candida species in cultures are helpful to diagnose. C.dubliniensis is found in human oral flora and is responsible for oral candidiasis in HIV (+) patients. In literature searching 'candida', 'pericarditis', 'dubliniensis' keywords no case of pericarditis caused by C. dubliniensis was found, and systemic infections caused by C.dubliniensis were seen as 'pneumonia' and 'endocarditis'.

Case: The 61-year-old male patient was referred with septic shock to ICU of our hospital from where aortic replacement and CABG operation were performed. Acute liver and kidney failure occurred with severe bilirubin level elevations. He had been treated with broad-spectrum antibiotics (meropemem and vancomycin), norepinephrine, and there was a yeast signal in the blood culture. Echocardiography reported EF:50, no valve insufficiency, 25 mm fibrinous adjacent to LV wall, 17 mm adjacent to RV and 11 mm adjacent to posterior wall. Pericardial fluid was observed which didn't cause compression and tamponade in the widest part, fluconazole was added to prescription. Despite his broad-spectrum antimicrobials, patient's clinic was deteriorated, so caspofungin and tigecycline antibiotics were added. Echocardiography was performed again and it was observed that pericardial fluid increased and compressed, and pericardiocentesis was performed. After draining 800cc of fluid. C.dubliniensis was grown in the culture of pericardial fluid. The patient was diagnosed with candida pericarditis based on culture result. Despite high-dose norepinephrine and adrenaline infusion, the patient's deep hypotension due to septic shock persisted, cardiac arrest occurred on 5th day of ICU admission and was declared as exitus.

Conclusions: 'Siller et al., reviewed at 2022 39 candida pericarditis case, 24 of them were C.albicans (66.7%), 4 of them were C.tropicalis (10.3%), 4 of them were C.glabrata (10.3%), 2 of them were c.parapsilosis (5%, 1), 1 was C.kruzei (2.6%), 1 was C.guillermondili (2.6%), and C.dubliniensis was not found. Due to its high mortality, early recognition of this clinical condition is important for the surveillance of the patient. We shared the information that C.dubliniensis, an uncommon species, can cause candida pericarditis, in order to remind clinicians and to initiate the correct empirical treatment early.

Reference

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Keywords: Candida, Dubliniensis, Pericariditis, Septic Shock

Cardiovascular - Other

Abstract:0388

RECURRENT MYOPERICARDITIS ASSOCIATED WITH ANABOLIC ANDROGENIC STEROIDS IN A YOUNG MALE

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Introduction: Anabolic-androgenic steroids (AASs) are derived from testosterone and its related precursors. AASs are widely used by adolescents, athletes and especially by bodybuilders, both for aesthetic uses and as performance enhancers. Their long-term use can lead to serious health problems, especially the occurrence of cardiovascular risk. Toxicity effects of AASs on the cardiovascular system range from left ventricular hypertrophy to sudden cardiac death. In this case report, we present a patient with recurrent myopericarditis associated with anabolic-androgenic steroids.

Case: A 22-year-old male patient was admitted to the emergency department with stabbing chest pain. In the history of the patient who had been on AASs, it was learned that coronary angiography was performed one year ago due to chest pain, angiography was found to be normal, it was evaluated as myopericarditis and non steroid anti inflammatory drugs were used. Physical examination was normal and there was no evidence of heart failure. The electrocardiography revealed sinus tachycardia. There was no ST-T change. In lab values Troponin-T: 261 ng/ml, white blood cell:15870 /uL, C-reactive protein: 39 mg/L, N-terminal pro-brain natriuretic peptide:804 ng/L. Other blood workup was normal. Transthoracic echocardiography showed a mild global left ventricle hypokinesia, ejection fraction of 55%, increased pericardial brightness. Cardiac MRI could not be performed due to technical problems. He was hospitalized in intensive care unit with clinically suspected myopericarditis. Lipid and thyroid hormone profile, autoimmune parameters, viral respiratory panel were checked and found negative in the patient without cardiovascular risk factors. He had no sign of infectious or systemic disease. It was evaluated as myopericarditis associated with AASs. He was treated with aspirin, colchicine, intravenous immune globulin and methylprednisolone due to recurrent history. During his hospitalization, there was no anginal complaint and the troponin decreased. He was discharged with the recommendation of exercise restriction for at least 3 months and cessation of AASs use. It was learned that the patient who continued to use AASs had myopericarditis again.

Conclusions: AASs abuse leads to adverse effects in all body tissues and organs. The cardiovascular adverse effects include myocardial hypertrophy, changes in heart relaxation and contractile function, cardiomyopathy and myocarditis. A temporary rise in blood pressure,

prothrombotic effects, and impaired lipid metabolism increase the risk of coronary artery disease. AAS abusers are at increased risk of arrhythmias, leading to sudden cardiac death. Abuse of AASs has increased in recent years. It would appear that anabolic steroid abuse should be considered in any young athletic individual presenting with an acute vascular event.

Keywords: myocarditis, anabolic androgenic steroids, drug toxicity

Kidney - Renal replacement therapy

Abstract:0389

DISSEMINATED INTRAVASCULAR COAGULATION INDUCED BY UNFRACTIONATED HEPARIN DURING DIALYSIS IN AN ANEPHRIC PATIENT: A CASE REPORT

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Introduction: We presented an anephric patient with disseminated intravascular coagulation related dialysis with unfractionated heparin.

Case: Right laparoscopic nephroureterectomy, cystoprostatectomy, left ureterectomy and bilateral iliac lymph node dissection operation was performed on a patient aged 74, who had hypertension, diabetes, chronic renal failure, had not received dialysis before, and had left nephrectomy operation for polycystic kidneys 10 years ago. He was extubated at the postoperative 14th hour. The patient, who was on dialysis without heparin postoperative 20th hour due to hyperkalemia, was followed up with a Glasgow coma scale of 15, conscious, cooperative, oriented, and hemodynamically stable. No problems were encountered during the first dialysis without heparinization. On the post-operative 2nd day, the patient was hemodynamically stable for 4 hours on dialysis with heparin, which was performed due to hyperkalemia and anephric symptoms. It was observed that there was a progressive and rapid increase in alanine transaminase, aspartate transaminase, and creatinin values. Nausea, vomiting and hypotension developed in the 16th hour after the end of the second dialysis in the patient who came out with 350 ml of hemorrhagic fluid from the abdominal drains. 750 mg tranexamic acid, 3 U erythrocyte suspension, 3 U FFP, 1 U platelet suspension, 2 ampoules of calcium gluconate were administered to the patient who needed high-dose dopamine, noradrenaline, and ephedrine. The patient was found to have platelet 155,000, leukocytes 16,300, a PTT: 28.9, PT: 15.7, INR: 1.1. D- dimer values >5500. After the ACT value of the patient was determined as 216 sec, 20000 U of protamine sulfate was administered. The patient was intubated and taken to emergency operation due to the increase in the amount of blood coming from the drains and the development of respiratory failure and clouding of consciousness in the patient, free fluid in the abdomen in the abdominal ultrasonographic evaluation, and a 7x9 cm hematoma in the right kidney lodge. In the patient who could not detect a clear active bleeding focus, the hematoma in the abdomen and kidney site was cleaned and the operation was terminated. The patient with severe increase in ALT, AST, creatinine and phosphorus levels, followed by liver failure and ARDS, died on the 5th day of hospitalization due to cardiac arrest.

Conclusion: Treatment of enhanced-fibrinolytic-type DIC with unfractionated heparin alone may increase bleeding. Monitoring ACT can be done, hemodialysis without anticoagulation may be a therapeutic option in such patients at least postoperative period.

Keywords: dialysis, unfractionated heparin, disseminated intravascular coagulation

Fluid and electrolyte disorders

Abstract:0391

SALINE INTOXICATION - A DANGEROUS CONDITION IN INTENSIVE CARE UNIT POST HYDATID CYST IRRIGATION

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Introduction: Cystic echinococcosis (CE), caused by *Echinococcus granulosus* is a very common disease in Turkey. CE is treated with anthelmintics and surgery or percutaneous drainage. The cyst contents are usually irrigated with %20 hypertonic saline (HS) solutions. Cyst hydatid disease with HS irrigation can cause a rare but fatal complication such as hypernatremia. It requires careful monitoring and treatment.

Case: A 24-year-old female patient was processed under general anaesthesia in interventional radiology for percutaneous drainage for pelvic cyst hydatid disease. After propofol induction, the patient was intubated by giving fentanyl and rocuronium after providing sufficient muscle relaxation. During the procedure, the inside of the cysts was irrigated with 30% HS solution. No hemodynamic disorder was detected during the procedure and the patient was extubated in the operating room and transferred to the recovery unit without any problems. Sodium value was 190 mmol/l (reference 135-145 mmol/) in the blood tests taken due to the regression of the patient's consciousness in the recovery unit in the 1st postoperative hour. The patient was taken to PACU for the treatment of acute hypernatremia that developed after 2 hours of the intervention. The patient whose Glasgow Coma Scale 3 was intubated. Acute intracranial pathologies were excluded with cranial CT and MR images. Dialysis was not recommended for the patient whose arterial blood gases had pH:7,005, BE: -21 mmol/l, HCO3: 8 mmol/l, hypotonic intravenous fluid (5% dextrose) and irrigation through the nasogastric tube were started. The patient, whose Na level was decreased by 36 mmol/l (190-156 mmol/l) in 24 hours, regained consciousness 24 hours later, although the sedative drugs were discontinued. She was transferred from the intensive care unit to ward on the postoperative 4th day without any neurological damage.

Conclusion: Acute severe hypernatremia is potentially lethal. Extreme caution should be exercised in the use of hypertonic saline. In order to detect hypernatremia intraoperatively, blood tests should be taken at regular intervals and treatment should be started without delay. Hypernatremia should be kept in mind in patients who cannot recover after a procedure in which HS is used and whose conscious state gradually worsens.

Keywords: Severe hypernatremia, cystic echinococcosis, hypertonic saline

Infections and antimicrobials

Abstract:0404

SEVERE INTRAVASCULAR CATHETER RELATED INFECTIVE ENDOCARDITIS INFECTION THAT IS INOCULATED WHILE SWIMMING IN THE LAKE

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Introduction: Infective endocarditis (IE) is a life-threatening inflammation of the endocardium and heart valves. Staphylococcus (S) hominis is a gram-positive, coagulase-negative bacteria that occurs as a normal commensal organism on the skin and may rarely cause IE.

Case: Here we present a 44-year-old stage 4 non-small cell lung cancer patient who was currently on immunotherapy. He was treated for methicillin-sensitive staphylococcus catheter infection 4 months priorly with broad spectrum antibiotics (BSA) with removal of the infected central catheter. In current admission, he complained about oliguria and high fever. He became hypotensive and tachypneic during the first few hours of admission and he was supported with non-invasive mechanical ventilator and vasopressor agents. Blood results showed leukocytosis and bicytopenia. Creatinine was detected high with 7.89 mg/dL which was tested normal a week priorly. Acute phase reactants were detected as high. He started BSA and underwent hemodialysis for several days. The blood culture test extracted from the central catheter, on admission day showed S.hominis infection. Catheter was removed and BSA was continued. However, he continued to have a high fever. With a preliminary diagnosis of IE, an echocardiography was ordered. It was compatible with IE with 13x9mm vegetation on the tricuspid valve. He was not considered for surgical treatment because of his high level of vasopressor and inotropes. Several more sets of blood cultures were taken for other possible pathogens. Two sets of peripheral blood cultures were positive for S.aureus that is sensitive to methicillin on antimicrobial susceptibility tests. He had recurrent bouts of S.hominis, S.aureus, S.epididymis and S.haemolyticus bacteremia. In the second week of IE treatment valvular vegetation became 20x30mm. With four weeks more BSA treatment his heart vegetation disappeared fully without any sequela. He was discharged from hospital with full recovery on the 7th week of admission.

Conclusion: This is a very severe IE case successfully treated with optimal antibiotherapy and supportive treatments. Tricuspid valve IE is less common than left-sided IE, encompassing only 5–10% of cases of IE. In this patient, bacteria was probably inoculated from chemotherapy requiring long term central venous catheter. The patient stated that he was careful about the hygiene of his catheter, since he had a catheter infection before. However, he informed me that he had carelessly entered the lake once. We wanted to share this unique case to be aware of the condition.

Keywords: Infective endocarditis, Staphylococcus, Gram-positive coagulase-negative bacteria, Immunosuppression,

Cardiovascular - Other

Abstract:0406

A RARE CASE OF CARDIAC HYDATID CYST

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Introduction: Echinococcosis is a zoonotic parasite that often infects humans. The liver is the most frequently affected organ. The lung is the second common location of hydatid cyst. Cardiac hydatid cyst is a rare presentation of echinococcosis with approximate incidence of 0.03-1.1%. Hydatid cyst infection is often asymptomatic for years due to slow growth. Here we present a successfully treated left ventricular hydatid cyst case.

Case: A 28-years-old woman presented with atypical chest pain that she had been suffering for two years. She never smoked and her medical records were unremarkable. Cardiac examination and electrocardiogram were normal. Transthoracic echocardiography revealed a well-defined myocardial cystic mass with normal ejection fraction. Magnetic resonance test showed 3x3 cm cystic mass on the left apical border of myocardium. With a preliminary diagnosis of hydatid cyst, a serologic test was ordered. However, it was negative for echinococcal infection. Hepatic and pulmonary examinations were normal with no sign of hydatid cyst. Subsequently, she was operated on and the cyst was carefully drained and removed. Histological results were compatible with hydatid cyst including a nucleated germinal membrane, an acellular cuticle membrane and an acellular proteinaceous fluid.

Conclusion: Cardiac hydatid cyst could be life-threatening when it is ruptured. The incidence of complicated hydatid cyst is reported to be as high as 60%. It is important to manage patients with surgical intervention before it is complicated.

Keywords: Cardiac hydatid cyst, Cardiac parasitic infection, Hydatid cyst, Cardiac echinococcosis

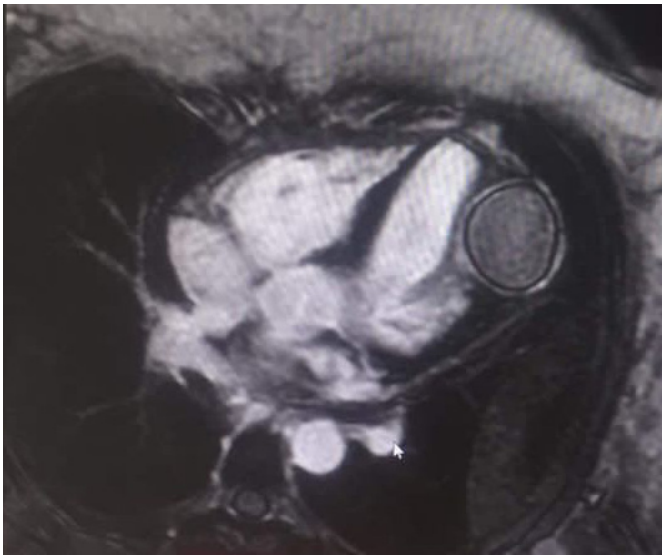

Figure 1. Cardiac magnetic resonance image that revealed hydatid cyst on the left ventricule apex.

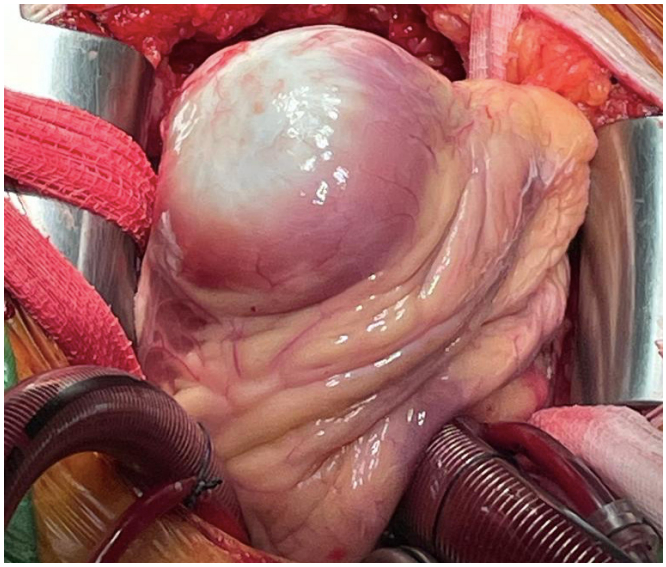

Figure 2. An operation view of hydatid cyst on the left ventricule apex.

Infections and antimicrobials

Abstract:0473

COXIELLA BURNETII CAUSING HAEMOPHAGOCYTIC SYNDROME: A RARE COMPLICATION OF AN UNUSUAL PATHOGEN

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Introduction: Q fever caused by Coxiella burnetii is a worldwide zoonosis. The clinical presentation of Q fever is polymorphic. The most common clinical syndromes in acute cases are self-limiting febrile illness, hepatitis and pneumonia. Here, a case with Q fever-associated hemophagocytic syndrome, which is very rare in the literature is presented.

Case: A 28-year-old male patient has no known disease or history of drug use. He applied to the emergency department with complaints of high fever, nausea, vomiting, and muscle pain. In the anamnesis, it was learned that the profession of the patient was a butcher and that he had been slaughtering dead illegal animals about 20 days ago. On physical examination, blood pressure was 110/70 mmHg, pulse: 75 bpm, body temperature: 37 0C. System examinations were normal. Widespread echymotic areas were seen in the bilateral lower extremity (picture 1). Laboratory values were as follows; SGOT(AST):208 U/L, SGPT(ALT):237 U/L alkaline phosphatase:197 U/L, gamma-glutamyl transferase:201 U/L, Total bilirubin:2.27 mg/dL, direct bilirubin:2 mg /dL, urea:140 mg/dL, Creatinine:1,75 mg/dL, C-reactive protein:320 mg/L, INR:3.1 in hemogram; white blood cell: 25.6 10^3/μL hemoglobin: 16.5 g/dL platelet count: 70 10^3/μL creatine kinase (CK): 747 IU/L, lactate dehydrogenase (LDH): 741 U/L troponin T: <15 ng/L, fibrinogen: 223 mg/dL ferritin: 5137 μg/L. No pathology was found in radiological imaging. Decreased platelets were seen in peripheral blood smear. A zoonotic agent panel was sent to the patient who had pancytopenia and myopathy accompanying multi-organ failure and had contact with a dead animal in his history. In the follow-up, triglyceride (TG):606 mg/dL progressed to CK: 3211 IU/L, ferritin: 8335 μg/L. From the sent zoonosis panel, Coxiella burnettii (Q fever) IgG (Phase 2) IFA was found positive at a titer of 1/128. Q fever-related hemophagocytic syndrome was considered in the patient with persistent fever, hypertriglyceridemia, thrombocytopenia, and hyperferritinemia. Intravenous immunoglobulin and doxycycline were started. After 3 days, liver and kidney functions returned to normal.CK:167 IU/L LDH: 214 U/L TG:162 mg/dL ferritin: 540 μg/L INR:1.4. After 7 days of observation, the patient was discharged with recovery.

Conclusion: Although many infectious etiologies have been associated with hemophagocytosis, Q fever has rarely been described in this context. The diagnosis of Q fever is often overlooked, especially when the picture is atypical. Here, the successful management of hemophagocytic syndrome developed during follow-up in a case of Q fever diagnosed with multiorgan failure is presented.

Keywords: Q fever, Coxiella burnetii, multiorgan failure

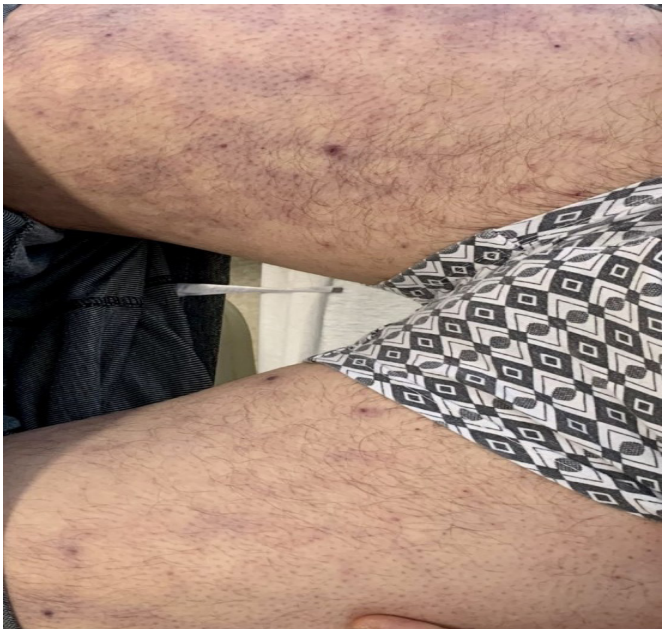

Figure 1

Hematology and Oncology

Abstract:0476

CARDIAC ADVERSE COMPLICATIONS DEVELOPED DURING LOW-DOSE REMISSION INDUCTION IN A PATIENT WITH NEW DIAGNOSIS ACUTE MYELOID LEUKEMIA IN ACCORDANCE WITH CURRENT GUIDELINES AND OUR MANAGEMENT PROCESS IN OUR INTENSIVE CARE UNIT: A CASE STUDY

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Introduction: Acute myeloid leukemia (AML) is in the group of hematological diseases that usually occur in patients aged 65 and over, difficult to manage with the addition of comorbidities and additional genomic factors. Responses to standard chemotherapy protocols are limited by hemodynamic instability. Therefore, new salvage therapy combines a lower dose potent bcl-2 inhibitor

with a hypomethylating agent in the older age group. Venetoclax-Azacitidine in AML salvage treatment and bridge to allogeneic cell transplantation. Our aim is to present the early diagnosis of rare cardiac complications due to chemotherapy and treatment management in the intensive care unit.

Case: A 74-year-old female patient applied to the hematology clinic due to complaints that had been going on for 2 weeks, and after the bone marrow biopsy, the patient's treatments were continued with a prediagnosis of AML. With cardiology approval, personalized low-dose combined chemotherapy regimen is started on 03.03.23. Azacitidine 75mg/m2 1x1, 7 days; Venetoclax 400mg 1x1, 21-day protocol was planned. Severe pleural and pericardial effusion were detected in Thorax CT. The patient is being evaluated by cardiology and thoracic surgery with the recommendation of close follow-up.

On the 15th day of the combined therapy, the patient with a pre-diagnosis of tamponade and septic shock with increased respiratory distress was admitted to our general intensive care unit. The patient was re-evaluated by cardiology. Pericardiocentesis was cancelled again because of the high risk of complications. However, the patient, who had respiratory distress, was quickly intubated. On the second day, the echocardiography was found to have newly developed hypokinesia in the segmental-inferoposterior area. The troponin-I level measured was significantly high. In the patient whose troponin follow-up was continued, the day after she was taken to coronary angiography with apheresis platelet and fresh frozen plasma replacement. A drug-eluting stent was placed in the circumflex artery. In the follow-up, only acetylsalicylic acid could be given as an antiaggregant and the patient developed acute renal failure. Despite all the medical interventions, she died on 23.03.23.

Conclusion: In addition to the complications that are frequently encountered after chemotherapy (CTx) in the middle and advanced age group, early diagnosis and treatment of cardiac complications that may develop during CTx are of vital importance. In common consensus with hematology, we think that MI and tamponade clinics were related to chemotherapeutics. In this regard, more studies and data are needed, both in the treatment dosage and management of complications.

Keywords: AML Management, Chemotherapy induced Cardiotoxicity, Chemotherapy induced Cardiogenic shock

Infections and antimicrobials

Abstract:0503

BOTULISM AFTER GASTRIC BOTOX APPLICATION

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Introduction: Botulism is a syndrome caused by a toxin acting on the nervous system produced by a bacterium called Clostridium botulinum, which can rarely be life-threatening and is accompanied by strokes. Iatrogenic botulism, on the other hand, is a rare condition that can occur after botulinum toxin injection for therapeutic/cosmetic purposes. While botulinum toxin weakens and paralyzes skeletal muscles, its effect is temporary. Affected muscle functions return to normal within 2-3 months. We also aimed to present the patient who developed botulism after gastric botox.

Case: A 36-year-old female patient with a BMI of 32.3 and no additional disease had stomach botox performed in another hospital 20 days ago. After the procedure, the patient with complaints of inability to walk, constipation, blurred vision, dysphagia and shortness of breath was re-admitted to the hospital where the operation was performed and the patient was discharged 4 days later. The patient applied to our emergency department with complaints of respiratory distress, muscle weakness and dysphagia. In the evaluation of the patient, blurred vision, 3/5 decrease in bilateral muscle strength in the lower and upper extremities, dysphagia, and hypoactive bowel movement were found. The patient who was considered to have botulism was admitted to the intensive care unit to be given antitoxin. In the intensive care unit, the patient's BP:155/71 mmHg, HR: 83/min, SpO2: 97%, RR: 24/min. While BAT® [Botulism Antitoxin Heptavalent (A, B, C, D, E, F, G) – (Equine)] was given to the patient as an antitoxin, the patient experienced flushing, dyspnea, decrease in SpO2, and itching in the throat and ear in the 1st minute could not be given. After electromyography (EMG), the patient was started with Pyridostigmine bromide 60 mg 3x1 and discharged to the service 3 days later (Figure 1).

Conclusion: The classic manifestation of botulism is acute onset, symmetrical, progressive muscle weakness from top to bottom, and bilateral paralysis of the cranial nerves. In cases such as the absence of fever, the presence of symmetrical neurological deficits, normal sensory and cognitive functions, normal or slowed heart rate, normal blood pressure, and the absence of sensory deficits other than blurred vision, botulism should definitely be considered in the differential diagnosis. The treatment of botulism mainly consists of anti-toxin therapy and intensive care support to support organ systems, including breathing, if necessary. In botulism, which is largely fatal if left untreated, mortality rates can be reduced to 5-8% with early diagnosis and treatment.

Keywords: Botox, Systemic Botulism, Gastric Botox, Intensive Care Unit, Antitoxin

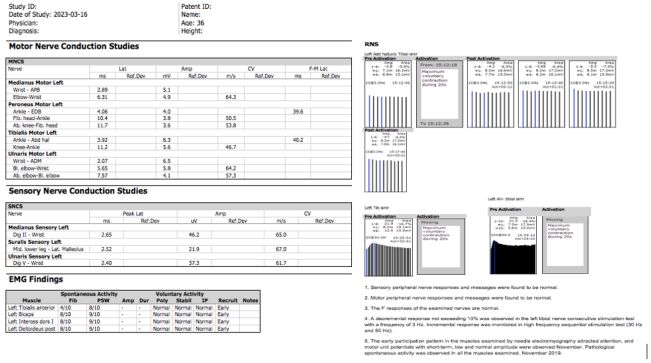
Study ID:
Date of Study: 2023-03-16
Physician:
Diagnosis:
Patient ID:
Name:
Age: 36
Height:
Motor Nerve Conduction Studies
Sensory Nerve Conduction Studies
EMG Findings
RNS

Figure 1. Electromyography (EMG) reports of the patient

Respiratory - ARDS and acute respiratory failure

Abstract:0579

EVENTRATION OF LEFT HEMIDIAPHRAGM WITH TYPE 2 RESPIRATORY FAILURE AND REVERSIBLE LEFT BUNDLE BRANCH BLOCK

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Introduction: Diaphragmatic eventration is defined as a permanent elevation of a hemidiaphragm without defects of continuity (1). In adults, it is usually caused by diaphragmatic palsy and may present with dyspnea. Long lasting eventration may result in corpulmonale (2).

Case: A 65-year-old male, admitted in high- dependency emergency ward of a 700- bedded tertiary care hospital of Kashmir, North India came with chief complaints of worsening breathlessness of 3 days duration, orthopnea, swelling of legs and altered sensorium with

similar history for last one year. Clinical examination on admission revealed signs of respiratory distress with mediastinal shift to the right. Apex beat was not palpable in its usual location. On auscultation, hyper resonant gurgling sounds were heard over the left chest. ABG revealed Type 2 respiratory failure. Chest X-ray (PA) showed elevated left dome of diaphragm with bowel loops and fundic air bubble way up in the left thoracic cavity. (Fig.1A) ECG revealed sinus tachycardia and LBBB (Fig.2A). High Resolution CT chest showed elevated left hemidiaphragm with displacement of large bowel, stomach and spleen towards left thoracic cavity. Diagnosis of eventration of left hemidiaphragm with congestive cardiac failure and type 2 respiratory failure was made. After clinical stabilization, definitive management was done by performing laparoscopic diaphragmatic plication surgery.

However, a point of interest is that post surgery there was complete reversal of LBBB and his ECG normalized completely. (Fig 2B) The patient was discharged in a stable condition with normal ABG analysis. A repeat ECHO done 3 months after surgery revealed significant drop in patient's pulmonary arterial pressures with normal ABG analysis. Postoperatively, spirometry didn't reveal any evidence of obstructive lung disease.

Conclusion: By reporting this case, our aim is to bring forward this interesting ECG finding, the possible and plausible explanation for it being the temporary mechanical cardiac displacement and/or pressure on the heart by the eventrated viscera.

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Keywords: Diaphragmatic eventration, Hypercapnic Respiratory Failure, Left Bundle Branch Block

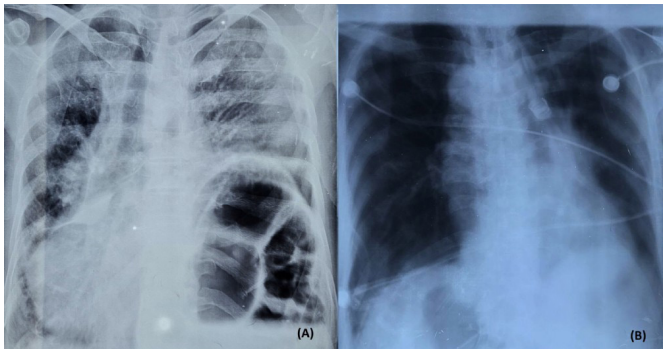

Figure 1. Chest Xray pre and post operative
Chest X-ray (A) Before surgery. (B) After laproscopic fundoplication.

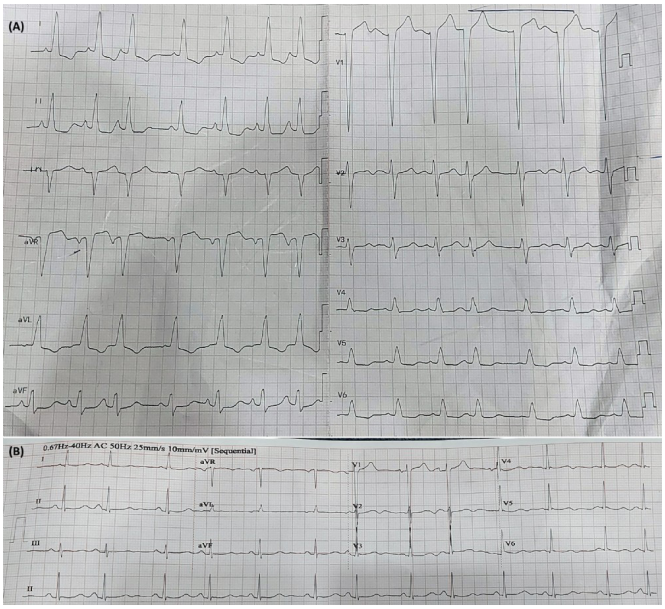

Figure 2. ECG pre and post operative
(A) ECG at presentation showing LBBB with atrial ectopics. (B) Post laproscopic fundoplication ECG showing reversal of LBBB.

Perioperative critical care

Abstract:0583

PRIMARY POSTPARTUM HAEMORRHAGE IN A MULTIPAROUS WOMAN: A MORTALITY AVERTED

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Introduction: Primary postpartum haemorrhage (PPH) is a common cause of maternal mortality. Vigilance monitoring and prompt intervention is imperative in improving outcome.

Case: A 41-year-old G7 Para 6+0 (4 alive) woman who presented with a 2 hour history of vaginal bleeding after a vaginal birth 2 hours prior, delivery was attended by a midwife in a primary health center with birth of an asphyxiated baby and early neonatal death. She was observed to be pale, with altered level of consciousness, tachycardic and hypotensive (80/30). A diagnosis of hypovolemic shock secondary to PPH with uterine atony was made. Resuscitation commenced immediately with oxygen delivery, intravenous fluids, oxytocin infusion and misoprostol suppository. Following no effect of oxytocics, emergency laparotomy was planned. Anesthesia involved ketamine induction, and suxamethonium to facilitate endotracheal intubation and maintenance with ketamine, surgery revealed an atonic uterus with a posterior vaginal wall tear necessitating a subtotal hysterectomy and repair of tears. The blood loss was 2L and received 4 units of blood, 1L of colloid, 2.5L of crystalloid and 1g of tranexamic acid, the drain had 450mls of fresh blood immediately and DIC was suspected, necessitating admission to the intensive care unit and consulted to the Haematology department. Sedation with ketamine 10mg and vasopressor support were ordered. She received 4 more units of whole blood, 2 units of fresh frozen plasma and tranexamic acid 500mg for 8 hours. After 36 hours in the ICU, she was noticed to be pale, PCV was 22.6%, platelet of 67000/mm3 while the abdominal drain at this point had drained 2.5L of frank blood over

a few hours, obvious abdominal distension and ultrasound scan that showed massive fluid for which haemoperitoneum was queried, she was transfused with 2 units of packed cells,1L of N/S and moved for emergency reexploration. Surgical findings were a slipped ligature at the angle of the right broad ligament, haemoperitoneum of 1.2L, and bleeds from the uterine stump. She received 2 more units of packed cells post op, was extubated 8 hours later, with minimal drainage by abdominal drain, a PCV of 35.8%, PLT- 227000/mm3. By the 5th day in ICU, abdominal drain had been empty for 72 hours, PCV was 33.5%, Hb: 11.7g/dl, PLT-164000/mm3. She was transferred to the ward and discharged home on the 13th day of admission.

Conclusion: Maternal critical care is important in improving maternal outcomes. Early involvement of the haematologist in cases of haemorrhage improves care.

Keywords: postpartum, haemorrhage, intensive care,

Sepsis - Other

Abstract:0587

A RARE RESISTANT AND MORTAL CANDIDA AGENT: CANDIDA AURIS

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Introduction: Candida auris is a rare, mortal invasive candidiasis agent associated with invasive healthcare and high immunosuppression. The agent, which has been rarely isolated in Istanbul, Izmir and Ankara until now in our country, has never been isolated in Antalya. In our case, we will describe Candida auris candidemia observed in a hematology patient.

Case: Twenty-six-year-old male patient, followed up with Diffuse B-Cell Lymphoma (DBCL), has no additional disease other than malignancy. He had been followed up in the hematology ward for about 4 months due to disease progression. He had a history of using multiple intravenous (IV) antibiotics due to febrile neutropenia attacks during his follow-up. After one week of without antibiotics, the patient started to have abdominal pain and bloodless diarrhea. In his vital signs, fever was 38.5 degrees and blood pressure was 90/60. On physical examination, there was tenderness and defense in the abdomen on palpation. Meropenem and Vancomycin IV treatment was started with a preliminary diagnosis of sepsis. Abdominal computed tomography (CT) was planned for the patient to rule out febrile colitis. A loculated fluid was detected in the abdomen, but sampling could not be performed because the platelet count was 15,000. Due to the increased oxygen and vasopressor requirements, the patient was followed up in the Internal Medicine Intensive Care Unit. It was informed that the blood culture taken during the febrile period and the urine culture had Candida Auris. Since the sensitivity is not yet known, Caspofungin IV was started empirically. Considering the minimum inhibitory concentration (MIC) values of the antifungals studied for susceptibility, Caspofungin treatment was discontinued, and Micafungin IV treatment was started instead. The patient, who was given to the ward, was taken back to the intensive care unit due to the development of septic shock with vasopressor requirement during the follow-up. In the 3-day intensive care follow-up, the patient died.

Conclusion: Because of the high intrinsic azole resistance, it is recommended to start treatment with an echinocandin. Close follow-up with control blood cultures is recommended because resistance can easily develop during treatment. Close contact isolation, hand hygiene and follow-up with high disinfection are recommended by the Centers of Disease Control and Prevention. We wanted to present our case because this very mortal factor is very rare both in our region and in our country.

Keywords: candida auris, rare, intrinsic resistance

Respiratory - Other

Abstract:0589

A SLE CASE WITH DIFFUSE ALVEOLAR HEMORRHAGE MANIFESTATION

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Introduction: Systemic lupus erythematosus (SLE) is an autoimmune connective tissue disease which has the potential to affect almost every organ in patients with autoantibody and immune complex production. Pleuropulmonary involvement is seen at a rate of 50-70% and often manifests as pleuritis and pleural effusion. Diffuse alveolar hemorrhage (DAH) is a rare finding of SLE, mostly seen in young adults with an underlying autoimmune disease such as systemic vasculitis or Goodpasture's syndrome. We wish to present SLE case which has DAH manifestation.

Case: A 19-year-old female patient with a diagnosis of SLE disease was admitted to the rheumatology service with complaints of diffuse edema, exertional dyspnea, and oliguria. Laboratory data at hospital admission was ANA:1/1000 granular homogeneous staining pattern, anti dsDNA:>800, ANCA: Negative, C3:0.4, c4:0.03, Sedimentation:97, CRP:7.5, creatinine:1.9, 24 hours urine protein: 2151 mg. With the diagnosis of lupus nephritis, the patient was followed up with 500 mg cyclophosphamide and pulse steroid followed by 1 mg/kg methylprednisolone. During her follow-ups, she was admitted to the intensive care unit due to her dry cough, hemoptysis, decrease in hemoglobin and decrease in oxygen saturation to 85. HRCT was performed with suspicion of hemorrhage on chest X-ray. Pleural and pericardial effusion, bilateral diffuse intralobular nodular densities (mimics diffuse alveolar hemorrhage) were seen in her HRCT (Figure 1). Bronchoscopy was performed following the findings. Bronchoalveolar lavage (BAL) was taken from the right middle lobe of the lung in bronchoscopy. BAL fluid was hemorrhagic and as it continued to be taken, its hemorrhagic character increased, suggesting alveolar hemorrhage. After the bronchoscopy, the patient was admitted to the intensive care unit. Then, 16 hours later, the patient was intubated after massive hemoptysis and desaturation was developed in the patient. She received 1000 mg of pulse steroid for 2 days in the service follow-up and 3 days after he was admitted to the intensive care unit, and apheresis was also applied. The patient was extubated 48 hours after intubation.

Conclusions: When dyspnea develops in patients with SLE, rare alveolar hemorrhages should also be kept in mind as a differential diagnosis. DAH has high mortality and early recognition and the treatment is life-saving for the patient. We experienced that our patient was extubated in the early period after early diagnosis and rapid pulse steroid and apheresis treatments.

Keywords: SLE, Diffuse Alveolar Hemorrhage, ICU

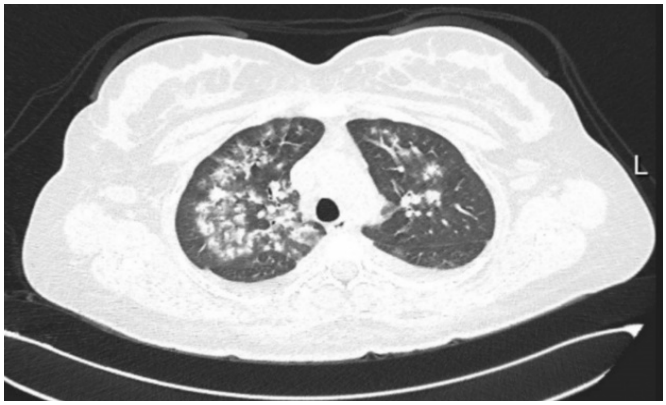

Figure 1. HRCT

Neuro-intensive care

Abstract:0602

A CASE REPORT ON PERIMORTEM CAESAREAN SECTION AND NEUROCRITICAL CARE

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Introduction: Maternal cardiopulmonary arrest is a challenge to caregivers when it happens. It has a prevalence of 1:12,500 pregnancies and the survival of both the mother and the child depends on a quick and focused resuscitation.

Case: The patient is a 40-year-old G3 P2+0 (2A) admitted as a case of bleeding PV at 32 weeks 3 days gestation. She had polyhydramnios and preeclampsia in this index pregnancy. She had an episode of fits on Day 4 and a decision was taken to deliver the baby via cesarean section. While preparing for surgery, she suffered a cardiac arrest, a code blue was called and the ICU team joined the resuscitation. CPR was commenced and she was intubated with a size 7.0 endotracheal tube. A perimortem cesarean was done and a live male neonate was delivered with an Apgar score of 1 and 6 at 1 and 5 minutes. The return of spontaneous circulation was less than 8 minutes (about 2 minutes after baby was delivered). The patient was then given Ceftriaxone 1g, vecuronium 6mg and was ventilated mechanically till surgery was over. In the ICU, mechanical ventilation was continued, and antibiotics were changed to meropenem and metronidazole after samples were taken for culture and sensitivity. Mannitol was administered. MgSo4 was continued according to the Prechard regimen.

Conclusion: In this case, the perimortem cesarean section proved to be useful as there was a return of ROSC and there was a delivery of a live baby. Conversion of the perimortem cesarean section to general anaesthesia could have played a part in the recovery process of the patient as it proly prevented injury to adjoining structures. When the intestines were disturbing surgery after the baby had been delivered, ketamine and vecuronium was administered and she was connected to a transport ventilator. The careful usage of antibiotics and mannitol could have helped by reducing cerebral edema. Another factor that probably worked positively is the social support from the family. The husband and father were given unhindered access to her and they kept talking to her and encouraging her before she became conscious until she was discharged. The success of this case report will be the prompt delivery of the baby and its impact on resuscitation, converting to general anaesthesia after ROSC, the use of mannitol in the immediate postoperative period and the prompt use of preemptive antibiotics.

Keywords: postmortem caesarean cardiac arrest eclampsia, cerebral edema, general anaesthesia

Respiratory - Other

Abstract:0608

STRIDOR: AN UNCOMMON SIGN THAT MAY WARN CARCINOMA OF ESOPHAGUS

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Introduction: Stridor is a variable, high pitched respiratory sound that can occur either during inspiration or expiration or both, most typically during inspiration. An obstruction or narrowing in upper airway of variable pathology may present with stridor. It's a medical emergency that may requires urgent intervention. Rarely an esophageal or thyroid or neck or chest growth may present with stridor other than the upper airway pathology, infection or inflammation.

Case: A 61-year-old pleasant lady with known DM (Diabetes Mellitus), CLD (Chronic Liver Disease), presented to a tertiary level Intensive Care Unit from a district hospital with endotracheal tube in situ having the history of severe respiratory distress and stridor for four days followed by cardiac arrest with ROSC (Return Of Spontaneous Circulation) after 15 minutes. She had history of cough with occasional shortness of breath for one month. CT (Computed tomography) of chest done after 10 days of her symptoms as per advise of a general physician, showing a neck mass behind the trachea, which was thoroughly neglected. On query patient denied of any complain of difficulty in deglutition, loss of appetite, neck pain or swelling, weight loss. After admission in our hospital repeat CT neck done with CT guided FNAC (Fine Needle Aspiration Cytology) which revealed mixed density lesion in pre and right paravertebral location at C7 to D2 levels, that compressed and invade the posterior wall of trachea causing significant narrowing of adjacent airway, as well as esophagus is compressed and displaced towards left. FNAC was suggestive of metastatic squamous cell carcinoma irrespective of the origin. To find out the primary site, Bronchoscopy was done, which showed inflamed mucosa without any visible growth. Endoscopy was the next, which revealed polypoid growth on the posterior wall from 25 to 30 cm from the teeth line. Histopathology of the growth revealed poorly differentiated squamous cell carcinoma of esophagus. Subsequently patient underwent tracheostomy and after stabilization shifted under care of oncology for further management.

Conclusion: Stridor is a sign that must be addressed and properly investigated to find out the cause when there is no visible local finding in oropharynx and laryngopharynx. Esophageal carcinoma may present with stridor other than it's usual presentation due to mass effect. Clinician should always have high index of suspicion of carcinoma in an elderly patient presenting with stridor.

Keywords: Stridor, FNAC (Fine Needle Aspiration Cytology), Endoscopy, Carcinoma of Esophagus

Sepsis - Management

Abstract:0609

INTRAVENOUS DRUG USE RELATED INFECTIVE ENDOCARDITIS

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Introduction: Infective endocarditis (IE) is a rare infectious disease that still maintains its importance due to the high morbidity and mortality. Predisposing factors include rheumatic heart diseases, artificial heart valves, and intravenous drug use. We present our case of infective endocarditis, a 36-year-old patient with intravenous (IV) drug addiction, followed up for septic shock and multiple organ failure after pregnancy loss.

Case: A 36-year-old, 33-week pregnant patient with bipolar disorder and IV drug abuse was being followed up in an external center due to a prediagnosis of HELLP syndrome, intrauterine ex fetus, and blurred consciousness. After the development of septic shock and multiple organ failure, she was admitted from another center for advanced diagnosis and treatment. Due to the development of cyanosis in both fingers and toes, upper and lower extremity doppler ultrasonography was made; no stenozis, triphasic normal flow detected.

Blood Results: CRP:122 mg/L,Procalcitonin>100μg/L,creatinin 2,26 mg/dL, GFR:27mL/dk/1.73m2, PLT:30000, d-dimer:36 μg/L, WBC:15000, HCV AB: positive, HCVRNA:9990 IU/ml, Total Bilirubine 4 mg/dL Direct Bilirubine:3.5 mg/dL,AST:203 U/L, ALT:126 U/L, C3:0,50 (normal 0.9-1.8), C4 normal

Renal Doppler ultrasonography was normal, no abscess was observed in abdominal ultrasonography. Transthoracic echocardiography (TTE) performed by us on the first day of his hospitalization showed a mass image similar to myocardial echogenicity in the right ventricle, a cardiology consultation was requested, their TEE report: right cavities wide, the ejection fraction was evaluated as 60%, no mass image was observed. Since the clinic was thought to be compatible with infective endocarditis, meropenem and vancomycin treatment was started on the first day of hospitalization. The blood culture obtained was concluded on the 4th day of hospitalization. Staphylococcus aureus was detected and the patient was screened again for infective endocarditis. Colistin and fluconazole were added to treatment based on blood culture result. Roth spot was found bilaterally positive. Transesophageal echocardiography (TEE) performed on the 6th day of hospitalization revealed vegetation in the non-coronary cusp of the aortic valve. Cyanotic lesions on fingers and toes were accepted as septic embolism, vasodilator agents were started. Since the patient in septic shock did not respond to the treatment, she was accepted as exitus on the 19th day.

Conclusion: Patient's history and physical examination findings are very important in the diagnosis of infective endocarditis. We presented our patient, who used intravenous drugs, and was progressing with septic shock after pregnancy loss, in order to raise awareness of this disease and to emphasize the importance of ECO use in the intensive care unit.

Keywords: infective endocarditis, iv drug use, cardiac thrombus

Kidney - Acute kidney injury

Abstract:0614

NOSOCOMIAL SARS-COV-2 INFECTION PRESENTING WITH ACUTE KIDNEY INJURY

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Introduction: Coronavirus disease 2019 (COVID-19) has been predominantly associated with respiratory symptoms, but it has also been shown to cause acute kidney injury (AKI), leading to increased hospitalization and mortality. Nosocomial Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) infections are a concern in intensive care units (ICUs), especially with relaxed mask mandates and visitor permissions. Early detection and treatment of nosocomial COVID-19 are crucial for effective care.

Case: We present a case of a 91-year-old female patient with a negative initial nasopharyngeal swab for SARS-CoV-2 upon ICU admission. The patient had a medical history of multiple comorbidities and presented with deterioration of consciousness, poor oral intake and respiratory distress. Although her initial creatinine level was 1,39 mg/dL (0.50–0.90 mg/dL), it improved to normal values with hydration. On the follow-up she developed an acute kidney failure. Despite investigations and treatment for possible causes, the renal function did not improve. A nosocomial SARS-CoV-2 infection was suspected and confirmed by a subsequent positive nasopharyngeal swab. The patient's condition continued to deteriorate, requiring mechanical ventilation and eventually resulting in cardiac arrest and death.

Conclusion: This case highlights the importance of considering COVID-19 in the differential diagnosis of patients with AKI. Sepsis, dehydration, and nephrotoxic medications are common causes of AKI in the ICU, however; COVID-19 should be evaluated and considered alongside other etiologies of AKI when the renal function does not improve despite appropriate treatment. Nosocomial SARS-CoV-2 infections pose a significant challenge in ICUs, with factors such as advanced age, prolonged ICU stay, and invasive procedures increasing the risk. COVID-19-induced AKI is thought to involve inflammatory and immune responses, endothelial injury, hypercoagulability, and the renin-angiotensin pathway. Older age, hypertension, and a history of renal disease are important risk factors for COVID-19-induced AKI and are associated with higher mortality rates. Prompt identification and isolation of COVID-19 patients in hospitals are crucial for controlling the spread of the virus and reducing in-hospital mortality. Increased awareness and implementation of preventive measures are necessary to control the transmission of COVID-19 in healthcare settings.

Keywords: nosocomial infection, COVID-19, acute kidney injury

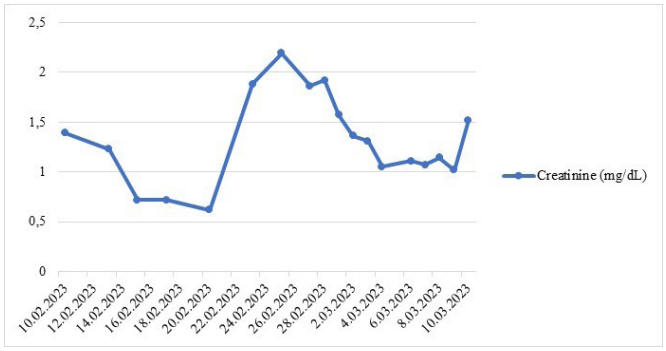

Figure 1. Creatinine trend during hospitalization
The patient's initial elevated creatinine level was evaluated as prerenal acute kidney failure. 4 days later, after appropriate hydration, her creatinine levels were measured within the normal range. However, on the 12th day of hospitalization, it increased from 0,62 mg/dL to 1,88 mg/dL, subsequently attributed to SARS-CoV2 infection.

Infections and antimicrobials

Abstract:0615

ISONIAZID AND RIFAMPICIN RESISTANT TUBERCULOUS MENINGITIS WITH INTRACRANIAL TUBERCULOMA: A CASE REPORT

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Introduction: Forms of central nervous system (CNS) infection due to Mycobacterium tuberculosis (MT) include meningitis, tuberculoma, spinal arachnoiditis, and transverse myelitis. Tuberculoma may present as a clinically evident mass lesion of the brain in the absence of tuberculous meningitis (TBM). The establishment of a cortical or meningeal tubercle, with subsequent rupture into the subarachnoid space, is a critical event in the development of TBM. Resistance to isoniazid and rifampicin in tuberculosis cases is quite rare. In this report, a case of TBM with tuberculoma caused by resistant MT and confused with an intracranial mass is presented.

Case: A 25-year-old male patient has complaints of headache and weight loss for 1 month. He was brought to emergency room by his relatives after he had a seizure. No features on his resume GCS:15. Routine tests are unremarkable. Elisa is negative. Brain CT showed a mass of 15 mm in the left frontal area and brain edema in its vicinity. Patient was admitted to neurosurgery clinic. On 3rd day, patient started to have speech and gait disturbances. Upon the development of nuchal rigidity and fever, LP was performed with the preliminary diagnosis of meningitis. CSF examination showed acid-fast bacilli, MT-PCR was reported as positive. Patient was transferred to infectious disease clinic and quadruple antituberculosis treatment was started. Thorax and abdomen CT revealed multiple lymph nodes, true-cut biopsy was obtained from the abdominal lymph node considering the preliminary diagnosis of lymphoma, it was reported as reactive. On the 10th day, the patient who developed status epilepticus was intubated and transferred to ICU. An increase in hydrocephalus was observed in the control brain-CT. CSF drainage was impaired secondary to meningitis. Neurosurgery clinic performed mass excision and decompression surgery. EZN stain of material sent from brain tissue and dura showed MT bacillus and necrotizing granulomatous inflammation in its pathology. Thus, it was determined that intracranial mass was tuberculoma. On the 10th day of ICU, rifampicin and isoniazid resistance was reported. It was revised as MT treatment (Pyrazinamide, Cycloserine, Prothionamide, Paraaminosalicylic Acid, Moxifloxacin, Amikacin). PPD and ARB in the respiratory secretion were negative 3 times. The patient had no history of contact. LP was renewed in patient with intermittent fever which showed mononuclear cell dominance and high protein content continued. In the follow-up, P.aeruginosa and panresistant K.pneumoniae were grown in BAL culture. Ceftazidime-avibactam were added to the treatment. On the 40th day patient died due to secondary infections.

Conclusion: In endemic areas, tuberculosis may present as a mass lesion of brain. The presented case is important in that it is a rare dual-drug-resistant extrapulmonary tuberculosis case reported in our country and presents with intracranial mass effect.

Keywords: Mycobacterium tuberculosis, meningitis, tuberculoma, drug resistance, intracranial mass

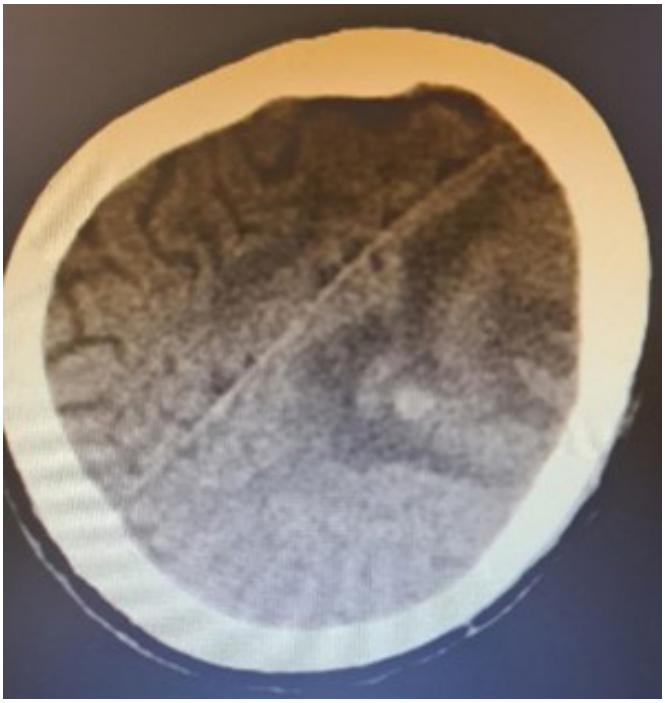

Figure 1. Brain CT image at admission

Infections and antimicrobials

Abstract:0616

DIFFERENTIAL DIAGNOSIS OF SUSPECTED STATUS EPILEPTICUS IN INTENSIVE CARE: A CASE OF SPORADIC CREUTZFELDT-JAKOB AND ITS END-OF-LIFE DECISION

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Introduction: Creutzfeldt-Jakob disease (CJD) is a rare. Its prevalence is between 1 and 1.5 cases per 1 million. Sporadic cases (sCJD) are the most frequent forms (85%). This infectious disease is difficult to diagnose, especially in its early forms, and may have very polymorphic clinical presentations. Our aim was to highlight this exceptional beginning of sCJD (new-onset refractory status epilepticus: NCSE) and draw attention to the end-of-life decision.

Case: A 67-year-old female patient with no comorbidities other than hypertension was admitted to the intensive care unit for diagnosis of limbic encephalitis. Antibiotics were started because the suspicion of meningitis could not be ruled out due to the patient's febrile values, resistant hypotension and high cerebral fluid protein. One month before the admission, she had symptoms of anxiety, forgetfulness, progressive gait disorder, involuntary movements in the upper extremity and myoclonus-like movements, and difficulty in speaking. On Magnetic Resonance Imaging (MRI), there was a cortical diffusion restriction, predominantly in the bilateral basal ganglia and parietal region in the left hemisphere. Herpes was considered due to the lesion in the temporal region and antiviral treatment was started. EEG pattern characterized by very frequent subclinical seizures originating from the left hemisphere and spreading to the opposite hemisphere was observed. Pulmonary thromboembolism was detected in the imaging taken due to sudden respiratory failure and thrombolytic therapy was given. Glasgow coma scale regressed to 3 and she was intubated due to increased oxygen demand. The patient developed a seizure pattern compatible with NCSE. Typical MRI and EEG findings and a positive 14-3-3 protein led to the diagnosis of sCJD. Despite the cessation of sedation in the follow-up of the patient, no awakening was observed and was agreed to Do-Not-Attempt-Resuscitation (DNAR). Until the patient with tracheostomy and percutaneous gastrostomy was transferred to palliative care, continued to have seizures despite quadruple antiepileptic treatments during the 2-month follow-up.

Conclusion: We believe that the differential diagnosis needs to be carefully evaluated in order not to miss a possible reversible condition. sCJD should be considered in the differential diagnosis of patients with rapid cognitive decline and EEG changes consistent with NCSE. On the other hand, there are case reports arguing that the treatment should be terminated when this diagnostic triad is positive like our patient, whom we decided DNAR and sent to the palliative care center. This is a controversial topic.

Keywords: Creutzfeldt-Jakob Syndrome, status epilepticus, tau Proteins, Intensive Care Units

Trauma

Abstract:0623

LIFESAVING EARLY SPECIALIST INTERVENTION FOR MULTIPLE TRAUMA PATIENT IN TURKEY EARTHQUAKE DISASTER

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Introduction: Natural disasters such as earthquakes cause unexpected deaths. Early intervention is crucial in reducing the life-threatening risk of multiple trauma patients rescued from the rubble (1). The importance of expert intervention in trauma patients in the emergency, operating, and intensive care units (ICU) are evident. On February 6, 2023, two earthquakes occurred with a magnitude of 7.7 and 7.6 occurred in Kahramanmaras. We present the successful management of the multiple trauma patient after this earthquake disaster.

Case: A 62-year-old man, rescued by relatives from the rubble, was brought to the emergency room of Sanlıurfa Hospital within 15 minutes. Imaging examinations revealed hemopneumothorax and multiple rib fractures on the right, retroperitoneal minimal hematoma, and a fragmented fracture in the distal left tibia with a possible cut in the tibial artery (Figure). Upon arrival at the hospital, the patient underwent a right tube thoracostomy, and an erythrocyte transfusion was started. Within the first hour, the orthopedist reduced the patient's fracture and the cardiovascular surgeon performed the tibialis posterior artery anastomosis. An external fixator procedure was applied, and the patient was admitted to the ICU as intubated due to hypoxia. The intensivist observed that the patient had flail chest. He preferred pressure-controlled mode over volume-controlled mode to avoid increasing pneumothorax. In addition, he tried to ensure effective volume by stabilization by applying a bandage between the sternum and thoracic vertebrae. The patient was extubated on the 9th day after the earthquake and was transferred to the orthopedic ward on the 11th day.

Conclusion: In our case, a chest tube was inserted within 30 minutes after injury and damage control surgery was performed within the first hour of the first earthquake. The patient survived fatal injuries like hypothermia, crush syndrome, pneumothorax, hemothorax, flail chest and arteria tibialis laceration with early specialist intervention. Despite the magnitude of the earthquake, reaching this positive outcome makes the situation different. Studies have shown that rapid intervention by a trauma team determined by protocols positively affects patient outcomes (2). This case report highlights the importance of early intensivist and other specialist intervention, multidisciplinary teamwork, and resuscitation of multiple trauma patients during major disasters.

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Keywords: intensive care, hemopneumothorax, Kahramanmaras earthquake

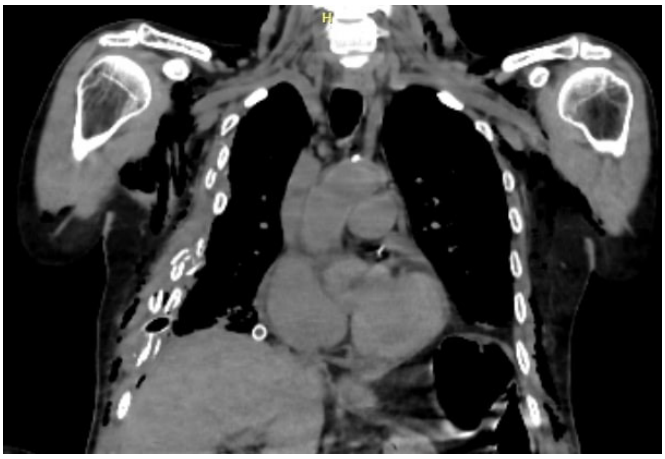

Figure 1. Chest computed tomography(coronal plane)

Respiratory - Other

Abstract:0630

JOUBERT SYNDROME: DIFFERENTIAL DIAGNOSIS OF HYPERPNEA

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Introduction: Joubert syndrome is an autosomal recessive disease characterized by episodes of hypotonia, ataxia, and episodic hyperpnea. The disease can be diagnosed by evaluating the clinical and radiological findings together. Clinically, abnormal eye movements, nystagmus, hyperpnea-apnea episodes, and mental-motor retardation are observed. Radiologically, the leading central nervous system anomalies are the cerebellum vermis, brain stem hypoplasia, and "molar tooth sign" detected in brain radiology.

Case: A 70-year-old male patient with known diagnoses of COPD, diabetes mellitus, hypertension, and chronic kidney failure is brought to the emergency department due to a change in respiratory pattern. He is admitted to the intensive care unit due to respiratory distress with a diagnosis of subsegmental pulmonary embolism and pneumonia. Extubation was attempted twice after treatment, but the patient was tachypneic and whose respiratory pattern changed. He was investigated in terms of central pathologies. According to the anamnesis taken from the patient's relatives, the patient with motor-mental developmental delay had balance (ataxia) and vision problems at later ages. The samples were applied to the medical genetics department for examination for the diagnosis of Joubert syndrome. In the neurological examination, see-saw nystagmus was observed in the eye, and no pathology was observed in the eye, cardiac, and abdominorenal system examinations. The clinical correlation was provided when the brain MR images showed a molar tooth image (Figure 1) and cerebellum dysplasia in the brain. Patient relatives' consent was obtained for the case report.

Conclusion: Joubert syndrome should be considered in the differential diagnosis with clinical correlation in patients with episodic hyperpnea attacks and weaning difficulties. The diagnosis of the disease can be made by evaluating the clinical and radiological findings together and genetic studies.

Keywords: Joubert syndrom, hyperpnea, episodic

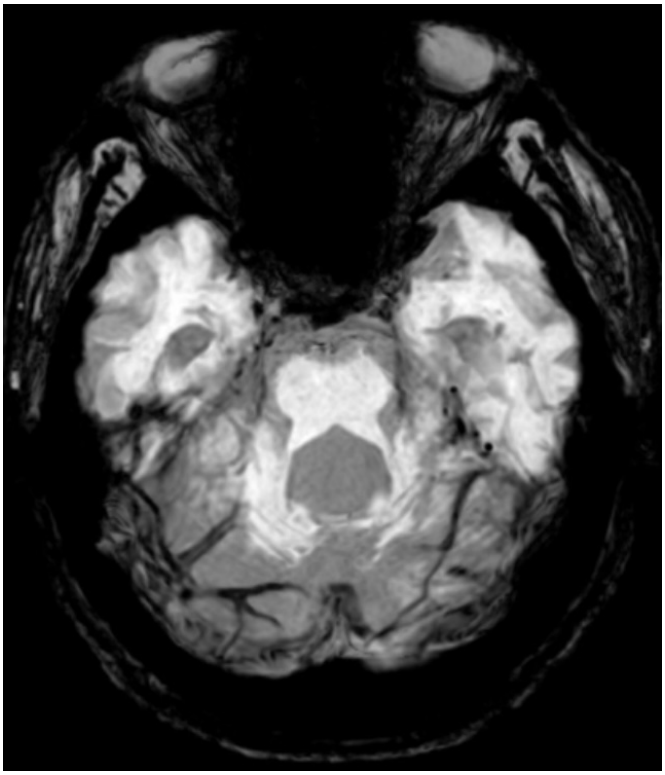

Figure 1. Molar tooth sign on MRI

Respiratory - Other

Abstract:0638

RARE AND SIGNIFICANT: RITUXIMAB INDUCED INTERSTITIAL LUNG DISEASE TREATED WITH PLASMAPHERESIS

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Introduction: Rituximab is an anti-CD20 antibody that can be used to treat many hematologic and immunologic diseases, but rarely it can cause a less known, but fatal; rituximab-induced interstitial lung disease. In this case, we present a 33-year-old man diagnosed with myasthenia gravis who was treated in our intensive care unit after administration of rituximab.

Case:A 33-year-old male ex-smoker with a history of myasthenia gravis had been treated with rituximab every 6 months since 2019. The patient tested positive for COVID-19 in May 2022 and presented to our clinic in October 2022 with a cough. Our initial differential diagnosis included long COVID and rituximab-induced lung disease (R-ILD). Bronchoalveolar lavage and transbronchial lung biopsy showed normal cell differentiation and benign cytology with reactive epithelial changes. To confirm our differential diagnosis, we suggested a change in rituximab treatment. Rituximab treatment was delayed for one month while the patient underwent corticosteroid treatment and experienced dyspnea. After one month, rituximab was administered with a persistent suggestion from the neurology department. After administration, due to progressive desaturation with a non-rebreather mask, the patient was admitted to the intensive care

unit (ICU). In the ICU, the patient continued to desaturate, requiring the use of HFNC (60L/dk, %100 FiO2) for adequate oxygen delivery. Given the diagnosis of R-ILD, we initiated pulse steroid therapy and 0.4 g/kg IVIG for three days. Due to progression despite all treatments, plasmapheresis was performed. After plasmapheresis, a rapid decrease in oxygen demand was observed. After a few more days in the ICU, the patient was discharged to the ward.

Conclusion: This case report highlights the rare but significant occurrence of rituximab induced lung disease. Our patient was diagnosed with myasthenia gravis and receiving rituximab as part of his treatment. Healthcare providers should be aware of this potential complication to ensure safe and effective use of rituximab. It has been demonstrated that dramatic improvements can be achieved with a single session of plasma exchange therapy in these patients who have few cases and treatment options in the literature.

Keywords: Rituximab, Plasmapheresis, Respiratory Insufficiency

Kidney - Other

Abstract:0641

MANAGEMENT OF PATIENTS WITH FAMILIAL MEDITERRANEAN FEVER IN THE ICU - CASE REPORT

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Introduction: Familial Mediterranean Fever (FMF) is an autosomal recessive genetic disorder, which though rare worldwide, affects 1 in 1000 people of Mediterranean origin. This autoinflammatory disease is caused by mutations in the Mediterranean fever gene located on chromosome 16 (16p13), which encodes a 781–amino acid protein called pyrin. FMF causes recurrent episodes of fever and serositis, leading to painful attacks during early childhood. The most fatal complication of FMF is amyloidosis. Patients suffering from FMF with secondary amyloidosis have an average life span of 24-53 months from diagnosis. FMF has been associated with various vasculitis, especially Henoch-Schonlein Purpura (HSP) and Polyarteritis nodosa (PAN). Deposition of amyloid damages the tunica media and adventitia of affected vessels resulting in stenosis of the vascular lumen and fragmentation of the internal elastic lamina. These changes may lead to fibrinoid necrosis and microaneurysms predisposed to hemorrhage.

Case: A 49-year-old patient was admitted to ICU with hemodynamically unstable TA 70/40, HR 110, Sp02 96%, intubated and mechanically ventilated. The patient had been undergoing regular dialysis treatment 3 to 4 times per week for his end-stage kidney disease. He was in septic shock, his ABG showed metabolic acidosis, and his laboratory results showed anemia and thrombocytopenia with massive epistaxis from the nasopharynx and oropharynx. Hemostasis tests were normal. Nasal, anterior and posterior oropharyngeal tamponade were performed. The patient was given blood derivates, fresh frozen plasma and platelets, tranexamic acid, prothrombin complex concentrate and inotropes, and was put on corticosteroid therapy. The CT scan showed intracerebral hemorrhage, pleural effusion with pulmonary atelectasis, and hypoplastic kidneys. Despite expert efforts from rheumatology, nephrology and neurosurgery, the patient expired.

Conclusion: We present a rare case of amyloidosis complications in a patient with FMF accompanied by massive hemorrhage and end-stage renal disease. FMF patients with PAN-like vasculitis may suffer from distinct small and medium vessel vasculitis, with early onset and more perirenal hematoma and CNS involvement. The vasculitis diagnosis can either precede the FMF diagnosis or occur during a previously known FMF. The link between these two conditions is unknown and requires further studies. Prevention and early management of FMF are crucial for avoiding serious complications.

Keywords: amyloidosis, FMF, management

Kidney - Acute kidney injury

Abstract:0643

SYSTEMIC DRUG AND VITILIGO: A RHABDOMYOLYSIS STORY

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Introduction: Rhabdomyolysis, a recognized adverse outcome of statin therapy, poses an increased risk when combined with drugs that inhibit cytochrome p450-3A4 (CYP3A4).

Case: We present a case study of a 45-year-old patient with acrofacial vitiligo who experienced generalized weakness, muscle pain, decreased urine output, and these symptoms persisted for three days. The patient had previously undergone intensive treatment involving multiple immunosuppressive drugs and simvastatin in Egypt. Then he returns home from Egypt. Following that, the patient drove from Aden airport to Sana'a for 12 hours without stopping, resulting in acute rhabdomyolysis and admittance to the critical care unit. On general examination, the patient exhibited consciousness, orientation, normal body temperature, and no signs of pallor, cyanosis, or jaundice. Vital signs were as follows: blood pressure of 139/72 mm Hg, pulse rate of 80 beats per minute, respiratory rate of 30-36 breaths per minute, and central venous pressure of 5 cmH2O. Upon admission, initial investigations revealed the following results: hemoglobin level 15.3 g/dL, white blood cell count 16.1 × 109/L, neutrophils 81.3%, lymphocytes 3.4%, platelet count 393 × 109/L, creatinine level 4.16 mg/dL, urea level 202 mg/dL, potassium level 7.62 mmol/L, ALT level 837 IU/L, AST level 147 IU/L, serum albumin level 3.4 g/L, CPK level 4033 IU/L, arterial blood gas measurements showing pH 7.35, PCO2 22.6 mmHg, HCO3 12.6 mmHg, SaO2 98%, and lactate level 1.9 mmol/L.

The treatment plan included aggressive intravenous fluid therapy with normal saline and sodium bicarbonate, along with diuretic administration to promote urine output. A hemodialysis catheter was inserted for an urgent hemodialysis session, with monitoring of fluid intake and ultrafiltration. An anti-hyperkalemia protocol was implemented, a proton pump inhibitor was prescribed, and heparin prophylaxis was initiated. On 2nd day patient was arrested and connected to mechanical ventilator and improved on hemodialysis. A sepsis management plan involving cultures and sensitivities was established, and empirical antibiotic therapy was administered. These interventions aimed to address the patient's condition and manage potential complications associated with rhabdomyolysis.

Conclusion: This case emphasizes the need to be aware of drug interactions related to statins and immunosuppressive drugs, as well as the importance of early detection and therapy of rhabdomyolysis.

Keywords: Rhabdomyolysis, Vitiligo, Statin, Immunosuppressive drugs

Neuro-intensive care

Abstract:0644

A CLINICAL CASE REPORT OF BRAIN ABSCESS CAUSED BY NOCARDIA OTIDISCAVARUM IN AN IMMUNOCOMPROMISED PATIENT

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Introduction: Nocardia is an aerobic, gram-positive bacteria from the actinomycetes group that can cause opportunistic infections, usually in immunocompromised hosts. The central nervous system is the most common site of extrapulmonary. In this case, we presented an immunocompromised patient who developed multiple brain abscesses caused by Nocardia otidiscavarum. (1)

Case: A 50-year-old male patient with the diagnosis of idiopathic thrombocytopenic purpura and hypertension was admitted to the emergency department of our hospital with complaints of headache, dizziness, and inability to walk. In her neurological examination, he was conscious, prone to sleep, pupillary isochoric, and light reflex were observed. Cranial computed tomography (CT) was performed for his neurological complaints. On CT imaging, linear hyperdensity of approximately 5 mm in the right cerebellar hemisphere, multiple nodular lesions in the supratentorial brain parenchyma, the largest in the plane of the centrum semiovale, approximately 16 mm in the parafalcian area, and hypodensities compatible with edema around it. (figure 1). High-resolution pulmonary CT was performed on the patient describing hemoptysis. (figure 2). In the intensive care follow-up, the patient's consciousness worsened. On MR imaging, there is multiple hyperintense lesions with limited edema and diffusion of surrounding lesions due to the abscess content. (figure 3) Due to the progressive deterioration of the patient's neurological status, the patient was evaluated in the multidisciplinary council. Diagnostic brain biopsy was performed. Nocardia-compatible bacteria and hyphal structures in the gram-positive filamentous structure were detected in the tissue biopsy culture, and Nocardia otitidiscaviarum was grown in the culture.

Conclusion: The incidence of nocardial infection has been increasing gradually due to the increase in the elderly population and the number of patients receiving immunosuppressive therapy (2). In the literature, it has been shown that Nocardial brain abscesses are caused mainly by common Nocardia species such as Nocardia abscessus, Nocardia farcinica, and Nocardia asteroide. Nocardia otidiscavarum was seen in only 3% of nocardial infections.

The treatment of Nocardia brain abscesses relies on long-term antibiotic therapy and surgical drainage. The neurosurgical approach is essential in the rapid diagnosis and source control of brain abscesses. However, the optimal management of cerebral Nocardiosis is not yet clear.

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Keywords: Nocardia otidiscavarum, Cerebral Nocardiosis, brain abscess, treatment, effective antibiotics

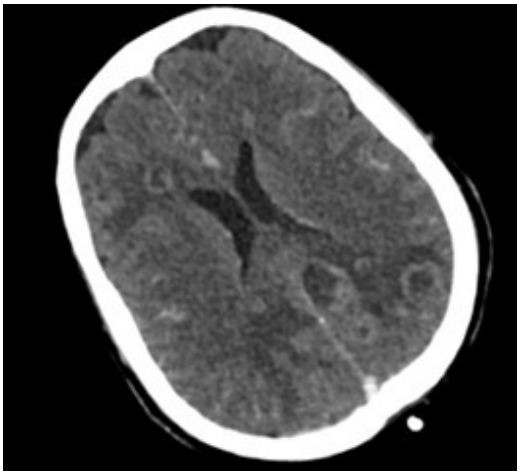

Figure 1. Contrast enhanced axial CT image
Contrast enhanced axial CT image shows multiple ring- enhanced nodular lesions with surrounding edema.

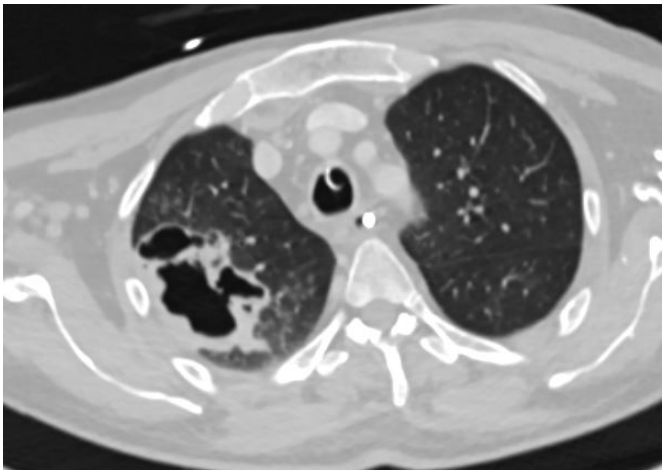

Figure 2. High-resolution CT
High-resolution CT showed a 5.5 cm cavitary lesion in the right lung's upper lobe apical and posterior segment.

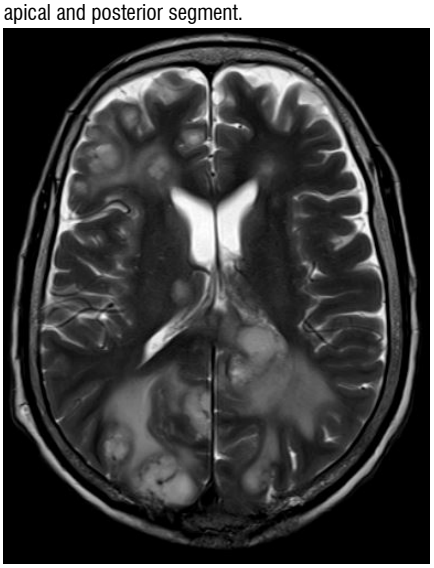

Figure 3A. Axial T2-weighted image
Axial T2-weighted image demonstrates multiple hyperintens lesions with surrounding edema

Infections and antimicrobials

Abstract:0656

MILIARY TUBERCULOSIS AND TUBERCULOMA IN PREGNANCY-A RARE FINDING

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Introduction: Central nervous system tuberculosis (CNS-TB) is rarely suspected in pregnancy because its presentation may mimic other common conditions in pregnancy, such as pre-eclampsia or eclampsia (1). In high tuberculosis endemic areas like India, CNS-TB should be suspected with a high degree of suspicion among immunosuppressed states like pregnancy (1).

Case: We report a case of a 24-year-old primigravida in 3rd trimester of pregnancy with complaints of pain over right side of face and head followed by unilateral left side weakness, diplopia in right eye, epigastric pain and low grade fever with respiratory distress with raised BP and deranged liver function tests. CE-MRI brain and spine revealed ill-defined areas of altered signal intensity which were hypointense on T2WI with surrounding T2/Flair hyperintensity were seen in bilateral cerebral hemispheres predominantly at grey-white matter junction, right caudate nucleus, right hypothalamus, left middle cerebellar peduncle and in bilateral cerebellar hemispheres, thus multiple ring-enhancing lesions in supratentorial and infratentorial brain parenchyma. The lesions showed diffusion restriction on DWI with a nodular enhancing lesion in the spinal cord at D4 vertebral level suggestive of infective granuloma, likely Tuberculosis. Fundoscopy revealed choroid tubercles. A preterm cesarean section was performed at 32 weeks gestational age due to preterm rupture of membranes. One day after delivery patient developed respiratory distress with a fall in saturation and patient got intubated. CTPA was done which revealed lobar consolidation in left lower lobe with confluent areas of patchy consolidation and ground glass opacities in right upper lobe, right middle lobe and left upper lobe and normal pulmonary angiography. Tracheal CBNAAT was positive suggesting miliary tuberculosis with Tuberculoma. Patient was started on antitubercular drugs. The patient was extubated after 2 days with steady neurological improvement and shifted to the ward.

Conclusion: This case highlights the role of imaging for early diagnosis and treatment.

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Keywords: Tuberculosis, tuberculoma, pregnancy

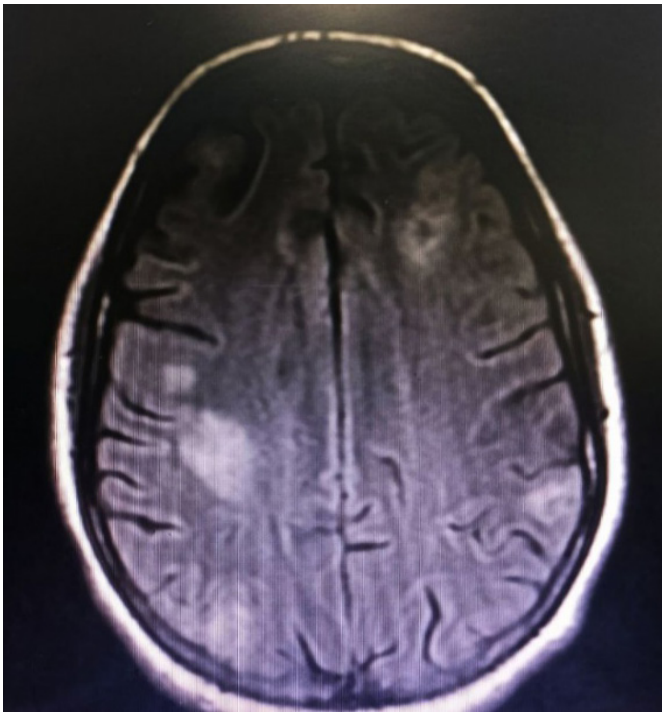

Figure 1. CE-MRI Brain
Multiple ring enhancing lesions

Respiratory - Other

Abstract:0661

ANTIFUNGAL THERAPY IN ICU: WHEN IS THE RIGHT TIME TO START?

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Introduction: Aspergillus species are a major cause of life-threatening infections in immunocompromised patients. Depending on the underlying disease, decreased host resistance, neutropenia, use of corticosteroids and other immunosuppressive agents are facilitating factors for invasive infection. The aim of this case report is to show how the initiation time of antifungal therapy affects the clinical course and mortality of patients with IPA.

Cases: 1) A 61-year-old male patient was admitted to our clinic with complaints of shortness of breath. The patient was receiving methylprednisolone with a diagnosis of secondary ITP. The patient's chest CT showed scattered opacities of various sizes in both lungs, some of which tended to cavitate (Figure 1). The patient with respiratory failure was transferred to our intensive care unit. The patient who had immune suppression and compatible radiological involvement on admission was consulted with infectious diseases for urgent antifungal treatment after respiratory tract cultures were taken. However, the infectious diseases department did not start antifungal treatment. Aspergillus was found in sputum culture on the 3rd day of hospitalization and antifungal treatment was started. The patient, was intubated on the 5th day of hospitalization with clinical deterioration. The patient, did not respond to treatment and died on the 7th day of hospitalization.

2) A 62-year-old male patient was admitted to the hospital with symptoms of fever, cough, and a preliminary diagnosis of ARDS and viral pneumonia. Oxygen support was provided with a reservoir mask in addition to FiO2:100% high flow oxygen support. Despite 5 days of steroid treatment, the patient had a fever above 38 degrees. Due to the progression on the chest X-ray, ampiric piperacillin-tazobactam was discontinued and switched to vancomycin, meropenem, TMP-SMX, colimycin. On the 17th day of hospitalization, patient underwent a thorax CT scan due to skeptical cavitary lesion on the patient's chest X-ray (Figure 2). Patient found compatible with necrotizing pneumonia and antibiotic revision was performed (Tigecycline, ambisome and TMP-SMX). Despite all supportive treatment, the patient was intubated on the 26th day of hospitalization and died on the same day after cardiac arrest.

Conclusion: Since Aspergillus is a fungus capable of rapid proliferation and vascular invasion (highly fatal in immunocompromised hosts), empirical treatment should be started as soon as invasive aspergillosis is suspected without waiting for the results of definitive diagnostic procedures. As seen in our cases, mortality is inevitable when treatment is delayed, especially in patients who require intensive care management.

Keywords: IPA, ımmunsupresif, early recognition, ICU

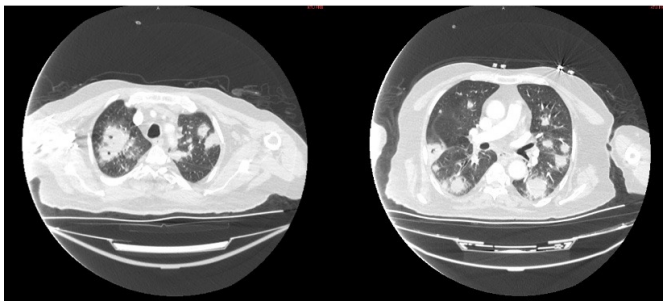

Figure 1. Thorax CT of Patient 1

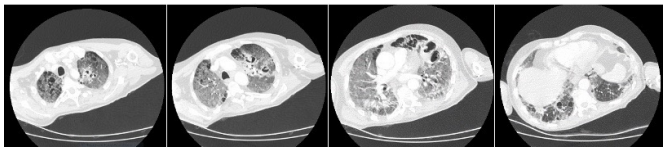

Figure 2. Thorax CT of Patient 2

Critical care pharmacy and drug monitoring

Abstract:0662

SPONTANEOUS PNEUMOTHORAX IN MIXED POISONING PRESENTED AS DAISLEY BARTON SYNDROME

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Introduction: Herbicide has toxicological importance because some of are associated with high mortality rates, due to respiratory failure. OPC and Paraquat self-poisoning is a major clinical and public health problem in low and middle-income countries across much of South Asia. Paraquat was not used as a common suicidal agent previously in Bangladesh. We report a case of 15 years old female admitted to the ER with history of nausea and vomiting after ingestion of unknown substance in suicidal attempt, later identified mixed poisoning with OPC and Paraquat. She was initially asymptomatic, but later developed renal shutdown with lung injuries as well as pneumothorax, referred to as Daisley Barton Syndrome. This case report aims to alert spontaneous pneumothorax in mixed poisoning on uncommon forms of presentation. Pneumothorax in a patient with paraquat poisoning is a less unusual but underdiagnosed findings. It has high index of early mortality.

Case: Patient's attendant complains about nausea followed by vomiting, which was non projectile and contains undigested food materials first then gastric juice later. After a few hours she also complained of urinary retention. Her family members treated her with some home remedies for her initial symptoms, but all attempts were failed. After admission, patient was initially asymptomatic. Through repeated history taking her attendant showed a bottle of OPC in liquid form, which they suspected that she may have ingested some of liquid from that bottle accidentally or attempted of suicide. Management started for OPC poisoning. She responded well initially but 4th day of admission patient's condition deteriorated. After the workout with family member 2nd bottle of pesticide was discovered which was paraquat.

Conclusion: Physicians should be aware of the symptoms of mixed poisoning and the timely use of urine dithionate testing for early detection and treatment. Pneumothorax is an early predictor of mortality in patients with paraquat poisoning.

Keywords: Pneumothorax, Dithionate; OPC; Herbicide, Suicide

Respiratory - ARDS and acute respiratory failure

Abstract:0663

PRONNING IN POLYTRAUMA INDUCED ARDS WITH ITS CONCERNS AND OUTCOME- A CASE SERIES

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Introduction: Pronning is a proven therapy for severe Acute Respiratory Distress Syndrome (ARDS) as concurred by the PROSEVA trial showing definite mortality benefit. In ARDS patients, the change from supine to prone position generates a more even distribution of the gas–tissue ratios along the dependent–nondependent axis and a more homogeneous distribution of lung stress and strain (1). The change to the prone position is generally accompanied by a marked improvement in arterial blood gases, which is mainly due to better overall ventilation/perfusion matching. Improvement in oxygenation and reduction in mortality are the main reasons to implement prone position in patients with ARDS. The main reason explaining a decreased mortality is less overdistension in non-dependent lung regions and less cyclical opening and closing in dependent lung regions. ARDS is a common complication in polytrauma victims, particularly those with chest injuries, and a major cause of mortality and morbidity (2). However, in polytrauma patients, positioning poses a trial for fear of dislocations or destabilization.

We present a case series where three male patients of polytrauma with lung contusion induced ARDS were undertaken for pronning using appropriate precautions to prevent destabilization. Proper positioning plays a pivotal role here. The first male had a stabilized acetabular fracture along with flail chest and pulmonary contusions. The second male had multiple rib fractures with humerus and femur fracture, while the third was a young male with bilateral hemopneumothorax with multiple rib fractures and L1L3 fracture with clavicular fracture with pulmonary contusion and trauma induced ARDS. There was uniform improvement in all three cases with improved oxygenation parameters and gradual weaning from ventilator without any

pronning related complications like arrythmia, elevated ICP, pressure sores. The first patient died of intestinal perforation one month after trauma while the second succumbed to secondary blood stream infection but the third patient survived and was discharged after decannulation of the tracheostomy tube.

Conclusions: Although unconventional pronning with adequate precautions can be beneficial in polytrauma patients with ARDS.

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Keywords: ARDS, Proning, Polytrauma

Metabolism - Endocrinology

Abstract:0672

HYPEROSMOLAR HYPERGLYCEMIC STATE COMPLICATED BY AORTIC THROMBUS: CASE REPORT

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Introduction: Hyperosmolar hyperglycemic state is an emergency with a poor prognosis. Significant dehydration (8-10 liters), electrolyte loss and hypotension, focal neurological signs are present. Here, an example of a case resulting with serious morbidity will be presented.

Case: A 46-year-old female patient with a known diagnosis of Type 2 diabetes mellitus and multinodular goiter, who did not use oral antidiabetics regularly, applied to the emergency department with epigastric pain, nausea, vomiting, fainting, and chest pain. It was observed that the patient was severely dehydrated on arrival. Vital signs were BP: 90/60 mmHg, pulse: 80/min, fever: 36.8 °C, respiratory rate: 25/min, O2 saturation: 99. High blood glucose levels (691mg/dl) and metabolic acidosis were detected. Insulin infusion (12 units/h) and iv fluid replacement (200 cc/h) were started. The laboratory parameters of the patient are given in Table 1. ST elevation was found in v1-v2 on her ECG and troponin level was elevated. The patient was consulted with the cardiology clinic. The dysarthric patient who was conscious and had difficulty in forming sentences, was consulted to neurology. Motor strength was 4/5 in the left upper extremity. No acute pathology was detected in cranial CT. During follow-up, urine output was absent despite a total of 10 liters of iv fluid replacement and the acidosis was deepened. Patient's nausea and epigastric pain continued. High blood glucose levels were persistent although acidosis was improved. The patient's troponin level elevation was continued, in echocardiography right chambers were dilated, Mac Cunnel sign was positive, medium tricuspit regurgitation, pap 40, pulmonary artery dilated 35 mm, EF 65%. The patient's creatinine level increased up to 1.4 mg/dl and hemodiafiltration treatment was started in hemodynamically unstable patients and ultrafiltration was performed for 5 days. After starting the noradrenaline treatment short-term ventricular fibrillation developed but was discontinued. In diffusion MRI, ischemia in the acute-subacute process was detected on centrum semiovale, right frontal precentral gyrus and parietal deep white matter. Acetylsalicylic acid 300, clopidogrel 75 mg, and enoxaparin 2x0.6 treatment were administered. Instead of enoxaparin and ASA, apixaban started because of heparin-induced thrombocytopenia.

Conclusion: It should be kept in mind that hyperosmolar hyperglycemic state can be fatal with cardiac and neurological complications.

Keywords: Acute cerebrovascular disease, hyperosmolar hyperglycemic state, ischemic hepatitis, aortic thrombus

Table 1. Laboratory parameters of the patient

Parameter	At the Arrival	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
pH	7.17	7,26	7,21	7,40	7,45			7,44	
pCO2(mmHg)	38	26	32	37	45			39	
HCO3(mmol/l)	13	11,6	12	22	31			26	
O2 Sat(%)	62	89	32	42	33			64	
Lactate(mmol/l)	8,3	6,7	5	3,5	2,6			2,5	
WBC(103/ml)	11,640	17,500	26,160	15,460	12,040	10,510	10,850	12,690	
NEU(103 ml)	8,010	15,290	21,220	11,800	8,480	6,930	7,350	9,400	
HB(gr/dl)	8,6	8,5	8,8	7,7	7,8	8,1	7,6	7,6	
PLT	277	248	114	85	67	73	100	111	
INR	1,18		1,61	1,86	1,31		1,25		
APTT(second)	23		32	37	31		32		
BUN(mg/dl)	12	19	34	17	18			8,2	
Creatinine(mg/dl)	0,7	0.8	1.48	1,15	0,74			0,7	
Glucose (mg/dl)	691	364	324	122	149	139			
AST(U/L)	38	899	8412	3503	1902	951	427	235	107
ALT(U/L)	300	761	5153	4299	3577	2233	1407	1198	591
CRP(mg/L)	0,44	12	32			31			
Troponin T (pg/ml))	316	679	2478	1953	884	721	418	441	360

Urinary pH was 6.0, glucose 3+, ketone negative, density was 1033. Hba1c was 13.1. (Ferritin: 24 ml/ng Iron: 23 µmol/l TIBC: 470) Iron deficiency anemia is detected. Ammonia level: 29 µmol/l. Thyroid function tests and hepatitis markers are normal range.

Respiratory - Other

Abstract:0673

A CASE OF MICROSCOPIC POLYANGIITIS

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Introduction: Microscopic polyangiitis (MPA) is a granulomatous, non-inflammatory vasculitis that involves small vessels. In MPA, 80% of patients are positive for Perinuclear Antineutrophilic Cytoplasmic Antibody (p-ANCA) and 40% for Cytoplasmic Antineutrophilic Cytoplasmic Antibody (c-ANCA). MPA is frequently associated with kidney and lung involvement. Pulmonary involvement which manifests as alveolar hemorrhage is observed in 30-40% of patients with MPA and is one of the causes of increased mortality (1). The first sign of renal involvement in MPA is microscopic hematuria and proteinuria. In this manuscript, we reviewed MPA features in a patient with reno-pulmonary syndrome presenting with progressive cough, dyspnea and renal failure.

Case: A 75-year-old woman was admitted to the emergency department with a cough, blood coming from her mouth and respiratory distress. The patient was admitted to the intensive care unit with the diagnosis of acute renal and respiratory failure. Her medical history revealed a diagnosis of chronic kidney disease (CKD). Chest X-ray

(CXR) showed bilateral patchy non-homogeneous density increase. Thorax computed tomography showed scattered areas of consolidation in both lungs and reported as the findings could be due to significant for alveolar hemorrhage, pulmonary edema or pneumonia. On physical examination, the patient's breathing was spontaneous, respiratory rate was 30/min. Laboratory examinations were as hematocrit: 25%, sedimentation: 128 mm/h, C-reactive protein: 103mg/L, creatinine: 7.35mg/dl. Complete urinalysis revealed +3 hemoglobin, 108 erythrocytes was seen in urine sediment. In immunological tests Anti-nuclear Antibody, Anti-smooth muscle antibody, c-ANCA was negative and p-ANCA positive in the patient who was considered to have an immune system disease with reno-pulmonary etiology (Table1). The patient was diagnosed as MPA with p-ANCA positivity as no sinusitis or nasal polyp pathologies of the upper respiratory tract were detected clinically. The patient with diffuse alveolar hemorrhage and MPA was treated with 1 g/day corticosteroid for 3 days followed by 1 mg/kg corticosteroid. Pneumonia could not be excluded and broad spectrum antibiotics were started. Hemodialysis and plasmapheresis were performed for 10 days. Non-invasive mechanical ventilation and high-flow oxygen therapy were applied. On the 12th day of intensive care treatment, CXR was improved (Figure1). The patient was transferred to the ward.

Conclusion: MPA is one of the most common causes of reno-pulmonary syndrome. When investigating the etiologies of CKD, hemoptysis and alveolar hemorrhage, the patient should also be evaluated for vasculitis and MPA by excluding frequently observed diseases.

References

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Keywords: Diffuse alveolar hemorrhage, Renopulmonary syndrome, Microscopic polyangiitis

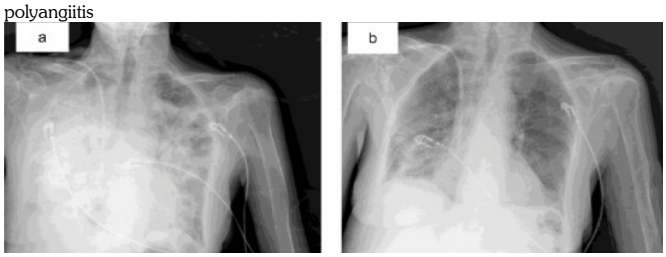

Figure 1. Chest-X ray of the patient before (a) and after (b) treatment

Table 1. Laboratory parameters

	Addmission	Values
Hemogram		
Hemoglobin	7,5 gr/dl	4-15 gr/dl
Hematocrit	%25	%38–42
Biochemistry		
Blood urea nitrogen	220 mg/dl	5–23 mg/dl
Creatinine	7,5 mg/dl	0.51–0.95 mg/dl
IgG	18,5 mg/l	7-16 g/l
gM	0,7 mg/l	0,4- 2,3 g/l
IgA	2,6 mg/l	0,7-4 g/l
Complete urinalysis		
Density	1013	1005–1030
Ph	5	4,8–7,4
Protein	+2	
Hemoglobin	+3	
Erythrocyte	108	0-5
Serology		
C-Reaktif Protein	103 mg/dl	0-5 mg/l
C3	1.25 gr/l	0,9–1,8 gr/l
C4	0.29 gr/l	0,1–0,4 gr/l
Sedimentation rate	128 mm/saat	6-22 mm/saat
Elisa		
Anti HIV	Negative	
Anti HCV	Negative	
Immunulogy		
ANCA (P - ANCA)	Positive	
ANCA (C-ANCA)	Negative	
Anti Kidney Mikrozomal Antikor	Negative	
Anti smooth muscle Antibody	Negative	
Anti Kidney Mikrozomal Antikor	Negative	

Perioperative critical care

Abstract:0677

PREOPERATIVE OPTIMIZATION FOR SURGERY IN PATIENT WITH THYROTOXICOSIS

Alekandra Jankovska

Introduction: Thyrotoxicosis is a clinical condition characterized by excessive thyroid hormone activity, caused by excessively high thyroid hormone levels in the bloodstream. Palpitations, tachycardia, tremor, anxiety, weight loss, sweating, polydipsia are some of the symptoms. Thyrotoxicosis affects about 2% of women and 0.2% of men.

Case: We present a 20-year-old female patient with thyrotoxicosis for thyroidectomy, who complains of difficulty in breathing and swallowing. Thiamazole 2x20 mg and Propranolol 40mg 2x1/2 are given to treat the patient. In the OR she had the following vital parameters:

TA 170/90; HR 140 / min; SaO2 98%. Laboratory tests: TSH 0.022 mIU / L; T3 14, 4 pmol / L; T4 38.4 pmol / L. Esmolol, fentanyl and midazolam were administrated intravenous, but the tachycardia still persisted. The intervention was postponed and the patient was referred to the Institute of Pathophysiology for further treatment. After 2 months, with adjusted therapy, the patient was re-admitted to OR with the following vital parameters: TA 140/80; HR 115 / min; SaO2 98%. Laboratory tests: TSH 0.206 mIU / L; FT4 28.18 pmol / L. Induction in general anesthesia was performed with midazolam, lidocaine, fentanyl, propofol, rocuronium, continued analgesia with remifentanil, and continued sedation with propofol. The patient was intubated with ETT 7.0 with very careful manipulation. During the intervention, Cefazol, Dexamethasone, Pantoprazole, Chloropyramine, Ca gluconate, Methylprednisolone were given. Throughout the intervention, the patient maintained stable vital parameters with TA around 120/80 and HR around 80 / min.

Conclusion: Thyroid disease is very frequent. Preoperative preparation and optimization of thyroid physiological status might reduce intraoperative morbidity. With careful manipulation and good preoperative assessment, we managed to avoid a thyroid storm.

Keywords: Thyrotoxicosis, Preoperative, Optimization

Neuro-intensive care

Abstract:0679

WEST NILE ENCEPHALITIS COMPLICATED BY SEVERE DEMENTIA AND CO-INFECTION WITH COVID-19

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Introduction: Since December 2019, COVID-19 is an important viral infection that has affected all over the world. West Nile virus is an RNA virus belonging to the family of Flaviviridae that is transmitted to humans by Culex mosquitoes and causes involvement of the central nervous system in the form of encephalitis, meningitis, or acute flaccid paralysis. In this paper, we reported a patient with West Nile encephalitis who developed impaired consciousness on the fifth day of COVID-19 infection.

Case: In July 2021, a 40-year-old male patient working in Tanzania was taken to the clinic with fever, cough, and headache. He had been found to be COVID-PCR positive and isolated, and treatment was initiated. The patient developed impaired consciousness and speech impairment on the fifth day of COVID-19 infection and was admitted to the intensive care unit with the diagnosis of encephalitis. On the 12th day of the disease, the patient was brought to Turkey by his relatives and admitted to our neuro-intensive care unit. On neurological examination, he was confused and apathetic, speech was dysarthric and incomprehensible, and there were no lateralizing deficits. EEG demonstrated delayed activity asymmetry in the left frontotemporal area, while MRI revealed bilateral temporoparietal hyperintense lesions, bigger on the left side. Cerebrospinal fluid testing indicated West Nile virus Ig M positivity. The patient regained consciousness on the 20th day of hospitalization after receiving supportive care, and his speech was partially understandable. The patient is still being followed up on in the outpatient clinic, despite having severe dementia and being able to mobilize without assistance.

Conclusion: Although West Nile Encephalitis is a rare entity, it should be considered in the differential diagnosis in cases with atypical clinical and radiologic course.

Keywords: COVID-19, West Nile Virus, Encephalitis, Dementia

Respiratory - Other

Abstract:0690

HIT PROGRESS IN THE PATIENT WITH MASSIVE PULMONER EMBOLI

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Introduction: Heparin-induced thrombocytopenia (HIT) is an important life-threatening complication of heparin exposure. Patients have thrombocytopenia and thrombocytopenia is often accompanied by thrombosis. Since it is associated with high mortality, the diagnosis of HIT should be kept in mind and after the diagnosis, all heparin derivatives administered to the patient should be stopped and alternative anticoagulation drugs and/or surgical methods should be applied.

Case: A 54-year-old female patient with no systemic disease or regular drug use in her history passed out in the bus terminal. When it was noticed that there was no pulse, she was brought to the emergency room by getting cardiopulmonary resuscitation (CPR) for about 10 minutes. During the thorax tomography angiography, massive thrombus was detected in both pulmonary arteries and after 50 mg of alteplase was given to the patient, she was followed up in the intensive care unit. Enoxaparin of 2x60mg was started to be given at the 12th hour of her hospitalization. The patient extubated in her 24th hour. On the 7th day of her hospitalization, she developed cardiac arrest again and an additional dose of 50 mg alteplase was administered to the patient in whom recurrent pulmonary embolism was detected. In her examination after 24 hours, ecchymotic lesions were detected in the right hand dorsum and left inguinal region. In addition, there was an increase in diameter in the right lower extremity of the patient compared to the left. Blood tests showed acute renal failure (GFR <30) and thrombocytopenia (platelet <50,000 UI). A thrombus was detected in the right popliteal vein in Doppler Ultrasound. His HIT score was calculated as 6, and the enoxaparin she was taking was stopped immediately. 'Bivaluridine' treatment was recommended by the Hematology department for the patient who could not be given fondaparinux due to his GFR <30. After appropriate hydration, when GFR >30, the patient was started fondaparinux of 7.5 mg. The patient, who was extubated again 72 hours later and her platelet count improved, was transferred to the ward on the 16th day of her hospitalization.

Conclusion: HIT syndrome, which is often associated with unfractionated heparin and can progress with fatal complications when it is not considered, can develop with enoxaparin, as in our case, and to transfer our experience of alternative treatment methods, the patient we followed in the intensive care unit was deemed worthy of presentation.

Keywords: massive pulmonary embolism, heparin-induced thrombocytopenia, fondaparinux

Neuro-intensive care

Abstract:0692

TREATING CENTRAL FEVER WITH CYTOKINE REMOVAL THERAPY: A CHALLENGING CLINICAL ENTITY

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Introduction: Disruption of mesencephalic mechanisms, production of cytokines IL-1, IL-6 and TNF-α by monocytes and macrophages, release of prostaglandin E2 via activation of COX2 enzyme, systemic pyrogens such as IL-1 underlie the pathophysiology of central fever. The clinical implications of using cytokine adsorption as a method to reduce cytokine levels in fever thought to be of central origin and resistant to appropriate antibiotherapy and supportive care were discussed in 3 cases.

Case:A 47-year-old male patient was admitted to our intensive care unit after in-vehicle traffic accident. He intubated after accident with many bone fractures, and subarachnoid hemorrhage. High fever with no response to medical treatment was attributed to multiple trauma-related cytokine release. The patient with high fever and hemodynamic instability was treated with cytokine removal therapy with Cytosorb.

A 36-year-old male patient brought to the emergency department after fainting, GCS dropped to 8 and he was intubated. He had a hematoma in the left periventricular region, which was operated after 6 hours due to enlargement of the hematoma. He had high grade central fever, and hypotensive progression requiring inotropic support in intensive care unit. Clinical improvement was achieved with cytokine removal therapy with Cytosorb due to central fever refractory to medical treatment.

A 49-year-old female patient whose cardiac rhythm stopped after chest pain was intubated in the ambulance. She was diagnosed with NSTMI and stents were placed. In the intensive care unit she had high grade central fever and increasing need for inotropes. Clinical improvement was achieved with cytokine removal therapy with Cytosorb due to central fever resistant to medical treatment. In the table, important parameters of the cases are presented before and after the procedure.

Conclusion: Inflammatory markers causing fever may be triggered by extreme physiologic stress in acute neurologic injury. Monocytes and macrophages produce the cytokines IL-1, IL-6, and TNF-α, which act on the organum vasculosum of laminae terminalis. Systemic pyrogens, such as IL-1, appear to enter the brain at regions where there is an incomplete blood-brain barrier (circumventricular organs) and act on the preoptic area of the hypothalamus to induce fever. With these theories, cytokine removal therapies may be an effective way to control central fever. As a matter of fact, decrease in fever, mean arterial pressure and vasopressor needs were observed after cytokine removal therapies were applied in these 3 patients. Cytokine removal should be kept in mind as a treatment option for uncontrolled fever that may have catastrophic consequences in intensive care unit.

Keywords: Cytokine Removal Therapy, Treating Central Fever, Critical Care, Cytosorb

Clinical and laboray values of patients before and after cytokine adsorbtion therapy

Table X. Clinical and laboray values of patients before and after cytokine adsorbtion therapy*

	before adsorbtion therapy					after adsorbtion therapy				
	CASE 1.1	CASE 1.2	CASE 2	CASE 3	mean	CASE 1.1	CASE 1.2	CASE 2	CASE 3	mean
pH	7,492	7,545	7,320	7,453	*7,4525*	7,545	7,476	7,400	7,489	*7,478*
pCO_2 (mmHg)	37,7	32,7	34,3	35,2	*35,0*	32,7	34,8	35,2	41	*35,9*
pO_2 (mmHg)	79	86,8	95,1	90,8	*87,9*	96,8	92,3	98,9	94,2	*95,6*
HCO_3 (mmol/L)	29	28	24	27,7	*27,2*	28	25,2	25	29,8	*27,0*
Lactate (mmol/L)	1	1,2	1,2	2,2	*1,4*	1,2	1	1,9	1,2	*1,3*
Temperature (°C)	38,2	38,8	38,9	38,8	*38,7*	38,8	37,7	37,5	37,6	*37,9*
MAP (mmHg)	75	85	75	55	*72,5*	85	110	85	100	*95*
Systolic pressure (mmHg)	100	110	100	70	*95*	110	120	110	130	*117,5*
Heart beat (beat/min)	90	90	100	120	*100*	90	90	95	100	*94*
Noradrenaline dose (mcg/kg/min)	0,35	0,4	0,2	0,1	*0,26*	0,4	0,25	0,15	0	*0,20*

*the worst values were noted 12 hours period before or after the cytokine ads. therapy
MAP donates mean arterial pressure

Neuro-intensive care

Abstract:0696

ACUTE BULBAR PALSY WITH RARE GBS VARIANT

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Introduction: The Guillain-Barre Syndrome (GBS) is the most frequent reason of acute flaccid paralysis among adults today. There are rare variants of this syndrome that do not cause motor loss in extremities, the most important of which is symmetrical flaccid paralysis. The young patient with a rare variant of GBS is considered noteworthy.

Case: The 28-year-old male patient with no pathology all his life but allergic asthma started to have complaints about shifting his mouth to the right and not being able to close the right eyelid. In 24 hours, the same complaints occurred halfway on the other side, too. When in hours, not being able to swallow, having difficulty in speaking and laboured breathing added to his complaints, the patient applied to neurology polyclinic. He had bilateral peripheric facial paralysis, hypophonia, vomiting and loss in swallowing reflex in his first neurologic examination. The deep tendon reflexes couldn't be taken globally. In a short time, his breathing problem got worse and the patient was electively intubated and taken into intensive care unit. The patient with a positive plasma VDRL test had normal blood examinations except high CRP levels (186 mg/dl). The patient with negative FTA-ABS, had long peroneal F wave responses on both sides in nerve conduction/neurotransmission studies. Elongation/prolongation of tibial, peroneal and ulnar nerve distal latency and a decrease in motor transmission speed were present. Both amplitudes of ulnar nerve DSAP decreased. Albuminostic disassociation was present in the BOS sample and BOS VDRL was negative. The patient was considered as acute bulbar palsy plus variant GBS and was given IVIG of 0,4gr/kg for 5 days. The patient was given penicillin for 14 days suggesting that he might get infectious diseases. Afterwards, he did not develop any new symptoms. The patient who had tracheostomy on the 10th day of his hospitalization was disconnected on the 22nd day and taken to physical treatment hospital.

Conclusion: The patient arriving at the hospital with acute bulbar involvement and no extremity weakness who had atypic beginning and prognosis for classical GBS is considered to be worth mentioning in order to draw attention to rare GBS forms.

Keywords: Guillain-Barré Syndrome, bulbar palsy, plasmapheresis

Cardiovascular - Other

Abstract:0697

POST-TRAUMATIC MITRAL CHORDAE RUPTURE MIMICKING ACUTE RESPIRATORY DISTRESS SYNDROME

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Case: A 19-year-old male patient applied to the emergency department with a complaint of a plank hitting his sternum at work. The stable patient, whose thorax computed tomography images were normal at the first admission, reapplied 24 hours later with increasing dyspnea and hemoptysis. His new thorax computed tomography showed bilateral patchy pneumonic consolidation and ground glass areas, and he was intubated due to oxygen unresponsiveness. The patient with a negative respiratory viral panel was admitted to intensive care unit with a preliminary diagnosis of acute respiratory distress syndrome (ARDS), pulmonary contusion and alveolar hemorrhage. Echocardiography was performed immediately after the patient's admission for the differential diagnosis. Mitral chordae rupture was detected and the patient was taken to emergency valve surgery in hours. The patient, who was clinically compatible with acute respiratory distress syndrome, was discharged from the hospital on the 7th day after emergency valve surgery with completely normal chest X-ray and recovery. Traumatic chordae rupture, which is very rare, is a pathology that can be completely healed by emergency surgery. The radiological examinations are non-typical for pulmonary edema is probably due to pulmonary trauma has altered the classic presentation of cardiogenic pulmonary edema for thorax computed tomography.

Signs and symptoms of pulmonary contusion may continue to worsen up to 48 hours after trauma, but evolution of the patient without a rib fracture to an ARDS clinic is cautionary in the case. In chest trauma, chordae rupture should always be considered in the differential diagnosis as a treatable cause of respiratory failure. Acute severe valvular regurgitation will be more fatal if left unoperated than ARDS associated with lung contusion.

Keywords: Traumatic chordae rupture, adult respiratory distress syndrome, acute mitral valve failure

Cardiovascular - Other

Abstract:0699

SUCCESSFUL TREATMENT OF PERICARDIAL TAMPONADE DUE TO ESOPHAGEAL RUPTURE

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Introduction: Purulent pericarditis is a rare type of bacterial pericarditis with a high risk of mortality and morbidity. In untreated patients, mortality rate may reach to 85%. In between 42%-77% of the cases with purulent pericarditis, cardiac tamponade develops, leading to rapid clinical deterioration via septic shock.

Case: A 25-year-old man with no significant medical history presented to emergency department with deteriorated general state, epigastric pain, shortness of breath, and syncope. He had sought medical care with a complaint of cough 10 days ago and was given treatment for pneumonia. Contrasted thorax computed tomography (CT) showed esophageal leakage to mediasten at the level of T4. Gastric endoscopy showed erythematosus in the same area. The patient had a Glasgow coma scale 9/15 (E2V2M5) and was admitted to the Intensive Care Unit (ICU) with a preliminary diagnosis of mediastinitis due to esophageal rupture and was intubated. The patient was in deep shock state and had high central venous pressure (CVP). For differential diagnoses, transthoracic echocardiography was performed which showed presence of pericardial tamponade. The tamponade could not be drained via pericardiocentesis, and a pericardial window was opened with thoracotomy. Through pericardial window, two liters of purulent fluid was drained. In the fluid culture anaerobic bacteria was grown. The patient was given broad spectrum antibiotics and continuous hemadsorbtion with Oxiris filter was carried out for three days. During monitoring, the patient developed multiple organ failure and continuous venous-venous hemodialysis (CVVHD) was initiated. Once the patient's condition stabilized, a control gastroscopy was performed showing that the esophageal rupture has been repaired. The patient, whose breathing pattern and hemodynamics were stabilased, and was weaned on the 19th day of admission. Three weeks after weaning, he had spontaneous breathing in open air, was conscious and hemodynamically stable and was discharged from the intensive care unit.

Conclusion: Pericarditis is a life-threatening complication of esophageal rupture with a reported incidence of approximately 13%. We highlight our successful management and treatment of a patient with purulent pericarditis and cardiac tamponade with drainage of around two liters of fluid and broad-spectrum antibiotics. The follow-up examination showed complete resolution of the condition. Due to the need to make a prompt diagnosis and early treatment, clinicians should consider cardiac tamponade among the severe complications of esophageal rupture in the presence of variable and vague symptoms such as shortness of breath, cough, and epigastric pain.

Keywords: esophageal rupture, pericardial tamponade, pericardiocentesis, mediastinitis

Sepsis - Other

Abstract:0706

SEPSIS BY EXTENSIVELY RESISTANT SALMONELLA TYPHI IN ANGOLA: CASE REPORT

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Introduction: Infections caused by multidrug-resistant strains represent a growing public health threat worldwide. In particular, recent decades there has been an increase in isolates of *Salmonella typhi* strains resistant to quinolones and more recently to third-generation cephalosporins (extensively resistant) (1,2).

Case: A 30-year-old female presented with a 5 day history of fever, associated with abdominal pain, vomiting and diarrhea without blood. She self-medicated with amoxicillin/clavulanic acid, without improvement. At admission she was dehydrated, sub-icteric, with painful hepatomegaly 2 cm below the costal margin, without peritoneal reaction. Admission blood tests showed anemia with hemoglobin 9.4g/dL, thrombocytopenia of 113×109/L, white cell count 4.8x109/uL, C-reactive protein 21.3 mg/dL; hyperbilirubinemia (3.4 mg/dL), elevated transaminases three times above the upper limit; negative malaria serology and thick blood film for *Plasmodium falciparum* and serology for dengue, human immunodeficiency virus and liver virus negative. Chest radiography showed a mild bilateral interstitial infiltrate in basal region, for which reason empirical ceftriaxone was started after collection of blood and urine cultures. On the 3rd day the patient worsened clinically and analytically with increased abdominal pain and hypoxemia, without neurological and hemodynamic dysfunction. Thoracic and abdominal tomography showed bilateral homogeneous consolidation of the lower lobes with air bronchogram, small/medium volume bilateral pleural effusion and homogeneous hepatomegaly. She was admitted to the intensive care, the blood culture was positive for *Salmonella typhi*, resistant to chloramphenicol, ampicillin, trimethoprim-sulfamethoxazole, quinolones, third generation cephalosporins and sensitive to cefepime and aztreonam. Antibiotic therapy with cefepime was started, and she was discharged on the 15th day.

Conclusion: Sepsis caused by multidrug-resistant agents represents a huge therapeutic challenge, as limited therapeutic options are more expensive and less accessible in countries with limited resources. The identification of *Salmonella typhi* extensively resistant in Angola, joins the recent cases identified in the world, in endemic countries such as Pakistan and non-endemic countries such as Canada, justifying the need to carry out epidemiological studies and profiles of antimicrobial resistance in African countries.

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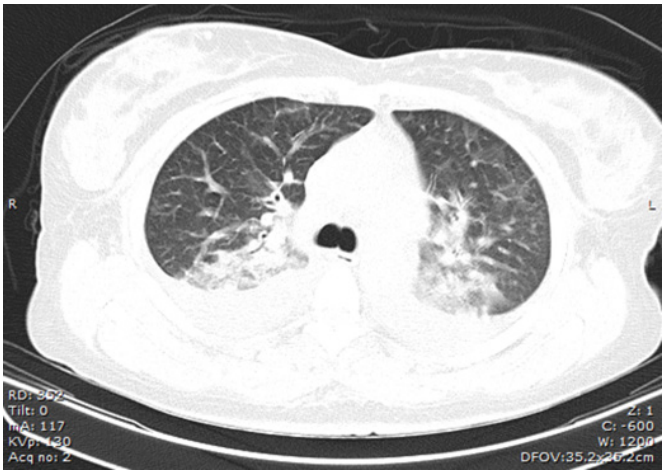
Keywords: Salmonella typhi, Drug-resistant, Sepsis

Angio Thoracic CT



Figur 1. Shows consolidation with air bronchogram and bilateral pleural effusion.

Metabolism - Other

Abstract:0708

DIVING-INDUCED SERIOUS AIR EMBOLISM: CASE REPORT

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Introduction: The most common diseases associated with diving are air embolism and decompression sickness. In this case report, a case with serious air embolism due to diving will be presented.

Case: A 54-year-old female patient is brought to our emergency department because of respiratory and cardiac arrest at home. Except for known hypertension and insulin-independent diabetes, the patient, who had a history of sleeve gastrectomy surgery, revealed that he had been active diving for 3 days, 1 week ago, when his anamnesis was deepened. It was determined that the patient, who had nausea and vomiting after the last dive, traveled with a long-term land trip the next day while his symptoms continued, and his symptoms gradually worsened after the trip. The patient is brought to our center after muscle fatigue, myalgia, cessation of oral intake, and arrest at home 4 days after diving. Patient was transferred to the intensive care unit. GCS determined E1M1Vt. High-dose inotropic was given. In laboratory analysis; BUN: 89, Creatine: 10.11, Na: 136, K: 7.86, Cl: 92, glucose: 267, pH: 6.78, pCO2: 27.9, pO2: 65,

lactate: 9.7, HCO3: 4.6, CK: 241, SpO2:75 determined. Necessary replacements were made. Antibiotherapy was started. Neurology was consulted for the patient who had uninterrupted seizures in the form of convulsions. Antiepileptic treatments were started. Acidosis in arterial blood gas was broken and reached normal levels in follow-up. After dialysis treatment, creatinine regressed and urination started. The need for inotropic therapy decreased, but on the 4th day of intensive care treatment, brain death was diagnosed by neurological examination. Apnea test was planned to confirm, but oxygen saturation value decreased rapidly and patient was accepted as exitus for donation before the apnea test could be completed. Consent was obtained from the relatives of the patient for the case report.

Conclusion: Arterial gas embolism is rare at altitude and is not related to depth-time exposure in diving. Bubbles can have mechanical, embolic, and biochemical effects with manifestations ranging from trivial to fatal. Clinical manifestations can be caused by direct effects from extravascular bubbles such as mechanical distortion of tissues causing pain, or vascular obstruction causing stroke-like signs and symptoms. Hypotension can occur in severe cases. While no triggering cause is sought for decompression sickness, triggering factors such as obesity, long travel, and dehydration are sought, especially for arterial air embolism. Late admission to the hospital and progressive organ dysfunction resulted in death due to that he and his relatives did not have enough information about such diving-related conditions. Divers should be aware of this. Decompression and air embolism should be considered in cases with a history of diving.

Keywords: Diving, Induced, Air, Embolism

Respiratory - Other

Abstract:0710

CRITICAL CARE MANAGEMENT OF VIRAL ENCEPHALITIS: 2 CASE REPORTS

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Introduction: Viral encephalitis, a condition characterized by brain inflammation caused by viral infections, presents significant challenges in critical care management. Its rapid progression and high morbidity and mortality rates demand careful monitoring and intervention. This article presents two case reports with viral encephalitis.

The aim is to provide insights into critical care management strategies and highlight key considerations in viral encephalitis.

Case:1) The first patient was a 26-year-old male who developed encephalitis associated with Epstein-Barr virus (EBV) infection. The patient presented with symptoms of respiratory distress, altered consciousness, and sore throat. Analysis of cerebrospinal fluid revealed increased levels of albumin and protein, along with decreased levels of sodium and chloride. The treatment included administration of clindamycin, acyclovir, antiepileptic drugs, and steroids.

2) The second patient was a 39-year-old female who experienced encephalitis due to influenza A virus infection. The patient presented with respiratory symptoms, myalgia, altered consciousness, dizziness, nausea, and vomiting. Cerebrospinal fluid analysis showed elevated protein levels and increased albumin. The treatment involved intravenous antibiotics and antiviral medications. Broad-spectrum antibiotics like ceftriaxone, acyclovir, and vancomycin were used considering the potential causative agents of the infection.

All patients initially presented with common symptoms such as altered consciousness, seizures, and fever. Standard intensive care methods, including respiratory support, intravenous antiviral treatment, fluid support, and seizure control, were implemented during the treatment process.

Conclusions: Viral encephalitis requires a comprehensive approach that includes early diagnosis, prompt initiation of antiviral therapy, and diligent critical care management. This case series underscores the significance of close neurological monitoring, appropriate imaging, and cerebrospinal fluid analysis in guiding treatment decisions. Furthermore, optimizing supportive care measures such as airway management, hemodynamic stability, and seizure control plays a crucial role in achieving favorable outcomes.

Keywords: viral encephalitis, intensive care, critical care, antiviral treatment

Neuro-intensive care

Abstract:0712

IATROGENIC BOTULISM AFTER BOTULINUM TOXIN INJECTION FOR HYPERHIDROSIS

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Introduction: Botulinum toxin-A (BTA) has been used for therapeutic purposes since 1980. Today, botulinum toxin injection is the most common procedure performed worldwide, with an estimated 3 million injections per year. It is frequently used in a wide area such as spasticity, chronic migraine, strabismus, hyperhidrosis treatments and for aesthetic purposes. It should be kept in mind that the toxin can spread hematologically from vascular structures and its systemic effects can be life-threatening. In this case report, we discussed a patient who developed iatrogenic botulism after palms BTA treatment for hyperhidrosis.

Case: A 23-year-old female patient, was admitted to the emergency department with complaints of dysphagia, fatigue, blurred vision, nausea and vomiting. She had no known disease, chronic drug use, recent infection history, and no family history of neuromuscular disease. One week before the onset of symptoms, the patient had been injected with BTA (Dysport 750 units; IPSEN) in both palms for hyperhidrosis. After the BTA injection, double vision and ptosis developed on the 4th day, dysarthria and difficulty in holding the neck on the 5th day, and difficulty in swallowing on the 6th day. On the 7th day, when she admitted to the emergency department, there was ptosis in both eyes and weakness was detected in the upper extremity muscles. Trunk and lower limb muscles were clinically preserved. Deep tendon reflexes and sensory examination were found to be normal. Due to the risk of developing respiratory distress, she was admitted to the intensive care unit to be followed closely and her examinations were performed. Routine laboratory examinations including complete blood count, renal and liver function, c-reactive protein and sedimentation rate were all within normal limits. EMG results were evaluated in accordance with the early findings of botulism. Ptosis, upper extremity weakness, dysarthria and dysphagia continued for 4 days following her hospitalization.

Conclusion: As seen in this case, systemic iatrogenic botulism involvement should be considered in botulinum toxin applications regardless of the site and dose relationship. Intravascular injection, deep tissue invasion risks should be considered while applying BTA injection. Before the treatment by BTA, patients should receive a complete information about the potential side effects. To decrease the complications, only justified and trained physician should be allowed to injection.

Keywords: Iatrogenic botulism, botulinum, toxin, injection

Respiratory - ARDS and acute respiratory failure

Abstract:0713

REPEATED BRONCHOSCOPIC THERAPEUTIC LAVAGE FOR PERSISTENT PULMONARY HEMORRHAGE DURING VENOVENOUS EXTRACORPOREAL MEMBRANE OXYGENATION

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Case: A 24-year-old obese patient with autism was admitted to the emergency department due to dyspnea that became symptomatic 1 month ago and was taken to our intensive care unit due to type 2 respiratory failure. The patient, who weighed 214 kilograms, could not undergo computed tomography imaging due to obesity. Ejection fraction was 60% and pulmonary artery pressure was 45mmHg on echocardiography, and no valve pathology was detected. He was intubated 3 days after his hospitalization with insufficient oxygen response and type 2 respiratory failure. After 3 days of intubation, the patient developed low PaO_2/FiO_2 ratio and hypercapnia despite 100% FiO_2. Inhaled nitric oxide, diuretic, antibiotic and steroid treatments were added and a veno-venous extracorporeal membrane oxygenator (vv-ECMO) was started. Although the targeted range of aPTT and ACT levels could not be reached under heparin or bivaluridine infusions, abundant active bleeding was observed from endotracheal tube and all anticoagulation therapy was stopped. The patient was not treated with anticoagulants for 7 days, and daily bronchoscopy was performed to prevent the dysfunction of the airways and lungs with blood plugs. During vv-ECMO treatment, aPTT, INR and ACT levels remained below the targets set for effective anticoagulation, and platelet counts were within normal limits. However, in the patient who developed pulmonary hemorrhage, the basic coagulation parameters were within the normal range, except for the low fibrinogen levels during the hemorrhage. During the 7-day anticoagulation-free period, no deterioration in the functions of the membrane oxygenator was observed, and no thrombotic complications were observed. As a result of 7-day follow-up and repeated bronchoscopies, the continuation of the decrease in the need for ventilator support was achieved. He was successfully weaned from vv-ECMO on the 22nd day of vv-ECMO.

The coagulation system may be incalculably affected as the duration of vv-ECMO is prolonged, and the physician may have to make difficult decisions between the risk of bleeding and thrombosis. For correction of these complications, weaning the patient from vv-ECMO may be the most effective way. Successful weaning from vv-ECMO depends on supporting the recovery processes of the ventilator-dependent lung and ensuring lung protection. Here, it is aimed to emphasize that repeated therapeutic bronchoscopy sessions may be beneficial in massive pulmonary hemorrhage due to anticoagulant therapy. After the case was weaned from vv-ECMO, it was also successfully weaned from the mechanical ventilator.

Keywords: vv-ECMO, pulmonary bleeding, anticoagulation,therapeutic bronchoscopy

Respiratory - Other

Abstract:0714

SPLITTED THROMBOLYTIC THERAPY IN A HIGH-RISK PULMONARY EMBOLISM PATIENT CAN BE AFFECTIVE IN THE CASE OF HEMORRHAGIC COMPLICATION: CASE REPORT

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Introduction: High-risk pulmonary embolism refers to the blockage of more than 50% of the pulmonary arterial tree, leading to a critical condition where the right ventricle is overloaded, resulting in acute and severe cardiopulmonary failure. High-risk pulmonary embolism is treated with different methodologies including systemic thrombolytic treatment. Thrombolytic treatment is usually recommended as 100 mg dose in two hours. Splitted thrombolytic therapy is not specified as a generally used protocol.

Case: We present a case of 73-year-old female patient with massive pulmonary embolism who has PESI score class V. She had congestive heart failure, and a history of bilateral femoral arterial thrombus and iliofemoral endarterectomy after Biontech® vaccine in August 2021. She was operated due to femoral neck vascular insufficiency fracture 2 weeks ago and the patient has used low molecular weight heparin (LMWH) 4000 IU 1*1/day for 2 weeks, and she discontinued for 2 days. She presented to the emergency department with complaints of sudden onset of shortness of breath and stabbing chest pain. She was diagnosed with massive embolism based on thorax computed tomography angiography. As she was not clinically stable, she was intubated in the emergency department and dopamine infusion was started and admitted to pulmonary intensive care unit. She was administered 30 mg r-TPA thrombolytic therapy. Thrombolytic therapy was stopped and heparin infusion was started due to the occurrence of hematochesia during the treatment. On echocardiography, systolic pulmonary artery pressure (sPAP) was 90 mmHg and ejection fraction was 30%. After 30 mg of systemic thrombolytic, sPAP was measured as 95 mmHg. Afterwards, some improvement in right heart functions was observed and total 70 mg more thrombolytic was given on the 6th and 7th day to the patient who had no signs of bleeding. Patient's laboratory findings were seen in Table 1. She was extubated on the 11th day of ICU follow-up. Dopamine infusion was discontinued because of patient's normotensive course. The patient, whose general condition was stable, was transferred to the pulmonary medicine ward.

Conclusion: Thrombolytic therapy is a life-saving treatment option in patients with massive embolism, however this treatment brings complications as well, such as intracranial hemorrhage, gastrointestinal bleeding, anaphylaxis, myocardial rupture as a late complication. In this case report, the patient developed hematochesia, and therefore thrombolytic therapy was discontinued. In her follow-up thrombolytic treatment was started and 100 mg dose completed according to guidelines and clinical improvement was achieved.

Keywords: Pulmonary embolism, thrombolytic, thrombosis

Table 1. Patient's laboratory findings

Parameters	10.May	11.May	12.May	13.May	14.May	15.May	16.May	17.May	18.May	19.May	20.May	21.May
Arterial Blood gase												
PH	7,24	7,37	7,31	7,38	7,42	7,45	7,39	7,43	7,43	7,48	7,53	7,37
Pc02 mmHg	37	32	46	41,8	36,5	33,2	30,5	37,5	38,5	33,5	33	47
P02 mmHg	70	60,4	70	68,8	63,5	84,3	76,1	74,2	78,6	73,8	63	68,2
S02 %	86.9	91,2	89,7	92,1	90,6	96,6	94,1	95,4	95	92,6	92,2	93,5
Hc03 mmol/L	15,50	18,10	22,80	24,30	23,20	22,60	18,40	24,40	25,40	24,50	27,90	27,60
Lac	1,90	1,32	1,73	1,22	1,47	1,41	1,16	1,43	1,42	1,34	1,24	2,01
Echocardigraphy findings												
Ejection Fraction %	35	35	35		50	55	55	50		55		
Systolic Pulmoner Artery Blood Pressure mmH	90	70	65		65	65	90	65		60		
Fi02 support %	50	40	50	35	40	60	50	40	35	35	35	30
Pa02/Fi02	140	151	140	196,57	158,75	140,5	152,2	185,5	224,57	210,86	202,2	227,33
nT- BNP pg/L	31000	>35000				>35000	33000			17000	3000	
hs- Troponin T ng/L	99	103				164	178			58	52	
Vital signs												
Blood pressure mmHg	108/55	120/75	124/78	119/51	101/52	95/48	108/52	112/62	121/60	115/69	125/71	123/80
Pulse rate /min	62	101	57	94	87	106	93	96	89	91	78	93
Respiratory rate /min	32	18	20	21	22	26	20	20	19	18	20	23
Body tempature °C	36.5	36.1	36.3	36.7	36.2	36.4	36.5	36.6	36.5	36.2	36.3	36.6
Oxygen saturation %	93	96	94	97	95	92	94	93	92	95	96	97