CASE REPORT

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# Beyond the Respiratory Tract: Severe Influenza A-Associated Rhabdomyolysis with Anuric Acute Kidney Injury and Respiratory Failure

Kemal Yetiş Gülsoy,[1] Burcu Ancın[2]

## Abstract

Although rare, influenza A-associated rhabdomyolysis can rapidly progress to multiorgan failure. A previously healthy 22-year-old man returned to the emergency department eight hours after his initial visit with dark urine and rapidly deteriorating clinical status. Influenza A was detected. Creatine kinase peaked at 320,050 U/L, accompanied by severe hyperkalemia, profound mixed acidosis, hypercapnic respiratory failure, hypotension, and anuric acute kidney injury. The patient required invasive mechanical ventilation, vasopressor support, urgent intermittent hemodialysis, and continuous renal replacement therapy. Diuresis resumed during the first week, continuous therapy was discontinued on hospital day 9, and the final hemodialysis session was performed on day 19. The patient recovered without requiring long-term dialysis. Dark urine or disproportionate enzyme elevation during influenza infection should prompt immediate creatine kinase testing and serial monitoring of electrolytes, acid-base status, renal function, and urine output. Timely organ support may enable recovery despite extensive muscle injury.

**Keywords:** Acute kidney injury; Influenza A virus; respiratory failure; rhabdomyolysis.

## Introduction

Influenza predominantly affects the respiratory tract but can cause extrapulmonary complications, including acute myositis and rhabdomyolysis.[1] Skeletal muscle breakdown releases creatine kinase (CK), myoglobin, potassium, phosphate, and other intracellular constituents; severe muscle injury may lead to hyperkalemia, acidemia, arrhythmias, and acute kidney injury (AKI).[2,3] Infections are recognized nontraumatic causes of rhabdomyolysis, with influenza A being among the best-described viral triggers.[1,4,5] Direct viral injury to myocytes and systemic inflammatory effects have been proposed as potential mechanisms; influenza A(H1N1) strains can infect and lyse differentiated human myotubes.[6] Adult cases involving extreme CK elevation, anuric AKI, renal replacement therapy, and invasive mechanical ventilation remain uncommon.[5,7,8] We report the case of a previously healthy young man whose CK level reached 320,050 U/L and

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[1]Intensive Care Unit, Burdur State Hospital, Burdur, Türkiye
[2]Department of Thoracic Surgery, Hacettepe University Faculty of Medicine, Ankara, Türkiye

**Address for correspondence:**
Burcu Ancın, Department of Thoracic Surgery, Hacettepe University Faculty of Medicine, Ankara, Türkiye.
E-mail: ancinburcu@gmail.com

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who recovered following sequential intermittent hemodialysis and continuous renal replacement therapy (CRRT).

## Case Report

A previously healthy 22-year-old man who was not taking any regular medications presented with a two-day history of cough and fatigue. He denied trauma, crush injury, strenuous exercise, and substance exposure. His vital signs, serum creatinine and potassium levels, and white blood cell count were within reference ranges, whereas C-reactive protein (CRP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and lactate dehydrogenase (LDH) levels were mildly elevated. High-sensitivity troponin I was within the reference range. Total CK was not measured, and the patient was discharged with a presumed upper respiratory tract infection.

Eight hours later, he returned with fever, chills, diaphoresis, and dark, tea-colored urine. His temperature was 37.8°C, and his heart rate was 122 beats/min. Repeat laboratory testing revealed marked elevations in aminotransferases and LDH, leukocytosis, increased CRP, hyperkalemia, and hypocalcemia (Table 1). Serial blood gas analyses during the repeat emergency department assessment revealed mixed respiratory and metabolic acidosis. One sample showed a pH of 7.148, partial pressure of arterial carbon dioxide ($PaCO_2$) of 63.2 mmHg, and standard bicarbonate level of 16.1 mmol/L; another showed a pH of 7.05, $PaCO_2$ of 85 mmHg, and partial pressure of arterial oxygen ($PaO_2$) of 74 mmHg. A respiratory pathogen panel detected influenza A(H1N1), whereas all other tested pathogens, including severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), were negative. Toxicological screening did not identify an alternative toxic trigger for rhabdomyolysis.

Progressive respiratory distress prompted admission to the intensive care unit (ICU). Before intubation, the patient was tachypneic with intercostal retractions but reported no myalgia or muscle weakness. Muscle strength was preserved in all four extremities, and palpation elicited no tenderness. Serial limb examinations, including daily assessments of peripheral pulses, were performed to monitor for compartment syndrome. No limb swelling, muscle tenseness, or changes in skin color or temperature were observed initially or during follow-up. Noninvasive pressure-support ventilation (PSV: 10 $cmH_2O$; positive end-expiratory pressure [PEEP]: 5 $cmH_2O$; fraction of inspired oxygen [$FiO_2$]: 0.60) was initiated. Despite this support, respiratory acidosis worsened (pH: 6.94; $PaCO_2$: 117 mmHg; $PaO_2$: 78.4 mmHg), accompanied by confusion and lethargy, prompting intubation and invasive mechanical ventilation. Subsequent blood gas analysis showed a pH of 7.48, $PaCO_2$ of 31.3 mmHg, and $PaO_2$ of 117 mmHg. Chest computed tomography revealed bilateral ground-glass opacities consistent with pulmonary involvement in the setting of influenza A infection. On hospital day 1, CK was 146,970 U/L, potassium was 7.11 mmol/L, and creatinine was 1.48 mg/dL. During the first 24 hours, the patient received 7,120 mL of crystalloid, and urine output was 2,500 mL. Blood, tracheal aspirate, and urine samples were collected for culture before antimicrobial treatment was initiated. Following the detection of influenza A on the respiratory pathogen panel, oseltamivir was initiated at ICU admission, approximately two days after symptom onset. Empiric piperacillin-tazobactam and levofloxacin were also administered. All cultures subsequently showed no growth. Profound acidemia and life-threatening hyperkalemia prompted urgent intermittent hemodialysis.

On day 2, CK peaked at 320,050 U/L, and the patient developed anuria. Hypotension necessitated norepinephrine and hydrocortisone administration. Given the persistent hemodynamic instability and vasopressor requirement, further intermittent hemodialysis was deferred, and CRRT was initiated to allow more gradual fluid and solute management. Anuria persisted through day 5 despite declining muscle enzyme levels. By day 6, CK had decreased to 4,267 U/L, and urine output had resumed at 100 mL/day.

The patient was extubated on day 8. On day 9, urine output increased to 1,200 mL/day, and norepinephrine requirements decreased markedly, allowing CRRT to be discontinued. Later that day, worsening hypoxemia despite continuous positive airway pressure (CPAP) support, together with inadequate airway secretion clearance, necessitated reintubation. Because renal dysfunction and volume overload persisted, intermittent hemodialysis with ultrafiltration was subsequently resumed while low-dose norepinephrine support was maintained. These sessions were tolerated without hypotension or hemodynamic deterioration. By day 14, urine output had increased to 2,100 mL/day, and CK had decreased to 132 U/L. The patient was successfully extubated on day 15, and vasopressor support was discontinued on day 17.

**Table 1.** Early clinical and laboratory findings in a patient with influenza A-associated rhabdomyolysis and acute kidney injury

| Parameter | Initial emergency department visit | Second emergency department visit (~8 h later) | Intensive care unit day 1 | Intensive care unit day 2 |
|---|---|---|---|---|
| Creatine kinase (U/L) | — | — | 146,970 | 320,050 |
| Aspartate aminotransferase (U/L) | 68 | 2,169 | 4,980 | 4,001 |
| Alanine aminotransferase (U/L) | 93 | 392 | 913 | 880 |
| Lactate dehydrogenase (U/L) | 478 | 1,906 | 4,639 | 4,344 |
| Creatinine (mg/dL) | 0.97 | 1.09 | 1.48 | 1.95 |
| Blood urea nitrogen (mg/dL) | 19 | 21 | 29 | 31 |
| Potassium (mmol/L) | 4.24 | 5.33 | 7.11 | 5.70 |
| Total calcium (mg/dL) | 8.4 | 6.7 | 8.4 | 7.8 |
| Phosphate (mg/dL) | — | — | 10.2 | 4.2 |
| White blood cell count (×10³/µL) | 8.96 | 15.94 | 19.89 | 24.97 |
| C-reactive protein (mg/L) | 33.1 | 77.3 | 179.4 | 153.3 |
| Procalcitonin (µg/L) | — | — | 1.42 | — |
| pH | — | 7.148 | 6.94 | 7.27 |
| Partial pressure of carbon dioxide (mmHg) | — | 63.2 | 117 | 51 |
| Standard bicarbonate (mmol/L) | — | 16.1 | 18.1 | 19.6 |
| Lactate (mmol/L) | — | 2.40 | 4.24 | 3.95 |
| Urine appearance/output | — | Dark tea-colored urine | 2,500 mL/24 h | Anuria |

An em dash (—) indicates that data were unavailable in the medical records. Laboratory values reported for intensive care unit (ICU) day 1 were obtained from serial measurements during the first 24 hours of ICU admission and do not necessarily represent measurements collected at the same time.

Renal recovery lagged behind the decline in CK. On day 17, creatinine peaked at 6.16 mg/dL, and blood urea nitrogen was 128 mg/dL (Fig. 1). The final hemodialysis session was performed on day 19. On day 21, the patient was transferred to the ward awake, breathing spontaneously, hemodynamically stable, tolerating oral intake, and ambulatory. Renal function subsequently recovered without the need for long-term dialysis.

## Discussion

Influenza-associated rhabdomyolysis has been reported with varying degrees of organ involvement. Velho et al.[9] described marked CK elevation without AKI, whereas Benaini et al.[10] reported dialysis-requiring AKI associated with influenza A infection, strenuous exercise, and dehydration. Previous reports have documented CK levels exceeding 1,000,000 U/L,[5] influenza-associated AKI,[7,8,11,12] and severe cases requiring both invasive ventilation and renal replacement therapy.[5,7] Against this background, our case is notable for the progression from initially normal creatinine and potassium levels to dark urine and mixed acidosis within eight hours, followed by anuric AKI, severe hypercapnic respiratory failure, and vasopressor dependence, with subsequent recovery without long-term dialysis. Although the initial respiratory failure was characterized by severe hypercapnia, the deterioration preceding reintubation on day 9 involved hypoxemia and inadequate airway secretion clearance.

The subsequent clinical course demonstrated a clear dissociation between declining CK levels and renal recovery. CK had decreased to 132 U/L by day 14, whereas creatinine peaked at 6.16 mg/dL on day 17, and hemodialysis remained necessary until day 19. This course illustrates why declining CK levels should not be equated with recovery of renal function.

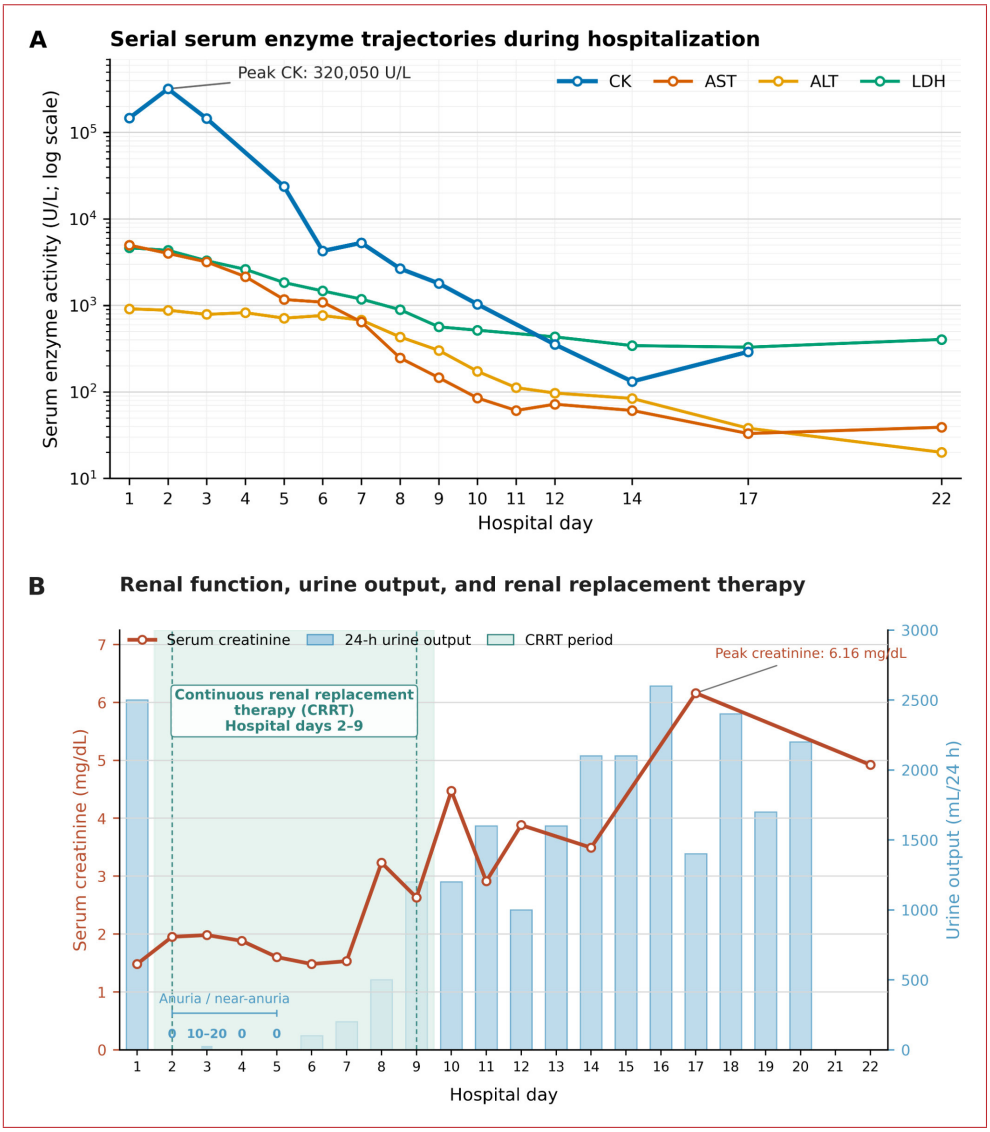



**Figure 1. Clinical course during hospitalization. (A) Serum creatine kinase, aspartate aminotransferase, alanine aminotransferase, and lactate dehydrogenase levels are presented on a logarithmic scale. Creatine kinase peaked at 320,050 U/L on hospital day 2. (B) Serum creatinine (line, left axis), 24-hour urine output (bars, right axis), and the duration of continuous renal replacement therapy (shaded area, hospital days 2–9) are shown. Urine output recovered before serum creatinine levels normalized, with serum creatinine peaking at 6.16 mg/dL on hospital day 17.**

**ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CK: Creatine kinase; CRRT: Continuous renal replacement therapy; LDH: Lactate dehydrogenase.**

Alternative causes of rhabdomyolysis include trauma, prolonged immobilization, muscle ischemia, strenuous exercise, temperature extremes, drugs or toxins, electrolyte disturbances, endocrine disorders such as hypothyroidism, autoimmune diseases, inherited muscle disorders, and other infections.[4] Our patient reported no trauma, strenuous exercise, regular medication use, or substance exposure, and toxicological screening identified no alternative toxic trigger. The respiratory prodrome and detection of influenza A support an infection-associated episode, although an underlying predisposition or other contributing factors cannot be completely excluded.

The pathogenesis is likely multifactorial. Direct viral invasion, cytokine-mediated injury, metabolic stress, hypoperfusion, and systemic inflammation may contribute.[1,4-6] Pigment-associated AKI involves renal vasoconstriction, heme-mediated tubular toxicity, oxidative injury,

and tubular obstruction, particularly in the presence of hypovolemia and aciduria.[2-4] Subsequent hypotension may have exacerbated renal injury. In severe influenza, AKI may also result from hemodynamic compromise, systemic inflammation, sepsis, and nephrotoxic exposure.[13]

At the initial assessment, aminotransferase and LDH levels were already elevated, although CK was not measured. Elevated CK levels have been associated with worse outcomes in patients with influenza,[14] but CK alone does not determine the risk of kidney failure. A multivariable assessment incorporating renal function and metabolic abnormalities is more informative.[15] Therefore, serial monitoring of urine output, potassium, phosphate, calcium, bicarbonate, and creatinine should accompany CK measurements.

Management focuses on prompt treatment of the underlying cause, individualized crystalloid resuscitation, and correction of complications.[2-4] Renal replacement therapy is indicated for conventional reasons, such as refractory hyperkalemia, severe acidemia, anuria, uremic complications, or fluid overload, rather than on the basis of CK concentration alone.[2,3] In our patient, intermittent hemodialysis addressed the initial hyperkalemia and acidemia, whereas CRRT provided continued support during hemodynamic instability.

This report is limited by its single-patient design, the lack of total CK measurements during the first two emergency department visits, and the inability to determine the individual contributions of viral muscle injury, hypoperfusion, and treatment to the patient's renal outcomes. Nevertheless, serial biochemical measurements and urine output data document both the rapid progression and reversibility of severe disease.

## Conclusion

Influenza A-associated rhabdomyolysis can rapidly lead to life-threatening hyperkalemia, profound acidemia, respiratory failure, and dialysis-requiring AKI in previously healthy adults. Early CK measurement, serial metabolic and renal monitoring, and timely organ support are essential. Recovery without the need for long-term dialysis remains possible.

**Ethics Committee Approval:** This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

**Informed Consent:** Written informed consent was obtained from the patient for publication of the case and the accompanying clinical data and figure.

**Conflict of Interest:** The authors have no conflicts of interest to declare.

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