CASE REPORT

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West Nile Virus Encephalitis in a Kidney Transplant Patient

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Abstract

West Nile virus encephalitis is a neuroinvasive condition with significant diagnostic challenges, especially in immunosuppressed patients. We describe the case of a 54-year-old male patient with a history of kidney transplantation and systemic comorbidities. He presented with fever and altered mental status, and neuroimaging revealed bilateral thalamic lesions and leptomeningeal enhancement. West Nile virus was confirmed via polymerase chain reaction from cerebrospinal fluid. The patient was managed with supportive care and close monitoring. Early recognition and comprehensive diagnostics are crucial for effectively managing high-risk patients, despite the lack of a specific treatment for West Nile virus, as with most other viral diseases.

Keywords: Encephalitis; Kidney transplantation; Leptomeningeal involvement; West Nile virus.

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Introduction

West Nile virus (WNV), a single-stranded RNA virus belonging to the *Flaviviridae* family and classified under the *Flavivirus* genus, is primarily transmitted by mosquitoes, particularly those of the *Culex* genus. Transmission can also occur through the bite of an infected animal, and in rare cases, via blood transfusions and solid organ transplants.[1,2] The majority of WNV cases are asymptomatic or present with flu-like symptoms. WNV with neurological involvement, such as encephalitis and meningitis, can be fatal in one out of every 150 cases.[3] The West Nile virus has also been shown to induce dysfunction in the cranial nerves.

We describe a case of West Nile virus encephalitis with a comprehensive evaluation of a patient who presented with fever, altered consciousness, bilateral thalamic lesions, and pathologic contrast enhancement in the right trigeminal nerve and both oculomotor nerve cisternal segments on cranial magnetic resonance imaging. In this case, we aimed to emphasize the difficulties in identifying West Nile virus and the increased exposure to opportunistic pathogens with greater numbers of organ transplantations. Signed consent was obtained from the patient's guardian.

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Case Report

A 54-year-old male patient with a history of type 2 diabetes mellitus, hypertension, coronary artery disease, and kidney transplantation secondary to diabetic nephropathy was admitted to the emergency department with a fever of 38.3°C, decreased communication, nausea and vomiting, diarrhea, and poor oral intake. The Glasgow Coma Scale score at admission to hospital was 15. Vitals signs were: peak heart rate 96 beats/min, blood pressure 142/81 mmHg, SpO_2 97%, and respiratory rate 23/min. Although the quick Sequential Organ Failure Assessment (qSOFA) score was 1, the patient was considered to have sepsis given the clinical picture. The immunosuppressive drugs he was taking included prednisolone, tacrolimus, and mycophenolate mofetil. Mycophenolate mofetil was discontinued in consultation with the transplantation team, as the patient also had a history of kidney transplantation, and stress-dose corticosteroid methylprednisolone treatment was started.

Brain Magnetic Resonance Imaging (MRI) was performed to investigate the possibility of cerebrovascular events, given the patient's altered consciousness. A right thalamic infarction was detected on diffusion-weighted MRI (Fig. 1), and the patient was referred to Neurology. In the patient presenting with diarrhea and a history of broad-spectrum antibiotic use within the past three months, testing for *Clostridioides difficile* toxin A/B was requested to evaluate for possible antibiotic-associated enterocolitis. Cytomegalovirus (CMV) polymerase chain reaction (PCR) testing was also performed to investigate a viral etiology. CMV DNA PCR was below 500 quantitative copies, and *C. difficile* toxin A/B was negative. One day after admission to the hospital, the patient exhibited signs of somnolence. He opened his eyes in response to painful stimuli, made incomprehensible sounds, and his Glasgow Coma Scale (GCS) score dropped from 15 to 7. The patient underwent orotracheal intubation to ensure airway safety. On the second day of intensive care monitoring, a GCS of 3 was observed, and an electroencephalogram (EEG) was performed. Rare sharp-slow wave activity was observed in the left frontotemporal and occipital regions on EEG. These findings were interpreted as indicative of a tendency toward hypersynchronization in the left frontotemporal and left occipital regions based on widespread irregularity. Unconsciousness, rightward gaze deviation, eye ptosis, and absent pupillary light reflex in the left eye were observed. Therefore, contrast-enhanced brain MRI was performed, and chronic ischemic gliotic hyperintensities were observed in the cerebral white matter. The millimetric acute ischemic diffusion restriction areas defined in the previous examination at the bilateral thalamic level showed marked regression (Fig. 2). Minimal leptomeningeal contrast enhancement was observed in the cerebellar folia. Contrast enhancement was also found in the right trigeminal nerve and both oculomotor nerve cisternal segments.

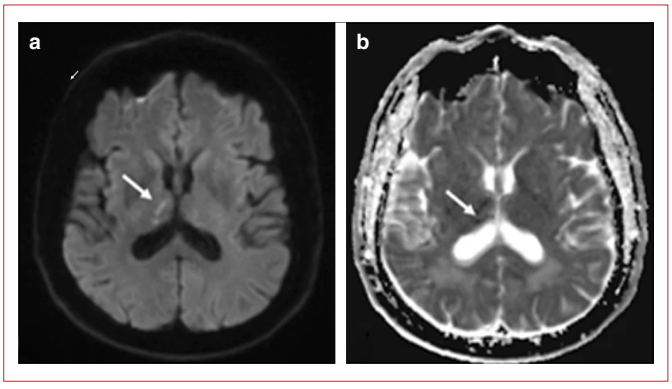

Figure 1. Diffusion-weighted magnetic resonance imaging (MRI) scan. (a) Right and left thalamic cytotoxic edema is observed as diffusion restriction in a linear pattern (arrow). (b) Apparent diffusion coefficient (ADC) maps show ADC hypointensity confirming right thalamic cytotoxic edema (arrow).

Considering the preliminary diagnosis of meningitis or leptomeningeal/perineural involvement of a systemic or neoplastic process, a lumbar puncture was performed and cerebrospinal fluid (CSF) analysis was conducted. The glucose level in the CSF was 153 mg/dL, while the simultaneous blood glucose level was 192 mg/dL, and the protein level was 169 mg/dL. CSF showed 40/μl leukocytes and 60/μl erythrocytes, comprising 80% basophils and 20% lymphocytes. Infectious Diseases and Clinical Microbiology (IDCM) consultation was requested. The meningoencephalitis panel FilmArray (BIOFIRE®) was negative. It was planned to repeat CSF sampling and investigate atypical agents (West Nile virus, *Treponema pallidum*, oligoclonal bands, CMV, *varicella zoster virus (VZV), herpes simplex virus (HSV), Toxoplasma gondii, adenovirus*), immunoglobulin G (IgG) index, beta-D-glucan, fungal culture, flow cytometry, and paraneoplastic/autoimmune encephalitis panel tests. Paraneoplastic/autoimmune encephalitis panels, ganglioside panels, and tests for *Brucella, Borrelia, and Mycobacterium tuberculosis* were also performed in blood. The PCR test for West Nile virus, performed at the Ministry of Health, yielded a positive result. Since the clinical and laboratory findings

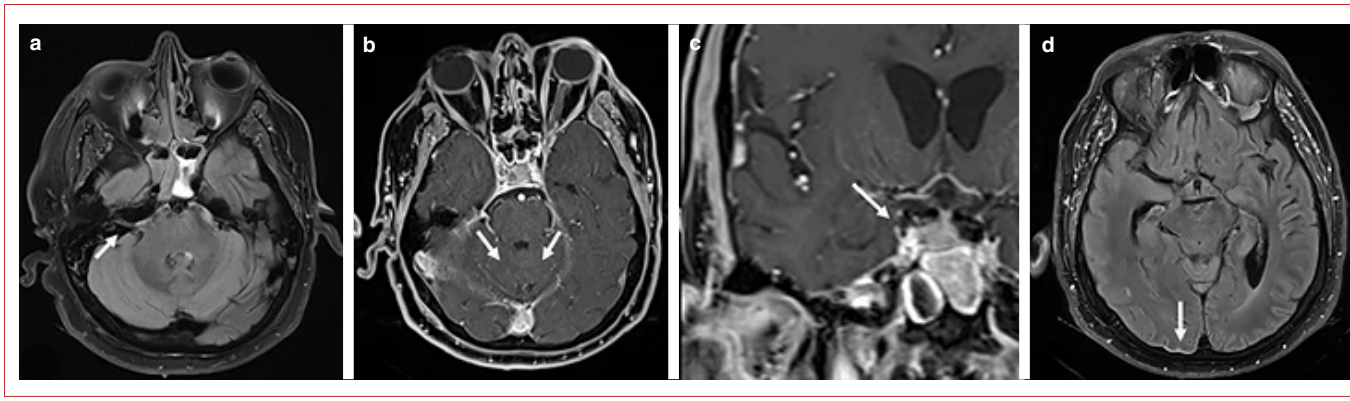

Figure 1. Postcontrast magnetic resonance imaging (MRI). (a) Post-contrast fluid-attenuated inversion recovery (FLAIR) scan shows focal perineural contrast enhancement in the right trigeminal nerve intracanalicular segment (arrow). (b) Post-contrast axial T1A series shows leptomeningeal contrast enhancement in the superior cerebellar follicles (arrow). (c) Post-contrast coronal T1A series shows contrast enhancement in the right oculomotor nerve cisternal segment (arrow). (d) Post-contrast FLAIR scan shows mild dural contrast enhancement in the right occipital convexity (arrow).

were compatible, the patient was diagnosed with West Nile virus encephalitis, and empirical antibiotic treatment was discontinued as no other agent was detected.

Discussion

In this report, we describe the clinical course, diagnostic workup, and therapeutic process of a rare case of WNV-associated encephalitis in a kidney transplant recipient. The present WNV case is among the few reported in the literature, characterized by severe neurological symptoms that necessitated admission to the intensive care unit and a permanent tracheostomy. With increasing awareness and reported case numbers, WNV is of epidemiological importance in various regions. According to European Centre for Disease Prevention and Control (ECDC) data, 1,202 cases of WNV were reported by November 2024.[4]

Immunosuppressed patients, particularly solid organ transplant (SOT) recipients, hematopoietic stem cell transplant (HSCT) recipients, and patients receiving B-cell-depleting therapies, can significantly alter the epidemiological profile of WNV transmission. Donor-derived transmission through organ or blood transfusion has been documented. Additionally, the onset of infection in transplant recipients has been reported to occur up to approximately 50 months after transplantation.[5] Our patient underwent a kidney transplant 36 months earlier, and there is a possibility of infection from the donor. There is also a possibility of WNV infection from red blood cells and fresh frozen plasma transfusions. Organ donors are not routinely screened for WNV, and both organ donors and transplant recipients often receive multiple blood transfusions. Therefore, screening both SOT donors and blood donors for WNV may allow for earlier detection of the virus.[6]

Approximately one-fifth of patients infected with WNV develop fever, headache, neck pain, and flu-like symptoms. It has been shown that neuroinvasive disease can develop in one out of every 150 to 250 cases.[7] In solid organ transplant recipients with donor-derived WNV infection, up to 75% develop neuroinvasive disease, which may present as meningitis, encephalitis, or acute flaccid paralysis.[5,8] Subsequently, neurological symptoms may manifest, including altered mental status, weakness, and abnormal movements.[5]

Neuroimaging plays an important role in the evaluation of suspected WNV encephalitis. Brain computed tomography (CT) scans are often normal in the early stages of infection; MRI has higher sensitivity in detecting inflammatory changes in the brain parenchyma. Characteristic MRI findings include T2-weighted hyperintensities and diffusion restrictions in regions such as the thalamus, basal ganglia, brainstem, and white matter, though these findings are not always present. Leptomeningeal contrast enhancement may also be observed, indicating more widespread meningeal involvement.[8] Our patient had T2-weighted hyperintense areas in the white matter and leptomeningeal contrast enhancement.

For diagnostic confirmation, if there is a clinical suspicion or detailed patient history suggesting the possibility of WNV infection, the Centers for Disease Control and Prevention (CDC) guidelines recommend a more nuanced approach: if neurological symptoms are present,

PCR testing on CSF should be considered as an initial diagnostic tool. In cases of PCR positivity, the diagnosis is considered confirmed, and the patient should be managed accordingly. Conversely, in cases where only serological testing (immunoglobulin M, IgM positivity) is observed, the patient should be treated as a suspected case, and further monitoring is required.[1,9]

Like most viral infections, there is no specific antiviral treatment for WNV; patients are typically given symptomatic and supportive care. In selected cases, reduction of immunosuppressive therapy has been associated with clinical improvement. Despite interventions, a 30% mortality rate has been reported in patients with severe encephalitis who have undergone kidney transplantation with neuroinvasive WNV. Survivors of WNV encephalitis may experience long-term neurological sequelae, including cognitive impairment, motor deficits, and neuropsychiatric disorders, all of which contribute to a decline in quality of life.[10-12]

In light of current data and considering the high morbidity and mortality associated with WNV encephalitis in kidney transplant patients, preventive measures are of great importance. Although routine screening of organ donors for WNV is not mandatory in all regions, targeted screening is recommended in endemic areas or during periods of high WNV activity.[11,13]

Conclusion

West Nile virus infection has been shown to cause meningoencephalitis. Patients who have undergone kidney transplantation are at higher risk due to immunosuppressive therapy. Especially in endemic areas, advanced donor screening is a critical measure that can reduce the incidence of WNV transmission from donors. Early diagnosis and prompt supportive treatment are crucial for optimizing patient outcomes. Therefore, the education of organ transplant surgeons and infectious disease specialists is important. Further clinical studies are needed to better understand the disease and optimize treatment strategies.

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

Informed Consent: Signed consent was obtained from the patient's guardian.

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