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An Uncommon Neuroimaging After A Recent Viral Infection: A Case Report

[1.]Dr Bharat Rajkumar, [2]Dr Cherukara Nihil , [3]Dr Karthika Aravindakshan, [4]Dr Lihil CK , [5]Dr Ajay Nathan, [6]Dr Roshan Thomas, [7]Dr Rose Maria Jose

[1]Neurology Consultant, Department of Medicine, INHS Sanjivani Hospital, Kochi - 682004 India
[2]District Medical Officer, CGDHQ4, Indian Coast Guard, Kochi, 682001 India
[3]Medical Officer, Kochi - 682036 India
[4]Resident, Department of Radiodiagnosis ,INHS Asvini Hospital, Mumbai - 400005 India
[5]Consultant, Department of Medicine, INHS Sanjivani Hopsital, Kochi - 682004
[6]Medical Officer, Trivandrum - 695006 India.
[7]Medical Officer, Trivandrum - 695006 India
bharat.rajkumar@gmail.com[1] cknihilrock@gmail.com[2] karthikaaravindakshan@gmail.com[3] lihilck69@gmail.com[4] nathanajay@gmail.com[5] roshanthomas6737@gmail.com[6] rosemariajose@gmail.com[7]
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ABSTRACT

Background: The West Nile virus is an enveloped, single-stranded RNA arbovirus that can cause disease in humans. The majority of individuals infected with West Nile Virus remain asymptomatic. Symptoms can vary from fever, body aches, skin rash, and swollen lymph nodes to significant neurological impairments resulting from encephalitis and neuroinvasive disease. We present a case of West Nile viral infection with rapid neurological worsening, and neuroimaging showing an unusual pattern with only one literature report.

Case presentation: A 42-year-old female presented with vertigo followed by loss of consciousness. She had history of high-grade fever without chills of six days duration two weeks prior, associated with body ache, arthralgia and reduced appetite. Routine blood investigations were within normal limits. Clinical examination revealed a GCS of E2V1M5 which worsened to E1V1M1on next day, pupils pinpoint, moving all four limbs in response to a painful stimulus, bilateral symmetrical sluggish deep tendon reflexes and bilateral extensor plantar reflex. Her MRI brain showed extensive T2 and FLAIR hyperintense lesions in bilateral cerebral hemisphere predominantly white matter more than grey matter with sparing of the thalamus and basal ganglia. Lesions showed significant diffusion restriction.

Discussion and conclusion: This case highlights a rare and atypical neuroimaging finding in West Nile virus infection, emphasizing the importance of recognizing such findings in post-viral neurological syndromes, particularly in endemic regions.

Keywords: West Nile Virus, CNS vasculitis, neuroimaging, post-infectious demyelination

BACKGROUND

West Nile Virus (WNV) is a zoonotic, mosquito-borne arbovirus primarily spread by mosquitoes of the *Culex* genus. Most individuals infected with WNV remain asymptomatic; however, approximately 25% develop fever and symptoms resembling a viral syndrome, while a smaller proportion, about 1 in 200, progress to neuroinvasive disease. Severe complications, such as encephalitis and marked neurological impairments, occur in approximately 1% of cases, with increased morbidity observed in individuals over 50 years old.

Here, we report a case of West Nile viral infection in a 42-year-old female from South Kerala, presenting with vertigo followed by loss of consciousness. Neuroimaging revealed predominant white matter involvement, significant diffusion restriction, and absent blooming—an atypical pattern rarely documented in the literature.

CASE PRESENTATION

A 42-year-old female from South Kerala presented with vertigo followed by loss of consciousness. She reported a high-grade fever without chills two weeks prior, lasting six days. Associated symptoms included body ache, arthralgia, and reduced appetite. Fever was not accompanied by rash, headache, vomiting, seizures, or altered sensorium.

Clinical examination revealed; a GCS of E2V1M5, pupils pinpoint, moving all four limbs in response to a painful stimulus, bilateral symmetrical sluggish deep tendon reflexes and bilateral extensor plantar reflex. Next day her GCS worsened to E1V1M1.

Her blood tests, including complete blood counts, blood sugar, liver function, renal function and electrolytes were normal. Her CSF examination showed no cells, protein of 60 mg/dl, glucose 56 mg/dl with corresponding blood glucose of 82 mg/dl.

Routine blood investigations, Viral markers, Malarial parasite, Anti TPO antibodies, ANA profile, ANCA, Serum and CSF VDRL, IgM JE, CSF neuro 9 panel, H1N1, and IgM scrub were negative. Serum and CSF dengue IgM were negative. Serum ammonia and ACE levels were normal. Serum and CSF West Nile IgM was positive.

CT Brain was normal. MRI brain showed extensive T2 and FLAIR hyperintense lesions in bilateral cerebral hemisphere predominantly white matter more than grey matter with sparing of the thalamus and basal ganglia. Corpus callosum, brainstem structures, cerebellar peduncles and cerebellar hemispheres were involved but less extensively than cerebral hemispheric involvement. Lesions showed significant diffusion restriction (Figure 1a,b and c). Post contrast images showed no enhancement in lesions. There was no evidence of blooming. MR angiogram and venogram was normal.

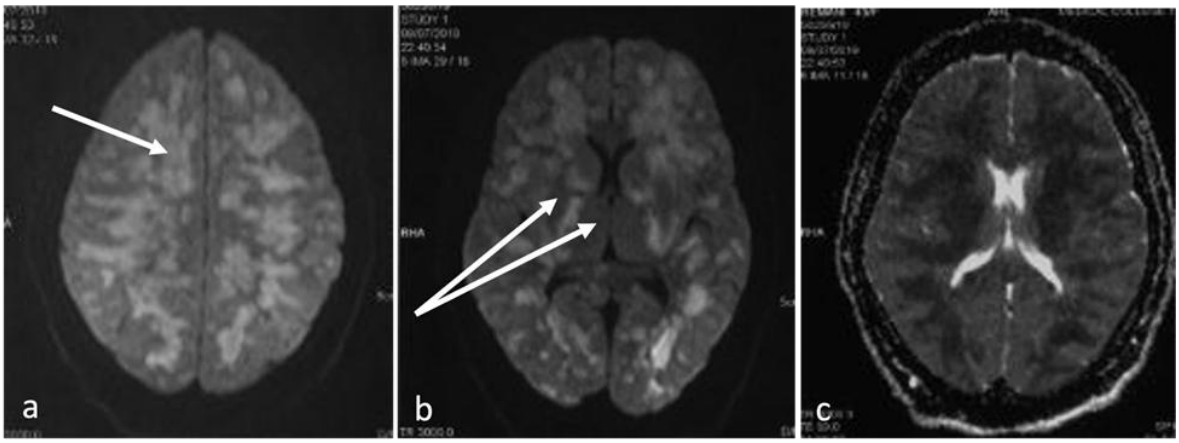
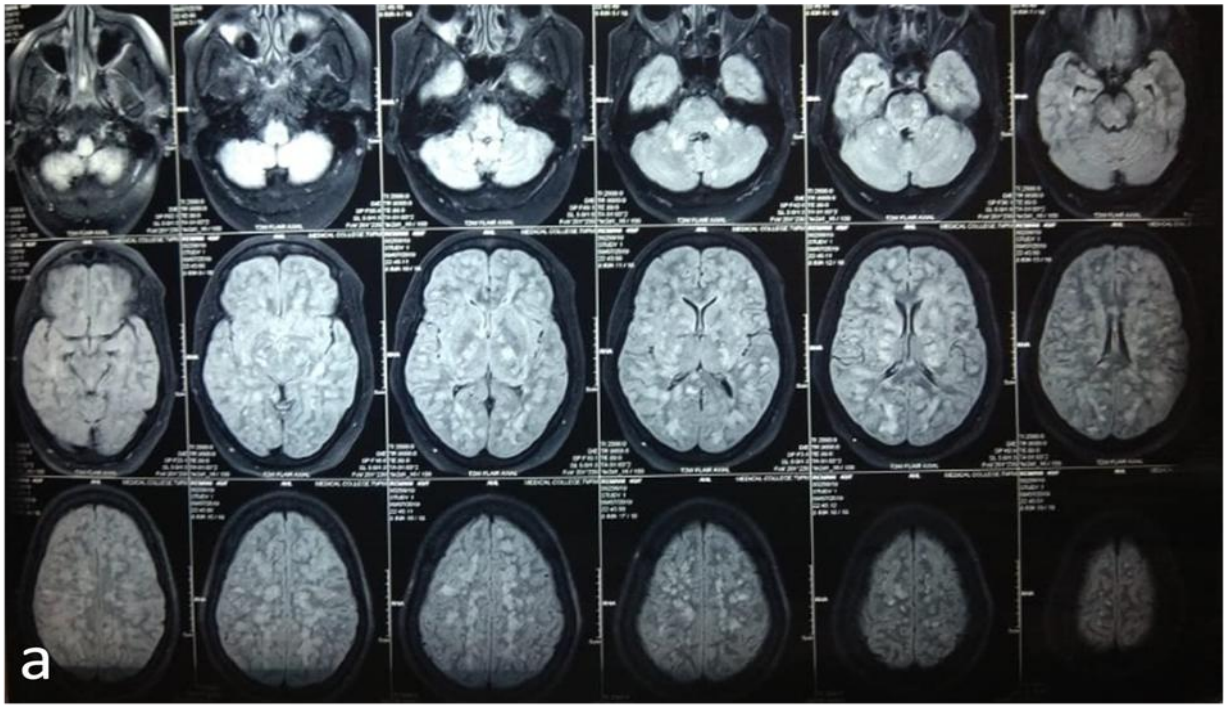


Figure 1: a, b and c: Diffusion Restriction involving subcortical and deep white matter sparing cortical and deep grey matter



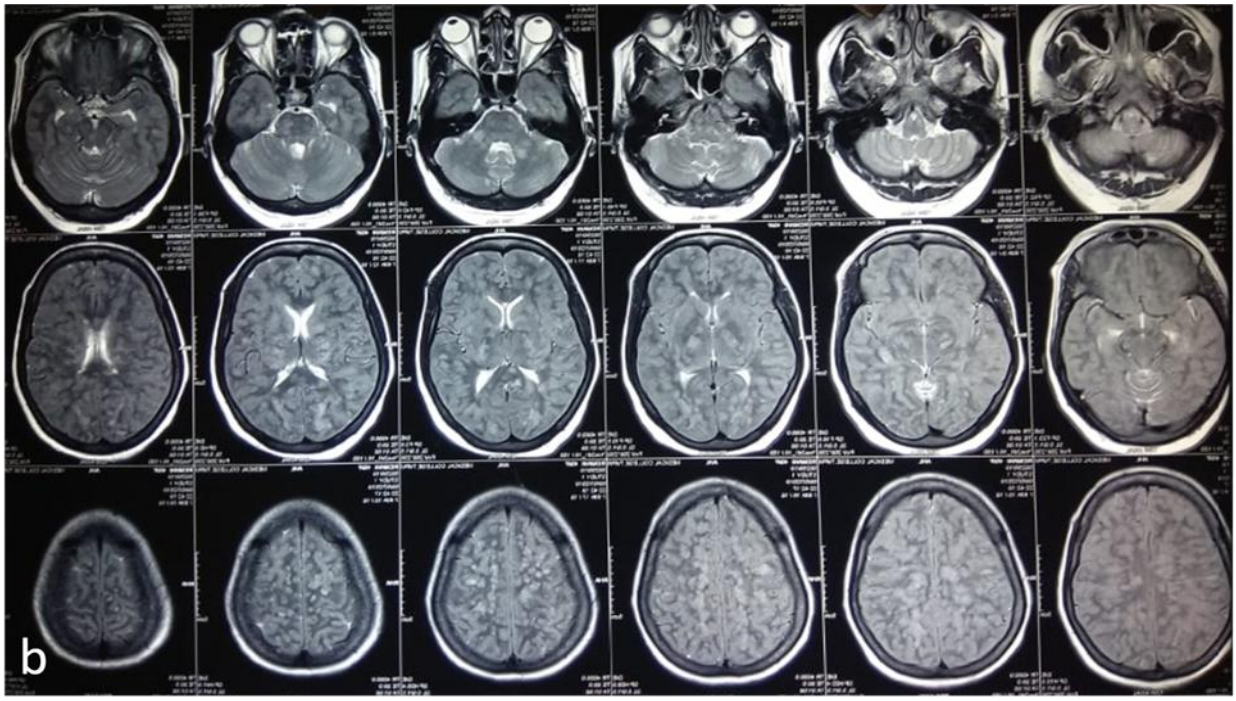


Figure 2: a & b T2/FLAIR hyperintensities involving subcortical and deep white matter sparing basal ganglia and thalamus.

DISCUSSION

West Nile disease is predominantly asymptomatic, with clinical presentation varying based on the severity of the host's response to the virus. While the majority of individuals remain symptom-free, approximately 20% develop symptoms characteristic of viral fever, referred to as West Nile fever. Less than 1% of infected individuals progress to neuroinvasive disease, typically occurring in those with advanced age or compromised immune systems.

In this case, a 42-year-old female presented with vertigo followed by loss of consciousness and a history of febrile illness two weeks prior. Given her recent viral infection, post-infectious central nervous system (CNS) demyelination or vasculitis were initially considered as the primary differential diagnoses.

The acute onset of neurological symptoms, coupled with rapid deterioration in sensorium, prompted an MRI brain study. Imaging revealed bilateral symmetric T2 /FLAIR hyperintensities in subcortical white matter, and posterior fossa structures with sparing of the thalamus and basal ganglia.

Simultaneously, she was evaluated for her recent febrile episode. Serological and cerebrospinal fluid (CSF) analyses confirmed a diagnosis of West Nile viral infection.

However, the imaging findings in this case are unusual, as arboviral encephalitis typically demonstrates predominant involvement of the thalamus, basal ganglia, and brainstem. The acute neurological deterioration following recent West Nile virus infection raises the possibility of post-infectious demyelination or vasculitis as underlying mechanisms. In acute disseminated encephalomyelitis (ADEM), imaging findings are typically characterized by patchy, rounded lesions with poorly defined margins and prominent involvement of deep grey matter nuclei, the thalamus, brainstem, subcortical white matter, and U-fibres. However, the imaging in this case does not align with the classic radiological features of ADEM, suggesting an alternative pathology.

The extensive diffusion restriction observed on imaging is consistent with infarction. The absence of contrast enhancement may be attributed to imaging performed within two weeks of the onset of illness. Large vessel occlusion is unlikely, given the normal MR angiography findings and the bilateral symmetrical involvement.

We postulate that post-infectious CNS vasculitis, affecting small cerebral vessels and resulting in multiple cerebral infarctions, is the underlying cause of this presentation. Notably, the patient hails from a geographical region in South Kerala where West Nile infection has been previously reported.

This represents the first documented case of West Nile virus infection associated with such extensive MRI changes, making it a significant and novel contribution to the literature.

CONCLUSION:

In conclusion, this case highlights a rare presentation of West Nile virus infection with extensive MRI changes, including bilateral symmetrical diffusion restriction and subcortical white matter involvement, which are atypical for arboviral encephalitis. The findings suggest post-infectious CNS vasculitis as the likely underlying mechanism, contributing to multiple cerebral infarctions. This case underscores the need for heightened awareness of atypical neuroimaging patterns in post-viral neurological syndromes, especially in endemic regions.

LIST OF ABBREVIATIONS:

GCS - Glasgow Coma Scale

T2 - T2-weighted (MRI imaging)

FLAIR - Fluid Attenuated Inversion Recovery

MRI - Magnetic Resonance Imaging

WNV - West Nile Virus

CSF - Cerebrospinal Fluid

TPO - Thyroid Peroxidase

ANA - Antinuclear Antibodies

ANCA - Antineutrophil Cytoplasmic Antibodies

VDRL - Venereal Disease Research Laboratory

IgM - Immunoglobulin M

JE - Japanese Encephalitis

H1N1 - Influenza A

ACE - Angiotensin-Converting Enzyme

ADEM – Acute Disseminated Encephalomyelitis

DECLARATIONS:

Competing interests: The authors declare that they have no competing interests.

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AUTHORS CONTRIBUTIONS:

1. Contributed to the clinical management of the patient, conceptualized the case report, collected patient data, drafted the initial manuscript, provided critical revisions, and approved the final version for submission.

2. Reviewed relevant literature and assisted in drafting the discussion section. Also contributed to the preparation of the final manuscript by reviewing existing publications and integrating relevant findings.

3. Contributed to the manuscript's methodology section and reviewed the final manuscript, including a review of existing literature to inform the case discussion.

4. Assisted with reviewing the manuscript.

5. Reviewed the manuscript and contributed to the final version. Responsible for gathering and verifying references to ensure completeness and accuracy in the final manuscript.

6. Reviewed the manuscript and contributed to the final version. Responsible for gathering and verifying references to ensure completeness and accuracy in the final manuscript.

7. Reviewed the manuscript and contributed to the final version. Responsible for gathering and verifying references to ensure completeness and accuracy in the final manuscript.

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