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Case Series

Clinical profile and outcome of viral encephalitis patients in a tertiary care hospital

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ABSTRACT

Early detection and management of viral encephalitis patients presenting with various symptoms result in better outcomes. This study seeks to address gaps in timely diagnosis and management to reduce long-term neurological deficits. To study the clinical profile and outcomes of viral encephalitis patients admitted to a tertiary care hospital. Retrospective, observational study of 30 patients admitted at Travancore Medical College Hospital from April 2023 to May 2024. Fever, altered sensorium, and seizures were the most common presentations. Recovery was slower among immunocompromised patients, particularly those with chronic kidney disease. Common etiologies included Dengue, Herpes Simplex Virus (HSV), and Varicella Zoster Virus (VZV). Viral encephalitis remains a significant neurological emergency. Early recognition and management improve outcomes and reduce morbidity.

Keywords: Viral encephalitis, Dengue, Herpes simplex virus, Varicella zoster virus

INTRODUCTION

Viral encephalitis, characterized by inflammation of the brain parenchyma caused by viral agents, is a significant public health concern. It presents with varied clinical manifestations, making timely diagnosis and management challenging. This study evaluates the clinical features, diagnostic approaches, and outcomes of viral encephalitis cases in a tertiary care setting. Viral encephalitis remains a significant global health concern due to its high morbidity and mortality rates. It affects approximately 3.5 to 7.5 individuals per 100,000 annually, with children and the elderly being most vulnerable.[4,6] The causative agents of viral encephalitis include herpesviruses, arboviruses, and enteroviruses, among others. Among these, herpes simplex virus (HSV) is the leading identifiable cause globally.[3] Advances in neuroimaging and molecular diagnostic tools, such as polymerase chain reaction (PCR) and next-generation sequencing (NGS), have improved our ability to identify viral pathogens.[4] Despite these advancements, a substantial number of cases remain idiopathic, underscoring the need for better diagnostic approaches.[5] The pathogenesis of viral encephalitis involves direct viral invasion and immune-mediated neuronal damage, resulting in a wide spectrum of clinical presentations ranging from mild fever to severe neurological deficits

Commonly seen virus

Arbovirus

Japanese encephalitis, St. Louis encephalitis, West Nile encephalitis, equine encephalitis, Venezuelan equine encephalitis.

Buniya virus

Californian encephalitis.

Reovirus

Colorado tick fever encephalitis.

Herpes

Herpes simplex virus I and Herpes simplex virus II, Varicella zoster, Epstein-Barr, Cytomegalo, Human herpes virus 6, B- virus.

Myxo and Paramyxo

Influenza, measles, mumps, parainfluenza, Nipah virus. Adeno, Parvo and Rhabdovirus.[4-6]

Clinical presentation

Fever, headaches, convulsions, and altered mental status are the most prevalent signs and symptoms. Neuropsychiatric symptoms are frequently observed, including behavioural abnormalities, hallucinations, and/or cognitive deterioration. Additionally, patients may experience additional symptoms or test results that are unique to a particular virus. For instance, herpes zoster encephalitis is characterised by rash and cutaneous vesicles, whereas EBV is typically linked to lymphadenopathy and splenomegaly. Aphasia, memory impairments, and mental symptoms are frequently observed in cases of HSV encephalitis, which affects the temporal and frontal lobes.

However, because arboviruses primarily attack the basal ganglia, some of them elicit motor symptoms such parkinsonian movements and choreoathetosis. Features: Extrapyramidal symptoms, which resemble Parkinson disease, may be brought on by JE. Enterovirus 71 may cause tremor, myoclonus, ataxia, pulmonary edema, and cranial nerve palsies. Nipah virus has been shown to produce brainstem and cerebellar signs, hypertension and segmental myoclonus. Microcephaly is typical of Zika virus infection.[7,8]

Prevalence

Viral encephalitis affects 3.5 to 7.5 persons per 100,000, with the elderly and young having the highest frequency. With time, the epidemiology of some viral encephalitis causes has altered. For instance, the incidence of encephalitis caused by the measles and mumps has decreased as a result of immunisation.

However, because EBV and CMV encephalitis affect immunocompromised people, such as those with AIDS, transplant recipients, and chemotherapy patients, they are becoming increasingly common. Other important epidemiologic factors include the time of the year, geography, and animal or insect exposure.[9-11]

To study the clinical profile and outcome of viral encephalitis patients in a tertiary care hospital. This retrospective observational study was conducted at Travancore Medical College Hospital, Kollam, from April 2023 to May 2024. Patients presenting with fever, headache, seizure, altered sensorium, behavioral changes, or limb weakness were admitted to the Neuro Care Center. Selection criteria included adults aged 18 and above diagnosed with viral encephalitis. Sampling was purposive, and data were collected from medical records. Routine metabolic screening, MRI Brain, CSF analysis, and Biofire viral panel studies were performed. Patients received tailored treatment based on their condition. Ethical approval was obtained from the institutional ethics committee.

It is a retrospective Observational study of viral encephalitis patients admitted in Travancore medical college hospital, Kollam from April 2023 till May 2024. Presenting to casualty with fever/headache/seizure/ Altered sensorium/Behavioral changes/limb weakness were admitted in Neuro care center.

Routine metabolic screening followed by MRI Brain done. CSF analysis done for Sugar, protein, cell count, ADA, Cell cytology, Culture and sensitivity, Gram stain), Biofire viral panel study. Empirically patient was started on intravenous antivirals and intravenous antibiotics at meningitis dose (GFR adjusted dosage), antiepileptics, anti edema measures and ventilatory support if needed. Intravenous immunoglobulins and plasmapheresis done in selected patients. Patients were mobilised upon recovery and rehabilitation procedures were done.

Informed consent

Written informed consent will be taken from all the participants before the conduct of study.

CASE SERIES

This section includes summaries of 30 cases of viral encephalitis treated at the Travancore Medical College Hospital. The following cases highlight the diversity in clinical presentations and outcomes.

Case 1

35/F-Fever, tremor, status epilepticus. Normal MRI. Leukocytosis. Immunocompetent. Unknown etiology.

Case 2

40/M-Fever, tremor, headache. Dengue positive. Recovered.

Case 3

45/M-Fever, headache, altered sensorium. Unknown etiology. Recovered.

Case 4

28/M-Fever, comatose, refractory status epilepticus. ADEM. Dengue positive.

Case 5

22/F-Fever, seizure, comatose. Bilateral thalamic hyperintensity. Leukocytosis. Dengue positive.

Case 6

65/F-Fever, diplopia, ataxia. Normal MRI. Leukocytosis. GQ1b+ (Bickerstaff encephalitis).

Case 7

55/F-Fever, headache, seizure. Normal MRI. Leukocytosis. Immunocompetent.

Case 8

18/F-Fever, refractory status epilepticus, myocarditis. Panencephalitis. Leukocytosis. Dengue positive.

Case 9

30/M-Fever, altered sensorium, paraparesis. Hemorrhagic myelitis. Dengue positive.

Case 10

66/M-Fever, altered sensorium, mutism. Bilateral temporal hyperintensities. HSV1+ PCR.

Case 11

22/F-Fever, headache. Normal MRI. Leukocytosis. Unknown etiology.

Case 12

12/M-Fever, altered sensorium, quadriparesis. ADEM. Leukocytosis. Biofire nil.

Case 13

66/F-Fever, diplopia, quadriparesis. Normal MRI. Leukocytosis. GQ1b+ (Bickerstaff encephalitis).

Case 14

62/M - Fever, paraparesis, urinary retention. Myelitis. Leukocytosis. COVID-19 positive.

Case 15

60/M - Fever, altered sensorium, varicella zoster lesions. Normal MRI. Leukocytosis. CKD. VZV.

Case 16

58/F - Fever, myocarditis, status epilepticus. Temporal hyperintensities. HSV PCR+. CKD.

Case 17

40/M-Fever, headache, seizure. Normal MRI. Leukocytosis. Unknown etiology.

Case 18

55/F-Fever, hallucinations, seizure. Normal MRI. Leukocytosis. On immunosuppressants for RA.

Case 19

72/M-Fever, status epilepticus. Cortical atrophy. Leukocytosis. CKD. Dengue positive.

Case 20

66/F - Fever, diplopia, ataxia, quadriparesis. Normal MRI. Leukocytosis. GQ1b+ (Bickerstaff encephalitis).

Case 21

40/F - Fever, VZV lesions, altered sensorium. Normal MRI. Leukocytosis. CKD.

Case 22

60/M-Fever, VZV lesions, cortical atrophy. Leukocytosis. CKD.

Case 23

70/M-Fever, status epilepticus. Bilateral temporal hyperintensities. HSV PCR+. Alcohol use.

Case 24

62/M-Fever, myoclonus, altered sensorium. Bilateral thalamic hyperintensity. Dengue positive.

Case 25

61/F-Fever, altered sensorium, myocarditis. Pancytopenia. ADEM. SLE. Recovered.

Case 26

30/M-Fever, seizure. Normal MRI. Leukocytosis. Unknown etiology.

Case 27

70/M-Fever, diplopia, quadriparesis, respiratory distress. Normal MRI. GQ1b+ (Bickerstaff encephalitis).

Case 28

28/F - Fever, delirium. Normal MRI. HSV PCR+. On antipsychotics.

Case 29

60/F-Fever, altered sensorium, seizure. Cortical atrophy. Leukocytosis. CKD.

Case 30

71/M-Fever, myoclonus, seizure. Chronic changes on MRI. Leukocytosis. CKD.

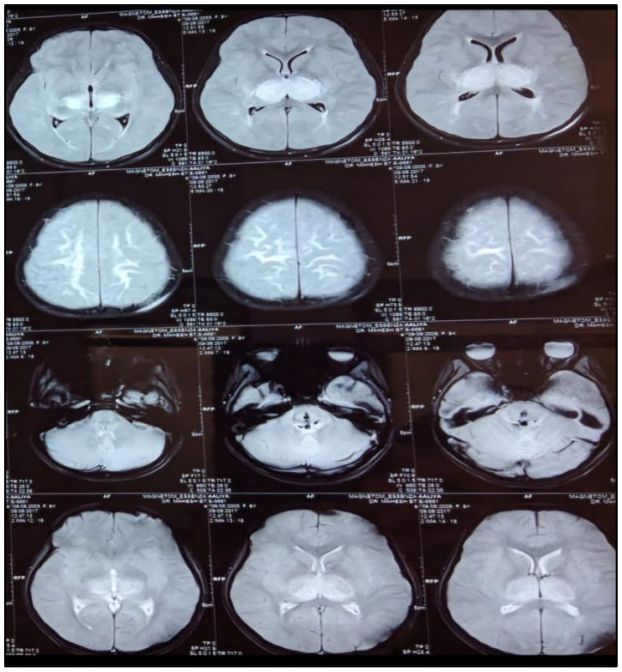

Figure 1: Acute haemorrhagic encephalitis in dengue fever.

Figure 2: Myelitis (Parainfectious)- MRI spin.

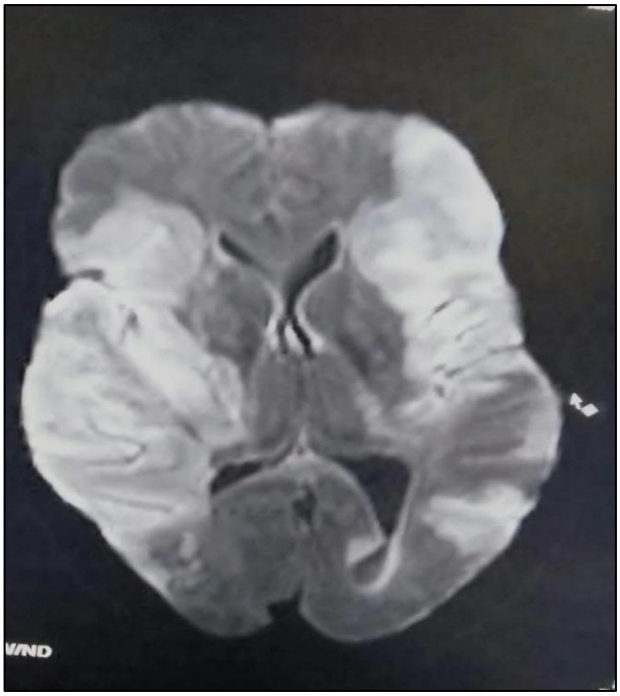

Figure 3: MRI brain-herpes encephalitis.

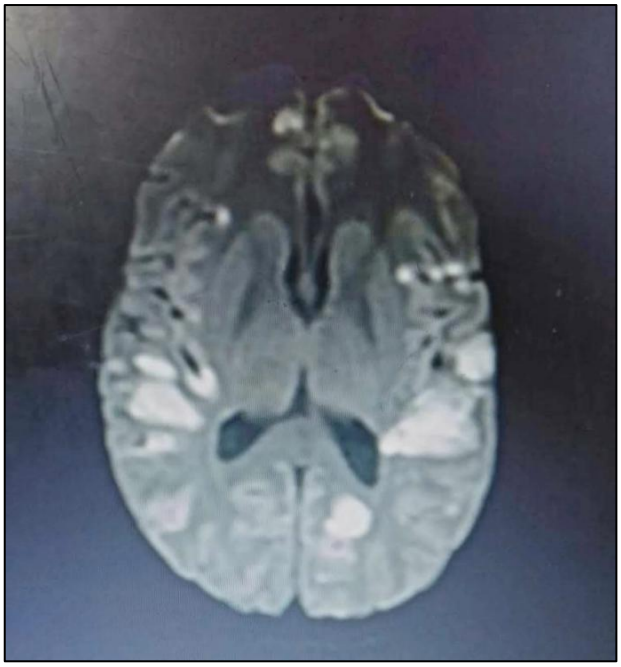

Figure 4: MRI brain in acute disseminated encephalomyelitis.

DISCUSSION

This study emphasizes the heterogeneity of viral encephalitis presentations and the challenges in timely diagnosis. immunocompromised patients, such as those with chronic kidney disease, demonstrated prolonged recovery and required extensive rehabilitation. Comparison with existing literature underscores the need

for early suspicion and comprehensive management to reduce morbidity and mortality.[9,10]

Our study highlights the critical importance of early diagnosis and timely intervention to reduce the morbidity and mortality associated with viral encephalitis. For example, HSV encephalitis is well-recognized as a leading cause globally.[3,7]

Strengthening diagnostic infrastructure and implementing region-specific diagnostic protocols can significantly improve patient outcomes.[6] further research is required to explore novel therapeutic options, especially for cases with unknown etiology or refractory symptoms.[11]

This study underscores the importance of rapid diagnostic tools like Multiplex PCR and Next-generation sequencing (NGS) to identify rare or atypical pathogens.[4,5] Our findings align with global epidemiological data showing HSV and Arboviruses as leading causes, though regional variation necessitates tailored diagnostic protocols.[10] The utility of Neuroimaging in identifying specific patterns of brain involvement, such as thalamic hyperintensities in dengue-related encephalitis, has been demonstrated in prior studies and correlates with our findings.[5]

CONCLUSION

Viral encephalitis is a critical neurological emergency requiring prompt diagnosis and tailored management. Delayed treatment initiation, especially in immunocompromised individuals, was associated with poorer outcomes, underscoring the need for early empirical antiviral therapy.

This study highlights the importance of multidisciplinary care and advanced diagnostic tools in improving outcomes. Further research is required to explore novel therapeutic options, especially for cases with unknown etiology or refractory symptoms.

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