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Research Article

Clinical Presentation, Cerebrospinal Fluid Findings and MRI Patterns in Viral Encephalitis: A Hospital-Based Prospective Study

Upma Gupta[1*], Nikhil Sahu [2], and Divya Saxena[3]

[1-3]Assistant Professor, Department of Pathology Dr B.S. Kushwah Institute of Medical Sciences, Kanpur

[2]Associate Professor, Department Of Neurology, GSVM Medical College,Kanpur

***Corresponding Author**
Dr. Upma Gupta
Nks1103@gmail.com

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Abstract: Background: Viral encephalitis is an important cause of acute encephalopathy and neurological morbidity in adults. The diagnosis is made on the basis of clinical examination, analysis of cerebrospinal fluid (CSF) and magnetic resonance imaging (MRI). **Methods:** It is a prospective hospital-based observational study done on 50 adult patients of viral encephalitis admitted in a tertiary care center. Patients had acute encephalopathy lasting >24 hours with supporting clinical, CSF, or neuroimaging findings. Patients with bacterial, fungal, parasitic, tuberculous and non-infectious encephalopathies were excluded. Clinical features, CSF parameters and MRI patterns were analysed. **Results:** The study included 28 males (56.0%) and 22 females (44.0%) with a mean age of 42.5 years (range: 19-74 years). Fever was present in 48 (96.0%), altered sensorium in 44 (88.0%), headache in 38 (76.0%) and seizures in 30 (60.0%). Mean CSF cell count was 85 cells/mm3 with 90% lymphocytic predominance. Mean CSF protein and glucose were 75 mg/dL and 65 mg/dL, respectively. Abnormal MRI was detected in 35 (70.0%) patients. The most common finding was hyperintensity in the temporal lobe (18 patients, 36.0%). **Conclusion:** Viral encephalitis commonly presented as acute febrile encephalopathy with seizures, lymphocytic CSF pleocytosis, preserved CSF glucose and MRI abnormalities. Temporal lobe involvement was the most frequent MRI finding but nearly one-third of patients had normal initial MRI findings.

Keywords: Viral Encephalitis, Cerebrospinal Fluid, Magnetic Resonance Imaging, Encephalopathy

INTRODUCTION

Encephalitis is an inflammatory process in the brain parenchyma associated with neurological dysfunction especially altered mental status. Infectious causes include viruses, bacteria, fungi, parasites and Mycobacterium tuberculosis, whereas autoimmune, metabolic, toxic, neoplastic and post-infectious disorders may mimic encephalitis. Viral infections are an important cause of encephalitis worldwide and can result in substantial mortality, cognitive dysfunction, behavioural changes, epilepsy and other long-term neurological sequelae [1-4].The International Encephalitis Consortium proposed a standardised case definition in which encephalitis is defined as the presence of altered mental status of 24 hours or greater duration without an identifiable alternative cause, plus supportive clinical, laboratory, electrophysiological, or neuroimaging criteria. Supportive features include fever, generalised or focal seizures, new focal neurological deficits, CSF pleocytosis, abnormal electroencephalography or brain imaging abnormalities consistent with encephalitis [1]. Clinical manifestations depend on the viral agent, immune status of the host, age of the patient and anatomical distribution of inflammation. Fever, headache, altered sensorium, seizures, behavioural abnormalities and focal neurological deficits are frequently reported in acute viral encephalitis [2-4]. These presentations overlap with bacterial meningitis, tuberculous meningitis, fungal encephalitis, autoimmune encephalitis, metabolic encephalopathy and toxic encephalopathy, so a systematic clinical assessment and targeted investigations are needed [1,2,5]. CSF analysis is included in the evaluation. The CSF in viral encephalitis usually shows mild to moderate pleocytosis with a predominance of lymphocytes, normal CSF glucose and normal or mildly elevated CSF protein. However, early infection may have normal CSF leukocyte count. Occasionally viral infections may show neutrophilic pleocytosis initially. Therefore, CSF results should be interpreted according to the duration of illness, neuroimaging and pathogen-specific testing [1-3,6]. MRI is more sensitive than CT in identifying inflammatory parenchymal lesions in encephalitis and can give clues as to the underlying aetiology by the distribution of the lesions. Typical patterns of herpes simplex virus encephalitis are temporal and limbic T2/FLAIR hyperintensities. Japanese encephalitis and other viral infections may show thalamic and basal ganglia involvement. However, MRI may be normal in early disease and a normal baseline scan does not exclude encephalitis [1,3,7-9]. The objective of this study was to report clinical presentation, CSF findings and MRI patterns in adults with viral encephalitis admitted to a tertiary care center.

MATERIALS AND METHODS

Study Design and Setting

This prospective, hospital-based observational study was conducted at a tertiary care center over a defined study period designed to capture seasonal variations in viral central nervous system infections.

Study Population

Fifty patients aged 18 years or above who fulfilled diagnostic criteria for viral encephalitis were included. Encephalitis was defined as acute encephalopathy characterized by altered or depressed level of consciousness lasting 24 hours or more, accompanied by at least two of the following: fever, seizures, focal neurological deficits, CSF pleocytosis, or neuroimaging findings compatible with encephalitis. The criteria were adapted from the International Encephalitis Consortium case definition.

Patients with bacterial, fungal, parasitic, or tuberculous central nervous system infection were excluded. Patients with non-infectious encephalopathy, including metabolic, toxic, autoimmune and other alternative causes, were also excluded.

Data Collection

A detailed clinical history, including the onset and progression of symptoms, was obtained from each participant or an appropriate caregiver. Demographic data included age and sex. Clinical variables included fever, headache, altered sensorium, seizures, vomiting and focal neurological deficits.

Lumbar puncture was performed in all patients. CSF analysis included total leukocyte count, differential leukocyte count, protein concentration and glucose concentration. MRI brain was performed using either a 1.5-Tesla or 3-Tesla MRI system. T2-weighted and FLAIR sequences were reviewed for hyperintensities involving the temporal lobes, thalamus, basal ganglia and cerebral cortex.

Statistical Analysis

Categorical variables are presented as number and percentage. Continuous variables are presented as mean with range or predominant pattern.

RESULTS

A total of 50 patients who met the diagnostic criteria for viral encephalitis were included in the final analysis. The demographic profile showed a slight male predominance, with 28 males (56.0%) and 22 females (44.0%). The mean age of the study population was 42.5 years (range: 19 - 74 years).

Clinical Profile

The clinical onset was predominantly acute. Fever was the most consistent symptom, recorded in 48 patients (96.0%). A defining feature of the cohort was altered sensorium, present in 44 patients (88.0%). Seizures, which were primarily generalized tonic-clonic but occasionally focal, occurred in 30 patients (60.0%) Table 1.

CSF Analysis

Lumbar puncture was safely performed in all patients. The CSF findings were characteristic of a viral etiology. The mean leukocyte count was moderately elevated at 85 cells/mm^3, with a strong lymphocytic predominance (mean 90%). Crucially, CSF glucose levels remained normal (mean 65 mg/dL) and protein levels were only mildly to moderately elevated (mean 75 mg/dL) Table 2.

Table 1: Clinical Presentation of Patients with Viral Encephalitis

Clinical Feature	Number of Patients (n = 50)	Percentage
Fever	48	96.0
Altered Sensorium / Encephalopathy	44	88.0
Headache	38	76.0
Seizures	30	60.0
Vomiting	22	44.0
Focal Neurological Deficits	12	24.0

Table 2: Cerebrospinal Fluid (CSF) Characteristics

CSF Parameter	Mean Value	Range / Predominance
Total Cell Count (cells/mm^3)	85	10 - 320
Lymphocyte Percentage (%)	90	70 - 100
Protein (mg/dL)	75	45 - 150
Glucose (mg/dL)	65	50 - 95

Table 3: MRI Brain Patterns (T2/FLAIR Hyperintensities)

MRI Finding (Location)	Number of Patients (n = 50)	Percentage
Temporal Lobe(s)	18	36.0
Normal Initial MRI	15	30.0
Thalamus / Basal Ganglia	10	20.0
Diffuse / Cortical Edema	7	14.0

Radiological Profile

MRI brain scans were pivotal in evaluating parenchymal involvement. While 15 patients (30.0%) had normal MRI findings upon initial presentation, the remaining 70.0% exhibited significant abnormalities. Temporal lobe hyperintensities (frequently bilateral but asymmetrical) were the most common structural finding, noted in 18 patients (36.0%) Table 3.

DISCUSSION

Discussion

This was a prospective study in 50 adults with viral encephalitis, with a slight male predominance. The mean age was 42.5 years old suggesting that viral encephalitis affected a wide range of adult ages. The demographic profile of encephalitis differs between populations as disease occurrence is affected by the causative pathogen, vector activity, host factors, geography and season [1,3,4]. Fever was observed in 96.0% and altered sensorium in 88.0% of the study population. These findings fit within the established clinical paradigm of encephalitis, in which the central clinical requirement is protracted altered mental status, with fever being an important supporting feature. Fever, seizures, focal neurologic deficits, CSF inflammation and abnormalities in neuroimaging may further support brain inflammation in patients with encephalopathy [1]. Venkatesan *et al.* emphasised.Headache was reported by 76.0% of subjects and seizures were reported by 60.0%. Seizures can be generalised or focal. They are usually due to cortical irritation from inflammatory injury to the brain. "Acute viral encephalitis is usually characterised by seizures, altered mental status, headache and focal neurological abnormalities," Tyler said. In a similar note, Venkatesan and Geocadin emphasised the significance of seizure and altered consciousness as urgent neurological manifestations that require prompt investigation and supportive management [2,3,10]. The average CSF white blood cell count was 85 cells/mm^3 with a marked lymphocytic predominance of 90%. The mean CSF protein was 75 mg/dL, suggestive of mild-to-moderate protein elevation and CSF glucose was relatively preserved (65 mg/dL). This profile is consistent with viral encephalitis in which the CSF typically shows a lymphocytic pleocytosis, normal glucose concentration and variable protein elevation [1-3,6]. Solomon *et al.* also reported lymphocytic pleocytosis with normal glucose as a typical pattern of CSF in viral encephalitis [11]. However, CSF findings alone are not enough to diagnose a viral aetiology and microbiological testing, especially CSF nucleic acid amplification testing when available, is required to identify specific viral pathogens [2,3,11]. MRI abnormalities were found in 70.0% of patients. The most common MRI pattern was temporal lobe hyperintensities (36.0% of the cohort). Temporal and limbic signal abnormalities on T2-weighted and FLAIR sequences are particularly associated with herpes simplex virus encephalitis, but imaging patterns need to be interpreted with clinical presentation and virological testing, as MRI is not pathogen-specific in isolation [3,7]. Tyler highlighted the temporal lobe involvement as a common radiological pattern in herpes simplex virus encephalitis [4]. Granerod *et al.* also found good agreement among neuroradiologists for radiological identification of herpes simplex virus encephalitis, particularly when there were abnormalities in the temporal and frontal lobes [8,12]. Thalamic or basal ganglia hyperintensities were observed in 20.0% of patients. Involvement of deep grey matter is a known MRI pattern of viral encephalitis and may be indicative of particular viral aetiologies, particularly Japanese encephalitis in the correct epidemiological context [8]. Diffuse or cortical oedema was observed in 14.0% of patients and suggested more widespread inflammatory brain involvement [10]. In 30.0% of patients, the initial MRI was normal. This has clinical importance as the absence of MRI abnormalities at presentation does not exclude the diagnosis of viral encephalitis [1,2,7,9]. Granerod *et al.* [12] reported that aetiology and time of imaging influence imaging performance and interpretation. Diagnostic decisions in suspected encephalitis should combine clinical features, CSF results, EEG where indicated, neuroimaging and microbiological testing, recommend Venkatesan and Geocadin [10]. The limitation of this study is the lack of pathogen-specific CSF PCR or serological data to correlate specific clinical and MRI patterns with specific viruses. EEG data, treatment details, serial imaging and clinical outcomes were not available. The MRI distribution patterns should be interpreted with consideration of these limitations.

CONCLUSION

Viral encephalitis in adults commonly presented as acute febrile encephalopathy with headache and seizures. The CSF profile was lymphocytic pleocytosis, mildly elevated protein and preserved glucose concentration. Most patients had an

abnormal MRI, with the predominant pattern being hyperintensities of the temporal lobe; however, 30.0% of patients had normal initial MRI findings. The accurate diagnosis of aetiology and early management requires the integration of clinical findings, CSF analysis, MRI and pathogen-specific microbiological testing.

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