ORIGINAL ARTICLE

Clinical, Radiological, and Cerebrospinal Fluid Characteristics of Patients with Acute Viral Encephalitis: A Series of 93 Cases

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Abstract:

Introduction: Acute viral encephalitis (AVE) is a severe neurological condition associated with significant morbidity and mortality, often caused by neurotropic viruses such as herpes simplex virus (HSV) and Japanese encephalitis virus (JEV). Early recognition is challenging due to overlapping clinical features with other central nervous system disorders and variable laboratory and imaging findings. Objective: To describe the demographic characteristics, clinical presentations, neuroimaging findings, and cerebrospinal fluid (CSF) parameters of patients with clinically suspected AVE in a tertiary care hospital in Bangladesh. Methods: This observational study was carried out at the Department of Neurology during December-2024 to November-2025 in, Chittagong Medical College Hospital (CMCH), Chattogram, Bangladesh. A total of 93 patients irrespective of age and sex with clinically suspected acute viral encephalitis (AVE) were consecutively enrolled during the study period. Clinical evaluation, laboratory investigations, MRI, and CSF analysis were performed for all patients. Data were analyzed usingStatistical Package for Social Sciences(SPSS) version 23.0.Ethical approval for the study was obtained from the Institutional Review Board (IRB) of Chittagong Medical College, and confidentiality of all patients' information was strictly maintained throughout the research process in accordance with the Declaration of Helsinki-1964. Results: The mean age of the study patients was 45.87 ± 20.13 years, with nearly equal gender distribution (52.7% male). Fever (100%) and altered consciousness (96.8%) were the most common clinical features, followed by confusion (80.7%), headache (54.8%), and vomiting (51.6%). MRI abnormalities were observed in 52.7% of patients, including restricted diffusion (50.5%), FLAIR T2 changes (50.5%), and hypodense areas of hypoxia (43%). Focal neurological deficits were present in 22.6% patients, and extensor plantar response predominated in (65.6%) patients. CSF analysis revealed a median glucose of 78 mg/dL, CSF/blood glucose ratio of 0.54, protein 53.7 mg/dL, RBC count 550 cells/µL, leukocyte count 8 cells/µL, neutrophils 10%, and lymphocytes 98%. Conclusion: Patients with suspected AVE in this setting presented with non-specific but severe neurological manifestations, with approximately half showing MRI abnormalities. CSF analysis provided supportive evidence of viral etiology, emphasizing the importance of integrating clinical, imaging, and laboratory data for early diagnosis and management.

Key words: Acute viral encephalitis, cerebrospinal fluid, MRI, clinical features, Bangladesh, observational study

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Introduction

Acute viral encephalitis (AVE) is a life-threatening neurological condition characterized by inflammation of the brain parenchyma in response to viral infection, leading to significant global morbidity and mortality. Despite improvements in neurocritical care and diagnostic methods, AVE continues to pose a major public health challenge, particularly in tropical and subtropical regions where vector-borne viruses are endemic[1,2]. A wide spectrum of neurotropic viruses, such as herpes simplex virus (HSV), varicella-zoster virus (VZV), enteroviruses, adenoviruses, Epstein Barr virus (EBV), and arboviruses including Japanese encephalitis virus (JEV) and West Nile virus (WNV) are implicated in its pathogenesis[3,4]. Among these, HSV remains one of the most common causes of sporadic encephalitis worldwide, while JEV accounts for a substantial proportion of cases in Asia[5].Clinically, AVE often begins with non-specific symptoms such as fever, headache, and vomiting, followed by altered mental status, seizures, and focal neurological deficits as the disease progresses[6]. These manifestations frequently overlap with other central nervous system (CNS) disorders, including bacterial meningitis, cerebral malaria, autoimmune encephalitis, and metabolic encephalopathies making early clinical diagnosis challenging[7]. Furthermore, the variability in clinical presentation between different viral etiologies complicates decision-making, particularly in settings where advanced diagnostic technologies are limited. Cerebrospinal fluid (CSF) examination remains a cornerstone of evaluation, but classical viral patterns (lymphocytic pleocytosis, mild protein elevation, normal glucose) may not always be present, especially in early disease or in immunocompromised patients[8]. Neuroimaging, especially magnetic resonance imaging (MRI), plays a pivotal role, yet radiological abnormalities are absent in a considerable proportion of patients, leading to diagnostic uncertainty. Molecular diagnostic methods, particularly multiplex PCR (mPCR), have significantly improved pathogen detection and have become essential for confirming viral etiologies and guiding targeted management[9]. However, their availability and affordability remain limited in many low-resource healthcare settings, forcing clinicians to rely heavily on clinical suspicion. Understanding the demographic and clinical characteristics of patients with suspected AVE is crucial for improving timely recognition, initiating early empiric therapy, and strengthening diagnostic pathways. Such data are especially important in regions with evolving viral epidemiology, shifting population immunity, and changing environmental conditions that influence disease transmission[10,11]. Despite its clinical relevance, there is limited context-specific evidence describing the characteristics of patients presenting with suspected AVE in tertiary care settings. This study aims to address this gap by examining the demographic characteristics, clinical presentations, neuro-imaging findings, and cerebrospinal fluid (CSF) parameters of patients with clinically suspected AVE in a tertiary care hospital in Bangladesh. By documenting their symptoms, neurological findings, imaging results, and laboratory parameters, this work seeks to contribute to improved clinical awareness and support future research and policy strategies for more effective encephalitis management.

Methodology:

This observational study was carried out at the Department of Neurology during December-2024 to November-2025 in, Chittagong Medical College Hospital (CMCH), Chattogram, Bangladesh. A total of 93 patients irrespective of age and sex with clinically suspected acute viral encephalitis (AVE) were consecutively enrolled during the study period. Patients with confirmed bacterial meningitis, metabolic encephalopathy, pre-existing chronic neurological disorders, or traumatic brain injury were excluded from this study. After obtaining

informed written consent, Routine laboratory and clinical investigations were performed for all patients following standard procedures. MRI was performed on all patients and findings were categorized as normal or abnormal and were further assessed the clinical features associated with MRI findings. Then all 93 patients underwent cerebrospinal fluid (CSF) analysis following lumbar puncture under aseptic conditions. CSF parameters measured included glucose level, CSF-to-blood glucose ratio, total protein, red blood cell (RBC) count, leukocyte count, and differential cell percentage (neutrophils and lymphocytes). Values were summarized using median and interquartile range (IQR). All clinical, laboratory, neuroimaging and demographic data were collected using a restructured case record form(CRF). Collected data were analyzed using Statistical Package for Social Sciences(SPSS), version 23.0 (IBM Corp., Armonk, NY, USA). Continuous data were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR), depending on their distribution. Categorical data were summarized as frequencies and percentages, and results were presented in tables and charts. Ethical approval for the study was obtained from the Institutional Review Board (IRB) of Chittagong Medical College, and confidentiality of all patients' information was strictly maintained throughout the research process in accordance with the Declaration of Helsinki-1964.

Results:

A total of 93 patients with clinically suspected acute viral encephalitis were included in the study. The majority (23.7%) patients were aged <26 years, followed by those aged ≥67 years (20.4%), 27–36 years (15.1%), 47–56 years (15.1%), 37–46 years (14%), and 57–66 years (11.8%). The mean age of the participants was 45.87 ± 20.13 years, with a median age of 43 years, mode of 65 years, and age range of 17–85 years. The gender distribution was nearly equal, with males comprising 49 (52.7%) and females 44 (47.3%) of the study population (Table 1).The mean vital signs recorded at presentation were: pulse rate 101.34 ± 17.72 beats/min, respiratory rate 22.32 ± 5.67 breaths/min, temperature 38.70 ± 1.08 °C, systolic blood pressure 121.07 ± 29.20 mmHg, and diastolic blood pressure 74.13 ± 15.68 mmHg. Laboratory assessments showed mean hemoglobin level 12.07 ± 1.73 g/dL, WBC count 11.43 ± 4.44 × 10^3/μL, random blood sugar 157.64 ± 64.98 mg/dL, serum creatinine 0.98 ± 0.44 mg/dL, sodium 138.20 ± 8.09 mmol/L, and potassium 3.84 ± 0.53 mmol/L. The mean Glasgow Coma Scale scores were: eye response 2.80 ± 1.04, motor response 4.07 ± 1.14, and verbal response 3.15 ± 1.28 (Table 2).Comorbidities were absent in 53 (57%) patients. Among those with comorbidities (n=40, 43%), hypertension was the most common (24.7%), followed by diabetes mellitus (15.1%), and ischemic heart disease (3.2%) (Table 3).Fever was present in all patients 93 (100%). Other common clinical presentations included altered consciousness (96.77%), confusion (80.65%), headache (54.84%), vomiting (51.61%), photophobia (45.16%), seizures (38.71%), and status epilepticus (11.83%) (Table 4).MRI findings were abnormal in 49 (52.69%) patients, while 44 (47.31%) showed no abnormalities. According to clinical associated features with MRI, focal neurological deficits were documented in 21 (22.58%) patients. Plantar responses were predominantly extensor in 61 (65.59%) patients. Oedema was present in 25 (26.89%), restricted diffusion in 47 (50.54%), hypodense areas of hypoxia in 40 (43.01%), FLAIR T2 abnormalities in 47(50.54%), and contrast enhancement in 20 (21.50%) (Table 5).CSF biochemical and cytological parameters demonstrated the following median (IQR) values: CSF glucose 78 mg/dL (67–92), CSF/blood glucose ratio 0.54 (0.43–0.67), CSF protein 53.7 mg/dL (39.8–70.65), CSF RBCs 550 cells/μL (225–1000), CSF leukocytes 8 cells/μL (5–15), CSF neutrophils 10% (10–30), and CSF lymphocytes 98% (90–100) (Table 6).

Table 1: Age and sex distribution of the study patients (N=93).

Age groups(years)	**Frequency**	**Percentage**
<26	22	237
27-36	14	15.1
37-46	13	14
47-56	14	15.1
57-66	11	11.8
≥ 67	19	20.4
Total	93	100.0
Age: (Mean ±SD) (years):	45.87±20.13	
Median	43	
Mode	65	
Range	17-85	
Gender:		
Male	49	52.7
Female	44	47.3

Table 2: Distribution of vital signs and laboratory parameters (N=93).

Vital Signs	**Mean ± SD**
Pulse rate (beats/min)	101.34 ± 17.72
Respiratory rate (breaths/min)	22.32 ± 5.67
Temperature (°C)	38.70 ± 1.08
Systolic BP (mmHg)	121.07 ± 29.20
Diastolic BP (mmHg)	74.13 ± 15.68
Laboratory Parameters	
Hemoglobin (g/d	12.07 ± 1.73
WBC ($\times 10^3/\mu L$)	11.43 ± 4.44
RBS (mg/dL)	157.64 ± 64.98
Serum Creatinine (mg/dL)	0.98 ± 0.44
Serum Electrolytes	
Sodium (mmol/L)	138.20 ± 8.09
Potassium (mmol/L)	3.84 ± 0.53
Glasgow Coma Scale (GCS)	
Eye response	2.80 ± 1.04
Motor response	4.07 ± 1.14
Verbal response	3.15 ± 1.28

Table 3: Distribution of comorbidities (N=100).

Distribution of comorbidities	Frequency	Percent
Absent	53	57
Present:	40	43
Hypertension(HTN)	23	24.7
Diabetes Mellitus(DM)	14	15.1
Ischemic HeartDisease(IHD)	3	3.2

Table 4: Distribution of clinical presentation (N=100).

Clinical presentation	Frequency	Percent
Fever	93	100
Headache	51	54.84
Vomiting	48	51.61
Seizure	36	38.71
Status epilepticus	11	11.83
Altered Consciousness	90	96.77
Confusion	75	80.65
Photophobia	42	45.16

Table 5: Distribution of MRI findings and associated clinical features (N=100).

Clinical findings	Frequency	Percent
MRI Appurtenance		
Normal	44	47.31
Abnormal	49	52.69
Focal deficit		
Absent	72	77.42
Present	21	22.58
Planter Response		
Flexor	32	34.41
Extensor	61	65.59
Oedema		
Absent	68	73.11
Present	25	26.89
Restricted diffusion		
Absent	46	49.46
Present	47	50.54

Clinical findings	Frequency	Percent
Hypodense areas of hypoxia		
Absent	53	56.99
Present	40	43.01
FLAIR T2 abnormalities		
Absent	46	49.46
Present	47	50.54
Contrast enhancement		
Absent	73	78.50
Present	20	21.50

Table 6: Distribution of CSF biochemical and cytological parameters (N=100).

CSF Parameter	N	Median (IQR)
CSF Glucose (mg/dL)	93	78 (67-92)
CSF/Blood Glucose Ratio	93	54 (43-67)
CSF Protein (mg/dL)	93	53.7 (39.8-70.65)
CSF RBCs (cells/µL)	93	550(225–1000)
CSF Leukocytes (cells/µL)	93	8 (5–15)
CSF Neutrophils (%)	93	10 (10-30)
CSF Lymphocytes (%)	93	98 (90–100)

Median (IQR) represents median with interquartile range (25th–75th percentile).

Discussion:

This study provides a comprehensive assessment of the demographic, clinical, radiological, and cerebrospinal fluid (CSF) characteristics of patients with clinically suspected acute viral encephalitis. The age distribution showed a distinct concentration among younger adults (<26 years) and older individuals (≥67 years). This bimodal pattern is consistent with previous Bangladeshi data that likewise identified early adulthood and advanced age as periods of heightened vulnerability to neuroinvasive viral infections[12]. South Asian studies from India and Nepal have similarly reported that younger adults and the elderly represent major risk groups, likely due to differing immune dynamics and increased exposure risks in developing regions[13,14]. European data also reflect a broad age susceptibility, though peaks often occur in slightly older age groups due to demographic and etiological differences[15]. The nearly equal male–female ratio in our findings corresponds with several regional and Western studies, indicating that sex does not significantly influence susceptibility to viral encephalitis in most settings[15,16]. The clinical features observed in this cohort—particularly fever (100%), altered consciousness (96.77%), and confusion (80.65%)—are typical of viral encephalitis and are in strong agreement with Bangladeshi hospital-based findings, where fever and impaired consciousness were nearly universal[16]. Likewise, neurological symptoms such as headache, vomiting, photophobia, and seizures are frequently seen in South Asian encephalitis cases, reflecting meningeal irritation and cortical involvement, as reported in Sri Lanka and India[17,18].

Seizures occurred in 38.71% of our patients, which corresponds closely with regional data reporting seizure rates of 30–45%[18]. The rate of status epilepticus (11.83%) is of clinical significance, as it matches findings from European multicentre analyses where approximately 10–15% of viral encephalitis patients experience severe seizure activity[19], highlighting the global relevance of early seizure control. Radiologically, MRI abnormalities were identified in 52.69% of the patients, indicating moderate diagnostic yield. This pattern aligns with Bangladeshi studies that also reported abnormal MRI in about half of encephalitis cases, reflecting the variable nature of viral CNS involvement[20]. Restricted diffusion and FLAIR/T2 abnormalities were the most common imaging findings in our cohort (50.54% each). Similar neuroimaging manifestations have been emphasized in Indian and European studies, where diffusion restriction and FLAIR hyperintensity are recognized hallmarks of viral neuronal injury, particularly in herpesvirus-associated encephalitis[21–23]. The presence of oedema (26.89%) and contrast enhancement (21.50%) further supports active inflammation, comparable to reports from India and Europe showing 20–30% frequency of these features[22,23]. Additionally, focal neurological deficits were documented in 22.58% of our study population, echoing the findings from previous Bangladeshi and regional studies that reported focal deficits in roughly one-quarter of encephalitis patients[24]. The CSF parameters documented in this study support a predominantly viral etiology. The mild leukocytosis (median 8 cells/μL) with strong lymphocytic predominance (median 98%) is characteristic of viral CNS infections and closely matches the cytological profile described in Bangladeshi and Indian cohorts[25]. The CSF protein elevation (median 53.7 mg/dL) falls within the typical viral range, lower than expected in bacterial infections but higher than normal physiological levels. The CSF/blood glucose ratio (median 0.54) also remained within the range commonly noted in viral encephalitis across South Asian and European studies, suggesting intact glucose transport despite CNS inflammation[25,26]. Collectively, these CSF profiles reinforce the likelihood of viral etiology and underscore the diagnostic value of combining biochemical and cytological markers in low-resource settings. Overall, the findings of this study are highly consistent with existing Bangladeshi, South Asian, and European evidence. Despite regional differences in viral epidemiology, the clinical, imaging, and CSF patterns observed show substantial overlap with global trends. This alignment strengthens the clinical confidence in the diagnostic approach used and highlights the need for continued emphasis on early recognition, neuro imaging, and CSF analysis to improve diagnostic accuracy in suspected viral encephalitis.

Limitations:

This study was limited by its single-center design and relatively small sample size, which may affect generalizability. Viral confirmation by PCR or serology was not available for all patients, potentially leading to misclassification. Advanced neuroimaging and follow-up outcome data were also limited. Despite these constraints, the study provides important insights into the clinical, CSF, and imaging profiles of viral encephalitis in Bangladesh.

Conclusion:

In this study of 93 patients with clinically suspected viral encephalitis, fever, altered consciousness, and confusion were the most common clinical features. MRI abnormalities were present in about half of the patients, with restricted diffusion and FLAIR/T2 changes being predominant. CSF analysis showed lymphocytic pleocytosis and mildly elevated protein, consistent with viral etiology. These findings highlight the importance of combined clinical assessment, neuroimaging, and CSF evaluation for early diagnosis and management of viral encephalitis in Bangladesh.

Recommendations:

Early recognition of viral encephalitis is crucial to improve patient outcomes. We recommend routine use of combined clinical assessment, MRI, and CSF analysis in suspected cases. Strengthening molecular diagnostic capacity for viral identification and establishing standardized treatment protocols in tertiary centers can further enhance timely diagnosis and management.

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