

Epstein–Barr Virus Meningoencephalitis Presenting with Extrapyraxidal Symptoms in an Immunocompetent Adult Female: A Rare Case Report

Case Report

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Abstract

Epstein–Barr virus (EBV) is an infrequent yet important cause of meningoencephalitis, especially in immunocompetent individuals. [1] We present young patient, previously healthy woman who developed progressive neuropsychiatric and motor symptoms following a ritualistic holy dip. Her course was complicated by rural tremors, dystonia, rigidity, and quadriplegia. Despite broad-spectrum antimicrobial and antiviral therapy, the clinical response was initially limited. CSF polymerase chain reaction (PCR) ultimately revealed EBV as the causative pathogen. The patient underwent prolonged hospitalization with supportive neuro-rehabilitative care and gradual clinical recovery. This case emphasizes the need for high suspicion of EBV in atypical neuro-infections and the importance of early PCR-based diagnosis.

Introduction

[1] Epstein–Barr virus (EBV), a member of the Herpesviridae family, is among the most prevalent human viruses globally, with over 90% of the adult population estimated to be seropositive. [2] Though primarily recognized for causing infectious mononucleosis, EBV is also associated with a variety of malignancies, autoimmune conditions, and—less commonly—central nervous system (CNS) disorders. [3] Neurological manifestations of EBV are under reported in the immunocompetent population due to their subtle presentation and diagnostic challenges. Nevertheless, when CNS involvement does occur, the consequences can be severe and long-lasting. [4] EBV has the capacity to infect both epithelial cells and B lymphocytes, leading

to latent infection within the host. [5] The neurological sequelae may stem from direct viral invasion of the CNS or, more commonly, from immune-mediated processes. The virus can enter the CNS via hematogenous spread or through retrograde axonal transport. The resulting clinical syndromes can include encephalitis, meningitis, cerebellitis, myelitis, cranial nerve palsies, and movement disorders such as parkinsonism or opsoclonus-myoclonus. In some cases, acute disseminated encephalomyelitis (ADEM) or Guillain–Barré syndrome may also occur.

Case Presentation

A 24-year-old lady, recently married, presented with subacute neurological symptoms for 15 days after participating in a religious

holy dip at Murudeshwar. Initially asymptomatic, she developed high-grade fever with chills that persisted for over a week. This was followed by the insidious onset of severe diffuse headache, giddiness, and visual disturbances characterized by blurring and diplopia. Over the next few days, she experienced progressive unsteadiness of gait, behavioral changes including altered sensorium, and involuntary movements of the limbs.

She had no history of seizures, trauma, animal bites, recent vaccination, skin rash, ear discharge, herpes zoster or arthralgia. There was no significant past medical and surgical history. The symptoms progressed to include dysarthria, emotional lability, and difficulty in performing daily activities, prompting hospital admission.

Clinical Examination

On admission, she was febrile with a pulse rate of 120 bpm and blood pressure of 120/80 mmHg. She had a normal respiratory rate and oxygen saturation at room air. General examination revealed evidence of purulent vaginal discharge and lax anal sphincter are raising concern for a systemic or neurogenic cause.

- Neurologically, she was confused, with a 'Glasgow Coma Scale' score of 14 (E4 V4 M6). Notably, she had dysarthria and dysphonia, though there was no facial asymmetry. Her optic fundi were normal with no papilledema, and pupils were reactive. She exhibited opsoclonus, rubral tremors, finger dystonia, generalized rigidity, and quadriparesis with a Medical Research Council (MRC) grade of 3–4/5. Deep tendon reflexes were brisk, plantar responses were extensor bilaterally, and neck stiffness was present—suggestive of meningeal irritation.

Investigations

- **Routine Laboratory Workup** revealed low haemoglobin (10.3 g/dl), Leucocytosis (Total leucocyte count of 26,100/cumm) raised ESR of 36 mm/hr. Liver and renal function tests within normal limits.
- **CSF Analysis** revealed lymphocytic pleocytosis (total cells-40), normal protein and glucose levels. Meningitis panel is Positive for Epstein–Barr Virus DNA by PCR and antibodies for other viral markers were not detected
- **Electrophysiological Studies:** EEG revealed generalized slowing with diffuse theta and delta activity, Visual Evoked Potentials are within normal limits, Reduced amplitude of V wave with normal interpeak intervals on auditory evoked potential. Somatosensory Evoked Potentials (SSEP) revealed delayed central conduction in tibial nerve (Right 39.0 ms, Left 33.0 ms).

Treatment

The patient was started on empirical treatment for meningoencephalitis: Antibacterial (Ceftriaxone+Ampicillin), antiviral (Acyclovir), Levodopa+carbidopa and trihexyphenidyl for extrapyramidal symptoms, prophylactic antiepileptic and anticoagulation therapy as a part of DVT prophylaxis.

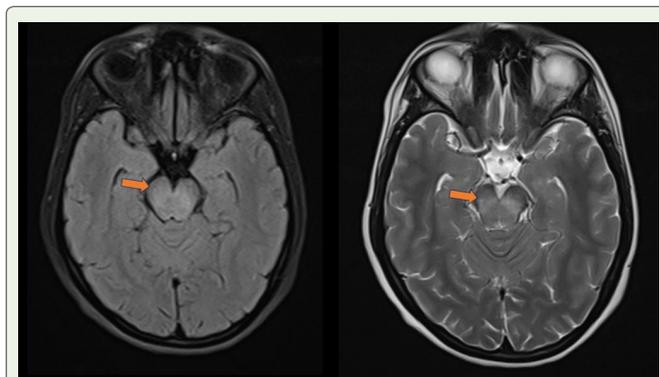


Figure 1: MRI Brain(T2/FLAIR): Bilateral symmetric hyperintense lesions involving midbrain and substantia nigra.

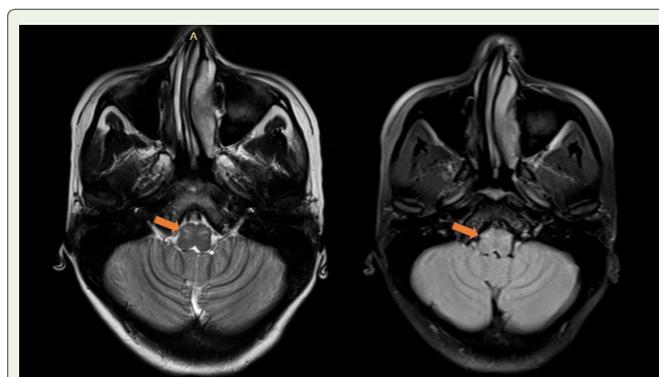


Figure 2: MRI Brain(T2/FLAIR): Bilateral hyperintense lesions involving superior colliculus of midbrain.

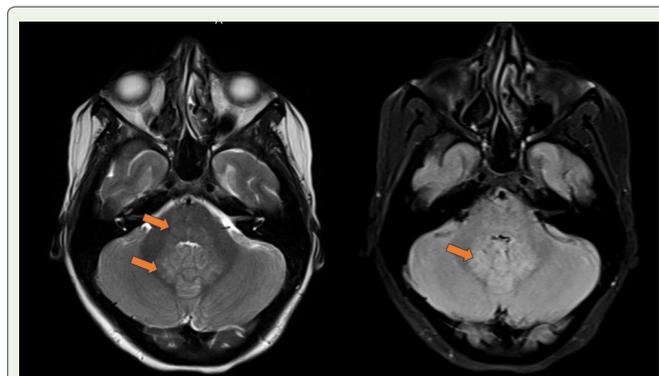


Figure 3: MRI Brain(T2/FLAIR): Hyperintense lesion involving vermis of cerebellum.

Despite treatment, her symptoms progressed initially with persistent bulbar signs and behavioural abnormalities. Over several weeks, multidisciplinary care, physiotherapy, and careful tapering of medications led to steady improvement.

Discussion

This case report illustrates the varied and complex presentation of 'Epstein–Barr virus meningoencephalitis' in an immunocompetent adult. The patient's symptoms began with a febrile prodrome and

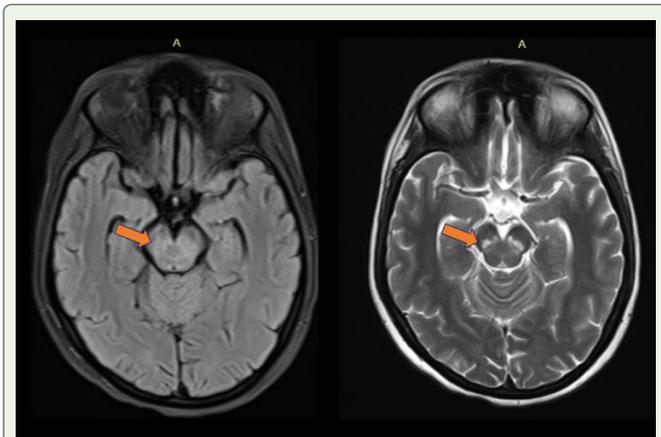


Figure 4: MRI Brain(T2/FLAIR): Bilateral symmetrical hyperintense lesion showing atrophy and calcification of substantia nigra .

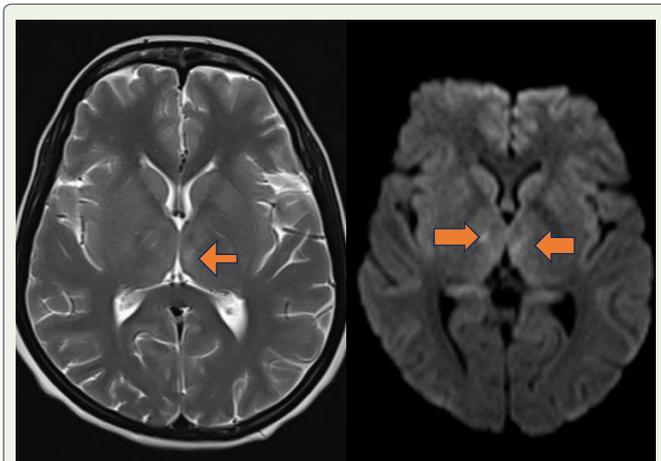


Figure 5: MRI Brain(T2/DWI): Hyperintense lesion on bilateral thalami (left>right) .

progressed to severe neurobehavioral, extrapyramidal, and bulbar manifestations. There is no systemic evidence of Epstein Barr virus infection in our patient. EBV-related CNS involvement is relatively rare in adults with intact immunity, its consequences can be devastating, particularly when diagnosis and targeted supportive care are delayed. [6]

The extrapyramidal signs—including rubral tremors, rigidity, and dystonia—suggest involvement of the basal ganglia or midbrain tegmentum, a region frequently implicated in EBV encephalitis, as documented in case series and radiological reviews. Opsoclonus and dysarthria point toward cerebellar and brainstem involvement. Behavioural disturbance like altered sensation and mood disturbance suggests the involvement of thalamus (supratentorial region). These diverse neuroaxis involvements reinforce the idea that EBV-associated encephalitis does not follow a single anatomic pattern, unlike HSV which typically involves the temporal lobes, or Japanese encephalitis which has predilection for the thalami and substantia nigra. Although there are nonspecific manifestations like vaginal discharge and lax anal sphincter, they may be a secondary infection

and most unlikely in Epstein barr virus infection where vulval ulcers are more common.

The pathophysiology is believed to be multifactorial, involving both direct viral invasion and immunologically mediated damage. EBV can disrupt the blood-brain barrier either directly or through cytokine-mediated inflammation, allowing infiltration of activated lymphocytes that contribute to neuronal injury. Molecular mimicry and the generation of autoreactive antibodies are also hypothesized mechanisms for the immune-mediated component, which may explain some of the persistent symptoms even after viral clearance.

In our case report, diagnostic confirmation was achieved through CSF PCR, which remains the gold standard for identifying active EBV infection in the CNS. This emphasizes the importance of including EBV in multiplex PCR panels when evaluating undifferentiated viral meningoencephalitis.

Treatment is typically supportive. Although acyclovir was administered due to its activity against herpesviruses, evidence for its efficacy against EBV in CNS infection remains limited. The use of Levodopa and Trihexyphenidyl in this case was targeted at controlling extrapyramidal symptoms. Adjunctive therapies for movement disorders and psychotropic interventions for behavioural disturbances are frequently required.

The patient's prolonged hospital stay and slow neurological recovery reflect the chronic nature of post-viral sequelae. Behavioural disturbances including mood lability and pseudobulbar features, along with mild gait impairment, persisted for months despite overall improvement in motor power and coordination. These long-term outcomes are consistent with literature reports indicating that up to 25–40% of patients with EBV encephalitis may have residual neuropsychiatric symptoms.

Radiological follow-up, though not detailed in this case, can show resolution or progression to cerebral atrophy depending on the severity of the initial insult. Rarely, EBV encephalitis may lead to complications such as brainstem haemorrhage, subdural empyema, or transformation to CNS lymphoma, especially in immunocompromised patients.

Comparative literature:

- **N Soni et al. (2023)** documented pediatric CNS involvement by EBV, showing that extrapyramidal signs and cerebellar dysfunction were among the presenting features, which were also evident in our adult patient. [12]
- **Huang et al. (2021)** described a rare case of EBV encephalitis complicated by brainstem hemorrhage in an immunocompetent adult, highlighting the need for high clinical suspicion and prolonged monitoring.[13]
- **S Vyas et al. (2020)** summarized radiological manifestations of EBV encephalitis, noting basal ganglia and thalamic involvement as key MRI features, consistent with our patient's clinical profile. [14]

This case report exemplifies how Epstein–Barr virus can cause severe CNS disease even in healthy individuals. The atypical

presentation, delayed diagnosis, and prolonged recovery underscore the need for greater awareness of this entity among neurologists, intensivists, and infectious disease specialists. Comprehensive evaluation including CSF PCR, neuroimaging, evoked potentials, and multidisciplinary rehabilitation is essential for optimal outcomes.

Conclusion

Epstein–Barr virus (EBV) meningoencephalitis, though rare in immunocompetent individuals, can present with severe and atypical neurological features such as extrapyramidal symptoms, opsoclonus, and behavioral changes. This case report highlights the importance of early CSF PCR testing for diagnosis and the need to consider EBV in the differential diagnosis of unexplained meningoencephalitis. Despite an initially poor response, the patient showed gradual improvement with supportive care, symptomatic therapy, and rehabilitation. Long-term follow-up is crucial, as neuropsychiatric sequelae may persist. A high index of suspicion and multidisciplinary care are essential for timely diagnosis and favourable outcomes in such complex cases.

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