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Postinfectious Cerebellitis: An Unusual Presentation of Acute Infectious Mononucleosis

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INTRODUCTION

Epstein-Barr virus (EBV), the causative agent of infectious mononucleosis, has been linked to the secondary development of several neurologic disorders (Table 1). Here, I present the case of an 18-year-old female with acute cerebellar dysfunction consistent with postinfectious cerebellitis secondary to a previously undiagnosed EBV infection.

CASE PRESENTATION

History

An 18-year-old Caucasian female presented to the emergency department with a two-day history of vertigo, slurred speech, ataxia, poor hand-eye coordination, and impaired depth perception. The patient mentioned that a few weeks earlier she had experienced a prolonged upper respiratory tract infection with fatigue, sore throat, and nasal congestion but denied any other significant past medical history.

Physical Exam

Physical examination was remarkable for posterior cervical lymphadenopathy, palpable splenomegaly, mild ataxia, and difficulty with tandem gait to either side. Impaired depth perception was demonstrated when she walked into a wall.

Diagnostic Data

Initial laboratory studies revealed a normal white blood cell count with lymphocytic predominance and mild transaminitis. A later CBC with differential revealed the presence of atypical lymphocytes. CBC and CMP were otherwise unremarkable. Epstein-Barr virus serology was consistent with an acute EBV infection (Table 2). HIV by ELISA was negative. Urine drug screen was negative. Cerebrospinal fluid analysis (Table 3) revealed no abnormalities, including negative EBV by PCR. Neuroimaging was unremarkable, including CT of the head without contrast, MRI of the brain with and without contrast, and MRA of the head and neck without contrast. An abdominal ultrasound confirmed mild splenomegaly.

Clinical Course

Based on the above history, physical exam, and diagnostic studies, our patient was diagnosed with postinfectious cerebellitis secondary to acute infectious mononucleosis. She was treated supportively but remained symptomatic over the next eight days with no significant change in her clinical picture. On the ninth day of her illness, she was discharged home in stable condition with neurology follow-up. She experienced complete symptom resolution within four weeks.

DISCUSSION

Introduction

Postinfectious cerebellitis (PIC) is a rare, immune-mediated complication of Epstein-Barr virus, characterized by rapid development of cerebellar dysfunction. The condition has been linked to several other microbiologic agents and some vaccinations as well (Table 4).

Pathogenesis

PIC is believed to be an immune-mediated condition, as neurologic symptoms frequently follow resolution of the infectious prodrome. The inability to detect the presence of infection in the cerebrospinal fluid supports this theory. To date, there have been several autoantibodies isolated in patients with PIC.¹

Clinical features

Patients may present with sudden onset of ataxia, dysarthria, saccadic eye movements, dysmetria, vertigo, nausea, and vomiting.² Some patients exhibit the cerebellar cognitive affective syndrome, which is characterized by cerebellar dysfunction, cognitive impairment and affective changes.³ Cerebellar symptoms tend to appear within four weeks of the onset of the preceding infection. In cases of EBV-associated PIC, the preceding infection may go unrecognized or the patient may only recall a recent viral-like illness, as was the case with our patient.

Diagnosis

Patients with PIC due to Epstein-Barr virus will typically have a positive heterophile antibody test and

express both positive serum EBV VCA IgM and IgG, consistent with acute EBV infection.

CT and MRI are classically normal. There is suggestion that abnormalities in cerebellar enhancement may be seen on MRI prior to the onset of neurologic symptoms with near normalization by the time the patient is symptomatic (Figures 1 & 2).⁴ Thus, it may be possible to detect mild cerebellar changes on MRI if obtained very early in the course of the illness; however, this is not thought to be typical. Brain perfusion single photon emission tomography (SPECT) examination using 99mTc-HMPAO, on the other hand, may be useful during the symptomatic phase, demonstrating cerebellar hyperperfusion (Figure 3).⁵

The CSF cell count is frequently normal, with a mild lymphocytic pleocytosis seen in only 25-50% of patients.¹ CSF protein may be normal or slightly elevated. No direct evidence of CNS infection is found on CSF analysis by cultures or PCR.

Prognosis

PIC is usually self-limited, with the majority of patients experiencing complete resolution of symptoms within 1-4 weeks. However, 10-30% of patients experience persistent cerebellar dysfunction manifested by dysarthria or ataxia.¹ In severe cases, cerebellar edema may result in death by transtentorial or transforaminal herniation.⁴

Treatment

Treatment is primarily supportive, as most cases are self-limited. However, in rare cases with significant disability or a prolonged course, total plasma exchange or IV Ig may hasten recovery.⁶ Steroids may be beneficial in moderate to severe cases, however, direct viral invasion should be ruled out by negative CSF PCR prior to the initiation of any immunosuppressive treatment.⁷

Conclusion

Clinicians should be aware of this condition so that they may avoid unnecessary diagnostic testing, provide appropriate medical treatment when intervention is warranted, and prevent erroneous diagnoses that may be attached to a poor prognosis or negative stigma.

TABLE 1.⁸

Acute Neurologic Complications of Epstein-Barr Virus Infection
Encephalitis
Cerebellitis
Cranial neuritis
Transverse myelitis
Peripheral neuritis
Autonomic neuropathy
Guillan-Barre syndrome
Alice in Wonderland syndrome

TABLE 2

EBV SEROLOGY	
Heterophile antibody	Positive
VCM IgM antibody	Positive
VCA IgG antibody	Positive
EA antibody	Positive
EBNA antibody	Negative

TABLE 3

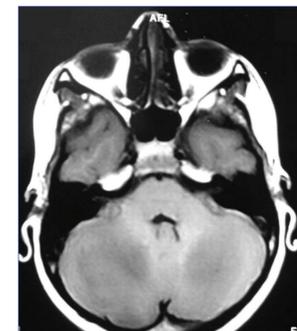
CEREBROSPINAL FLUID ANALYSIS	
RBC (/cmm)	0
WBC (/cmm)	4
Protein (mg/dL)	69
Glucose (mg/dL)	28
EBV by PCR (copies/mL)	<200 (negative)
CMV by PCR (copies/mL)	<500 (negative)
VZV by PCR (copies/mL)	<100 (negative)
Oligoclonal bands	No bands

TABLE 4¹

Microbiologic Agents Associated with Postinfectious Cerebellitis

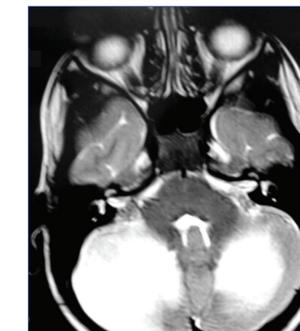
Viruses	
Epstein-Barr virus	Legionella pneumonia
Varicella-zoster virus	Neisseria meningitidis
Enterovirus	Salmonella typhi
Parvovirus	Bordetella pertussis
Mumps virus	Streptococcus pyogenes
Hepatitis A virus	Corynebacterium diphtheriae
Human herpes virus 6	
Herpes simplex virus	
Parasites	
	Plasmodium falciparum
Vaccines	
	Varicella-zoster virus
	Hepatitis B virus
	Rabies virus

FIGURE 1.⁴



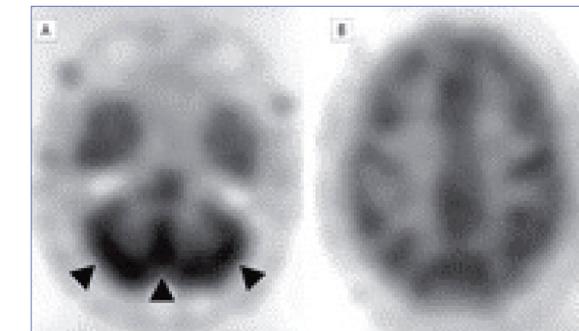
T1-weighted MRI image demonstrating hypointensity of the cerebellar grey and white matter.⁴

FIGURE 2.⁴



T2-weighted MRI image demonstrating a hyperintense signal in the cerebellum.⁴

FIGURE 3.²



Axial single photon emission tomographic sections through the cerebellum (A) and cerebral hemispheres (B). Increased cerebellar perfusion is demonstrated by high photon flux in the superficial regions (arrow heads).²

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