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Acute Epstein-Barr Virus Infection Presenting with Bilateral Facial Palsy: A Case Report

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Introduction: Bilateral facial palsy is a rare clinical presentation, occurring in less than 2% of all facial nerve palsies. Well-recognized causes include Guillain-Barré syndrome and Lyme disease.

Case Report: We present an unusual case of bilateral facial palsy related to acute Epstein-Barr virus infection in an immunocompetent 40-year-old man. We describe the emergency department diagnostic pathway, cerebrospinal fluid and magnetic resonance imaging findings and compare the case with previously reported cases.

Conclusion: Emergency physicians should consider Epstein-Barr virus in the differential diagnosis of bilateral facial palsy, particularly in patients with concurrent systemic viral symptoms and lymphocytic cerebrospinal fluid pleocytosis. Early recognition, appropriate imaging, and multidisciplinary consultation facilitate optimal management and patient outcomes. [Clin Pract Cases Emerg Med. 2026;X(X):XXX–XXX.]

Keywords: *bilateral facial palsy; Epstein-Barr virus; infectious mononucleosis; Bell's palsy; case report.*

INTRODUCTION

Bilateral facial palsy represents approximately 1% to 2% of all facial nerve palsies and poses a diagnostic challenge in the emergency department (ED).[1] Whereas unilateral facial palsy is idiopathic in most cases, bilateral involvement is idiopathic in approximately 20% of cases and is more likely to reflect an identifiable systemic cause.[2] The differential diagnosis is broad and includes infectious, inflammatory, neoplastic, metabolic, and traumatic etiologies. Epstein-Barr virus (EBV) is a recognized but relatively rare infectious cause. Reviews of the limited reported cases have identified Lyme disease and autoimmune disease, principally Guillain-Barré syndrome, as leading triggers.[3,4]

Epstein Barr virus is a human herpesvirus that causes infectious mononucleosis, typically presenting with fever, pharyngitis, lymphadenopathy, and splenomegaly. Neurologic complications occur in less than one percent of acute EBV infections and include meningitis, encephalitis, Guillain-Barré syndrome, transverse myelitis, and cranial neuropathies.[5,6] Although rare, nearly 40% of all EBV-associated facial nerve palsies reported in the literature are bilateral, with most cases occurring in children and young adults.[2,7] Bilateral facial palsy attributable to EBV in an otherwise healthy adult is described far less frequently.[8-10]

CASE REPORT

A 40-year-old previously healthy man presented to the ED with progressive bilateral facial weakness. Seventeen days prior to presentation, he developed bilateral retro-orbital pressure, headache, neck pain, and flu-like symptoms while visiting Florida. He initially presented to urgent care, where COVID-19 and influenza testing were negative. Despite brief initial improvement, he subsequently developed night sweats, chills, and subjective fever. Five days prior to ED presentation, he noticed right-sided facial weakness that progressively involved the left side, resulting in complete bilateral facial paralysis. He denied dysphagia, dysarthria, diplopia, or limb weakness. There was no reported tick bite or rash.

Outpatient workup at his primary care office revealed leukocytosis with lymphocyte predominance, elevated transaminases and alkaline phosphatase. Serologic testing demonstrated positive EBV viral capsid antigen immunoglobulin M (IgM) and immunoglobulin G (IgG), negative EBV nuclear antigen antibody, and positive heterophile antibody, consistent with acute EBV infection. Lyme IgM was equivocal and IgG negative. HIV, hepatitis B and C, cytomegalovirus, gonorrhea, and chlamydia testing were all negative. He was started on doxycycline, prednisone, and valacyclovir by his primary care physician and referred to the ED for further evaluation.

On arrival to the ED, vital signs were the following: temperature, 97.8 ºF; heart rate, 61 beats per minute; blood pressure, 120/56 mm Hg; respiratory rate, 18 breaths per minute; and SpO_2, 99% on room air. Physical examination was notable for complete bilateral facial nerve palsy with inability to close eyes, raise eyebrows, or smile. Pupils were equal, round, and reactive to light with full extraocular movements. The remainder of the cranial nerve examination was normal. Motor strength was 5/5 in all extremities with normal sensation and symmetric deep tendon reflexes. No rash or lymphadenopathy was appreciated.

Laboratory studies again showed lymphocyte predominant leukocytosis with elevated transaminases. Computed tomography of the head without contrast and computed tomography venogram were unremarkable without evidence of venous sinus thrombosis. Lumbar puncture yielded clear cerebrospinal fluid with lymphocytic pleocytosis. Cerebrospinal fluid Gram stain was negative, and meningitis/encephalitis polymerase chain reaction panel including herpes simplex virus and varicella zoster virus was negative.

The patient was admitted, and neurology and infectious disease consultations were obtained. Magnetic resonance imaging (MRI) of the brain with and without gadolinium demonstrated enhancement of the labyrinthine segments of both facial nerves extending to the fundus of the internal auditory canal bilaterally, consistent with infectious or inflammatory bilateral facial nerve palsy (Images 1 and 2).

No other cranial nerve enhancement, parenchymal edema, or intracranial abnormality was identified. Additional cerebrospinal fluid testing for EBV deoxyribonucleic acid, Lyme antibody, West Nile virus, and dengue were ultimately returned negative. Serum angiotensin-converting enzyme and Sjögren antibodies were negative. The patient was empirically treated with ceftriaxone (2 g) intravenously daily to cover for possible central nervous system Lyme disease, given the equivocal Lyme serology and endemic area (western Massachusetts). He continued valacyclovir (1 g) orally three times daily and prednisone (60 mg) orally daily.

By hospital day 2, the patient reported subjective improvement in left facial movement with better eye closure. Repeat EBV serologies confirmed acute infection, and a Lyme western blot was negative. Based on recommendations by the infectious diseases team, ceftriaxone and valacyclovir were discontinued, and the patient received oral doxycycline (100 mg twice daily) for 7 days with a prednisone taper. The patient was discharged on day 3 with neurology and infectious diseases follow-up and a referral for facial nerve physical therapy.

CPC-EM Capsule

What do we already know about this clinical entity?
Bilateral facial palsy is rare (<2% of facial palsies); Lyme disease and Guillain-Barré syndrome (GBS) are leading causes. Epstein-Barr virus (EBV) is an uncommon but recognized trigger.

What makes this presentation of disease reportable?
Adult with EBV bilateral facial palsy mimicking Lyme in an endemic area (equivocal IgM), with lymphocytic cerebrospinal fluid (CSF) and bilateral labyrinthine nerve enhancement on MRI.

What is the major learning point?
Bilateral facial palsy warrants a full workup; CSF and serologies help separate EBV from GBS and Lyme, and empiric Lyme therapy is reasonable pending results.

How might this improve emergency medicine practice?
A structured emergency department approach can identify EBV and treatable mimics in bilateral facial palsy.

DISCUSSION

This case illustrates bilateral facial palsy as a rare neurologic manifestation of acute EBV infection in an immunocompetent adult. The pathophysiology of EBV-associated facial palsy is not fully understood but is thought to involve either direct viral invasion of the facial nerve or an immune-mediated inflammatory response.[11] Epstein-Barr virus has tropism for B lymphocytes and epithelial cells; researchers have detected viral DNA in facial nerve ganglia from a random postmortem sample.[12] The bilateral nature of facial nerve involvement may reflect systemic viremia with symmetric neural inflammation or a heightened immune response affecting both nerves simultaneously.

The diagnosis of acute EBV infection in this case was supported by positive EBV viral capsid antigen IgM and IgG with negative EBV nuclear antigen antibody, a pattern



Image 1. Left facial nerve, demonstrating enhancement on axial T1 weighted magnetic resonance imaging (arrow) consistent with inflammatory or infectious Bell's palsy in a patient with bilateral facial nerve palsy.



Image 2. Right facial nerve, demonstrating enhancement on axial T1 weighted magnetic resonance imaging (arrow) consistent with inflammatory or infectious Bell's palsy in a patient with bilateral facial nerve palsy.

consistent with recent primary infection.

The presence of heterophile antibody provided additional confirmatory evidence. Lymphocytosis and elevated transaminases are characteristic laboratory findings in infectious mononucleosis and were present in this patient (Table). The cerebrospinal fluid demonstrated lymphocytic pleocytosis with elevated protein, consistent with viral meningoencephalitis. Importantly, the absence of albuminocytologic dissociation (markedly elevated protein with normal or minimally elevated cell count) argued against Guillain-Barré syndrome.[13]

Magnetic resonance imaging played a crucial role in this case by demonstrating bilateral facial nerve enhancement along the labyrinthine segments. This finding is consistent with inflammatory or infectious facial neuropathy and helped exclude other structural causes such as neoplasm or demyelinating disease. The absence of other cranial nerve enhancement or parenchymal lesions further supported the diagnosis of isolated bilateral facial nerve involvement related to acute EBV infection.

The differential diagnosis of bilateral facial palsy is broad and warrants a structured workup in the ED with Lyme serology, cerebrospinal fluid sampling with cell counts and polymerase chain reaction, EBV serology, HIV and syphilis serology, and neuroimaging as available. Physical exam must include careful evaluation of extraocular movements and deep tendon reflexes. Important mimics include Guillain-Barré syndrome (particularly the Miller Fisher variant), Lyme disease (especially in endemic areas), sarcoidosis, HIV, syphilis, and leukemic or lymphomatous meningeal infiltration.[3,4] The patient's recent travel to Miami raised concern for emerging infectious diseases such as Oropouche virus, which has been associated with neurologic complications including Guillain-Barré syndrome.[15] However, comprehensive infectious workup including tick-borne panel, dengue, West Nile virus, and Lyme disease was negative, making these etiologies unlikely.

Most EBV-associated bilateral facial palsies have occurred in infants and young children, such as the 14-month-old described by Terada et al and a 3-year-old boy reported by Grassin et al. Adult presentations are uncommon, and include an adult with concomitant COVID-19 as well as other reports of EBV-associated bilateral facial palsy.[8-10] Our patient's course featured similar features to others, including lymphocyte predominant cerebrospinal fluid pleocytosis, facial nerve enhancement on MRI, and (as in Grassin) early empiric treatment for central nervous system Lyme disease until it had been definitively ruled out. The patient described in Coddington et al notably had jaundice in addition to facial palsy.

From an emergency medicine perspective, we emphasize

Table. Laboratory and diagnostic findings at outpatient, emergency department and inpatient evaluation.

Test	Outpatient (PCP)	ED and inpatient	Reference Range
White blood cell count (/μL)	15,000 (68% lymph)	11,200 (50% lymph)	4,000-11,000
Alkaline phosphatase (U/L)	255	194	30-130
Aspartate aminotransferase (U/L)	111	43	10-40
Alanine aminotransferase (U/L)	249	129	7-56
EBV VCA IgM / IgG	Positive / Positive	Confirmed on repeat	Negative
EBV nuclear antigen antibody	Negative	N/A	Negative
Heterophile antibody	Positive	N/A	Negative
Lyme IgM / IgG	Equivocal / Negative	Western blot negative	Negative
CSF appearance	N/A	Clear	Clear
CSF white cells (/μL)	N/A	19 (100% lymph)	0-5
CSF protein (mg/dL)	N/A	64	15-45
CSF glucose (mg/dL)	N/A	60	40-70
CSF Gram stain / ME PCR panel	N/A	Negative / Negative	Negative
CSF EBV DNA PCR	N/A	Negative	Negative
HIV, hep B/C, CMV, GC/chlamydia	Negative	N/A	Negative

CMV, cytomegalovirus; *CSF*, cerebrospinal fluid; *dL*, deciliter; *DNA*, deoxyribonucleic acid; *EBV*, Epstein-Barr virus; *ED*, emergency department; *GC*, gonococcus; *hep*, hepatitis; *HIV*, human immunodeficiency virus; *IgG*, immunoglobulin G; *IgM*, immunoglobulin M; *L*, liter; *lymph*, lymphocytes; *ME*, meningoencephalitis; *mg*, milligram; *μL*, microliter; *N/A*, not applicable; *PCP*, primary care physician; *PCR*, polymerase chain reaction; *U*, units; *VCA*, viral capsid antigen.

the importance of bilateral facial palsy as a red flag that should prompt serious consideration for neuroimaging and lumbar puncture. Our patient underwent MRI as an inpatient, as well as consultations by neurology and infectious disease specialists. At departments without such resources, transfer to a tertiary care center may be necessary.

Management of EBV-associated bilateral facial palsy remains largely supportive. Corticosteroids are commonly used based on their established benefit in Bell's palsy.[16] Our patient received prednisone (60 mg daily) with a taper, which is consistent with standard protocols for facial nerve palsy. The role of antiviral therapy is controversial because antivirals reduce viral shedding but do not clearly improve clinical outcomes in infectious mononucleosis or its neurologic complications.[17] Valacyclovir was initially prescribed but ultimately discontinued based on infectious diseases recommendations and lack of robust evidence for benefit. Eye care is critical in patients with facial palsy to prevent corneal exposure and injury, and artificial tears should be prescribed for incomplete eye closure.[14]

The empiric use of antibiotics for Lyme disease in this case warrants discussion. The patient resided in an endemic area and had an equivocal Lyme IgM antibody result, raising concern for early Lyme neuroborreliosis. Current guidelines recommend empiric treatment in patients with suspected Lyme disease and neurologic involvement, particularly facial palsy.[19] Doxycycline was chosen over ceftriaxone after negative confirmatory testing because it provides adequate central nervous system penetration for early disease and is the preferred agent for outpatient management.

The prognosis for EBV-associated facial palsy is generally favorable, with most patients experiencing complete or near-complete recovery over weeks to months. Recovery time is variable and depends on the severity of nerve damage. Physical therapy exercises targeting facial muscles can facilitate recovery and prevent synkinesis.[18] Close follow-up with neurology is needed to monitor recovery and address complications.

This case highlights several important teaching points for emergency physicians. First, bilateral facial palsy requires a thorough diagnostic evaluation to identify treatable causes, with particular attention to infectious and inflammatory etiologies. Second, acute EBV infection should be considered in patients presenting with bilateral facial palsy, especially when accompanied by systemic viral symptoms, lymphocytosis, and elevated transaminases. Third, cerebrospinal fluid analysis can help differentiate between infectious or inflammatory causes (lymphocytic pleocytosis with mild protein elevation) and Guillain-Barré syndrome (albuminocytologic dissociation). Fourth, MRI is valuable for confirming facial nerve inflammation and excluding structural mimics. Finally, a multidisciplinary approach involving neurology and infectious disease consultation facilitates optimal management.

This case report has several limitations. As a single case, it does not establish causation between EBV infection and bilateral facial palsy, although the temporal relationship and exclusion of alternative etiologies are suggestive. Cerebrospinal fluid EBV DNA polymerase chain reaction was

negative, which could indicate that the facial nerve involvement was immune-mediated rather than due to direct viral invasion, although false-negative cerebrospinal fluid polymerase chain reaction results can occur. Long-term follow-up data regarding complete recovery were not available at the time of this report.

CONCLUSION

Bilateral facial palsy is a rare but important presentation that requires systematic evaluation for infectious, inflammatory, and structural etiologies. Acute Epstein-Barr virus infection should be included in the differential diagnosis, particularly in patients with concurrent systemic viral symptoms and lymphocytic cerebrospinal fluid pleocytosis. A comprehensive workup that includes serologic testing, lumbar puncture, and MRI with gadolinium helps establish the diagnosis and guide treatment. Management is primarily supportive with corticosteroids and eye care, although empiric antibiotics may be warranted in endemic areas with concern for Lyme disease. Early recognition and multidisciplinary consultation facilitate optimal outcomes for this uncommon but challenging clinical presentation.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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