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CE - PHYSICAL EXAMINATION

Facial palsy associated with Epstein–Barr infection in an adult patient: case report

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A 42-year-old white male with an unremarkable past medical history was admitted in our ward (Internal Medicine) because of fever, myalgia and asthenia. Upon admission, patient's temperature was 99.5 F, blood pressure 130/90 mmHg, heart rate 100 beats/min and respirations 20 breaths/min. Physical examination showed only mild hepatosplenomegaly in the absence of lymphadenopathy and rashes. His neurological examination was normal.

Among initial chemistries, CBC revealed white count 12,100/mm^3, hemoglobin 14.0 g/dL, and platelets 238,000/mm^3. White cell differential was notable for 26% neutrophils, 11% lymphocytes, 51% atypical lymphocytes, and 6% monocytes. Peripheral blood smear confirmed the presence of large atypical lymphocytes. Liver function tests showed albumin of 3.9 g/dL, total bilirubin of 2.0 mg/dL, alkaline phosphatase of 179 U/L and aspartate aminotransferase of 508 U/L. Prothrombin time and activated partial thromboplastin time were within normal limits.

He had positive Lyme titers by EIA at 5.4 U/mL (negative <5.0), but western blot did not show sufficient bands for a sierological diagnosis of *Borrelia burgdorferi*. Serum antibody titers for HIV, cytomegalovirus (CMV), herpes simplex (HSV) type I–II, toxoplasma, Hepatitis A, B, C and E and for chikungunya were also negative. Chemiluminescence assays of antibody titers to Parvovirus B19 antigens showed IgM of 1.3 index (positive if > 1.1 index), but with the absence in the blood of Parvovirus B19 DNA. Heterophile antibody test was positive. Chemiluminescence showed the absence of EBNA IgG antibodies, Viral Capsid Antigen (VCA) (IgM) of 20.03 index (positive if > 1.0 index), VCA (IgG) of 2.83 index, and the presence in the blood of Epstein–Barr virus (EBV) DNA: 12.288 UI/mL. These findings were suggestive of an acute primary EBV infection, also called infectious mononucleosis (IM). Screening was completed with tests for antinuclear antibodies, antibodies to extractable nuclear antigen, anti-mitochondrial antibodies and antimicrosomal antibodies, which were all negative. A toxicology screen was negative. Chest and abdomen CT scan showed not specific mild lymphoadenomegalies and splenomegaly (longitudinal diameter of 16 cm).

On third day from admission, the patient complained of weakness on the left side of his face with no tenderness. On physical examination, facial asymmetry with complete lower motor neuron-type left facial nerve paralysis was observed. Facial weakness was assessed as grade IV of the House–Brackmann scale, involving incomplete closure of the eye lid. No vesicles in the oral mucosa or in the auditory canal indicated active HSV or varicella zoster virus (VZV). Due to recent fever, a head CT scan was performed and it was negative. Moreover, to rule out a neuroborreliosis, we proposed to the patient a rachicentesis to investigate IgM and IgG for Borrelia in the cerebrospinal fluid, but the patient refused. A diagnosis of left peripheral facial nerve palsy (FP) of viral origin, EBV, was made.

The patient was started on a course of prednisone 75 mg daily and valaciclovir 1 g three times daily, and vitamin B complex; eye protection and artificial tears were also provided. A complete recovery was not achieved 3 weeks after presentation. He started physical facial re-training and subsequently improved and there were no sequelae.

FP is the most common single nerve infection. Most cases are idiopathic, nonetheless a relevant percentage is caused by potentially treatable etiologies including infections. Henkel et al.[1] analyzed retrospectively all the cases of newly diagnosed FP at their center. An infection, due to serological findings or based on cerebrospinal fluid (CSF) analysis, was found in 19% of adult patients, caused by *B. burgdorferi*, VZV or HSV, but no cases due to EBV [1]. Involvement of the nervous system is unusual in IM [1–5] and it has been reported to range from 0.37 to 7.3%, although changes in the CSF are found to occur in about 26.5% of cases [2].

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Moreover, FP as the only neurological manifestation is rare, in particular in adult patients [1–5]. Gsell [2] presented for the first time one case each of unilateral and bilateral FP in two young men with clinical IM. Davidson and Salter [3] described a case of IM presenting with facial diplegia in the initial absence of any of the more commonly recognized features of the disease. Grose et al.[4] reported three cases of FP following serologically documented IM without other neurological deficits. The authors suggested that unilateral facial palsy could represent a mononeuritic variant of Guillain–Barrè syndrome associated with IM. The main differential diagnosis for those patients includes Lyme disease or some rarer viral cause. Although Borrelia serum IgM antibodies were positive in our patient, clinical features were not suggestive of Borrelia infection because of lack of arthralgia and erythema migrans. Moreover, Borrelia IgM positivity in the setting of IM may be interpreted as a cross-reaction phenomenon. It could also be the case for Parvovirus IgM antibodies' positivity. Indeed, we decided not to investigate for a seroconversion in the serum with an antibody for Borrelia because Lyme disease is not endemic in our region and our patient had a complete, even if delayed, resolution of symptoms with IM treatment plus physical therapy.

The prognosis for neurological complications during EBV infection is usually good. In Bell's palsy, the most frequent idiopathic form of FP, for whom evidence is more consistent, it has been seen that: (a) predictors of incomplete recovery include severe FP, length of time prior to onset of recovery, and persistent pain; (b) the combination of oral corticosteroids plus antiviral therapies is associated with lower rates of incomplete recovery compared with oral corticosteroids alone; (c) although not recommended for all the patients suffering from Bell's palsy, there are subgroups of patients for whom there is evidence supporting the use of physiotherapy.

Our patients had no pain and therapy with oral corticosteroids plus antiviral drug was started within 3 h from onset of symptoms. Nonetheless, an early incomplete recovery was observed and we could be able to achieve a significant recovery only with the start of facial rehabilitation.

The present case confirms the occurrence of FP as the sole neurological manifestation in IM. Moreover, this case highlights that management of this neurological complication could be complex.

Compliance with ethical standard

Conflict of interest The authors declare that they have no confict of interest.

Statement of human and animal rights All procedures performed in this study involving human participant were in accordance with the ethical standards of the institutional or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from the patient at the discharge from our department.

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