Neurological Sciences (2022) 43:6983–6985
https://doi.org/10.1007/s10072-022-06327-x

LETTER TO THE EDITOR

Bilateral facial palsy as the first sign of HIV infection

Fabrizio Giammello[1,2] · Chiara Vitale[2] · Vincenzo Rizzo[2] · Antonio Toscano[2] · Paolo Girlanda[2]

Received: 10 July 2022 / Accepted: 5 August 2022 / Published online: 9 August 2022

Dear Editor,

Bilateral facial palsy (BFP) is a rare clinical condition, representing 0.2–3% of facial paralysis cases [1]. Etiology is often unclear. Peripheral facial palsy (PFP) is the most frequent cranial neuropathy associated with human immunodeficiency virus (HIV), whereas only few BFP cases have been described in literature. We report a case of BFP in a previously asymptomatic patient, as initial presenting symptom in advanced stage of HIV infection.

A 37-year-old man was referred to the emergency room of our Hospital with difficulty in closing eyes and mouth drooping on both side. He had been treated with oral prednisone for 10 days until hospital admission, without improvement. A month earlier, the patient experienced a flu-like episode characterized by fever, vomiting, diarrhea, and maculopapular skin rush. His previous medical history was negligible.

Neurological examination was unremarkable except for a bilateral peripheral facial palsy (House-Brackmann stage IV). Blood examination, also including cytomegalovirus (CMV), Epstein-Barr virus (EBV), and herpes simplex virus type 1 and 2 (HSV) antibodies, was normal except for a slight rise in white blood cell count (10,930/mm^3; normal value: 4000–9000) with 44% neutrophils and 48% lymphocytes.

Brain magnetic resonance imaging (MRI) revealed no intracranial abnormalities. Specifically, gadolinium administration did not reveal facial nerve enhancement or signs of inflammation, although motion artifacts may have affected the image quality. Direct facial nerve conduction studies were normal, whereas blink reflex examination revealed prolonged latencies of both R1 responses and a decreased amplitude of all recorded responses. The cerebrospinal fluid (CSF) analysis showed clear and colorless appearance, with an increased protein content (100 mg/dl; nv: 20–50) and cell count (57/ mm3; nv: 0–5), but no oligoclonal bands. However, ELISA and Western blot analyses of serum sample were clearly positive for HIV-1 infection. The CD4 count was 13% (nv: 31–60) and the CD8 was 75% (nv: 13–41) with a CD4/CD8 ratio of 0.17 (nv: 1.0–3.5), revealing an asymptomatic HIV in advanced stage [2]. The patient was not aware of the disease, but he used to conduct high-risk sexual behaviors, having had unprotected receptive intercourse with casual partners. Despite not reporting further risk factors, we performed serologic testing for Lyme disease using both ELISA and Western blot, which resulted negative. He was referred to the infectious disease specialist for clinical and laboratory follow-up and promptly started an antiretroviral therapy with Triumeq (dolutegravir/abacavir/lamivudine). No viral load measurement was available at that time. At the 30th day follow-up the patient had a decrease in white blood cell count (7630 mmc/mm3; normal value: 4000–9000) with 59% neutrophils and 31% lymphocytes. The CD4 count decreased to 8% (nv: 31–60), and therefore, the CD4 cell count calculated from total lymphocyte count (2365/mm3; nv: 1000–4800) was 189/mm^3, confirming diagnosis. His clinical condition progressively improved with a BFP almost complete resolution in about 6 months.

Discussion

Neurological involvement in HIV is quite common (75–90% of HIV patients) and all neuraxis levels could be involved [1, 3]. PFP is the most frequent cranial neuropathy associated with HIV [3]. The association of PFP and HIV was firstly described by Snider et al. in 1983 [4]. BFP is much less common than the unilateral PFP, occurring in 0.3 to 2% of the general population [1]. However,

Fabrizio Giammello and Chiara Vitale contributed equally to this work.

✉ Fabrizio Giammello
fabrizio.giammello@gmail.com;
fabrizio.giammello@unime.it

[1] International PhD Translational Molecular Medicine and Surgery, Department of Biomedical, Dental Science and Morphological and Functional Images, Polyclinic Hospital, University of Messina, Via Consolare Valeria 1, 98125 Messina, Italy

[2] Department of Clinical and Experimental Medicine, Polyclinic Hospital, University of Messina, Messina, Italy

among HIV patients, BFP is quite rare: during the HIV course, excluding the present case, only 20 other cases have been reported [4]. Both unilateral and BFP may develop any time throughout the course of HIV infection and it is most commonly observed as an early stage complication [4, 5]. PFP occurring in acute HIV infection may precede seroconversion by 4–6 weeks, and it could be complicated by an aseptic meningitis [4]. Frequently, in BFP, facial muscle weakness does not occur simultaneously on both sides but, firstly, affects one side and, within few days, the opposite side [6]. PFP pathogenesis in HIV is not entirely understood: at an early asymptomatic stage, some authors hypothesized a direct lesion of facial nerve caused by a neurotropic virus. Another hypothesis is an immunological hyper-reaction to HIV viremia, producing demyelination of cranial nerves, similar to a Guillain–Barre syndrome (GBS). Finally, in the advanced stages, impaired cellular immunity may originate PFP by altering patient defenses against opportunistic infections [5]. BFP requires an extensive differential diagnosis because it can present in several diseases, being GBS the most common [4]. Other etiologies include Bickerstaff's brainstem encephalitis, tuberculous meningitis, herpes zoster, HSV, CMV, EBV, neurosarcoidosis, lymphomatous or carcinomatous meningeal infiltration, Lyme disease, traumatic, diabetes mellitus, and toxic [4]. Consequently, the work up should include complete blood and CSF examinations, very useful to exclude other neurologic infections. Although the patient did not report further risk factors, apart from high-risk sexual behaviors, we performed serologic testing for Lyme because of the possible risk of coinfection [7]. Recently, Cabrera Muras et al. [8] reported a case of severe BFP associated with EBV and SARS-CoV-2 coinfection. However, our patient first presented before the pandemic breakthrough; hence, we could not consider such a differential diagnosis at the time. Contrast-enhanced MRI scan may detect lesions of the central nervous system. Skull base, meninges, and cerebellopontine angle are the brain regions that must be studied above all. In this case, a specific diagnostic work up allowed us to exclude other diagnoses, and therefore, BFP was easily linked to the diagnosed HIV infection. Moreover, another quite unique aspect was that the patient was asymptomatic despite an advanced disease stage (absolute CD4 cell count < 200/mm^3 or percentage < 14% [2]). The prognosis of PFP in HIV is usually favorable. Most cases have a spontaneous recovery within 15 days to 3–6 months [1]. The treatment of PFP in HIV infection is mainly based on administration of famciclovir or of a high-dose acyclovir [1]. Steroids use is controversial: according to a recent Cochrane systematic review [6], some authors support their use in order to reduce the immunologically mediated neural inflammation of facial nerve. On the other hand, the use of corticosteroids could increase the possibility of opportunistic infections in HIV immunocompromised patients. Although the antiretroviral treatment has been successful in our BFP case, the efficacy of these therapeutic agents on HIV-associated PFP recovery is still unknown [5].

Conclusion

BFP is a very rare entity that should be rapidly investigated when occurring, because it may indicate the onset of a systemic disease or represent the sole symptom of active underlying disease process. The early diagnosis of HIV infection in patients with BFP is crucial, considering that a timely management of HIV infection can produce the greatest benefit for the patient, as well as a preventive awareness, in order to avoid possible transmission of the virus.

Author contribution F. G., C. V., and P. G. conceptualized the study. F. G. and C. V. wrote the original draft. V. R., A. T., and P. G. reviewed and critiqued the manuscript.

Declarations

Ethics approval The paper does not report on primary research. All data analyzed were collected as part of routine diagnosis and treatment.

Informed consent Unfortunately, to account for the restrictive measures in place due to COVID-19, a handwritten signature is not possible. However, we obtained an audio recording of oral consent from the patient. We thank the patient and his family for their kind cooperation.

Conflict of interest The authors declare no competing interests.

References

1. Serrano P, Herná Ndez N, Arroyo JA, M[a] De Llobet J, Domingo P. Bilateral Bell Palsy and acute HIV type 1 infection: report of 2 cases and review. https://doi.org/10.1086/511876
2. CDC (2014) Revised surveillance case definition for HIV infection. Morb Mortal Wkly Rep 63: 1–13
3. McArthur JC, Brew BJ, Nath A (2005) Neurological complications of HIV infection. Lancet Neurol 4:543–555
4. Abboud O, Saliba I (2008) Isolated bilateral facial paralysis revealing AIDS: a unique presentation. Laryngoscope 118:580–584
5. Sathirapanya P et al (2018) Peripheral facial paralysis associated with HIV infection: a case series and literature review. Clin Neurol Neurosurg 172:124–129

6. Madhok VB, Gagyor I, Daly F, Somasundara D, Sullivan M, Gammie F, Sullivan F (2016) Corticosteroids for Bell's palsy (idiopathic facial paralysis). Cochrane Database Syst Rev 7(7):CD001942. https://doi.org/10.1002/14651858.CD001942.pub5
7. Garcia-Monco JC, Frey HM, Villar BF, Golightly MG, Benach JL (1989) Lyme disease concurrent with human immunodeficiency virus infection. Am J Med 87:325–328
8. Cabrera Muras A et al (2021) Bilateral facial nerve palsy associated with COVID-19 and Epstein–Barr virus co-infection. Eur J Neurol 28: 358–360