

A Rare Cause of Bilateral Facial Palsy

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Abstract HIV infection can cause neurological complications at all stages of the disease. Bilateral facial paralysis is not common in HIV, and more likely to have a systemic cause such as GBS, brain stem encephalitis, sarcoidosis, Lyme disease, syphilis or bacterial meningitis. Although HIV-associated bilateral facial palsy is very rare, it should be included in the differential diagnosis in unexplained cases, particularly in high-risk patients. We report a unique case of bilateral facial nerve palsy as an initial presenting symptom of AIDS, with an updated literature review.

Keywords: facial palsy, HIV infection

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1. Introduction

HIV infection can cause neurological complications at all stages of the disease [1,2].

These may involve both central and peripheral nervous systems, including aseptic meningitis, encephalitis, meningoencephalitis, cranial neuropathy, myelitis, peripheral nerve palsy and peripheral neuropathy [3,4,5,6]. HIV infection-associated facial palsy is usually unilateral [7,8]; bilateral facial paralysis associated with HIV is exceedingly rare [9,10].

Bilateral facial paralysis (BFP) usually occurs as a result of systemic disease [10,11,12,13]. In addition, several central and peripheral nervous system diseases can cause BFP. When HIV is entertained as a causative etiologic agent in BFP, an extensive work-up should be performed, considering both to exclude central and peripheral lesions, before making a final diagnosis.

Herein, we report a unique case of BFP as an initial presenting symptom of AIDS, with an updated literature review.

2. Case Presentation

A 44 year-old male with past medical history of an inactive seizure disorder, chronic migraine, anxiety, depression, and syphilis was admitted to hospital for drooling, facial weakness, blurred vision and left arm numbness. The latter two complaints resolved within hours. He first noticed difficulties with speech and facial movement two days before admission. Facial weakness was initially more prominent on the right, but he eventually developed severe facial weakness on both sides. He had lost about 20 pounds in recent months.

He had been treated for syphilis approximately 12 years earlier. He was homosexual, having sex exclusively with males, but with consistent condom use. Multiple previous tests for HIV had all been negative. His mother had rheumatoid arthritis; family history was otherwise unremarkable except that his father and his mother were siblings.

Examination revealed no rash or fever, but bilateral cervical adenopathy. Neurological exam showed bilateral, fairly severe 7th nerve palsies that involved all aspects of facial function. The remainder of his neurological examination, including other cranial nerves, motor exam, sensory exam, reflexes, coordination and gait, was within normal limits.

Cranial MRI, with and without gadolinium contrast, was normal. Cerebrospinal fluid examination showed mildly elevated protein, 68 mg/dl (normal range 15-45), 30 white blood cells/mm³ with lymphocyte predominance (normal range 0-10), and unremarkable results for other tests including glucose, Venereal Disease Research Laboratory, enterovirus culture, HSV-1 and 2 polymerase chain reaction (PCR), varicella-zoster virus DNA PCR; CSF culture and gram stain, and cryptococcal antigen. Fourth generation serum HIV Ag/Ab, 4th was reactive, HIV copy number was 4,230,758/ml (normal range <20 copies/ml). Rapid plasma reagin was reactive at a low titer of 1:2, as was the fluorescent treponemal antibody test. Varicella zoster IgG, but not IgM was elevated in serum. Also negative/unremarkable were: blood count, creatinine, serum chemistries, Lyme ELISA test, EBV VCA IgM, Cytomegalovirus IgM, tests for rickettsial diseases, C-reactive protein, *Borrelia burgdorferi* DNA and mono spot test. CD4⁺ count was 342 cells/mm³ (15%).

The positive syphilis tests were considered to reflect remote, treated infection. Thus, he was not retreated for syphilis. After review of the entire picture, it was concluded that his BFP were related to acute HIV infection. He was discharged to be followed by an

infectious disease specialist closer to his home. When contacted approximately 10 weeks after discharge he had not had any remarkable improvement in his BFP and reported no new symptoms following discharge.

3. Discussion

In this report, we present a case with BFP as the initial presenting neurological symptom of AIDS. Extensive investigations, including MRI of brain and CSF analysis were performed to rule out central and peripheral causes of BFP. Several earlier HIV tests had been had been negative.

Patients with HIV can develop a wide spectrum of neurological complications [3,4,5]. The frequency of HIV related peripheral nervous system involvement is 14 - 27% [3,5,14], with cranial nerve involvement in approximately 1% - 4% [7,11,15]. PFP can be seen as the first presenting symptom of HIV infection [16]. PFP associated with HIV is usually unilateral; BFP associated with HIV is extremely rare. Including the present case, only 27 cases of BFP associated with HIV have been reported [3,5,16,17,18].

In BFP associated with HIV, contralateral facial nerve involvement usually starts within 30 days [3,18]. In our case, the patient developed contralateral PFP within 3-5 days.

BFP associated with HIV can be diagnosed after exclusion of the other central and peripheral causes [3,9,19,20,21]. Well-described differential diagnoses of BFP are Bell's palsy, Guillain-Barre syndrome, syphilis, Lyme disease, poliomyelitis, EBV, meningeal/pontine tumor and sarcoidosis. Detailed investigation including MRI of brain, CSF analysis and serum tests must be performed to rule out all other possible infectious and systemic diseases [2,6,17,18,22,23,24,25]. On the other hand, PFP has a high predictive value for HIV-1 infection in patients with high rates of seroconversion. Thus, HIV-1 testing should be part of the routine work-up for facial paralysis in high-risk patients [20,25,26].

The diagnosis of HIV infection can be challenging and difficult at the beginning of infection [12,13,15,25]. Serology can be negative. BFP can be the initial presenting symptom of AIDS [15,19]. In our case, the patient had recent negative HIV tests before presentation with BFP, at which time repeat HIV tests became positive. We recommend repeating HIV testing for patients with BFP who have had previously negative HIV tests, if etiology remains unclear.

The prognosis of BFP associated with HIV is usually benign, but depends upon the HIV stage. Unilateral PFP usually completely resolves within months [9,20,21]. Of 27 reported cases, 21 showed a good recovery of the paralysis within several weeks after onset, and three had partial improvement [3,7,8,9,12,15,18,19,20,21,25,27]. Most of the patients with BFP associated with HIV infection have spontaneous recovery over 2-24 months [3,8,10,20,21].

There is no recommended treatment approach for facial paralysis associated with HIV infection [2,24]. The role of antiretroviral, acyclovir or steroid treatment is controversial [19,24,25,28]. There have been no controlled studies on the effectiveness of antiretroviral therapy or

acyclovir on facial paralysis associated with HIV infection [3,8,18,19,24]. Corticosteroid treatment for facial paralysis in HIV patients may increase risk of life-threatening opportunistic infections [9,19,20,24,25]. Therefore, risk and benefit assessment should be done carefully; if benefits are judged to exceed risks, then short-term corticosteroid can be administered, particularly in patients with early stage disease. In our case, the patient received no specific treatment and had not improved by day 75 after discharge; however, recovery can take up to 24 months.

4. Conclusion

Facial palsy is common, and may be caused by several autoimmune, inflammatory and infection diseases. BFP is less common and more likely to have a systemic cause such as GBS, brain stem encephalitis, sarcoidosis, Lyme disease, syphilis and bacterial meningitis. HIV-associated BFP is very rare, but should be included in the differential diagnosis in unexplained cases, particularly in high HIV risk patients. Careful and detailed examination and investigation are warranted. We report this case to increase awareness of clinicians that HIV should be include in differential diagnosis of unexplained cases of BFP.

Disclosure

The authors declare that there is no conflict of interest.

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