To the Editor

Bilateral Peripheral Facial Paralysis Combined with HIV Meningitis

Bilateral peripheral facial paralysis (PFP) is a very rare clinical manifestation that occurs in acute HIV-1–infected patients, and only 17 cases (including the present case) have been reported worldwide over the last 30 years[1-3]. Here, we report the first case of bilateral PFP in a Chinese patient who also had acute retroviral syndrome (ARS) and aseptic meningitis with high HIV-1 viral load in the cerebrospinal fluid (CSF).

Case Report

A 38-year-old homosexual man had been in normal health until 21 days prior to admission, when he developed high fever (up to 39°C), chills, headache, fatigue, general malaise, nausea, and vomiting. Ten days prior to admission, he experienced peripheral facial numbness, and then suddenly experienced right-side facial paralysis; failure to close both eyes; and difficulty sucking, smiling, and talking. Three days later, the left side of his face became paralyzed.

On physical examination at admission, the patient was febrile (37.5°C) and had cervical, axillary, and groin lymphadenectasis. Neurological examination revealed bilateral drooping of the lower lips and cheeks, as well as failure of bilateral eye closure (palsy severity was grade 4 according to the House-Brackmann system). Results of the other physical and neurological examination were normal.

Cerebrospinal fluid (CSF) analysis revealed pleocytosis (30 cells/mm^3, 30 leukocytes/mm^3, 93% monocytes), a marked increase in micro total protein content of 1.27 g/L (normal level, 0–0.4 g/L), and a decreased chloride level of 113.9 mmol/L (normal level, 118–129 mmol/L). The CSF intrathecal IgG synthesis was found to have markedly increased to 41.85 mg/24 h (normal level, <7.0 mg/24 h); also found were a high IgG index of 0.95 (normal level, <0.85) and high blood–brain barrier permeability (18.61×10^{-3}; normal level, $<5.0 \times 10^{-3}$). The CSF myelin basic protein level was high (4.02 μg/L; normal, <3.5 μg/L) but that of blood was normal. Blood biochemistry parameters, chest radiographs, head computed tomography and magnetic resonance imaging scans, skin tuberculin test, as well as CSF Gram-stained smear revealed no abnormalities. Cultures of CSF for bacterial, mycobacterial, viral and fungal organisms were also negative. PCR for cytomegalovirus, Epstein-Barr virus, type1 and 2 herpes simplex virus, enterovirus, adenovirus, coxsackie virus, toxoplasma gondii, treponema pallidum, and cryptococcus were negative in CSF.

HIV-1 serology was found to be positive by ELISA but weak positive by western blot. Fifteen days later, the western blot test became positive. The CD4+ cell count was 180 cells/mm^3 (normal, 706–1,125 cells/mm^3) at admission, with a marked decrease in the CD4/CD8 ratio of 0.12, and improved to 466 cells/mm^3 one month later. The plasma viral load was 9,903 copies/mL, while that of CSF obtained ten days before plasma collection was 95,575 copies/mL. One month later, his blood HIV-1 viral load was 9,895 copies/mL, but that of the CSF was only 260 copies/mL. The patient's condition improved steadily and he experienced complete resolution of the bilateral PFP after two months. As there was no evidence of any other underlying systemic illness, and based on his HIV antibody–negative report four months before admission, the patient was diagnosed with bilateral PFP combined with HIV-1 meningitis associated with ARS.

Discussion

Acute HIV-1 infection (AHI) is an early stage of HIV-1 infection. It is estimated that 40–90% of patients with AHI develop ARS, characterized by fever, myalgia, rash, headache, lymphadenopathy, and so on[1]. PFP can be the first symptom of HIV-1 infection, but bilateral PFP is extraordinarily rare[1] even among patients with AHI; only 17 cases (including the present case) have been reported. Aseptic meningitis was present in all of these reported patients, but plasma viral load test was performed for only six cases (35.29%), and only the present reported case underwent a CSF HIV-1 viral load test. To the best of our knowledge, this is the first report of simultaneous ARS, bilateral PFP, and aseptic meningitis with high CSF HIV-1 viral load.

The pathogenic mechanisms of PFP in AHI patients are not well understood. A nerve lesion directly resulting from HIV-1 or an immunologically mediated inflammatory polyradiculopathy have been proposed[4]. The CSF viral load is strongly correlated with neurological symptoms[5]. In our patient, bilateral PFP occurred with lymphocytic pleocytosis, increased proteins, and high CSF HIV-1 viral load, supporting the direct lesion hypothesis. Interestingly for this patient, the CSF HIV-1 viral load was almost ten times that of the viral load in plasma, which was obtained ten days later (CSF, 95,575 copies/mL vs. plasma, 9,903 copies/mL); the reason for this is not completely understood. One possible explanation is that the CSF viral load was transient and that it decreased after that of the plasma viral load during the acute HIV infection, decreasing to 260 copies/mL rapidly, and accompanying improvement of the clinical condition of PFP after one month.

Unilateral and bilateral PFP occurs with higher frequency in the HIV-infected population. The clinician should have a high index of suspicion of AHI when a patient presents with PFP and a recent flu-like illness, especially patients with high-risk behavior or who have been in endemic HIV areas. An accurate diagnosis of acute/early HIV infection is particularly important at the public health level, as approximately 50% of new sexual transmissions take place when a person is at the primary phase of infection. Moreover, early initiation of treatment and education to limit disease transmission is necessary.

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