ABSTRACT

Introduction: Bilateral facial nerve paralysis is a rarely reported but recognized complication that follows acute HIV-1 infection. HIV seroconversion illness or acute HIV infection is present in approximately 40-90% of primary HIV infected patients. Patients with acute HIV-1 infection typically present with fever, rash, myalgias, arthralgias, and lymphadenopathy. Most of these appear in two to four weeks following the initial infection. We report a patient experiencing HIV-1 seroconversion as described who presents with bilateral facial nerve paralysis. Related literature of this subject is also reviewed. Case Report: We report a 33-year-old Hispanic male, with no significant past medical history, who presented with bilateral facial nerve paralysis in the setting of a viral-like prodrome of fevers, malaise, muscle aches and sore throat. After a full workup, the patient was subsequently diagnosed with an HIV-1 infection. His facial paralysis resolved over a course of five months. Discussion: Unilateral and bilateral facial paralysis occur at over 100-fold greater frequency in HIV-infected patients. However, even among patients with an acute HIV infection, bilateral facial paralysis is exceedingly rare, as only 12 cases have been reported worldwide over the last 20 years. Because the onset of facial paralysis closely follows HIV-1 seroconversion, its presence can facilitate early diagnosis and treatment of those newly infected with HIV-1. Conclusion: In a patient who is at risk and presents with a new facial paralysis, HIV infection should be considered as a possible underlying etiology.

Keywords: Bilateral facial paralysis, HIV-1

INTRODUCTION

Acute HIV infection or primary HIV infection is the second stage that occurs soon after the incubation stage of the infection, usually two to four weeks after exposure to HIV-1. It is characterized by the body developing antibodies against the HIV antigen. Clinically, this manifests itself when the body mounts a symptomatic response to the viral infection. Influenza and mononucleosis-like symptoms including fever, malaise, myalgias, mouth sores, pharyngitis and generalized rash are some symptoms experienced by the majority (40-90%) of those with a primary HIV infection [1]. Those infected may experience all, some or none of these symptoms for an average course of 28...
days. Moreover, because of their non-specific nature, primary HIV infection is often missed. Occasionally, patients may also have neurological sequel including aseptic meningitis, neuropathy, encephalopathy, myelopathy and brachial neuritis. Bell’s palsy has also been reported in those chronically infected with HIV. However, facial Bell’s palsy experienced at the time of seroconversion is much more infrequent, and first reported by Wechsler et al. in 1989 [2].

CASE REPORT

We report case of a 33-year-old Hispanic male with an unremarkable past medical history who was admitted to the hospital with bilateral facial diplegia. Three weeks prior to admission, he experienced subjective fevers, sore throat and malaise. He also reported eruption of a vesicular rash on his forehead and right cheek and severe dull occipital headaches. The patient received a three day course of cefuroxime after which the rash resolved. One week prior to his admission, he noted onset of painless, right-sided facial paralysis. Initially, he was not able to open or move the right side of his mouth. Soon the weakness involved the entire right side of his face. A couple of days later, the weakness also involved the left side of his face. He reported difficulty in speaking, inability to close his eyes and hypoguesia. He was able to produce tears and saliva and denied hyperacusis or hypoacusis. He denied any swelling of the face, joint pain, chest pain, palpitations or the presence of circular rashes. In general, all of his symptoms were localized to his face (Figure 1).

On physical examination, he was alert and oriented to person, place, time and event. He was afebrile and the only abnormality was weakness of bilateral facial musculature. The rest of the neurological examination was unremarkable. Results of blood biochemistry, hematocrit and platelet counts were within normal limits. His white blood count was 6.2x10^9 cells/L (53% neutrophils, 41% lymphocytes and 6% monocytes). Liver enzymes were within normal limits. Blood cultures and seriological studies for infection with herpes simplex virus 1 and 2, human herpes virus 6, enterovirus, campylobacter virus, cytomegalovirus, hepatitis viruses A, B, C, D, E, Epstein-Barr virus, Toxoplasma gondii, Brucella species and syphilis were negative. Angiotensin-converting enzyme level was within normal limits. A lumbar puncture was performed and cerebrospinal fluid analysis revealed glucose content of 2.4 mmol/L, protein content of 1.8 g/L, and 39 lymphocytes/mm^3. A gram-stained smear and culture of CSF for bacteria and mycobacteria was negative.

In light of no explanation for his symptoms, the patient was assessed for risk factors for HIV infection. He denied intravenous drug use. He did engage in regular homosexual activity. Previous HIV screening tests were all negative, the most recent of which was nine months prior. A screening ELISA was HIV positive and this was confirmed by Western blot. CD3^+ count were 1646 cells/mm^3, CD4^+ count were 871 cells/mm^3 (41%) and CD8^+ count was 855 cells/mm^3 (40%). CD4^+/CD8^+ ratio was 1. The HIV-1 load was 6.5x10^7 copies/mL. An MRI of the head showed subtle enhancement of the bilateral fundal and labyrinth segments of the facial nerve. The patient was discharged on an eight day course of acyclovir and five day course of prednisone. Over a course of five months, his facial palsy completely resolved.

DISCUSSION

From the first time that the bilateral facial nerve paralysis was reported in 1989 to date, 12 patients with bilateral Bell’s palsy with acute HIV-1 infection have been reported [3]. This patient’s transient bilateral facial nerve paralysis most likely resulted from an acute HIV seroconversion syndrome and not from any other infection or malignant etiology. In addition, his aseptic meningitis could have contributed to his facial palsy. Cerebrospinal fluid abnormalities are commonly
reported in HIV-1 positive patients evaluated with unilateral and bilateral Bell's palsy [4] and it has been hypothesized that the delayed onset of such symptoms is indicative of a delayed host response to the infection and not the result of the virus itself [5]. Also, it has been postulated that the facial nerve is compressed in the fallopian canal secondary to the inflammation associated with autoimmune demyelination of neurons similar to a regional Guillain-Barre syndrome in response to HIV-1 infection of the central nervous system [6].

Bilateral facial nerve paralysis is more often due to systemic causes in comparison to unilateral paralysis and a wide differential diagnosis must be considered. The diseases often associated with bilateral peripheral paralysis are neurosarcoidosis, multiple idiopathic cranial neuropathies, brain stem encephalitis, benign intracranial hypertension, Guillain-Barre syndrome, bacterial meningitis, leukemia, syphilis, lyme disease and orofacial granulomatosis (Melkersson-Rosenthal syndrome) [7]. The annual incidence of unilateral Bell’s palsy is approximately 20-30/100,000 population and bilateral facial paralysis is even less common, occurring in only 0.3-2.0% patients with facial nerve palsy [7]. Both unilateral and bilateral facial nerve paralysis occurs with a 100-fold greater frequency in the HIV-1-infected population: 4.1% vs. ~0.04% in the population [8]. Even among HIV-1 patients, bilateral Bell’s palsy is extremely rare, as only 12 cases have been reported worldwide over the last 20 years.

CONCLUSION

This case and literature review demonstrates that it is important to consider acute HIV infection in the differential diagnosis of unilateral and bilateral facial nerve paralysis in someone who is sexually active and presents with a prior febrile prodrome and non-specific flu-like symptoms. If initial screening diagnostic tests are negative, ordering a more specific test such as the Western blot can be considered. Early diagnosis is critical as early treatment can reduce the morbidity associated with the disease.

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Author Contributions
Roman Leonid Kleynberg – Conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Critical revision of the article, Final approval of the version to be published
Joshua Leonid Kleynberg – Conception and design, Critical revision of the article, Final approval of the version to be published

Guarantor
The corresponding author is the guarantor of submission.

Conflict of Interest
Authors declare no conflict of interest.

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REFERENCES