



Acute Retroviral Seroconversion Presenting as Bilateral Facial Palsy

KEYWORDS

facial palsy , HIV, acute retroviral syndrome

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ABSTRACT *Bilateral facial palsy is of very infrequent incidence and generally has a systemic cause than local cause. The most common causes of bilateral facial palsy are neuro-sarcoidosis, Lyme disease, Guillain Barre disease and Syphilis. Acute retroviral seroconversion is a very rare cause of bilateral facial palsy. We present here a case of 30 year old married female developing bilateral facial palsy due to acute HIV infection and presented during window period and later was diagnosed with HIV infection by ELISA test.*

Introduction

Acute HIV syndrome is experienced by 40-90% of patients undergoing sero-conversion. The common features are fever, malaise, generalized body ache, headache and occasional lymphadenopathy which are features of most of the viremia. Major complications in form of encephalopathy, meningitis, encephalitis and neurological deficits can occur in acute HIV 1 infection. Bilateral facial palsy is a very rare neurological complication of acute HIV 1 infection. The first case of bilateral facial nerve palsy due to acute retroviral infection was documented by Wechsler and Ho in 1989.

Case report

30 year old married female presented inability to close both the eyes with loss of movement of facial muscles on both the sides since 5 days. She developed weakness on right side and within 3 days also affected left side. She had history of fever, myalgia and dull headache 2 weeks prior to the episode. There was no previous episodes of facial palsy. There was no history of any facial swelling or painful vesicular eruptions on the face or pinna. Patient had no history of any drug abuse, blood transfusion or any other high risk behavior. 9 months ago, the patient had medical termination of pregnancy followed by check cur-retage at a primary health center in a village. There was no history of any chronic medical illness.

On physical examination, bilateral lower motor neuron palsy of seventh cranial nerve was present. There was no other neurological deficits. There were no signs of meningitis or meningism. There was no cervical lymphadenopathy or rash around the pinna. No other abnormality was detected on systemic examination.

Haematological investigations revealed total leukocyte count of $9.3 \times 10^9/\text{ml}$ with 37% neutrophils and 61% lymphocytes. Serological tests for Herpes simplex virus 1 and 2, Epstein-Barr virus, Cytomegalovirus, Human Herpes Virus 6, Enterovirus, Hepatitis viruses A, B, C, D, E, Toxoplasma gondii, Brucella species and Syphilis were negative. Serum angiotensin converting enzymes were 56 (normal range -8 -50 U/l). Chest X Ray showed no parenchymal lesion or any hilar lymphadenopathy. CSF studies showed glucose level 2.8 mmol/L and protein level of 2.2 g/L with lymphocytes 38 per cubic millimeter. CSF was negative for any gram positive or negative bacteria, mycobacteria and Cryptococcus. Patient had underwent HIV testing 6 months back which was negative but due to high suspicion, it was repeated which came out to be positive for HIV 1.

CD4+ counts was $455/\text{mm}^3$ and CD8+ counts was $1733/\text{mm}^3$. Viral load was not done. No significant abnormality was detected in MRI brain. Three drug regimen anti-retroviral therapy was started consisting of Zidovudine, Lamivudine and Nevirapine. Patient was also given a course of steroids with acyclovir simultaneously. Facial nerve functioning was completely regained at end of 4 weeks.

Discussion

HIV has emerged as rapidly growing endemic in last few decades. These patients present with variable different clinical features. Isolated Neuropathy and aseptic meningitis are very rare manifestations for retroviral disease. Facial palsy (seventh cranial nerve - lower motor neuron palsy) can occur during the window period of retroversion. The annual estimated incidence of Bell palsy in immunocompetent subjects is 20-30 patients per 100,000 population.¹ Bilateral facial paralysis is much less common than unilateral paralysis, and occurs in only 0.3%-2% of patients. Unilateral and bilateral facial nerve paralyzes occur with a 100-fold greater frequency in the HIV-1-infected population: 4.1% vs. ~0.04% in the general population.²

Bilateral facial generally has a systemic cause than local cause as seen in cases of unilateral facial palsy. Hence in cases of bilateral facial palsy, there should be a comprehensive search for the underlying causes. The most common causes of bilateral facial palsy are Sarcoidosis, Lyme's disease, Neurosyphilis and Guillain Barre syndrome. The other causes are benign intracranial hypertension, brainstem encephalitis, bacterial meningitis, leukemia and Melkersson-Rosenthal syndrome (oro-facial granulomatosis syndrome).^{3,4} Serological work up for antibodies against the pathogen Lyme's disease and syphilis is important. Serum angiotensin converting enzymes level are increased drastically in cases of sarcoidosis but is known to be marginally raised in cases of acute retroviral syndrome as was the case in our patient. Chest x ray can be helpful in cases of sarcoidosis which may reveal hilar lymphadenopathy and endo-bronchial biopsy will prove the granulomatous disease proving sarcoidosis. Cerebrospinal fluid studies are essential to rule out bacterial meningitis and Guillain Barre syndrome. In bacterial meningitis, sugar will be low with presence of pus cells and gram stained smears will reveal cocci or bacilli. In Guillain Barre syndrome, there is "albumin-cytological dissociation" in which there is an elevated protein levels with low number of white blood cells in CSF, this distinguishes GBS from lymphoma and poliomyelitis where both the protein and cell counts are elevated.

The cause and the exact mechanism of HIV 1 induced neuropathy is not clear. Various different theories has been proposed amongst which two school of thoughts have gained popularity. First one suggest direct neurological insult by HIV itself and the second theory suggest role of immunological reaction to HIV type 1 resulting in inflammation secondary to autoimmune demyelination of facial nerve causing nerve compression in the narrowest segment of the nerve (exit from internal auditory canal).⁵ The second theory can explain the time lag between the appearance of general viral symptoms and onset of the facial palsy. This time lag is due to time taken by the immune system to develop antibodies against the retrovirus which actually cross reacts with the neurological structures.

Serrano et.al (2006) reviewed 12 patients starting from 1989 till 2006, they concluded that The median interval between the onset of symptoms of HIV-1 infection and the development of Bell palsy was 15 days (range, 2–180 days). Aseptic meningitis was present in all patients for whom Bell palsy was reported, and a maculopapular rash was present in 4 patients (36.4%). All but 2 patients had a CD4+ cell count 1500 cells/mm³, and the range of values of the CD4+:CD8+ cell ratio was 0.09–0.49 . Two patients had additional neurological symptoms when a diagnosis of Bell palsy was made. No patient died. Only 1 patient received antiretroviral therapy. All other patients except 1 (8.3%) experienced recovery with transient residual Bell palsy over a period of 2–24 weeks.⁶

There has been no protocol laid down for management of facial palsy due to acute HIV 1 infection. There has been no trial for the assessing the efficacy and safety of steroids, antiretroviral drugs and acyclovir in these cases. It is been argued that use of long term steroids, as used in the management of Bell's palsy , can result in life threatening diseases in immunocomprised patients. But the risk of developing ophthalmic and further neurological complications outweigh the risks of use of steroid. Hence short course of steroid can be used in cases of facial palsy secondary to acute HIV 1 infection. We suggest use of acyclovir and start anti-retroviral therapy which help in reducing the risk of herpes zoster re-activation and reduce HIV viral load respectively.

Conclusion

Acute retroviral syndrome should be considered as a differential diagnosis in cases of bilateral facial palsy especially in cases where serological test for bacterial and autoiimmune disorder are negative. Prompt diagnosis and early initiation of treatment reduces the duration of recovery and prevent other complication secondary to HIV infection.

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