

Facial Diplegia and Vestibular Neuritis Secondary to HIV Seroconversion Syndrome

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Peripheral nerve involvement is a frequent complication of Human Immunodeficiency Virus (HIV) infection¹. Among cranial nerves, the seventh is the most commonly affected². However, facial diplegia is a rare clinical entity in the HIV infected population. We recently observed a case that is of particular interest, since the patient developed facial diplegia and unilateral vestibular neuritis revealing HIV infection.

CASE REPORT

The patient is a 60-year-old heterosexual male with a past medical history of morbid obesity and chronic obstructive pulmonary disease. He had been in his usual state of health until approximately four weeks prior to admission, when he developed low grade fever, rhinorrhea, sore throat and diffuse arthromyalgias. Fever resolved promptly after treatment with amoxicillin and clavulanic acid. Later on he was admitted to our institution with a five-day history of constant dizziness, unsteadiness and imbalance. By the time of admission he additionally experienced perioral numbness followed suddenly by lack of facial movements, with failure of both eyes to close and difficulty in sucking, smiling and talking. Neurological examination revealed bilateral peripheral facial palsy, right-deviation tendency in past pointing and Quix tests, and mild unsteadiness with gait deviation to the right. The remainder of physical and neurological examination was normal, including intact reflexes. Laboratory studies disclosed an erythrocyte sedimentation rate of 51 mm/h. White blood cell count was 12.4×10^9 cells/l, with 35.6% neutrophils, 48.6% lymphocytes and 12.3% monocytes. Red blood cell and platelet counts were within normal limits. Autoantibody screening tests and blood biochemistry parameters, including angiotensin converting enzyme, were also normal. Cerebrospinal fluid (CSF) contained 30 cells/ μ l with 93% lymphocytes, protein 1.64 g/l and glucose 2.6 mmol/l (serum glucose 6.7 mmol/l). Cytology was negative. No organism was seen on Gram-stained smear, and cultures of CSF for bacterial, mycobacterial, viral and fungal organisms were also negative. Polymerase chain reaction and serologies for citomegalovirus, varicella zoster virus, Epstein-Barr virus, type 1 and 2 herpes simplex virus, *Toxoplasma gondii*, *Cryptococcus*, *Brucella*, *Treponema pallidum*, and *Borrelia burgdorferi* were negative in both serum and CSF. Human Immunodeficiency Virus testing was positive by ELISA and was confirmed by Western blot. CD4⁺ cell count was 252 cells/ μ l and CD8⁺ cell count was 5027 cells/ μ l, with CD4/CD8 ratio at 0.05. Viral RNA load was 197 000 copies/ml. A gadolinium enhanced brain magnetic resonance imaging showed normal findings. Electrophysiological study confirmed severe impairment of

trigemino-facial reflex, with no motor evoked potentials recorded from the orbicularis oculi muscle of both sides after stimulating the supraorbital nerve. No evidence of a generalized demyelinating polyneuropathy was found on nerve conduction studies. Since there was no evidence of any other underlying systemic illness, the diagnosis of right vestibular neuritis and facial diplegia in relation to HIV acute seroconversion syndrome was made. After five days unsteadiness and facial palsy steadily improved, with complete resolution of neurological signs within the following week. At three-months follow-up the patient remained free of all neurological features.

DISCUSSION

Bilateral facial palsy, defined as the appearance of paresis of the contralateral facial nerve within 30 days of the onset of the first side³, is due to a systemic cause in a much higher incidence than unilateral palsy. The diseases most commonly associated with facial diplegia are Guillain-Barre syndrome, multiple idiopathic cranial neuropathies, brainstem encephalitis, Melkersson-Rosenthal syndrome, syphilis, sarcoidosis, Lyme disease, bacterial meningitis, poliomyelitis, Epstein-Barr Virus and other herpesvirus infections, leukemia, meningeal carcinomatosis, pontine tumours, Moëbius syndrome, idiopathic intracranial hypertension and, more rarely, HIV infection^{4,5}. Although both unilateral and bilateral facial paralyses occur with a greater frequency in HIV infected patients than in the general population, bilateral palsy is exceedingly rare even among them⁵. Seventh nerve involvement is the most frequent cranial neuropathy associated with HIV². As in our patient, it mainly occurs in the early stages of the disease, in the setting of lymphocytic pleocytosis and antibodies directed against HIV in CSF, supporting the hypothesis of a direct lesion of the facial nerve by the neurotropic virus⁵; however, an immunologically mediated inflammation of the cranial nerves has also been suggested as a possible pathophysiologic mechanism, given the similarities with other demyelinating peripheral neuropathies associated with HIV seroconversion⁶. Other causes of facial palsy such as Ramsay-Hunt syndrome, syphilis, type-2 herpes

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simplex infection, mononeuritis multiplex, and lymphomatous or sarcomatous infiltration of the nerve, must be considered in the later stages^{6,7}. Our case is original in that facial palsy appeared and resolved simultaneously in both sides, in a complete symmetrical way. Moreover, its association to vestibular neuritis revealing HIV infection is a very rare clinical picture, which could have been easily misdiagnosed due to its autolimited course. To the best of our knowledge, this is the first report of simultaneous facial diplegia and vestibular neuritis revealing HIV infection. This diagnosis could be made since the extensive search for other causes was negative, serological testing for HIV infection was positive and all symptoms resolved spontaneously.

Without a high index of suspicion, physicians may miss the diagnosis of acute HIV infection. Symptoms, if present, are nonspecific and resolve spontaneously without treatment. Moreover, patients generally have no findings on physical examination except for possible lymphadenopathy. This report emphasises that cranial nerve involvement such as facial palsy or vestibular neuritis can be the first symptom of HIV infection. Therefore HIV should be considered in the investigation of these entities, more concretely facial diplegia, especially in patients with high-risk behaviours even in absence of the mononucleosis-like syndrome. Given that patients are typically highly infectious during acute HIV due to a very high viral load⁸, the identification of these patients, who are unaware of their seropositive status, is clearly important from a public health perspective.

REFERENCES

1. Parry GJ. Peripheral neuropathies associated with human immunodeficiency virus infection. *Ann Neurol*. 1988;23 Suppl: S49-53.
2. Casanova-Sotolongo P, Casanova-Carrillo P, Casanova-Carrillo C. Diseases of the peripheral and visual nervous system during infection with human immunodeficiency virus. *Rev Neurol*. 2003;37:481-5.
3. Gevers G, Lemkens P. Bilateral simultaneous facial paralysis: differential diagnosis and treatment options. A case report and review of literature. *Acta Otorhinolaryngol Belg*. 2003;57: 139-46.
4. Keane JR. Bilateral seventh nerve palsy: analysis of 43 cases and review of the literature. *Neurology*. 1994;44:1198-202.
5. Serrano P, Hernandez N, Arroyo JA, de Llobet JM, Domingo P. Bilateral Bell palsy and acute HIV type 1 infection: report of 2 cases and review. *Clin Infect Dis*. 2007;44:57-61.
6. Abboud O, Saliba I. Isolated bilateral facial paralysis revealing AIDS: a unique presentation. *Laryngoscope*. 2008;118:580-4.
7. Kohler A, Burkhard P, Magistris MR, Chofflon M. Isolated peripheral facial paralysis and HIV infection: 7 cases. *Rev Neurol*. 1995;151:332-7.
8. Daar ES, Moudgil T, Meyer RD, Ho DD. Transient high levels of viremia in patients with primary human immunodeficiency virus type 1 infection. *N Engl J Med*. 1991;324:961-4.