Alerts, Notices, and Case Reports

Bilateral Bell's Palsy and Aseptic Meningitis in a Patient With Acute Human Immunodeficiency Virus Seroconversion

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SINCE FIRST REPORTED in 1985,[1] the acute retroviral syndrome has been increasingly recognized in patients infected with the human immunodeficiency virus (HIV). Cases typically present with fever, myalgia, headache, rash, and lymphadenopathy and can easily be mistaken for mononucleosis or influenza. Occasionally neurologic complications—including aseptic meningitis, encephalopathy, neuropathy, myelopathy, and brachial neuritis—develop in association with primary infection. Except for the encephalopathy, which occurs simultaneously during the acute phase, the other manifestations tend to be seen about three weeks after the onset of the primary illness.[2] Bell's palsy has been reported during these episodes and has also been seen in patients already known to be HIV-seropositive.[3,4] Wechsler and Ho in 1989 first reported the case of a patient with bilateral Bell's palsy at the time of seroconversion.[5] Cerebrospinal fluid findings were not reported because no lumbar puncture was done. We report the case of a similar syndrome in a patient with acute seroconversion and the additional finding of aseptic meningitis.

Report of a Case

The patient, a 32-year-old male homosexual intravenous drug injector, was seen because of bilateral facial weakness. Four weeks before being seen he had been admitted to hospital because for three days he had had abdominal pain, fever, sweats, chills, and lethargy but no headache. An examination showed a temperature of 39.1°C, mild abdominal tenderness, and diffuse lymphadenopathy. No rash was noted. He had no nuchal rigidity or other neurologic abnormalities. His leukocyte count was 3.3×10^9 per liter (3,300 per μl) with 0.47 polymorphonuclear leukocytes, 0.34 bands, and 0.12 lymphocytes. An absolute $CD4^+$ count was 390 cells $\times$ 10^6 per liter, the $CD4^+$:$CD8^+$ ratio was 1.4, and an enzyme-linked immunosorbent assay (ELISA) test for HIV was negative. Over the next few days, all of his symptoms resolved, and he was discharged home without a definitive diagnosis.

One week before we saw him, he noted the painless onset of right-sided facial weakness. Three days later he noted that the weakness now involved the left side of his face. He otherwise felt well and had no other complaints, but on questioning said that he did have a mild nonlocalized headache. He lived in Sacramento, California, and had no recent travel history. He was admitted to the neurology service for evaluation.

On examination, he was afebrile, appeared comfortable, with the only abnormality being bilateral peripheral facial musculature weakness, right greater than left, with decreased taste discrimination on the right anterior two thirds of the tongue. Detailed neurologic, ophthalmologic, and mental state examinations elicited no other abnormalities. There was no nuchal rigidity. Laboratory studies included unremarkable values on a liver panel, an erythrocyte sedimentation rate of 9 mm per hour, and a leukocyte count of 8.6×10^9 per liter with 0.38 polymorphonuclear leukocytes, 0.18 bands, and 0.31 lymphocytes. A chest x-ray film was normal, and heterophile and VDRL tests and blood cultures were negative. Head computed tomographic scan and magnetic resonance imaging with gadolinium were normal. Lumbar puncture revealed a cerebrospinal fluid (CSF) glucose level of 3.1 mmol per liter (55 mg per dl; a blood glucose level was 4.3 mmol per liter [78 mg per dl]), protein 1.66 grams per liter (166 mg per dl), leukocyte count 78×10^6 per liter (0.99 lymphocytes), and no erythrocytes. No organisms were seen on Gram's stain, and cultures were negative for bacterial, fungal, viral, and mycobacterial organisms. The CSF specimen was tested for *Coccidioides immitis* antibodies, *Toxoplasma gondii* antibodies, *Cryptococcus neoformans* antigen, and VDRL, all of which were negative. Serum antibody tests were negative for *Borrelia burgdorferi*, herpes simplex virus, and varicella zoster virus. A Tensilon test was negative. Electromyography and nerve conduction studies were interpreted as consistent with primarily peripheral facial nerve lesions, right greater than left, without evidence of demyelination or abnormality of the neuromuscular junction. The $CD4^+$ count was now 718 cells $\times$ 10^6 per liter, with a $CD4^+$:$CD8^+$ ratio of 0.3. A repeat ELISA and Western blot assays were both positive for HIV type 1.

During a period of observation in the hospital, without antiretroviral or other therapies, he had spontaneous lessening of his facial palsy and resolution of his headache. A repeat lumbar puncture a week after the first one continued to show lymphocytic pleocytosis. By four weeks after his symptoms began, the palsy had nearly completely resolved, and he continued to feel well.

Discussion

This patient's transient facial diplegia probably di-

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ABBREVIATIONS USED IN TEXT

CSF = cerebrospinal fluid
ELISA = enzyme-linked immunosorbent assay
HIV = human immunodeficiency virus

rectly resulted from his acute HIV seroconversion and was not a consequence of any other infectious or malignant process. His initial hospital admission was almost certainly due to acute HIV infection with its characteristic transient decline in CD4+ cell count followed by the development of seropositivity and a rebound in T-helper cells. In addition, he had evidence of an ongoing aseptic meningitis, which may have been the cause of the palsy. Cerebrospinal fluid abnormalities were reported in all seven previously known HIV-positive patients evaluated for unilateral Bell's palsy[4] and are a common finding in HIV-infected patients without focal neurologic abnormalities.[6,7] It has been postulated in cases like this that the delayed occurrence of neurologic abnormalities until after the acute primary illness has resolved may reflect an exaggerated host response to the infection rather than a direct result of the virus itself.[2] In contrast to the progressive HIV-related neurologic abnormalities that can develop later in the disease course, these acute complications are much more likely to resolve spontaneously in a few weeks, as seen in this patient.[8,9]

The nearly normal CD4+ count during this patient's second hospital admission is also a good example of the possible lack of correlation between the degree of immune dysfunction and the onset of acute HIV neurologic disease. In a series from Africa, Belec and colleagues observed that "facial palsy can affect healthy, symptom-free HIV carriers with only a slight decrease in immunological indices."[3(p1421)] It has been hypothesized that neurotropic HIV strains differ biologically from those linked to rapid immune dysfunction.[8] Therefore, in a patient at risk, despite a normal CD4+ count or lack of other systemic symptoms, HIV infection should be considered as the possible cause of newly occurring facial palsy. If initial screening serologic tests are negative, consideration should be given to repeating them later, ordering a Western blot test, or doing one of the new, more sensitive assays such as the polymerase chain reaction.[10]

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Recurrent Angioedema and Urticaria

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ANGIOEDEMA is a potentially life-threatening condition in which a well-demarcated localized edema involves the deeper layers of mucous membranes and the skin, including the subcutaneous tissue. During an acute episode, patients may have edema of all or part of an extremity, the face, tongue, epiglottis, larynx, respiratory tract, and gastrointestinal tract. Involvement of the respiratory tract can cause dyspnea, dysphasia, stridor, wheezing, and respiratory arrest.

We describe the case of a patient with recurrent angioedema of the face, neck, and pharynx severe enough to require tracheal intubation and an urticarial skin rash of the distal upper and lower extremities. This case illustrates the possible severity of angioedema and the need to investigate thoroughly cases of recurrent angioedema for causative factors.

Report of a Case

The patient, a 60-year-old woman, had hypertension that had been treated with enalapril maleate, an angiotensin-converting enzyme (ACE) inhibitor, for 20 months before admission; chronic polyarthritis treated with piroxicam; and macrocytic anemia associated with chronic alcohol abuse. Since starting the enalapril therapy, the patient was evaluated on six different occasions in a military medical center for recurrent angioedema of the face and urticaria on the upper and lower extremities. Each time her symptoms were relieved with epinephrine, antihistamines, and corticosteroids. The patient did not have sensitivity to sunlight, cold, or heat and had no apparent renal, hepatic, intestinal, or central nervous system dysfunction. Her family history was negative for an-

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