

## Bilateral Peripheral Facial Palsy in a Patient with Human Immunodeficiency Virus (HIV) Infection

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Neurological complications are important causes of morbidity and mortality in patients with human immunodeficiency virus (HIV) infection. They can occur at any stage of the disease and can affect any level of the central or peripheral nervous systems. In the literature, several cases of HIV-associated facial paralysis have been reported; however, bilateral facial palsy is rarely reported. In this paper, we present the first case in Korea, of a bilateral facial palsy occurring as the first clinical manifestation of HIV infection.

**Key Words:** HIV infection, facial paralysis, central nervous system

### INTRODUCTION

With the increase of HIV infection many opportunistic infections are now being recognized. Among them, neurologic complications represent an important cause of morbidity and mortality in patients with HIV infection,<sup>1</sup> and can occur at all stages of infection, involving any level of the central or peripheral nervous system.<sup>2</sup> Complications include aseptic meningitis, meningoencephalitis, encephalitis, and peripheral nerve palsy. Peripheral neuropathy is only 5 to 20% of total neurological complications,<sup>3,4</sup> and facial nerve palsy, especially bilateral, is quite rare.<sup>5</sup>

Although there have been several reports of facial palsy as a late complication of HIV infec-

tion,<sup>6,7</sup> it has mainly been considered an early stage neurological complication. Two French studies reported<sup>8,9</sup> a 15.4% HIV seropositivity rate in peripheral facial palsy cases, with an average age of 34 years.

In this report, we present a case of bilateral facial palsy as the initial clinical manifestation of HIV infection. To the best of our knowledge, this is the first case in Korea.

### CASE REPORT

A 26-year-old heterosexual male with no significant past medical history presented with a three-day history of headache, nausea, and vomiting. Two weeks prior, he developed fever, chills, rhinorrhea, and sore throat. His first sexual partner was a prostitute nine years ago; since then he has had approximately thirty more sexual contacts. He denied history of intravenous drug abuse or transfusion. At admission, temperature was 37.4 °C, blood pressure 120/80 mmHg, pulse 80 beats/min, and respiration 20 breaths/min. Physical examination was unremarkable except for neck stiffness. There were no abnormalities in mental status, cranial nerve function, or motor and sensory function on neurologic examination.

Complete blood cell count included white blood cell count of 12,290/μL with 47.2% neutrophils and 38.0% lymphocytes, hemoglobin of 14.9 g/dL, and platelet count of 350,000/μL. Cerebrospinal fluid (CSF) analysis revealed opening pressure of 210 mm H<sub>2</sub>O, and leukocyte count of 70 cells/μL with 89% lymphocytes. CSF protein was 73.7 mg/dL and glucose was 64 mg/dL (serum glucose 143

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mg/dL). No organism was seen on Gram stain, and cultures were negative for bacterial or fungal organisms. Tuberculosis polymerase chain reaction, smear, and culture, as well as cryptococcal antigen in CSF were negative. Cytomegalovirus, Epstein-Barr virus, herpes simplex virus, varicella zoster virus, and *Treponema pallidum* antibody tests were negative in both serum and CSF.

Brain magnetic resonance imaging (MRI) showed normal findings. Initially, conservative management for aseptic meningitis was started, however, on the sixth day, right facial palsy (peripheral type) developed (Fig. 1), and on the eighth day, left facial palsy developed.

At that time, HIV infection was diagnosed by enzyme-linked immunosorbent assay (ELISA) and Western blot; CD4+ T lymphocyte count was 327/ $\mu$ L and viral load was 350,000 copies/mL.

HAART (Zidovudine<sup>®</sup>, lamivudine<sup>®</sup>, Kaletra<sup>®</sup>) was started for HIV neuropathy. After seven days, generalized symptoms improved, except for facial palsy resolution, and the patient was discharged to be followed up on in the outpatient clinic.

## DISCUSSION

Literature review confirmed that peripheral facial palsy can occur at any stage of HIV infection,<sup>10</sup> however, it is more likely to be present in healthy HIV carriers than in patients with AIDS.<sup>7</sup> Though there have been several reports of facial palsy as a late complication of infection,<sup>1</sup> this neuropathy occurs mainly in the early stages of



**Fig. 1.** Patient with right side facial palsy on sixth day of admission.

virus infection, as in our patient.<sup>7</sup> Thus, peripheral palsy can be divided into two types according to etiology; an early stage-related palsy and a late stage-related palsy.<sup>6</sup> In stages I and II of HIV infection, patients may develop either Bell's palsy or Guillain-Barre syndrome. In stages III and IV, as cellular immunity begins to decline, herpes zoster-related facial palsy, facial nerve involvement secondary to meningeal lymphomatosis, and peripheral facial palsy, as manifestation of various chronic peripheral neuropathy, may also occur.<sup>10</sup>

HIV is generally neurotropic and virus localization in the facial nerve or geniculate ganglion can cause intraneural edema and fiber swelling, resulting in increased signal enhancement of the affected facial nerve in brain MRI.<sup>11</sup> However, brain MRI of our patient showed normal findings.

Histological findings of nervous tissue in early stage associated peripheral facial palsy include a degenerative and non-suppurative inflammatory process, however, pathophysiology remains obscure.<sup>1</sup>

Several authors suggest that host immune response to infection may trigger facial nerve compression, degeneration, and paralysis.<sup>6,12,13</sup> Yet, others authors disagree, and suggest that the relationship between Bell's palsy and HIV infection may be coincidental, especially in countries with high HIV prevalence.<sup>2</sup>

Kohler et al.<sup>14</sup> reported seven cases of isolated peripheral facial palsy, with one being a bilateral palsy. This patient had also presented with bilateral facial palsy, with no evidence of trauma, otitis media, parotitis, tumor, or any other underlying systemic illness, and the facial palsy was considered to be secondary to HIV infection.

It is apparent that there is an increase in facial palsy incidence proportionate to the increase of HIV infection.<sup>15</sup> Since facial palsy is considered an initial clinical presentation of HIV infection, it can aid in early diagnosis. As early treatment and prevention of HIV dissemination are recommended, it is important to recognize the first signs of HIV infection; and it should be considered as a possible etiology of facial palsy, especially in young age groups.

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