A Case of Trigeminal Neuropathy

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Trigeminal neuropathy is a disorder characterized by the rapid onset of sensory deficit in the distribution of the trigeminal nerve[1,2]. It may be accompanied by pain, concomitant motor paresis or taste disturbance. There are many different causes of trigeminal neuropathy, i.e., neoplasms, infections, connective tissue disorders and multiple sclerosis[2]. Among these, only two reports related to viral infection are available[3,4]. However, there have been no reports which indicate that the trigeminal neuropathy is related to the infection by Epstein-Barr (EB) virus. The case reported herein is considered to provide evidence of the possible relationship of this condition to EB virus.

Report of a Case

A 47-year-old man, complaining of painless numbness on the left side of the face and taste disturbance on the ipsilateral side of the tongue, came to our hospital on November 20, 1986. On September 25, 1986, he had suddenly noticed numbness on the left side of his face and taste disturbance of the tongue when he was washing his face. Although he consulted a neurologist and an otolaryngologist, the etiology of his disorders remained unclear. Two months after the onset of symptom, he consulted our clinics. On the day of his consultation, neurological examination revealed disturbance of the sensation of touch, pain and temperature (cold and warm) in the area of the distribution of the left trigeminal nerve. The sensory disturbance was more pronounced in the distribution of the second and third divisions of the trigeminal nerve than in the first division of the nerve. Taste was lost in the anterior two-thirds of the left side of the tongue. Electrogustametry showed remarkable impairment of function of taste in the area of distribution of the left chorda tympani. An electromyogram of the left masseter muscle revealed signs of partial disturbance on biting. The right trigeminal nerve as well as all the other cranial nerves were intact. The patient had no particular past history except for diabetes mellitus. Laboratory examination revealed a fasting blood sugar level of 135 $mg \cdot dl^{-1}$ and a 75-g oral glucose tolerance test later disclosed a diabetic curve. No glucose was present in the urine. He had been treated with gliclazide 20 $mg \cdot day^{-1}$. Cerebrospinal fluid examination, roentgenogram and computed tomography of the head revealed no abnormalities. Erythrocyte sedimentation rate was normal (11 $mm \cdot hr^{-1}$), and rheumatoid factor and C reactive protein were negative. The serum was positive for anti-EB virus antigen (IgG) at 1:640 dilution. The antibodies for other neurotropic viruses (Herpes Simplex viruses, Cytomegalovirus and Varicella-Zoster virus) were negative.

Four days after his admission, he told the alleviation of sensory disturbances on the re-

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gion of the first division of left trigeminal nerve. Sensory disturbances gradually disappeared, and one month later they remained around the region of left upper lip and brow. Taste disturbance remained unchanged for two weeks and it gradually recovered. Two months later, sensory and taste disturbances completely disappeared. Electrogustametry also revealed the recovery of taste disturbance. Diabetes was well controlled with a diet of 1500 kcal·day^{-1} and gliclazide 20 mg·day^{-1}. The fasting blood sugar level was around 120 mg ·dl^{-1} and urine sugar was almost negative. The anti-EB virus titer remained at 1:640 for one month and decreased to 1:320 when the patient's symptoms had completely disappeared. The patient received twenty four stellate ganglion blocks with 5 ml of 1% mepivacaine during his admission in hospital for about 1 month.

Discussion

The common causes of trigeminal disorder which may produce facial numbness are neoplasm arising from the nasopharynx, the base of the skull, the cerebellopontine angle or the region of the gasserian ganglion, facial or dental trauma, viral infection, diabetic neuropathy, connective tissue disease and multiple sclerosis[2]. Among these, the most probable cause was neoplasm. Horowitz[5] reported trigeminal neuropathy associated with neoplasm. In his report, 24 cases out of 64 involved neoplasm. However, in the present case, laboratory investigation revealed no evidence of tumor. In multiple sclerosis, facial numbness is the initial symptom in 2–3% of patients[6]. The disease is diagnosed on the basis of involvement of other non-contiguous central nervous structures. The present patient had no disorders of the cranial nerves other than the trigeminal nerve. Connective tissue disease may initially be manifested as facial numbness. Bennett et al.[7] reported that about 10% of cases of mixed connective tissue disease were accompanied by trigeminal neuropathy. Our laboratory findings also excluded the possibility of connective tissue disease.

In cases of diabetic mononeuropathy, one of the cranial or spinal nerves may be involved[8]. Diabetic mononeuropathy tends to occur in mildly affected patients, has a short duration and is independent of the fasting blood glucose level. However, the cranial nerves most affected in diabetic mononeuropathy are the third, fourth, seventh and sixth cranial nerves[8]. Only two reports on trigeminal neuropathy associated with diabetes mellitus have appeared[9,10].

The relationship of viral infection to trigeminal neuropathy was first postulated by Blau et al[1]. Fisher[3] reported a case of trigeminal neuropathy which could have been interpreted as subacute herpes simplex ganglionitis. Mandal and Allbeson[4] also reported that trigeminal neuropathy occurred after typical viral hepatitis. In the present case, the antibody (IgG) for EB virus antigen was positive at a titer of 1:640, while those for other neurotropic viruses were negative. These findings suggest that facial numbness might have been caused by EB virus infection.

In summary, we reported a case of trigeminal neuropathy with painless numbness on the left side of the face and taste disturbance on the ipsilateral side of the tongue. The patient had no relevant past history except for diabetes mellitus. Two months later, the patient completely recovered from the accompanying sensory disturbances. Serological tests for neurotropic viruses revealed the possible relationship of this condition to Epstein-Barr virus.

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