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Herpes Zoster Ophthalmicus and Delayed Contralateral Hemiparesis

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Central nervous system is often involved by herpes zoster but it is very rarely seen that contralateral hemiparesis or hemiplegia developed after herpes zoster ophthalmicus. We report a case of herpes zoster ophthalmicus followed by the delayed contralateral hemiparesis. A 33-year-old man developed acute cerebral infarction and resultant right hemiparesis 44 days after herpes zoster ophthalmicus in the left side. Brain CT disclosed hypodense area in the left basal ganglia. Cerebral angiography revealed segmental narrowing of M1 portion of the right middle cerebral artery.

Key Words: *Herpes zoster ophthalmicus, contralateral hemiparesis, cerebral infarction, direct invasion, granulomatous angitis.*

INTRODUCTION

Herpes zoster ophthalmicus(HZO) associated with delayed contralateral hemiparesis or hemiplegia is such a rare clinical event that about 56 cases have been reported(Luigi et al., 1987) since the original description by Dumary in 1896(Baudouin and Lantuéjoul, 1919). Temporal dissociation between the onset of HZO and that of contralateral hemiplegia was in the range of 1 week to 6 months (Reshef et al., 1985). Although it is controversial, the pathogenesis of this crossed zoster syndrome is believed to be granulomatous angitis (Kolodny et al., 1968, Sato et al., 1971, Rosenbulum and Hadfield, 1972, Linnemann and Alvira, 1980, Hilt et al., 1983). For a definite diagnosis of zoster arteritis, in a strict sense, biopsy of intracranial vessel is required. But it is also possible to document the diagnosis of contralateral hemiparesis or hemplegia complicating HZO by cerebral angiography or brain CT.

The purpose of this paper is to describe the clinical, angiographic, and CT scan features in a patient presenting with contralateral hemiparesis following HZO.

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CASE REPORT

On November 3, 1986, this 33-year-old, right handed man was admitted to Hanyang University Hospital for the evaluation of headache and speech disturbance. On August 10, gnawing left crainal pain developed suddenly followed by HZO in the distribution of the left ophthalmic division of the trigeminal nerve. He noticed facial weakness, right hemiparesis, and slurred speech on the morning of september 23.

Right hemiparesis had been almost disappeared but considerable pain was noticed when he was seen by us. On admission, vital signs were stable. There were no audible carotid bruits bilaterally. There were multiple brown-colored depressed scars in his left forehead. Neurologic examination revealed that speech was dysarthric, visual acuity was O.D.: 20/20,

and O.S.: 20/25. Slit lamp examination showed a few cells, flares and subendothelial corneal opacities in left eye. Both fundi were normal. There were no limitations of motion in all extraocular muscles. Corneal sensibility was not decreased. A central type facial weakness was present in the right face. There were no sensory deficits. No conspicuous weakness of the extremities was noted. Deep tendon reflexes were hyperactive and plantar response was extensor on the right side. No meningeal irritation signs were noted.

Routine laboratory data including CBC, SMA, electrolyte, urine analysis, lipid study, and EKG were all within normal limits. EEG showed no abnormal findings. Pre and postcontrast CT scan revealed a round hypodense area in the left globus pallidus and internal capsule (Fig. 1). Lumbar puncture revealed that an opening pressure of 130 mmH20 of clear fluid with a cell count of 1 lymphocyte and 2 red blood cells/ml, protein 16 mg%, sugar 65 mg%. Angiography was refused. During his hospitalization of 9 days, speech disturbance and weakness of the right face was noted to be slightly improved.

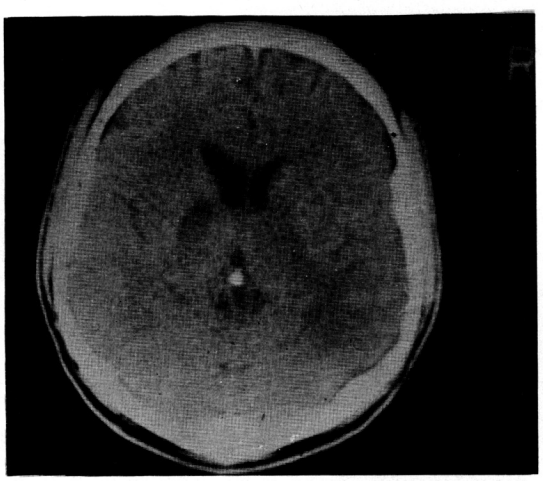

Fig. 1. Computerized tomography showed a round hypodensity in the left globus pallidus and internal capsule.

He was readmitted via outpatient clinic due to severe intolerable headache on January 6. He was depressed. His speech was dysarthric. Visual acuity in his left eye was further decreased to 20/40. There were no cells and flares but subendothelial opacities were still present. Right facial weakness was also remained. Deep tendon reflexes were still increased but plantar response was changed into flexor in the right side. Left carotid angiogram revealed segmental narrowing of proximal horizontal portion of the middle cerebral artery (Fig. 2). Right carotid and vertebral angiogram showed normal findings. The patient was discharged with a considerable improvement of the headache, dysarthric speech, and right facial weakness 12 days later.

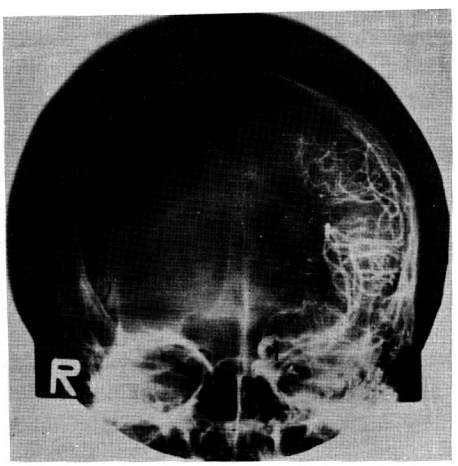

Fig. 2. Anteroposterior projection of left carotid angiogram. The arrow indicates proximal segmental narrowing of left middle cerebral artery.

DISCUSSION

There were clues which enable us to diagnose hemiparesis associated with HZO without difficulty in this case. First, there was typical time lapse, that is, hemiparesis was preceded by contralaterl HZO. Second, no major attributes of cerebral infarction such as hypertension, seruim lipid changes, diabetes, or carotid bruits were present. Third, the age of the patient is younger than that of usual victim of cerebral infarction.

The modes of involvement of the CNS by herpes zoster virus(HZV) can be fallen into 4 groups: mononeuritis, encephalitis, myelitis, and hemiplegia associated with HZO(Cope and Jones, 1954, Acers, 1964). The last is known to be the least common syndrome which was described first by Dumary in 1896. Although it is an unusual clinical event, this crossed zoster syndrome have been reported with increasing frequency.

The typical clinical history was gradual resolution of cutaneous HZO followed by acute onset of contralateral hemiparesis or hemiplegia, hemisensory symptoms or aphasia (Hilt et al., 1983). There was, in general, interval of 1 week to 6 months between the onset of HZO and that of contralateral hemiparesis, with an average of 7.3 weeks (Reshef et al., 1985). Thus, the time span, 44 days, in our case is not unusual. Then, how can this various time lapse be explained? Eidelberg et al. (1986) provided a feasible explanation, they suggested that host factors might play a role in determining the chronicity and cellular reactivity of the lesion.

Reshef et al. (1985) reviewed 51 cases which had been reported in the literatures. There were 29 men and 22 women. The mean age of the patients was 58.1 with a range of 7 to 96. Thirty patients had HZO on the left side and 21 on the right. Twenty-four patients (47%) exhibited diffuse CNS symptoms such as stupor, somnolence, general disorientation, confusion, and/or memory deficit at or following or prior to contralateral hemiparesis. But these were not present in our case. Aphasia was present in 15 patients. Seizure and cerebellar signs are among presenting symptoms reported rarely.

The neurological manifestations are usually acute and monophasic (Hilt et al., 1983) but Bourdette et al. (1983) suggested that stroke syndromes might be presented as TIAs, stroke-in-evolution, and multiple cerebral infarction in addition to isolated cerebral infarction. There was only one case that contralateral hemiplegia after HZO was resulted from intracerebral hematoma (Luigi et al., 1987).

Various possible pathogenic mechanisms for crossed zoster syndrome have been proposed. These have been reviewed by Acers (1964). Among these, direct invasion theory is believed to be the most probable mechanism (Rosenbulum and Hadfield, 1972, Linnemann and Alvira., 1980, MacKenzie et al., 1981, Doyle et al., 1983, Hilt et al., 1983). MacKenzie et al. (1981) emphasized segmental narrowing of the proximal middle and anterior cerebral arteries in the M1, A1, and A2 segments in their patients. The fact that these vessels are known to have dense trigeminovascular connections is supporting the hypothesis of direct spread. Since Kolodny et al. (1968) who provided the first description of HZO complicating contralateral hemiplegia with granulomatous angitis of the CNS, many authors have reported the pathologic angitis in crossed zoster syndrome. Rosenbulum and Hadfield (1972), Linnemann and Alvira (1980) who provided the first evidence of viral particle in the vessel wall, and Doyle et al. (1983) also suggested that granulomatous angitis resulted from direct invsion of blood vessels by HZV.

Direct spread is not, however, the sole mechanism of HZV spread (Hilt et al., 1983). Eidelberg et al. (1986) suggested that other possibilities such as trigeminal infection without rash, hematogenous seeding to nerves, or spread via sympathetic nervous system pathways might be exist.

The findings of CSF, which was obtained from 40 patients, was abnormal in 28 patients during the course of their disease (Reshef et al., 1985). Typically, the CSF had elevated white cells (range 0 to 1,200), elevated protein (range 48 to 445 mg%), and normal levels of glucose. Mononuclear cells were predominant.

Angiographic findings have been reported by many authors (Sato et al., 1971, Rosenbulum and Hadfield, 1972, Gursoy et al., 1980, Mackenzie et al., 1981, Bourdette et al., 1983, Doyle et al., 1983, Hilt et al., 1983, Eidelberg et al., 1986, Luigi et al., 1987). Sato et al. (1971) were the first who described angiographic evidence of segmental constriction in the proximal middle cerebral artery ipsilateral to HZO, as in our case.

CT findings in crossed zoster syndrome have been reported by many authors, too (Bourdette et al., 1983, Doyle et al., 1983, Hilt et al., 1983, Reshef et al., 1985, Eidelberg et al., 1986, Luigi et al., 1987). In general, lesions appeared in the deep gray matter or internal capsule and were consistent with infarction in the distribution of the approximate middle cerebral artery (Hilt et al., 1983). He also reported progressive white matter lucencies representing demyelination.

In addition to clinical findings, brain CT, and cerebral angiography, other diagnostic methods were suggested. Bieger et al. (1977) showed elevated CSF HZV antibody titers and Gershon et al. (1977) demonstrated the usefulness of indirect immunofluorescence assay for HZV antibody in CSF of patients with associated neurological syndromes. Temporal artery biopsy may be useful in confirming arteritis after HZO, and differentiating it from other similar arteritides (Gursoy et al., 1980, Landi et al., 1981).

Some patients have been treated with corticosteroid, acyclovir, adenine arabinoside, and anticoagulants with variable degree of benefits (MacKenzie et al., 1981, Doyle et al., 1983, Hilt et al., 1983, Reshef et al., 1985). But optimal therapy still remains to be defined. Prognosis was guarded and mortality rate was about 20% (Reshef et al., 1985), or 25% (Hilt et al., 1983).

In conclusion, it seems that granulomatous angitis, caused by direct spread of HZV into the blood vessel, may result in delayed contralateral hemiparesis or hemiplegia and a number of other stroke syndromes in patients with HZO. And it can be confirmed by cerebral angiography, brain CT, and biopsy.

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