CASE STUDY
HERPES ZOSTER MYELITIS TREATED WITH ACYCLOVIR

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Varicella zoster virus (VZV) is a human herpes virus which causes both varicella (chickenpox) and herpes zoster (HZ). HZ is a sporadic illness and is characterized by a painful vesicular cutaneous eruption along the distribution of cutaneous sensory dermatomes. The neurological complications include segmental motor or sensory loss, cranial nerve involvement, encephalitis, myelitis, Guillain-Barré syndrome, cerebral vasculitis, multifocal leucoencephalitic syndrome and post herpetic neuralgia (Kennedy, 1987). Clinical myelopathy from HZ is distinctly unusual and usually develops in the immunocompromised host. In large series, the incidence varies from 0 to 0.8% in the general population or in immunosuppressed patients, respectively (Devinsky, 1991). In the past, treatment was only symptomatic. Recently antiviral chemotherapy has been effective for patients with VZV, particularly acyclovir (Prober *et al,* 1982; Shepp *et al,* 1986). However the therapeutic efficacy of this drug in HZ myelitis could not be assessed because of limited data. We report a case of HZ myelitis and the results of therapy with acyclovir.

A 54-year-old woman was admitted to Srinagarind Hospital, Khon Kaen, in December 1991 because of left leg weakness. She was healthy until about 2 weeks before admission when she experienced difficulty in walking due to progressive weakness of the left leg. Abnormalities of bowel/bladder function and pain sensation were not observed. About 3 months prior to admission she had a HZ eruption on the left flank region which subsided with oral acyclovir therapy. Significantly, her past history included well-controlled diabetes mellitus and hypertension for 10 years.

On examination, the patient was alert with normal body temperature. A post-herpetic scar lesion was noted on the left flank in the T11-12 distribution. She had muscle weakness of the left leg (muscle power grade III-IV) with hyperreflexia of both knee and ankle jerks. Babinski's sign was present bilaterally. Pin prick sensation, joint positional sensation and vibration sensation were intact. The rest of general and neurological examinations were within normal limits.

Laboratory results of complete blood count, blood glucose, urea, creatinine and electrolytes were normal. A myelogram was performed which disclosed no abnormalities. The CSF was clear and colorless with no white blood cells or red blood cells, normal protein concentration and glucose level.

Intravenous acyclovir 500 mg every 8 hours was given for 7 days. Muscle power of the left leg was improved dramatically within 2 days after initiation of treatment. The patient was discharged with a nearly normal muscle power. On follow-up 3 months later she was in a good condition.

A diagnosis of a VZV-induced neurological syndrome is usually made on clinical grounds when characteristic CNS symptoms develop in the context of varicella or herpes zoster. The association is generally clear when the characteristic rash is present and precedes the symptoms, and when the time interval between the exanthem and the onset of neurological disease is not unusually long.

An extensive review of HZ myelitis by Devinsky *et al* (1991) revealed that this complication occurred in both sexes with a mean age about 50 years (range 16-86 years). About 40-50% had underlying diseases, particularly hematologic diseases. Onset of spinal cord dysfunction usually developed within 2 weeks (range, 8 days before to 10 weeks) after the initial rash. Most patients presented with subacute progression and maximal deficit usually occurred within 2-3 weeks. The most common presenting symptom was leg weakness, ipsilateral to the rash and was usually unilateral. When the disease progressed, neurological deficits presented with symptoms and signs of spinal cord dysfunction such as paraparesis, sensory loss and bladder

dysfunction. Laboratory tests were more useful in excluding the other causes of myelopathy than in specifically identifying VZV. The myelogram was normal in all cases in which it was performed. The CSF profile usually included a mononuclear pleocytosis with normal or elevated protein and normal sugar level. Inflammatory cells may be absent or minimal or may include polymorphonuclear cells.

Pathological changes of the spinal cord segment, corresponding to the involved dermatome, varied according to the severity of any associated myelitis. The finding included perivascular infiltration with lymphocytes and plasma cells, microglial proliferation, hemorrhagic necrosis, focal destruction of axons and demyelination, type A intranuclear inclusion bodies, positive VZV antigens by immunohistochemical detection and positive cultures of VZV from spinal cord tissue (Rose *et al,* 1964; McCormick *et al,* 1969; Hogan and Krigman, 1973; Devinsky *et al,* 1991). These findings suggested that the pathogenesis should be the result of direct viral invasion from the infected sensory ganglia or the consequence of an immune-mediated postinfectious mechanism.

The characteristic delayed development and slow evolution of HZ myelitis suggested that early therapy with an effective antiviral drug, especially acyclovir, was beneficial. However only few cases of HZ myelitis, treated with acyclovir therapy, were presented. Acyclovir monotherapy was used in 1 non-AIDS patient who improved, and in 2 AIDS patients. One of the latter improved during treatment but deteriorated several weeks later and the other showed no response because of delayed medication. The role of corticosteroids in this disease was less clear.

Our case demenstrates the typical presentation of HZ myelitis and rapid improvement with acyclovir therapy. Good prognosis depends on early recognition and treatment with this anti-viral drug.

REFERENCES

Devinsky O, Cho E-S, Petito CK, *et al.* Herpes zoster myelitis. *Brain* 1991; 114 : 1181-96.

Hogan EL, Krigman MR. Herpes zoster myelitis. Evidence for viral invasion of spinal cord. *Arch Neurol* 1973; 29 : 309-13.

Kennedy PGE. Neurological complications of varicella-zoster virus. In : Kennedy PG, Johnson RT, eds. Infections of the nervous system. London : Butterworths, 1987 : 177-208.

McCormick WF, Rodnitzky RL, Schuchet SS, *et al.* Varicellar-zoster encephalomyelitis : a morphological and virologic study. *Arch Neurol* 1969; 21 : 559-70.

Prober CG, Kirk LE, Keeney RE. Acyclovir therapy of chickenpox in immunosuppressed children-A collaborative study. *J Pediatr* 1982; 101 : 622-5.

Rose FC, Brett EM, Burston J. Zoster encephalomyelitis. *Arch Neurol* 1964; 11 : 155-72.

Shepp DH, Dandliker PS, Meyers JD. Treatment of varicella-zoster virus infection in severely immunocompromised patients : a randomized comparison of acyclovir and vidarabine. *N Engl J Med* 1986; 314 : 208-12.