

Letters

Herpes zoster myelitis treated with vidarabine

Sir: Herpes zoster is a common disease with an annual incidence of 3.4 per one thousand persons.¹ Classically, it produces a vesicular eruption and sensory abnormalities in the distribution of one or several adjacent dermatomes. However, the virus may be responsible for five other conditions: encephalitis, myelitis, polyneuritis, segmental muscle atrophy and segmental radiculitis. The first three conditions are rare. Thomas and Howard reported that of 1,210 patients with herpes zoster seen over a ten-year period at the Mayo Clinic, 61 (5%) were found to have zoster-induced segmental muscle weakness. Only one had myelitis.² We present the clinical history and findings of a 59-year-old man with myelitis following intercostal herpes zoster and report the results of treatment.

A right handed caucasian business executive, age 58 years, was admitted to the Neurology Unit, Wayne State University, with the chief complaint of weakness of the right lower extremity. He had developed herpes zoster approximately five weeks previously with vesicles in the distribution of T4 on the right side. His temperature rose to 39.4°C orally at the onset and he had severe pain on the right side of the chest radiating to the spine followed by nausea and vomiting. The family physician treated him with tetracycline. Approximately two weeks prior to admission he developed another small crop of vesicles over the distribution of T8 on the right. One week prior to admission he developed extreme difficulty climbing stairs because of weakness of the right lower extremity and loss of sensation, including temperature perception, which was noted on the left lower extremity. At the same time he developed acute right chest pain radiating to the spine followed by a recurrence of vomiting. He was allergic to penicillin by history, smoked cigarettes until 15 years prior to admission, and had a moderate alcohol intake. Review of symptoms was otherwise unremarkable. At the time of admission he was taking antacids for reflux esophagitis, tetracycline for herpes zoster and erythromycin stearate for an infection in his toe. General physical

examination revealed a well developed, well nourished male in no acute distress with a blood pressure of 120/80 mm Hg, pulse 84 per minute and regular, respirations 12 per minute, temperature 37°C orally, weight 90 kg. There were no other abnormalities except for the healing vesicles of herpes zoster. Neurological examination revealed an alert, cooperative male who was oriented in all three spheres. Judgment, recent memory, recall and remote memory were intact. The neck was supple and Kernig's sign was negative. Cranial nerves were within normal limits. Power was normal in the upper extremities. The tone was normal but there was slight weakness in the left lower extremity and severe weakness in the right lower extremity. Coordination was intact. Sensory examination showed that light touch, pain and temperature sensations were lost between T4 and T8 on the right and below T4 on the left. Proprioception was absent below T4 on the right side. The stretch reflexes were normal and symmetrical in the upper limbs, knee jerks were also normal but the ankle jerks were absent. There was a bilateral extensor plantar response. A complete blood count with differential was within normal limits and the sedimentation rate was 8 mm/hour (Westergren). The cerebrospinal fluid was clear and colorless with 11 white cells, all monocytes, protein 0.69 g/l, glucose 4.8 mmol/l, IgG 0.073 g/l, with no growth on culture. There was no significant abnormality on cervical or thoracic myelography.

The patient was treated with corticosteroids but continued to deteriorate and by the end of the first week in the hospital he had become incontinent of urine and faeces. The corticosteroids were discontinued and treatment with vidarabine 15 mg/kg iv per day was started. Two days later he suddenly became erythematous, sweaty and drowsy and felt unwell. Treatment was stopped and he soon recovered. Vidarabine was started again at 10 mg/kg iv per day for a further 8 days. A second lumbar puncture showed a clear CSF with 5 RBC, 12 WBC, all monocytes, glucose 3.3 mmol/l, protein 0.35 g/l, IgG 0.031 g/l. The patient began to improve about 8 days after the commencement of treatment with vidarabine. After 12 days' treatment he could walk 50 feet with a cane. The sensation showed marked improvement in both legs. He was discharged one month after admission and was walking well without assistance. He has continued to improve since that time

and the only abnormality is a slight spasticity and increase in stretch reflexes in the lower limbs.

Since herpes zoster myelitis was first reported in 1876,³ few cases have appeared in the literature. There are no reports of treatment with antiviral agents. Recent findings have suggested that it is mediated by direct viral invasion of the spinal cord and cell lysis.⁴ Our patient was given vidarabine since this drug is known to be effective in herpes zoster infections.⁵ We suggest that vidarabine may be useful in the management of herpes zoster myelitis.

PA CULLIS

J GILROY

Department of Neurology

R CUSHING

Department of Internal Medicine,

Wayne State University School of Medicine,
Detroit, Michigan 48201, USA

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