Radiculomyelopathy associated with herpes simplex genitalis treated with adenosine arabinoside

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Summary

A case of herpes simplex genitalis producing radiculomyelopathy with urinary retention is reported. The patient was treated with adenosine arabinoside intravenously and made a complete recovery over 4 weeks. Although herpes simplex genitalis is common, neurological sequelae, particularly cord involvement, are distinctly rare. It should, nevertheless, be considered in the differential diagnosis of urinary retention, with or without long-tract signs, in young patients.

KEY WORDS: herpes simplex genitalis, radiculomyelopathy.

Introduction

Herpes simplex infection of the nervous system may manifest clinically as meningitis, encephalitis, myelitis or as a radiculopathy. A syndrome comprising radiculopathy with urinary retention has been associated with herpes simplex genitalis, but spinal cord involvement in such cases appears rare. We have found only one other similar case reported in the literature.

We report a case of herpes simplex genitalis producing radiculomyelopathy with urinary retention, in which complete recovery occurred, after treatment with intravenous adenosine arabinoside, an antiviral agent. Aspects of this case have been previously discussed (Handler and Perkin, 1982).

Case report

A 20-year-old white female, with no previous history of herpetic infection, developed vulval pain two days after sexual intercourse with a new partner.

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A few days later, she had dyspareunia with dysuria and then developed urinary retention. She was catheterized temporarily. The following day, the appearance of lower limb paraesthesiae and weakness led to her admission to hospital.

On examination, she was afebrile and had a palpable bladder. There was a moderate flaccid paraparesis with diminished knee and ankle tendon reflexes and equivocal plantar responses. There was sensory level to light touch and pin prick at the umbilicus. The vulva showed vesicles and ulcerating lesions.

A urinary catheter was inserted. Cultures from the vulval lesions, the cervix and urine revealed herpes simplex type 2. A full blood count and biochemical screen were normal. The erythrocyte sedimentation rate was 38 mm/hr. Acute and convalescent sera showed a diagnostic rise (1:8-1:320) of complement fixation antibodies to herpes simplex type 2. Serum VDRL and TPHA were negative. The serum IgM level was slightly depressed, 0.4 g/litre (normal range 0.5-3.1 g/litre). A serum autoantibody screen was negative.

The cerebrospinal fluid (CSF) contained 15 lymphocytes/mm³, two red cells/mm³ and a protein concentration of 0.64 g/litre. IgG and albumin levels were 44 mg/litre and 325 mg/litre respectively with a normal ratio of IgG to albumin in CSF compared with serum. Repeat CSF examination 10 days later showed a rise in protein concentration to 1.60 g/litre with 5 lymphocytes/mm³. Complement fixation titres for herpes simplex antibodies showed no diagnostic rise.

A diagnosis of herpes simplex radiculomyelopathy was made. The patient was treated with topical lignocaine jelly with idoxuridine, together with adenosine arabinoside 600 mg/24 hr intravenously for 5

days. Leg power returned to normal and the patient was walking normally within 2 weeks. Full recovery of sensory function occurred over the next month. The urinary catheter was withdrawn 5 days after admission.

Discussion

Two types of herpes simplex virus have been identified (Nahmias and Roizman, 1973a). Differing patterns of neurological involvement are described according to virus type. In the adult, type 1 virus is most definitely associated with encephalitis, myelitis or radiculitis and type 2 with meningitis, myelitis or radiculitis (Nahmias and Roizman, 1973b). Genital herpes is usually but not invariably, due to type 2 virus transmitted during sexual intercourse.

A syndrome of lumbosacral radiculomyelopathy with urinary retention has been described secondary to herpes genitalis (Caplan, Kleeman and Berg, 1977). Diagnosis in these cases was established either on the basis of typical genital lesions, positive viral cultures or complement fixation tests. All patients developed urinary retention. Six of the eleven had paraesthesiae in the perineum and lower limbs. Examination revealed altered sensation in sacral dermatomes in some patients with decreased sphincter tone. Lower limb weakness does not seem to have occurred and the state of the tendon reflexes was not given. It is not clear why the diagnosis was lumbosacral radiculomyelopathy as the signs they recorded indicated a sacral radiculopathy without evidence of spinal cord involvement.

A spinal cord disturbance secondary to herpes simplex infection, either of type 1 or 2, appears distinctly uncommon. A case of myelitis, confirmed at autopsy, secondary to type 1 infection has been reported (Klatersky *et al.*, 1972). The diagnosis was based on virus isolation from the cerebrospinal fluid, which paradoxically, showed a predominance of neutrophil leucocytes and a depressed glucose concentration. The patient eventually developed a tetraparesis with facial weakness though the authors failed to refer to brain stem changes at autopsy. A less convincing case, due to type 2 infection, has been recorded by Craig and Nahmias (1973). The patient had a previous genital infection. Spinal cord involvement was deduced from a sensory disturbance ascending to the umbilicus and a questionable plantar response on one side. Sensory symptoms and

sphincter impairment predominated in this case. Motor function appears to have been normal though there was lower limb areflexia. The association with herpes infection was based on virus isolation from vulval lesions.

The diagnosis of herpes simplex genitalis in our patient was based on a typical clinical appearance combined with virus isolation from the genital lesions and the urine. In some respects, the case was similar to those described by Caplan *et al.* (1977) with urinary retention and lower limb sensory symptoms. The natural history of these cases of radiculopathy was one of spontaneous resolution. It is difficult, therefore, to attribute the improvement in our patient to adenosine arabinoside alone. In addition, there was prominent motor dysfunction, a finding previously reported only in the case due to type 1 virus discussed by Klatersky *et al.* (1972). It must be admitted that the evidence for spinal cord involvement in this case was indirect. Sphincteric involvement alone is clearly insufficient. A prominent flaccid paraparesis occurred in none of the cases described by Caplan *et al.* (1977) and this, together with the truncal sensory level, a finding rare in peripheral neuropathy (Poser, 1981), leads us to consider the likelihood of a myeloradiculopathy in this case, secondary to herpes simplex genitalis infection.

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