The MR of the brainstem showed a small infarction of the left dentate and medial medulla without any further pathology.

Until confirmation of the total occlusion of the left vertebral artery the patient was treated with cumarine to prevent arterial emboli. The clinical symptoms progressively disappeared over the next 30 days and the patient left hospital with only a slight ataxic gait and mild dysaesthesia within the left C6 segment.

Discussion

The patient presented with an anterior dislocation and fracture of the left facet joint between the C5 and C6 vertebral bodies and radicular pain in the left C6 distribution, and subsequently developed a left vertebral artery dissection with left brainstem ischaemia. It is suspected that the dissection occurred during the 19 day delay with subsequent occlusion including the posterior infracerebellar artery. By contrast with our case, most reports during the past decade concerning posterior circulation ischaemia connected with vertebral dissection, occurred spontaneously or were loosely related to minor trauma during sport or neck manipulation during chiropraxy. These typically occur in the young middle aged and equally between the sexes.1 Although traumatic fractures of the cervical spine occur often, injuries of the vertebral arteries with or without clinical symptoms are rarely evident. The incidence is postulated to be between 3% and 10%.2

In a retrospective study Parent and coworkers reviewed some 640 patients sustained fractures of the cervical spine, 96 of these had facet involvement and in only five was injury of the vertebral artery diagnosed by initial major neurological deficits such as cerebellar infarction, cortical blindness, or pontine infarction, which have been documented by postmortem examination in two cases.3 All these patients had cervical fractures located at C5-C6 and in one case in combination with a fracture at C6-C7. Radiographs showed anterior dislocation at C5-C6 in four cases and at C6-C7 in one case. Bilateral facet fractures were evident in four cases.

One prospective study exists that considers the combination of facet joint dislocation of the cervical spine, the incidence of vertebral artery injury, and the neurological deficit.2 From 12 consecutively examined patients with facet joint dislocation (C5/C6 in seven, C6/C7 in three, and C4/C5 in two) nine showed an occlusion of at least one vertebral artery. Of these nine patients only two with bilateral facet joint dislocations had a transient neurological deficit. Further indications of a traumatic or spontaneous dissection of the vertebral artery are neck pain and symptoms of a C6 radicular palsy.

Thus a combination of neurological and radiological findings could lead to an early diagnosis and may indicate development of a dissection of the vertebral artery. We suggest that patients with the clinical symptoms and type of injury described here are prone to development of a dissection or occlusion of the vertebral artery. Early diagnostic procedures by non-invasive diagnostic techniques such as MR angiography and ultrasound techniques coupled with treatment at the onset of a possible dissection may help to prevent the formation of a microembolism or arterial occlusion.

Correspondence to: V Dietz, Paraplegic Centre, Bulgarist, Poststrasse 340, CH-8008 Zürich, Switzerland.


Hypersimple virus type 2 ascending myeloradiculitis: MRI findings and rapid diagnosis by the polymerase chain method

Although neurotropic viruses are often suspected of causing spinal cord injuries, confirmation by early diagnosis is difficult. Ascending myelitis related to herpes simplex virus type 2 (HSV-2) infection has seldom been reported and the diagnosis could be established only at postmortem examination.1-3 We report the case of an elderly woman with a subacute ascending myeloradiculitis. MRI showed spinal cord and sacral root involvement and the polymerase chain reaction allowed the rapid identification of HSV-2 DNA in the CSF.

A 76 year old woman was referred because of urinary retention and paraparesis. Three weeks previously, she had noted the progressive onset of anorexia, fever (38°C), weight loss (4 kg), and low back pain. Evaluation performed in another hospital showed negative bacterial cultures from blood and urine and CT of the thorax, abdomen, and lumbarosacral spine was normal. Three days before admission she complained of right sciatalgia and rapidly developed lower extremity weakness and spinchter disturbances.

Neurological examination showed a flaccid paraplegia, a T10 sensory level, and a distended bladder. Deep tendon reflexes were absent in the lower limbs and plantar responses were both extensor. In the upper extremities strength was normal but reflexes were brisk and a bilateral Hoffman sign was noted. Mental state and cranial nerves were normal.

Non-enhanced T1 weighted images of the spine were normal. T2 weighted sequences showed a hyperintense signal at the T10 level and within the conus medullaris. T1 weighted images with contrast injection showed an enhancement of both the posterior meninges and the roots of the cauda equina.

Her CSF contained 73 lymphocytes/mm3 (97% lymphocytes), 132 mg/dl protein and 68 mg/dl glucose. Electrophoresis of CSF protein showed 26% γ-globulins with the oligoclonal distribution and a raised IgA/albumin ratio (0.56; normal<0.25). A polymerase chain reaction was performed on CSF with a pair of primers that allowed the simultaneous detection of four viruses of the herpes group. A strong signal was obtained on ethidium bromide staining. Characterisation of HSV-2 DNA was achieved by restriction analysis of the amplified product. α-Interferon in CSF was normal. The patient had no history of recurrent herpes genitalis. There was no serological evidence for borrelia, HIV-1 or HIV-2, HTLV-I, Q-fever, lysteriosis, cytomegalovirus, measles, varicella zoster, or Epstein-Barr virus infection. CD4 counts were normal and no case for immunodepression could be identified.

Parenteral acyclovir (30 mg/kg daily) was given for 10 days and the patient’s neurological status remained stable. No recurrence was seen after admission, sparse vesicular lesions appeared on the patient’s buttocks, internal aspects of the thighs, and lower part of the abdomen. Ten days later, numbness in both hands appeared. Examination showed bilateral arm and shoulder weakness and the disappearance of upper limb reflexes. The patient then became confused and drowsy, developed hyponatraemia and hypona-
after admission, a week before the appearance of the cutaneous eruption.

The pathogenesis of HSV-2 myelitis in this case remains ill defined. Although IgM antibodies were detected in the first serum sample we are not sure that the patient's myelodacilitis reflected real HSV-2 primary infection because reappearance of IgM antibodies can be detected in herpes virus reactivation. All but one previously reported case of HSV-2 myelitis were fatal and occurred in immunocompromised hosts. This patient was neither diabetic nor HIV-infected, but despite normal CT examinations of the thorax and abdomen, a hidden malignancy cannot be excluded. Reactivation of a latent infection within dorsal root ganglia neurons with a contiguous spread via sacral ganglia to the spinal cord has already been proposed in HSV-2 ascending myelitis. It is of note that despite antiviral treatment, our patient died within six weeks. Thus an immunologically mediated injury triggered by the herpes infection could also be involved here, as in another patient with HSV-1 myelitis whose necropsy examination showed patchy demyelination of the spinal cord.1

EMMANUEL ELLIE
Service de Neurologie, Hospital du Haut-Levêque, CHU Bordeaux, France
FLORE ROZENBERG
Service de Bactériologie-Virologie, Hôpital Saint-Vincent de Paul, Paris, France
VINCENT DOUSSET
Service de Neurologie, CHU Bordeaux, France
MARIE BEYLOT-BARRY
Service de Dermatologie, CHU Bordeaux, France

Correspondence to: Dr Emmanuel Ellie, INSERM U394, 33077 Camoline-Saint-Sains, 33077 Bordeaux Cedex, France.


Peduncular hallucinosis and right hemiparkinsonism caused by left mesencephalic infarction

Since the original description of Lhermitte1 several causes of peduncular hallucinosis have been reported, but all in relation to a bilateral mesencephalic lesion. We describe here a patient with prolonged visual hallucinations and right hemiparkinsonism.

Axial T2-weighted MR image obtained with a 0–T unit shows an area of hypointensity in the left cerebral peduncle, consistent with infarction.