Letters

presence of small ischaemic lesions it would appear to be the ideal method for assessing neurological damage in these patients.

DG MENZIES
IW CAMPBELL
Department of Medicine,
Victoria Hospital,
Kirkcaldy, Fife KY2 5AH
DM KEAN
NMR Imaging Unit,
University of Edinburgh,
Edinburgh

Correspondence to Dr Menzies, Department of Medicine, Royal Infirmary of Edinburgh, Edinburgh EH3 9YW, UK

References


Accepted 23 May 1988

Paraplegia as a presenting feature of meningococcal meningitis

Sir: Recent attention has been focused on the need for the early diagnosis and treatment of meningococcal meningitis. Involvement of the spinal cord in this condition is rare and tends to occur either as a late sequela or in the context of a fulminating illness.1 We report a case where paraplegia was the first major symptom.

A previously fit 17 year old male “A” level student developed non-specific headache, nausea and myalgia which he attributed to influenza. The next day he noticed some weakness of his legs which progressed over the next 24 hours, by which time he had to be carried to the toilet. This was not reported to the deputising general practitioner who visited the next day and prescribed oral amoxycillin. Thirty six hours later he was admitted to hospital with acute urinary retention and referred to our department. He was able to give his own history.

On examination he was afebrile and there was no rash. There was a minor degree of neck stiffness and Kernig’s sign was positive bilaterally. He was fully orientated but whilst he performed standard tests of higher cerebral function accurately, the speed did not seem appropriate to his educational status. Fundoscopy and pupillary reactions were normal. He complained of diplopia on right lateral gaze. The other cranial nerves were normal as was examination of the arms. There was a symmetrical paraparesis (MRC Grade 2) affecting all lower limb muscles. Muscle tone and tendon reflexes were unremarkable and the plantar responses were flexor but the lower abdominal reflexes were absent. There was a loss of pinprick and temperature sensation below T12 with sparing of vibration and proprioception.

He had a neutrophil leucocytosis (12.7 x 10⁹/l) and a raised blood urea (16.1 mmol/l). On pre and post contrast cerebral CT scans the basal cisterns were indistinct and there was slight dilatation of the temporal horns. A full length myelogram was normal but turbid fluid was obtained which contained 24,000 x 10⁶/l white blood cells (99% polymorphs), a protein of 3.2 g/l and glucose of less than 1 mmol/l. A gram stain revealed occasional gram negative diplococci and a co-agglutination test for meningococcal antigen (Phadecact CSF Test—Pharmacia Diagnostics) was positive.

Intravenous benzyl penicillin (24 megaunits/day) and chloramphenicol (3-6g/day) were given for 10 days. Four hours after the first dose a fever was noted which persisted for 5 days. The first signs of improvement were noted on the fifth day after admission and by the twelfth day he could raise his legs off the bed. There was also some return of bowel and bladder sensation. By the seventeenth day he was able to bear weight and the urinary catheter was removed. The sensory level was still present and the plantar responses were now extensor. He was walking unaided by the third week and could manage stairs by the sixth week, by which time sphincter control was socially acceptable with the help of emperonium bromide. At 8 weeks there was no detectable sensory level, there was only minimal weakness of hip flexion and slight increase in tone but the plantar responses remained extensor.

Our patient developed a thoracic cord syndrome as the first major neurological feature of a meningococcal meningitis. Even on admission several days later he had only mild meningism and a subtle impairment of higher cerebral function though the antibiotic started 24 hours after the development of the spinal cord problem might have influenced the later course of the disease. In his review of spinal cord lesions in association with meningococcal meningitis, Turner found only one case of acute transverse myelitis and one case of an acute conus medullaris syndrome, which he distinguished from the later spinal cord sequelae caused by diffuse arachnoiditis.

Two cases of early cord damage associated with meningococcal infection have been reported since the introduction of penicillin. Graus et al2 described a patient who developed a partial Brown-Squard syndrome with a spinocerebellar syndrome level at T10 within hours of the onset of headache, vomiting and fever though the patient was still orientated at the time of first examination. As in our patient, proprioceptive sensations were preserved and the patient made a full recovery. Gotshall et al3 reported a patient who had signs suggestive of a conus medullaris syndrome within 24 hours of the onset of fever and vomiting but the patient had been unconscious when first examined.

The pathology of the acute cord damage is uncertain though an intramedullary vascular lesion seems most likely. Our patient, like the other cases above, had a myelogram which did not show any paraspinal collection of arachnoid adhesions. Banks and McCartney4 described focal encephalitis and myelitis in some of their patients with meningococcal meningitis who died having survived the first few days. This took the form of small haemorrhagic foci with thrombosed capillaries and perivasculary cuffs with polymorphs. Lesions were described both within the brain and upper spinal cord. The relative sparing of proprioception in our patient and that of Graus et al is compatible with occlusion of a perforating branch of the anterior spinal artery but there has not been pathological confirmation of this. Our case emphasises that serious neurological damage may occur before the more florid “classical” features of meningococcal meningitis.

BR BOOTHMAN
JM BAMFORD
MR PARSON

The General Infirmary at Leeds
Great George Street
Leeds LS1 3EX, UK

References


Accepted 27 May 1988