Acute paraparesis with tuberculous meningitis

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A 30-year-old-man who had low-grade intermittent fever and occasional vomiting of 2 months duration, presented with acute onset of complete weakness of both lower limbs and urinary retention. There was no family history or past history of tuberculosis. Examination revealed right 6th nerve palsy, marked neck rigidity and positive Kernig's sign. The patient had paraparesis with power 0/5 on the MRC scale. His bladder was distended and was palpable in the suprapubic region. Deep tendon reflexes were brisk in the lower limbs. Both plantars were extensor, and abdominal and cremastic reflexes were not elicitable. He had loss of all forms of sensations below the umbilicus; power in the upper limbs was normal.

All haematological, serum biochemical and urine parameters were normal. Cerebrospinal fluid (CSF) examination revealed protein 0.14 g/l, cells $336 \times 10^6/l$ (all mononucleocytes), sugar 1.3 mmol/l. Enzyme-linked immunosorbent assay (ELISA) for antituberculous antibodies was strongly positive. Cranial computed tomography (CT) revealed hydrocephalus, exudates in basal cisterns and marked gyral enhancement. Radiographs of thoracic spines, chest and a lumbar myelogram were normal. Contrast-enhanced magnetic resonance imaging (MRI) of spine revealed an intensity-enhancing disc lesion at the thoracic 8–9 level of the spinal cord (figure).

Questions

- 1 What is the probable diagnosis ?
- 2 What are possible causes of paraparesis in a patient with tuberculous meningitis ?

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Figure Contrast-enhanced MRI of spine

Causes of paraplegia in tuberculous meningitis

- compressive myelopathy (granuloma): extradural, intradural-extramedullary, intramedullary
- transverse myelitis
- myeloradiculopathy (with or without spinal meningitis)
- syrinx formation

Answers

QUESTION 1

This patient, having features consistent with the diagnosis of tuberculous meningitis, had presented with acute transverse cord syndrome. The myelogram showed free flow of contrast media. There was no clinical or radiological evidence of radiculopathy. However, on MRI, an enhancing lesion at thoracic levels 9 and 10 suggestive of granuloma was seen.

QUESTION 2

Possible aetiologies which can produce paraparesis in a patient with tuberculous meningitis are listed in the box.

Discussion

Spinal tuberculomas are rare and usually present as transverse myelitis. They are often associated with bony abnormalities such as collapse of vertebrae and loss of pedicles, especially in extradural and subdural forms. Intramedullary tuberculomas are more rare and usually associated with normal CSF parameters.^{1,2}

Paraparesis is an uncommon manifestation in patients with tuberculous meningitis. Frequently it is due to tuberculous spinal meningitis involving pia and arachnoid matter, in a pattern of myeloradiculopathy. The acute form presents with fever, backache and radiating root pains (radiculopathy) accompanied by rapid onset of lower limb weakness and a definite sensory level on the trunk (myelopathy). In the subacute form, root pain and bladder involvement are less frequent. The chronic form, usually localised to a few segments, presents as progressive spinal cord tumour-like syndrome. Tuberculous spinal meningitis should be suspected when there are root signs in addition to cord signs, for example, spastic paraparesis with brisk ankle jerks and absent knee jerks in an appropriate clinical setting.³⁻⁵

Intramedullary tuberculoma are exceedingly rare lesions and must be differentiated from other nonosseous forms of spinal extramedullary spinal tuberculosis. Dastur⁶ reviewed 74

cases of tuberculous paraplegia without evidence of Pott's disease and discovered that extradural granulomas occurred in 64%, arachnoidal lesions without dural involvement in 20%, subdural lesions in 8%, and intramedullary lesions in 8% of the patients. Isolated intramedullary tuberculomas are rarely reported in western literature and a prevalence of two in 100 000 cases of tuberculosis has been cited.⁷ Reports of intramedullary spinal cord tuberculoma, even from India (a highly endemic area of tuberculosis), have been sporadic.¹ Granulomatous involvement of the spinal cord compared to that of brain has been cited to occur in the ratio of 1:42.⁸ Granuloma formation in brains of patients with tuberculous meningitis can occur during antituberculous therapy.⁷

Isolated intramedullary spinal tuberculomas tend to occur in young persons, and are usually associated with the pulmonary form of the disease. The lesions are more common in the thoracic segments of the spinal cord. The mycobacterial bacilli reach the central nervous system by haematogenous spread secondary to tuberculosis elsewhere in the body. Small tuberculous lesions (Rich's foci) develop in the central nervous system, either during the stage of bacteraemia of the primary tuberculous infection or later. These initial small tuberculous lesions in the meninges, brain or spinal cord can become active after a dormant period, even years after initial infection. Rich's foci tend to develop in subpial or subependymal regions of the brain and may rupture in CSF spaces, leading to development of tuberculous meningitis.⁹ Rarely, foci develop deep in the parenchyma of spine and brain; possibly, these lesions develop into clinically manifest tuberculoma.

Non-caseating granulomas are more common in the spinal cord while caseating lesions are seen in the brain. In non-caseating granulomas the lesion usually appears hypointense on T1-weighted MRI and iso- or hyperintense on T2-weighted images; after contrast administration the lesion shows intense homogenous disc enhancement.²

Although intramedullary tuberculoma often respond to antituberculous therapy, some patients show progressive neurological deficits despite treatment, possibly due to swelling in the lesion as the bacilli release toxic material in response to treatment.⁷ In cases in which the patient deteriorates, surgery is indicated.

Final diagnosis

Intramedullary spinal tuberculoma in a case of tuberculous meningitis.

Keywords: spinal tuberculoma; tuberculous meningitis

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Pyrexia of unknown origin

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A 32-year-old Indian man presented with a 4-week history of intermittent pyrexia with rigours, associated with malaise and weight loss. Physical examination was normal except for pyrexia without evidence of lymphadenopathy. The white cell count was $13.2 \times 10^9/l$ (3.5–11.0) with increased lymphocytes. Erythrocyte sedimentation rate and C-reactive protein were elevated. Liver function tests showed mildly raised liver enzymes. Blood and sputum cultures were negative for acid-fast bacilli. A computed tomography (CT) scan of the abdomen revealed a multiloculated mass in the head of the pancreas with peripancreatic and mesenteric lymphadenopathy (figure).

Figure CT scan of the abdomen showing an enlarged head of the pancreas with focal hypodense lesions

Questions

- 1 Suggest the most likely differential diagnosis in this case.
- 2 What further investigations should be performed?

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