

# Ring-enhancing lesions in the brain and spinal cord

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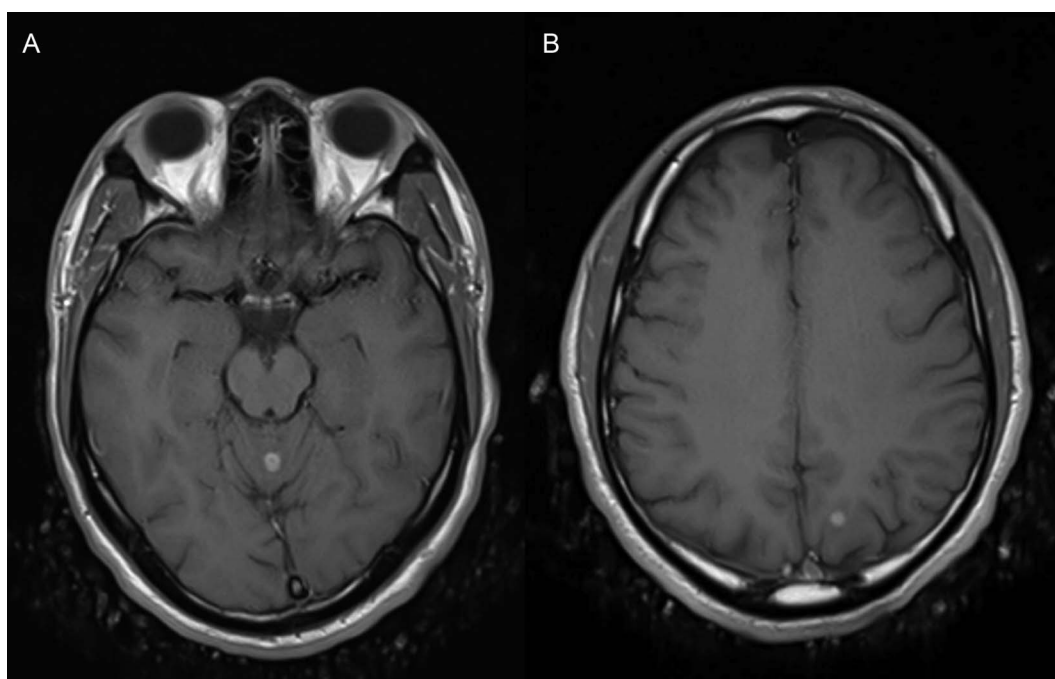
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## DESCRIPTION

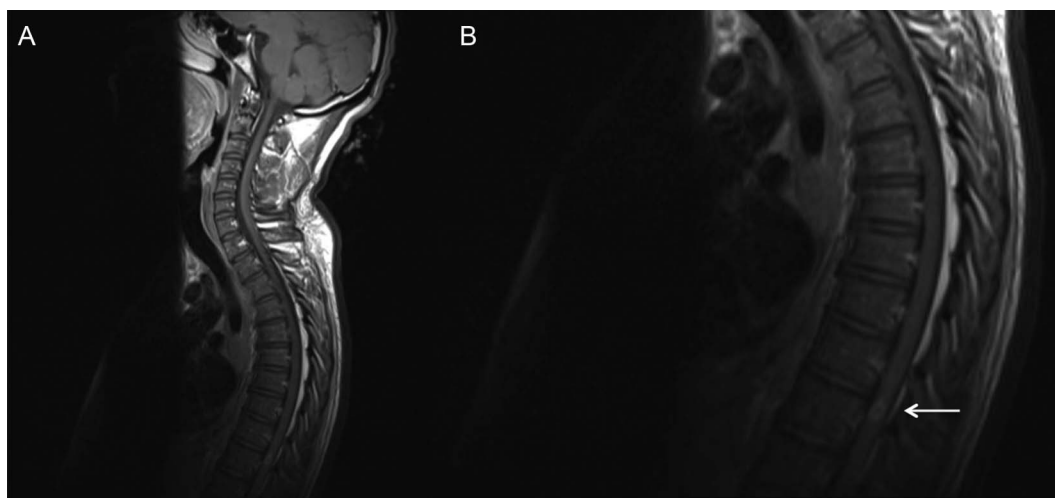
A 31-year-old Afro-Caribbean man, previously well, volunteered a 3-week history of drenching night sweats, unsteadiness, weak numb legs, constipation and difficulty passing urine. Examination elicited increased tone in the right leg, distal weakness of left leg, bilateral weak hip flexion, absent reflexes, muted plantar responses, sensory

level at T11. Cranial nerve, upper limb and back examinations were normal: no meningism.

Haemoglobin 11.2 (13.0–17.0 g/dL), mean corpuscular volume 89 (80–100 fL), white cell count 7.7 ( $4.0\text{--}11.0 \times 10^9$ ), C reactive protein 17 (0–4 mg/L), erythrocyte sedimentation rate 58 (0–15 mm/h), urea 5.1 (1.7–8.3 mmol/L), thyroid stimulating hormone 1.39 (0.27–4.20 mIU/L).



**Figure 1** Gadolinium-enhanced T1-weighted axial MRI brain imaging showing ring-enhancing lesions in the superior vermis (A) and the left posterior parietal lobe (B).



**Figure 2** Gadolinium-enhanced T1-weighted sagittal MRI spine imaging showing an intramedullary ring-enhancing lesion at T10 (A) and magnified image (B) with arrow highlighting abnormality.

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MRI revealed ring-enhancing foci in the superior vermis and the left posterior parietal lobe of the brain (figure 1), hyperintensity of the cord mid-thoracic level to conus and an intramedullary ring-enhancing lesion at T10 (figure 2), isointense on T2-weighted imaging.<sup>1–3</sup> X-ray chest was normal. CT chest, abdomen and pelvis demonstrated subcarinal and paratracheal lymphadenopathy and mid-oesophageal thickening. Positron emission tomography-CT indicated tracer uptake in mediastinal nodes and oesophagus.

Autoimmune and infectious serological screens, HIV and trans-thoracic echocardiogram were unremarkable. Lumbar puncture was normal. Endoscopic oesophageal biopsies: non-specific inflammation. Endobronchial ultrasound-guided fine needle aspiration of enlarged mediastinal lymph nodes revealed necrotising granulomatous lymphadenitis and acid-fast bacilli on Ziehl-Neelsen staining. *Mycobacterium tuberculosis* was cultured. The Health Protection Agency identified an infectious cluster suggesting this was likely primary infection. Family screening was negative. He is responding to quadruple therapy and steroids but progress is slow owing to medication side effects.

More common causes of transverse myelitis, viral infection, autoimmune (eg, lupus) and demyelination (eg, multiple sclerosis) are usually treated with steroids. Undiagnosed tuberculosis is likely to be calamitous.

### Learning points

- ▶ MRI is the non-invasive modality of choice to characterise cerebrospinal lesions.
- ▶ An intramedullary lesion with ring enhancement on T1-weighted imaging, isointense on T2-weighted imaging, is consistent with a tuberculoma, but is not diagnostic.
- ▶ Tuberculosis of the spinal cord is both very rare and difficult to diagnose. Cerebrospinal fluid may be normal. Other sites in the body should be sought to recover the organism. Failing this, spinal cord biopsy may be necessary.

**Contributors** JG developed the concept of the case report and prepared the draft. RH was involved in critical revision of the manuscript.

**Competing interests** None.

**Patient consent** Obtained.

**Provenance and peer review** Not commissioned; externally peer reviewed.

### REFERENCES

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