Paradoxical expansion of cerebral tuberculomas during therapy for Pott’s spine

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Cerebral tuberculoma is the commonest extrapulmonary lesion in tuberculosis, presenting with symptoms of an intracranial space occupying lesion. Computed tomography (CT) has been a major advance in its diagnosis and management. For unknown reasons, these tuberculomas sometimes become manifest, or expand, during successful treatment for tuberculosis elsewhere in the body. So far, only about 20 cases have been described, and we think that the following case is the first of its kind to be reported from India, though the country is flooded with tuberculosis.

CASE HISTORY
A woman aged 28 was diagnosed as having tuberculosis of the spine (Pott’s disease) with paravertebral abscess and was prescribed isoniazid 300 mg, rifampicin 450 mg, ethambutol 800 mg, and pyrazinamide 1.5 g together with pyridoxine 20 mg all daily. Also, a plaster jacket and bed rest were advised. Her symptoms improved and she was walking with support within 35 days. However, after 44 days’ treatment without a break, she complained of global headache with severe vomiting. Next day she was admitted to hospital where she had a focal seizure in her right foot with secondary generalization. Postictally she complained of weakness in the right half of the body, which began to improve after 2 h. There was no history of head trauma. She was in lactational amenorrhoea.

On neurological examination she had bilateral papilloedema, hypotonia with muscle power grade 3/5 in right upper and lower limbs, and bilateral upgoing plantar responses. On the second day there were right-sided grasp and palmo-mental reflexes, with positive glabellar tap. From the sixth day onwards, right-sided frontal lobe signs could not be elicited. Other laboratory examinations, including ELISA for HIV, were normal. Her haemoglobin was 9 g/dL. Contrast enhanced CT scan of skull revealed multiple hypodense areas in the left cerebral hemisphere with ring enhancement and surrounding oedema suggestive of tuberculomas (Figure 1).

Tuberculosis chemotherapy was continued unchanged, and the patient was given decompressive agents—mannitol 40 g IV in 30 min four times on day 1; glycerol 30 g orally four times daily for 15 days; and dexamethasone 8 mg IV three times daily for 10 days, with substitution of prednisolone 60 mg daily by mouth, tapering to zero over 2 months. In addition, she received phenytoin 100 mg three times daily, intravenously then orally, for the first 2 months. Pyrazinamide and ethambutol were stopped after 3 months. She improved clinically and papilloedema resolved completely over 2 months. Four months after discharge the patient had a further right focal seizure with secondary generalization and raised intracranial pressure. Contrast CT scanning again revealed tuberculomas with surrounding oedema. Decompression therapy and phenytoin again led to clinical improvement. At present the patient continues on isoniazid, rifampicin and pyridoxine with prednisolone 20 mg daily.

DISCUSSION
Why should intracranial tuberculomas expand during chemotherapy? The explanation is not irregular medication, drug resistance, or failure of drugs to penetrate the blood–brain barrier. Some workers suggest that the reaction may be due to an immune response at the site of these lesions, with products that cannot be discharged as they could from
the lungs\textsuperscript{1,4,5}. Tuberculomas have usually shown themselves after about 3 months' treatment, though the interval has been as short as 30 days and as long as 12 months\textsuperscript{1,3}. Our patient showed the paradoxical response on two separate occasions, after 44 days and after 4 months, while on successful treatment for Pott's disease, and others have recorded such events even after completion of treatment\textsuperscript{4}.

Any course of antituberculosis chemotherapy should be completed, and intracranial pressure should be controlled. The condition may eventually resolve with medical treatment alone\textsuperscript{1}. Until the results of medical treatment have been rigorously assessed, these patients should probably be kept under observation for several years.

**Intra-abdominal haemorrhage from mesenteric angiosarcoma**

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Malignant mesenteric angiosarcoma is a rare tumour. The histological diagnosis is difficult, probably leading to underestimation of its true incidence. We describe a case that presented with haemorrhage.

**CASE HISTORY**

A 61-year-old man presented as an emergency with a 1 day history of right iliac fossa pain and nausea. His past medical history was unremarkable apart from a myocardial infarct in France 1 year previously, for which he was on warfarin. On examination, he was afebrile, had a regular pulse of 84 bpm but was hypotensive with a blood pressure of 94/68 mmHg. He was tender in the right iliac fossa with peritonism and slight abdominal distension.

Initial blood tests were normal apart from haemoglobin of 11.8 g/dL and an International Normalized Ratio (INR) of 3.6. Abdominal ultrasound scan showed free fluid and a mass in the right iliac fossa. After resuscitation with intravenous fluids and fresh frozen plasma, urgent laparotomy was performed.

During operation, 2.6 L of blood was found in the abdominal cavity, the bleeding originating from a tumour connecting the distal ileum to the appendix, initially thought to be a Meckel's diverticulum.

*En bloc* resection of the ileum containing the tumour and appendicectomy were performed. The postoperative period was uneventful, and the patient was discharged 1 week later.

Macroscopically, the tumour comprised a ruptured thick, dark grey tissue mass arising in the small bowel

**REFERENCES**


Figure 1 Medium power view of the tumour stained with haematoxylin and eosin, demonstrating the characteristic epithelioid cells with irregular anastomosing vascular channels. In places, these epithelioid cells pile up to form papillary projections