Paradoxical development of brainstem tuberculoma during treatment for tuberculous meningitis

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The development of intracranial tuberculoma during treatment for tuberculosis have been described previously. A decrease in steroid therapy was thought to be responsible. We report a 30-year-old woman with tuberculous meningitis who developed brainstem tuberculoma while being treated with corticosteroids and anti-tuberculous therapy.

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Case report

A 30-year-old woman with prior good health was admitted with symptoms of fever, cough, and headache. Her symptoms lasted for one week and did not respond to ciprofloxacin. On admission, her body temperature was 36.8°C but there was neck stiffness. A computed tomography (CT) scan of the brain was normal, and a spinal fluid examination showed a white cell count of 284×10^9/L (normal range, $0\text{-}5 \times 10^9$/L) [90% lymphocytes, 10% polymorphonuclear leukocytes], protein of 3.4 g/L (normal range, < 0.40 g/L), glucose level of 2.5 mmol/L (normal range, 2.8 - 4.4 mmol/L) [concurrent blood glucose, 7.6 mmol/L, normal range, 3.9 - 6.1 mmol/L].

A diagnosis of tuberculous meningitis was made and she was given rifampicin 600 mg, isoniazid 300 mg, pyrazinamide 2 g, ethambutol 750 mg, and streptomycin 0.75 g, daily; dexamethasone 2 mg was also given intravenously three times daily. Ampicillin and cefotaxime were also given. Both her fever and headache started to decrease one week later, and she became afebrile two weeks after starting treatment. Five weeks later, her CSF culture grew *Mycobacterium tuberculosis*. Dexamethasone was continued at 1mg daily with rifampicin, isoniazid, and pyrazinamide at the same dosages as before.

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Two months later, she complained of the return of her headache for two weeks and that it was much worse than before. There was a nonspecific numbness on the left side of her body, although examination could not demonstrate any objective neurological deficit. In particular, all cranial nerves, fundi, and motor power signs were normal. A magnetic resonance image (MRI) of the brain showed a discrete lesion in the brainstem with a co-existing hydrocephalus which had not been present in the first CT (Fig 1).

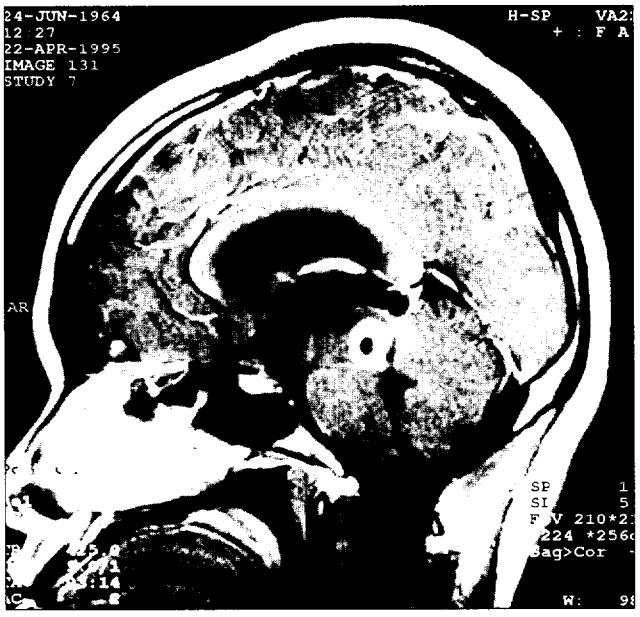

Fig 1. Tuberculoma with ring enhancement in the midbrain region on T1-weighted image after injection of IV gadolinium

The therapy was changed to rifampicin 600 mg, isoniazid 300 mg, pyrazinamide 2 g daily and dexamethasone 4 mg, four times daily. She remained afebrile throughout her second admission. Her headache started to decrease after two weeks. The numbness took two months to decrease. A ventriculo-peritoneal shunt was performed to relieve the hydrocephalus. Two weeks later, the dexamethasone was decreased to 8 mg daily and then to 6 mg daily and this was maintained for three months. Another MRI of the brain five months later (Fig 2) showed that the brainstem tuberculoma was still present. Clinically, she had no symptoms and could return to work, and she is still receiving rifampicin, isoniazid, and pyrazinamide.

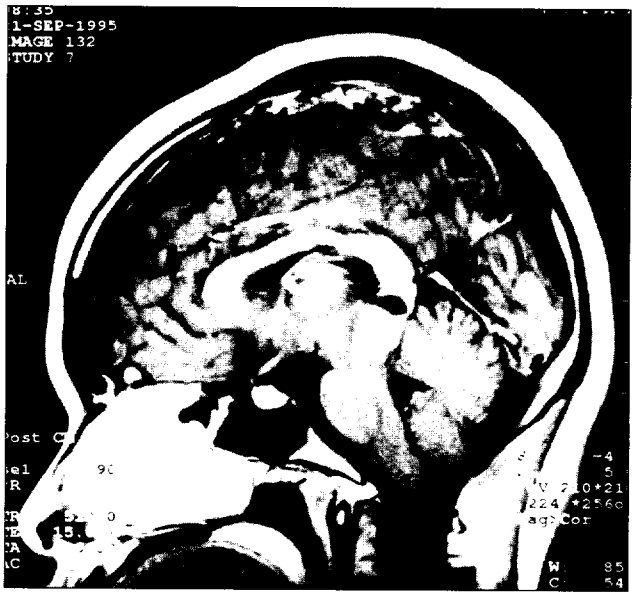

Fig 2. Tuberculoma is still present in the same region five months later

Discussion

The duration of symptoms before presentation was only one week. A review by Teoh et al shows the latent period to be between 1 month to 18 months, the mean period being 5.2 months.[1] It is unusual to have a short latent period, but it can occur and it is important, as tuberculosis is still common in our locality. Such a short latent period is shown by Talamas whose patient also had a latent period of one week.[2]

The first CT of the brain was normal. The first MRI that was done two months later showed brainstem tuberculoma and hydrocephalus. Since MRI is much better at showing up a brainstem lesion with its sagittal images, and can pick up additional information which may have been missed in the traditional CT scan, we cannot be certain that the brainstem lesion was absent at the beginning.[6] But the clinical setting of tuberculous meningitis, and the development of hydrocephalus strongly suggest that the tuberculoma developed and expanded during the first two months of treatment.

Bouchama et al report a brain biopsy positive rate of 12 of 15 patients with tuberculoma.[7] Their lesions were mostly located in the cerebral hemisphere (9 lesions), with three lesions in the cerebellum, and three with multiple sites. A brainstem lesion was present in one patient. In the analysis by Talamas in which 11 patients with brainstem tuberculoma were evaluated, 10 of 11 were treated with medical therapy alone, and seven remained well without adverse reactions.[2] One patient was operated on and died 36 hours after surgery.

The reason why tuberculoma expands during treatment is thought to be related to a disturbance in immunity when the patient is responding to therapy.[4] The question is whether this implies a failure in medical treatment or is a result of the low dose steroid.[8,5] In our case, the tuberculoma still developed even when dexamethasone was reduced to 1 mg daily. The best treatment for avoiding the development and expansion of intracranial tuberculoma is still to come. Anti-tuberculous therapy and corticosteroids have been accepted as the cornerstones for treatment, because more patients have a better functional recovery when compared with those who received surgery.[1,9]

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