

Intracranial Tuberculoma Manifesting During Treatment

S.K. Ajay
B.B. Lakhkar
N. Bhaskaranand

Tuberculoma is one of the commonest intracranial space occupying lesions in children in India and is known to respond well to antituberculous treatment (ATT). One peculiar characteristic is appearance of new tuberculomas or expansion of already existing ones in patients who were on ATT and were responding well. Thirty two such cases have been reported in the English literature(1) but only one such case is reported in the Indian literature(2) though there are two Indian reports published in other foreign journals(3,4). We hereby report a patient with miliary tuberculosis in whom intracranial tuberculoma manifested during chemotherapy, while the other manifestations of the disease were satisfactorily responding.

Case Report

An 8-year-old girl, second of four siblings, from a low socioeconomic class, developed high grade fever which lasted

for about 20 days. X-ray of the chest showed miliary mottling. Rest of the investigations for pyrexia were negative. The family history revealed that two siblings were treated for tuberculosis based on a positive mantoux test and X-ray chest. One sibling died due to high grade fever of long duration; this patient was not investigated.

The patient was treated with rifampicin (13 mg/kg), isoniazid (10 mg/kg). Pyrazinamide (26 mg/kg) and ethambutol (22 mg/kg). Ethambutol was stopped four months later and pyrazinamide was discontinued after seven months. Rifampicin and isoniazid were however continued. A repeat X-ray chest after eight months of ATT showed clearing of miliary mottling.

In the ninth month of treatment, she was referred to this hospital, with early morning headache and vomiting of two weeks duration and giddiness and diplopia of six days duration. Examination revealed, that the child was ataxic, had a divergent squint, lateral gaze nystagmus (fast component to the left), hypotonia and positive Babinski reflex on the left side. Other cerebellar signs were bilateral but more on the left side. A contrast CT scan of the head showed multiple hypodense lesions in the posterior fossa with ring enhancement and hydrocephalus (*Fig 1*).

The doses of rifampicin and isoniazid were increased to 20 mg/kg and 14 mg/kg respectively and pyrazinamide (25 mg/kg) was restarted. Streptomycin was added as the fourth drug. Suboccipital craniotomy was done. The vermis had herniated into the cisterna magna. Around 10 cc of pus was aspirated after splitting the vermis and entire abscess capsule was resected out.

*From the Department of Pediatrics,
Kasturba Medical College, Manipal 576
119.*

*Reprint requests: Dr. B.B. Lakhkar, Associate
Professor, Department of Pediatrics,
Kasturba Medical College, Manipal 576
119.*

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A modified ventriculocisternal shunt was introduced. Histopathological report of the aspirate showed features suggestive of tuberculosis. ELISA test for HIV antibodies was negative.

Two weeks after surgery the child was discharged from the hospital with improvement in cerebellar symptoms, squint and the general condition. The plantar reflexes were flexor on both sides. ATT was continued for one and half years postoperatively. Follow up, a year after the surgery, showed only minimal residual weakness of the left lower limb which was evident only on toe and heel walking.

Tuberculomas of the brain are isolated foci of caseous or proliferative tuberculosis. They may remain silent for a long time and later create symptoms. They routinely respond to antituberculosis treatment.

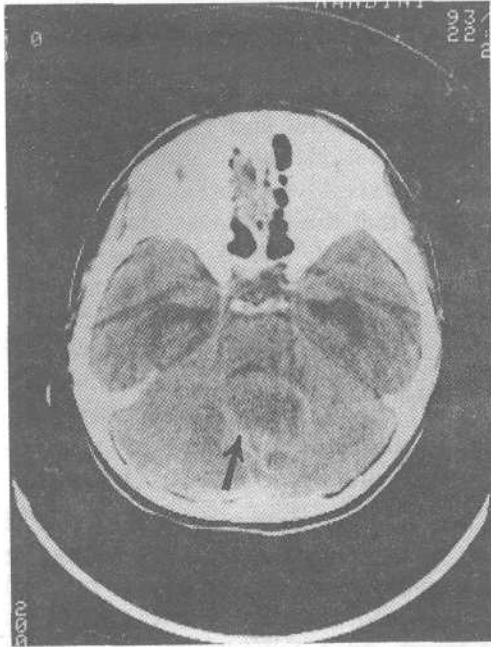


Fig 1. Contrast CT scan showing multiple hypodense lesions in the posterior fossa with ring enhancement.

Surgical management is needed very rarely. Paradoxical increase or appearance of tuberculomas during chemotherapy is an interesting finding.

Teoh, *et al*.(1) reviewed 10 cases of intracranial tuberculomas developing during treatment for tuberculosis. They found that the latent period *i.e.*, the time between the start of chemotherapy for original tuberculous infection and presentation of the intracranial tuberculoma was as early as two weeks to as late as 20 weeks. In another reported case latent period was 18 months(5). In our case it was eight months.

The original infection in tuberculoma could be pulmonary, meningeal or miliary(1,3). The chemotherapy in our case was appropriate as seen by improvement on the X-ray chest repeated eight months after starting ATT. Tuberculomas still developed despite the use of drugs with good central nervous system penetration. It, therefore, appears that drug dosage, drug resistance and central nervous system penetration have limited role in development of tuberculomas while on therapy. Teoh, *et al.* also noted similar findings(1). Some authors have tried increasing the dosage of ATT or have added a new drug when tuberculomas developed but others maintained the same dosage. In all above circumstances patients responded well. Our patient responded to increase in the dosage and the surgical management. The exact mechanism of development of intracranial tuberculomas while on ATT is uncertain. It possibly has an immunological basis similar to the enlargement of regional lymph nodes seen in patients being treated for glandular tuberculosis(6). It is possible that treatment improves the ability of the host to localize the disease with more cellular reaction becoming visible on CT scan.

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