Tuberculous abscess of the brain

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Summary

A bacteriologically confirmed case of tuberculous abscess of the brain is reported. Tuberculous brain abscess unlike tuberculoma does not exhibit the typical granulomatous changes and the diagnosis is confirmed by demonstration of tubercle bacilli either by staining or culture. An antituberculous regime should be started immediately the diagnosis has been established.

Introduction

Although involvement of the brain by tuberculosis is not uncommon on the Indian subcontinent, tuberculous brain abscess, an encapsulated collection of pus containing viable tubercle bacilli is quite rare. Bannister (1970) described one case and mentioned five others (Evans and Smith, 1931; Rand, 1935; Singh, Pandya and Dastur, 1968). Devadiga *et al.* (1969) reported one case of bacteriologically confirmed tuberculous brain abscess. Recently, Rab *et al.* (1975) described one case of tuberculous brain abscess proved by demonstration of tubercle bacilli in the pus, and by histology.

In several series of tuberculomas of the brain it was mentioned that pus or pus-like material was found. Obrador and Urquiza (1948) recorded one case in which the pus did not show any organisms and the culture was also negative. Arseni (1958) had only one case containing pus in his series of 201 tuberculomas of the brain treated surgically. Higazi (1963) mentioned one tuberculoma containing pus but did not give operative or post-mortem details. Dastur and Desai (1965) in a series of 107 tuberculomas, described eight containing pus-like material but it was not mentioned whether the pus contained tubercle bacilli or whether the material was caseated tubercular debris. One case of cystic tuberculoma was reported by Dastur, Desai and Dastur (1962) and another by Rao, Subrahmanyam and Sathe (1963).

The authors now describe a case of tuberculous brain abscess which was confirmed bacteriologically.

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The histological sections of capsule showed chronic non-specific infection.

Case report

A 20-year-old female was first admitted in the psychiatry unit in July, 1974, with a 10-year history of progressive impairment of memory, and insomnia. For one year she had had intermittent headaches, vomiting, right-sided focal convulsions and had experienced several attacks of brief unconsciousness. On examination, she was anaemic and ill nourished. Fundi showed papilloedema. Motor and sensory systems were normal. There was no neck stiffness. She was transferred to the authors' unit for investigations. Her haemoglobin was 8 g/dl; total leucocyte count $8 \times 10^9/l$, differential count polymorphs 64%, lymphocytes 32%, eosinophils 2%, monocytes 2% and ESR 17 mm in the first hour. Tuberculin test was negative. Chest X-ray film showed no abnormality. X-ray skull revealed erosion of dorsum sellae and posterior clinoid processes. Echo-encephalography showed shift of midline echo by 6 mm to the right. Left carotid angiography revealed proximal rounded shift of anterior cerebral artery to the right on A-P view and posterior and downward displacement of pericallosal artery on lateral view. Operation was refused by the husband and the patient was taken home.

The patient was re-admitted 2 years later with 2-months' history of continuous headache; frequent vomiting; right-sided weakness; incontinence of urine and faeces; altered sensorium and right-sided focal fits. She was drowsy and disorientated. She had optic atrophy, left ptosis, upper motorneurone right facial palsy, right hemiparesis and dysphasia. Tendon reflexes were brisk on the right side with extensor plantar. Investigations showed her haemoglobin was 9 g/dl, total leucocyte count $12.5 \times 10^9/l$, differential count polymorphs 68%, lymphocytes 27%, eosinophils 5%, and ESR 22 mm in the first hour. Tuberculin test was negative. Chest X-ray was normal. Skull X-ray showed signs of increased intracranial pressure. Echo-encephalography showed shift of midline echo by 8 mm towards the right.

Left frontal craniotomy revealed a deep, medial

placed frontal lobe abscess with a thick capsule containing 60 cm³ of thick pus. Only partial excision of the capsule was possible. The post-operative period was uneventful. A direct smear from the pus prepared with Ziehl Neelsen stain revealed no acid-fast bacilli. Histological studies of the abscess and its wall showed chronic non-specific inflammatory changes. Culture of the pus for pyogenic organisms was sterile. The patient was discharged on anticonvulsants.

One month later the patient was admitted with meningitis. On examination she was responding only to deep painful stimuli and was toxic. There was marked neck stiffness and Kernig's sign was positive. There was no improvement on treatment with gentamicin. Six weeks after operation *Mycobacterium tuberculosis* was cultured from some pus. She was put on antituberculous chemotherapy and steroids and showed remarkable improvement.

Discussion

Ramamurthi and Varadarajan (1961) and Dastur and Desai (1965) have reported that about 20% of all intracranial space-occupying lesions in India are tuberculomas. In Africa, where tuberculomas and tuberculous encephalopathy are common occurrences, 30% of intracranial tumours are tuberculomas (Scrinshaw, Garden and Taylor, 1968). In 1933 Garland and Armitage found eighty-nine tuberculomas in 13 000 post-mortems performed in Leeds, England. Evans and Courville (1938) found forty-three cases of tuberculomas in 15 000 post-mortems. However, these reports do not mention tuberculous brain abscess. Thus, tuberculous brain abscess is rare compared to tuberculoma.

According to Rand (1935), tuberculous brain abscess, unlike tuberculoma, does not show the typical granulomatous changes such as collection of epithelioid cells and giant cells around central area of caseation. Instead, tuberculous abscess shows only chronic non-specific inflammatory changes as seen in the present case. Diagnosis is established only with the demonstration of tubercle bacilli. In the present case a tuberculous aetiology of the abscess was confirmed by positive culture.

If the tuberculous aetiology is not confirmed and anti-tuberculous treatment is not enforced at the

earliest date, there is every likelihood that the infection will spread, leading to death of the patient. It is the authors' opinion that all chronic brain abscesses should be treated as being tuberculous unless proved otherwise. Anti-tuberculous chemotherapy should be discontinued only after excluding a tuberculous aetiology.

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