Tuberculous brain abscess

Case report

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The authors describe a case of tuberculous cerebral abscess of the frontal lobe that developed 1 year after an episode of acute miliary tuberculosis. The development of such a lesion indicates a persistence of infection and an immunological breakdown which may partly have been due to protein malnutrition.

Key Words • brain abscess • tuberculosis

Tuberculous brain abscess, an encapsulated collection of pus containing viable tubercle bacilli, is quite rare. Bannister described one case and mentioned five others in the literature as fully investigated and undoubted tuberculous abscesses. However, the pus was not analyzed and the clinical and histopathological findings were not described in two of three cases reported by Sinh, et al. Therefore, only four cases of tuberculous abscess have been fully documented and bacteriologically confirmed. Pus or pus-like material is mentioned in isolated reports of cerebral tuberculomas as well as in several series of intracranial tuberculous space-occupying lesions. Obrador and Urquiza recorded one case in which the pus did not show any organisms and the culture was also negative. Arseni, in an analysis of 201 tuberculomas treated surgically, found one lesion containing pus. Higazi mentioned one tuberculoma containing pus but did not give operative or postmortem details. Dastur, et al., in a series of 107 tuberculomas mentioned eight containing pus-like material but again gave no details as to whether the material was caseated tubercular debris or frank pus containing tubercle bacilli.

A bacteriologically confirmed case of tuberculous cerebral abscess is described here. The clinical evolution of the lesion and the angiographic and histopathological findings are discussed.

Case Report

A 14-year-old girl was admitted on April 23, 1973, after 1 month of fever, chills, abdominal pain, and jaundice. There was no history of tuberculosis in the family.

First Admission. The patient was found to be emaciated, anemic, and toxic, with frank jaundice. Her pulse was 110/min, respiration 15/min, and blood pressure 120/75 mm Hg. Her temperature varied between 100 and 103.5°F. There were fine crepitations at both lung bases; the liver was tender and enlarged.
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5 cm below the costal margin. Optic fundi showed choroid tubercles. Other systems were normal.

Laboratory investigations were as follows: hemoglobin 9.2 gm%; leucocytes 4200/mm³ with neutrophils 68%, lymphocytes 27%, monocytes 3%, and eosinophils 2%; erythrocyte sedimentation rate fell 10 mm in the first hour. Platelet count was 85,000/mm³; prothrombin time 27 sec with a control of 14 sec. Her serum bilirubin was 4.5 mg%. Total protein was 5.15 gm with albumin 2.7 gm%, alkaline phosphatase 15 B.L. units, serum glutamic pyruvic transaminase (SGPT) 21, serum glutamic oxaloacetic transaminase (SGOT) 120, lactic dehydrogenase (LDH) 200. Thymol turbidity was 9. Tuberculin test was negative in all dilutions. The cerebrospinal fluid (CSF) was normal. Urine showed a trace of albumin, bile pigments, and plenty of erythrocytes. Chest x-ray films showed bilateral miliary opacities. An electrocardiogram done on admission was normal but during her stay in the hospital, the patient developed transient changes suggestive of pericarditis along with a pericardial friction rub which disappeared after a week. A liver biopsy showed normal lobular architecture interspersed with multiple granulomas showing epithelioid cells and giant cells. A kidney biopsy was normal.

The patient was treated with streptomycin sulphate 1 gm/day intramuscularly, isonicotinic acid hydrazide (INH), 300 mg/day, ethambutol 1000 mg/day, and paraaminosalicylic acid, 10 gm/day. At the time of her discharge from the hospital on September 17, 1973, she was afebrile, chest x-ray films had returned to normal, and the liver enlargement and choroid tubercles had regressed. She was maintained on ethambutol, INH, and PAS and was followed as an outpatient; however, her attendance was erratic.

Second Admission. The patient was readmitted on March 14, 1974, with a 10-day history of headache, vomiting, and drowsiness. On examination, her temperature was 100°F; she was conscious and oriented, but demonstrated some inattention and apathy. She had signs of meningeal irritation, a slight right lower facial weakness, and bilateral papilledema. Her liver was palpable 4 cm below the right costal margin and was tender. Other systems were normal.

Laboratory investigations showed the following findings: leucocytes 11,600/mm³ with neutrophils 70%, lymphocytes 26%, monocytes 3%, eosinophils 1%, and ESR 25 mm fall in the first hour. Tuberculin test was negative in all dilutions. Urinalysis, chest x-ray, and electrocardiographic findings were normal. Lumbar puncture showed a rise in pressure with a protein of 55 mg% and 8 lymphocytes/mm³. Straight radiographs of the skull showed evidence of increased intracranial pressure. At this stage a diagnosis of intracranial space-occupying lesion was made.

A brain scan with Tc-99m showed an area of increased isotope concentration in the left frontal lobe. This was confirmed by electroencephalographic findings and by an echoencephalogram which showed a slight shift of midline structures to the right. Percutaneous carotid angiogram demonstrated a bilobulated abscess in the left frontal lobe (Fig. 1). In view of the past history of disseminated tuberculosis, and the absence of an obvious infection, it was assumed to be of tuberculous etiology.

Operation. A frontal craniotomy revealed that the cortex was under great tension. A brain needle was introduced posteriorly through the tip of the frontal lobe. A firm resistance was encountered at a depth of 2 cm. The needle was removed and the tip of the frontal lobe excised, exposing the anterior surface of the large abscess. The capsule of the abscess was penetrated with a brain needle and 40 cc of dark brown pus was evacuated, with a reduction of the intracranial tension. The abscess was subsequently enucleated by gentle dissection. The bilobulated abscess was delivered out of the cranium in two large pieces (Fig. 2).

Pathological Examination. On gross inspection, the abscess had a thick wall with three nodules projecting into it measuring 1 to 2 cm in diameter. A direct smear from the pus, prepared with Ziehl-Neelsen stain, revealed many acid-fast bacilli. A growth of *Mycobacterium tuberculosis* was obtained on culture of the pus after 5 weeks.

Histological studies showed what appeared to be the wall of an abscess. The wall was composed of dense hyalinized collagen with large vessels in it. In parts it appeared to be the dura. The fibrotic area was surrounded by a mixture of granulation tissue with a large
FIG. 1. Lateral (left) and anteroposterior (right) carotid angiograms showing the lobulated outline of the abscess in the venous phase.

number of young vessels and collections of epitheloid cells. No granuloma or giant cell or caseation necrosis was seen. A few areas of necrosis filled with cellular debris of the type seen in liquefactive abscess were also seen. Fresh imprints showed acid-fast bacilli (Fig. 3).

Postoperative recovery was uneventful. The patient was discharged home on May 18, 1974, on antituberculous chemotherapy. At the time of discharge she had gained weight, was afebrile, and showed no evidence of any neurological defect.

The angiographic findings in this case were characteristic of a chronic abscess. Capsular blush outlining a bilobed mass was seen in the late arterial phase; such an angiographic appearance has not previously been described in relation to a tuberculous cerebral abscess.

Discussion

Bacteriological and histological evidence confirmed the tuberculous etiology of the frontal lobe abscess in this case. The tuber-

FIG. 2. Photograph showing the two abscesses removed at surgery.

FIG. 3. Photomicrograph showing the histology of the abscess. Ziehl-Neelsen stain, × 400.
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culous infection in the brain probably dated back to the period when she had acute miliary tuberculosis. This may have left her with cerebral tubercles with viable bacilli. The subsequent development of the abscess could result either from bacterial multiplication or from immunological breakdown. While the former may have been due to erratic or unsatisfactory chemotherapy, this cannot be the entire explanation. Spontaneous recovery can take place in some patients with confirmed tuberculous infections of the cerebrospinal fluid.8 We believe the breakdown in immunity was more relevant in this case. She was anergic to tuberculin in both her illnesses, and serum albumin was low on both occasions.

The severity of tuberculosis in malnourished individuals has been well documented.16 Protein deficiency as well as vitamin A, D, and C deficiencies have all been known to increase the severity of the disease by reducing the resistance to tuberculous infection both in humans and experimental animals.10 Ward, et al.,18 found an association between previous tuberculous infection and cerebral gliomas; they suggested that the gliomas arose as did the tuberculosis in an environment where immunity was impaired. Another explanation might be that our patient's immune response may have been altered by the administration of corticosteroids; however, this is not tenable, because her course lasted only 1 month at which time she had apparently recovered from her previous illness, and also because she had been tuberculin-negative from the very beginning.

Tuberculous involvement of the brain is not uncommon. It is reported to be common on the Indian sub-continent.8 In Africa, where tuberculomas and tuberculobus encephalopathy are common occurrences, 30% of intracranial tumors are tuberculomas.16 Ramamurthi and Varadarajan14 and Dastur, et al.,14 have each reported that about 20% of all intracranial space-occupying lesions in India are tuberculomas. In 1933, Garland and Armitage8 found 89 tuberculomas in 13,000 autopsies performed in Leeds, England. Evans and Courville7 found 43 cases of tuberculomas in 15,000 autopsies. However, these reports do not mention tuberculous brain abscess.

Tuberculosis occurring in developing countries has many differences from that seen in Europe or North America.12 As a result, unusual presentations may be found in immigrant populations in countries that have eradicated tuberculosis.11 There are few similar reports because in countries with the facilities for sophisticated neurological investigations and treatment, the problem of tuberculosis does not exist; however, neurosurgeons should be aware of this treatable condition.

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