Tuberculous brain abscess
Case report

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An abscess was removed from the left occipital region in a 73-year-old woman with no previous history of tuberculosis. The patient later died from aspiration bronchopneumonia. Autopsy revealed a basilar tuberculous meningitis and miliary tuberculosis in the peritracheal lymphatic glands, the liver, the spleen, and in isolated areas of the lungs. No chronic tuberculous foci were noted in any area. Including this case, only 18 instances of tuberculous abscess have been reported.

KEY WORDS • brain abscess • tuberculosis

Tuberculous meningitis is the most frequent form of bacillary localization in the central nervous system (CNS). Tuberculomas of the CNS constitute 25% of the lesions occupying the brain in African and Asian countries, in contrast to approximately 1% in the United States and in European countries. Tuberculous abscess of the brain is rare, and the pathogenesis is not completely clear. Adams, in 1896, was probably the first to refer to this clinical entity. Thiébaut and Philippides pointed out the necessity of distinguishing tuberculous abscess of the brain from tuberculomas, which have a liquefied center. However, there are few reports of cerebral tuberculous abscesses that have been properly verified. In a series of 201 tuberculomas, Arseni encountered only one that contained pus. Higazi described a similar lesion but gave very few data. Dastur and Desai found eight lesions with pus or a pus-like substance among 107 tuberculomas, but no exact data were given about their characteristics. Mathai and Chandy found one case among 143 tuberculomas. Sinh, et al., found three such lesions among 70 tuberculomas, but in two of the three cases no attempt was made to look for the tubercle bacillus. More recently, Bannister described a case of tuberculous brain abscess with identification of tubercle bacilli in the pus. Dinakar and Rao reported another case with identification of the bacillus by histological study.

Whitener presented another case of tuberculous cerebral abscess, and made an exhaustive search of the world literature, finding 57 cases designated as tuberculous cerebral abscess, but only 16 fulfilled his rigid criteria based on the microscopic, histological, and bacteriological aspects. He discussed the theories of the pathogenesis and made a clinical analysis of the previous 16 cases.

This paper presents the autopsy findings of a new case of tuberculous cerebral abscess in a woman without previous history of tuberculosis. The abscess was excised, and a miliary dissemination to other parts of the body was discovered later at autopsy.

Case Report

This 73-year-old woman noted the onset of dizziness, frontoparietal headaches, and ringing in the ears in early December, 1975. Ten days later, she presented with a mild right hemiparesis, and slight mental confusion.

Examination. General physical examination was normal. The neurological examination demonstrated mental confusion alternating with psychomotor agita-
tion and aphasia. She had Grade II papilledema, and right hemiparesis, predominantly of the right arm, with a positive right Babinski sign. There were no meningeal signs. Blood count showed 10,000 leukocytes, 70% segmented polymorphonuclear leukocytes, 28% lymphocytes, and 2% band forms. Bleeding, coagulation, and prothrombin times were normal. The sedimentation rate was not determined on admission. Urinalysis was normal. The electroencephalogram showed a focal lesion in the left rolandic-temporal cortex.

Skull films were normal and the admission chest film did not demonstrate any pathology. A radionuclide brain scan showed a pathological accumulation of radioactive material in the left occipital region. Echoencephalography revealed a 5-mm deviation of the midline to the right. Left carotid angiogram demonstrated a forward displacement of the pericallosal and the Sylvian groups (Fig. 1). There was also a left-to-right displacement of the thalamostriate veins. Three days after admission, the patient was transferred to the neurosurgical service because of progressive neurological deterioration.

Operation. On January 11, 1976, a left parieto-occipital craniotomy was performed. The parietal and occipital convolutions appeared pale and distended, and yellowish pus was aspirated through a subcortical puncture and sent to the laboratory. The cortex was opened and the abscess capsule, which measured $3 \times 1 \times 2$ cm, was excised en bloc. Culture of the pus proved the presence of *Mycobacterium tuberculosis hominis*, sensitive to rifampicine (rifamycin), isonicotinic acid hydrazide (INH), and ethambutol.

Pathological Examination. Histological examination of the capsule demonstrated an internal necrotic zone with polymorphonuclear leukocytes. Outside this zone there was a granular reaction, rich in macrophages, of which some had foamy cytoplasm and others had an epithelioid appearance, with a tendency to form clumps. There were a few giant cells of the Langhans type, abundant lymphocytes, and a few plasmatic cells. These cell types were preferentially arranged in the adventitia of the vessels and in the area around them. The Ziehl-Neelsen stain showed acid-fast bacilli, some of which had been phagocytized by the foamy cytoplasmic macrophages. In the external zone, there was a glial reaction (Fig. 2).

Postoperative Course. The patient's hemiparesis, aphasia, and level of consciousness improved after the operation. She was treated with rifampicine, INH, and ethambutol. On January 30, 1976, she experienced fever, a decreased level of consciousness, and a worsening of the right hemiparesis. There was no neck rigidity. Two cerebrospinal fluid (CSF) studies demonstrated glucose levels of 30 and 37 mg/100 ml; total protein content of 166 and 174 mg/100 ml; and 30 and 60 white cells, respectively. Cultures of the CSF were negative. The blood count showed 7500 white cells, 57% polymorphonuclear cells, 10% band forms, 30% lymphocytes, and 3% monocytes. Sedimentation rate was 48 mm in the 1st hour. Blood urea nitrogen and blood glucose were normal, and a chest film showed no evidence of active tuberculosis.

A repeat left carotid angiogram demonstrated signs of a space-occupying mass at the previous site. On
February 13, 1976, the original flap was re-elevated and a zone of the right occipital lobe with areas of gliosis mixed with pockets of pus was excised. The histological and bacteriological studies were identical to those of the earlier specimen. The patient did not improve after the operation and died 10 days later from aspiration bronchopneumonia.

Postmortem Examination. The autopsy showed a basal acute meningitis with a large quantity of bacilli. Foci of pus and large zones of gliosis were seen at the operative site. In the rest of the parenchyma, multiple cortical foci of recent necrosis were found secondary to intravascular coagulation. In addition, there were foci of suppurative aspiration bronchopneumonia, thrombotic microangiopathy in glomerular capillaries, isolated miliary tubercles in the lungs, and disseminated tubercles in the lymphatic ganglia of the hilar portion of the lungs, in the liver, and in the spleen.

Discussion

Diagnosis of Tuberculous Abscess

This patient represents a case of tuberculous abscess diagnosed after surgery using strict criteria. In this patient, there had been no previous tuberculous involvement, and there was neither clinical nor radiological evidence of active tuberculosis. At postmortem examination an acute dissemination was discovered, but no chronic tuberculous foci were found. These findings led us to believe that the two operations had provoked the hematogenous dissemination which caused the recurrence of miliary tuberculosis. Tubercle bacilli can be carried to the brain by blood from the lungs; however, in this case there was neither clinical nor radiological evidence of any pulmonary lesion.

Bannister proposed that, in the absence of active tuberculous foci elsewhere in the body, the presence of small, inactive, calcified lesions in the lungs could act as a source of infection for a tuberculous cerebral abscess. Rab, et al. reported a similar case that appeared 1 year after the miliary pulmonary tuberculosis was healed. Whitener established the following rigid anatomical, histological, and bacteriological criteria which served as a basis for the selection of his 16 cases: 1) macroscopic evidence of a cavity with pus in the center; 2) the existence of an inflammatory reaction in the wall of the abscess, composed predominantly of granular vascular tissue with acute and chronic inflammatory cells, especially polymorphonuclear leukocytes; and 3) positive culture of Mycobacterium tuberculosis or demonstration of acid-fast bacilli in the pus or in the wall of the abscess.

The signs and symptoms of cerebral abscess in the 18 cases reported to date are as follows: focal neurological deficit, 71%; headache, 47%; fever, 46%; seizures, 35%; mental alterations, 24%; and neck rigidity, 17%. The period between onset and presentation ranges between 1 and 4 weeks. Our patient met most of these guidelines; however, she did not have...
fever, rigidity of the neck, or seizures. In agreement with the findings of Whitener,\textsuperscript{21} the glucose in the CSF was low in our patient, there were few cells, and the cultures were negative on two occasions.

We would like to stress the following points: 1) In the differential diagnosis of purulent abscess, it is important to test for tuberculosis in other parts of the body; 2) tuberculomas appear at earlier ages than do tuberculous abscesses, they have a slower evolution, and in those cases the glucose in the CSF remains within normal limits;\textsuperscript{17} 3) neck rigidity and the decrease of glucose in the CSF are regular and notable findings in tuberculous meningitis;\textsuperscript{19} 4) the patient's reaction to tubercular infection depends on the state of individual immunity, on the extension of the tubercular infection, on the type of tissue infected, and on the medical treatment given;\textsuperscript{18} 5) the patient's nutritional status is important: deficiencies of vitamins A, D, and C increase the severity of the tuberculous infection.\textsuperscript{22}

Pathogenesis, Treatment, and Prognosis

The inoculation of a large number of bacilli in a hypersensitive individual provokes an exaggerated exudative phase with massive caseation. The softening of the caseous material with an influx of polymorphonuclear leukocytes can form pure pus.\textsuperscript{4,16} Dannenberg and Sugimoto\textsuperscript{5} proved the participation of certain enzymes (proteinases, nucleases, and lipases) in the liquefaction of the caseous material. Whitener\textsuperscript{21} sought an explanation for the rare histological features presented by the tuberculous focus with infiltration of polymorphonuclear leukocytes, and thought that the formation of pus and the great multiplication of tubercle bacilli might be a possible pathogenic theory.

The treatment of tuberculous cerebral abscess should include the excision of the abscess and anti-tuberculous chemotherapy. Therapy must begin as soon as the diagnosis is made, with a combination of ethambutol, INH, and rifampicine, because these drugs cross the blood-brain barrier readily. This treatment should be given for 1 to 2 years. Recurrences of the cerebral abscesses have been reported upon termination and even during administration of the medication.\textsuperscript{8,21}

The preoperative diagnosis of cerebral tuberculous abscess presents difficulties, because its clinical and radiological manifestations are similar to those of purulent cerebral abscesses and tuberculomas. The recent incorporation of computerized tomography diminishes the possibility of error to a certain extent. A correct preoperative diagnosis and administration of specific chemotherapy can reduce the high mortality, which is around 40% even with chemotherapy. Evidence of tuberculosis, such as positive cultures from the CSF or positive radiological findings, along with a positive family history, could assist an early diagnosis.