A tuberculous abscess of the brain

Case report

CARYS M. BANNISTER, B.Sc.(Oxon), F.R.C.S.
Department of Neurosurgery, The General Infirmary at Leeds, Yorkshire, England

Even before the introduction of streptomycin in 1944 for the treatment of tuberculosis, tuberculous abscesses of the brain as opposed to the more common tuberculomas were rarely reported. Rand described one case and found nine others in the literature. Of these ten cases only two, that of Evans and Smith and Rand, were fully investigated and shown to be undoubted tuberculous abscesses. Recently Singh, et al., reported in detail three other cases. These five cases remain the only fully documented lesions of this sort in the literature, although in several recent series of cerebral tuberculomas pus was found. Higazi, describing a series of six tuberculomas, stated that one lesion contained pus, but he did not supply operative or postmortem details. Das, and Desai, reporting 107 tuberculomas, mentioned that in eight of the lesions pus was found, but again supplied no further details.

This report is a description of the clinical features and necropsy findings of a tuberculous cerebral abscess.

Case Report

The patient, a Yorkshireman of 54, was in good health and working as a hospital porter until the onset of his illness in December, 1966. The illness began with a left-sided focal fit, followed by a transient left hemiparesis. A week later he had a second left-sided focal fit, and after it there was a persistent weakness of the left leg. During the next week he had two further left-sided focal fits, and following them he was noted to have developed a hemiparesis involving the left side of the face and both left limbs.

Examination. The patient was admitted for investigation initially to a medical unit at the Leeds General Infirmary, but was transferred to the Neurosurgical Unit early in January, 1967. By that time there had been considerable deterioration in his condition; he was confused and disorientated, he had bilateral moderately severe papilledema, and a severe left hemiparesis involving the face, arm, and leg. The tone in the left limbs was increased, and the reflexes brisk. A left extensor plantar response was present. There was diminished sensation to pinprick over the whole of the left side of the body, and position sense was impaired in the distal joints of both left limbs. Plain x-rays of the skull were normal. A lumbar air encephalogram showed a depression in the roof of the right lateral ventricle. There was no significant displacement of the ventricular system toward the left. The lumbar cerebrospinal fluid contained 5 monocytes per cu mm and 60 mg% of protein.

A provisional diagnosis of a rapidly growing glioma in the right parietal lobe was made, and a biopsy of the lesion through a burr hole was planned.

Operation. Under local anesthesia, a cannula was passed through a right parietal burr hole into the brain, and at a depth of about 3 cm resistance was felt. With further pressure
From...I.N.A.H. throughout
specimen theloid
eroid organisms,
ques...500.

204
of penicillin,
wall pus.
numorous tubercle bacilli. H. & E., X 850.
showing necrotic cerebral
issue collections
of 100 mg of streptomycin, and 3 ml of
of the abscess cavity were instilled
nto the cavity.

Examination of the pus showed that it contained numerous pus cells. Routine techniques did not reveal any of the usual pyogenic organisms, but acid-fast bacilli were found. Subsequently, M. tuberculosis were cultured. Microscopic examination of the biopsy taken from the wall of the abscess cavity showed that it consisted of necrotic cerebral tissue and large collections of leucocytes. No epitheloid follicular systems were seen in the specimen even though a Ziehl-Neelsen stain revealed numerous acid-fast bacilli scattered throughout the pus (Fig. 1).

Postoperative Course. Once the infecting organism had been identified, the patient was given systemic streptomycin, P.A.S., and I.N.A.H. in appropriate doses. Emptying the abscess did not initially improve the patient. He remained confused, disoriented, and hemiparetic, and in addition, developed a dense left hemonymous hemianopia.

Six weeks later he deteriorated suddenly. Over a few hours he became deeply unconscious, developed a stiff neck, and a positive Kernig's sign. Examination of the lumbar cerebrospinal fluid showed 87 white blood cells per cu mm, 74% of which were monocytes, and a glucose content of 22 mg%. No tubercle bacilli were seen in the fluid nor were they cultured. In addition to the systemic medication, he was now given daily intrathecal streptomycin. On this regime he gradually improved and returned to consciousness after 48 hours. During the next 2 months he became rational and oriented; the left hemiparesis improved to the extent that he had almost full movement of the left arm and could walk with a stick. Serial x-rays of the skull showed that the abscess cavity was progressively decreasing in size.

The patient was sent to a convalescent home on full systemic antituberculous therapy, but within 2 weeks he had a second sudden relapse and had to be readmitted to the Neurosurgical Unit.

Second Examination. On admission he was unconscious and had a severe left hemiparesis with signs of meningitis. The cerebrospinal fluid contained 30 white blood cells per cu mm, the majority of the cells being monocytes. Again no tubercle bacilli were seen or cultured. Intrathecal streptomycin was recommenced.

Improvement was slow, but after 2 weeks the patient could answer simple questions. Two months later the hemiparesis had improved sufficiently to allow him to walk with the help of two people. His condition remained unchanged until July, 1967, when he began to deteriorate slowly. By September he could no longer answer questions, and the left hemiparesis had become total; by December, 1967, he had become unresponsive to all stimuli. Early in January, 1968, he developed hypostatic pneumonia and died. Right up to the time of death he was receiving full systemic antituberculous therapy and intrathecal streptomycin.

Postmortem Examination. Externally, the brain was swollen, and there was slight fibrosis of the basal meninges that included the foraminal exits of the fourth ventricle. A co-
Tuberculous abscess of the brain

Coronal section showed that there was a small abscess in the white matter of the right parietal lobe extending to the upper part of the outer angle of the body of the right lateral ventricle with which it communicated (Fig. 2). The abscess was filled with barium sulphate solution. It had a translucent gray capsule varying in thickness from being barely visible to up to 2 mm. Although the abscess cavity measured only 2 × 1 cm, opposed capsular tissue extended up to the surface in the superior parietal region, indicating that it was partially collapsed. The lateral ventricles were dilated, especially on the right side. The right foramen of Munro was almost obliterated by exudate. The fourth ventricle was moderately dilated. Many terminal secondary brain stem hemorrhages were seen.

Histological examination of the abscess showed that its cavity was entirely filled by macrophages containing barium sulphate particles. The capsule was composed of dense collagenous tissue infiltrated by small mononuclear cells, and bordered externally by a zone of gliosis (Fig. 3). There were no signs of tuberculous granulation tissue either in the abscess or in the ventricles.

The ependymal exudate was of two types. That lining the lateral ventricles consisted mainly of polymorphs with some small mononuclear cells; in places the ependymal cellular layer was deficient and subependymal gliosis had occurred. The second form of exudate consisted of small foci of macrophages containing barium sulphate particles together with fibroblasts and capillaries lying on the ependyma of the fourth ventricle and clustered around the exit foramina. Reactions to the barium sulphate may have been responsible for the blockage of the fourth ventricle. Both Gram and Ziehl-Neelsen stains failed to reveal any organism in either the ependymal exudate or in the wall of the abscess cavity.

Examination of the lungs showed widespread bronchopneumonia. There were also calcified nodules at both apices; although none of these appeared to be active tuberculous lesions, in the absence of foci elsewhere in the body, it must be presumed that one of the apical lesions acted as the source of infection for the cerebral abscess.

Discussion

During the early course of the patient's illness, excision of the abscess was considered, but before a decision could be made, he developed his first attack of meningitis. Be-

Fig. 2. Coronal section through the brain showing the abscess in the right parietal lobe, and the dilated ventricular system with its shaggy lining.

Fig. 3. Photomicrograph of a section through the abscess showing the cavity filled with macrophages, and its collagenous wall. H. & E., × 150.
cause he was making a satisfactory response to drug therapy, it was felt that an operative procedure carried too high a risk of causing further meningeal infection. Even after the second attack of meningitis, it was still hoped that the disease would finally be eradicated by drugs alone. In the latter part of the illness when it was obvious that drug therapy had failed, the patient's general condition was too poor to withstand the surgical procedure involved. In retrospect, early excision of the abscess after a short course of systemic antituberculous drugs would have been the treatment of choice.

It is interesting to note the similarity of the histological features of the abscess in Rand's case and ours. He found, as did we, a lesion consisting of a pus-filled cavity surrounded by a poorly-developed fibrous tissue wall in which there was little or no evidence of giant cell systems. The pus teemed with tubercle bacilli.

Summary

The clinical, laboratory, and necropsy findings in a patient with a tuberculous abscess of the right parietal lobe have been described. The disease ended fatally after nearly a year of antituberculous drug therapy given systemically and intrathecally.

Acknowledgments

I wish to thank Dr. Tallersall and Mr. Wall for permission to publish this case, and Dr. Harriman for the biopsy and necropsy reports.

References


Received for publication April 11, 1969.

Address reprint requests to: Carys M. Bannister, F.R.C.S., Department of Neurosurgery, The General Infirmary at Leeds, Great George Street, Leeds 1, Yorkshire, England.