Tuberculoma masquerading as a meningioma

Case report

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Intracranial tuberculomas may imitate, both clinically and radiologically, the more commonly observed intracranial tumors. This case report outlines the development of such a lesion masquerading as a typical meningioma of the frontal fossa. Discussion of intracranial tuberculomas follows, with special reference to clinical and radiological findings.

Key Words • tuberculoma • meningioma • angiography • intracranial pressure • brain tumor

Intracranial tuberculomas, both infratentorial and supratentorial, have been extensively reviewed in neurological and neurosurgical literature, particularly that of the developing nations. They are less frequent in Western Europe and North America, and may present themselves in unusual forms that challenge both the diagnostic capabilities of our clinicians and their investigative machinery. A review based on 2200 intracranial tumors seen in a single British neurosurgical unit from 1951 to 1972 revealed an incidence of 0.15%. A further report 3 years later warned of an increase in the frequency of intracranial tuberculomas, a reflection, perhaps, of increased immigration into Great Britain from India and surrounding regions. As many as 20% of intracranial tumors in India were found to be tuberculomas.

Tuberculomas producing intracranial mass effects with signs of raised intracranial pressure (ICP) and seizures occur rarely in the United States. Mayers, et al., reported a collection of cases in 1978, and emphasized the possible absence of extracranial tuberculosis in intracranial mass lesions of tuberculous origin. The following is a report of such a case, demonstrating geographical, radiological, and clinical features of a meningioma of the anterior frontal fossa.

Case Report

This 33-year-old Algerian electrical engineer suffered his first seizure in November, 1978, awakening with bilateral tremors of his arms, difficulty in breathing, and transient loss of consciousness. His wife witnessed a grand mal seizure.

First Admission. Neurological examination was normal. Initial investigations included a brain scan, which revealed a small concentration in the right frontal lobe, and a computerized tomography (CT) scan, which demonstrated a probable meningioma in the anterior portion of the falx.

Repeat CT scan and angiography were thought to be normal, and the patient was discharged after an unremarkable hospital course. Three subsequent seizures occurred in August, 1979, February, 1980, and May, 1980. All episodes occurred without an aura and lasted approximately 30 seconds. There was no loss of consciousness. Moderate right frontal headaches, experienced around the right eye, lasted about 1 hour when untreated; they were relieved by aspirin. During the 6 to 12 months previous to his present admission, the patient had noted an increasing prominence of the right eye with a sensation of slight protrusion. A similar feeling had involved the left eye for 1 to 2 months. In addition, during the previous 2 months, the patient had complained of wavy lines in his right visual field and intermittent flashes of light, lasting about 10 seconds, before both eyes. Ophthalmological examination at the beginning of June raised the possibility of bilateral early papilledema. A marked decrease in the patient's sense of smell accompanied the visual complaints. He also complained of occasional
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FIG. 1. Anteroposterior (left) and lateral (right) skull films demonstrating hyperostosis of the crista galli and planum sphenoidale.

bouts of "electrical current" in his stomach and head, lasting only a minute on the average. Functional enquiry revealed no exposure to infectious agents, fungal, tuberculous, or otherwise. There was no family history of these infections or of cancer. The patient had immigrated to Canada from Algeria at 20 years of age.

Second Admission. Anticonvulsant drugs were the patient's only medicine. A hemogram revealed a white blood cell count of 9300/cu mm, with an erythrocyte sedimentation rate (ESR) of 30 mm/hr (normal 0 to 20 mm/hr Wintrobe-corrected). Chest film was unremarkable. Skull films (Fig. 1) revealed hyperostosis of the crista galli and planum sphenoidale consistent with an olfactory groove meningioma without an accompanying abnormal calcification or vascular pattern. Tomograms of the crista galli demonstrated a minimal degree of hyperostosis with poor cortical definition. Sclerosis of the adjacent region of the planum sphenoidale was evident. A brain CT scan (Fig. 2) revealed significant frontal lobe edema, bilaterally surrounding a hyperdense lesion within the interhemispheric region of the frontal lobes. Enhancement of the lesion occurred with contrast infusion in addition to posterior displacement of the anterior horns of the lateral ventricles—again consistent with a falx meningioma. A slight backward displacement of the right anterior cerebral and left pericallosal arteries, presumed secondary to marked edema of the frontal lobes, was demonstrated on cerebral arteriography. The left anterior meningeal artery had a normal course, and no abnormal tumor vessels were seen. No blush was observed on the late venous phase. The most anterior portion of the superior sagittal sinus was not visualized. Electroencephalography (EEG) gave evidence of a mild intermittent disturbance of cerebral activity over both frontocentrotemporal brain regions without predominance on either side. Ophthalmological evaluation 1 month after his latter assessment disclosed a marked bilateral chronic papilledema with temporal retinal folding and some deep hemorrhages. Repeat examination 9 days later revealed no new pathology, even though the patient complained of transient episodes of bilateral visual obscurations.

Operation. The patient was maintained on diphenylhydantoin and began a course of dexamethasone 7 days before operation. He underwent a bifrontal craniotomy for a presumed falx meningioma. The operation revealed a tumorous expansion of the anterior falx which was avascular and rubbery in consistency, and extending outward in a butterfly fashion into both frontal fossa beds. Adhesions joined this mass to the medial frontal lobes. The sagittal sinus was patent. Both frontal lobes were extremely edematous. The dura overlying both frontal lobes was excised, the sagittal sinus and falx anterior to the coronal suture sectioned, and the tumor removed. As much of the tumor carpet as possible, extending into the frontal fossa beds, was also removed. The gross impression was that of a very scirrhous meningioma, although frozen section examination revealed only granulomatous disease.

Pathological Examination. The right frontal pole was sent for pathological examination. Histopathology of the excised tissue revealed multiple small necrotizing granulomas with surrounding lymphocyte accumulation, epithelial cells, and Langhans giant cells. No meningitis was found. Ziehl-Neelsen, silver methenamine, Giemsa, and periodic acid-Schiff stains failed to reveal the presence of any organisms. Auramine-rhodamine stain did not show fluorescent tuber-
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FIG. 2. Plain (left) and infused (center) preoperative computerized tomographic scans indicating enhancement of the frontal lesion and surrounding edema. Right: Postoperative scan showing marked reduction of edema.

cle bacilli. Cultures of the excised tissue proved negative for bacteria and tubercle bacilli.

Postoperative Course. Some improvement was noted funduscopically, with reductions in papilledema consistent with reduction in ICP after craniotomy. A postoperative Mantoux skin test was positive, at 12 mm induration after 48 hours. Steroids were tapered and discontinued, with institution of antituberculous therapy consisting of isonicotinic acid and hydrazide (INH) and rifampin. The patient was discharged from the hospital 2 weeks postoperatively under medication. He was in satisfactory condition, without further clinical seizure activity and without notable neurological deficit apart from decreased olfactory sensation.

Discussion

Clinically, tuberculomas resemble other intracranial tumors without any distinguishing pathognomonic features. The presence of a prolonged clinical course suggesting a slowly progressive lesion is common. In their review, Ramamurthi and Varadarajan categorized patients in two groups; those having signs of increased ICP with or without localizing neurological signs, and those with progressive neurological disability in the absence of elevated ICP. The patients of the latter group usually display features of tuberculosis extracranially.

As in any other pathological intracranial process, the clinical signs and symptoms of intracranial tuberculosis are related to the site of the lesion. A significant frequency of false clinical localization exceeding that found with gliomas has, however, been noted in cases of intracranial tuberculosis, particularly in cases of focal seizures and hemiplegia. The reported incidence of multiplicity has ranged from 16% to 32%.

Multiple lesions may go unnoticed in individual cases where evidence is not forthcoming radiographically, although the patient may display neurological abnormalities inconsistent with the presumed solitary lesion. In his series of 201 intracranial tuberculomas, Arseni reported a focal cerebral syndrome in 68% of cases, symptoms of raised intracranial tension in 72%, and generalized or focal epileptic activity in 85%.

A presumptive diagnosis of tuberculoma is possible on the basis of personal history of tuberculosis or simply on report of previous contact. However, as in the present example, this is not always the case. Reported evidence of concomitant extracranial tuberculous infection has ranged from 30% to 50% of cases, although some 50% may have normal chest radiographs. Fever in cases of intracranial tuberculoma does not appear as a prominent differentiating feature for cases of glioma, although fever can last longer in intracranial tuberculoma cases. Visual complaints and papilledema are frequent, but their rates of progression should not be used as a means of differentiating tuberculosis from other intracranial tumors. Eleva- tion of ESR is of questionable value as an aid to differential diagnosis, since it occurs in as few as 15% of cases, similar to the incidence in cases of glioma. Skin testing with purified protein derivative (PPD) has on numerous occasions been negative in proven cases of intracranial tuberculosis.

Radiological features abound, but the evidence may be conflicting and could be misleading. The low incidence of intracranial calcifications and occasional absence in some series should warn against depending on their presence. Neighboring bone hyperostosis and sclerosis have not been commented upon previously, although they can occur, as in the present case. Angiographically, two types of picture have been recognized: a deep subcortical avascular lesion acting like a space-occupying lesion often requiring surgery, and
2) a less common, more superficial, occasionally infiltrating vascular tumor requiring operation in selected cases but responding well to antituberculous drug therapy. The reverse situation has also been reported, with deeply situated vascular tuberculomas and more superficial hard, nodular avascular lesions poorly responsive to antituberculous agents.

Abnormal looping of terminal branches of major intracranial arteries seen during the arterial phase of angiography may serve as a clue to the location of a tuberculoma, although on occasion the wall of a tuberculoma may best be seen in the capillary and early venous phases. Naturally, vessels may be displaced, narrowed, or occluded by the mass effect of the tuberculoma. Wide dural attachments have been demonstrated in some tuberculomas, with an enlargement of feeding meningeal arteries leading to these lesions. The CT scanner will demonstrate only a nonspecific intracranial mass, but it can provide an objective noninvasive means to follow the course of therapy of intracranial tuberculomas. Electroencephalographic patterns are potentially useful in localizing cerebral hemispheric space-occupying lesions, with correlative findings demonstrated in 82.5% of cases and lateralization alone in a further 13.9%. Patterns suggestive of a slow-growing lesion were noted in 75% of cases of tuberculoma. The EEG, however, has on a number of occasions been reported as failing to detect or localize significant lesions, and CT scanning remains more conclusive regarding localization of tuberculomas of small to moderate size.

Therapy of intracranial tuberculomas has improved greatly with the advent of antituberculous agents. Tuberculous meningitis, which complicated surgical excision of these lesions before the introduction of streptomycin, significantly raised the mortality to four times that of the operative mortality alone. Since the introduction of multiple antituberculous therapy, the mortality has fallen significantly. Steroids can be given safely as long as effective chemotherapy has been undertaken beforehand.

References


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