

Large cerebellopontine angle tuberculoma: a case report

Duży gruzliczak kąta mostowo-mózdkowego – opis przypadku

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Abstract

Tuberculoma involving the cerebellopontine angle is very rare. Preoperative neuroradiological features of such lesions may mimic neoplastic lesions and postoperative histopathological study brings the ultimate diagnosis. Here we present a patient with a large tuberculoma at the cerebellopontine angle who had another small lesion at the right fronto-basal region and was managed by surgical excision of the cerebellopontine angle lesion along with post-surgical antitubercular therapy for 18 months. On the 14th postoperative day, the patient developed status epilepticus, left hemiplegia and left-sided complete hearing loss. Computed tomography showed right frontal oedema. Then he recovered his motor function slowly and incompletely but left-sided hearing loss remained unchanged. Magnetic resonance imaging of the brain at 18 months after surgery showed no residual lesion with right frontal cortical atrophy.

Key words: cerebellopontine angle, tuberculoma, status epilepticus, hemiparesis, hearing loss, cortical atrophy.

Streszczenie

Gruźliczak w kącie mostowo-mózdkowym jest dużą rzadkością. Guz ten w przedoperacyjnych badaniach obrazowych układu może naśladować guzy nowotworowe, a ostateczne rozpoznanie ustala się na podstawie pooperacyjnego badania histopatologicznego. W pracy przedstawiono przypadek pacjenta z dużym gruzliczakiem w kącie mostowo-mózdkowym i z drugą mniejszą zmianą w okolicy czołowo-podstawnej prawej, który był leczony za pomocą chirurgicznego wycięcia, a następnie przez 18 miesięcy otrzymywał leki przeciwgruźlicze. W 14. dobie po operacji wystąpił stan padaczkowy, niedowład połowiczny lewostronny i lewostronna głuchota. W tomografii komputerowej stwierdzono obrzęk prawego płata czołowego. Następnie niedowład powoli i niecałkowicie ustąpił, ale lewostronna głuchota się nie zmniejszyła. W badaniu za pomocą rezonansu magnetycznego wykonanym po 18 miesiącach od operacji nie stwierdzono pozostałości guza, obserwowano natomiast zanik korowy w płacie czołowym prawym.

Słowa kluczowe: kąt mostowo-mózdkowy, gruzliczak, stan padaczkowy, niedowład połowiczny, głuchota, zanik korowy.

Introduction

Approximately 10-15% of all patients with tuberculosis have central nervous system (CNS) involvement [1] and it is seen most commonly in the developing countries. Although large series from developing coun-

tries continue to be reported [2] and incidence has increased for the last 20 years due to human immunodeficiency virus (HIV) infection, people of any age can be affected and the mycobacterium usually reaches the CNS by haematogenous dissemination from pulmonary lesions. Tuberculoma is found in 15-30% of cases of

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CNS tuberculosis [3] and is hemispheric in most cases [2]. Other, rare locations include the sellar area, cerebellopontine angle, Meckel's cave, suprasellar cistern, and hypothalamic region [3,4].

Central nervous system tuberculoma presenting as a solitary mass in an extrinsic location is rare. The cerebellopontine angle is a very unusual site for tuberculoma [5]. Only a few reports have been published in the literature. Here we describe a patient with a large cerebellopontine angle tuberculoma with another small lesion in the fronto-basal region; the patient developed status epilepticus, hemiplegia and unilateral complete hearing loss early after the surgery. Hemiplegia recovered slowly and incompletely but hearing loss remained unchanged with frontal cortical atrophy following surgical excision of the cerebellopontine angle tuberculoma.

Case report

A 12-year-old boy presented with occipital headache for the last 3 years. He had no history of visual disturbance, hearing loss, vomiting or convulsions. On neurological examination, higher psychic function, and cranial nerves, including fundoscopy, were normal, but he had left-sided cerebellar signs. Hoffman sign and Babiński sign were found on the right. Deep tendon reflexes on the right side were exaggerated. Muscle power and tone, as well as sensation, were normal. Magnetic resonance imaging (MRI) of the brain showed a large ($4 \times 4 \times 3$ cm) lesion in the left cerebellopontine angle compressing the cerebellum and brainstem (Figs. 1A-C) and there was also a small lesion in the right frontobasal region. The lesion was excised completely through left retrosigmoid suboccipital craniectomy. The lesion was easily separable from the cerebellum. Its consistency was peculiar (some parts were firm, some others elastic, and yet another sandy) and it was not attached to any cranial nerve. Histopathology revealed tuberculosis. After the histopathological diagnosis, the patient was put on standard antitubercular chemotherapy for 18 months (four-drug antitubercular chemotherapy with isoniazid, rifampicin, ethambutol, and pyrazinamide for the first three months was started and then maintenance therapy with isoniazid and rifampicin was continued over the next 15 months). Postoperatively, he was also on an anti-convulsant (phenytoin) that was started preoperatively.

On the fourteenth postoperative day the patient developed status epilepticus and was rapidly referred to the intensive care unit. Immediate intravenous injection of

diazepam was given twice but it failed to stop convulsions; then phenytoin was given intravenously in a bolus dose that stopped the convulsions. Intravenous phenytoin was continued for 72 hours. Left-sided hemiplegia and complete hearing loss on the left were noted. Computed tomography of the head performed on the fourteenth postoperative day showed no residual tumour but there was oedema at the right frontal lobe (Fig. 1D). His left hemiplegia gradually improved to Medical Research Council grade 3+/5 in the left lower limb and 4+/5 in the left upper limb. Hearing loss on left side did not recover at the final follow-up after 18 months. Magnetic resonance imaging at 18 months after the surgery showed no residual lesion (Figs. 1E-F) but there was cortical atrophy of the right frontal lobe including the motor area (Fig. 2).

Discussion

With the increasing number of tuberculosis cases, CNS tuberculosis is also increasing [5]. According to the World Health Organization, 8-10 million new cases of tuberculosis are diagnosed worldwide each year. Several factors, including HIV infection [6], intravenous drug abuse, immunosuppression, advanced age, alcoholism, malnutrition, poverty, transplantation, aggressive chemotherapy, immigration [1], homelessness and crowding are mainly considered to be responsible for the development and for the annual increase of disease. Tuberculosis involving the leptomeninges, brain and skull base is thought to spread by haematogenous dissemination from a primary source outside the CNS. The meningitic process may affect the cranial cerebrospinal fluid (CSF) pathway, the spinal subarachnoid pathway, or both [7]. Central nervous system involvement by the tuberculous bacilli can also manifest as hard or soft granulomas, tuberculous abscess, tuberculous cerebritis, pachymeningitis, spinal arachnoiditis, and intraspinal tuberculoma [7]. Parenchymal disease usually presents as either solitary or multiple tuberculoma, and can occur with or without meningitis [8]. Tuberculomas are histologically round or oval masses, or they may assume a more lobular configuration with the fusion of several smaller nodules [3]. Only ten percent of them are associated with concomitant meningitis [9]. We did not perform a CSF study, but there was no clinical or radiological evidence of meningitis or intraventricular lesions apart from a small lesion in the right frontobasal area that was also thought to be tuberculoma. Cerebel-

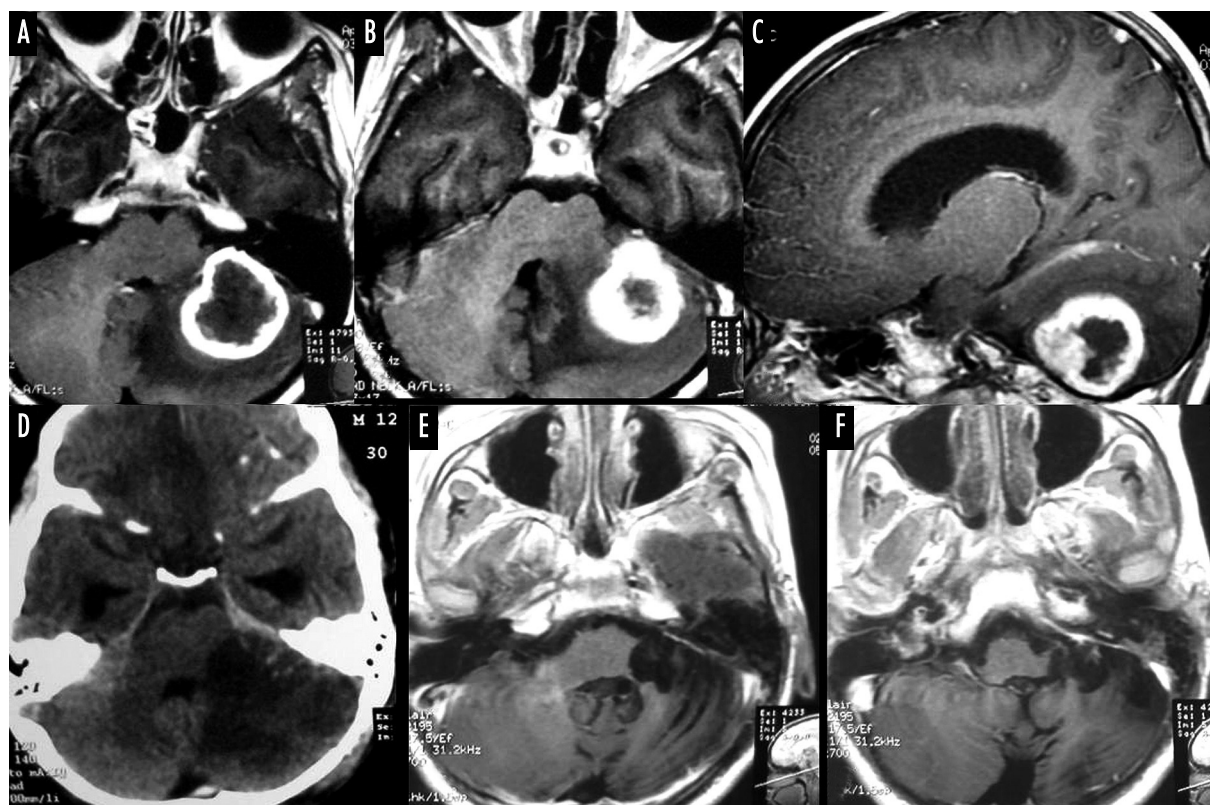


Fig. 1. Brain MRI in axial (A and B) and sagittal (C) plane showing left cerebellopontine angle lesion. D, postoperative brain CT at 14th day after the surgery showing no residual tumour but right frontal oedema. E and F, postoperative brain MRI in axial plane showing no residual lesion 18 months after the surgery

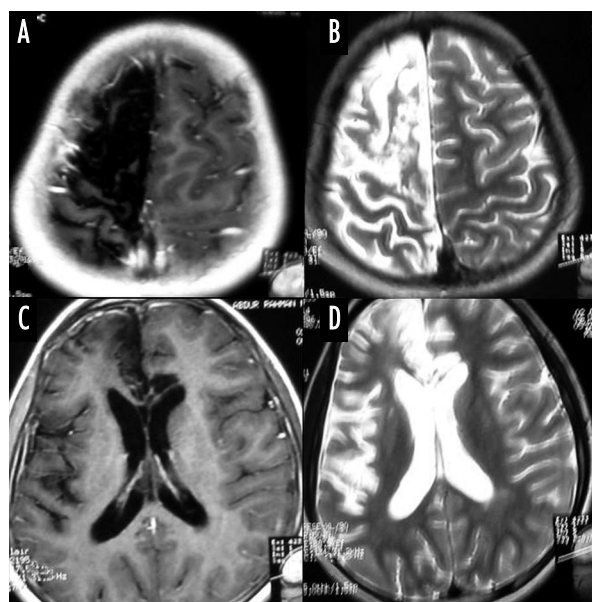


Fig. 2. Postoperative brain MRI (18 months after the surgery). T1-weighted (A and C) and T2-weighted (B and D) images in axial planes showing right frontal cortical atrophy

lopontine angle lesions usually present with hearing loss, or other cranial nerve deficit, cerebellar features, brain-stem compression features, hydrocephalus or headache. The only symptom in our patient was headache along with mild cerebellar and long tract signs.

Confirmation of CNS tuberculosis is only possible by isolation of *Mycobacterium* or histopathological identification of tuberculous granuloma, although various investigations including CT scan, MRI, magnetization transfer MRI [10], or in vivo proton MR-spectroscopy [11] have been suggested. The inappropriate neuroradiological diagnosis of meningioma, neurinoma, or even metastasis is usual in cases of tuberculoma [12].

Cerebellopontine angle tumours are most commonly acoustic schwannomas or meningiomas [5]. Other differential diagnoses include cholesteatoma, metastases, lymphoma, brain abscess, fungal infection, neurocysticercosis, sarcoidosis, etc. In our case, radiological findings suggested possible neoplastic lesions but the suspicion of tuberculosis was also high as the frequency of tuberculous lesions in India is high. Increased use of radiosurgery as a primary modality of treatment for

many CNS masses without prior pathological diagnosis is noted [5] but it may prove harmful and dangerous to the patient without histopathological diagnosis.

In such a paediatric patient, postoperative status epilepticus may be caused by any preexisting cortical lesion, by the ongoing antitubercular drug therapy or by the potentiating effect of both. Antitubercular drugs, especially isoniazid, can cause tonic-clonic seizures by the impairment of pyridoxine metabolism [13]. In the early postoperative period, the patient's status epilepticus was probably due to the combined effect of a frontal lobe lesion and the action of antitubercular drugs that caused severe oedema in that lobe and contralateral hemiplegia that recovered slowly and incompletely along with frontal lobe atrophy.

Disclosure

Authors report no conflict of interest.

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