Tuberculoma of the Pons

Sabine Knauer-Fischer, MD*, Jörg Schaper, MD†, Hans-Theo Janssen, MD*, and Felix Ratjen, MD*

A case of a pontine tuberculoma in a 12-year-old male from Somalia is presented. Cranial magnetic resonance imaging, performed in Abu Dhabi 1 month before admission in Germany revealed a tumorous lesion in the pons with surrounding edema; a presumptive diagnosis of a pontine glioma was made. Chest radiograph disclosed a pulmonary infiltrate with cavitation, as well as hilar and left mediastinal lymphadenopathy, suggestive of active tuberculosis. Tuberculostatic therapy led to an improvement of the patient’s clinical status and a significant reduction in the size of the pontine tuberculoma. Intracranial tuberculomas rarely are seen in industrialized countries. They should, however, be considered as an important part of the differential diagnosis of intracranial space-occupying lesions. In most cases, as in this patient, conservative therapy provides good or excellent results.

© 1999 by Elsevier Science Inc. All rights reserved.


Introduction

In Western industrialized countries, intracerebral tuberculomas represent only a small part of all intracranial space-occupying lesions; however, their incidence in developing countries is significantly higher [1-4]. Signs of systemic tuberculosis are missing in 40-70% of patients, making the diagnosis of intracerebral tuberculomas difficult, particularly because these lesions can have a multifocal morphology and variable clinical signs.

The most common presentation of tuberculomas is focal neurologic signs or seizures. They vary in size from 0.5-3 cm but may occasionally be larger. Singular lesions are more frequent, but 15-20% of patients have multiple lesions, and grape-like clusters of tuberculomas have been described [1,4]. Tuberculomas have a tendency to be peripheral but may occur anywhere in the parenchyma. Infratentorial tuberculomas are more frequent in affected children (59-63%), whereas lesions are mostly supratentorial in adults [5]. Symptomatic tuberculomas can be associated with a surrounding vasogenic edema [6].

The neurologic signs, especially partial seizures and focal neurologic deficits, correlate with the localization of the tuberculoma but frequently do not correlate with its extent [1]. Intracranial tuberculomas can cause all the signs of intracranial space-occupying lesions and must therefore be considered in the differential diagnosis of brain tumors.

A case of a 12-year-old male from Somalia, in whom a brain tumor was suspected initially but who had an intracranial tuberculoma, is reported.

Case Report

A 12-year-old male from Somalia living in Abu Dhabi presented to the authors’ hospital in Germany. A brain tumor was suspected, and he was transferred to the Department of Pediatrics for further diagnosis and therapy.

For 8 months the boy had severe headaches of changing localization, occasional vomiting, and dry cough. Five months before admission the headaches had worsened and vertigo, as well as gait disturbance, appeared. Dexamethasone therapy (1 mg twice daily) had been started 3 months before admission because a brain tumor was suspected. A few days before the patient’s departure from Abu Dhabi to Germany a pontine tumor was diagnosed by magnetic resonance imaging (MRI).

Physical examination on admission revealed a 12-year-old male who appeared ill, with a cushingoid habitus and reduced consciousness. Auscultation revealed rales over the left lung. The liver was palpable 2 cm inferior to the right costal margin; no enlarged lymph nodes were evident.

Neurologic examination revealed pupils of equal size, diplopia because of paresis of the right sixth nerve, paraplegia of both legs, and ataxic gait. Tendon reflexes were hyperactive, and the plantar response was flexor bilaterally.

On chest radiograph a pulmonary infiltrate was present in segment 6 of the left lower lobe, with cavitation and additional hilar and left mediastinal lymphadenopathy (Fig 1). Abdominal ultrasound revealed increased echogenicity of the liver parenchyma, without focal lesions and mild splenomegaly.

MRI of the brain revealed a pontine mass lesion with a maximal diameter of 2.7 cm and a hypointense (dark) wall on T₁-weighted and
T₂-weighted images. The lesion was hypointense on T₂-weighted and isointense on T₁-weighted images. The wall demonstrated strong enhancement on T₁-weighted images after intravenous gadolinium, with apparent nodular, granulomatous thickening of the wall itself and some lobulation of the mass. There was a large surrounding edema and slight dilatation of the third and lateral ventricles because of compression of the fourth ventricle (Figs 2 and 3).

Erythrocyte sedimentation rate was 40/70, blood leukocytes were 7.5x10⁹/L, bilirubin in serum was 1.5 mg/dL, lactate dehydrogenase (LDH) 445 U/L, and aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma glutamyl transferase, creatinine, electrolytes, coagulation, and blood gases were within normal ranges. The patient was negative for HIV-1 and -2 antibodies; HBs-antigen in serum, anti-HBc-antibodies, and Hbe-antigen were positive. A cytospin preparation of the cerebrospinal fluid (CSF) contained 26 cells/mm³ (94% lymphocytes, 1% neutrophils, 5% monocytes). The CSF protein concentration was elevated (300 mg/dL).

A Mendell-Mantoux test with 10 IU tuberculin was positive, with an induration exceeding 10 mm. Acid-fast bacilli were found in gastric fluid. There was neither microscopic nor cultural evidence of acid-fast bacilli in urine or CSF. Polymerase chain reaction for Mycobacterium tuberculosis in CSF was negative as well.

Tuberculostatic therapy with isoniazid 300 mg once daily, rifampin 600 mg once daily, pyrazinamide 1 gm twice daily, and streptomycin 1 gm daily intramuscularly was initiated; dexamethasone was maintained at a dose of 0.5 mg twice daily.

On his fifth day in the hospital, he complained of increasing frontal headaches and lost consciousness. His pupils were no longer reactive to light, and the patient developed rigor of both arms. Cranial computed tomography revealed a progressive occlusive hydrocephalus requiring external drainage of CSF for 14 days until a permanent ventriculoperitoneal shunt could be implanted. Four days after implantation of the external drainage the patient regained consciousness. Subsequently the patient recovered, and the protein content in the CSF returned to normal.

One week after the start of the quadruple tuberculostatic therapy, liver enzymes increased (ALT 97 U/L, AST 55 U/L, and LDH 431 U/L). Three weeks later a further increase (ALT 1,208 U/L, AST 725 U/L, LDH 601 U/L, and bilirubin 27.4 mg/dL) made it necessary to discontinue two of the hepatotoxic drugs (isoniazid and rifampin) after 27 days of therapy. On the twentieth and twenty-first day of tuberculostatic therapy no mycobacteria were detectable in gastric fluid.

The patient was discharged 4 weeks after admission. His neurologic status had returned to normal.

Follow-up MRI 1 year after specific therapy had been started revealed a significant reduction in the size of the pontine tuberculoma. A residual change in the central pons was seen, with a maximal diameter of 1 cm and signal characteristics suggestive of calcifications. There was no pathologic contrast enhancement of the lesion after intravenous gadolinium administration (Fig 4). Laboratory data were all...
within normal ranges, and the patient continues to be well without any neurologic deficits. All medications were discontinued.

Discussion

Intracranial tuberculomas are rare, and only 4% are located in the brainstem [1,4,7,8]. Typically, most pontine tumors in childhood are astrocytomas [9]. Because of the therapeutic implications, intracranial tuberculomas play an important role in the differential diagnosis of intracranial space-occupying lesions.

Tuberculomas mostly occur after 5 years of age and present with focal neurologic deficits or seizures. The diagnosis is difficult because an active tuberculous infection of other organs is frequently not, or no longer, apparent. As in the authors’ patient a brain tumor is often the initial clinical diagnosis. Depending on their size and location, intracranial tuberculomas can have many signs mimicking primary central nervous system tumors, such as high intracranial pressure, focal neurologic deficits, and seizures [1,3,4,7,10].

Hints with regard to the differential diagnosis of an intracranial space-occupying lesion for a tuberculous focus are a positive tuberculin skin test (25-75%), signs of an active or healed pulmonary tuberculosis on chest radiograph (25-50%), relapsing cough, and the appearance of the lesion on cranial computed tomography or MRI [2,11]. Laboratory tests, such as differential cell counts of blood leukocytes and erythrocyte sedimentation rate, are normal in most cases; often only the CSF protein level is raised. Cultures for Mycobacterium tuberculosis of CSF are rarely positive [4,12]. The polymerase chain reaction of CSF, which was negative in the authors’ patient, appears to be the most sensitive diagnostic tool, with a reported sensitivity of 75% [4].

The MRI finding of a mass lesion with a T1-weighted and T2-weighted hypointense rim with strong T1-weighted contrast enhancement, as well as T2-weighted hypointense content, is the characteristic appearance of a gummatous tuberculoma in comparison with caseous or other bacterial abscesses or central tumor necrosis with hyperintense signal [5]. With modern imaging facilities the granulomatous, nodular character of the wall becomes evident if the tuberculoma has reached a size of 1.5-2 cm.

Tuberculomas usually dissolve with tuberculostatic therapy within 3-6 months [4-6,12]. Isoniacid and pyrazinamide can reach therapeutic levels in CSF even when meninges are not inflamed; streptomycin and rifampicin achieve therapeutic levels only in the case of meningitis [13,14]. Although the authors’ patient received quadruple tuberculostatic therapy with rifampin, isoniazid, pyrazinamide, and streptomycin for only 4 weeks because of marked elevation of liver enzymes, subsequent therapy with pyrazinamid and streptomycin was successful in resolving his intracerebral lesions and neurologic symptoms.

References