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Retrospective study of 23 pathologically proven cases of central nervous system tuberculomas

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Abstract

Introduction: Extrapulmonary manifestations of tuberculosis involving the central nervous system (CNS) due to haematogenous spread are not a rare entity. It presents as meningitis or tuberculoma. Tuberculoma is a granulomatous inflammatory process mimicking a neoplasm radiologically, so usually a biopsy is performed.

Material and results: Our study consisted of 23 pathologically proven cases of tuberculomas between 1988 and 2003. Patients were discussed clinically, radiologically and histologically. Headache, fever, weight loss and weakness are the most common clinical manifestations. Our patient's ages vary from 3 to 67 years with a mean of 31.8 years. Ninety-five percent of patients had bad social, economic and nutritional conditions. None of them were infected by human immunodeficiency virus (HIV). All patients had similar contrast-enhancing lesions radiologically. The majority of tuberculomas were located supratentorially. Only one patient presented two foci of (cerebral and cerebellar) tuberculomas. Nineteen tuberculomas were intracerebral; two were located in the cerebellum and one was intramedullary. Among those lesions, one cavernous sinus tuberculoma and one sellar tuberculoma were identified. Only two patients underwent stereotactic biopsy and 21 patients underwent surgical excision. Histopathologic examination revealed granulomatous inflammation with central caseous necrosis in all patients.

Discussion: Diagnosis of tuberculoma can be difficult, and in most of our cases, the clinical diagnosis was 'neoplasm'. For this reason, clinicians must always be aware of it and consider it in the differential diagnosis of central nervous system mass lesions.
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Keywords: Central nervous system tuberculoma; Granulomatous inflammation; Meningitis

1. Introduction

The central nervous system (CNS) involvement comprises approximately 10–15% of all tuberculous infections [1]. It is considered the most feared complication of the disease. Even patients who are treated adequately are associated with a high morbidity and mortality rate [2]. Being a worldwide health problem, it is usually seen in all ages and usually caused by haematogenous dissemination of bacilli from pulmonary lesions. Tuberculous meningitis is especially common in patients younger than 5 years of age in many developing countries [3]. Diffuse exudative leptomeningitis, serous tuberculous meningitis, epidural or subdural abcess formation and intracerebral or intraspinal tuberculoma formation are the features of CNS tuberculosis. Cerebellar involvement is slightly more common in children (6 months to 6 years). Tuberculomas are mainly described in the cerebrum, but other common locations include tegmentum and paracentral lobulus. It is rarely found in the medulla spinalis. Major gross features of tuberculomas are small round or oval shaped nodules, ranging from 2 to 12 mm in size and tend to form a lobulary architecture. The central necrotic area is surrounded by an oedematous brain tissue with reactive gliosis. Oedema is much more remarkable in brain abscess [3,4].

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Tuberculoma is a tumour-like mass and its diagnosis necessitates preferably, a stereotactic biopsy followed by, a histopathological examination. Central caseous necrosis surrounded by epithelioid histiocytes and Langhans giant cells. Mycobacterium tuberculosis can be demonstrated by a histochemical reaction using Erlich Ziehl–Nielsen's (EZN) method. The presence of inflammatory exudate and bacilli is less common in supratentorial tuberculomas with cystic, fibrous and calcific features [2].

The purpose of this study is to stress that CNS tuberculomas may be misdiagnosed preoperatively as a tumour and discuss the difficulty arising in the demonstration of the bacilli, and also discuss the entities that should be considered in the histopathological differential diagnosis of CNS tuberculomas.

2. Results

Our retrospective study consisted of 23 patients diagnosed with central nervous system tuberculoma between the years 1988 and 2003, and all biopsies and surgical excisions were studied in a single center in our neuropathology department. Only two patients underwent stereotactic biopsy and 21 patients underwent surgical excision.

The age of the patients ranged from 3 to 67 years (mean 31.8 years). Thirteen patients were female (57%) and 10 patients were male (43%). Patients were mainly presented with solitary lesions.

Among 23 patients, only 1 had cerebral and cerebellar (4%) contrast-enhancing lesions (Figs. 1 and 2). Nineteen lesions were intracerebral (82%), two were located in the cerebellum (8%) and only one was intramedullary (4%). Among the cerebral lesions, eight frontal (42%), five parietal (26%), two parieto-occipital (10%), one cavernous sinus (5%), one occipital (5%), one cerebellopontine angle (5%) and one pituitary tuberculoma (5%) were identified.

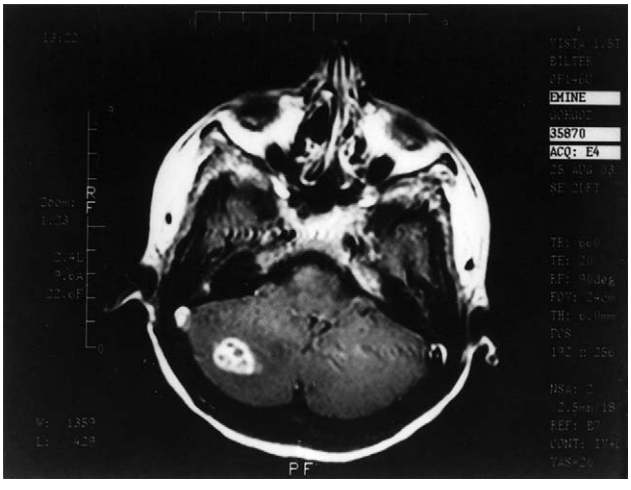

Fig. 1. T1-weighted axial MRI showing right cerebellar tuberculoma with contrast enhancement giving to it a multilocular appearance and perifocal oedema.

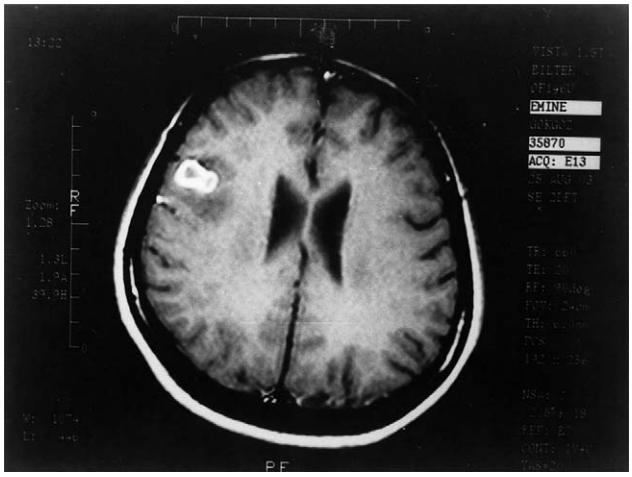

Fig. 2. T1-weighted axial MRI showing right parietal tuberculoma presenting thick contrast enhanced capsule and perifocal oedema.

Weight loss, weakness, fever, vomiting, nausea and headache were the most dominant clinical manifestations in all patients. Seizures and hemiparesia occurred in 50% of patients. The evidence of tuberculosis in the lungs and in the body elsewhere was found in three patients (13%). These cases should be accepted to be the milliar form of disease. CSF examination was performed in three patients with milliar features and the results of the examination were compatible with lymphocytic meningitis. Two patients presented with intracranial lesions while they were treated for meningitis. CSF cultures were positive in two patients and one patient had a positive PCR result in CSF. Three patients had a positive family history of tuberculosis or recent exposure to active tuberculosis patients. Two patients had underlying immunocomprimising conditions. None of them was infected by HIV. The first one had undergone splenectomy and had used corticosteroids before. The other one was a haemodialised patient, having chronic renal failure due to polyarteritis nodosa. All analyzed patients had low social and economic conditions.

Neuroimaging results are obtained from the biopsy forms filled out by clinicians. Localizations, the number of the lesions and available MRI and/or CT scan details were noted for all patients when the biopsy forms were reviewed. On CT scans, central hypodense lesions surrounded by a contrast-enhancing rims were usually observed. Differential diagnosis included necrotic tumours or pyogenic abscess, radiologically. We had the occasion to examine 10 patients' magnetic resonance imaging (MRI) findings, where lesion sizes varied from 0.5 to 4 cm. For this reason, we do not know the pattern enhancement and the distribution of the lesion size in all patients. Solitary lesions were mainly detected in our cases. Multiple lesions were observed in only one patient whom MRI finding is presented (Figs. 1 and 2). MRI showed space-occupying lesions: an iso-hipo intense in T1 sequence, a contrast-enhanced lesion in T1 sequence and hy-

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