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Case report

Cerebral tuberculoma: critical review of two cases

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Abstract

Central nervous system tuberculosis can have protean manifestations. We review the clinical and radiological findings in two cases of cerebral tuberculoma in immuoncompetent patients. These two cases represent the spectrum of variations of clinical and radiological features of cerebral tuberculoma and highlight the controversies in the approach to diagnosis and management.

Keywords: Cerebral tuberculoma; Intracerebral mass; Tuberculoma; Tuberculosis

INTRODUCTION

Manifestations of central nervous system tuberculosis include tubercular meningitis, tuberculoma, tuberculous abscess, and less commonly cerebral miliary tuberculosis, tuberculous encephalopathy, tuberculous encephalitis, and tuberculous arteritis[1]. Tuberculoma is encountered in only 15% to 30% cases of central nervous system (CNS) tuberculosis and is seen most commonly in the developing countries[2-4]. In developed countries intracranial tuberculoma has become a rarity where immigrants from endemic countries are usually affected [5-10].

CASE REPORTS

Case 1

A 35 year gentleman presented with progressive headache of one month duration, weakness of right upper and lower limbs and slurring of speech of 15 days duration. The headache was associated with vomiting and blurring of vision. There was no history of cough, fever or seizures. There was no history of contact with tuberculosis. There was no significant previous medical history. His general and systemic examination was unremarkable. Higher mental functions were normal except slurring speech. There were no meningeal signs. Cranial nerves were normal except bilateral papillodema. Tone was increased in right upper and lower limbs with grade 4/5 power. Deep tendon reflexes were exaggerated on right side with right plantar extensor. Motor power, reflexes were normal on left side with flexor plantar. Blood investigations were normal except raised ESR (30 mm in 1st hour). Chest X-ray was normal. A serological test for HIV was negative. Sputum for AFB was positive. Tuberculin skin test was positive. CT scan brain showed enhancing lesion suspected as high grade glioma (Figure 1). Computed tomography (CT) showed an isodense lesion on plain scan that was enhancing after contrast administration involving left fronto-temporal lobe with features of multiple coalescing nodules, diffuse perilesional oedema, mass effect and midline shift, features suggestive of high grade glioma (Figure 1). In view of diffuse mass and with a suspicion of malignancy he underwent surgical decompression of the mass le-

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sion. Intra-operatively there was a deep seated, well defined, firm avascular mass (Figure 2). With these feature a diagnosis of tuberculoma was suspected and it was confirmed with histopathological examination of the specimen that showed a dense infiltration of lymphocytes and few plasma cells around focal small aggregates of epithelioid cells with central caseation necrosis and Langhans type giant cells (Figure 3). He was started on anti-tubercular chemotherapy [Isoniazid (INH), rifampin, pyrazinamide and ethambutol] with steroids and making gradual recovery.

Case 2

A 45 year female presented with low grade fever, loss of appetite and weight and productive purulent cough of one month duration. Fever was persisting in spite of a course of antibiotics. She had one episode of generalized tonic-clonic seizures two days prior to admission. There was no significant previous medical history. On general examination she was ill looking, thin built and dyspnoeic. Pulse rate was 98/minute, respiratory rate was 30/minute and temperature was 1 000 F. chest examination showed bilateral crepitations. There was no hepatosplenomeagly or lymphadenopathy. Higher mental functions and cranial nerves were normal. Motor and sensory examination was normal. There were no meningeal signs. Fundus was normal. CT scan showed enhancing lesion. Computed tomography (CT) showed isodense lesion on plain scan, enhancing after contrast administration with peri-lesional oedema and mass effect in right frontal lobe (Figure 4). Blood investigations showed lecocytosis and raised ESR (60 mm in 1st hour). Chest X-ray showed military tuberculosis (Figure 5). A serological test for HIV was negative. Sputum for AFB was positive. Tuberculin skin test was positive. Based on clinical and radiological evidence a diagnosis of milliary pulmonary tuberculosis with cerebral tuberculoma was made. She was started on anti-tubercular chemotherapy [Isoniazid (INH), rifampin, pyrazinamide and ethambutol] and making gradually recovery.

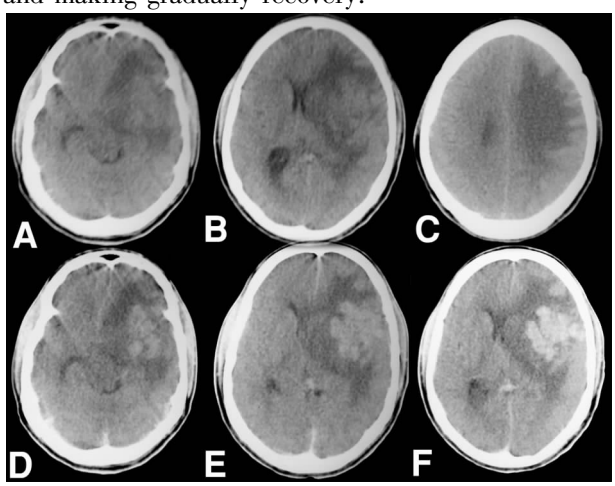

Figure 1 CT scans plain showing an isodense lesion in left fronto-temporal lobe showing coalescing nodular enhancement with peri-lesional oedema, mass effect and midline shift suspected as high grade glioma

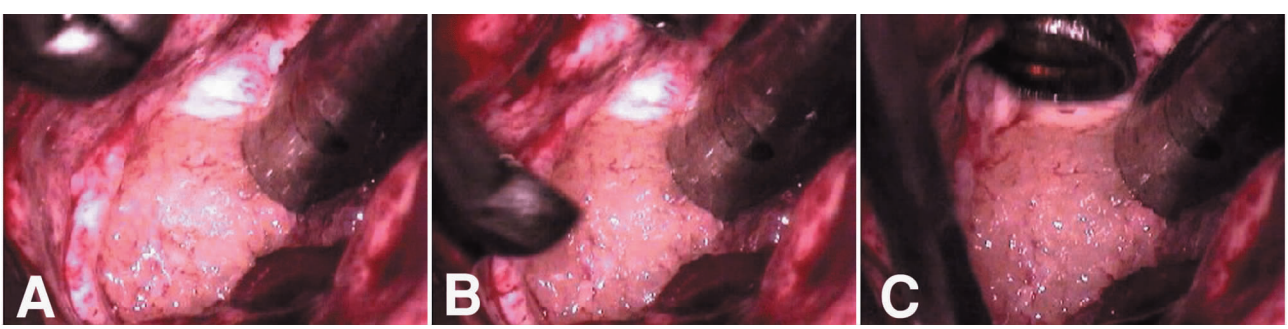

Figure 2 Intra-operative photograph showing pale yellow, avascular, well defined, deep seated mass suspicious of tuberculoma

DISCUSSION

Intracranial tuberculomas usually result from hematogenous spread from a distant focus of tuberculous infection and characterized by a conglomerate of small tubercles that join to form a mature tuberculoma with central caseation necrosis surrounded by a zone of fibroblasts, epithelioid cells, Langhans giant cells, and lymphocytes[11-14]. The diagnosis of intracranial tuberculomas is based on epidemiological, clinical and laboratorial information combined with radiological images and also an adequate response to

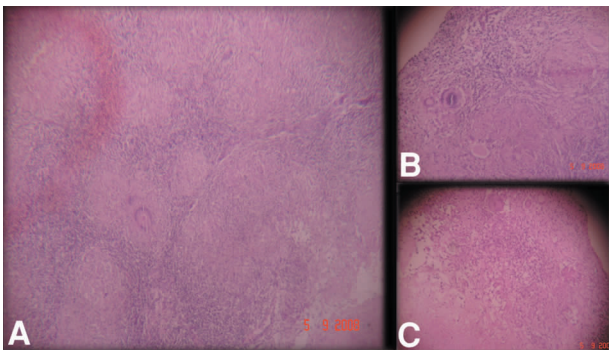

Figure 3 Photomicrographs showing a dense infiltration of lymphocytes and few plasma cells around focal small aggregates of epithelioid cells with central caseation necrosis and Langhans type giant cells (HE stain, A ×40, B and C × 100)

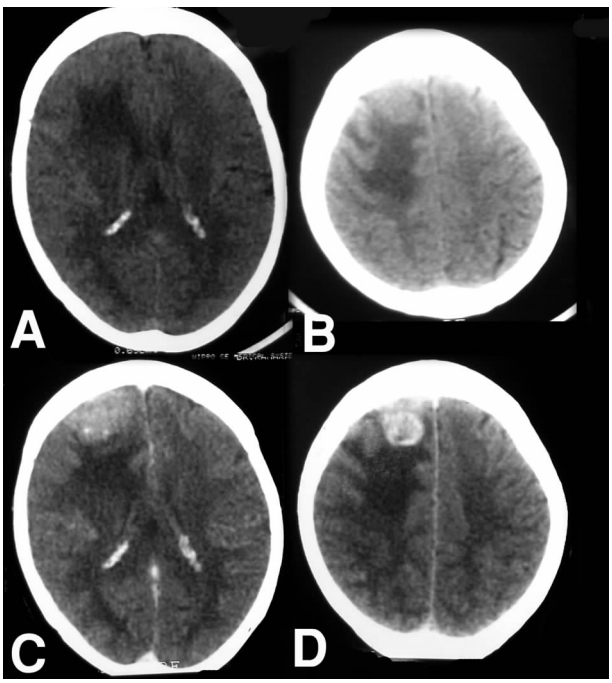

Figure 4 Plain CT scan (A and B) showing an isodense lesion enhancing after contrast administration (C and D) with an enhancing ring and a small central lucent area (D), note the associated perileiosnal oedema and mass effect

antituberculous treatment[1,8,15,16]. In many patients there may not be past history of tuberculosis (as in our both the cases) and both chest X-ray and erythrocyte sedimentation rate may be within normal limits[7,17] and the diagnosis of tuberculoma is possible only at the time of surgical excision (our case 1)[7,9]. Though tuberculosis involving the central nervous system is rarely observed in non immunocompromised hosts[18] m our both the patients did not evidence of immunocompromised state. The radiological investigation of choice for intracranial tuber-

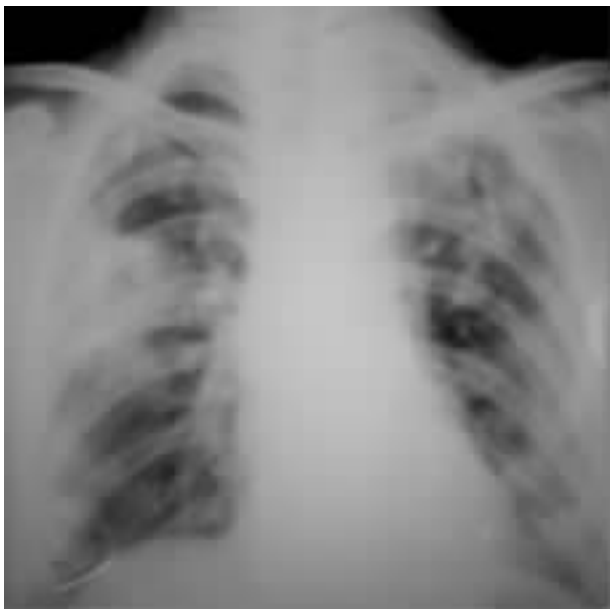

Figure 5 Chest X-rays showing widespread nodularity and infiltrates suggestive of military tuberculosis

culomas is contrast enhanced CT scan[7,19,20] and an adequate correlation between histopathological and tomographic findings of tuberculomas is documented[20,21]. Tuberculomas may be isodense or slightly hyperdense on plain scans and after contrast administration thaey may show different pattern of enhancement i.e. solid enhancing lesions, ring enhancing lesions, and mixed or combined forms of lesions[22]. Mature tuberculomas are well delineated round or oval ring enhancing masses[23], however sometimes, they appear as a ring enhancing lesion with a central area of enhancement or calcification ("target sign")[23]. The noncaseating granulomas are rounded or oval, multiple, isodense or lightly hypodense lesions, with nodular contrast enhancement[16]. Deep seated tuberculoma lesions may show multilocular ring-like enhancement with massive brain edema and can mimic high grade brain tumors (as in our 1st case), however surgical as well as histological findings will confirm the diagnosis[6,24]. The sensitivity of CT in diagnosing intracranial tuberculoma is 100% and its specificity is 86%, the positive predictive value has been reported to be as low as 33%[17,25]. Magnetic resonance imaging is shown slightly superior to CT scan in demonstrating the extent of lesions, especially in brainstem tuberculoma[20,26]. On imaging cerebral tuberculoma may be confused with many granulomatous[27,28] as well as neoplastic lesions[29-32] particularly when there are

no systemic manifestations of tuberculosis. However, based on clinical evidence and supportive investigations a diagnosis of tuberculoma can be suspected[20]. Almost all the intracranial tuberculoma patients can be managed with anti-tuberculosis short-course chemotherapy with excellent results[19]. Some studies recommend empirical anti-tubercular therapy, without the need for histological confirmation[33], while others recommend a definitive diagnosis is to be made before starting chemotherapy[7,10,33,34]. The indications for surgery include a tuberculoma with mass effect on the brain leading to raised intracranial pressure, non-response to chemotherapy[19,25,29] or when there is doubt regarding the diagnosis particularly in deep seated lesions stereotactic biopsy is recommended[34-36]. Paradoxical enlargement or development of new tuberculomas during antitubercular therapy is a documented phenomenon which can be overcome by continuation of antitubercular treatment[17,18,37,38]. and supplemented with systemic dexamethasone as adjuvant therapy for 4 to 8 weeks. 18,38 Early diagnosis, prompt and regular anti-tubercular treatment, and the clinical stage at which the patient presents are important and deciding factors for final outcome[1]. In summary these two cases represent the spectrum of variations of clinical and radiological features of tuberculoma and highlight the controversies in the approach to diagnosis and management.

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