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Tuberculous brain abscess—Case report

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ABSTRACT

In spite of recent advances in understanding of disease, tuberculosis still remains a major health problem, particularly in developing countries. Central nervous system tuberculosis may present as commonly encountered tuberculous meningitis or tuberculous mass lesions and rare tuberculous brain abscess (TBA). We report a case of tuberculous brain abscess in a patient of chronic liver disease with pulmonary hypertension and HCV infection. A 48 years old male presented with headache and abnormal behavior. There was no history of fever, vomiting, loss of consciousness, seizures, trauma and loss of weight and appetite. On examination patient was conscious but confused. No sensory– motor deficit was revealed on neurological examination. Chest x ray showed no abnormality. Mantoux test was positive. Magnetic resonance imaging of brain showed large, well defined marginally enhancing focal mass lesion in left frontal lobe. Evacuation of brain abscess done and frank creamy pus was aspirated and was sent for gram staining, Ziehl Neelsen staining, fungal smear and culture for both pyogenic and *Mycobacterium tuberculosis*. Gram staining revealed no microorganisms. No growth of pyogenic organisms obtained. No fungal hypha was seen. Ziehl Neelsen staining was positive for acid fast bacilli and growth of *Mycobacterium tuberculosis* was obtained. Patient was put on anti tubercular treatment. Patient responded well and discharged in satisfactory condition.

1. Introduction

In spite of recent advances in understanding of disease, tuberculosis still remains a major health problem, particularly in developing countries^[1]. Central nervous system tuberculosis may present as commonly encountered tuberculous meningitis or tuberculous mass lesions and rare tuberculous brain abscess (TBA). TBA are rarely encountered even in countries where CNS tuberculosis is relatively common^[2,3]. TBA was first described by Evans and Rand (early 1930s), and is still very rare in world literature^[2]. We report a case of tuberculous brain abscess in a patient of chronic liver disease with pulmonary hypertension and HCV infection

2. Case Report

A 48 years old male presented with history of headache of 15 days duration and abnormal behaviour. There was no history of fever, vomiting, loss of consciousness, seizures, trauma and loss of weight and appetite. Patient was on treatment for chronic liver disease with pulmonary hypertension. On examination patient was conscious but

confused. Patient has pulse rate of 64/min, respiratory rate 16/min and blood pressure of 136/90 mm of Hg. No sensory– motor deficit was revealed on neurological examination. Rest of physical examination was unremarkable.

Haemogram was within normal limits. Peripheral blood smear showed leucocytosis. Blood sugar was elevated. Renal function tests were within normal limits.

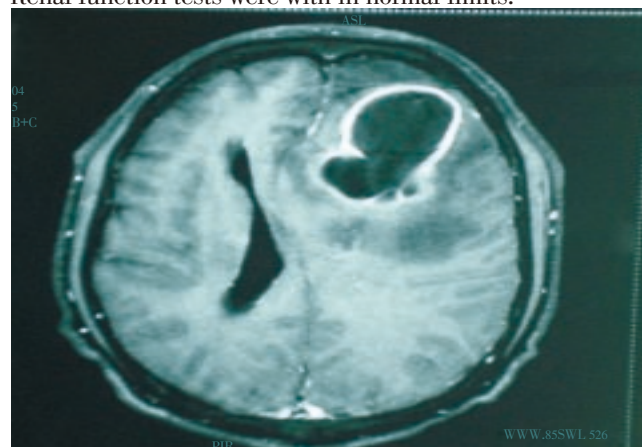


Figure 1. MRI brain showing large well defined lobulated focal mass lesion in left frontal lobe with mid–line shift.

Chest X ray showed no abnormality. Ultrasound abdomen revealed fatty liver and splenomegaly. Patient serum was tested negative for HBs Ag and Anti HIV antibodies but positive for Anti HCV antibodies. Mantoux test was positive

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with 15 mm induration. Magnetic resonance imaging (MRI) of brain showed large, well defined lobulated marginally enhancing focal mass lesion (56 mm×33 mm×40 mm) in left frontal lobe with moderate perilesional oedema causing mid line shift of 14 mm towards right suggestive of cerebral abscess (Figure–1). Treatment with injection metrogyl, mannitol, dilantin, lasix, amikacin, linezolid and insulin was started

Left frontal craniotomy was performed under aseptic precautions and evacuation of brain abscess done. Dramatic improvement was seen in patient's condition following evacuation and follow up MRI confirmed reduction in size of lesion.

Frank creamy pus was aspirated and was sent for gram staining, Ziehl Neelsen staining, fungal smear, culture for both pyogenic and *Mycobacterium tuberculosis*. Gram staining revealed no microorganisms. No growth of pyogenic organisms obtained. No fungal hyphae was seen. Ziehl Neelsen staining was positive for acid fast bacilli (Figure 2) and growth of *Mycobacterium tuberculosis* was obtained on Lowenstein Jensen medium. HPE of brain biopsy was consistent with tuberculosis.

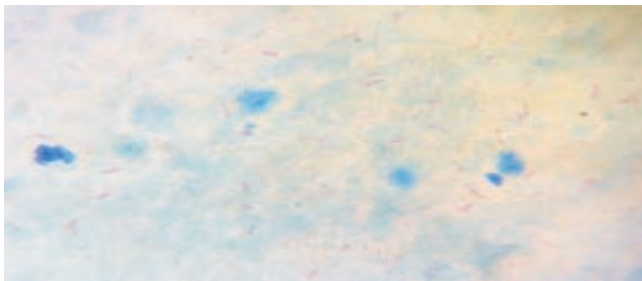


Figure 2. ZN staining of pus showing many AFB.

Patient was put on anti tubercular treatment ATT. Patient responded well and discharged in satisfactory condition with advice to continue ATT.

3. Discussion

The commonest forms of CNS tuberculosis are meningitis and tuberculomas. Tuberculous brain abscess is extremely rare^[3]. In a review of world literature by Whitner *et al* only 57 published cases were found out of which only 16 met rigid diagnostic criteria^[4]. Involvement of CNS in tuberculosis occurs more frequently in HIV positive patients (20%) than in general population (4–8%)^[5,6].

CNS tuberculosis occurs secondary to haematogenous spread of *Mycobacterium tuberculosis* from pulmonary koch's. Tubercle bacilli are immobilized in end arteries, which lead to formation of sub meningeal tubercle foci. This evokes a secondary immune reaction, which leads to formation of thick capsule with surrounding brain oedema and gliosis. In rare instances there may be secondary caseation, liquefaction and formation of an abscess. TBA occurs mostly in patients with abnormal cell mediated immunity^[7,8]. These lesions are devoid of granulomatous reaction characteristic of tuberculosis. Criteria for diagnosis are presence of pus within the brain and proof of tubercular origin demonstrated either by presence of acid fast bacilli on staining or culture or PCR^[9,10]. TBA unique in teeming with tubercle bacilli results from liquefaction of caseous center of a tuberculoma and may present as a single or multiple mass lesion with or without concomitant meningitis, with

more toxic features like headache, fever, delirium and focal neurological signs. Neurological drainage is usually required as drug treatment is seldom sufficient^[2,4]. Whitner found that TBA usually present acutely, often in the third and fourth decades, commonly have a supratentorial location with focal neurological signs, and are associated with historical and laboratory evidence of tuberculosis^[4]. Clinically diagnosis of TBA is difficult as it simulates pyogenic abscess, radiological appearance and failure to respond to treatment arouse suspicion. However gold standard of diagnosis remains on demonstration of innumerable tubercle bacilli in aspirated pus^[10].

A history of pulmonary tuberculosis may be present but in our case we could not demonstrate any extracerebral clinical manifestation of tuberculosis. In our study acid fast staining for mycobacteria on aspirated pus showed innumerable acid fast bacilli and culture proved *M. tuberculosis* similar to earlier reports^[3,10,11]. Chattopadhyay *et al*^[2] demonstrated acid fast bacilli on staining but in contrast to Kaushik *et al*^[9] where both staining and culture were negative but etiology confirmed by PCR for *Mycobacterium tuberculosis*. To conclude we highlight an unusual presentation of CNS tuberculosis presenting as brain abscess and diagnosis was confirmed by presence of acid fast bacilli on staining and growth of *Mycobacterium tuberculosis*.

Conflict of interest statement

We declare that we have no conflict of interest.

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