**INTRODUCTION**

A 24 year old man of Pakistani origin was admitted following a tonic-clonic seizure. He described altered sensation in his left hand over the previous 3 days. After recovering from the seizure, the only neurological abnormality was decreased sensation in his left hand. His medical history was notable for fully sensitive pulmonary tuberculosis diagnosed 9 years earlier; he completed only 2 months of treatment before moving abroad.

A CT scan of the brain showed mild ventriculomegaly with some sulcal prominence (Figure 1), and a chest radiograph revealed a widened upper mediastinum (Figure 2). He had further seizures and developed weakness in his left arm. Ceftriaxone and metronidazole were started empirically and phenytoin was commenced due to recurrent seizures. He had a low grade fever. MRI brain showed mild ventriculomegaly with some sulcal prominence (Figure 1), and CT thorax developed weakness in his left arm. Ceftriaxone and metronidazole were started empirically and phenytoin was commenced due to recurrent seizures. He had a low grade fever. MRI brain showed mild ventriculomegaly with some sulcal prominence (Figure 1), and CT thorax showed central necrosis from which both M. tuberculosis and streptococci were isolated, and M. tuberculosis and streptococci were isolated, and the history of partially treated pulmonary tuberculosis made CNS tuberculosis a distinct possibility in this case.

There was diagnostic uncertainty – radiological appearances could be consistent with either pyogenic or tuberculous abscess. Given the history of partially treated tuberculosis, empirical anti-tuberculous treatment was commenced, including dexamethasone: ceftriaxone was continued. Bronchoscopy for BAL and lymph node biopsy were requested but could not be performed in a timely manner. The cerebral abscess was amenable to biopsy, however it was decided after discussion with neurosurgery to sample the mediastinal lymph nodes initially as this represented a lower morbidity risk for isolating tubercle bacilli. This was done thoracoscopically (L2 and L4 paratracheal nodes) and caseous material was described. The lymph node and BAL sample were smear negative for AFB.

Three days later he developed a left facial palsy and worsening left sided weakness. Repeat MRI showed an enlarging abscess with significant oedema (Figures 5 and 6). The dexamethasone dose was increased and the patient was transferred to neurosurgery. Following aspiration of 20ml pus he improved significantly.

**IMAGING AND HISTOLOGY**

![Image 1. Day 1: CT head (non-contrast). Some sulcal prominence.](image1)

![Image 2. XR chest. Widened mediastinum.](image2)

![Image 3. Day 3: T1-weighted MRI head. Post-contrast ring-enhancement.](image3)

![Image 4. CT thorax (non-contrast): Calcified subcarinal node.](image4)

![Image 5](image5)

![Image 6](image6)

![Image 7a](image7a)

![Image 7b](image7b)

![Image 8](image8)

**RESULTS AND OUTCOME**

Lymph node histology (Figures 7a and 7b) showed central necrosis, numerous large granulomas and multinucleate giant cells, with sparse acid and alcohol fast bacilli seen on the Ziel-H-Nelsen stain (not shown). A fully sensitive M. tuberculosis was cultured from the lymph node biopsy and from bronchoalveolar lavage. Aspiration of the brain abscess was culture negative. On 16S PCR a streptococcal species was detected with 99% sequence homology to S. intermedius, S. milleri (previously strep miller group) which are well recognised causes of CNS abscesses involving the brain.1 TB PCR and culture were negative.

The patient completed 6 weeks ceftriaxone post aspiration and is making a good recovery. He is being treated for TB for 12 months.

**DISCUSSION**

Concomitant tuberculosis and pyogenic brain abscess is extremely rare and has seldom been reported in the literature; Siddiqui et al. reported two cases of brain abscesses from which both M. tuberculosis and streptococci were isolated, and Ramesh et al. reported a case of brain abscess with M. tuberculosis and staphylococcus aureus.2 The history of partially treated pulmonary tuberculosis made CNS tuberculosis a distinct possibility in this case. There was diagnostic uncertainty, clinically and radiologically, regarding the aetiology of the abscess. This case highlights the importance of keeping an open mind when treating complex infections prior to diagnostic results being available, whilst targeting common suspects with empirical therapy. It also illustrates the utility of 16S PCR in culture-negative samples.1

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**REFERENCES**

