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## 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.

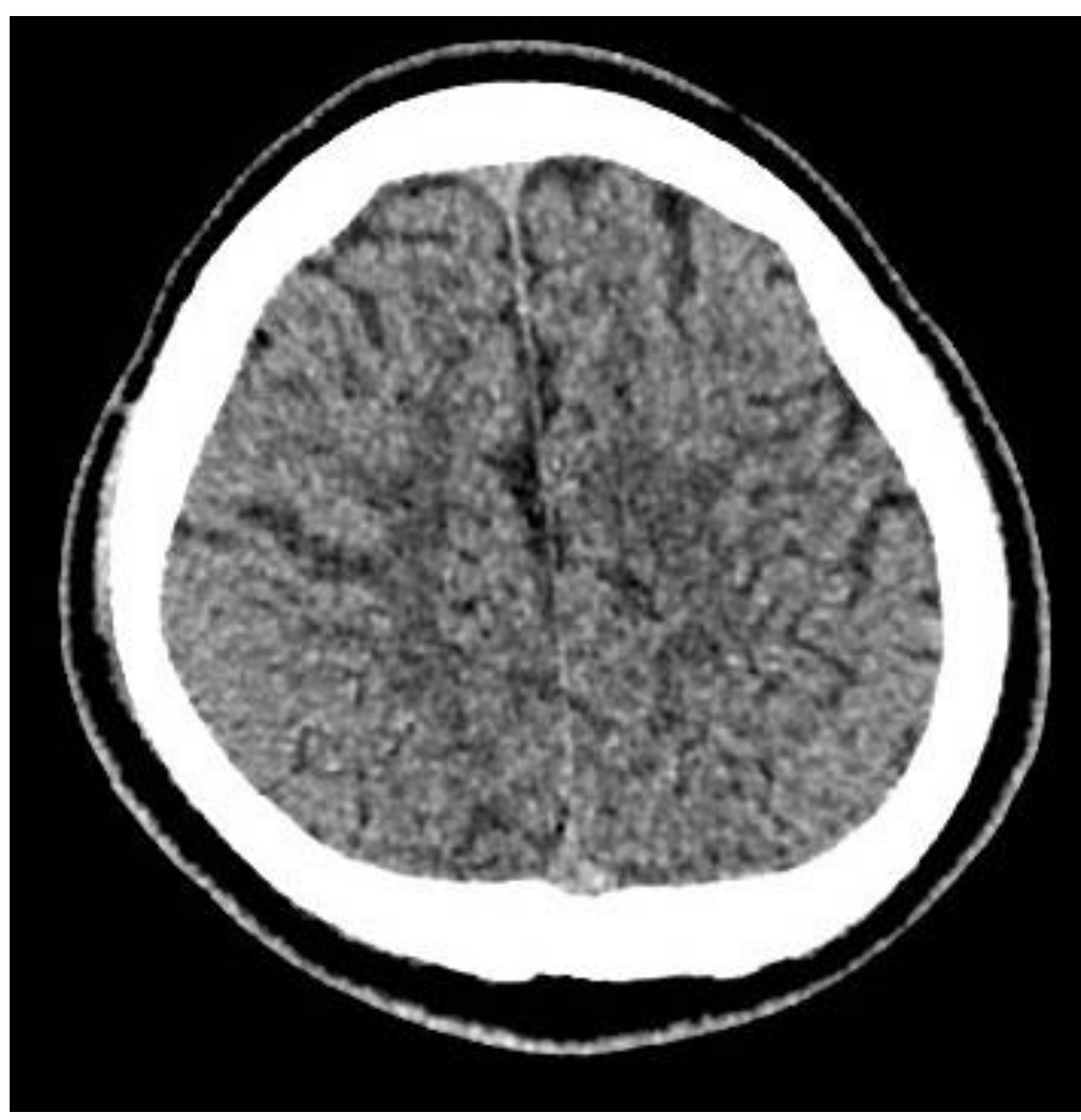
A CT scan of the brain showed mild ventriculomegaly with some sulcal prominence (Figure 1), and a chest radiograph revealed a widened upper mediastinum (Figure 2). He had further seizures and developed weakness in his left arm. Ceftriaxone and metronidazole were started empirically and phenytoin was commenced due to recurrent seizures. He had a low grade fever. MRI brain showed a 1.9 x 1.3cm space occupying lesion in the right motor cortex with ring enhancement, restricted diffusion and perilesional vasogenic oedema, consistent with an abscess (Figure 3), and CT thorax confirmed mediastinal lymphadenopathy (Figure 4).

## PROGRESS

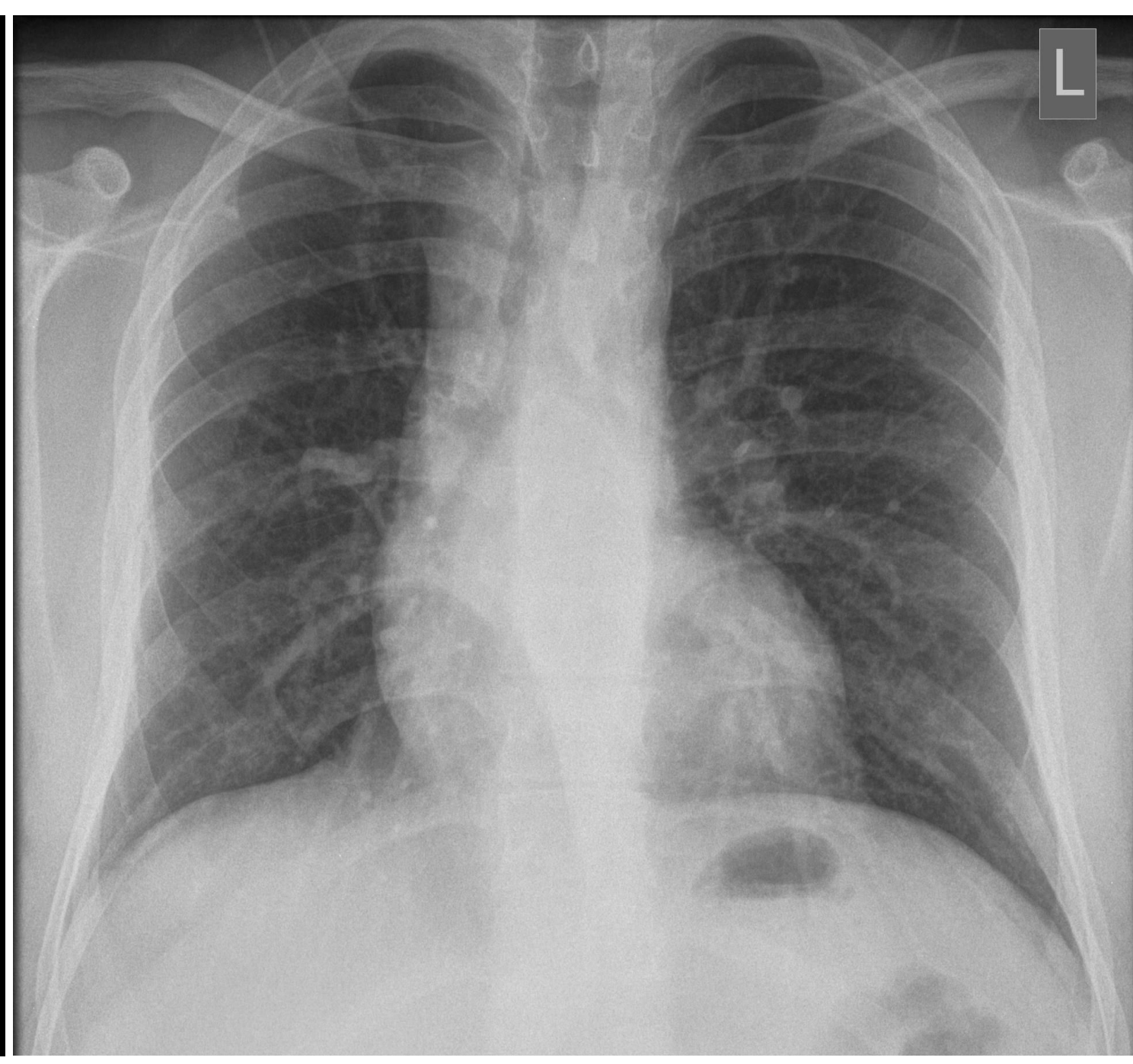
There was diagnostic uncertainty – radiological appearances could be consistent with either pyogenic or tuberculous abscess. Given the history of partially treated tuberculosis, empirical anti-tuberculous treatment was commenced, including dexamethasone; ceftriaxone was continued. Bronchoscopy for BAL 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 thoroscopically (L2 and L4 paratracheal nodes) and caseous material was described. The lymph node and BAL sample were smear negative for AAFBs.

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.

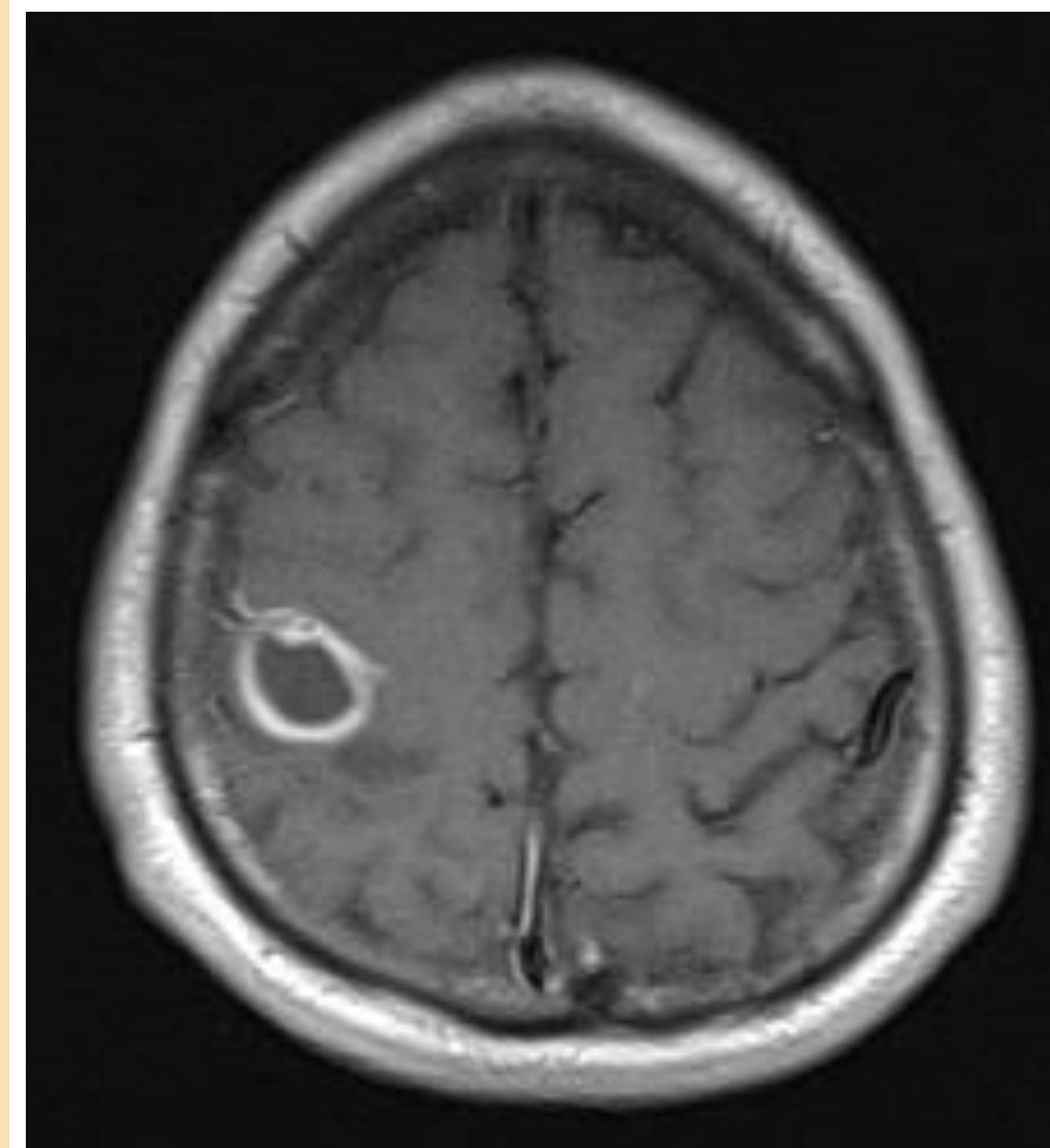
## IMAGING AND HISTOLOGY



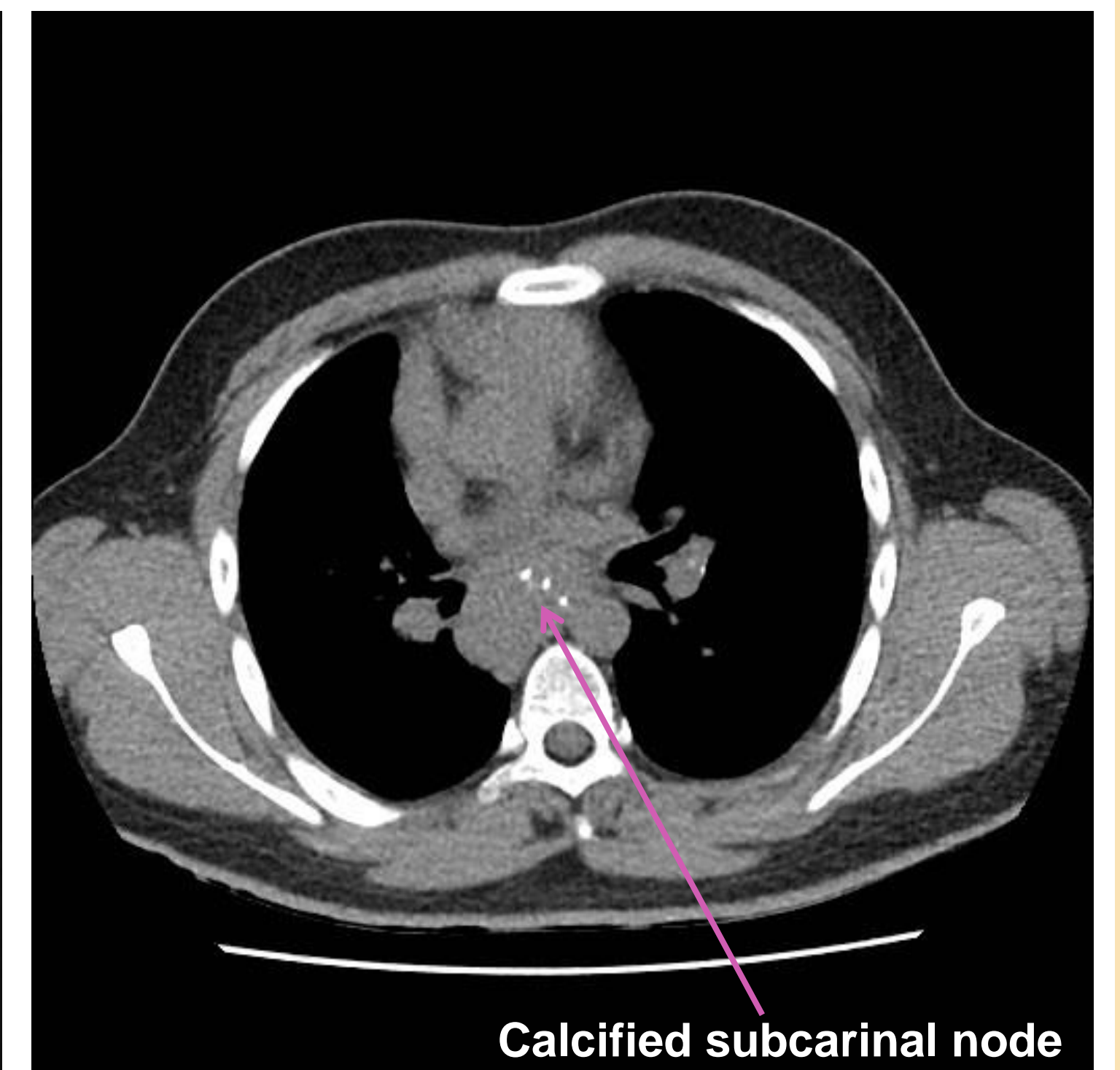
**Figure 1.** Day 1: CT head (non-contrast). Some sulcal prominence. In retrospect, some oedema is evident in the right frontal lobe



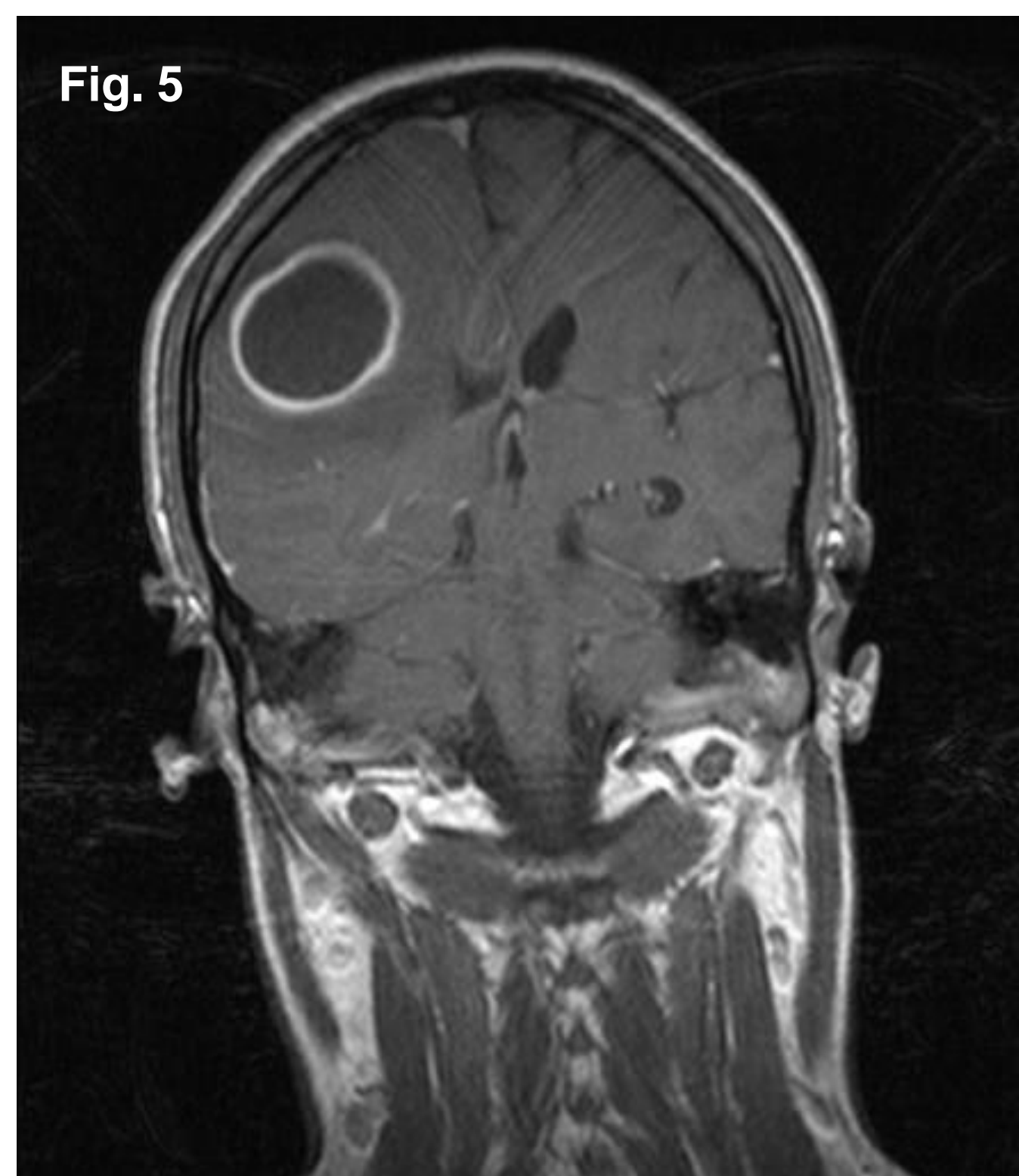
**Figure 2.** XR chest. Widened mediastinum



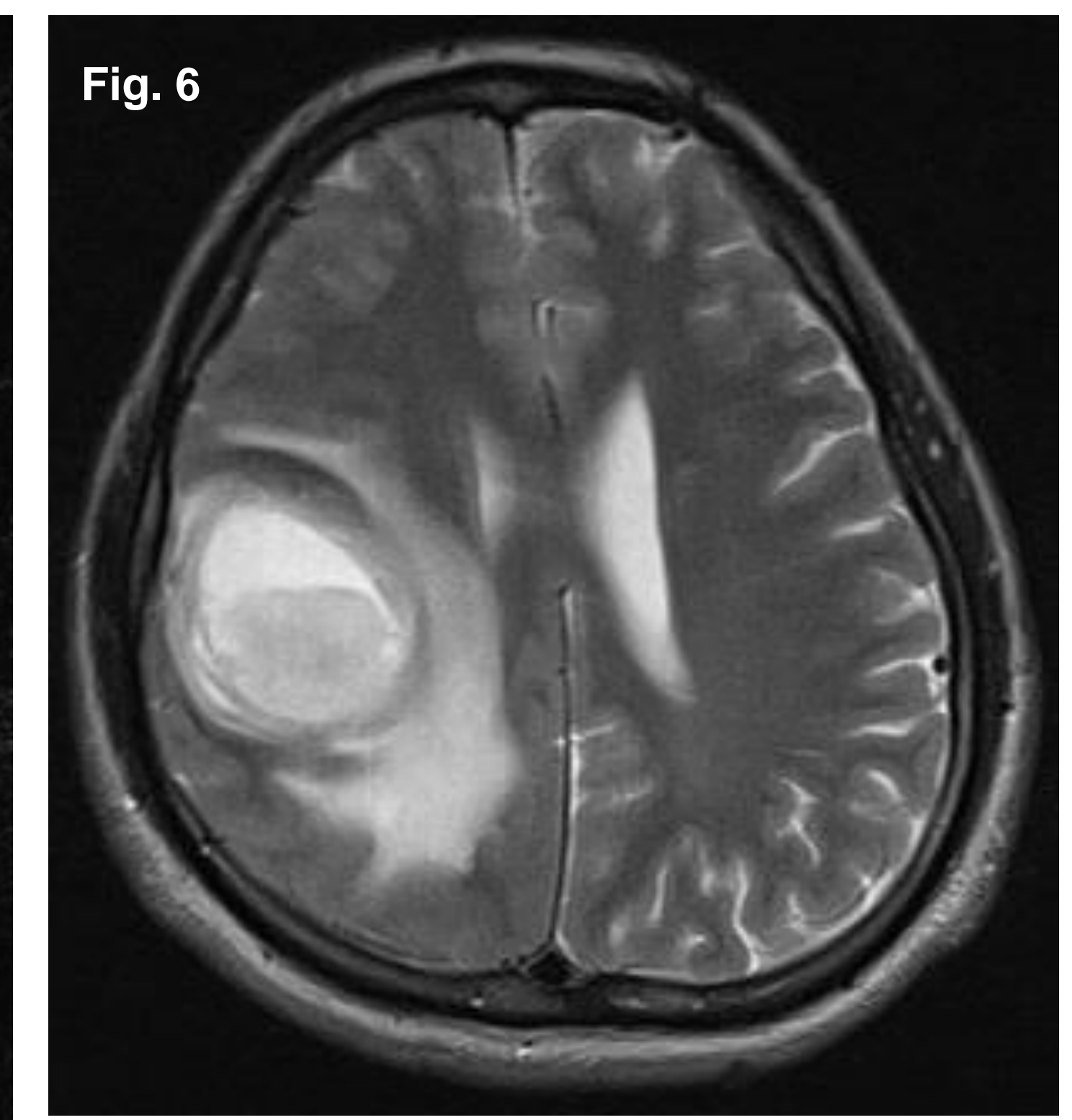
**Figure 3.** Day 3: T1-weighted MRI head. Post-contrast ring-enhancement



**Figure 4.** CT thorax (non-contrast): Calcified subcarinal node

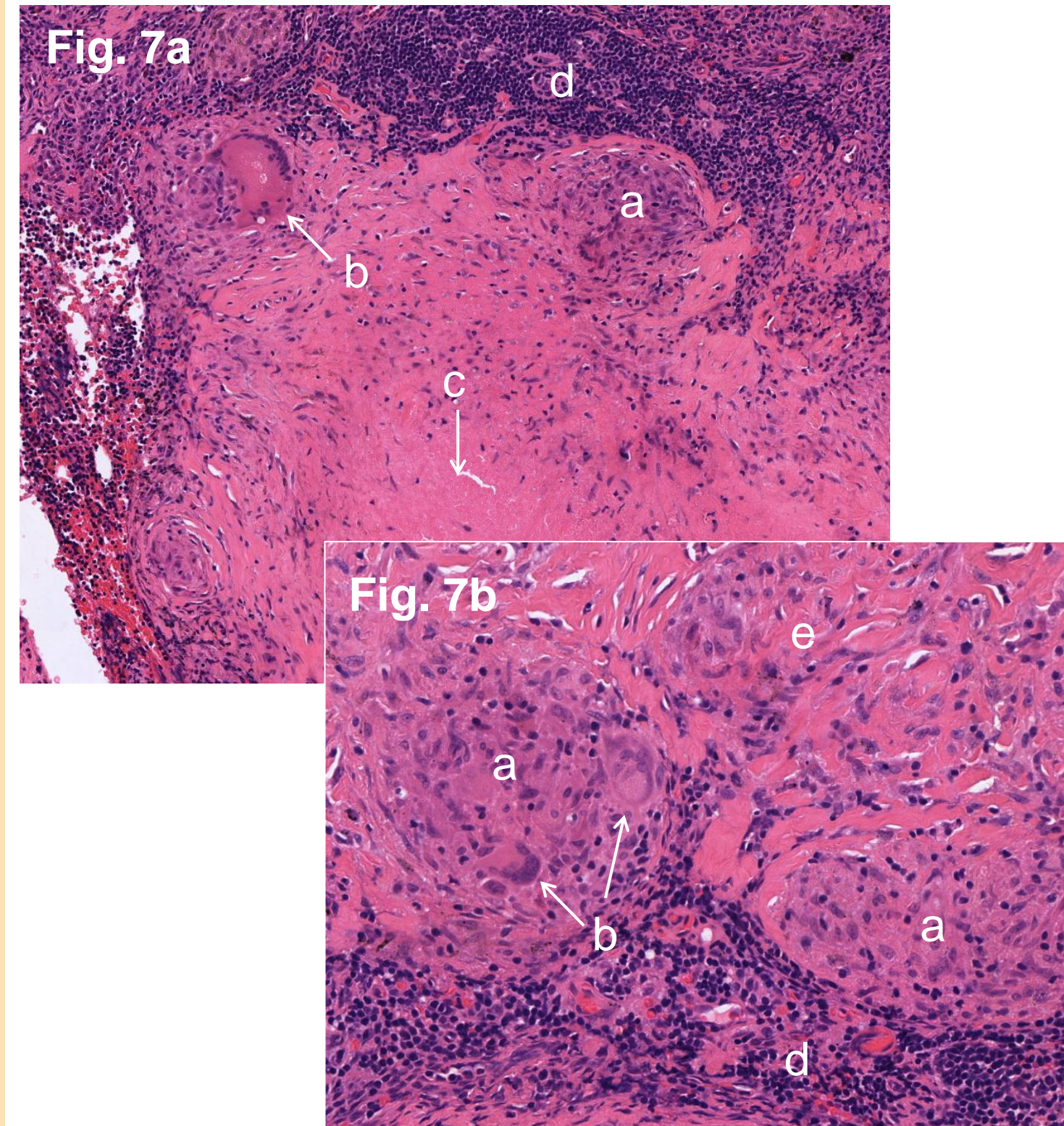


**Fig. 5**

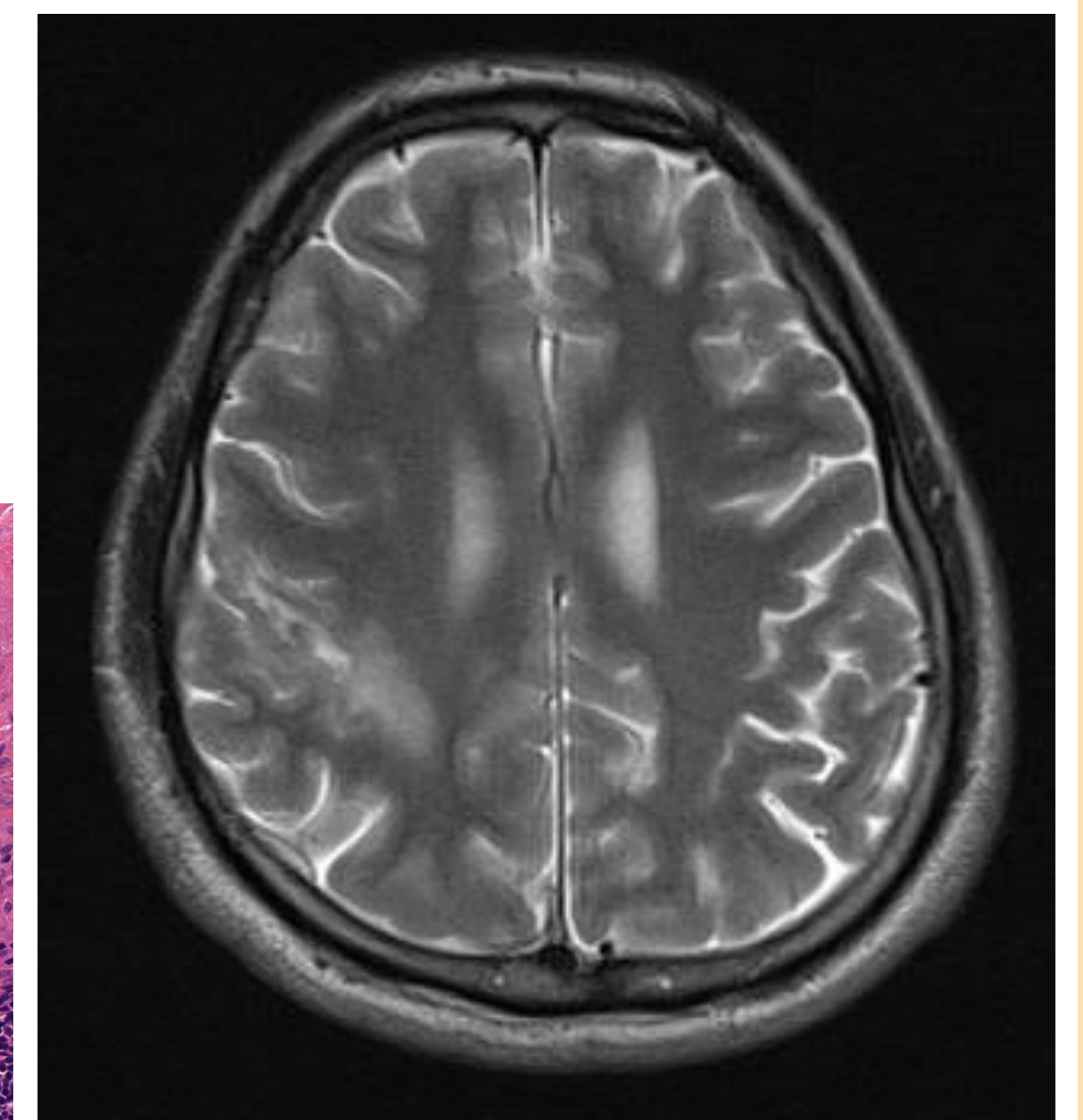


**Fig. 6**

**Figures 5 and 6.** Day 14: Sagittal and coronal T2 weighted MRI head with contrast. 3.2 x 4.4cm space-occupying lesion in right posterior frontal lobe with ring enhancement post contrast, restricted diffusion, echogenic oedema and effacement of the ipsilateral lateral ventricle



**Figure 7a and b.** Lymph node biopsy: **7a** H&E x100, **7b** H&E x200 ; granulomata (a), giant cells (b), necrosis (c), lymphocytic infiltrate (d), macrophages/fibroblasts (e)



**Figure 8.** 3 months post presentation: T2 weighted MRI head. Residual local inflammation.

## 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 Ziehl-Neelsen stain (not shown). A fully sensitive *M. tuberculosis* was cultured from the lymph node biopsy and from bronchoalveolar lavage.

Aspirate from brain abscess was culture negative. On 16S PCR a streptococcal species was detected with 99% sequence homology to *S. intermedius*, *S. anginosus* and *S. constellatus* (previously *strep milleri* group) which are well-recognised causes of CNS abscesses involving the brain.<sup>1</sup> 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, the longer duration chosen to cover the remote possibility of co-existent cerebral tuberculosis. Follow up imaging shows localised residual inflammation at the site of the previous abscess (Figure 8).

## 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,<sup>2</sup> and Ramesh et al. reported a case of brain abscess with *M. tuberculosis* and *staphylococcus aureus*.<sup>3</sup> 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.<sup>1</sup>

## ACKNOWLEDGEMENTS

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Neurosurgical colleagues at Queen Elizabeth Hospital, Birmingham.

## REFERENCES

1. Petti CA, Simmon KE, Bender J et al. Culture-negative intracerebral abscesses in children and adolescents from *Streptococcus anginosus* group infection: a case series. Clin Infect Dis 2008; **46**(10): 1578
2. Siddiqui AA, Sarwari AR, Chishti KN. Concomitant tuberculous and pyogenic tuberculous brain abscess. Int J Tuber Lung Dis 2001; **5**: 100-1
3. Ramesh V, Sundar K. Concomitant tuberculous and pyogenic cerebellar abscess in patient with pulmonary tuberculosis. Neurol India 2008; **56**: 91-2