

A CASE OF CORPUS CALLOSUM LESION ASSOCIATED WITH DISSEMINATED MYCOBACTERIUM TUBERCULOSIS INFECTION

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ABSTRACT

Isolated and transient splenial lesion in the corpus callosum has been reported in patients infected with influenza, rotaviruses, O-157 *Escherichia coli*, *Salmonella*, *Staphylococcus aureus* and *Legionnaires* infection. Here, is the first reported case of tuberculous meningitis presenting with an isolated splenial lesion in the corpus callosum. A 31-year old lady presented with a 2-week history of fever, chills, rigors and headache. Magnetic resonance imaging (MRI) revealed a lesion at the splenium of the corpus callosum. Her cerebrospinal fluid showed elevated protein, and, low glucose concentration with mononuclear pleocytosis suggestive of tuberculous meningitis. She was commenced on Isoniazid, Rifampicin, Ethambutol, Pyrazinamide and Dexamethasone for treatment of tuberculous meningitis. A repeat MRI 9 days after commencement of anti-tuberculosis treatment showed resolution of the corpus callosum lesion. In conclusion, a splenial lesion in the corpus callosum can be caused by tuberculous meningitis. This lesion is transient and can resolve with treatment of tuberculous meningitis.

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KEY WORDS

Tuberculous meningitis; Splenium of corpus callosum; *Mycobacteria tuberculosis*, MRI, Fever, Lumbar puncture.

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[I] INTRODUCTION

Isolated and transient splenial lesion in the corpus callosum was initially reported in epileptic patients. It occurs more frequently in those who had a sudden withdrawal of anti-epileptic drugs [1]. Since then, transient splenial lesion in the corpus callosum has been reported to be associated with influenza, rotaviruses, O-157 *Escherichia coli*, *Salmonella*, *Staphylococcus aureus* and *Legionnaires* infection [1]. Furthermore, transient splenial lesion in the corpus callosum has also been reported in people suffering from high altitude cerebral edema [1].

To my knowledge, this is the first reported case of *Mycobacteria tuberculosis* infection of the meninges presenting with an isolated splenial lesion in the corpus callosum.

[II] CASE REPORT

A 31-year old lady presented with a 2-week history of fever, chills, rigors and right lower quadrant abdominal pain. Physical examination revealed tenderness over the right iliac fossa with no rebound or rigidity. There was no evidence of pelvic inflammatory disease on clinical examination.

On preliminary investigation, her complete blood picture, erythrocyte sedimentation rate, C Reactive Protein, renal biochemistry and liver biochemistry were all normal. Septic work-up, computerized tomography of the whole abdomen, and echocardiogram were all unremarkable as well.

Her fever persisted despite empirical treatment with intravenous Meropenem, intravenous Amikacin and intravenous Azithromycin. On the fourth day after admission, she developed persistent headache over the bilateral occipital region. A magnetic resonance imaging (MRI) of the brain showed a focal homogenous T1W hypointense [Figure- 1A and Figure-1B], T2W/FLAIR hyperintense signal change at the splenium of the corpus callosum.

This lesion measured 2.7 cm across, 1.1 cm in anteroposterior dimension and 2.2 cm in craniocaudal dimension. It showed a bright signal change on diffusion weighted images sequence [Figure-1C] with corresponding dark signal change on apparent diffusion coefficients mapping images [Figure- 1D] that was consistent with focal area of restricted diffusion. There was only minimal mass effect. However, no corresponding contrast enhancement was detected.

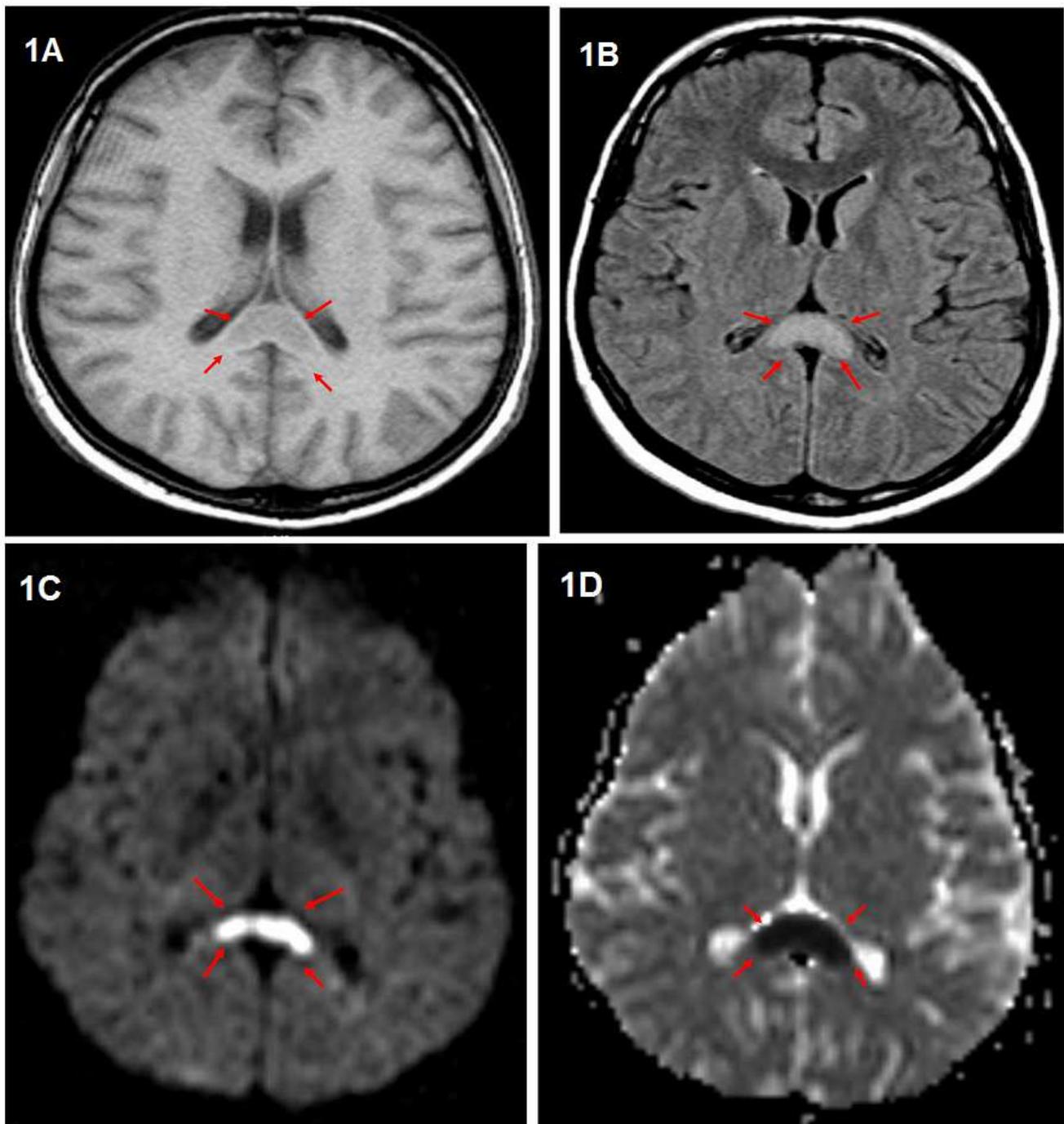


Fig. 1A: Magnetic Resonance Imaging showing focal homogenous T1W axial hypointense signal change at the splenium of the corpus callosum with symmetrical involvement. **Fig. 1B:** Magnetic Resonance Imaging showing T2W/FLAIR hyperintense signal change at the splenium of the corpus callosum. **Fig. 1C:** Magnetic Resonance Imaging showing bright signal change on diffusion weighted images (DWI) sequence. **Fig. 1D:** Magnetic Resonance Imaging showing corresponding dark signal change on apparent diffusion coefficients (ADC) mapping.

In view of the MRI finding, a lumbar puncture was performed. The cerebrospinal fluid (CSF) was clear and colorless. There were 124/ul white cells (normal range < 8) in the CSF and 99% of these white cells consisted of mononuclear cells. The CSF glucose, protein and lactate dehydrogenase (LDH) were 1.9 mmol/L (normal range 2.2-3.9), 230 mg/dl (normal range 15-45) and 34 IU/L (normal range 0-25), respectively. The corresponding spot serum glucose, protein and LDH were 9.8 mmol/L, 760 mg/dl and 148 IU/L, respectively. Only 2 red cells (normal range < 5) were detected in the CSF.

CSF for bacteria culture, acid fast bacilli smear, *Mycobacteria tuberculosis* polymerase chain reaction (PCR), cryptococcal antigen, Indian ink, fungal culture and viral titer were all negative. Nasopharyngeal aspirate for influenza A and B were also negative.

Although, the lady's CSF was negative for AFB smear and *Mycobacteria tuberculosis* PCR, the elevated protein, low glucose concentration with a mononuclear pleocytosis in her CSF was suggestive of tuberculous meningitis. She was started on Isoniazid, Rifampicin, Ethambutol and Pyrazinamide, along with Dexamethasone for treatment of tuberculous meningitis. A colonoscopy was performed on her 1-week after commencement of anti-tuberculosis treatment because of the persistent right lower quadrant abdominal pain. The colonoscopy showed multiple ulcers in the cecum and terminal ileum. Non-casating granulomas were seen on biopsy specimens obtained from the cecum. However, a Ziehl-Nielsen stain for AFB was negative.

An MRI of the brain was repeated 9 days after commencement of anti-tuberculosis treatment. This showed reduced swelling in the splenium of the corpus callosum. There was also less marked increase in T2W and diffusion signal intensity. Six weeks later, *Mycobacteria tuberculosis* was cultured from the stool and CSF.

[III] DISCUSSION

Mycobacteria tuberculosis involving the central nervous system would usually present as meningitis, tuberculoma or spinal tuberculous arachnoiditis. In the United States, they account for 1% of all cases of *Mycobacteria tuberculosis* and 6% of extrapulmonary *Mycobacteria tuberculosis* infection [2]. In one case series, 29% of patients with tuberculous meningitis had hydrocephalus on presentation [3]. The typical radiological features in tuberculous meningitis include basilar meningeal enhancement with hydrocephalus, cerebral edema with infarction [4-7].

The corpus callosum connects the left and right cerebral hemispheres. It enables the exchange of information between these 2 hemispheres. The exact pathogenesis of the splenial lesion in the corpus callosum in this case of tuberculous meningitis is uncertain. The reversible lesion in the splenium of

the corpus callosum is postulated to be caused by an increase in cytokines such as interleukin-6 and interleukin-10, and, intramyelinic edema [8].

Previous studies have shown that tuberculous meningitis is associated with an increase in the cytokine levels in the blood and CSF [8-10]. This would result in vasodilatation, and, vasogenic intramyelinic edema, resulting in the formation of splenial lesion in the corpus callosum.

These raised inflammatory cytokines will decrease following treatment of the tuberculous meningitis [9, 10], resulting in the resolution of the intramyelinic edema. This explains why the lesion in the splenium of the corpus callosum in tuberculous meningitis is only transient as seen in this patient.

[IV] CONCLUSION

In conclusion, this is the first reported case of tuberculous meningitis presenting with a splenial lesion in the corpus callosum. This lesion in the corpus callosum is transient and will resolve with treatment for tuberculous meningitis.

FINANCIAL DISCLOSURE

None

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None

CONFLICT OF INTERESTS

None

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