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Medical Imagery

‘Mini-boomerang sign’: reversible corpus callosal signal change in neuroleptospirosis

A 30-year-old male presented with a high-grade fever of 7-day duration, followed by acute onset confusion and poor verbal fluency. Conjunctival hemorrhage and signs of meningeal irritation were noted. Moderate leukocytosis, thrombocytopenia, and elevated alkaline phosphatase and creatinine phosphokinase with normal renal function were found. The cerebrospinal fluid was culture-negative. The serum anti-Leptospira IgM antibody level was 87 U/ml (cut-off <15 U/ml). Magnetic resonance imaging (MRI) of the brain (Figure 1) showed a hyperintense area involving the central part of the splenium of the corpus callosum (SCC), with decreased apparent diffusion coefficient (ADC) values. The patient was treated successfully with intravenous ampicillin. A repeat MRI of the brain (Figure 2) at the end of 4 weeks demonstrated complete resolution of the SCC signal change with normalization of ADC values, and the patient reported no residual neurodeficits.

Transient splenial hyperintensity has been found to be associated with various demyelinating, ischemic, metabolic, infective conditions.[1,2] A reversible semilunar hyperintense lesion in the SCC, or ‘mini-boomerang sign’, has never been described in association with neuroleptospirosis.[2] Neurological involvement of *Leptospira interrogans* is not uncommon and can range from aseptic meningoencephalitis to demyelinating lesions and intracerebral

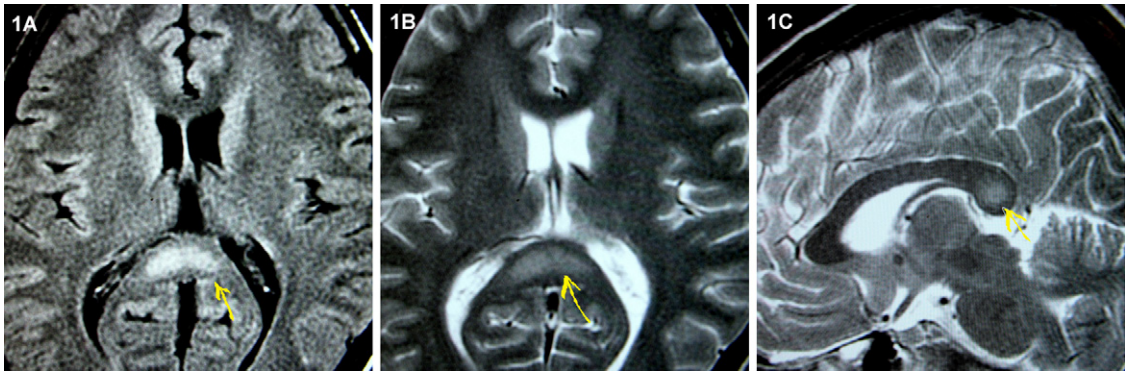

Figure 1. MRI of the brain on admission. (A) FLAIR, (B) axial T2-weighted, and (C) sagittal T2-weighted images showing a semilunar hyperintense lesion (yellow arrow) in the central portion of the splenium of the corpus callosum.

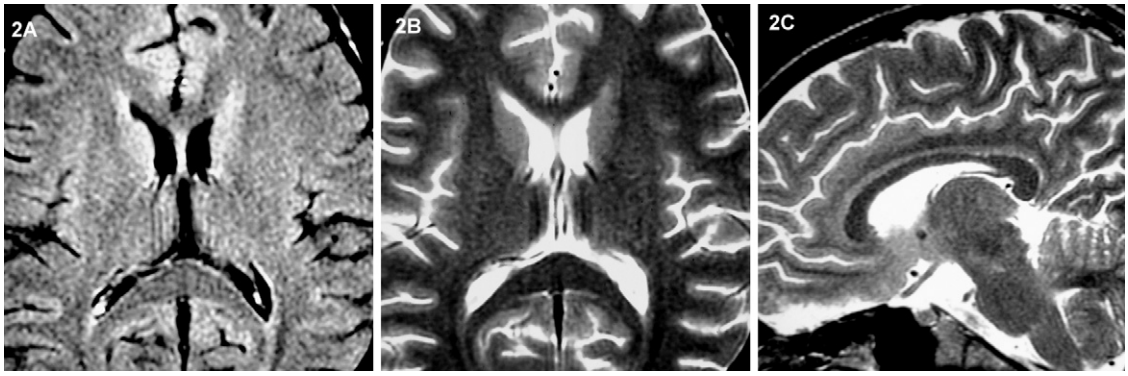

Figure 2. MRI of the brain after 4 weeks. (A) FLAIR, (B) axial T2-weighted, and (C) sagittal T2-weighted images showing complete resolution of the lesion in the central splenium of the corpus callosum.

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hemorrhage.[3,4] Possible mechanisms of SCC lesions include intramyelinic edema due to glial water uptake and influx of inflammatory cells, combined with cytotoxic edema.[5] The diagnosis of leptospirosis is often missed because of atypical presentations and the unavailability of definitive diagnostic modalities. SCC involvement in leptospirosis is benign and transient and thus aggressive diagnostic and therapeutic approaches are not indicated.

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Conflict of interest: No conflict of interest to declare.

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Dibbendhu Khanra*
Arunansu Talukdar
Department of Medicine, Medical College and Hospital, Kolkata, 88, College Street, Kolkata 700073, West Bengal, India

Corresponding Editor: Eskild Petersen, Skejby, Denmark

*Corresponding author
E-mail address: ddk3987@gmail.com (D. Khanra)