



BMH Med. J. 2026;13(2):43-46. **Case Report**

Lyme Neuroborreliosis Presenting as Acute Meningoencephalitis in a Post-Splenectomy Patient: A Diagnostic Challenge

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Abstract

Lyme disease, caused by *Borrelia burgdorferi*, is an emerging but under-recognized infection in India and can occasionally involve the central nervous system, presenting as Lyme neuroborreliosis. Its clinical manifestations often overlap with several tropical infections, making diagnosis challenging. We report a case of a young adult post-splenectomy patient who presented with acute meningoencephalitis characterized by fever, headache, neck pain, and behavioural changes. Initial investigations revealed lymphocytic meningitis, nonspecific MRI abnormalities, and isolated *Brucella* IgM seropositivity, raising suspicion for neurobrucellosis. However, the patient lacked systemic features of brucellosis, and cerebrospinal fluid cultures were negative. Subsequent testing showed *Borrelia burgdorferi* IgM positivity, and the constellation of clinical, CSF, and radiological findings strongly supported Lyme neuroborreliosis. The patient improved significantly with ceftriaxone, doxycycline, and rifampicin therapy. This case emphasizes the importance of cautious interpretation of *Brucella* serology, particularly isolated IgM, and highlights Lyme disease as an important differential diagnosis in patients presenting with subacute meningitis in endemic regions.

Keywords: Lyme neuroborreliosis; *Borrelia burgdorferi*; Meningoencephalitis; Post-splenectomy; CNS Lyme disease; Pachymeningeal enhancement

Introduction

Lyme disease, caused primarily by *Borrelia burgdorferi* and transmitted through Ixodes tick bites, is one of the most common vector-borne infections globally [1-3]. Although its incidence in India appears lower compared to Western countries, increasing case reports suggest that Lyme disease may be underdiagnosed due to overlapping clinical features with more prevalent tropical infections such as leptospirosis, brucellosis, scrub typhus, viral encephalitis, and tuberculosis [4].

Neurological involvement, termed Lyme neuroborreliosis, occurs in approximately 10-15% of untreated patients and can present with lymphocytic meningitis, cranial neuropathies, radiculoneuritis, encephalopathy, or encephalitis [3,5]. The diagnosis remains challenging due to nonspecific symptoms, variable imaging findings, and cross-reactivity in serological assays. CSF

abnormalities typically include lymphocytic pleocytosis, elevated protein, and normal to mildly low glucose. MRI may show leptomeningeal or pachymeningeal enhancement but may also be normal [6].

Post-splenectomy patients are more vulnerable to severe or atypical infections due to impaired bacterial clearance and altered immune responses [7]. This predisposition often complicates the diagnostic process.

In regions like India, where clinicians commonly consider brucellosis, tuberculosis, and viral etiologies for meningitis, Lyme disease may not be the first diagnostic consideration. Furthermore, false-positive *Brucella* IgM results are frequently reported due to cross-reactivity with other microorganisms, including *Borrelia burgdorferi* [8]. This case highlights the diagnostic complexities encountered when serological tests conflict with clinical and CSF findings.

Case Presentation

A young adult male with a history of splenectomy over a decade back following a road traffic accident presented with a one-week history of intermittent fever, severe headache, and posterior neck pain. He reported a history of a fly bite to the left eye one month earlier, followed by conjunctivitis and right eye infection, which initially improved with oral and topical medications. One week later, he developed intermittent fever, altered behaviour, vomiting, and abdominal discomfort.

At the referring hospital, blood investigations revealed leukocytosis and deranged liver function tests. He was then transferred to our centre for further neurological evaluation. On examination, he was conscious but restless, febrile (39 degrees Centigrade), and had significant neck stiffness. No focal motor deficits were observed. Fundus examination was normal, and abdominal examination showed mild epigastric tenderness.

CSF analysis revealed: WBC count: 297 cells/microliter (mixed pleocytosis with lymphocytes and monocytes), Protein: 181 mg/dL, Low glucose, Cytology: monocytes only. CSF meningoencephalitis PCR panel was negative. Wet mount for parasites was negative. Serology for Typhoid (IgG/IgM), Dengue NS1, *Leptospira*, Scrub typhus IgM, TB PCR, Adenovirus, ACE, CMV, EBV, Japanese Encephalitis, HIV, Hepatitis B and C were all negative. Peripheral smear showed neutrophilia with occasional atypical lymphocytes. He tested positive for *Brucella* IgM antibody. He was also tested positive for Lyme disease (*Borrelia burgdorferi* IgM antibody).

MRI brain revealed diffuse pachymeningeal enhancement in bilateral frontal and temporal lobes, with FLAIR hyperintensities in the left high frontal cortical sulci, suggestive of meningitis. MR venogram showed no venous sinus thrombosis. EEG and neck ultrasound were normal.

Given the lymphocytic CSF profile, lack of systemic brucellosis features, negative cultures, and positive Lyme serology, a diagnosis of Lyme neuroborreliosis was considered most likely.

The patient received parenteral ceftriaxone, doxycycline, and rifampicin. Over the next few days, his sensorium improved, fever subsided, and neck stiffness resolved. Repeat CSF study demonstrated normalization of cell count and protein. He continued to improve over 4 weeks of follow-up.

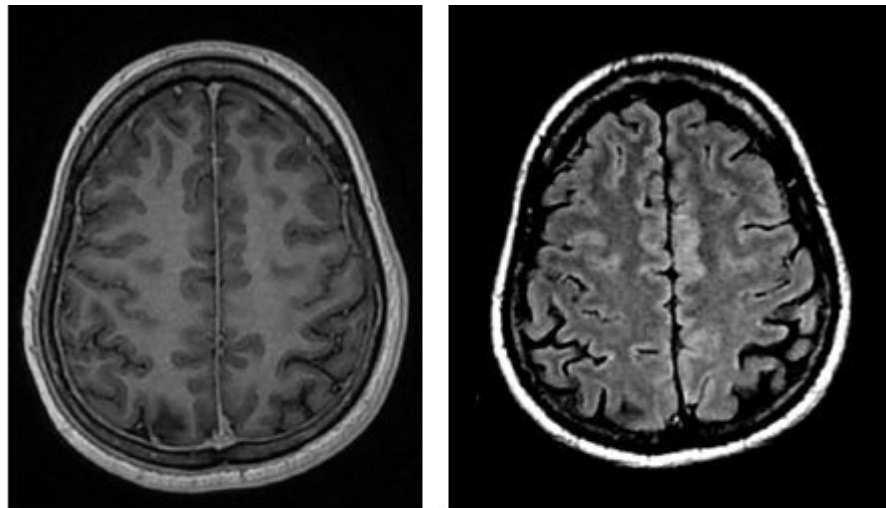


Figure 1a

Figure 1b

Figure 1:(a) Axial post-contrast T1-weighted MRI brain demonstrating diffuse pachymeningeal enhancement in the bilateral frontal and temporal regions. (b) Axial FLAIR MRI brain showing hyperintensities in the left high frontal cortical sulci, suggestive of meningeal inflammation.

Discussion

Lyme neuroborreliosis represents a significant neurological manifestation of Lyme disease, with patients commonly presenting with meningitis, meningoencephalitis, cranial neuropathies, and radiculitis [3,5]. In this patient, the subacute onset of fever, headache, neck stiffness, behavioural disturbances, and lymphocytic meningitis strongly suggested CNS involvement by *Borrelia burgdorferi*. The diagnostic process, however, was initially confounded by the presence of isolated *Brucella* IgM seropositivity, which raised suspicion for neurobrucellosis. *Brucella* IgM ELISA is notorious for its propensity to yield false-positive results due to cross-reactivity with several gram-negative organisms, including *Borrelia*, *Yersinia*, and *Escherichia coli* [8]. Furthermore, IgM antibodies can persist long after the resolution of prior infections or may be produced in a polyreactive manner, especially in individuals with altered immune function, such as those who are post-splenectomy [7]. Importantly, this patient lacked the typical systemic features of brucellosis, and there was no corroborative evidence from CSF or blood cultures. Taken together, the isolated IgM positivity was considered misleading and clinically insignificant when compared to the much stronger correlation between the patient's presentation and Lyme neuroborreliosis.

The CSF profile further supported the diagnosis of Lyme neuroborreliosis, as it demonstrated lymphocytic pleocytosis, raised protein levels and normal glucose - findings that are well recognized in CNS Lyme disease. In contrast, neurobrucellosis typically shows markedly reduced CSF glucose, granulomatous inflammation, and at times, vasculitic changes, none of which were observed in this case. Radiologically, the pachymeningeal enhancement seen on MRI was also more characteristic of Lyme disease, which is known to cause leptomenigeal or pachymeningeal enhancement and cortical or subcortical hyperintensities, although MRI findings may be normal in some patients. The diagnosis was further strengthened by the patient's rapid symptomatic improvement after initiation of ceftriaxone, and doxycycline, antimicrobials that have excellent penetration into the central nervous system and are standard therapy for neuroborreliosis [3,5].

This case also emphasizes the growing recognition of Lyme disease in India, where it has

historically been considered rare. The likelihood of underdiagnosis remains high due to overlapping clinical presentations with other endemic infections, low clinical suspicion among healthcare providers, and limited access to confirmatory testing. Increased awareness is essential to avoid delays in management, particularly in atypical or complex presentations such as this one. The patient's asplenic status added another layer of complexity by predisposing them to atypical immune responses and enhancing the risk of non-specific serological cross-reactivity, providing a plausible explanation for the misleading Brucella IgM result. Overall, the clinical presentation, laboratory findings, imaging features, therapeutic response, and immunological background collectively supported Lyme neuroborreliosis as the unifying diagnosis in this patient.

Conclusion

This case illustrates the significant diagnostic challenges associated with acute meningoencephalitis, particularly in regions where multiple infectious etiologies coexist and clinical presentations often overlap.

References

1. Stanek G, Wormser GP, Gray J, Strle F. Lyme borreliosis. *Lancet*. 2012;379(9814):461-473.
2. Mantur BG, Amarnath SK. Brucellosis in India - a review. *J Biosci*. 2008;33(4):539-547.
3. Mygland A, Ljostad U, Fingerle V, et al. EFNS guidelines on the diagnosis and management of European Lyme neuroborreliosis. *Eur J Neurol*. 2010;17(1):8-16.
4. Sharma A, et al. Emerging zoonotic infections in India. *Trop Doct*. 2018;48(2):93-98.
5. Halperin JJ. Nervous system Lyme disease. *Infect Dis Clin North Am*. 2015;29(2):241-253.
6. Hildenbrand P, et al. Lyme neuroborreliosis: manifestations of a rapidly emerging zoonosis. *AJNR Am J Neuroradiol*. 2009;30(6):1079-1087.
7. Di Sabatino A, Carsetti R, Corazza GR. Post-splenectomy and hyposplenic states. *Lancet*. 2011;378(9785):86-97.
8. Memish ZA, et al. Brucella serology: diagnostic pitfalls and cross-reactivity. *Clin Microbiol Infect*. 2015;21(8):707-712.