Case Report

The silent masquerader: Primary neuroleptospirosis mimicking pyogenic meningitis in a previously healthy young man

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ABSTRACT

Leptospirosis rarely announces itself through the nervous system alone. Most cases are self-limited febrile illnesses, or, at the severe end, Weil's disease with jaundice and renal failure; an isolated central nervous system (CNS) presentation is easy to mistake for bacterial meningitis. We describe a 32-year-old previously well man who developed status epilepticus days into a febrile illness following a bath in a village tubewell during the monsoon. His cerebrospinal fluid (CSF) showed neutrophilic pleocytosis with low glucose, indistinguishable from pyogenic meningitis, yet liver and kidney function stayed normal throughout. It was the exposure history, not the CSF report, that prompted leptospiral serology, immunoglobulin M-positive twice, 4 weeks apart. Magnetic resonance imaging showed bilateral frontal fluid-attenuated inversion recovery hyperintensities with an enhancing right ependymal nodule. He responded well to antibiotics, with near-normalization of CSF by discharge. This case is a reminder that leptospirosis can present as isolated CNS infection, and exposure history can matter more than the CSF numbers themselves.

Key words: Aseptic meningitis, Leptospirosis, Neuroleptospirosis, Status epilepticus, Zoonosis

Leptospirosis is caused by pathogenic, motile spirochetes of the genus *Leptospira*, usually picked up through contact with water or soil contaminated by infected animal urine [1]. The disease can look like almost anything: a mild flu-like illness in most people, and at its worst, the multi-organ failure of Weil's syndrome. Central nervous system (CNS) involvement, including meningitis, myocarditis, and pulmonary hemorrhage, is well recognized and can be life-threatening [2]. What makes neurological leptospirosis genuinely tricky is that it so often gets mistaken for other, more familiar causes of meningoencephalitis, which means the diagnosis depends heavily on someone asking the right question about water exposure at the right time. Recent multicenter data from an endemic setting found neuroleptospirosis accounted for roughly one in nine patients presenting with a primary CNS infection, suggesting it deserves more routine consideration than it typically receives [3].

We report a young man who presented with status epilepticus and was eventually diagnosed with neuroleptospirosis, without any of the hepatic or renal derangement usually expected with this disease.

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CASE REPORT

A 32-year-old man with no significant medical history came to us with 3 days of high-grade fever. About a week before that, he had bathed in a tubewell in his village during the rainy season. On the 4th day of his illness, he had a seizure that evolved into status epilepticus and was managed with antiepileptic drugs at another hospital before reaching us on day 15.

On examination, the patient was febrile but otherwise systemically unremarkable. Neurologically, he had neck stiffness with positive Kernig's and Brudzinski's signs, and was irritable, with clear frontal lobe features, disinhibited behavior and a noticeable change in personality.

His hemoglobin was 10.1 g/dL, and platelets were 1.1 lakh/µL. Liver enzymes were normal (bilirubin 0.8 mg/dL, serum glutamic pyruvic transaminase 38 U/L). A lumbar puncture showed an opening pressure of 25 cm H_2O, 900 cells/µL with 70% polymorphs, protein of 78 mg/dL, and glucose of 36 mg/dL against a blood glucose of 103 mg/dL, a cerebrospinal fluid (CSF) picture that looked, at first glance, like classic pyogenic meningitis. Gram stain and cultures were negative, as were India ink staining and cryptococcal antigen. We

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started him empirically on intravenous ceftriaxone, vancomycin, and dexamethasone.

It was only when we went back over his history, the tubewell, and the rainy season that leptospirosis entered the differential. Serum immunoglobulin M (IgM) came back positive, and he began improving on the same antibiotics. Magnetic resonance imaging of the brain showed bilateral frontal fluid-attenuated inversion recovery (FLAIR) hyperintensities and an enhancing nodule near the right ependyma (Figs. 1 and 2).

A repeat CSF examination after 2 weeks showed improvement; opening pressure down to 22 cm H_2O, cell count to 300/µL (90% neutrophils), protein 64 mg/dL, and glucose recovering to 65 mg/dL against a blood glucose of 98 mg/dL; alongside clinical resolution of his seizures, fever, and headache. A second IgM test at 4 weeks was still positive. He was discharged after 4 weeks in stable condition, with a third CSF sample showing just 10 cells and normal protein and glucose. He never came back for follow-up.

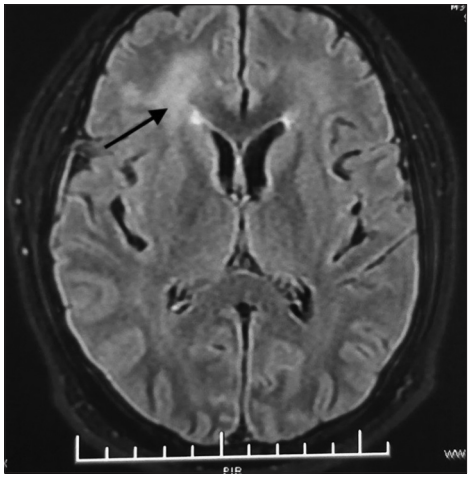

Figure 1: Axial T2/fluid-attenuated inversion recovery magnetic resonance imaging showing hyperintensity in the frontal lobe (arrow)

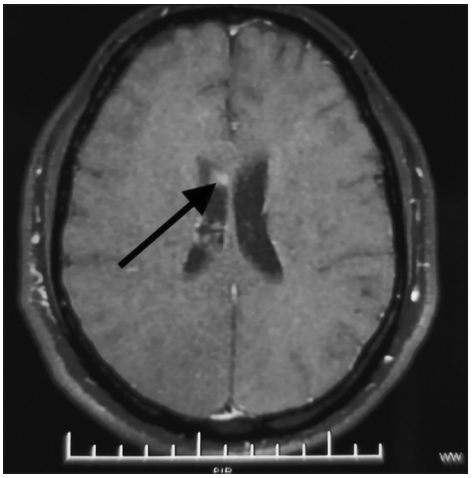

Figure 2: Post-contrast axial magnetic resonance imaging showing right ependymal nodular enhancement (arrow)

DISCUSSION

Aseptic meningitis is the neurological hallmark of leptospirosis, though it is probably underdiagnosed, since the symptoms are often mild enough to be dismissed [4]. The range of clinical presentation becomes wide: Cranial nerve palsies, Guillain-Barré-like syndromes, myelopathy, meningoencephalitis, movement disorders, dysautonomia, cerebellar involvement, mononeuritis, and even polymyositis, when the nervous system is involved, which happens in roughly 10–21% of cases [5].

The disease consists of two phases. Early, during the leptospiremic phase, the organism can sometimes be recovered from blood, urine, or CSF, often without much in the way of cytological change. Later, in the immune phase, the CSF starts to look more familiar: pleocytosis, high protein, low glucose, and positive serology; but by then the organism itself is usually gone [6,7]. Beyond meningitis, there are scattered reports of leptospirosis behind stroke-like hemiplegia, intracranial bleeds, cerebellar signs, abnormal movements, myelitis, and acute flaccid weakness, though these remain a small fraction of the aseptic CNS picture overall [8]. A presentation that is purely neurological, with no hepatic, renal, or hemorrhagic involvement at all, is genuinely uncommon [6,8].

The differential in a patient with neutrophilic CSF pleocytosis, fever, and an enhancing frontal lesion is wide, and apart from bacterial meningitis, other differentials were also considered. Tuberculous (TB) meningitis, being endemic to the country, deserves particular mention due to the subacute course; a recent analysis looked specifically at how neuroleptospirosis can be distinguished from TB meningitis, pertaining to CSF glucose and protein trends and the clinical response [9]. Herpes simplex encephalitis is also a differential in anyone with fever and behavioral change, though the bilateral, non-temporal pattern on FLAIR images in our case made that less likely; moreover, herpes simplex virus polymerase chain reaction came out to be negative. Fungal meningitis was ruled out with a negative India ink stain and negative cryptococcal antigen. Serology for scrub typhus was negative; dengue and malarial serology was not sent, as clinical history and blood workup were not pointing toward the same.

We started him on ceftriaxone and vancomycin empirically, before leptospirosis was considered, aimed at presumed pyogenic meningitis. Once the leptospiral IgM came back positive, we kept him on ceftriaxone rather than switching to penicillin. A randomized trial of 173 patients with severe leptospirosis found ceftriaxone and penicillin G performed identically, with the same fever duration and the same mortality [10], and a 2024 Cochrane review of the antibiotic trials in leptospirosis had a similar conclusion, while also noting that the overall certainty of evidence for antibiotic benefit in this disease remains very low [11]. Ceftriaxone also had the advantage of maintaining an empirical bacterial cover while a confirmed diagnosis was awaited. The trial-supported duration for severe leptospirosis is

seven days [10,11]; we continued treatment well beyond that in this patient due to his ongoing CNS symptoms and the slow pace of CSF recovery. That extension was based on our own clinical judgment. Our patient fits this uncommon pattern almost exactly since freshwater bathing in the rainy season is a classic risk factor for leptospirosis; yet his liver and kidney function tests were normal, and his CSF looked exactly like a bacterial infection. In the end, apart from the laboratory evidence, this particular history of freshwater bathing helped to reach a diagnosis.

CONCLUSION

In a patient whose CSF looks bacterial but whose cultures stay negative, a compatible water exposure history should be enough to prompt testing for leptospirosis, even when the liver and kidneys look normal.

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During preparation of this manuscript, the author used Claude (Claude Sonnet 5, Anthropic PBC), a large language model-based AI tool, for manuscript formatting to journal requirements, language and grammar editing, literature search and summarization of comparable published case reports, and drafting/formatting of reference citations. All AI-assisted output, including reference citations, was independently reviewed and cross-checked by the author against original sources for accuracy. AI was not used to collect, generate, or interpret any patient clinical data, laboratory results, or imaging findings; the clinical content and diagnosis reflect the author's own independent assessment. No images were AI-generated. The author takes full responsibility for the accuracy, integrity, and originality of this work.

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