Case Report

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Atypical Neurological Presentation in Brucellosis: A Rare Case Report

Internal Medicine Section

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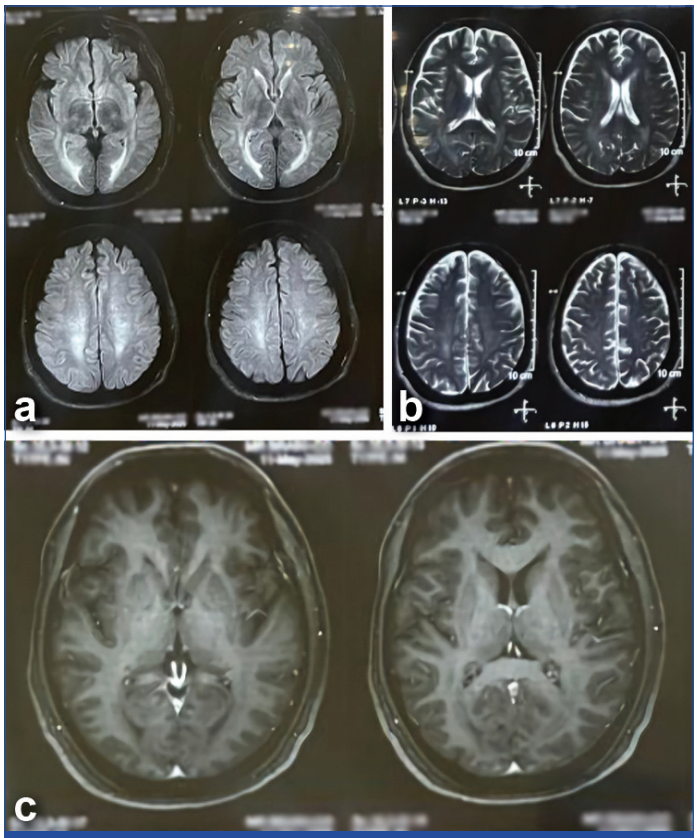
ABSTRACT

Neurobrucellosis is an atypical and grave complication of brucellosis. It can present with a wide range of neurological symptoms, often mimicking other Central Nervous System (CNS) disorders, making diagnosis challenging. The present case report describes neurobrucellosis in a 47-year-old male who presented with atypical neurological symptoms, including depressed mood, dysarthric speech, increased sleepiness for 15 days, and altered sensorium for two days. Initial investigations, including routine Cerebrospinal Fluid (CSF) analysis, were non-contributory, and neuroimaging suggested acute encephalopathy with acute lacunar infarct. However, a high index of clinical suspicion due to his occupation as a farmer with cattle exposure led to advanced serological testing, which showed positive *Brucella* Immunoglobulin M (IgM), confirming *Brucella* infection. The patient was diagnosed with neurobrucellosis and responded favourably to a prolonged course of combination antibiotic therapy. This case underscores the need to include neurobrucellosis in the differential diagnosis of unexplained neurological symptoms, particularly in endemic areas or when there is a history of potential, even minimal, exposure.

Keywords: Central nervous system, Encephalopathy, Neuroimaging viral encephalitis, Psychiatric manifestations

CASE REPORT

A 47-year-old male with a known history of hypertension for five years presented with a 15-day history of depressed mood, slowed speech, and increased sleepiness, followed by altered sensorium for two days, and was subsequently admitted to the critical care unit. The patient was a farmer by profession and had exposure to cattle. There was no significant past medical history or family history. He had been a tobacco chewer for 20 years, consuming two packets of tobacco daily. On admission, his pulse, respiratory rate, and blood pressure were within normal limits. General examination revealed bilateral pitting pedal edema. Neurological examination showed the patient to be drowsy with a Glasgow Coma Scale (GCS) score of E2M2V1, hyperreflexia, clonus, and a positive Babinski sign. The initial clinical provisional diagnosis was acute encephalopathy of undetermined aetiology. Other systemic examinations were non-significant. Laboratory investigations revealed a normal total leukocyte count (9,160 cells/µL), thrombocytopenia (platelet count: 86,000 cells/µL), mildly elevated liver enzymes {Serum Glutamic Pyruvic Transaminase (SGPT): 56 U/L, Serum Glutamic-Oxaloacetic Transaminase (SGOT): 44 U/L}, and a serum bilirubin level of 1.5 mg/dL. Investigations for malaria and dengue were negative. Ophthalmologic evaluation showed no evidence of papilledema or hypertensive retinopathy. Magnetic Resonance Imaging (MRI) of the brain demonstrated focal T2-weighted Fluid Attenuated Inversion Recovery (T2/FLAIR) hyperintensities in the periventricular deep white matter of the bilateral parieto-occipital regions with effacement of the occipital horns of the lateral ventricles. Additionally, a few acute infarcts appeared hyperintense on Diffusion-Weighted Imaging (DWI) with a corresponding signal drop on Apparent Diffusion Coefficient (ADC), involving the left parietal and occipital gyri, without significant post-contrast enhancement, findings suggestive of encephalopathy with acute lacunar infarcts [Table/Fig-1].



[Table/Fig-1]: a-c) Images shows focal T2/FLAIR hyperintensity around periventricular deep white matter in bilateral parietal occipital region with enhancement of occipital horns of bilateral lateral ventricles and acute lacunar infarct involving left parietal and occipital gyri with no post contrast enhancement.

The patient was initially treated with broad-spectrum empirical antibiotics, considering viral encephalitis as a differential diagnosis. He was started on ceftriaxone 2 g twice daily, vancomycin 15 mg/kg/day, and acyclovir 15 mg/kg three times daily. However, there was no improvement in the sensorium despite initiation of empirical therapy. The differential diagnoses considered were viral encephalitis, neurotuberculosis, metabolic encephalopathy, and a rare possibility of neurobrucellosis. CSF analysis was inconclusive, showing a total cell count of 2 cells (100% lymphocytes), protein of 43 mg/dL, glucose of 61 mg/dL (serum glucose 93 mg/dL), and an Adenosine Deaminase (ADA) level of 0.4 IU/L. CSF Acid-Fast Bacillus (AFB) smear and Cartridge-Based Nucleic Acid Amplification Test (CBNAAT) were negative, ruling out neurotuberculosis. The patient's CSF bacterial culture and Herpes Simplex Virus Polymerase Chain Reaction test (HSV PCR) were negative, and there was no improvement with acyclovir. The metabolic parameters were within

the normal range, so metabolic encephalopathy was ruled out. Considering his history of farming and exposure to cattle, *Brucella* IgM testing was performed, which was positive (1.3; reference <0.9 considered negative). Based on this result, the patient was started on doxycycline 100 mg twice daily and rifampin 600 mg once daily, while ceftriaxone was continued. A marked clinical improvement was noted within two days of initiating this treatment. The patient became fully conscious, oriented, and able to sit and ambulate with minimal support. After seven days of critical care admission, his condition stabilised and he was transferred to the general medicine ward. The patient was advised to continue antibiotics for at least six weeks. On discharge, the patient was conscious, oriented, and vitally stable. Symptoms of headache, drowsiness, and speech improved. On examination, gait and reflexes were normal. The patient was followed up with detailed neurological examinations every fortnight for two months. The last follow-up was two weeks after completion of the six-week antibiotic course, at which time the patient was asymptomatic with a normal neurological examination and was able to perform routine daily activities independently.

DISCUSSION

Brucellosis is a zoonotic disease in humans caused by four principal *Brucella* species: *B. suis*, *B. melitensis*, *B. abortus*, and *B. canis*. These organisms are small, Gram-negative, non-motile, non-spore-forming coccobacilli that act as facultative intracellular pathogens. Their primary animal reservoirs include swine (*B. suis*), sheep and goats (*B. melitensis*), cattle (*B. abortus*), and dogs (*B. canis*). Of these, *B. melitensis* is considered the most pathogenic, commonly leading to severe cases and is mainly contracted through unpasteurised dairy consumption or direct animal contact [1].

As noted in the review by Upadhyay AK et al., the prevalence of brucellosis in India shows marked regional variation, with Punjab reporting the highest incidence at 26.6%. In contrast, significantly lower rates have been documented in regions such as Kashmir (0.8%), Delhi (0.9%), Varanasi (6.8%), Gujarat and Belgaum (8.5%), Andhra Pradesh (11.5%), and Maharashtra (19.8%) [2].

The disease typically presents after an incubation period of 2-4 weeks with symptoms such as fever, musculoskeletal pain, and profuse sweating. If untreated, it can progress to a chronic form, with the osteoarticular and nervous systems being the most commonly affected [3]. Neurobrucellosis, though rare (0.5%-25% of cases), usually presents as meningioencephalitis but can also manifest as myeloradiculopathy, peripheral neuropathy, Guillain-Barré syndrome, spondylodiscitis, or intracranial abscesses [4]. Psychiatric manifestations like depression, personality changes, euphoria, and psychosis may also occur but are uncommon [5].

Diagnosing neurobrucellosis remains challenging due to the absence of distinct radiological hallmarks. Nevertheless, clinicians should maintain a high index of suspicion in patients presenting with neurological symptoms, CSF findings indicative of lymphocytic pleocytosis and elevated protein levels, positive *Brucella* Immunoglobulin G (IgG), and CSF culture confirmation-especially when imaging suggests infectious meningitis or meningoencephalitis [6].

In a case report by Vinaykumar PT et al., a 17-year-old female presented with frontal headache, vomiting, a recent generalised tonic-clonic seizure, and subtle behavioral changes such as slurred speech. Clinical evaluation revealed mild fever, stable vital signs, and no focal neurological deficits apart from generalised rigidity, with no notable systemic abnormalities. MRI of the brain with contrast showed an irregular, ill-defined, heterogeneously hyperintense area on FLAIR and T2-weighted images in the right frontal lobe, along with post-contrast and leptomeningeal enhancement. With a negative GeneXpert result, the patient underwent CSF analysis and a right frontal craniotomy with brain biopsy for further evaluation. Findings revealed positive *Brucella* antibodies on serological testing and evidence of active chronic inflammation on brain biopsy, confirming the diagnosis [6].

In a case report by Rossi M et al., a 57-year-old man with a history of recurrent Transient Ischaemic Attack (TIA) and progressive neurological symptoms including diplopia, ataxia, tremors, and hemifacial spasm was eventually diagnosed with neurobrucellosis after MRI changes and positive *Brucella* serology and CSF findings. He had a history of consuming unpasteurised ricotta cheese, suggesting exposure. Initial treatment with IV chloramphenicol and rifampicin was altered due to a new subdural lesion, and second-line therapy with rifampicin and Trimethoprim-Sulfamethoxazole (TMP-SMX) was administered. After five months, the patient showed marked neurological improvement, and follow-up CSF analysis showed only mild abnormalities [7].

In a case report by Soares CN et al., a 21-year-old Brazilian man presented with chronic headache, fever, and fatigue, and later developed neurological symptoms including blurred vision and hemiparesis. Initial investigations and CSF analysis suggested chronic meningitis, and he was empirically treated for tuberculous meningitis despite negative PCR results. While symptoms temporarily improved, persistent abnormal CSF findings prompted further investigation, eventually revealing positive *Brucella* serology. He was treated with rifampin, doxycycline, and trimethoprim-sulfamethoxazole, leading to normalization of CSF parameters and resolution of MRI abnormalities after ten months. Despite full recovery from most symptoms, he retained permanent visual impairment due to optic nerve atrophy [3].

In this study, a 47-year-old hypertensive male presented with depressed mood, altered sensorium, and neurological signs suggestive of acute encephalopathy, confirmed by MRI showing encephalopathic changes and acute infarcts. Initial CSF analysis was inconclusive, and broad-spectrum antibiotics and antivirals showed no improvement. Given the atypical presentation, *Brucella* IgM was tested and found positive. Upon initiation of targeted treatment with doxycycline, rifampin, and continued ceftriaxone, the patient showed significant clinical improvement within two days, regaining full consciousness and mobility with minimal support.

Neurobrucellosis can be diagnosed based on at least one of the following criteria: clinical signs and symptoms suggestive of neurobrucellosis; isolation of *Brucella* species from CSF and/or detection of anti-*Brucella* antibodies in CSF; CSF findings showing lymphocytosis, elevated protein, and reduced glucose levels; or characteristic abnormalities on cranial MRI or Computed Tomography (CT) imaging [8].

Timely and appropriate treatment can halt progression to chronic disease and minimise neurological complications such as myelitis, radiculoneuritis, cranial neuritis, brain abscess, epidural abscess, meningovascular syndromes, and psychiatric manifestations [9].

Doxycycline, rifampicin, and third-generation cephalosporins are considered the standard and preferred medications for treating neurobrucellosis, with a recommended treatment duration of at least six weeks [10]. Other antibiotics that can be used are trimethoprim-sulfamethoxazole and ciprofloxacin, as they have good CSF penetration and tolerability [11].

Neurobrucellosis is a rare presentation with no gold-standard diagnostic approach and requires a combination of clinical judgment and CSF and brain imaging findings, along with combination drug therapy for faster and better recovery [12].

CONCLUSION(S)

Neurobrucellosis should be considered a differential diagnosis in patients presenting with unexplained or atypical neurological symptoms, even in the absence of clear zoonotic exposure, particularly in endemic areas. This case emphasises the diagnostic challenges posed by its nonspecific presentation and the critical role of clinical suspicion in guiding appropriate investigations. Timely recognition and initiation of targeted antibiotic therapy can

result in significant neurological recovery and prevent irreversible complications. Increased awareness among clinicians is essential for early detection and improved patient outcomes.

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