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Research Article

A Case Report On Neurobrucellosis: A Forgotten Cause Of Meningoencephalitis

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A B S T R A C T

Human brucellosis caused by Brucella melitensis is the most prevalent zoonotic infection globally. This report highlights a case of neurobrucellosis in a male patient from rural India, underscoring the importance of timely clinical suspicion followed by microbiological confirmation. A 64-year-old farmer from Himachal Pradesh, India, presented to us with a short history of fever, moderate diffuse headache, and myalgia. He subsequently developed sudden-onset left hemiparesis, disorientation, bilateral sensorineural hearing loss, and neck rigidity. Laboratory investigations revealed abnormal blood counts, and cerebrospinal fluid analysis showed increased lymphocytes and protein content. Serological tests confirmed the diagnosis of neurobrucellosis. Prompt initiation of antibiotic therapy resulted in significant clinical improvement. This case highlights neurobrucellosis as a rare but a significant manifestation. It is important to consider it as a differential in a patient of atypical meningoencephalitis. A very specific history of contact with livestock or unpasteurised dairy is also imperative. The patient's swift response to targeted antibiotic therapy underscores the value of timely diagnosis and effective management in improving outcomes.

Keywords:

Introduction

Brucellosis is the most common bacterial zoonotic disease, with recent studies estimating that 1.6 to 2.1 million new human cases occur each year, a significant increase compared to the previously reported figure of 500,000 cases, which has remained unchanged for over a decade.1 The disease is widely distributed across the globe and is recognized as a zoonotic disease that is emerging in certain regions. It is listed among the neglected tropical zoonotic diseases by the World Health Organization. Brucella is an intracellular gram negative coccobacillus leading to abortion and infertility in the animals. Human brucellosis is a zoonotic infection which is primarily caused by four species named: Brucella melitensis (from sheep), B. suis (from pigs), B. abortus (from cattle), and B. canis (from dogs).2 Human brucellosis is transmitted through the consumption of contaminated milk, curd, and dairy products, as well as via aerosol exposure and occupational contact with infected sheep, cattle, and dogs. It is known to have systemic involvement presenting with a broad spectrum of symptoms and complications. It is classified into three forms: acute (septicemia), subacute (with secondary localization), and chronic (lasting longer than one year). Neurobrucellosis is

frequently misdiagnosed and can result in conditions such as meningoencephalitis, encephalitis, myelitis, cerebral venous thrombosis, and various psychiatric symptoms. Therefore, timely assessment and diagnosis are crucial.3 We present the case of a 64-year-old male farmer with neurobrucellosis, along with a comprehensive review of the relevant literature.

Case Presentation

A 64- year old male farmer from Himachal Pradesh, India presented to our hospital with fever, headache for four days which was moderate in intensity, diffuse, continuous throughout the day. Patient did not complaint of any light or sound intolerance. He also complained of loss of appetite since the onset of headache which was gradually worsening. Next morning, he was found to be disoriented on his bed sweating profusely, warm to touch and breathing heavily. There was history of spontaneous passage of urine in clothes as well. There were no complaints of abnormal body movement, up-rolling of eyeballs or frothing from mouth. The patient had a history of type II diabetes mellitus and hypertension for five years, with poor compliance to his prescribed medications. On examination, the patient had a regular pulse rate, and his blood pressure was within the normal range. His oxygen saturation was 78% on room air, improving to 91% with a simple oxygen mask at 4 litres per minute. He was tachypnoeic, with a respiratory rate of 30 breaths per minute, and exhibited a barrel-shaped chest. Additionally, he had tachycardia, and heart sounds were muffled. During the examination, the patient experienced a seizure characterized by left-sided facial twitching and tonic-clonic movements of the left upper limb. Bell's phenomenon was positive. His pupils were unequal, with an absent direct and consensual light reflex in the left eye. Tone was increased in the left half of body with flexion in upper limbs and extension in lower limbs, bulk was decreased symmetrically in all four limbs. Plantar reflex was mute bilaterally with positive Kernig's sign and neck rigidity. Although sensory examination was normal. In view of degrading sensorium with new onset seizure and left sided hemiparesis patient underwent a non-contrast Computed Tomography (CT) which returned with grossly normal results which further warranted a need for magnetic resonance imaging (MRI) of brain. Chest X-RAY showed emphysematous changes. All relevant blood investigations (Table 1) were sent and a lumbar puncture was performed with working diagnosis of acute meningoencephalitis -viral versus bacterial and samples were sent for Human simplex virus (HSV 1 / 2), acid fast bacilli (AFB), Cartridge based nucleic acid amplification test (CBNAAT) and mycobacteria growth indicator tube (MGIT) along with routine microscopic Cerebrospinal Fluid (CSF) examination

Table 1.Routine and special blood investigations

	DAY1	DAY 7	DAY 15	DAY 20
Haemoglobin(g/dl)	19.6	20.9	19.5	15.5
White Blood Cell (cells/mm^3)	16800	21000	20200	15400
Platelet (cells/µL)	120000	75650	290000	264000
Erythrocyte Sedimentation Rate (mm/hr)	18			
Quantitative C-Reactive Protein (mg/L)	31.8	64.4	55	40
Blood Urea Nitrogen (mg/dL)	28	64	39	19
Creatinine (mg/dL)	1	2.93	0.91	0.69
Sodium (mEq/L)	135	161	149	139
Potassium (mEq/L)	5.25	4.84	4.62	3.92
Chloride (mEq/L)	99	112	118	102
Corrected Calcium (mg/dL)	8.18			
Phosphorus (mg/dL)	2.5			
Vitamin D (ng/dL)	3.8			
Total Bilirubin (mg/dL)	0.93			
Direct Bilirubin (mg/dL)	0.17			
Aspartate Transferase (U/L)	33			

Alanine Transferase (U/L)	34			
Alkaline Phosphatase (U/L)	51			
Hepatits B serology	Non Reactive			
Hepatitis C serology	Non Reactive			
HIV 1 and 2	Non Reactive			
Nerve Conduction Study	Bilateral Peroneal Axonal Neuropathy			
Ultrasound Abdomen	Normal			
Anti-Nuclear Antibody by Hep 2	Negative			

Table 2. Cerebrospinal Fluid Analysis

Cerebrospinal Fluid Analysis Parameter	Value
Volume (mL)	4
Total Count (cells/mm^3)	32
Neutrophils (%)	11
Lymphocytes (%)	89
Protein (mg/dl)	118
Glucose (mg/dl)	21.5
Adenosine Deaminase (U/L)	<4
Gram Stain	Negative
Ziehl-Neelsen Stain	Negative
Culture Sensitivity	Negative
Cartridge Based Nucleic Acid Amplification Test for Tuberculosis	Negative
Herpes Simplex Virus 1 and 2	Negative

(Table 2). Cultures for blood and urine were also sent.

Patient was managed initially with intravenous(iv) ceftriaxone 2 g twice a day, iv vancomycin 1 g twice a day, iv ampicillin 2 g four hourly, iv acyclovir 600 mg thrice a day, iv pantoprazole 40 mg once a day and iv insulin thrice pre-meal with basal insulin and was also loaded with injection phenytoin 1 g iv bolus followed by 100 mg iv thrice a day in view of possibility of status epilepticus.

On the third day of admission, iv Doxycycline was added to provide cover against rickettsia (endemic in Himachal Pradesh) and other atypical organisms. Human immunodeficiency virus, scrub typhus, dengue, typhoid workup came out to be negative but in view of improved sensorium doxycycline was continued. Magnetic resonance imaging reports showed signs of old lacunar infarct in left frontal lobe with mild cerebral and cerebellar atrophy. CSF of the patient showed a cell count of 32 cells mm-3 with lymphocytes and monocytes comprising about 89% of total, with protein of 118 mg/dl which was elevated and glucose of 21.5mg/d which was reduced (Table 2). CSF CBNAAT was negative for mycobacterium tuberculosis and was also negative for HSV 1/ 2. Subsequently CSF cultures also came out to be negative for bacteria and fungus. Injection acyclovir was omitted on day three as soon as the reports of CSF were available. Patient developed thrombocytopenia on seventh day following which iv ceftriaxone and iv vancomycin were omitted on account of probable drug induced thrombocytopenia. In the light of an increasing total leukocyte count (Table1) and worsening crepitation in the right lung fields, iv Amikacin and iv Piperacillin-tazobactam were added. Patient sensorium was completely improved on the next day. Subsequently, patient started complaining of pain in his lower back which was radiating to his left leg following which an MRI whole spine was done which unveiled a collapsed vertebra at D5 level, osteoporotic collapse, altered T1 signal intensity in superior endplate of L3 vertebra, a dual energy X-ray absorptiometry (DEXA) scan also manifested osteoporosis, 25hydroxy vitamin D levels were significantly low. The patient was supplemented and tablet Risedronate was added after calcium correction. A Nerve conduction study showed peroneal axonal neuropathy bilaterally.

Even after sixteen days of antibiotic cover the patient was unable to maintain saturation on room air with persistent fever spikes which compelled us to re-evaluate and find the missing piece in this puzzle. The patient had been experiencing episodes of fever with night sweats, decreased appetite without any weight loss and decreased hearing (documented B/L sensory neural hearing loss on pure tone audiometry) all since last three years. Importantly, the patient mentioned that he used to consume curd made from raw milk of his cow with history of recurrent abortions which was coincidentally bought three years back. Hereinafter, Brucella IgM serology was sent which came out to be positive with titres of 35.80(>12 being positive).

Under this clinical vignette and microbiological confirmation, patient was labelled as a case of neurobrucellosis. We put the patient on Gentamicin 240 mg once a day, Rifampicin 600 mg once a day and Doxycycline 100 mg twice a day. He showed drastic improvement in the form of normalising lab parameters and clinical wellbeing. The patient was discharged on the next day on two drugs, that is Rifampicin and Doxycycline for three months. Patient was completely recovered on subsequent follow ups.

Discussion

Brucellosis, traditionally referred to as "undulant fever," is primarily transmitted to humans through direct or indirect contact with infected animals or their unprocessed products, particularly unpasteurized dairy. The risk of transmission is significantly heightened in endemic areas, where traditional farming practices and close contact with livestock are common. As emphasized in public health guidelines, pasteurization of milk and dairy products is a critical preventive measure to reduce the risk of human infection in such regions.[4]

Human brucellosis presents with a broad clinical spectrum, affecting multiple organ systems beyond the typical febrile illness. Common complications include osteoarticular involvement, epididymo-orchitis in men, hepatic inflammation (often manifesting as granulomatous hepatitis), and, rarely, endocarditis. Central nervous system (CNS) involvement occurs in approximately 5-7% of cases and is associated with a poor prognosis. Neurological manifestations may include meningoencephalitis, brain abscesses, and demyelinating lesions, reflecting the organism's capacity to affect virtually any organ system.5 Meningoencephalitis is the most common form of nervous system involvement. A subtype, basal meningitis, may lead to severe headache, visual disturbances, and raised intracranial pressure.6 Notably, vestibuloacoustic neuritis is the most common peripheral manifestation and was observed in our patient as well.7 These symptoms can occur at any stage of the disease and are driven by direct neuroinvasion by the organism, circulating exotoxins, and the host's immunologic and inflammatory response.8 Among the various virulence factors identified, the VirB protein and lipopolysaccharide play a key role in disease progression and immune evasion.9

Given its nonspecific presentation, neurobrucellosis closely mimics tubercular meningitis, particularly in countries like India. The overlap extends to CSF analysis, which in both conditions typically reveals lymphocytic pleocytosis and elevated protein, as well as to imaging findings such as meningeal enhancement, infarcts, and raised intracranial tension.10 Thus, differentiating between the two conditions is essential to prevent misdiagnosis and treatment delays.

In such scenarios, serological tests such as Enzyme Linked Immunosorbent Assay (ELISA), standard tube agglutination test, indirect Coombs test, Rose Bengal test, and complement fixation test become vital for confirmation.11 However, the absence of standardized diagnostic criteria often results in missed diagnoses. Kochhar et al. proposed diagnostic criteria that include: (i) neurological symptoms unexplained by other conditions, (ii) CSF abnormalities with lymphocytic pleocytosis and elevated protein, (iii) positive CSF culture or serology for Brucella, and (iv) clinical improvement following specific antimicrobial therapy.12 These criteria remain essential for timely diagnosis and management.

Prompt diagnosis and treatment are crucial, although there remains some controversy regarding the optimal number and choice of antibiotics.13 Standard treatment typically involves a combination of rifampicin, doxycycline, and an aminoglycoside, administered over an extended period, to effectively eradicate Brucella.6

This case report underscores the importance of raising awareness about common zoonotic infections in at-risk populations, as well as implementing veterinary control measures such as routine testing, vaccination of livestock, and pasteurization of dairy products. Most importantly, it emphasizes the need for timely notification of cases to public health authorities. Clinicians must maintain a high index of suspicion in endemic regions and utilize appropriate serological and microbiological investigations to diagnose human brucellosis effectively.

Conclusions

Neurobrucellosis is a great clinical masquerader, frequently eluding timely diagnosis due to its overlapping and nonspecific clinical manifestations. In endemic regions, it remains an important yet under recognized cause of neurological morbidity and, at times, mortality-particularly among individuals with identifiable risk factors such as the consumption of unpasteurized dairy products and frequent exposure to livestock.This case highlights the critical importance of adopting a holistic approach that considers not only the pathogenic mechanisms of Human

Brucellosis but also host-related factors, environmental exposures, and regional epidemiology. A high index of suspicion, coupled with a broader clinical outlook toward tropical and zoonotic infections, can significantly enhance early recognition. Clinical acumen that looks beyond the common differentials, supported by appropriate diagnostic tools and thorough history-taking, is key to identifying and successfully managing this rare but serious condition. Ultimately, the intersection of vigilant clinical practice, public health awareness, and preventive veterinary strategies forms the foundation for reducing the burden of neurobrucellosis in endemic populations.

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