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Case Report

Neurobrucellosis presenting as acute stroke with brain abscesses: a case report

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Abstract: Brucellosis is a prevalent zoonotic disease that is associated with consuming produce from animals infected with Brucella species, usually in the form of unpasteurized milk and milk products. The involvement of the central nervous system (CNS) is an uncommon but dangerous indication of neurobrucellosis. Neurobrucellosis can cause specific imaging findings on magnetic resonance imaging (MRI). In this study, we report a very rare case of neurobrucellosis presenting with stroke-like symptoms that were treated using antibiotics. A 38-year-old male was referred with a presenting complaint of headache, ataxia, and dysarthria. He was admitted one month prior for possible cerebral vascular accident (CVA), which was ruled out. On neurological examination, left-central facial paralysis and bilateral positive Babinski sign were observed. The brain magnetic resonance imaging (MRI) performed with and without contrast demonstrated an acute ischemic stroke in the right middle cerebral artery (MCA) territory and extra-axial heterogeneous ring-enhancing lesions, respectively. Brucellosis was confirmed on serological assessment. In rare instances, neurobrucellosis can cause stroke-like symptoms and brain abscesses. Neurobrucellosis should be considered in such patients when other neurological disorders cannot explain neuroimaging abnormalities.

Keywords: Neurobrucellosis, stroke, brain abscess

Introduction

Brucellosis is a prevalent zoonotic disease caused by infection with Brucella species. The most frequent infection causes include domestic animals such as sheep, dogs, cats, goats, horses, camels, and swine. Although direct contact with diseased animals is rare, the most common way for humans to contract brucellosis is through eating infected meat or unpasteurized milk products [1]. The regions with the highest incidence are Mexico, Central and South America, the Peninsula, and the Mediterranean. In contrast, developing countries such as India and others are impacted less [2].

The typical clinical manifestations of brucellosis include fever (often intermittent or cyclical), chills, fatigue, sweating (often heavy), joint and muscle pain, headache, loss of appetite, weakness, malaise, weight loss, and back pain. Osteoarticular complications, such as arthritis and spondylitis, are common, and genitourinary involvement, including testicular pain and swelling, can occur. Constitutional symptoms such as asthenia and malaise are highly prevalent, often affecting more than 90% of patients. Symptoms can vary widely, but fever, joint pain, and fatigue are consistently the most reported manifestations in research studies [3, 4].

Brucellosis is a systemic disorder comprised of complications affecting multiple organs. Out of the total number of patients diagnosed with brucellosis, 30% experience focal complications, 2% develop cardiovascular involvement, 2%-6.5% manifest central nervous system (CNS) involvement, and vascular complications occur very rarely [5]. The involvement of the

CNS is an uncommon but dangerous indication of brucellosis [6]. The main clinical manifestations of neurobrucellosis include mononeuritis, meningitis, myelopathy, brain abscess, polyradiculitis, epidural abscess, meningoencephalitis, and cerebrovascular involvement [7].

Recognizing clinical symptoms and conducting a comprehensive patient history assessment is crucial for diagnosing and managing brucellosis. Early detection and treatment are critical for achieving positive outcomes in cases of neurobrucellosis. Sustaining a high level of suspicion is essential for diagnosing patients from regions where the disease is prevalent. Diagnosis often depends on neurological symptoms, serological tests, and brain imaging that indicate the presence of the illness. In patients with laboratory-confirmed brucellosis, the diagnosis of neurobrucellosis may be established based on the following criteria [8]: 1) Presentation of signs and symptoms consistent with neurobrucellosis, such as fever, headache, and cranial nerve palsies; 2) Identification of cerebrospinal fluid (CSF) abnormalities compatible with brucellosis, including CSF lymphocytic pleocytosis, low glucose levels, high protein levels in the CSF, detection of anti-Brucella antibodies in the CSF, or isolation of Brucella from the CSF; 3) Recognition of imaging abnormalities indicative of brucellosis, particularly evident in CT scan and MRI analyses.

Neurobrucellosis can cause specific magnetic resonance imaging (MRI) findings. These findings can be classified as either normal, inflammation, white matter alterations, or vascular changes [9]. White matter changes are nonspecific and may be confused with other inflammatory disorders or infectious diseases [10]. Erdem et al. [11] reported that 45% of neurobrucellosis cases exhibited MRI changes, including meningeal inflammation with post-contrast enhancement, cranial nerve involvement, brain abscesses, enhancement of spinal nerve roots, arachnoiditis, granulomas, white matter, and vascular changes. When interpreting MRI results, it is important to consider infectious processes with a skull base predominance, such as tuberculosis, or granulomatous diseases like sarcoidosis.

In the current study, we report a very rare case of neurobrucellosis presenting with stroke-like symptoms that was treated using antibiotics.

Case report

A 38-year-old male was referred to the Ayatollah Kashani Hospital of Isfahan University of Medical Sciences presenting with headache, ataxia, and dysarthria. The patient was admitted one month prior due to upper limb weakness, dysarthria, and assessment for possible cerebral vascular accident (CVA). At that time, hydrocephalus was observed as the only finding on the brain computed tomography (CT) scan and magnetic resonance imaging (MRI). For this reason, an external ventricular drain (EVD) was implanted for the patient. However, as the patient's hydrocephalus improved, the EVD was removed prior to discharge.

The patient had no past medical history of note and was not on any regular medications. He mentioned that he is a current heavy smoker (> 40 pack-years) and divulged that he is a heroin user. He mentioned that he has been dependent on heroin for the past three to four months.

His observations showed a temperature of 37.2°C, a heart rate of 68 beats/min, a respiration rate of 17 breaths/min, and a blood pressure of 116/45 mmHg. Examination of his abdominal, respiratory, and cardiovascular systems was unremarkable. His Glasgow Coma Scale at the time of presentation was 15.

Neurological examination was carried out on the patient. In the cranial nerve evaluation, the only positive finding was left-central facial paralysis due to right frontal lobe ischemia. Power was assessed using the Medical Research Council (MRC) muscle power scale. There was a power of 5/5 bilaterally in both distal and proximal muscles of the upper and lower limbs. On assessment, deep tendon reflexes (DTRs) in both upper and lower limbs were normal. However, his plantar (Babinski) reflex was upward bilaterally. When walking, the patient displayed an ataxic gait.

Laboratory analysis of blood was performed. Results showed a raised lactate dehydrogenase (LDH). The complete blood count (CBC), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), renal function, liver function, and blood sugar (BS) were all normal (**Table 1**). The rapid antibody/antigen and fourth-generation tests were negative for human immunodeficiency virus (HIV). Due to the hydrocephalus

Table 1. Laboratory finding of our case

Test	Result	Normal	Test	Result	Normal
WBC (/m^3)	6400	4000-11000	AST (U/L)	32	1-37
Neutrophils (%)	70.4	50-70	ESR (1 hour) (mm/hour)	14	0-15
Lymphocyte (%)	21.7	20-40	BS (mg/dL)	97	70-100
RBC (10^{12}/L)	5.66	4.5-5.9	CPK (U/L)	57	1-171
Hb (g/dL)	14.8	14-17.5	Cr (mmol/L)	0.7	0.7-1.3
MCV (fl)	81	80-96	BUN	10.5	8-25
PLT (10^3/μL)	276	150-450	K (mmol/L)	4.1	3.8-5
CRP (mg/dL)	3.5	< 5	Na (mmol/L)	144	136-145
ALT (U/L)	33	1-41	Mg (mmol/L)	2.2	1.8-2.6
LDH (U/L)	574	0-480			

WBC: white blood cell, HB: hemoglobin, PLT: platelets, RBC: red blood cell, CRP: C-reactive protein, MCV: mean corpuscular volume, CPK: creatine phosphokinase, Cr: creatinine, BUN: blood urea nitrogen, LDH: lactate dehydrogenase, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, ESR: Erythrocyte sedimentation rate, BS: Blood sugar.

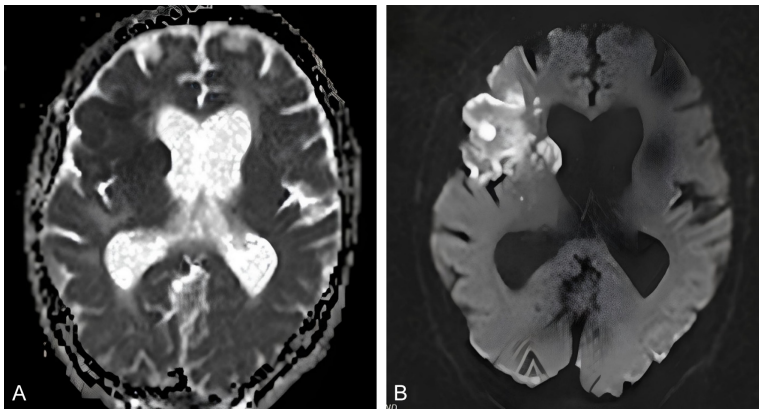

Figure 1. A and B. ADC map and DWI images show Wedge shape true restriction in right frontal lobe and basal ganglia due to acute ischemic infarction.

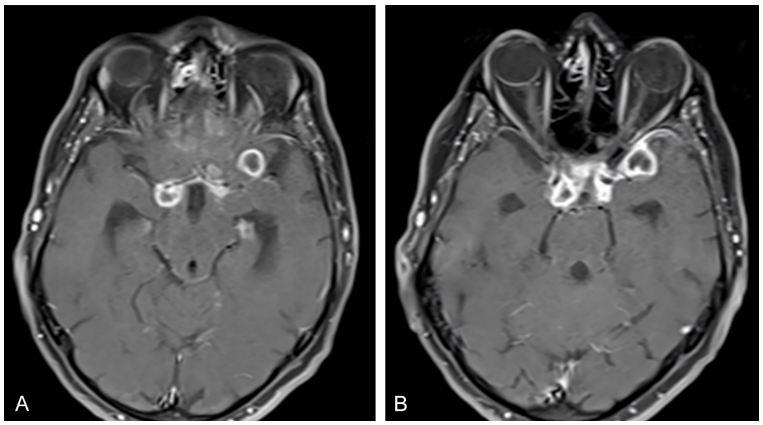

Figure 2. A and B. T1W non fat-suppressed Post-contrast axial images demonstrate extra axial ring enhancing lesions within suprasellar cistern and bilateral middle cranial fossa indicating abscess formation.

observed on the patient's CT scan, a lumbar puncture (LP) was contraindicated to evaluate the cerebrospinal fluid (CSF). However, no evidence of infection was observed in the CSF culture during the patient's previous hospitalization.

An echocardiogram was performed to rule out the possibility of any thromboembolic event and no abnormality was observed.

The MRI of the brain demonstrated an acute ischemic stroke in the right frontal lobe and basal ganglia in the middle cerebral artery (MCA) territory (**Figure 1**). Gadolinium-enhanced magnetic resonance imaging revealed extra-axial heterogeneous ring-enhancing lesions within the suprasellar and middle cranial fossa accompanied by white matter high signal changes and communicating hydrocephaly in FLAIR and T2-weighted images (**Figures 2**, **3**). This suggests an infective process, especially tuberculosis (TB) and brucellosis. As a result, additional diagnostic procedures were conducted to determine the exact cause of these findings. The IFN-γ release assays (IGRA) test for TB and galactomannan (GM) test for aspergillosis were negative. However, the Wright (1/640), Coombs

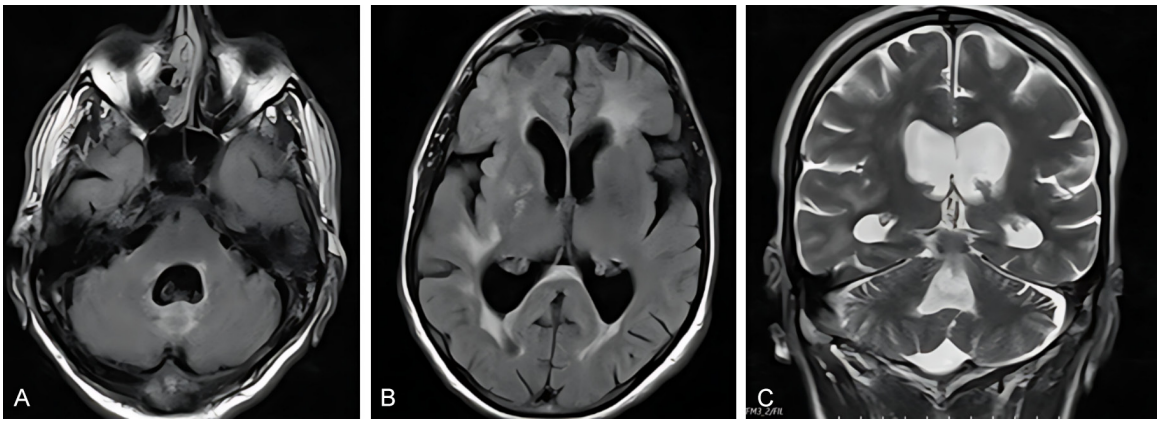

Figure 3. A and B. Axial FLAIR images show periventricular and deep white matter confluent high signal change due to chronic encephalopathy. C. Coronal T2W image illustrates communicating hydrocephaly and brain atrophy.

Wright (1/1280), and 2-Mercaptoethanol (2-ME) (1/320) tests were positive for active brucellosis.

Following the diagnosis of brucellosis, treatment with oral doxycycline 100 mg/day, rifampin 600 mg/day, and intravenous gentamicin 80 mg/day was prescribed. The patient was discharged from the hospital after completing the course of antibiotic therapy. After two months of follow-up, the patient still had dysarthria, but the patient's ataxic gait had partially improved.

Discussion

We present a challenging case of a 38-year-old man with neurobrucellosis presenting as acute ischemic stroke, highlighting the diagnostic complexity and treatment considerations for this rare but serious complication of brucellosis. Our patient's initial presentation with headache, ataxia, dysarthria, and focal neurological deficits (left-central facial paralysis and bilateral positive Babinski sign) exemplifies the diverse neurological manifestations of neurobrucellosis. The concurrent presentation of acute ischemic stroke in the right MCA territory with ring-enhancing lesions created a diagnostic challenge that required systematic evaluation, emphasizing the importance of maintaining a high clinical suspicion for neurobrucellosis in patients with unexplained neurological symptoms and an appropriate exposure history.

Brucellosis is globally recognized as the most prevalent zoonotic infection. 'Neurobrucellosis' is the medical term for the condition involving the nervous system [8]. Neurobrucellosis involving the central nervous system occurs in roughly 2-6.5% of brucellosis cases [5]. Due to the non-specific clinical symptoms and low sensitivity of conventional culture testing, neurobrucellosis is difficult to diagnose [12]. The exact mechanisms through which Brucella infection causes ischemic stroke remain to be elucidated. Possible pathophysiological processes include invasion of the endocardium by Brucella, resulting in vegetative formations that can embolize and occlude cerebral arteries. Furthermore, the inflammatory immune response triggered by Brucella or its endotoxins may induce vasospasm, small vessel vasculitis, or venous inflammation, increasing the risk of infarction, microbleeding, or venous thrombosis in the cerebral vessels [13]. These effects may collectively contribute to ischemic lesions, as observed in our patient.

The most common neurological symptom in patients with neurobrucellosis is severe headache, with possible presentations including cervical and peripheral neuropathies, myelitis, meningoencephalitis, or the rare cerebrovascular disease, which can be easily misdiagnosed and result in cerebral infarction [14]. The exact cause of Brucella-induced ischemic stroke remains unclear, with limited evidence of direct invasion of cerebral blood vessels. Potential mechanisms include Brucella invading the endocardium to form vegetative growths that embolize and cause cerebral artery infarctions, or the inflammatory immune response and endotoxins triggering vasospasm, venous inflamma-

tion, or small vessel vasculitis, thereby elevating risks of infarction, micro-bleeding, or venous thrombosis in arterial supply regions [15, 16].

Neurobrucellosis lacks established clinical diagnostic criteria or standardized therapeutic strategies, primarily due to its nonspecific symptoms and the frequent occurrence of negative results in routine laboratory tests, resulting in low diagnostic accuracy, high misdiagnosis rates, and challenges in distinguishing it from other neurological conditions, such as multiple sclerosis, meningitis, and tuberculosis [17, 18]. These factors create significant difficulties for clinicians. Overall, cerebral neurobrucellosis represents a rare complication of brucellosis, which can be diagnosed based on a history of Brucella exposure, neurological manifestations, positive serological tests, CSF analysis indicating elevated protein levels and lymphocytic pleocytosis, and neuroimaging evidence of parenchymal lesions or meningeal enhancement [8].

In 2023, Liu et al. [15] reported a case of a 44-year-old man whose initial neurobrucellosis symptoms mimicked a stroke, featuring multiple acute infarcts in the bilateral cerebellar hemispheres and brainstem, specifically within the perforator blood supply areas of the vertebral arteries and bridging vessels, indicating that Brucella may trigger inflammatory disease in small arteries. Similarly, Ceran et al. [16] examined atypical presentations in 18 confirmed neurobrucellosis patients, including one instance of left MCA vasculitis that caused an acute infarction in the vessel's feeder region. Another study by Jochum et al. [19] described acute neurobrucellosis in a young man presenting with thalamic apoplexy; although cerebral arteries appeared normal, MRI revealed an acute left thalamic infarction, and the case was characterized as cerebral vascular disease with symptoms resembling intracranial venous thrombosis, though the lesion did not align with the typical distribution of thalamic feeding arteries.

Cranial MRI is an important tool for supporting the diagnosis of neurobrucellosis, as it reveals characteristic abnormalities in the brain and meninges. The most common MRI finding is basal meningeal enhancement after gadolinium contrast administration, which is highly suggestive of meningeal inflammation. Additional MRI features that can be seen in neurobrucellosis are focal cortical or subcortical lesions that demonstrate ring enhancement with surrounding edema, white matter hyperintensities, nodular lesions, and areas of demyelination. These lesions typically involve the basal nuclei, thalamus, brainstem, and cerebellum. MRI may also show signs of brain swelling or hydrocephalus in severe cases [20]. While such imaging findings are supportive of neurobrucellosis, they are not pathognomonic alone and must be interpreted in the context of clinical symptoms and laboratory findings, including cerebrospinal fluid analysis. MRI abnormalities can sometimes resemble other infectious or inflammatory conditions, making a combination of imaging, clinical, and serological data essential for an accurate diagnosis [20, 21].

A radiological finding known as a ring-enhancing lesion can be seen in disorders such as neurocysticercosis, tuberculoma, and brain abscess. Within the framework of neurobrucellosis, ring-enhancing lesions may manifest on the brain due to the disease's varied radiological manifestations. Neurobrucellosis may manifest as basal meningitis, affecting multiple cranial nerves in approximately fifty percent of instances. Rarely, meningoencephalitis can result in hydrocephalus and intracranial hypertension. Postcontrast images of uncomplicated meningitis may exhibit enhancement of the meninges or nerve roots, although the imaging findings are typically normal. Predominantly observed abnormal enhancement occurs in the perivascular region, suprasellar region, basal, and dural meninges [22, 23]. Images that demonstrate meningeal enhancement are the most advantageous, and the ideal sequences to use for this are fluid-attenuated inversion recovery (FLAIR) and contrast-enhanced T1-weighted images. Other infectious etiologies, such as sarcoidosis, fungal infection, and TB, are included in the differential diagnosis [10, 24]. However, the GM and IGRA test were negative in our case.

In 2015, Reggio et al. [25] reported a 57-year-old man with a history of recurrent transient ischemic attacks (TIAs) who was referred with a presenting complaint of diplopia for one week. At the time of admission, his neurological evaluation identified several abnormalities: a wide-based gait, ataxia, right sixth nerve palsy, postural and kinetic tremors in both hands, and a

positive Romberg's sign. Furthermore, he mentioned that he had recently consumed cheese made from unpasteurized milk, increasing the likelihood of contracting brucellosis. The serological test confirmed brucellosis infection. In addition, an MRI of the brain revealed a peripheral ring enhancement-encircling subdural nodular lesion and a distended and enhanced dura matter in the right frontal region.

The main current treatment approach for brucellosis, including neurobrucellosis, as in our case, is a combination antibiotic regimen for several weeks to months because of the intracellular location of Brucella bacteria and the high relapse rates with monotherapy. The World Health Organization and many studies support a two-drug regimen, including doxycycline plus an aminoglycoside (streptomycin or gentamicin) or doxycycline with rifampin, which is generally administered for 6 weeks or longer [26]. In complicated cases, such as spondylitis, arthritis, endocarditis, or CNS involvement with stroke-like symptoms and abscesses (as seen here), triple therapy (doxycycline, rifampin, and an aminoglycoside) and a longer treatment duration are recommended to improve efficacy and reduce relapse and treatment failure rates [26]. Studies have shown relapse rates of approximately 4.8% and treatment failure rates of approximately 7.4% with combination therapy, and that streptomycin and gentamicin have similar effectiveness when combined with doxycycline [27]. In our patient, the prescribed triple therapy (doxycycline 100 mg/day, rifampin 600 mg/day, and intravenous gentamicin 80 mg/day) aligned with these guidelines, leading to partial improvement in ataxia at the 2-month follow-up, although persistent dysarthria underscores the need for extended monitoring and potential adjunctive therapies. Serial neurological examinations and repeat MRI at 4-6 weeks are essential to assess treatment response, as clinical improvement may be gradual and incomplete. While controversial, short-term corticosteroids may be considered in cases with significant cerebral edema or mass effect from ring-enhancing lesions; however, this requires careful monitoring for potential immunosuppression. The decision to initiate anticoagulation therapy in neurobrucellosis-associated stroke should be individualized, considering the inflammatory nature of the vasculopathy and the bleeding risk from potential hemorrhagic transformation.

Our patient's persistent dysarthria at the 2-month follow-up highlights several important management considerations. Extended treatment duration beyond standard protocols may be necessary, along with early speech therapy referral for patients with bulbar symptoms and long-term neurological monitoring, as recovery may take months. Additional imaging to rule out treatment-resistant lesions is also warranted. Post-treatment dysarthria may result from a variety of underlying causes, with neurological injury or dysfunctional muscle control being the primary causes. Various neurological disorders, including amyotrophic lateral sclerosis (ALS) and Parkinson's disease, as well as severe head trauma, can cause neurological damage [17, 21]. Dysarthria can result from diseases such as muscular dystrophy or myasthenia gravis, which weaken the speaking muscles. Speech therapy is a crucial part of the treatment, and recovery may differ depending on the underlying cause and personal circumstances. Communication skills can be considerably improved with customized interventions and routine follow-ups.

Based on our experience and a literature review, factors associated with better outcomes include early diagnosis and treatment initiation, combination antibiotic therapy, absence of hydrocephalus or brainstem involvement, and younger patient age. For patients presenting with stroke-like symptoms in endemic areas, clinicians should obtain a detailed exposure history and, if suspicion is high, immediate Brucella serology alongside the standard stroke workup. If positive serology with neurological involvement is confirmed, triple therapy should be initiated with monitoring of response at 4-6 weeks through clinical and imaging assessments, and therapy should be extended based on clinical response and lesion resolution.

Although neuroimaging tests are important for diagnosing neurobrucellosis, their diagnostic utility depends on the patient's clinical history. It is necessary for radiologists to increase their knowledge of Neurobrucellosis. In cases where simple cerebrovascular lesions fail to account for unexplained stroke manifestations, clinicians require a more comprehensive understanding of the patient's medical history to

make an accurate and timely diagnosis. Our case emphasizes that a systematic approach combining clinical suspicion, appropriate serological testing, and aggressive combination therapy can lead to favorable outcomes, even in complex presentations involving both strokes and parenchymal lesions.

Conclusion

Neurobrucellosis may mimic a variety of central and peripheral CNS diseases, resulting in misdiagnosis and treatment delays. We present a rare case of neurobrucellosis manifesting as stroke-like symptoms accompanied by a brain abscess. Consequently, it's critical to rule out neurobrucellosis when neuroimaging abnormalities are present and cannot be well explained by other neurological disorders.

Disclosure of conflict of interest

None.

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