SVOA Neurology

e-ISSN 2753-9180

ScienceVolks Open Access

Case Report

Neurobrucellosis: A Great Mimicker in Disguise

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DOI: https://doi.org/10.58624/SVOANE.2026.07.007

Received: February 02, 2026

Published: February 18, 2026

Citation: Akshinthala KL, Rao RV, Garapati DT, Yareeda S, Turaga SP, Sharma V, Shaik RS. Neurobrucellosis: A Great Mimicker in Disguise. *SVOA Neurology* 2026, 7:1, 33-37. doi. 10.58624/SVOANE.2026.07.007

Abstract

Background: Neurobrucellosis is a rare clinical complication of brucellosis that can manifest in isolation without systemic features. Subacute to chronic onset and progression of sensory neural hearing loss (SNHL) secondary to isolated affection of the eighth nerve can be a diagnostic clue.

Methods: Descriptive study of a case of a 28-year-old gentleman with a progressive painless bilateral sensory neural hearing loss (SNHL) of 4 months duration and spastic dysarthria of 2 months duration. His neurological examination revealed bilateral SNHL and a spastic ataxic dysarthria with pan cerebellar dysfunction. A clinical diagnosis of intraxial brainstem syndrome was considered. Workup with imaging, serology and cerebrospinal fluid analysis was advised.

Results: Imaging showed hyperintensities in the subcortical and juxtacortical areas of the left frontal and bilateral temporal lobes. Serum IgG Brucella Antibody by enzyme-linked immunosorbent assay(ELISA) had a titre of 92 RU/ml(Normal range <22 RU/ml). Cerebrospinal Fluid(CSF) analysis was performed on three occasions, consistently showing lymphocytic pleocytosis, elevated protein, and low glucose levels. Isolated affection of the eighth cranial nerve is a known clinical phenotype in Neurobrucellosis.

Conclusion: Neurobrucellosis is an underrecognized neuroinfection in India, despite its endemic status for this infection. Our case exhibits several distinctive features. The absence of fever in our case doesn't exclude Neurobrucellosis.. The presence of demyelinating features on imaging, CSF oligoclonal bands (OCB), and CSF suggestive of neuroinfection hints at an interplay between infection and immunity.

Keywords: *Neurobrucellosis, Sensory Neural Hearing Loss (Snhl), Igg Brucella Antibody, Enzyme-Linked Immunosorbent Assay (Elisa), Isolated Cranial Nerve Eight*

Introduction

Neurobrucellosis is a rare clinical complication of brucellosis that can manifest in isolation without systemic features.[1] Tuberculosis is the closest mimic, given the chronic neurological manifestations.[2] Subacute to chronic onset and progression of sensory neural hearing loss (SNHL) secondary to isolated affection of the eighth nerve can be a diagnostic clue of Neurobrucellosis.[1] The sources of infection include consumption of unpasteurized dairy products (most common), soil and skin abrasions, and inhalation of contaminated aerosols.[2] Certain professions, such as slaughterhouse workers, veterinarians and laboratory personnel, are at a higher risk of infection owing to exposure to animals and animal products.[2] Chronic brucellosis usually affects the osteoarticular system and the nervous system.[3] Neurobrucellosis could be due to direct neuropathic effect and/or deleterious cytokine or endotoxin release following the infection and/or an inflammatory/immunologic host reaction to *Brucella* within the CNS.[4]

Case Presentation

We present a case of a 28-year-old gentleman with a history of progressive painless bilateral sensory neural hearing loss (SNHL) of 4 months duration and spastic dysarthria of 2 months duration. He never had a history of fever or other constitutional symptoms. He has a history of handling cattle at his residence. He had no significant past medical history, no known comorbidities, and no notable family history. His general examination was unremarkable, and neurological examination revealed bilateral SNHL and a spastic ataxic dysarthria with pan cerebellar dysfunction. A possible diagnosis of an inflammatory illness like Cogan's syndrome, Susac's syndrome, Neurobehcet's syndrome or Neurosarcoidosis was considered. All routine laboratory parameters (complete blood count, complete urine examination, renal, liver, and thyroid function tests, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP)) were normal. Serum IgG Brucella Antibody by enzyme-linked immunosorbent assay (ELISA) had a titre of 92 RU/ml (Normal range <22 RU/ml). Cerebrospinal Fluid (CSF) analysis was performed on three occasions, consistently showing lymphocytic pleocytosis, elevated protein, and low glucose levels (Supplementary Table 1). CSF oligoclonal bands (OCB) were positive for type 2 bands. Magnetic Resonance Imaging (MRI) of the brain fluid attenuation inversion recovery (FLAIR) sequence showed hyperintensities in the subcortical, juxtacortical left frontal and bilateral temporal lobes (Figure 1) with no affection of the cranial nerves on the 3D CISS (Three-dimensional Constructive Interference in Steady State) sequence, with no evidence of meningitis in the T1 post contrast sequence, repeat MRI after 2 months showed increase in the size of the lesions (Figure 2). CSF and blood culture for Brucella remained negative. The auditory brainstem response (ABR) was abnormal bilaterally, suggesting cochlear and retrocochlear pathology on the right and left sides, respectively (Figure 3). Other etiologies, like other causes of neuroinfection and demyelination, were ruled out by performing Biofire, aquaporin (AQP4) antibody, and myelin oligodendrocyte (MOG) antibody tests on CSF and serum. With the above clinical presentation and supportive evidence of the investigations, a diagnosis of Neurobrucellosis was considered. Thus, the patient was initiated on triple antimicrobials (Parenteral Ceftriaxone, Oral Rifampacin, Oral Doxycycline) along with steroids. Patient reports a mild response in his SNHL.

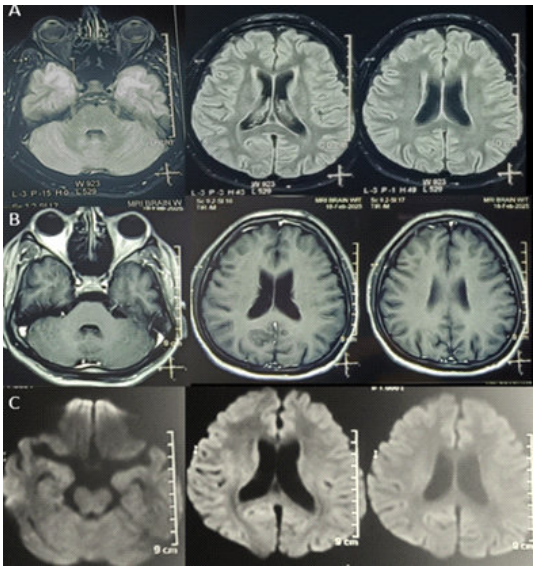


Figure 1. MRI Brain Done On 18.02.2025

A: Fluid attenuation inversion recovery (FLAIR) images of the brain show few discrete and confluent areas of hyperintensities in the left frontal and bilateral temporal lobes.
B: Post-contrast T1 images showing no enhancement.
C: Diffusion-weighted imaging (DWI) images show no areas of restricted diffusion.

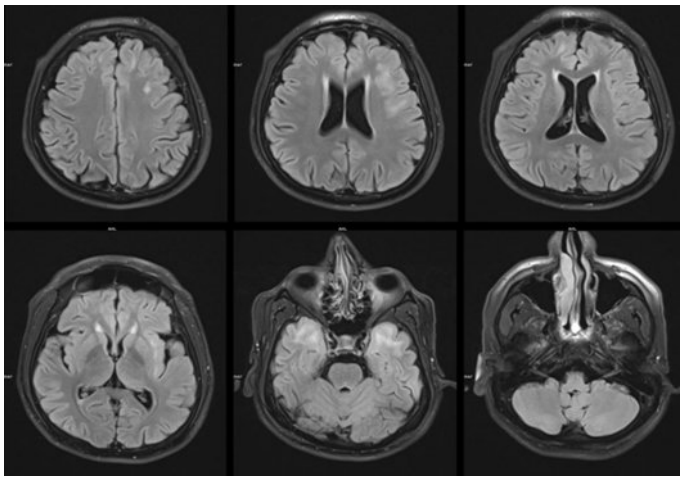
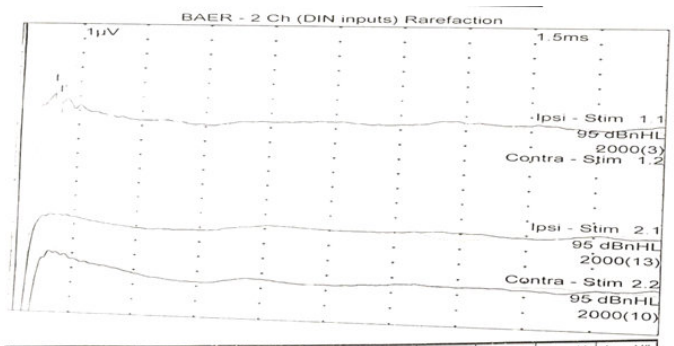


Figure 2. Repeat Fluid Attenuation Inversion Recovery (FLAIR) brain images done on 24.04.25 show progression of the disease activity in the form of new onset discrete lesions on the right frontal lobe and development of confluent lesions on the left frontal lobe.



Run	Stim	I ms	III ms	V ms	▲ I - III ms	▲ III - V ms	▲ I - V ms	Amp I µV	Amp V µV	Amp V/I
Rarefaction										
1	Left	0.9	NR	NR	NR	NR	NR	0.75	NR	
2	Right	NR	NR	NR	NR	NR	NR	NR	NR	NR

Figure 3. The auditory brainstem response (ABR) was abnormal bilaterally, suggesting cochlear and retrocochlear pathology on the right and left sides, respectively.

Discussion

This case emphasises the spectrum of various clinical phenotypes in Neurobrucellosis. Five clinical phenotypes have been described in Neurobrucellosis: acute meningoencephalitis, papilledema and increased intracranial pressure, meningovascular, central nervous system (CNS) demyelination, and peripheral neuropathy.[1] The most commonly used diagnostic criteria are as follows: “(1)symptoms and signs suggestive of Neurobrucellosis not explained by other neurological diseases,(2)positive CSF culture for *Brucella* organisms or positive *Brucella* IgG agglutination titer in the blood,(3)presence of lymphocytic pleocytosis and increased protein in CSF, and(4)response to specific antibiotics with a significant improvement in CSF parameters.” All four criteria are required for the diagnosis of Neurobrucellosis.[5] Though Standard Tube Agglutination(STA) remains a gold standard, ELISA have been widely used in chronic cases of brucellosis.[6]

Our patient in this case report had a demyelinating phenotype on imaging. In a study done over 12 years in the diagnosed cases of Neurobrucellosis, the authors proposed a radiological classification of four patterns: “normal, inflammation (abnormal enhancement: leptomeningitis, pachymeningitis, spinal radiculitis), white matter changes(demyelination affecting predominantly the arcuate fibres), and vascular changes(infarcts or haemorrhages).”[7]

Isolated cranial nerve eight affection in Neurobrucellosis is a well-known phenomenon, and a strong suspicion for Neurobrucellosis should be a rule in an endemic country like India.[7] SNHL in Neurobrucellosis has a poor prognosis; hearing rehabilitation and cochlear implantation in a selected few cases can be offered.[8] The exact pathophysiology of damage to the eighth cranial nerve is not known; various theories proposed are “direct invasion by the bacteria, demyelination, and hypoxia secondary to the inflammatory oedema.”[9] Brucella infection affects the astrocytes and the microglia, which create a microenvironment in the CNS by secreting pro-inflammatory cytokines, leading to destabilisation of the glial structure, damage to the blood-brain barrier(BBB) and neuronal death.[8]

Treatment in Neurobrucellosis is with doxycycline(100 mg, twice daily [bid],oral,6 weeks), rifampicin(600 mg/d, 6 weeks), ceftriaxone sodium(2.0 grams, bid, intravenous infusion[IV], 4–6 weeks).[8] In case of allergy to cephalosporins, ceftriaxone sodium can be replaced with levofloxacin hydrochloride(0.4 g, once daily[qd], IV,4–6 weeks). These should be considered both as standard and first-choice medications for Neurobrucellosis.[10] Treatment for systemic Brucellosis is usually for at least 6 weeks; however, in Neurobrucellosis, treatment for 6 to 12 months may be warranted till the patient has a clinical response and improvement in CSF parameters.[10] The role of steroids in Neurobrucellosis has not been validated in large studies; steroids have been given in patients with cranial neuropathies, myelopathy and arachnoiditis.[10] Prolonged disease duration and increased age of the patient increase the risk of developing Neurobrucellosis.[10] There could be a seasonal variation in the development of Neurobrucellosis, which correlates with the increased contact between humans and livestock during the lambing period and slaughtering time.[10]

Conclusion

Our case exhibits several distinctive features. The absence of fever in our case doesn't exclude Neurobrucellosis. Isolated affection of the eighth cranial nerve is a known clinical phenotype in Neurobrucellosis. The presence of demyelinating features on MRI, CSF OCBs, and CSF suggestive of neuroinfection hints at an interplay between infection and immunity, with a minimal response to treatment.

Patient Consent

Written informed consent has been obtained for this work.

Conflict of Interest

The authors have no conflict of interest to disclose.

Funding

This work required no funding.

Acknowledgement

The authors would like to thank Dr. Bhanu Prasad, Consultant in Infeinput Diseases, for his timely inputs regarding the treatment plan.

References

1. Saleh M. Al Deeb, Basim A. Yaqub, Hassan S. Sharif, Jayent G. Phadke, Neurobrucellosis, Journal Article, 1989, Neurology, 498-498, 39, 4, 10.1212/WNL.39.4.498 [doi] PMID – 2927673

2. Soares CN, Angelim AIM, Brandão CO, Santos RQ, Mehta R, Silva MTTD. Neurobrucellosis: the great mimicker. Rev Soc Bras Med Trop. 2022 Apr 8;55:e05672021. doi: 10.1590/0037-8682-0567-2021. PMID: 35416876; PMCID: PMC9009883.

3. Corbel MJ. Brucellosis in humans and animals. Geneva: WHO; 2006. 89p

4. Kizilkilic O, Calli C. Neurobrucellosis. Neuroimag Clin N Am. 2011;21(4):927–937. doi: 10.1016/j.nic.2011.07.008.

5. Marques IB, Marto N, Raimundo A, Gil-Gouveia R. Myelitis and Polyradiculoneuropathy With Severe Pain: Unusual Neurological Manifestations as Presenting Symptoms of Brucellosis. Neurologist. 2018 Jul;23(4):131-134.

6. Khan MZ, Zahoor M. An Overview of Brucellosis in Cattle and Humans, and its Serological and Molecular Diagnosis in Control Strategie. Trop Med Infect Dis. 2018;14(3(2)):65–65. doi: 10.3390/tropicalmed3020065.

7. M Al-Sous MW, Bohlega S, Al-Kawi MZ, Alwatban J, McLean DR. Neurobrucellosis: clinical and neuroimaging correlation. AJNR Am J Neuroradiol. 2004 Mar;25(3):395-401. PMID: 15037461; PMCID: PMC8158553

8. Guneri EA, Kirkim G, Serbetcioglu BM, Erdag TK, Guneri A. Cochlear implantation in neurobrucellosis. Otol Neurotol. 2009 Sep;30(6):747-9. doi: 10.1097/MAO.0b013e3181b285ec. PMID: 19638936

9. Rodríguez AM, Delpino MV, Miraglia MC, Giambartolomei GH. Immune Mediators of Pathology in Neurobrucellosis: From Blood to Central Nervous System. Neuroscience. 2019 Jul 1;410:264-273. doi: 10.1016/j.neuroscience.2019.05.018. Epub 2019 May 23. PMID: 31128159.

10. Zhao S, Cheng Y, Liao Y, Zhang Z, Yin X, Shi S. Treatment Efficacy and Risk Factors of Neurobrucellosis. Med Sci Monit. 2016 Mar 28;22:1005-12. doi: 10.12659/msm.897947. PMID: 27018084; PMCID: PMC4815993