The Canadian Journal of Neurological Sciences (2025), 1–3
doi:10.1017/cjn.2025.55

Canadian Journal of Neurological Sciences
Journal Canadien des Sciences Neurologiques

Letter to the Editor: New Observation

Intractable Focal Epilepsy from Neurobrucellosis

Zachariah I. Hasan[1], Jacqueline Barnes[1], Sarah Ganji[1], Alexander Andrews[1], Prishanya Pillai[2] and Gholam K. Motamedi[1]

[1]MedStar Georgetown University Hospital Department of Neurology, Washington, D.C., USA and [2]MedStar Georgetown University Hospital Department of Infectious Diseases, Washington, D.C., USA

Keywords: Epilepsy; CNS inflammation; electroencephalogram; epidemiology; MRI; neuroinfectious disease

Neurobrucellosis is a rare manifestation of systemic brucellosis, which is one of the most common zoonotic infections worldwide. Brucellosis is endemic to many parts of the world with suboptimal food safety and/or livestock care standards and poses a global public health challenge with an estimated greater than 2 billion people at risk of exposure worldwide.[1] Neurological complications are estimated to occur in less than 10% of systemic brucellosis cases, with predominantly acute syndromes of meningitis, encephalitis, focal myelitis, radiculitis and polyneuropathies of peripheral or cranial nerves reported.[1,2] Seizure is a rare complication of neurobrucellosis, and cases in the literature report seizures mostly in the setting of acute meningoencephalitis from *Brucella spp*. leading to cortical excitability.[4–6] Here, we report a case of chronic intractable focal epilepsy in the setting of a *Brucella melitensis* granuloma, expanding our understanding of the neurological manifestations of brucellosis.

A 40-year-old previously healthy immunocompetent man presented to the emergency room after a first-time focal seizure described as right face and arm tonic stiffness with preserved awareness that progressed after a few minutes to loss of consciousness with generalized tonic-clonic shaking and tongue bite. He reported one month of intermittent headaches, right arm burning pain, malaise, tremor and subtle right-sided weakness prior to the seizure. He denied fever or night sweats. On arrival, he was afebrile and without nuchal rigidity or altered mental status. Examination was notable for right face and limb hemisensory loss and weakness. He had immigrated to the USA from Peru 8 months prior to presentation and reported ingesting unpasteurized milk and other dairy products regularly. MRI of the brain with and without gadolinium contrast administration revealed an intra-axial mass in the left frontal lobe with areas of solid enhancement and extensive associated vasogenic edema (Figure 1). Laboratory analysis was notable for an elevated serum white blood cell count of 16.7 K/uL with neutrophil predominance. Pertinent negative serum studies included a negative HIV polymerase chain reaction (PCR) screen, syphilis screen, Lyme PCR, interferon gamma release assay (T-spot) TB test, histoplasma serologies, toxoplasma serologies, coccidioidomycosis serologies and blood cultures. Lumbar puncture with CSF analysis was unremarkable with 3 white blood cells/mm^3, 18 red blood cells/mm^3, 28 mg/dL protein, 63 mg/dL glucose, a negative meningitis/encephalitis viral/bacterial/fungal CSF screen and negative CSF cultures. Pathology from an excisional brain biopsy revealed necrotizing granulomatous inflammation, without evidence of any neoplastic process, viral cytopathic changes or vasculitic changes. Cultures from the biopsy revealed fastidious growth of gram-negative rods, which speciated into *B. melitensis*. A diagnosis of neurobrucellosis was made.

While hospitalized, dual therapy with levetiracetam and lacosamide was needed for breakthrough seizure control, and IV dexamethasone was added to reduce vasogenic edema. Long-term video electroencephalogram monitoring found left frontal focal slowing and intermittent left frontal epileptiform abnormalities (Figure 2). The patient then had a subtotal resection of the frontal granulomatous lesion. He was treated with 6 weeks of IV ceftriaxone 2 g daily, followed by 6 months of oral doxycycline 100 mg BID and rifampin 600 mg daily. Interval imaging at 6 months revealed improvement, but portions of the granuloma with enhancement were still present, so oral antibiotic treatment was extended 6 additional months. A brain MRI 14 months after initial presentation and post-completion of the antibiotic regimen showed near complete resolution of the nodular enhancing lesion. Subsequently, he was admitted to the epilepsy monitoring unit for a controlled seizure medication wean. Unfortunately, this led to breakthrough focal seizures requiring continued treatment with both lacosamide and levetiracetam. With physical rehabilitation, the patient gradually regained much of his strength in his right arm and leg but still has residual sensory deficits.

Brucella spp. likely invade the central nervous system (CNS) through hematogenous spread to the meninges.[3] Damage to neurologic tissues is suspected to be due to direct neuropathic effects of the bacteria and/or abnormal host inflammatory response to organisms.[3] Neurobrucellosis has most often been described in cases of acute meningitis and meningoencephalitis.[1–4] Within the peripheral nervous system, peripheral neuropathy and radiculopathy have been described including acute demyelinating polyneuropathy (Guillain–Barre syndrome).[1–4] In the CNS, brain abscesses, mycotic aneurysms, cerebral vasculitis, subarachnoid hemorrhage and spinal cord myelitis have been seen.[1–4]

Corresponding author: Zachariah Hasan; Email: Zachariah.I.Hasan@medstar.net

Cite this article: Hasan ZI, Barnes J, Ganji S, Andrews A, Pillai P, and Motamedi GK. Intractable Focal Epilepsy from Neurobrucellosis. *The Canadian Journal of Neurological Sciences*, https://doi.org/10.1017/cjn.2025.55

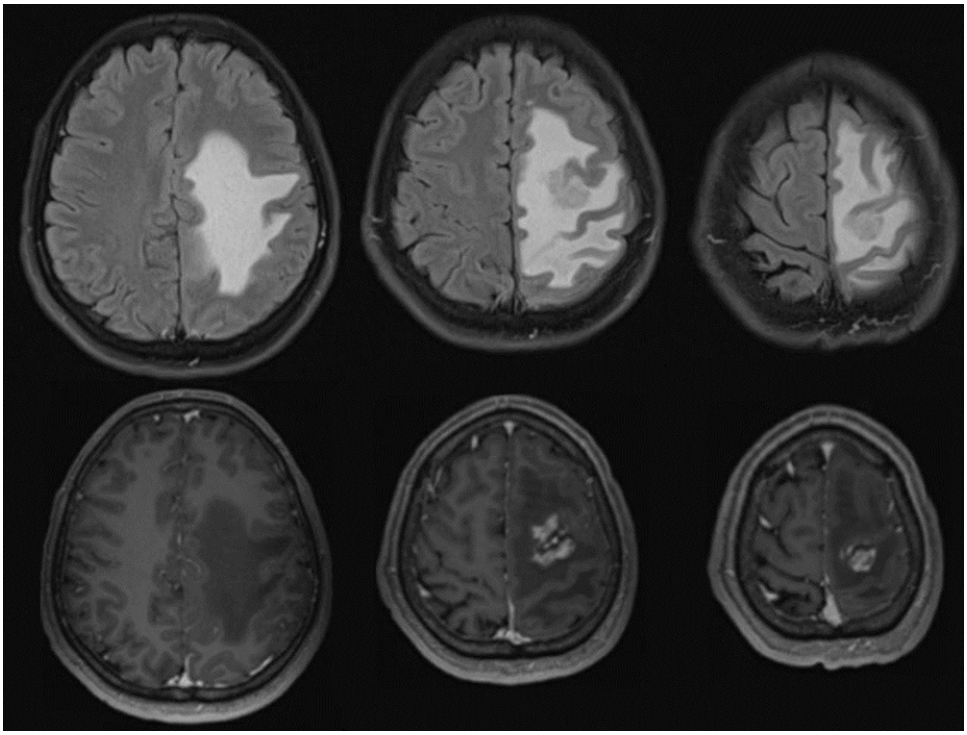

Figure 1. Row 1: Fluid attenuated inversion recovery (FLAIR) axial MRI. Row 2: T1 post-gadolinium contrast axial MRI. Presence of hyperintense signal abnormalities with cortical and subcortical involvement. Patchy enhancing foci along the cortex and adjacent subcortical white matter involving the left motor gyrus in the posterior left frontal lobe.

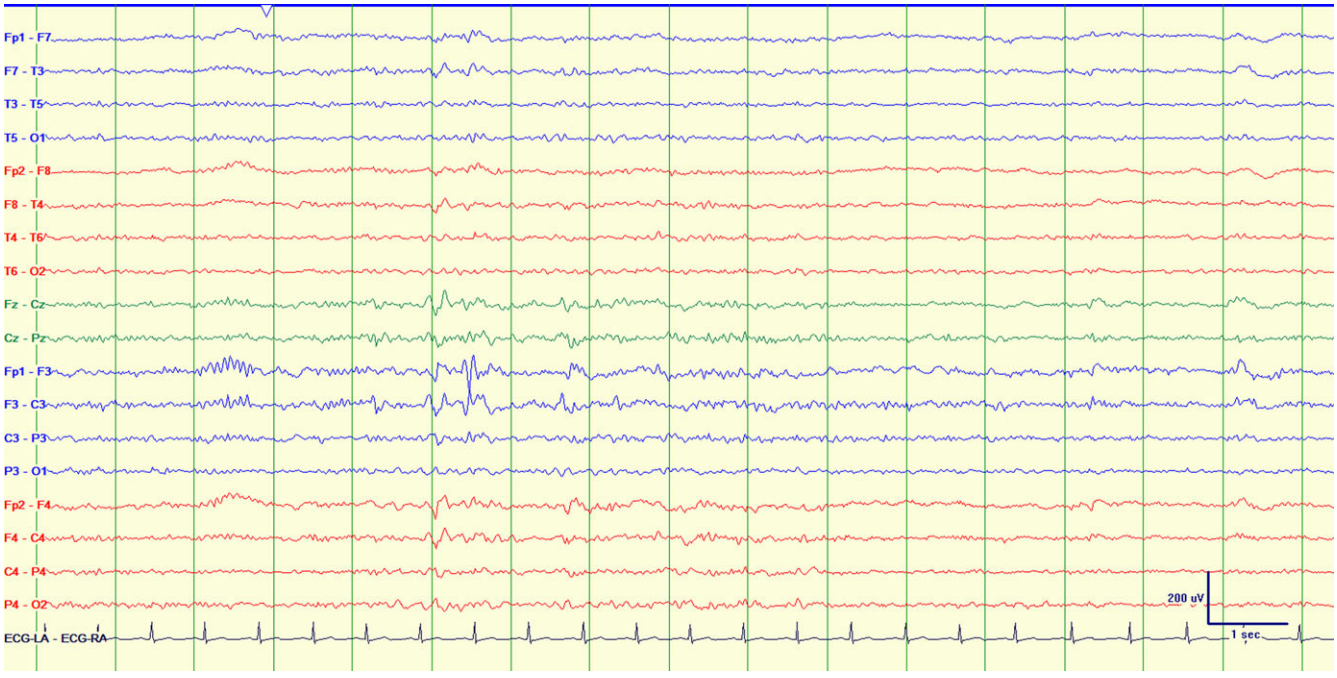

Figure 2. Electroencephalogram recording with evidence of interictal left fronto-central sharp and slow discharge, maximum at the F3 electrode. This localization correlates with the cortical irritability most likely caused by the left frontal granuloma seen on imaging and indicates the potential seizure focus.

Granulomatous inflammation from neurobrucellosis, as seen in this case, has rarely been described in the literature, and the associated clinical syndrome is less well defined.[3–5] Seizures associated with neurobrucellosis have been reported in the setting of acute meningitis, encephalitis and vascular complications, but cases of chronic epilepsy are rare.[4–6] For our patient, direct cortical irritation from the granuloma likely contributed to a chronic focal epilepsy. There have been pediatric cases of CNS brucella abscess leading to epilepsy reported in the literature.[6] One case from Saudi Arabia describes persistent epileptic auras without major seizures in an immunocompromised patient post-renal transplantation, who developed a CNS brucella granuloma.[5] In contrast, this case describes a CNS brucella granuloma leading to epilepsy in an immunocompetent patient.

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There are few guidelines for the optimal approach to treatment of neurobrucellosis as data is limited to observational and retrospective studies.[2–3,7–8] Duration of treatment could potentially be tailored to the presence of symptoms and neuroimaging response to therapy.[7–8] In this case, after continued presence of enhancement of the Brucella lesion despite resection and 6 months of antibiotic therapy, a multidisciplinary decision was made to continue oral antibiotics for an additional 6 months.

This case of neurobrucellosis presenting as focal epilepsy highlights important considerations in the presentation and sequelae of the disease. Brucellosis remains an important zoonosis to better characterize, given it is endemic to much of the world, with billions at risk of exposure annually.[1] In retrospect, repeated ingestion of unpasteurized dairy products while living in Peru was the likely source of infection for our patient. Globalization could bring more cases from endemic regions into the health systems of non-endemic regions. As such, increasing awareness of neurobrucellosis and additional research into the pathophysiology, clinical manifestations and potential treatments of neurobrucellosis could have broad public health benefits.

Acknowledgments. We thank the patient for his willingness to share his story and for consenting to its publication. We also thank the multitude of healthcare professionals who have assisted in the diagnosis and management of this patient.

Author contributions. Z.H., J.B., S.G., A.A., P.P. and G.M. all contributed to the conceptualization, design and content of this manuscript. Z.H., J.B., S.G. and P.P. contributed to manuscript drafting, background research, creation of figures and revision. A.A. and G.M. contributed to supervision and manuscript editing.

Funding statement. None.

Competing interests. Z.H., J.B., S.G., A.A., P.P. and G.M. (all authors) have no relevant disclosures and have completed ICJME disclosure forms that have been submitted to the editorial board.

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