# Mass-like Brainstem Lesion as an Unusual Presentation of Neuroborreliosis: A Case Report

Natia Burjanadze[1*], Medea Chichua[1], Tamta Andrazashvili[1], Khatuna Gordeladze[1]

[1]K. Eristavi National Center of Surgery, Department of Diagnostic Radiology, Tbilisi, Georgia

Swiss Journal of Radiology and Nuclear Medicine - SJORANM GmbH - Rosenweg 3 - CH-6340 Baar - Switzerland

## Abstract

Neuroborreliosis is an uncommon manifestation of Lyme disease, typically presenting with peripheral neuritis, radiculopathy and/or meningitis. We present an atypical case of central nervous system involvement characterized by a mass-like brainstem lesion and additional supratentorial white matter lesions, radiologically mimicking an inflammatory demyelinating process and posing a significant diagnostic challenge.

**Keywords:** Lyme disease, neuroborreliosis, lyme encephalitis.

[*]Corresponding author: Natia Burjanadze - received: 25.01.2026 - peer reviewed, accepted and published: 28.02.2026

## Introduction

Lyme disease is a tick-borne infectious disease caused by the spirochete Borrelia burgdorferi. The most common early manifestation is erythema migrans, but the infection may disseminate and result in multisystem involvement. Although Lyme disease remains relatively uncommon, approximately 65.500 cases are reported annually in Europe [1]. Neuroborreliosis, a neurological manifestation of Lyme disease, is even less frequent and typically presents with lymphocytic meningitis, cranial neuritis and/or peripheral radiculopathy [2]. Parenchymal central nervous system involvement is rare and may create a significant diagnostic challenge, particularly when imaging findings mimic demyelinating or infiltrative disorders. We present a rare case of neuroborreliosis with atypical CNS involvement, radiologically resembling a demyelinating process, underscoring the importance of integrating imaging findings with clinical and laboratory data to achieve an accurate diagnosis.

## Case report

A 30-year-old male presented with gradually progressive left-sided hemiparesis, paraesthesia and mild gradual cognitive changes. He was referred by a neurologist for contrast-enhanced brain MRI. MRI scan demonstrated heterogeneous, mass-like lesion centered in the right pons with surrounding edema, causing mild mass effect on the fourth ventricle. The lesion was hypointense on pre-contrast T1-weighted images. There was no T2 hypointense rim or significant susceptibility effect. No diffusion restriction was observed. Following contrast administration, heterogeneous enhancement was noted (Figure 1).

A smaller lesion was identified in the left corona radiata, exhibiting an irregular morphology with pronounced hyperintensity on T2-weighted images and a peripheral hyperintense rim on FLAIR with mild peripheral contrast enhancement. No diffusion restriction was observed. Additional ill-defined white matter lesions were present contralaterally, demonstrating only subtle T2/FLAIR hyperintensity and mild, more homogeneous post-contrast enhancement (Figure 2). The differential diagnosis included an inflammatory demyelinating process, particularly tumefactive demyelination, as well as infiltrative or infectious etiologies. Given the initial consideration of a demyelinating pro-cess, additional MRI of the cervical spine was performed to assess for further lesions; no abnormal findings were identified.

Open Access · CC BY 4.0 · © 2026 The Author(s) · Published by SJORANM GmbH, Switzerland

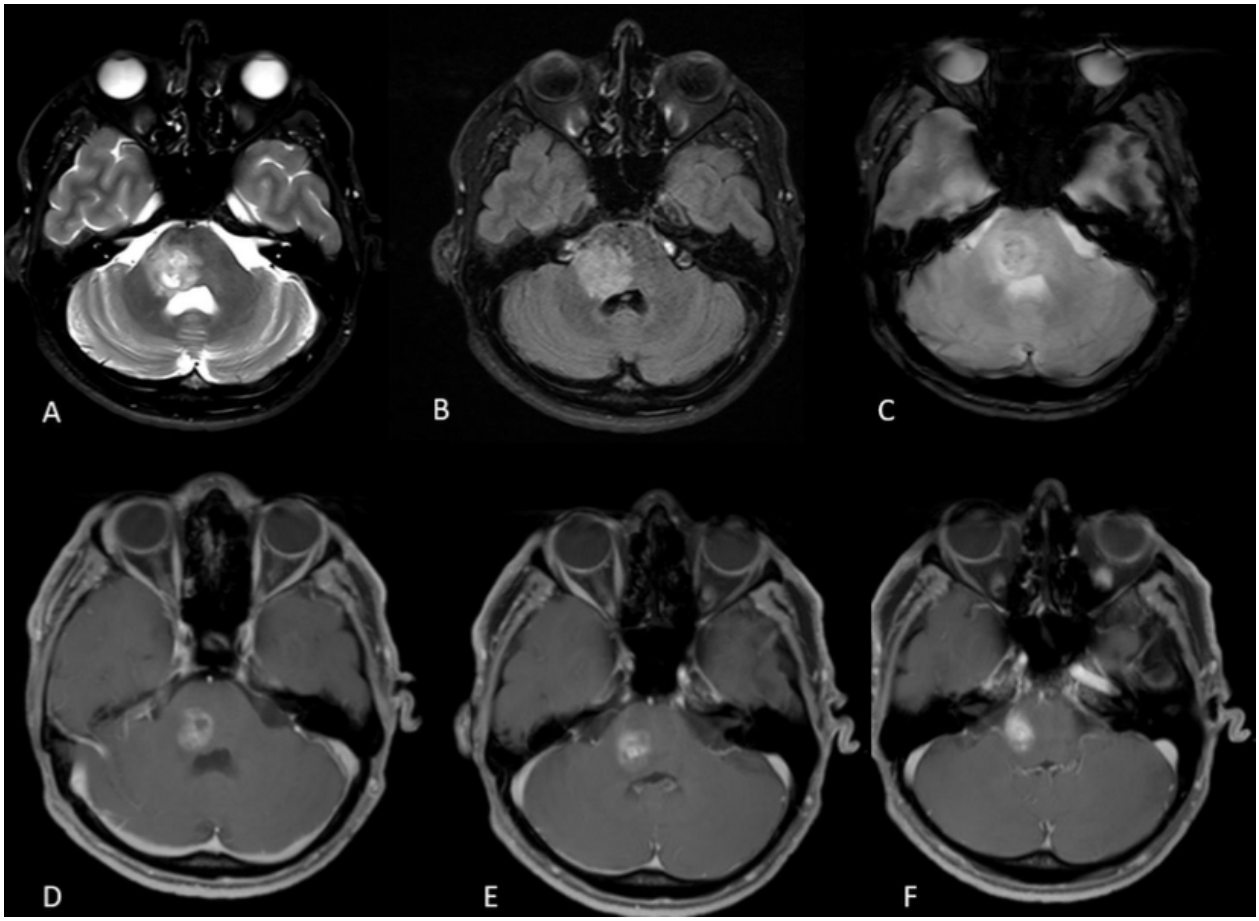



**Figure 1: MRI of the brain demonstrating a mass-like pontine lesion on initial imaging.** (A) Axial T2-weighted image, (B) axial FLAIR image, (C) Axial T2*-weighted gradient-echo image, (D–F) post-contrast axial T1-weighted images. A heterogeneous, mass-like lesion centered in the right pons with surrounding edema, causing mild mass effect on the fourth ventricle. The lesion demonstrates central hyperintensity on T2-weighted images. No T2 hypointense rim or significant susceptibility effect is present. Post-contrast imaging shows heterogeneous enhancement.

CSF analysis revealed pleocytosis and absent oligoclonal antibodies. Despite the absence of systemic infectious symptoms, serological testing demonstrated strongly positive Borrelia burgdorferi–specific IgG antibodies with positive antibody index. Establishing the diagnosis of neuroborreliosis. The patient was treated with a 14-day course of antibiotic therapy and underwent follow-up MRI three weeks after completion of treatment. Clinically, he demonstrated a favorable response, with marked improvement of neurological symptoms; only mild residual paraesthesia of the left lower limb persisted.

Follow-up MRI revealed near-complete resolution of the pontine lesion, while the smaller supratentorial white matter lesions remained largely unchanged in size but showed mildly reduced contrast enhancement (Figure 3, 4).

## Discussion

Lyme neuroborreliosis refers to involvement of the nervous system in Lyme disease and may manifest with both central and peripheral nervous system involvement. The precise mechanism by which Borrelia burgdorferi gains access to the central nervous system remains unknown. Proposed pathways include hematogenous dissemination across the blood–brain barrier as well as perineural spread, both of which have been suggested in previous studies [3]. The most common manifestation of neuroborreliosis is peripheral nervous system involvement, classically described as Bannwarth syndrome. It is characterized by painful radiculopathy and/or cranial neuritis. In contrast, primary central nervous system involvement is rare and may present as encephalitis and/or myelitis [4]. In the present case, the patient exhibited isolated CNS involvement, which contributed to diagnostic difficulty. Clinically, the young age, gradual onset of

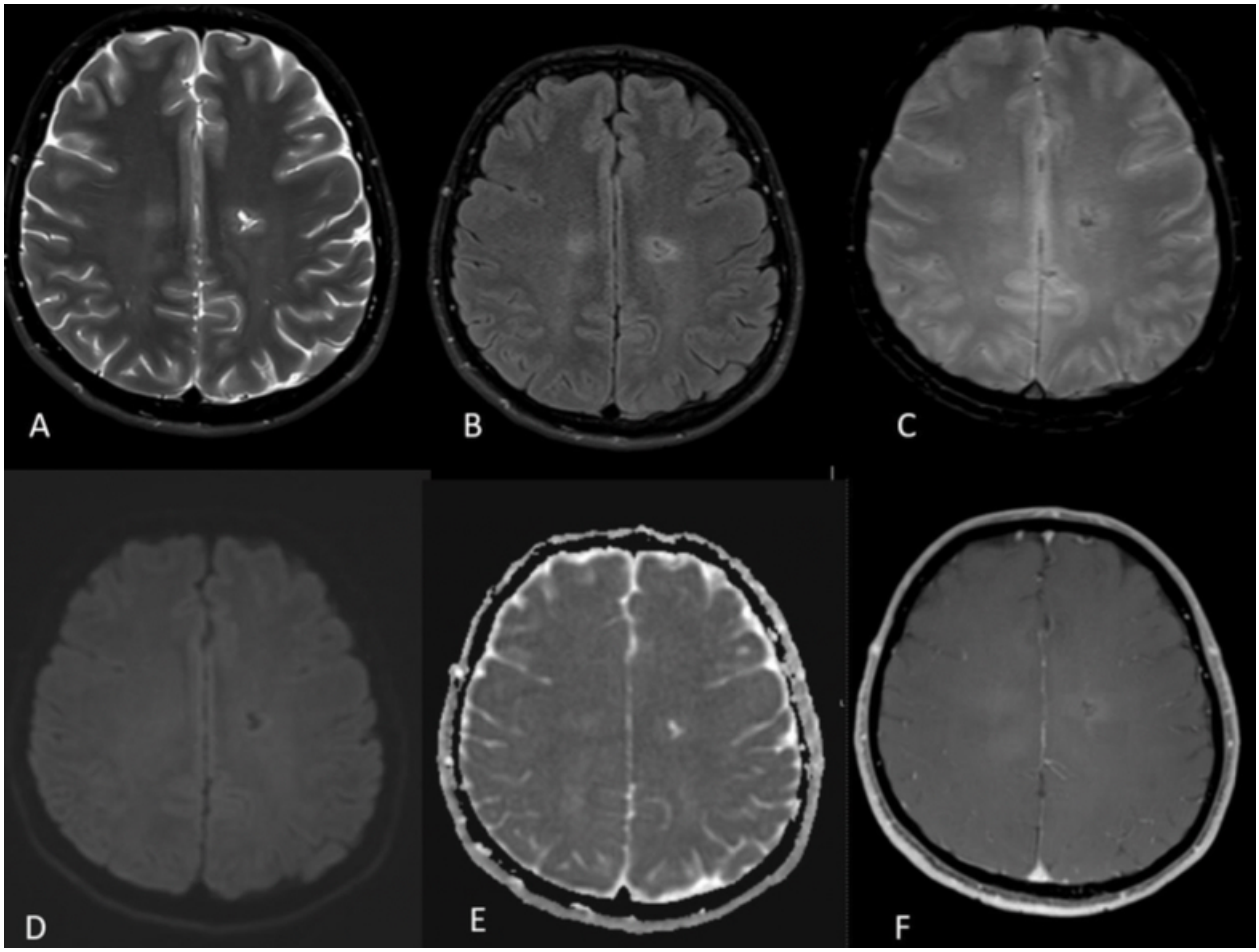



**Figure 2: MRI of the brain demonstrating supratentorial white matter lesions on initial imaging.** (A) Axial T2-weighted image, (B) axial FLAIR image, (C) axial T2*-weighted gradient-echo image, (D) axial high b-value diffusion-weighted image, (E) axial apparent diffusion coefficient (ADC) map, and (F) axial post-contrast T1-weighted image. A smaller lesion is identified in the left corona radiata, exhibiting an irregular morphology with marked hyperintensity on T2-weighted images and a peripheral hyperintense rim on FLAIR, associated with mild peripheral contrast enhancement. No diffusion restriction is observed. Additional contralateral white matter lesions are ill-defined, demonstrating only subtle T2/FLAIR hyperintensity and mild, more homogeneous post-contrast enhancement.

hemiparesis, associated paraesthesa and confusion, in the absence of pain or systemic infectious symptoms, were particularly misleading. From an imaging standpoint, the presence of a large, contrast-enhancing pontine lesion with mild mass effect, together with a smaller supratentorial lesions led two experienced neuroradiologists to initially favor a demyelinating etiology, with tumefac-

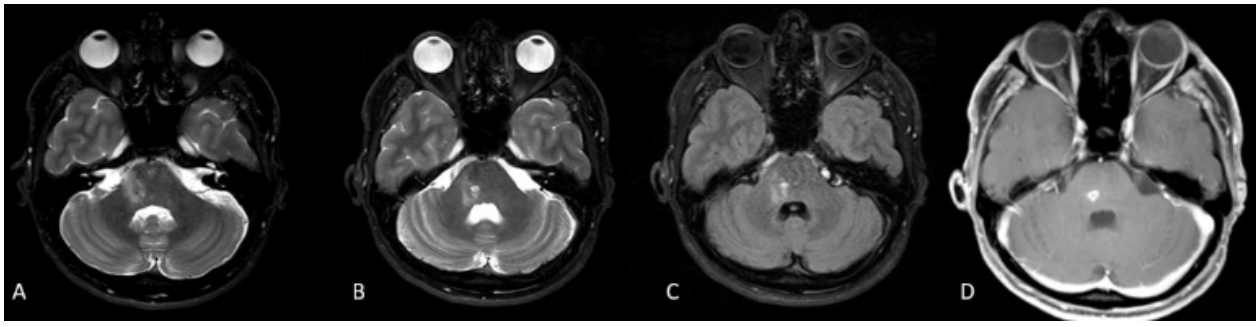



**Figure 3: Follow-up brain MRI demonstrating treatment response of the pontine lesion.** (A, B) Axial T2-weighted images, (C) axial FLAIR image, and (D) axial post-contrast T1-weighted image. Follow-up imaging demonstrates near-complete resolution of the previously described pontine lesion, with marked reduction of surrounding edema and no residual mass effect.

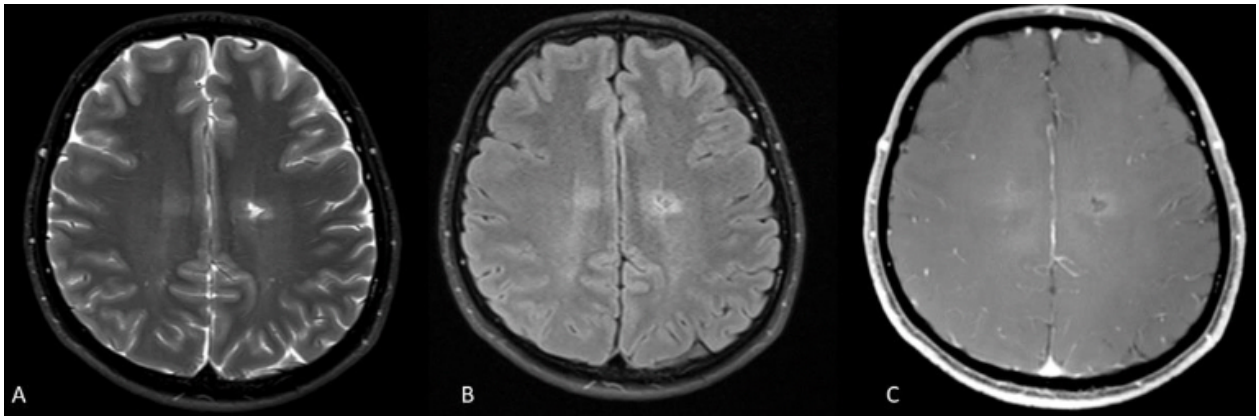



**Figure 4: Follow-up brain MRI demonstrating supratentorial lesions.** (A) Axial T2-weighted images, (B) axial FLAIR image, and (C) axial post-contrast T1-weighted image. Supratentorial white matter lesions remained largely unchanged in size but showed mildly reduced contrast enhancement.

tive demyelination considered the leading differential diagnosis.

## Conclusion

This case illustrates an atypical presentation of neuroborreliosis with isolated central nervous system involvement mimicking a demyelinating lesion on MRI. Given the substantial imaging overlap between demyelinating, infiltrative and infectious processes, integration of radiological findings with clinical and cerebrospinal fluid analysis is essential for accurate diagnosis.

***Correspondence to:***
Natia Burjanadze
natia.burjanadze@gmail.com
K. Eristavi National Center of Surgery, Department of Diagnostic Radiology, Tbilisi, Georgia

## Declarations

Consent for publication: The author clarifies that written informed consent was obtained and the anonymity of the patient was ensured. This study submitted to Swiss J. Rad. Nucl. Med. has been conducted in accordance with the Declaration of Helsinki and according to requirements of all applicable local and international standards. All authors contributed to the conception and design of the manuscript, participated in drafting and revising the content critically for important intellectual input, and approved the final version for publication. Each author agrees to be accountable for all aspects of the work, ensuring its accuracy and integrity.

Competing interests: None.
Funding: No funding was required for this study.

**Conflict of interest:**

The authors declare that there were no conflicts of interest within the meaning of the recommendations of the International Committee of Medical Journal Editors when the article was written.

**Disclaimer/Publisher's Note:**

The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of Swiss J. Radiol. Nucl. Med. and/or the editor(s). Swiss J. Radiol. Nucl. Med. and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.

**License Policy:**

This work is licensed under a Creative Commons Attribution 4.0 International License.
This license requires that reusers give

credit to the creator. It allows reusers to distribute, remix, adapt, and build upon the material in any medium or format, even for commercial purposes.

**SJORANM-LinkedIn:**

Check out our journal's LinkedIn profile with over 11K registered followers from the Radiologic & Nuclear Medicine Imaging field.

### *References*

1. Rizzoli A, Hauffe H, Carpi G, Vourc'h HG, Neteler M, Rosa R. Lyme borreliosis in Europe. Euro Surveill. 2011;16(27):19906. https://doi.org/10.2807/ese.16.27.19906-en
2. Garkowski A, Łebkowska U, Kubas B, Garkowska E, Rutka K, Gawarecka E., Zajkowska J. Imaging of Lyme neuroborreliosis: a pictorial review. Open Forum Infect Dis. 2020;7(10):ofaa370. https://doi.org/10.1093/ofid/ofaa370
3. Rupprecht TA, Koedel U, Fingerle V, Pfister HW. The pathogenesis of Lyme neuroborreliosis: from infection to inflammation. Mol Med. 2008;14(3–4):205–212. https://doi.org/10.2119/2007-00091.Rupprecht
4. Mygland A, Ljøstad U, Fingerle V, Rupprecht T, Schmutzhard E, Steiner I; European Federation of Neurological Societies. EFNS guidelines on the diagnosis and management of European Lyme neuroborreliosis. Eur J Neurol. 2010;17(1):8–16.e1–4. https://doi.org/10.1111/j.1468-1331.2009.02862.x