

Baló-Type Inflammatory Pseudotumoral Lesion Mimicking a Cerebral Metastasis: A Diagnostic Challenge

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Abstract

Baló's concentric sclerosis is a rare inflammatory demyelinating disorder characterized by concentric layers of demyelinated and relatively preserved myelin. Its clinical and radiological presentation may mimic an intracranial neoplasm, particularly when a solitary enhancing lesion is associated with diffusion restriction. We report the case of a 34-year-old woman who presented with acute right-sided motor symptoms involving the lower limb and fine movements of the right hand. Initial brain magnetic resonance imaging (MRI) revealed a solitary 15 mm left posterior frontal intra-axial lesion with T2/fluid-attenuated inversion recovery (FLAIR) hyperintensity, central T2 shine-through and peripheral diffusion restriction, intense contrast enhancement, and minimal surrounding edema. A metastatic lesion was initially considered the leading diagnosis. However, the patient experienced spontaneous clinical improvement without corticosteroid treatment. The lesion initially regressed in size and enhancement on serial MRI, with subsequent fluctuations in lesion size, while a new small left posterior subinsular lesion appeared during follow-up. Extensive oncological investigations, including thoracoabdominopelvic computed tomography, breast imaging, and dermatological examination, did not identify an underlying malignancy. Cerebrospinal fluid analysis demonstrated the presence of intrathecal oligoclonal bands. Subsequent MRI demonstrated a central vein within the initial left frontal lesion, providing an additional supportive, although nonspecific, feature of inflammatory demyelination. Retrospective MRI review also suggested a subtle, incompletely expressed concentric configuration, although the characteristic concentric-ring pattern was not clearly demonstrated. Although the clinical and radiological findings suggested an inflammatory demyelinating disorder within the multiple sclerosis spectrum, the diagnostic criteria for multiple sclerosis were not fulfilled because dissemination in space was not demonstrated. The final working diagnosis was a clinically isolated syndrome associated with a suspected Baló-type inflammatory pseudotumoral lesion, without histopathological confirmation. This case highlights the importance of serial imaging and multidisciplinary assessment when evaluating a solitary enhancing brain lesion in a young adult, particularly when spontaneous clinical and radiological improvement occurs.

Categories: Neurology, Radiology, Neurosurgery

Keywords: baló's concentric sclerosis, brain mri, central vein sign, clinically isolated syndrome, multiple sclerosis, pseudotumoral lesion, tumefactive demyelination

Introduction

Baló's concentric sclerosis is an uncommon inflammatory demyelinating disorder of the central nervous system. It is generally considered part of the spectrum of inflammatory demyelinating diseases related to multiple sclerosis, although its clinical course and imaging manifestations can be heterogeneous. The characteristic pathological pattern consists of alternating concentric layers of demyelinated and relatively preserved myelin, producing an onion-bulb or concentric-ring appearance on magnetic resonance imaging (MRI) [1-4]. However, this characteristic concentric-ring pattern may be absent or only subtly visible, making the radiological diagnosis challenging, particularly when the lesion mimics an intracranial neoplasm.

Patients may present with acute or subacute neurological symptoms, including motor deficits, cognitive disturbances, headache, or seizures [5]. In some patients, the radiological presentation is pseudotumoral, characterized by a focal enhancing lesion that may resemble a metastasis, glioma, lymphoma, abscess, or other intracranial mass [6,7]. Diffusion restriction and contrast enhancement may further complicate the differential diagnosis.

We report a case of a young woman presenting with acute right-sided motor symptoms and a solitary left frontal enhancing lesion initially considered suspicious for metastasis. The lesion initially regressed spontaneously, while a new posterior subinsular lesion appeared during follow-up. The case illustrates the diagnostic value of clinical evolution, serial MRI, cerebrospinal fluid analysis, oncological investigations, and advanced MRI biomarkers in guiding diagnostic and management decisions.

Case Presentation

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A 34-year-old woman with no significant medical history and no documented previous neurological episodes was admitted because of acute right-sided neurological symptoms. She initially experienced heaviness and impaired control of the right lower limb, followed by altered fine movements of the right hand, handwriting difficulties, and right-sided paresthesia. The initial neurological examination was largely unremarkable, and brain computed tomography showed no acute abnormalities. The initial brain MRI was performed 11 days after symptom onset. Initial laboratory investigations showed a white blood cell count of $9.79 \times 10^3/\mu\text{L}$ and a C-reactive protein level of 1.8 mg/L. Antinuclear and antineutrophil cytoplasmic antibodies were negative.

Initial brain MRI demonstrated a solitary, rounded, approximately 15 mm intra-axial lesion in the left posterior frontal region, corresponding to the left centrum semiovale. The lesion was hyperintense on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images and demonstrated heterogeneous diffusion signal, with central apparent diffusion coefficient (ADC) hyperintensity consistent with T2 shine-through and peripheral areas of true diffusion restriction. It showed intense contrast enhancement and was associated with minimal surrounding edema without significant mass effect or midline shift (Figure 1). Despite the patient's young age and absence of known malignancy, a solitary metastatic lesion was initially favored on radiological grounds, particularly because of the lesion's rounded morphology and intense contrast enhancement. However, an inflammatory demyelinating lesion remained an important differential diagnosis. Although the lesion measured only 15 mm and did not meet the conventional size threshold of 2 cm often used to define tumefactive demyelinating lesions, the term "pseudotumoral" was used to describe its tumor-like imaging appearance rather than its size.

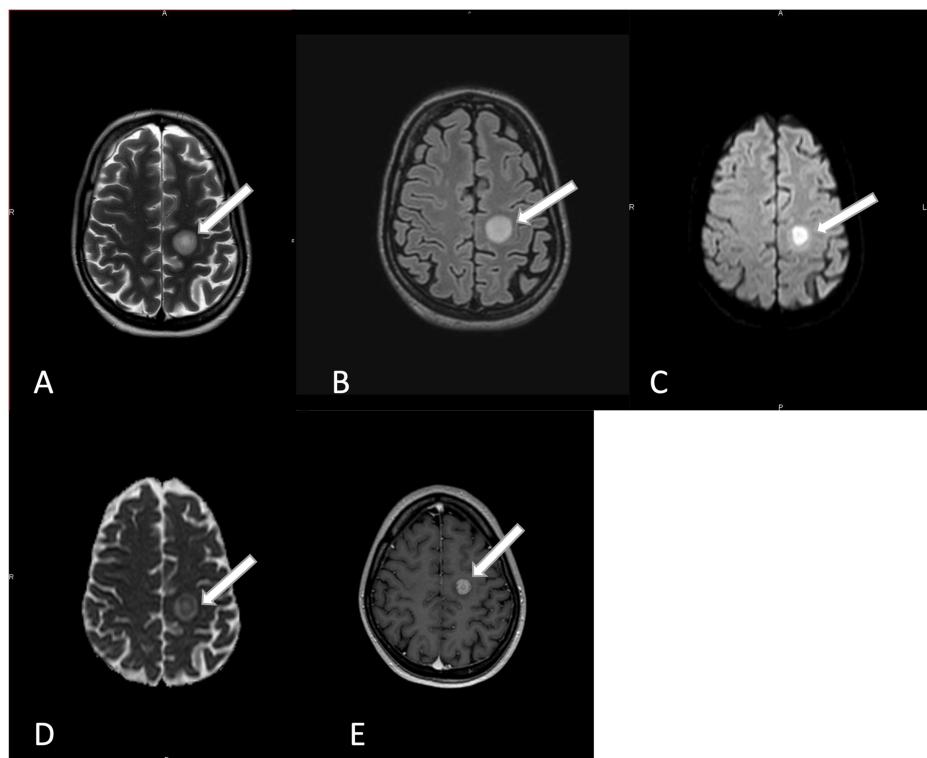


FIGURE 1: Initial brain MRI.

(A-E) Axial T2-weighted, FLAIR, diffusion-weighted, ADC, and post-gadolinium T1-weighted images demonstrating the approximately 15 mm left posterior frontal lesion, with T2/FLAIR hyperintensity, heterogeneous diffusion signal characterized by central T2 shine-through and peripheral true diffusion restriction, and intense contrast enhancement. White arrows indicate the lesion in all panels.

MRI, magnetic resonance imaging; FLAIR, fluid-attenuated inversion recovery; ADC, apparent diffusion coefficient.

The patient's neurological symptoms improved progressively without corticosteroid treatment. Following hospital discharge, residual difficulties with fine movements and coordination persisted despite overall clinical improvement. Lumbar puncture was performed three days after symptom onset, eight days before the initial brain MRI and prior to all follow-up MRI examinations. Cerebrospinal fluid analysis demonstrated intrathecal oligoclonal bands, with otherwise unremarkable findings (Table 1).

Parameter	Result
White blood cell count	2 cells/mm ³
Red blood cell count	3 cells/mm ³
Protein	28.8 mg/dL
Glucose	66.3 mg/dL
Lactate	15.6 mg/dL
Intrathecal oligoclonal bands	Present
Aerobic culture	Negative
Anaerobic culture	Negative

TABLE 1: Cerebrospinal fluid analysis.

Spinal MRI revealed no definite demyelinating lesion, and somatosensory, motor, and visual evoked potential studies were normal. An extensive oncological work-up, including thoracoabdominopelvic computed tomography, breast imaging, and dermatological examination, did not identify an underlying malignancy. Additional investigations included two brain 18F-fluoroethyl-L-tyrosine (FET) positron emission tomography (PET) examinations. Although the initial examination demonstrated tracer uptake within the frontal lesion, the subsequent examination showed a favorable evolution, supporting an inflammatory rather than neoplastic etiology.

Serial brain MRI demonstrated regression of the initial left frontal lesion. At the first follow-up examination, approximately three weeks after the initial MRI, it measured approximately 12 × 10 mm, with persistent enhancement and diffusion restriction but increased surrounding edema. At a subsequent follow-up examination approximately two months later, it had decreased to approximately 8 mm, with marked reduction in enhancement and perilesional edema (Figure 2).

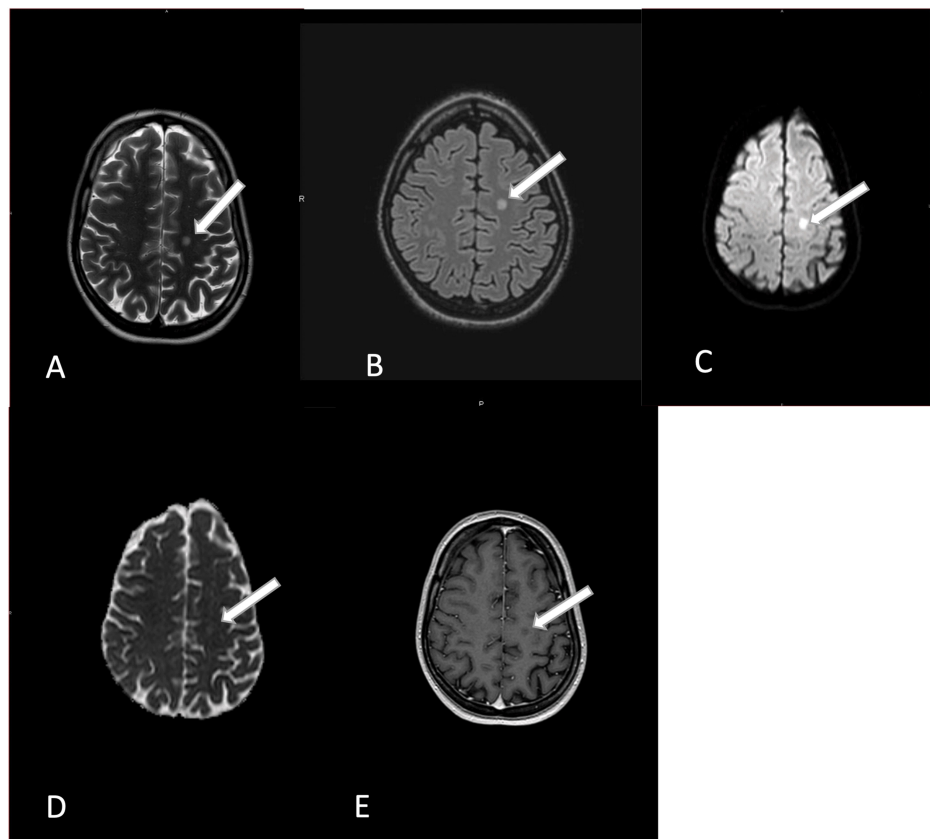


FIGURE 2: Follow-up brain MRI demonstrating spontaneous regression of the initial lesion.

(A-E) Axial T2-weighted, FLAIR, diffusion-weighted, ADC, and post-gadolinium T1-weighted images demonstrating marked spontaneous regression of the initial left frontal lesion, measuring approximately 8 mm on follow-up imaging, with substantial reduction in contrast enhancement and surrounding edema. White arrows indicate the residual lesion in all panels.

MRI, magnetic resonance imaging; FLAIR, fluid-attenuated inversion recovery; ADC, apparent diffusion coefficient.

At that time, a new small lesion was identified in the left posterior subinsular white matter, near the periventricular region. The lesion demonstrated increased signal on diffusion-weighted imaging without corresponding ADC reduction, suggesting T2 shine-through rather than true diffusion restriction (Figure 3).

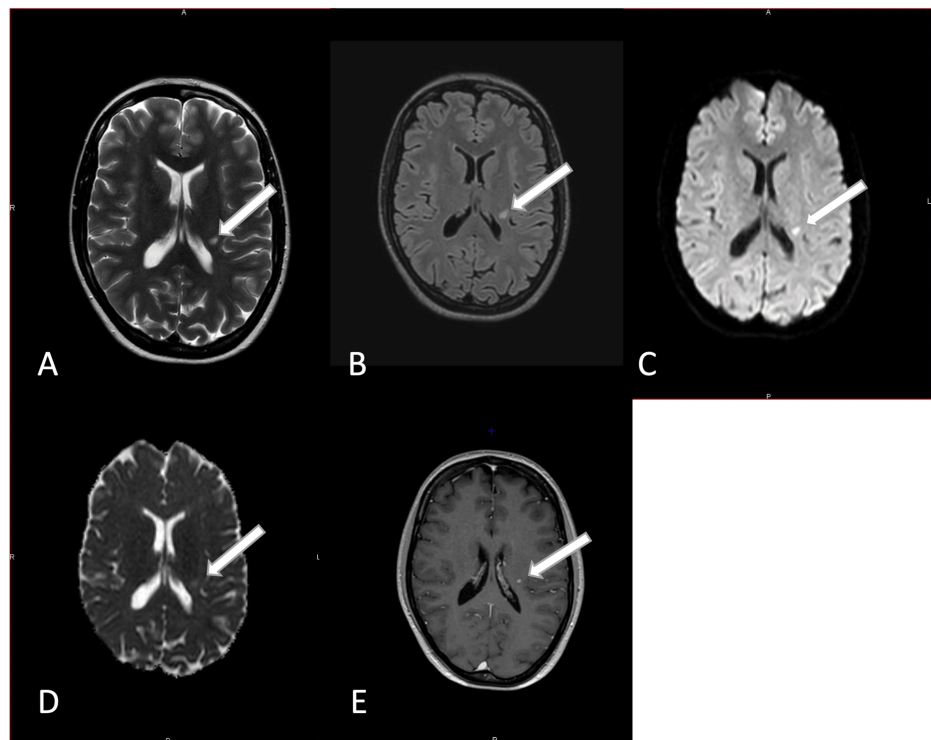


FIGURE 3: Development of a new left posterior subinsular lesion during follow-up.

(A-E) Follow-up T2-weighted, FLAIR, diffusion-weighted, ADC, and post-gadolinium T1-weighted images demonstrating the appearance of a new small lesion in the left posterior subinsular white matter during follow-up, in addition to regression of the initial lesion. The new lesion shows diffusion-weighted hyperintensity without corresponding ADC reduction, suggesting T2 shine-through rather than true diffusion restriction. White arrows indicate the new lesion in all panels.

FLAIR, fluid-attenuated inversion recovery; ADC, apparent diffusion coefficient.

No hemosiderin deposition was identified on the preceding MRI. Approximately six weeks later, a subsequent brain MRI demonstrated further evolution of the initial lesion and the new left posterior subinsular lesion. The initial lesion showed an increase in size compared with the preceding examination and mild contrast enhancement, while the second lesion showed reduced enhancement. A central vein was identified within the left frontal lesion on three-dimensional susceptibility-weighted imaging (Figure 4). This finding was considered supportive of an inflammatory demyelinating process but was not regarded as diagnostic of multiple sclerosis or Baló's concentric sclerosis. This fluctuating radiological course, characterized by initial spontaneous regression followed by transient lesion enlargement, was considered compatible with an evolving inflammatory demyelinating process, although it was not diagnostic on its own.



FIGURE 4: Retrospective MRI findings supporting an inflammatory demyelinating process.

(A-C) T2-weighted, FLAIR, and post-gadolinium T1-weighted images of the initial lesion. Retrospective review suggested a subtle, partially visible concentric or onion-bulb-like configuration. White arrows indicate the lesion in panels A and B, while the white arrow in panel C highlights the suspected concentric configuration. (D) Susceptibility-weighted imaging demonstrating a central vein within the left frontal lesion (white arrow).

MRI, magnetic resonance imaging; FLAIR, fluid-attenuated inversion recovery.

Anti-myelin oligodendrocyte glycoprotein and anti-aquaporin-4 antibodies were negative. The case was discussed by a multidisciplinary team including neurologists, neurosurgeons, neuroradiologists, and multiple sclerosis specialists. Given the lesion's location in the motor region, its spontaneous regression, the negative oncological assessment, and the potential neurological risks of biopsy or resection, brain biopsy and surgical resection were deferred, and close clinical and radiological surveillance was undertaken while further management options were discussed.

The total radiological follow-up period was approximately 11 months, from June 2025 to May 2026. Although transient enlargement of the initial lesion was observed in October 2025, subsequent brain MRI examinations performed in February and May 2026, approximately four months and three-and-a-half months later, respectively, demonstrated stable lesion burden without new lesions or evidence of active inflammation.

Following multidisciplinary discussion, disease-modifying treatment with rituximab was considered as an alternative to close clinical and radiological surveillance, given the severity of the initial neurological episode and the previously observed inflammatory activity. No new neurological symptoms were documented during subsequent follow-up, and the patient eventually achieved near-complete clinical recovery, with a normal neurological examination and an Expanded Disability Status Scale [8] score of 0. The chronological relationship between the clinical course and serial brain MRI findings is summarized in Table 2.

MRI date	Interval from previous MRI	Radiological findings	Clinical evolution
June 28, 2025	Initial MRI (11 days after symptom onset)	Solitary 15 mm left posterior frontal lesion with intense enhancement, heterogeneous diffusion signal, and minimal surrounding edema.	Acute right-sided motor and sensory symptoms.
July 17, 2025	19 days	Partial regression of the initial lesion to approximately 12 × 10 mm, with increased surrounding edema.	Progressive improvement without corticosteroid treatment; residual fine motor and coordination difficulties after discharge.
September 9, 2025	54 days (~8 weeks)	Further regression of the initial lesion to approximately 8 mm, with reduced enhancement and edema. Appearance of a new approximately 5 mm left posterior subinsular lesion.	No date-specific neurological assessment available.
October 19, 2025	40 days (~6 weeks)	Transient enlargement of the initial lesion with mild enhancement; regression of the second lesion. Central vein sign identified on SWI.	No date-specific neurological assessment available.
February 9, 2026	113 days (~4 months)	No new lesions or evidence of active inflammation.	No new neurological symptoms; normal neurological examination with an EDSS score of 0 documented during clinical follow-up.
May 27, 2026	107 days (~3.5 months)	Stable radiological findings without new lesions or active inflammation.	No date-specific neurological assessment available.

TABLE 2: Chronological correlation between clinical evolution and serial brain MRI findings.

Clinical findings are summarized according to the available medical documentation and do not necessarily correspond to neurological assessments performed on the exact MRI dates.

SWI, susceptibility-weighted imaging; EDSS, Expanded Disability Status Scale; MRI, magnetic resonance imaging.

Based on the clinical presentation, spontaneous regression of the initial pseudotumoral lesion, development of a new left posterior subinsular lesion, intrathecal oligoclonal bands, central vein sign, negative oncological work-up, and multidisciplinary assessment, the final working diagnosis was a Baló-type inflammatory pseudotumoral lesion in the context of a clinically isolated syndrome. The diagnostic criteria for multiple sclerosis were not fulfilled because dissemination in space was not demonstrated. Although cerebrospinal fluid-specific oligoclonal bands were present, the identified lesions did not meet the anatomical distribution requirements for dissemination in space. The patient was therefore considered to have a clinically isolated syndrome. No histopathological confirmation was obtained.

Discussion

Baló’s concentric sclerosis is a rare inflammatory demyelinating disorder characterized by concentric alternating layers of demyelinated and relatively preserved myelin. Although it is frequently discussed within the multiple sclerosis spectrum, its clinical and radiological presentation may be distinct. The disease can affect young adults and may present with focal neurological deficits, cognitive symptoms, headache, seizures, or a pseudotumoral syndrome [9].

The present case was challenging because the initial MRI demonstrated a solitary, intensely enhancing left frontal lesion with heterogeneous diffusion characteristics, including central T2 shine-through and peripheral true diffusion restriction. These features, particularly in the presence of a focal neurological deficit, raised concern for a neoplastic lesion. Metastasis was initially considered the leading diagnosis, despite the patient’s young age and the absence of an identified primary malignancy. Other relevant differential diagnoses included high-grade glioma, primary central nervous system lymphoma, and inflammatory demyelination [10]. Although the lesion measured only 15 mm and did not meet the conventional 2 cm threshold often used to define tumefactive demyelinating lesions, the term “pseudotumoral” was used to describe its tumor-like radiological appearance rather than its size.

Several features subsequently argued against an aggressive neoplasm. First, the patient's neurological symptoms improved spontaneously without corticosteroid therapy. Second, the initial lesion showed marked spontaneous regression in size and enhancement, followed by transient enlargement on subsequent MRI before eventual radiological stabilization. Third, the surrounding edema subsequently regressed. Finally, the appearance of a new small left posterior subinsular lesion during follow-up further supported an inflammatory demyelinating process, while extensive oncological investigations remained negative.

The MRI findings of Baló's concentric sclerosis are variable. The classical appearance consists of concentric alternating rings of abnormal and relatively preserved signal, best appreciated on T2-weighted and FLAIR sequences. Contrast enhancement and diffusion restriction may vary according to the stage of the inflammatory process. In some cases, the characteristic concentric architecture may be subtle or incompletely expressed.

In the present case, a typical concentric-ring appearance was not explicitly described in the original radiology reports. However, retrospective review of the available MRI images suggested a subtle, partially visible concentric or onion-bulb-like configuration within the initial lesion. This finding should therefore be interpreted cautiously. The working diagnosis of a Baló-type inflammatory demyelinating lesion was supported by the combination of the pseudotumoral presentation, subtle concentric features identified retrospectively, the central vein sign, spontaneous regression followed by transient lesion enlargement, the development of a new left posterior subinsular lesion, intrathecal oligoclonal bands, and the overall clinical and radiological evolution [11]. Although the overall findings strongly favored an inflammatory demyelinating process, they were not specific to Baló's concentric sclerosis. Therefore, the term "Baló-type" was used to describe the suspected radiological phenotype rather than a definitively established diagnosis, particularly given the absence of a clearly demonstrated concentric-ring pattern and histopathological confirmation.

The case also illustrates the potential role of advanced MRI biomarkers in the evaluation of suspected inflammatory demyelination. A central vein was identified within the left frontal lesion on susceptibility-weighted imaging. The central vein sign has been investigated as a marker supporting multiple sclerosis in appropriate clinical and radiological contexts [12,13]. However, the presence of a central vein is not sufficient to establish a diagnosis of multiple sclerosis or Baló's concentric sclerosis and should be interpreted only as a supportive imaging feature within the overall clinical and radiological context.

The presence of intrathecal oligoclonal bands further supported an inflammatory demyelinating process. Nevertheless, oligoclonal bands alone do not establish a diagnosis of multiple sclerosis. In this case, the diagnostic criteria for multiple sclerosis were not fulfilled because dissemination in space was not demonstrated, despite the presence of intrathecal oligoclonal bands. The patient was therefore considered to have a clinically isolated syndrome with a high suspicion of an underlying multiple sclerosis-spectrum disorder.

An important practical aspect of this case was the decision to defer an invasive diagnostic procedure. The lesion was situated in the left motor region, where biopsy or resection could have resulted in significant permanent neurological impairment. The spontaneous improvement, lesion regression, negative oncological assessment, and multidisciplinary discussion supported the decision for close surveillance rather than immediate biopsy, while maintaining diagnostic vigilance.

The main limitation of this case is the absence of histopathological confirmation. However, biopsy is not always appropriate in suspected inflammatory pseudotumoral lesions, particularly when the lesion is regressing and located in an eloquent functional region. The diagnosis in this case was based on longitudinal clinical and radiological evidence and is therefore best regarded as a suspected Baló-type inflammatory demyelinating lesion rather than a pathologically confirmed diagnosis.

Conclusions

Baló-type inflammatory demyelinating lesions may closely mimic intracranial neoplasms, particularly when the characteristic concentric-ring pattern is absent or only subtly visible. In such cases, the initial diagnosis should remain open to reassessment as new clinical, radiological, and laboratory findings become available.

A multidisciplinary approach, integrating serial MRI, complementary investigations, and careful clinical follow-up, is essential to guide diagnostic and therapeutic decisions. This approach may help prevent diagnostic errors and avoid unnecessary invasive procedures, particularly when lesions are located in eloquent brain regions. However, spontaneous lesion regression does not definitively exclude malignancy, and the need for histopathological confirmation should be reconsidered if subsequent findings raise concern.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Erika De Smet, Stephen Taylor, Corentin Delhayé

Acquisition, analysis, or interpretation of data: Erika De Smet, Stephen Taylor, Corentin Delhayé

Drafting of the manuscript: Erika De Smet, Stephen Taylor, Corentin Delhayé

Critical review of the manuscript for important intellectual content: Erika De Smet, Stephen Taylor, Corentin Delhayé

Supervision: Erika De Smet, Stephen Taylor, Corentin Delhayé

Disclosures

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