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(CASE REPORT)

Spinal ependymoma mimicking longitudinally extensive transverse myelitis: A case report

Zaid Ennasery [1,*], Salma Abouchiba [1], Moncef Ousafi [1], Hajar El Ouazzani [2], Ismail Chaouche [1], Amal Akammar [2], Meriem Haloua [2], Badreddine Alami [1], Moulay Youssef Alaoui Lamrani [1], Meryem Boubbou [2], Mustapha Maâroufi [1] and Nizar El Bouardi [1]

[1] *Department of Radiology and Interventional Imaging, CHU Hassan II, FEZ, Sidi Mohammed Ben Abdellah University, Fes, Morocco.*
[2] *Department of Radiology Mother and Child and Interventional Imaging, CHU Hassan II, FEZ, Sidi Mohammed Ben Abdellah University, Fes, Morocco.*

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Abstract

Longitudinally extensive transverse myelitis (LETM) refers to spinal cord involvement extending across three or more vertebral segments and is most commonly associated with inflammatory or infectious etiologies. Among the most frequent causes are neuromyelitis optica spectrum disorders (NMOSD) and granulomatous infections such as tuberculosis. However, although less common, certain low-grade intramedullary tumors such as spinal ependymomas may present with clinical and radiological features that mimic LETM, making diagnosis more challenging and potentially delaying appropriate management.

We report the case of a 17-year-old female admitted for ascending paralysis and respiratory distress. Initial spinal MRI revealed a cervico-thoracic intramedullary lesion hypointense on both T1- and hyperintense T2- weighted sequences, with peripheral contrast enhancement, along with an intradural extramedullary mass at the D11-D12 level, initially suggestive of an inflammatory or infectious origin. Due to the lack of clinical improvement following high-dose corticosteroid therapy and plasmapheresis, a follow-up MRI was performed. It demonstrated partial regression of the intramedullary abnormalities but a significant increase in the size of the extramedullary mass, now exerting a compressive effect on the spinal cord and suggesting secondary meningeal involvement. The lesion's well-defined and progressive radiological appearance raised suspicion for a neoplastic origin. Surgical biopsy and subsequent histopathological analysis confirmed the diagnosis of a low-grade spinal ependymoma.

This case highlights the importance of including tumoral etiologies in the differential diagnosis of LETM, particularly in cases with poor therapeutic response or atypical clinical evolution. Radiological monitoring and histopathological confirmation remain critical for establishing an accurate diagnosis and ensuring timely, appropriate treatment.

Keywords: Myelitis; Intradural extramedullary mass; MRI; LETM; Spinal ependymoma

1. Introduction

Longitudinally extensive transverse myelitis (LETM) is defined as a spinal cord lesion that spans three or more vertebral segments. Although most often linked to neuromyelitis optica spectrum disorder (NMOSD) [1], LETM is a nonspecific

* Corresponding author: Zaid Ennasery

imaging pattern observed in a wide range of infectious, autoimmune, vascular, paraneoplastic, and neoplastic conditions [2].

Among the neoplastic causes, spinal cord ependymomas, the most common intramedullary tumors in adults, also seen in children, can rarely mimic LETM on early magnetic resonance imaging (MRI) [3]. These tumors may initially appear as diffuse intramedullary T2 hyperintensity with cord swelling, without a well-defined mass, creating a risk of misdiagnosis as inflammatory myelitis [4].

Typical imaging features of ependymomas include a central location, sharp margins, homogeneous contrast enhancement, and occasional hemorrhagic foci or associated cysts [5]. Yet extensive edema, diffuse leptomeningeal enhancement, or delayed tumor demarcation can obscure these signs, especially in early or ambiguous cases [6].

When a patient is refractory to therapy and serological and cerebrospinal fluid studies remain negative, the initial diagnosis should be reconsidered. Serial imaging is then essential to uncover a hidden neoplasm [7].

We describe the case of a 17-year-old female whose presentation and first MRI suggested inflammatory LETM. Progressive imaging changes and clinical decline, however, ultimately revealed a spinal ependymoma.

2. Case presentation

A 17-year-old previously healthy woman was admitted to the emergency department with rapidly progressive neurological symptoms, including ascending paralysis and respiratory distress.

On examination, the patient was alert (Glasgow Coma Scale score 15), apyretic, and breathing spontaneously with an oxygen saturation of 96% on room air. Neurological assessment revealed generalized hypotonia, flaccid quadriplegia, abolished deep-tendon reflexes in all four limbs, and bilaterally indifferent plantar reflexes. A sensory level was identified at the T4-T5 dermatome, with impaired superficial and deep sensation in the lower limbs. Cranial-nerve examination was unremarkable. A distended bladder was palpable, consistent with neurogenic dysfunction.

Initial laboratory investigations were unremarkable. Cerebrospinal fluid analysis showed elevated protein concentration, normal glucose levels, and mild pleocytosis. Extensive infectious screening, including PCR for Mycobacterium tuberculosis and herpesviruses, was negative. Anti-aquaporin-4 antibodies were also undetectable (table 1)

Table 1 Comprehensive Laboratory Results (including CSF analysis and infectious screening) with Reference Ranges

Category	Test	Patient Result	Unit	Reference Range*	Comment / Method
Blood – Hematology	Hemoglobin	13.5	g/dL	♂ 13.0–17.0 / ♀ 12.0–15.5	
	Total leukocytes	7.2 ×10^3	/mm^3	4.0–10.0 ×10^3	Normal differential
	Neutrophils	60	%	40–75	
	Lymphocytes	30	%	20–45	
	Platelets	260 ×10^3	/mm^3	150–400 ×10^3	
Blood – Inflammation	C-reactive protein (CRP)	3	mg/L	<5	
	ESR (1 hour)	12	mm	♂ <15 / ♀ <20	
Blood – Biochemistry	Sodium	140	mmol/L	135–145	
	Potassium	4.1	mmol/L	3.5–5.1	
	Urea	4.5	mmol/L	2.5–7.1	

	Creatinine	72	µmol/L	♂ 62–106 / ♀ 44–80	
	AST (ASAT)	22	U/L	<35	
	ALT (ALAT)	24	U/L	<45	
	Alkaline phosphatase	78	U/L	40–130	
	Total bilirubin	11	µmol/L	<17	
	Serum glucose (simultaneous)	5.3	mmol/L	3.9–6.1	For CSF ratio
CSF – General Parameters	Appearance	Clear	—	Clear	
	Opening pressure	17	cm H_2O	10–20	
	Cell count	4	cells/mm^3	<5	Lymphocyte predominance
	Protein	0.85	g/L	0.15–0.45	Mildly elevated (common in spinal tumors)
	Glucose	3.2	mmol/L	2.5–4.5	CSF/serum ratio ≈ 0.60
	IgG index	0.6	—	<0.7	Normal
	Oligoclonal bands	Absent	—	Absent	Isoelectric focusing
Infectious screening	Cytology HIV Ag/Ab (4th gen) HBsAg	Negative Negative	— —	Negative Negative	
	Anti-HCV antibodies	Negative	—	Negative	
	Syphilis (VDRL/TPHA)	Negative	—	Negative	
	Quantiferon-TB Gold	Negative	—	Negative	
	PCR HSV/CMV/EBV (CSF)	Negative	—	Negative	Multiplex PCR
	Tumor markers (CEA, CA 19-9, etc.)	Not performed	—	—	Not relevant

Electromyography revealed features suggestive of myeloradiculopathy, raising the possibility of associated meningoradiculitis.

Electrophysiological testing showed a pattern compatible with myeloradiculoneuritis: motor nerve conduction studies of the lower limbs revealed reduced CMAP amplitudes, mildly slowed conduction velocities, and prolonged F-wave latencies, while sensory responses were preserved. Needle EMG demonstrated active denervation (fibrillation potentials and positive sharp waves) and reduced recruitment in muscles innervated by lumbosacral roots and paraspinal muscles, supporting the possibility of associated meningoradiculitis (table 2,3).

Table 2 Motor and Sensory Nerve Conduction Studies

Nerve (Side)	Segment	Distal Latency (ms)	Amplitude (CMAP/SNAP)	Conduction Velocity (m/s)	F-wave Latency (ms)	Reference Range	Interpretation
Tibial motor (R)	Ankle–AH	5.2	1.2 mV	38	62	DL < 6.0 ms; Amp > 4 mV; CV > 41 m/s; F < 56 ms	↓ amplitude, ↓ CV, F-wave prolonged
Peroneal motor (R)	Ankle–EDB	4.8	0.9 mV	39	61	DL < 6.0 ms; Amp > 2 mV; CV > 41 m/s; F < 56 ms	Similar abnormalities
Median motor (R)	Wrist–APB	3.5	6.0 mV	53	28	DL < 4.2 ms; Amp > 4 mV; CV > 49 m/s; F < 32 ms	Normal
Sural sensory (R)	Calf–Lat. malleolus	2.9	12 µV	48	—	DL < 3.6 ms; Amp > 6 µV; CV > 40 m/s	Normal
Superficial peroneal sensory (R)	Leg–Dorsum foot	3.1	10 µV	46	—	DL < 3.5 ms; Amp > 8 µV; CV > 40 m/s	Normal

Table 3 Needle EMG Summary

Muscle (Side)	Innervation / Root Level	Spontaneous Activity (Fibs/PSW)	Motor Unit Potentials (Duration/Amplitude)	Recruitment Pattern	Interpretation
Tibialis anterior (R)	Deep peroneal n. (L4–L5)	++	↑ duration, ↑ amplitude	Reduced / early recruitment	Active denervation & chronic reinnervation
Gastrocnemius medialis (R)	Tibial n. (S1–S2)	+	Slightly ↑ duration	Reduced	Radicular involvement
Paraspinal lumbar (R)	Dorsal rami (L4–S1)	++	N/A	N/A	Denervation at root level
Vastus medialis (R)	Femoral n. (L2–L4)	0	Normal	Normal	Proximal roots spared
First dorsal interosseous (R)	Ulnar n. (C8–T1)	0	Normal	Normal	Upper limb normal

Initial spinal MRI (Fig. 1) showed a longitudinal intramedullary lesion extending from the cervical to thoracic cord. It was isointense on T1-weighted images and hyperintense on T2-weighted sequences, with more pronounced changes at the cervical level. A central marked T2 hyperintensity created the classic "bright spotty lesion" appearance. After gadolinium injection, peripheral contrast enhancement was observed.

Dilation of the ependymal canal was noted at the thoracic level. Additionally, an intradural extramedullary lesion was identified at the D11-D12 level.

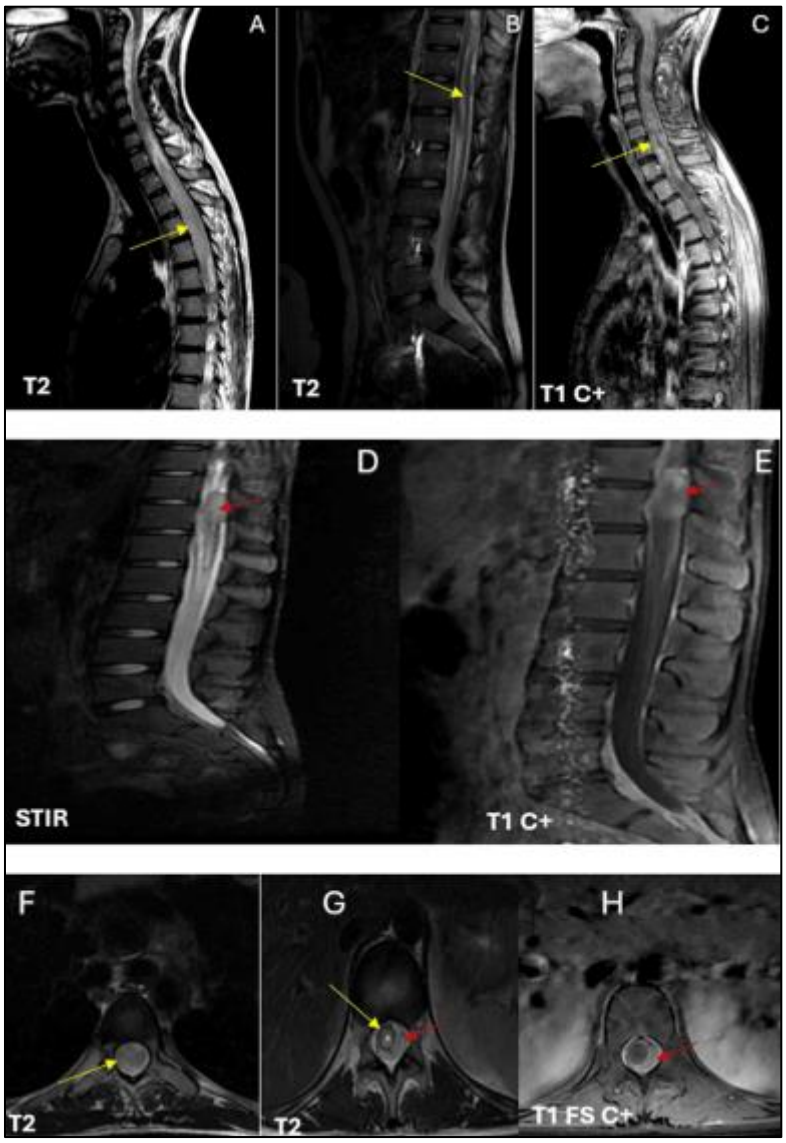

Figure 1 Initial cervico-dorso-lumbar MRI

Sagittal (A, B, C, D, E), Axial (F, G, H): Longitudinal T2 hyperintense intramedullary lesion with peripheral enhancement involving the cervico-dorsal spinal cord, associated with an enhancing intradural extramedullary pseudo-mass at the D11–D12 level.

Brain MRI (Fig. 2) revealed a focal FLAIR hyperintensity in the floor of the fourth ventricle, suggestive of bulbomedullary involvement typically seen in NMOSD.

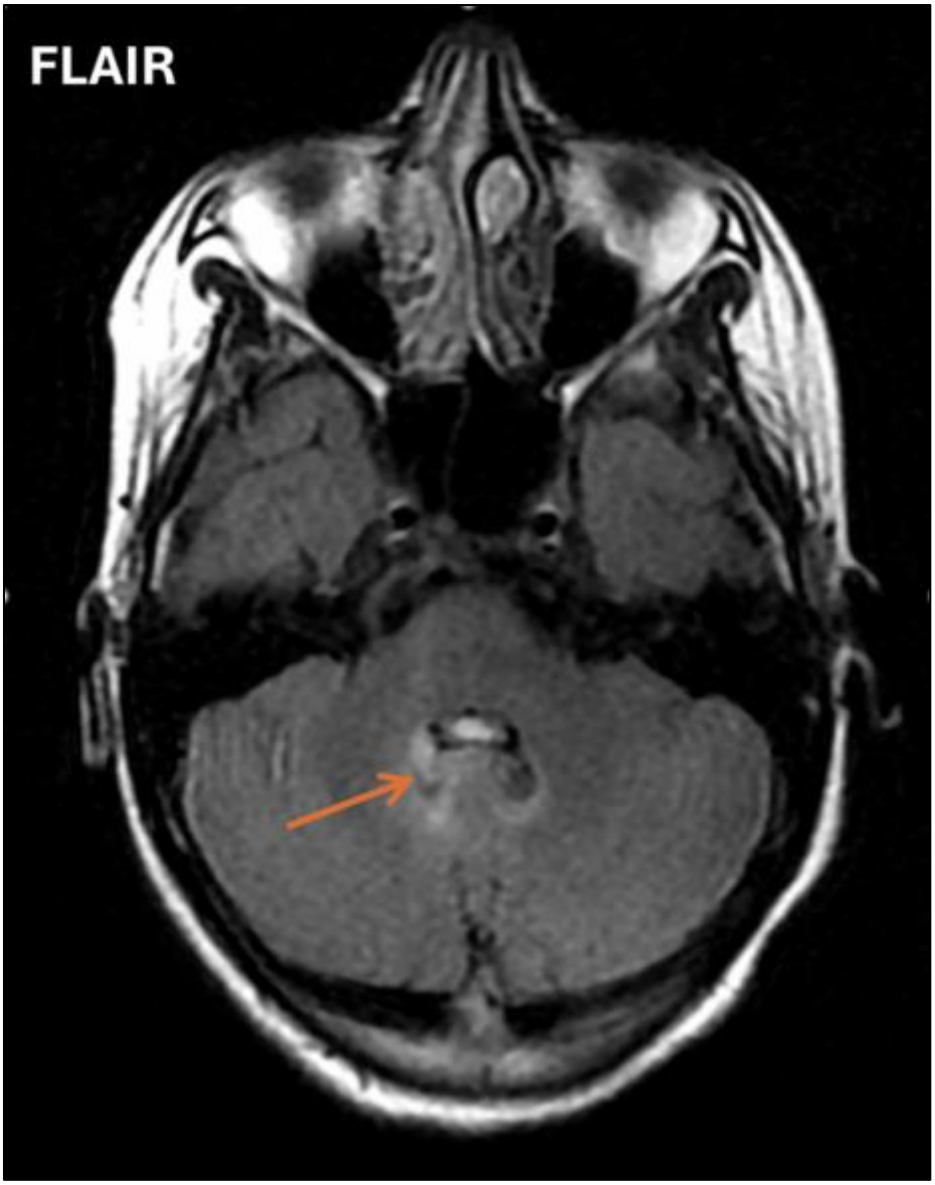

Figure 2 Initial Brain MRI

Axial: showed a focal FLAIR hyperintensity in the floor of the fourth ventricle, corresponding to the ependymal region rich in aquaporin-4 (AQP4), suggestive of bulbomedullary involvement typically observed in neuromyelitis optica spectrum disorder (NMOSD)

Given the initial suspicion of inflammatory LETM, the patient was treated with high-dose intravenous corticosteroids followed by plasmapheresis. The condition remained refractory to therapy.

Follow-up spinal MRI (Fig. 3) showed partial regression of the intramedullary changes. The D11-D12 extramedullary mass, however, had enlarged and now compressed the cord, suggesting secondary meningeal dissemination.

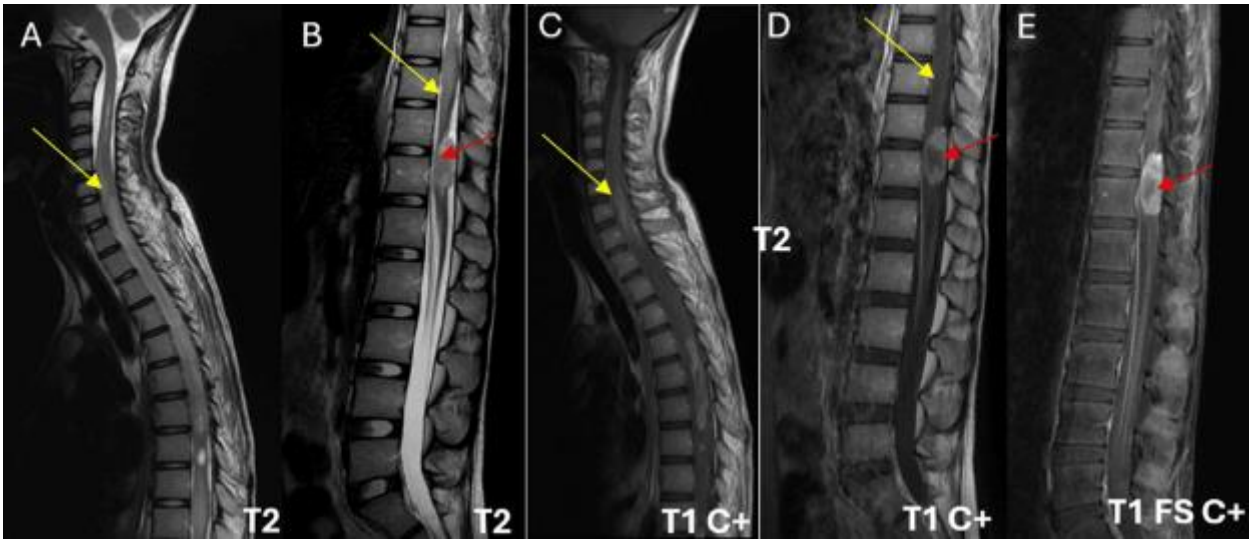

Figure 3 Follow-up cervico-dorso-lumbar MRI

Sagittal (A, B, C, D, E): Partial regression of the intramedullary T2 hyperintensity with persistent peripheral enhancement. Significant increase in the size of the well-defined, enhancing intradural extramedullary mass at D11–D12, now compressive on the spinal cord, suggestive of a low-grade glioma with secondary meningeal involvement.

A surgical biopsy was performed.Histopathological examination confirmed a low-grade spinal ependymoma. H&E sections (Fig.4) showed characteristic perivascular pseudorosettes composed of uniform cells with round to oval nuclei. A secondary dural nodule at T11-T12 displayed identical morphology, consistent with a dural metastasis. Immunohistochemistry revealed strong diffuse positivity for glial fibrillary acidic protein (GFAP), confirming the glial origin. Epithelial membrane antigen (EMA) showed focal "dot-like" staining, and the Ki-67

(MIB-1) proliferation index was low (~3%), supporting a low proliferative potential (Figure 4 and table 5).

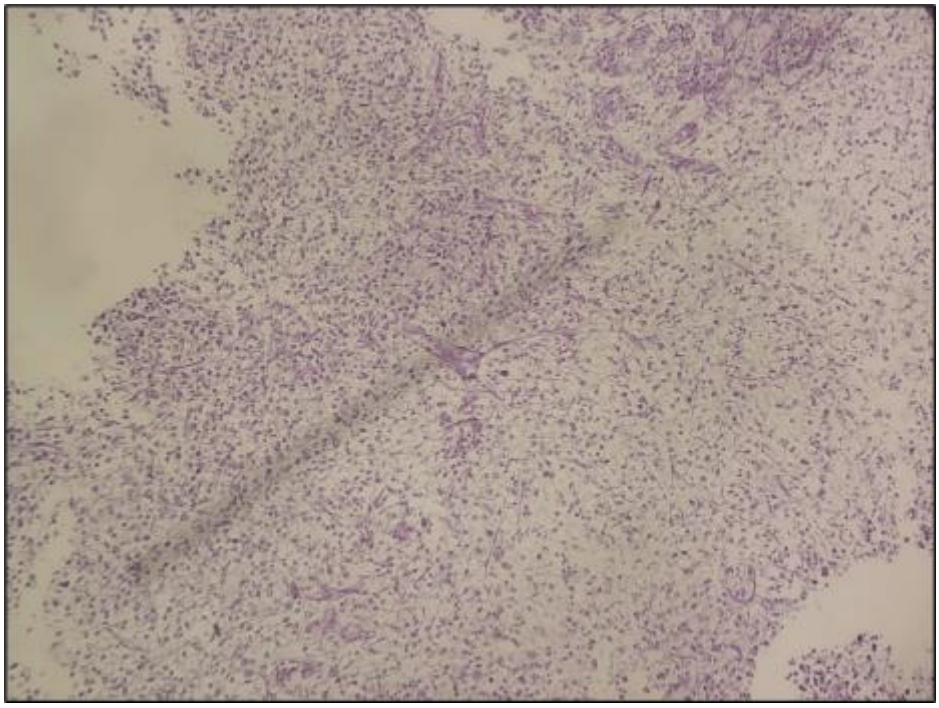

Figure 4 Tumor proliferation arranged on a fibrillary background (H&E,×100)

Table 5 Morphologic (H&E) Summary and Immunohistochemical Profile

Feature Evaluated	**Observation**	**Stain / Magnification**	**Interpretation**
Tumor architecture	Perivascular pseudorosettes, uniform round/oval nuclei	H&E, ×40 / ×200	Classic for ependymoma
Dural metastasis (T11–T12)	Dural nodule replicating primary tumor morphology	H&E	Secondary dural metastasis
Necrosis	Absent	H&E	Supports low grade
Microvasculature	Occasional hyalinized vessels; no endothelial proliferation	H&E	Compatible with low grade
Mitoses Rare H&E Low proliferative activity			
Marker	Result	Intensity / Extent	Interpretation
GFAP	Positive	+++ / >90% cells	Confirms glial origin
EMA	Focal "dot-like" positivity	+ / limited foci	Typical for ependymoma
Ki-67 (MIB-1)	~3%	Low index	Consistent with low grade
S100	Positive	++	Supports glial differentiation
OLIG2	Negative / very weak	0–/+	Often negative in ependymoma
p53	Low expression	+/−	No significant overexpression
Cytokeratins (AE1/AE3)	Negative	0	Rules out carcinoma
Synaptophysin / NeuN	Negative	0	No neuronal differentiation

Approximately two weeks after the biopsy, the patient experienced a sudden neurological decline, presenting with a Glasgow Coma Scale score of 4 and bilateral fixed, non-reactive pupils. She was intubated and transferred to the intensive care unit. The patient subsequently died, with no significant neurological recovery noted.

3. Discussion

Ependymomas are glial tumors originating from ependymal cells lining the central canal of the spinal cord. They account for approximately 30 % of intramedullary tumors across all age groups [8]. In adults, they represent the most common spinal glial tumor, accounting for up to 60 % of cases. These lesions, generally classified as grade II by the World Health Organization (WHO), are well circumscribed, slow-growing, and exhibit an expansive, non-infiltrative behavior. They respond favorably to complete surgical excision when diagnosed early [6, 9]. However, anaplastic forms (grade III) may appear, particularly in children and young adults [8].

From an epidemiological perspective, intramedullary ependymomas primarily affect adults, with a peak incidence between 30 and 50 years of age, and a slight male predominance. In children, the sex distribution is more balanced [9]. The most common locations are cervical and cervico-thoracic. The myxopapillary subtype mainly affects children and young adult males, with a preferential localization at the conus medullaris, filum terminale, or cauda equina [9].

On magnetic resonance imaging (MRI), the typical ependymoma appears as a centrally located lesion, well defined, iso- to hypointense on T1, hyperintense on T2, with homogeneous and intense enhancement after gadolinium injection. The

presence of polar cysts or microhemorrhages (cap sign) is frequently observed, facilitating diagnostic orientation [5, 9]. These characteristics generally allow differentiation of ependymomas from astrocytomas, which are often more infiltrative and eccentric [4]. However, in certain cases-particularly during early presentation, significant edema, or diffuse meningeal enhancement-these features may be obscured, making diagnosis more difficult [3, 6].

Atypical forms of ependymoma pose a real diagnostic challenge, particularly when they mimic inflammatory pathologies. Among them, leptomeningeal dissemination is an important entity. Although rare in low-grade forms, it may occur in locations near the conus medullaris or in cases of delayed diagnosis. It is observed in 10 to 15 % of ependymomas, particularly those located in the posterior fossa or lower spinal canal [4, 6, 9]. This dissemination may appear on MRI as diffuse or linear meningeal enhancement without a defined mass, thus mimicking infectious or inflammatory meningo-radiculitis. In this context, the absence of response to immunosuppressants constitutes a major warning sign [7].

Alongside this diffuse form, there is another equally misleading presentation: metastatic meningeal nodular lesions. These correspond to well-defined tumor implants in the subarachnoid space, visible on MRI as enhancing nodules-often compressive, as observed in our case. This nodular presentation, associated with a progressive extramedullary mass, fits within a picture of secondary focal meningeal dissemination and may appear late in the clinical course. These nodules can exert significant mass effect on the spinal cord, further complicating the differential diagnosis with infectious or granulomatous lesions. Their late detection constitutes a non-negligible factor in diagnostic delay [10].

The myxopapillary subtype, although classified as low-grade atypical, also presents a more aggressive behavior than expected, with a propensity for local recurrence and dissemination through cerebrospinal fluid. On MRI, it typically manifests as a lobulated extramedullary mass, isointense on T1, very hyperintense on T2, with intense post-gadolinium enhancement. This lesion may extend over several levels and infiltrate adjacent bone structures, requiring careful postoperative monitoring [9].

Certain highly aggressive forms, such as MYCN-amplified ependymomas described in children, present rapid progression and misleading MRI appearances. They may manifest as diffuse infiltrative spinal cord involvement, mimicking inflammatory conditions such as LETM. These rare entities are associated with poor prognosis and are often refractory to therapy [11].

However, in up to 11 % of LETM cases, a tumoral origin is identified, notably involving ependymomas or astrocytomas [3]. This non-negligible proportion highlights the need for increased vigilance in the presence of atypical presentations. LETM is a neuro-inflammatory syndrome defined by a continuous intramedullary lesion visible on T2-weighted MRI sequences, extending over three or more vertebral segments. These lesions are generally central, hyperintense, and sometimes associated with spinal cord swelling and gadolinium enhancement on T1-weighted images. Clinically, LETM resents with bilateral signs of acute or subacute spinal cord involvement: paresis or paralysis of the limbs, sensory disturbances (hypoesthesia, paresthesia, pain), and sphincter dysfunction (urinary retention or incontinence). The rapid onset of symptoms over a few hours to days reflects the aggressiveness of the underlying inflammatory process [1, 7].

Although classically associated with inflammatory disorders, particularly neuromyelitis optica spectrum disorder (NMOSD) [1], LETM can be the initial manifestation of a wide range of etiologies. These include autoimmune causes (lupus, Sjögren's syndrome, Behçet's disease, neurosarcoidosis), infectious agents (herpes, HIV, syphilis), vascular origins (dural arteriovenous fistulas), as well as neoplastic conditions, particularly ependymomas and astrocytomas [4].

MRI remains the central tool for diagnostic evaluation, but its interpretation must be dynamic and reassessed according to clinical evolution. T2, STIR, post-gadolinium T1 sequences, as well as diffusion (DWI) and susceptibility-weighted imaging (SWI), are essential to differentiate inflammatory etiologies from tumoral processes [12]. Nevertheless, in the absence of conclusive imaging or in case of persistent doubt, only biopsy with histopathological analysis can establish a definitive diagnosis [9, 12].

Our observation clearly illustrates this complexity. The initial imaging showed findings compatible with an inflammatory myelitis of the NMOSD type. A focal FLAIR hyperintensity was observed around the floor of the fourth ventricle (V4), suggesting brainstem involvement, typical of bulbo-medullary NMOSD. Additionally, spinal MRI revealed a T2 hyperintense signal extending over several segments, consistent with longitudinal spinal cord involvement, and associated with dilation of the ependymal canal at the thoracic level.

These radiological features are characteristic of NMOSD, particularly in the presence of anti-AQP4 antibodies. In fact, involvement of the V4 floor, a region rich in aquaporin-4, is frequently observed in this pathology. Longitudinally extensive spinal cord lesions (≥ 3 vertebral segments), central canal dilation, and brainstem involvement, especially in peri-ependymal areas, further support the diagnosis of NMOSD- associated myelitis.

The initial LETM-like presentation, absence of a visible mass, and refractory to therapy initially pointed to an inflammatory cause. Only upon repeated imaging, revealing a well-enhanced extramedullary nodular mass at D11-D12, was a tumoral hypothesis considered. This rapidly progressive evolution, with secondary appearance of a compressive meningeal nodular lesion, falls within the framework of focal secondary neoplastic dissemination, often unrecognized at an early stage.

Our case highlights the diagnostic complexity of certain LETM cases, particularly when the initial presentation suggests NMOSD, with bulbo-medullary involvement, extensive T2 hyperintensity, and central canal dilation. The rapid clinical deterioration, absence of specific antibodies, refractory to therapy, and the subsequent appearance of an extramedullary compressive meningeal nodule led to reconsideration of the initial suspected etiology.

The final diagnosis of a low-grade ependymoma with secondary nodular meningeal dissemination, an extremely rare entity, underscores the importance of including tumoral causes in the diagnostic spectrum of atypical or non-regressive LETM.

Ependymomas are primarily treated through surgical resection. Gross total resection is the mainstay of treatment for low-grade tumors and is associated with favorable outcomes when achieved. In cases where the tumor is not entirely resectable due to its location or adherence to neural structures, subtotal resection may be performed, followed by adjuvant radiotherapy. Radiotherapy is also considered in cases of recurrence or for high-grade lesions. Chemotherapy has a limited role in the management of spinal ependymomas, though it may be considered in specific pediatric or high-grade cases. Close postoperative surveillance with regular MRI follow-up is essential, especially in cases involving incomplete resection or atypical histologic features. Compared to literature recommendations [6, 9], our patient's condition was diagnosed too late to benefit from these therapeutic approaches, and her rapid deterioration prior to surgical management resulted in a fatal outcome.

The prognosis of spinal ependymomas largely depends on tumor grade, location, and extent of surgical resection. Low-grade ependymomas generally carry a good prognosis, particularly when complete excision is achieved. Myxopapillary ependymomas, while histologically low grade, have a higher risk of recurrence and cerebrospinal dissemination, which may negatively affect long-term outcomes. High-grade or anaplastic variants are associated with a significantly worse prognosis due to their aggressive nature and resistance to conventional treatments. Prognostic indicators also include patient age, tumor size, and presence of disseminated disease at diagnosis. Early detection and timely intervention are crucial in improving neurologic recovery and reducing long-term disability. Unfortunately, in our case, the diagnosis was delayed, and the rapid progression of the disease led to the patient's admission to intensive care, where she passed away, underscoring the severe prognostic consequences of late recognition [13].

In conclusion, in the presence of a rapidly progressive LETM refractory to therapy, the identification of radiological or biological warning signs should prompt diagnostic reassessment. Close MRI follow-up is essential to detect an underlying neoplastic etiology, particularly low-grade ependymomas with meningeal dissemination, for which early diagnosis directly influences neurological prognosis [3, 4, 7].

4. Conclusion

Extended transverse myelitis (LETM) remains a diagnostic challenge, particularly when clinical progression or imaging findings are atypical. In such cases, a tumor origin such as low-grade ependymoma should be considered, especially if the disease is refractory to therapy or shows atypical progression. This underscores the need to maintain a broad differential diagnosis. Although rare, secondary meningeal dissemination is a potential presentation that must be recognized. Close, ongoing clinical and radiological follow-up is essential to avoid diagnostic delays and improve outcomes by enabling timely intervention in complex cases.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

If studies involve use of animal/human subject, authors must give appropriate statement of ethical approval. If not applicable then mention 'The present research work does not contain any studies performed on animals/humans subjects by any of the authors'.

Statement of informed consent

"Informed consent was obtained from all individual participants included in the study."

References

[1] Wingerchuk DM, Banwell B, Bennett JL, et al.: International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. Neurology. 2015, 14:177-189. 10.1212/WNL.0000000000001729

[2] Trebst C, Raab P, Voss EV, et al.: Longitudinal extensive transverse myelitis—it's not all neuromyelitis optica. Nat Rev Neurol. 2011, 7:688-698. 10.1038/nrneurol.2011.176

[3] Nouvakis D, Natsis KS, Papamichalis E: Spinal cord ependymoma presenting as longitudinally extensive transverse myelitis: a case report. Neurol Disord Ther. Neurological Disorders and Therapeutics . 2017, 1:1- 5. 10.15761/NDT.1000124

[4] Bou-Haidar P, Peduto AJ, Karunaratne N: Differential diagnosis of T2 hyperintense spinal cord lesions . Part B. J Med Imaging Radiat Oncol. 2009, 53:152-159. 10.1111/j.1754-9485.2009.02067.x

[5] Carnero Contentti E, Hryb JP, Leguizamón F, et al.: Differential diagnosis and prognosis for longitudinally extensive myelitis in Buenos Aires, Argentina. Neurologia. 2017, 32:99-105. 10.1016/j.nrl.2015.06.013.

[6] Klekamp J: Treatment of intramedullary tumors: analysis of surgical morbidity and long-term results . J Neurosurg Spine. 2013, 19:12-26. 10.3171/2013.3.SPINE121063

[7] Jacob A, Weinshenker BG: An approach to the diagnosis of acute transverse myelitis . Semin Neurol. 2008, 28:105-120. 10.1055/s-2007-1019132

[8] Shatara M, Schieffer KM, Klawinski D, et al.: Clinically aggressive pediatric spinal ependymoma with novel MYC amplification demonstrates molecular and histopathologic similarity to MYCN-amplified spinal ependymomas. Acta Neuropathol Commun. 2021, 11:192. 10.1186/s40478-021-01296-2

[9] Shih RY, Koeller KK: Intramedullary masses of the spinal cord: radiologic-pathologic correlation . Radiographics. 2020, 40:1125-1145. 10.1148/rg.2020190196

[10] Ghasemi DR, Sill M, Okonechnikov K, et al.: MYCN amplification drives an aggressive form of spinal ependymoma. Acta Neuropathol. 2019, 138:1075-1089. 10.1007/s00401-019-02056-2

[11] Gemici Yİ, Çelik F, Göz ZZ, et al.: Anaplastic ependymoma and atypical refractory longitudinal expansive transverse myelitis due to immune reaction after COVID- 19: a case discussion that raises many unknown questions about COVID-19. Noro Psikiyatr Ars. 2023, 21:370-373. 10.29399/npa.28464

[12] Do-Dai DD, Brooks MK, Goldkamp A, Erbay S, Bhadelia RA: Magnetic resonance imaging of intramedullary spinal cord lesions: a pictorial review. Curr Probl Diagn Radiol. 2010, 39:160-185. 10.1067/j.cpradiol.2009.05.004

[13] Raffeld M, Abdullaev Z, Pack SD, et al.: High-level MYCN amplification and distinct methylation signature define an aggressive subtype of spinal cord ependymoma. Acta Neuropathol Commun. 2020, 8:101-10. 10.1186/s40478-020-00973-y