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Longitudinally extensive transverse myelitis as the first manifestation of probable neurosarcoidosis - case report

Paula Olszewska[1*], Maciej Olszewski[2], Oskar Bożek[3], Łukasz Mazurek[4], Tomasz Paleczny[5], Krzysztof Wierzbicki[2]

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ABSTRACT

Background: Sarcoidosis, as a rare disease, affects 0.01% of the population; involvement of the central nervous system (CNS) is even less common.

Case presentation: A 46-year-old man suffering from ulcerative colitis and epilepsy was hospitalized in the neurology department due to progressive, ascending paresthesia and quadriplegia accompanied by sphincter and balance disorders. Magnetic resonance imaging revealed extensive edematous changes extending from the medulla oblongata to the Th7 level and multiple enlarged mediastinal lymph nodes. Diagnostic workup was expanded to include a contrast-enhanced computed tomography scan of the chest, which revealed numerous enlarged lymph nodes in the mediastinum and scattered small nodular lesions in the lung parenchyma. After performing a histopathological examination of the subcarinal lymph node biopsy material, a diagnosis of sarcoidosis was made. The patient was diagnosed with longitudinal extensive transverse myelitis in the course of probable neurosarcoidosis and was treated showed clinical improvement following treatment.

Conclusion: Although CNS involvement is not a common manifestation of this disease, immediate medical attention is necessary if it occurs. The management of patients with neurosarcoidosis requires a multidisciplinary approach, which improves the patient's chances of achieving long-term remission.

Keywords: case report, LETM, neurosarcoidosis, sarcoidosis, ulcerative colitis.

Type of Article: CASE REPORT **Specialty:** Neurology

Correspondence to: Paula Olszewska
*Department of Allergology and Clinical Immunology, Medical University of Silesia, Katowice, Poland.
E-mail: paula.olszewska10@gmail.com
Full list of author information is available at the end of the article.

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Background

Sarcoidosis is a multisystemic immune-mediated granulomatous disease. Its etiology is not yet fully understood [1]. This disease is characterized by granulomatous inflammation of the affected organs, most commonly the lungs (90%), skin (15%), eyes (10%-30%), liver (20%-30%), lymph nodes (10%-20%), or, much less frequently, the nervous system (5%-15%) [2,3]. It affects young adults (with peak incidence in the third and fourth decades of life) and occurs at a rate of approximately 10-20 per 100,000 people [1,2].

A postmortem study of patients published in 2017 indicates that 34% of patients with systemic sarcoidosis also had neurosarcoidosis (NS) [3]. Sarcoidosis can affect the spinal cord through several mechanisms, including infiltration of the spinal cord parenchyma, meninges, epidural space, or extramedullary tissues, compressing the spinal cord [4,5].

A definitive diagnosis of NS is made in a small percentage of patients, because it requires histological confirmation of non-caseating granulomas in the affected nervous system tissue. A probable diagnosis is defined by evidence of nervous system inflammation on magnetic resonance imaging (MRI) (trident sign on axial images) or in cerebrospinal fluid (no specific markers for the onset of NS have been identified, but in the majority of patients, cerebrospinal fluid analysis reveals pleocytosis and elevated protein levels) combined with evidence of systemic sarcoidosis. Possible NS is defined by clinical suspicion and the exclusion of other diagnoses, but without meeting the aforementioned criteria [3–5].

MRI is the method of choice when a central nervous system (CNS) - occupying lesion is suspected. Gadolinium enhancement is not sufficiently specific for NS, but it is a helpful marker of active sarcoidosis in the CNS, has value as a biomarker of treatment response, and is crucial for clinical decision-making [6–8]. **Longitudinally extensive transverse myelitis (**LETM) diagnosis on MRI is supported by a submedullary gadolinium enhancement pattern involving ≥2 spinal segments and persistence of

enhancement for >2 months despite treatment. In patients with subacute myelitis, central canal enhancement and submedullary enhancements resembling a triangle on axial spinal cord MRI should raise suspicion of NS [4–7].

Case presentation

A 46-year-old man with a history of ulcerative colitis (UC) (treated with vedolizumab) and epilepsy was hospitalized in September 2024 in the neurology department of another hospital due to progressive, ascending paresthesia and quadriplegia accompanied by sphincter and balance disorders. Upon admission, a neurological examination revealed quadriplegia, a wide-based, ataxic gait, quadriparetic ataxia predominantly affecting the upper extremities, diminished superficial sensation in the lower and upper extremities and the trunk, and urinary dysfunction. Laboratory tests did not reveal any inflammatory parameters (no leukocytosis, normal CRP). Viral diseases and syphilis infection were excluded (HIV antigen, HBS antigen - negative, anti-HCV antibodies, antibodies against Treponema pallidum - negative). Cerebrospinal fluid analysis (CSF) revealed glucose 2,5 mmol/l (serum glucose level was not determined), cytosis (253 cells/µl; 99% mononuclears - monocytes and lymphocytes, 1% polymorphonuclears - neutrophils and eosinophils) and elevated protein concentration (216 mg/dl). Contrast enhanced magnetic resonance imaging (MRI) of the brain and spinal cord in the cervical (C) and thoracic (Th) segments revealed spinal cord edema extending from the medulla oblongata to the Th 7 segment. Additionally, the spinal cord enlarged by edema caused compression of the spinal cord in the cervical segment (Figure 1).

After intravenous contrast administration, there was strong enhancement in the posterior portion of the cord involving the posterior funiculus bilaterally, extending approximately 90 mm to the C2 to C6 levels (Figure 2).

Edematous or demyelinating changes throughout the entire spinal cord, extending from the Th1 to the Th7 level, encompassing the entire mid-spinal cord. Below, the areas were heterogeneous, partially involving the cord. The signal intensity of the gray and white matter of the brain was normal. Differential diagnosis of the spinal cord lesions included Guillain-Barré syndrome, Lyme disease, and NS.

Based on the above findings, a diagnosis of unspecified encephalomyelitis was made. The patient was initially treated with systemic steroid therapy (cumulative dose of methylprednisolone – 3 g). Due to a slow response to glucocorticoid therapy, the patient's treatment was modified to include immunoglobulins (human nonspecific immunoglobulin - 30 g per day for 5 days, cumulative dose 150 g). As a continuation of treatment, chronic oral

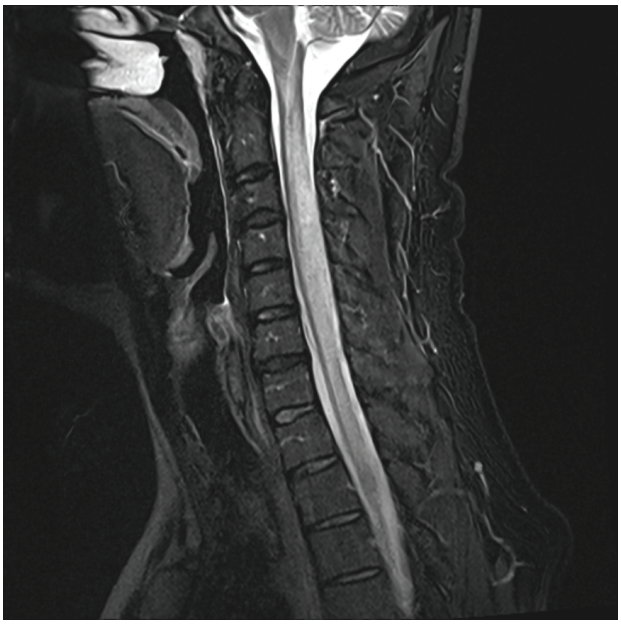


Figure 1. *Sagittal section of spinal cord edema extending from the medulla oblongata to Th segments.*

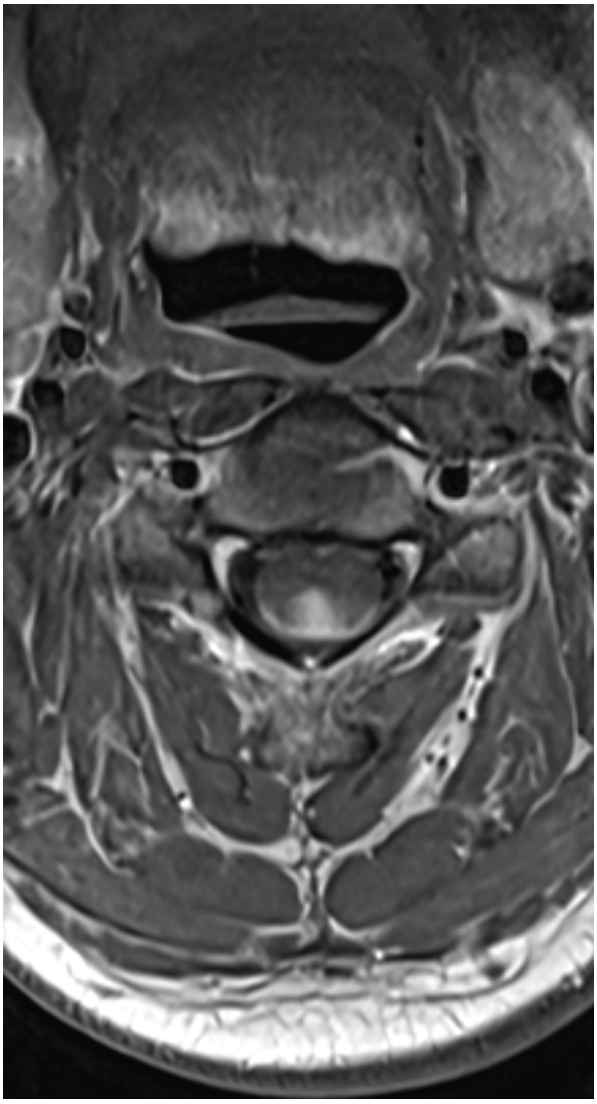


Figure 2. *A. cross-section of C spine, trident sign visible on an axial scan after gadolinium enhancement,*

steroid therapy (10 mg prednisone in gradually reduced doses for 1 month) was recommended. The patient underwent neurological rehabilitation and was discharged with a significant improvement in his clinical condition. The micturition disturbances resolved, the ataxic symptoms diminished, and he was moving more efficiently.

In January 2026, the patient was admitted to the Neurology Department of the Silesian Hospital in Cieszyn for further investigation of the etiology of the previously described inflammation. A neurological examination revealed exotropia of the left eye (a persistent symptom, post-corrective surgery), quadriparesis predominantly affecting the lower limbs, and hypoesthesia in the forearms and from the level of the costal arch (Th7). The patient underwent numerous laboratory tests, including virological tests (HIV, HCV, HBV serum levels, multiplex PCR from cerebrospinal fluid - negative) ruling out bacterial diseases (syphilis - VDRL, tuberculosis - TB-IGRA, Lyme disease - intrathecal antibody production negative), systemic diseases (antinuclear antibodies present, cytoplasmic fluorescence type 1:100, granular 1:320, anti-dsDNA - negative). Calcium and vitamin D serum levels were normal. Tumor markers (Ca-15.3, Ca-19.9, CEA) were negative; anti-GAD, antibodies against antineuronal diseases (anti-NMDA, anti-AMPA1, anti-AMPA2, anti-GABAR B1/B2, anti-LGI1, anti-CASPR2), anti-MOG and anti-aquaporin 4 antibodies detected by the cell-based assay were absent. A repeat lumbar puncture revealed normalized cytosis (5 cells/ul), normal glucose level (49 mg/dl, serum level 88 mg/dl), and a slightly elevated protein concentration (63.2 mg/dl). CSF culture revealed contamination with saprophytic bacteria (*Staphylococcus hominis*).

Contrast-enhanced head MRI did not reveal inflammatory lesions. The contrast-enhanced MRI of C and Th spine revealed thoracic cord with a subtle zone of increased signal in T2 and STIR sequences extending dorsally from the Th3 to Th7 level. A similar zone was observed at the Th10 to Th12 levels. Additionally, MRI revealed multiple enlarged mediastinal lymph nodes on the coronal and transverse scans. At the suggestion of the referring radiologist, a contrast-enchanced chest computed tomography (CT) scan was performed. It revealed numerous enlarged lymph nodes in mediastinum (Figure 3).

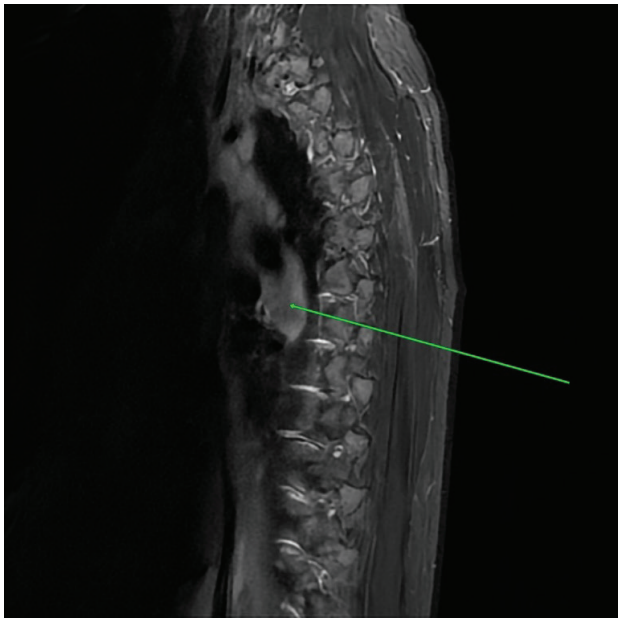
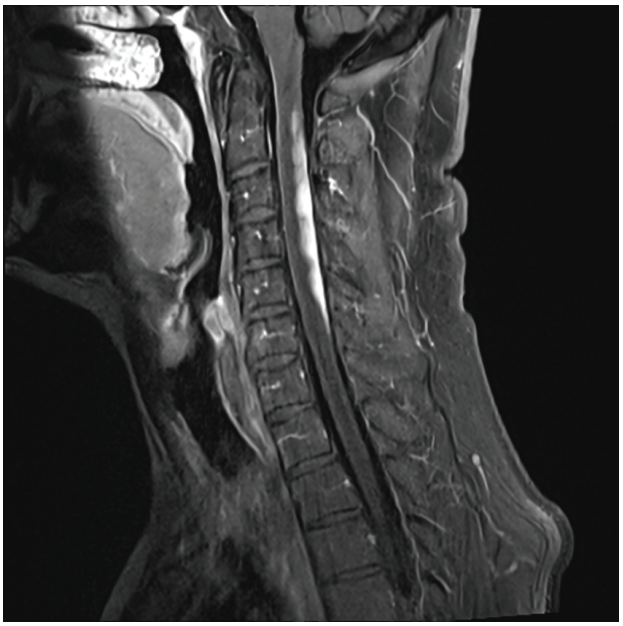
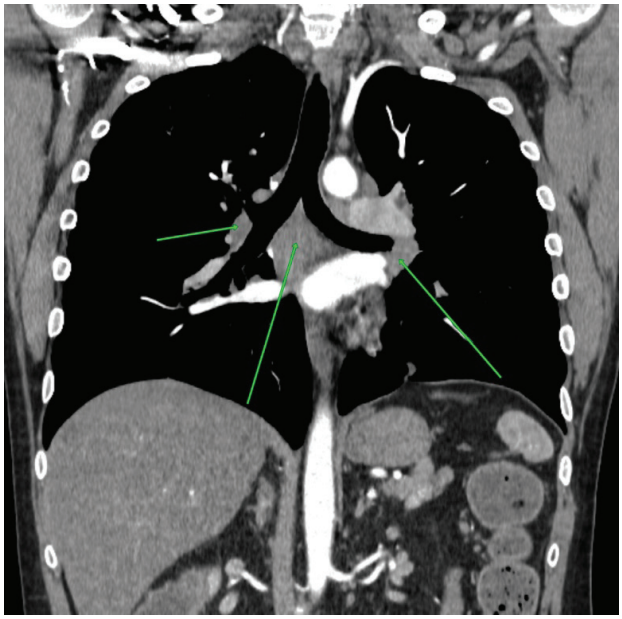


Figure 3. *A. sagittal section of Th spine, MRI images of enlarged mediastinal lymph nodes*



B. sagittal section of C spine, strong enhancement in the posterior portion of the cord after contrast administration.



B. longitudinal section of thoracic cavity, CT scan images of numerous enlarged lymph nodes in mediastinum

The findings, in conjunction with the clinical presentation, were consistent with changes typical of sarcoidosis. For this reason, the patient continued diagnostic evaluation at the Pulmonary Diseases Department of the Silesian Hospital in Cieszyn. During the stay, a lung X-ray examination and numerous lung function tests were performed (*transfer factor of the lung for carbon monoxide*, spirometry, and whole-body plethysmography), which were normal. During a bronchoscopy combined with endobronchial ultrasound, a subcarinal lymph node was biopsied and subjected to histopathological examination. Acid-fast bacilli smear and periodic acid-Schiff staining were negative. Examination of the obtained tissue revealed small lymphocytic infiltrates and small granulomas composed of epithelioid histiocytes. Immunochemical testing revealed CK (−), CD68 (+), and LCA (+) in these granulomas - which indicates that the image may be consistent with sarcoidosis. The patient also underwent an ophthalmological consultation - no ophthalmological complications associated with sarcoidosis were found. The neurological disorders described upon admission persisted. Based on the comprehensive diagnostic workup, the patient was diagnosed with LETM in the context of probable NS.

The patient was then referred to a gastroenterologist to determine further treatment for UC. Due to the coexistence of UC and sarcoidosis, a history of glucocorticoid intolerance, and hepatitis following azathioprine use, the patient was most likely considered for treatment with one of the tumor necrosis factor (TNF) inhibitors.

The patient currently resides in Spain, where he and his family have moved. Since May 2026, he is being treated with infliximab with good results. Sensory disturbances in his limbs persist, and neuropathic pain occasionally develops.

Discussion

According to Clinical Criteria for a Diagnosis of Neurosarcoidosis [8], the described patient meets the criteria for probable NS (clinical presentation and diagnostic evaluation suggest NS - MRI and CT findings typical for granulomatous inflammation after exclusion of other causes; pathologic confirmation of systemic granulomatous disease).

When choosing a course of treatment for NS, it is important to assess the benefit–risk ratio and consult with specialists in other fields to ensure that treatment addresses sarcoidosis involving other organs [7]. In more severe cases, when there is an incomplete response to GCS treatment or when there are specific concerns about adverse effects, early use of TNF inhibitors is being considered [4]. Among the TNF inhibitors available in Poland, sarcoidosis can be treated with infliximab or adalimumab [9,10]. Current ECCO guidelines recommend anti-TNF drugs for inducing remission in moderate to severe UC [9,10]. In sarcoidosis, TNF-α also plays a crucial role in the formation and maintenance of granulomas, so inhibiting TNF-α may lead to resolution of the granulomatous process [9].

Monitoring the response to treatment includes a medical history, physical examination, and typically MRI scans with and without contrast, which can detect worsening or recurrence of the disease before clinical symptoms appear [4,7,11].

It is possible that the disease may resolve, and some patients may be able to discontinue treatment, but there is a high (72%) risk of relapse after stopping therapy [4,7].

Conclusion

A broad differential diagnosis is important in the diagnosis of inflammatory lesions in the CNS. Rare diseases, such as sarcoidosis, should also be considered. Making an accurate diagnosis is crucial, primarily to enable the initiation of targeted treatment while also taking into account any co-existing conditions.

Acknowledgment

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Whats new?

NS **is a rare complication of sarcoidosis. It is particularly important to note that neurological symptoms are extremely rare as the initial manifestation of sarcoidosis and require prompt and targeted intervention to limit granulomatous inflammation.**

List of Abbreviations

Anti-AMPA1	Anti-AMPA receptor 1 antibodies
Anti-AMPA2	Anti-AMPA receptor 2 antibodies
Anti-CASPR	Anti-contactin-associated protein like 2 antibodies,
Anti-dsDNA	Anti-double-stranded DNA antibodies
Anti-GABA-RB1/B2	Anti-GABA-B receptor B1/B2 antibodies
Anti-GAD	Anti-glutamic acid decarboxylase antibodies
Anti-LGI1	Anti-leucine-rich-glioma-inactivated 1 antibodies
Anti-MOG	Anti-mielin oligodendrocyte glycoprotein antibodies
Anti-NMDA	Anti-N-methyl-D-aspartate receptor antibodies
C	Cervical
CD68	Cluster of differentiation 68
CEA	Carcinoembryonic antigen
Ca 15.3	Cancer marker 15.3
Ca 19.9	Cancer marker 19.9
CSF	Cerebrospinal fluid
CT	Computed tomography
HbsAg	Hepatitis B antigen
HCV	Hepatitis C virus
HIV	Human immunodeficiency virus
LCA	Leukocyte common antigen
LETM	Longitudinally extensive transverse myelitis
MRI	Magnetic resonance imaging
NS	Neurosarcoidosis

TB-IGRA	Tuberculosis interferon-gamma release assay
Th	Thoracic
TNF	Tumor necrosis factor
UC	Ulcerative colitis
VDRL	Venereal disease research laboratory

Conflict of interest
The authors declare that there is no conflict of interest regarding the publication of this article.

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Consent for publication
Written consent was obtained from the patient for the case report and the inclusion of medical imaging.

Ethical Approval
Ethical approval is not required for anonymous case reports.

Author details
Paula Olszewska[1], Maciej Olszewski[2], Oskar Bożek[3], Łukasz Mazurek[4], Tomasz Paleczny[5], Krzysztof Wierzbicki[2]
1. Department of Allergology and Clinical Immunology, Medical University of Silesia, Katowice, Poland
2. Department of Neurology, Silesian Hospital in Cieszyn, Cieszyn, Poland
3. Department of Radiology and Nuclear Medicine, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland
4. Tomograf Sp. z o.o. Independent Diagnostic Imaging Unit
5. Diagnostic Imaging Department, Silesian Hospital in Cieszyn, Cieszyn, Poland

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Summary of the case

1	Patient (gender, age)	46-years-old male
2	Final Diagnosis	LETM
3	Symptoms	progressive, ascending paresthesia and quadriplegia accompanied by sphincter and balance disorders
4	Medications	GCS, IVIG, vedolizumab, TNF
5	Clinical Procedure	Diagnostic imaging, pharmacotherapy, biopsy
6	Specialty	Neurology, gastroenterology, pulmonology