EJCRIM
European Journal of Case Reports in Internal Medicine

SEVERE CENTRAL DYSPHAGIA AS THE INITIAL PRESENTATION OF NEUROSARCOIDOSIS

Inês Domingues[1], Beatriz Mata Santos[2], Ana Maria Teodoro[1], Cláudia Nunes[1], Patrícia Ramos Santos[1], Fábio Pé D'Arca Barbosa[1], Vanda Spencer[1]

[1] Department of Internal Medicine, ULSAS – Hospital Garcia de Orta, Almada, Portugal
[2] Department of Rheumatology, Instituto Português Reumatologia, Lisbon, Portugal

Corresponding author's e-mail: ines.tdomingues@gmail.com

Received: 29/10/2026
Accepted: 06/11/2026
Published: 27/11/2026

Conflicts of Interests: The Authors declare no conflict of interest.
Patient Consent: A written informed and signed consent was previously obtained from the patient to secure permission for publishing his clinical history.
This article is licensed under a CC Attribution-NonCommercial-NoDerivatives 4.0 License

How to cite this article: Domingues I, Mata Santos B, Teodoro AM, Nunes C, Ramos Santos P, D'Arca Barbosa FP, Spencer V. Severe central dysphagia as the initial presentation of neurosarcoidosis. EJCRIM 2026;13:doi:10.12890/2026_007642

ABSTRACT

Introduction: Sarcoidosis is a multisystem inflammatory disorder characterized by non-caseating granulomas, with neurological involvement in 5–10% of cases. Brainstem involvement presenting as severe progressive dysphagia is exceptionally rare and can mimic neoplastic, infectious, or neuroinflammatory conditions.

Case description: A 47-year-old male presented with a six-week history of severe progressive dysphagia requiring nasogastric tube feeding. Additional symptoms included left peripheral facial palsy, dysarthria, limb paraesthesia and constitutional symptoms. Upper endoscopy excluded mechanical obstruction. Laboratory investigations revealed elevated erythrocyte sedimentation rate and angiotensin-converting enzyme levels. Magnetic resonance imaging of the brain revealed parenchymal and perivascular inflammation with pontine lesions and bilateral facial nerve enhancement, while cerebrospinal fluid analysis was normal. Thoracic imaging identified hilar lymphadenopathy. A biopsy of the hilar mass confirmed non-caseating granulomatous lymphadenitis. Infectious and malignant causes were excluded. Based on the clinical, radiological, and histological findings, a diagnosis of neurosarcoidosis with bulbar involvement was established. The patient was treated with high-dose glucocorticoids, leading to significant clinical improvement and resolution of dysphagia.

Discussion: Severe dysphagia may represent a rare initial manifestation of neurosarcoidosis. Maintaining clinical suspicion in patients with unexplained neurological symptoms and systemic involvement is essential. Comprehensive investigation, including systemic imaging and histological confirmation, enables timely treatment to prevent irreversible neurological sequelae.

Conclusion: This case highlights the importance of considering neurosarcoidosis in severe neurological dysphagia, emphasizing thorough clinical history and screening for systemic manifestations to ensure timely diagnosis and treatment.

KEYWORDS

Dysphagia, sarcoidosis, neurosarcoidosis, cranial neuropathy

EFIM
European Federation of Internal Medicine

Publisher
smc media srl

LEARNING POINTS

- Broaden the differential for dysphagia: While rare, neurosarcoidosis should be considered in the differential diagnosis of unexplained severe dysphagia, particularly when accompanied by other cranial neuropathies such as facial paralysis.
- A diagnosis of exclusion not to miss: Neurosarcoidosis is a diagnosis of exclusion and may present with normal cerebrospinal fluid findings requiring integration of neurological imaging, systemic evaluation and histological confirmation.
- The importance of early diagnosis on clinical outcome: Early diagnosis of neurosarcoidosis and prompt corticosteroid therapy are crucial to prevent irreversible neurological damage and improve functional recovery.

INTRODUCTION

Sarcoidosis is a multisystemic inflammatory disease of unknown aetiology, characterised by the formation of non-caseating granulomas[1,2]. Although it predominantly affects the respiratory system, neurological involvement occurs in approximately 5% to 10% of cases[2]. Diagnosing neurosarcoidosis (NS) remains a challenge for the clinician, particularly when neurological symptoms are the first or only manifestation of the disease, which occurs in nearly 50% of patients and often simulating other conditions such as infection or malignancy[2-4]. Although cranial neuropathies may occur, primarily affecting the facial (VII) or optic (II) nerves, involvement of the lower cranial nerves (IX, X, XII) or the brainstem nuclei is less frequent and bulbar dysfunction remains exceedingly rare[1,2,4].

This case report describes a 47-year-old male with severe and progressive dysphagia as a presenting symptom of neurosarcoidosis, a manifestation that is sparsely documented in the literature[1]. Furthermore, by integrating neurological findings with systemic screening, this case underscores the importance of a multidisciplinary internal medicine approach in reaching a 'probable' diagnosis when direct neural biopsy is not feasible[3].

CASE DESCRIPTION

A 47-year-old male with a medical history significant for hypertension and childhood poliomyelitis with right lower limb sequelae, was admitted with a six-week history of severe, progressive dysphagia for both solids and liquids. The condition had deteriorated to the point of requiring a nasogastric tube for nutritional support. Concomitantly, the patient reported a dry cough, low-grade fever, malaise and significant unintentional weight loss. Notably, he had been diagnosed with a left peripheral facial paralysis one month prior to the onset of swallowing difficulties. Upon admission, the physical examination revealed malnutrition and dehydration. Neurological assessment confirmed a left facial nerve palsy (House-Brackmann grade V) and non-specific limb paraesthesia. There were no palpable peripheral adenopathy or skin lesions. The diagnostic pathway initially focused on excluding a mechanical cause, so an upper gastrointestinal endoscopy was performed and ruled out mechanical obstruction or significant oesophageal pathology. The search for an aetiology to explain the severe dysphagia led to a contrast-enhanced brain magnetic resonance imaging (MRI) that revealed evidence of parenchymal and perivascular enhancement, specifically identifying pontine lesions and enhancement of the facial nerves *(Fig. 1)* consistent with inflammation. These findings suggested a central bulbar/pontine involvement as the cause of the swallowing dysfunction. Given the patient's constitutional symptoms, a broad differential diagnosis was pursued. A chest radiography *(Fig. 2)* and computed tomography *(Fig. 3)* revealed significant bilateral hilar adenopathy.

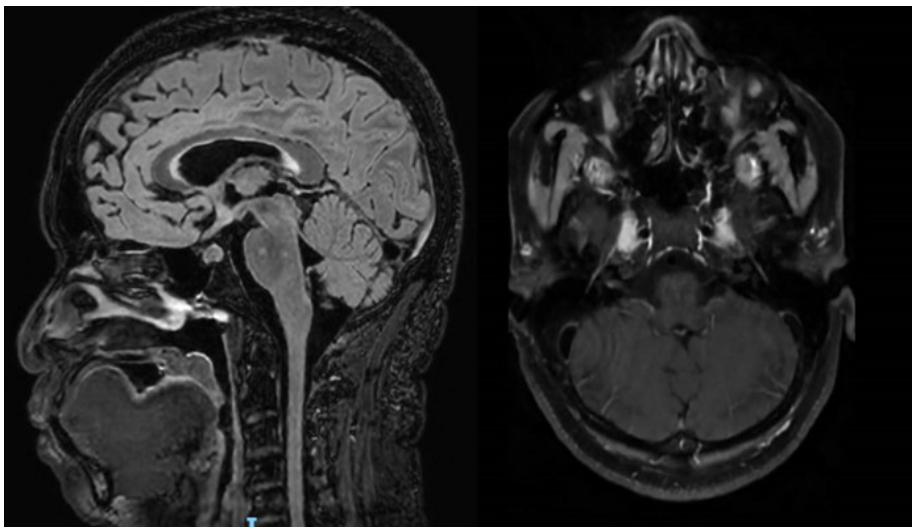


Figure 1. *Contrast-enhanced brain MRI showing parenchymal and perivascular enhancement, specifically identifying pontine lesions and enhancement of the facial nerves.*

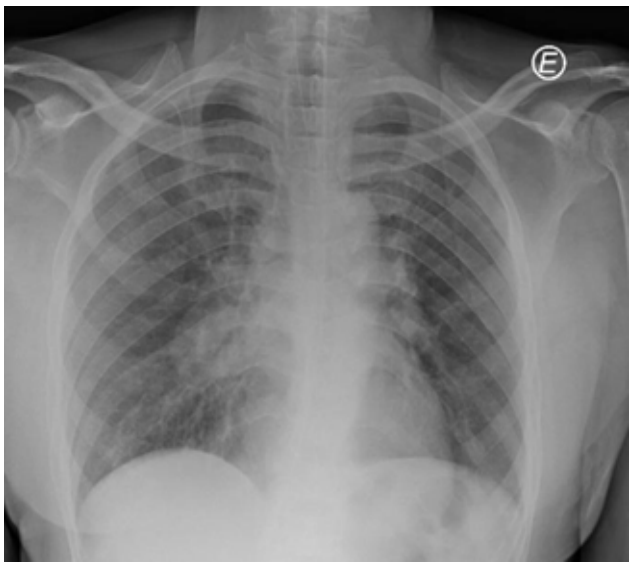


Figure 2. *Chest X-ray showing bilateral hilar adenopathy.*

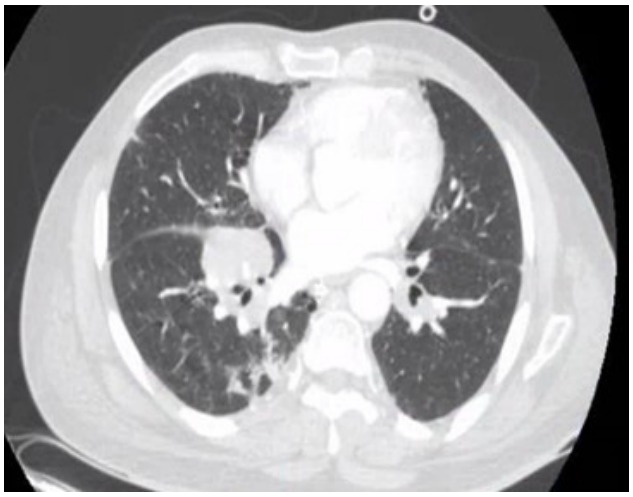


Figure 3. *Chest computed tomography showing bilateral hilar adenopathy.*

Laboratory investigations showed normocytic anaemia, an elevated erythrocyte sedimentation rate (111 mm/h) and significantly high angiotensin-converting enzyme levels (64 IU/l). A bronchoalveolar lavage was performed, showing a lymphocytic alveolitis with high CD4:CD8 ratio. Tuberculosis was a primary concern, but further testing including interferon gamma release assay, sputum smears and polymerase chain reaction for Mycobacterium tuberculosis was negative. Similarly, exhaustive screening for other neuro-infectious agents (syphilis, HIV and neurotropic viruses) and autoimmune markers proved unremarkable. A cerebrospinal fluid analysis was performed and showed no alteration; cytology for malignancy in the cerebrospinal fluid as well as in bronchial secretions was negative. Electromyography revealed a sensorimotor polyneuropathy with a 'sural sparing' pattern, supporting a systemic inflammatory process. A whole-body 18F-FDG PET scan *(Fig. 4)* was performed, revealing multiple hypermetabolic lymph node foci in both supra- and infra-diaphragmatic locations – most pronounced in the broncho-hilar region – alongside pulmonary and pleural involvement. Notably, hypermetabolic activity was also identified in several nerve roots, specifically involving the cranial nerves, a pattern highly suggestive of an active systemic inflammatory process. An endobronchial ultrasound-guided biopsy of the hilar mass provided histological evidence of non-caseating granulomatous lymphadenitis *(Fig. 5)*, confirming the diagnosis. Polymerase chain reaction testing for Mycobacterium tuberculosis on the biopsy was negative. Based on the combination of severe neurological deficits (dysphagia and facial palsy), MRI findings and biopsy-proven systemic disease, a diagnosis of probable neurosarcoidosis was established. The patient was started on high-dose glucocorticoid therapy (intravenous methylprednisolone



Figure 4. *Whole-body FDG-PET scan revealing multiple hypermetabolic lymph node foci in both supra- and infra-diaphragmatic locations—most pronounced in the broncho-hilar region.*

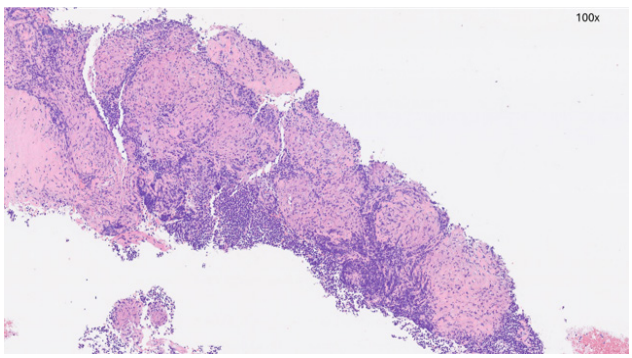


Figure 5. *Histological image showing non-caseating granulomatous lymphadenitis.*

1,000 mg/day for five days, followed by oral prednisone). He showed significant clinical improvement, regaining the ability to swallow effectively, which allowed for the removal of the nasogastric tube and the resumption of oral intake before his transfer to a rehabilitation unit.

DISCUSSION

This case illustrates an atypical and rare presentation of NS where severe, progressive dysphagia served as the presenting symptom. Diagnosing NS remains challenging for the internist as neurological symptoms are the first manifestation of the disease in nearly 50% of cases, often appearing non-specific and without a prior systemic manifestation[1,3]. NS is widely recognised as a 'great mimicker', with a clinical spectrum that overlaps significantly with infection and malignancy[1,2]. Cranial neuropathies are the most frequent neurological phenotype, occurring in 50% to 70% of patients[2] with NS and typically involve the facial (VII) and optic (II) nerves[1,2,4]. In this case, neurosarcoidosis presented as severe dysphagia and dysarthria as the primary neurological symptom, which is an exceptionally rare and uncommon manifestation with only few cases described in the literature. One example is the case described by Iyer et al. (2024), where vocal cord palsy (involvement of the X nerve) was the presenting sign of neurosarcoidosis[5]. In our patient, the diagnostic search for a neurological cause of severe dysphasia and dysarthria was confirmed by contrast-enhanced brain MRI, where the finding revealed perivascular enhancement and parenchymal involvement particularly affecting the brainstem with evidence of pontine lesions. This is highly characteristic of the disease's neuroimaging phenotype[1] and consistent with the literature where parenchymal involvement is present in approximately 30% to 50% of neurosarcoidosis cases. It has a predilection for the basal structures of the brain, including the hypothalamus, basal ganglia and brainstem, which encompasses the pons[1,2,4]. Parenchymal lesions in these areas typically present as T2-hyperintense regions with variable enhancement patterns, often mimicking neoplasia[2]. Such brainstem involvement and associated bulbar dysfunction directly justifies the severe dysphagia and dysarthria observed in this patient, and the identification of facial nerve enhancement is also a critical supporting element. While the facial nerve (VII) is the most commonly affected cranial nerve in neurosarcoidosis, occurring in 24% to 50% of cases, visible enhancement on MRI is only present in approximately 30% of patients[1,4]. Its presence in this case, when combined with the clinical history of preceding facial palsy, strongly pointed towards a cranial neuropathy secondary to the granulomatous process[1,2]. Additionally, our patient also exhibited peripheral nervous system (PNS) involvement, initially suggested by limb paraesthesia and later confirmed by electromyography, which revealed an asymmetric sensorimotor polyneuropathy with a sural sparing pattern, frequent associated with inflammatory processes. According to the literature, the PNS is involved in approximately 14% to 21% of neurosarcoidosis cases[1,2]. A diagnosis of 'probable' neurosarcoidosis was made, supported by the neurological alterations with evidence of brainstem inflammation and the systemic findings such as hilar lymphadenopathy on thoracic imaging. The associated biopsy confirmed non-caseating granulomatous lymphadenitis, after exclusion of diagnostic mimics such as tuberculosis and malignancy[1,3]. Given the risk of CNS biopsy, the possibility of systemic biopsy and the dramatic clinical recovery following high-dose corticosteroids, a definite diagnosis – according to the 2018 Neurosarcoidosis Consortium Consensus Group – was not confirmed[2,3].

CONCLUSION

This case emphasises that severe dysphagia from a neurological cause, although rare, must prompt the clinician to consider neurosarcoidosis in the differential diagnosis. A meticulous clinical history and the proactive screening for systemic signs and symptoms, such as hilar adenopathy, are essential to uncover this complex diagnosis and initiate timely and efficient therapy[1,3].

REFERENCES

1. Barreras P, Stern BJ. Clinical features and diagnosis of neurosarcoidosis – review article. J Neuroimmunol. 2022;368:577871. doi: 10.1016/j.jneuroim.2022.577871.
2. Sinha T, Tahir S, Namal F, Vineesha F, Warsha F, Ahmed Z, et al. Neurosarcoidosis: current perspectives on diagnosis, management, and future directions. Cureus. 2024;16:e69208. doi: 10.7759/cureus.69208.
3. Stern BJ, Royal W 3rd, Gelfand JM, Clifford DB, Tavee J, Pawate S, et al. Definition and consensus diagnostic criteria for neurosarcoidosis: from the Neurosarcoidosis Consortium Consensus Group. JAMA Neurol. 2018;75:1546–1553. doi: 10.1001/jamaneurol.2018.2295.
4. Gosselin J, Roy-Hewitson C, Bullis SSM, DeWitt JC, Soares BP, Dasari S, et al. Neurosarcoidosis: phenotypes, approach to diagnosis and treatment. Curr Rheumatol Rep. 2022;24:371–382. doi: 10.1007/s11926-022-01089-z.
5. Iyer S, Palegar T, Ojha A. Pearls & oy-sters: vocal cord palsy as the presenting manifestation of neurosarcoidosis. Neurology. 2024;103:e209955. doi: 10.1212/WNL.0000000000209955.