

Association Between Myasthenia Gravis and Sarcoidosis: A Case Report

Review began 10/01/2026

Review ended 10/08/2026

Published 10/17/2026

© Copyright 2026

Amanahou et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.118079

Maryam Amanahou ^{1, 2}, Mohamed Laghdaf Maouelainin ^{3, 2}, Abdellah Taous ^{4, 5}, Abdelfatah Zidane ⁶, Naji Amrani Hicham ^{1, 5}

¹. Department of Pulmonology, Oued Eddahab Military Hospital, Agadir, MAR ². Faculty of Medicine and Pharmacy, Ibn Zohr University, Agadir, MAR ³. Department of Radiology, Oued Eddahab Military Hospital, Agadir, MAR ⁴. Department of Neurology, Moulay Ismail Military Hospital, Meknes, MAR ⁵. Faculty of Medicine and Pharmacy, Sidi Mohamed Ben Abdellah University, Fez, MAR ⁶. Department of Thoracic Surgery, Avicenna Military Hospital, Marrakesh, MAR

Corresponding author: Maryam Amanahou, amanahou.maryam@gmail.com

Abstract

Sarcoidosis is a systemic granulomatous disease of unknown cause. Its association with myasthenia gravis has mainly been documented in isolated case reports and small series. We report double-seronegative myasthenia gravis for the antibodies tested, associated with stage I thoracic sarcoidosis and thymic hyperplasia without thymoma.

A 46-year-old woman was hospitalized in November 2021 for grade 3 exertional dyspnea according to the modified Medical Research Council scale, associated with fluctuating left-sided ptosis that worsened toward the end of the day. The neostigmine test was positive, with transient improvement in the ptosis. Low-frequency repetitive nerve stimulation demonstrated a decrement compatible with a postsynaptic neuromuscular transmission disorder. Antibodies against the acetylcholine receptor and anti-MuSK antibodies were negative, leading to the diagnosis of double-seronegative myasthenia gravis for the antibodies tested. At the same time, thoracic imaging showed multiple bilateral mediastinal and hilar lymphadenopathies without pulmonary parenchymal involvement. Mediastinoscopy with lymph node biopsy revealed non-necrotizing epithelioid and giant-cell granulomatous lymphadenitis compatible with sarcoidosis. Systemic corticosteroid therapy with prednisone was initiated. The neurological course was marked by worsening myasthenic manifestations, with increased ptosis, diplopia, painful dysphagia, and predominantly left-sided weakness. A course of intravenous immunoglobulin resulted in marked clinical improvement. Thymectomy performed in July 2023 revealed thymic hyperplasia without thymoma. At the latest available follow-up, the patient was asymptomatic and remained under neurological follow-up.

The coexistence of sarcoidosis and myasthenia gravis remains rarely described. In this case, the two diagnoses were supported by independent findings. Sarcoidosis was supported by radiological and histological findings, whereas the fluctuating and fatigable nature of the ptosis, the response to neostigmine, and the electrophysiological abnormalities supported the diagnosis of myasthenia gravis. Neurosarcoidosis was the main neurological differential diagnosis. The coexistence of the two diseases may be coincidental and, although an immune-mediated background may be considered, the available data do not demonstrate a specific or causal relationship between sarcoidosis and myasthenia gravis.

This case describes a rare association between stage I thoracic sarcoidosis and double-seronegative myasthenia gravis, with thymic hyperplasia without thymoma. It highlights the importance of documenting each condition separately and of investigating a neuromuscular transmission abnormality in the presence of fluctuating ptosis, diplopia, or bulbar involvement in a patient with sarcoidosis, rather than immediately attributing these manifestations to neurosarcoidosis.

Categories: Neurology, Radiology, Pulmonology

Keywords: myasthenia gravis, neuromuscular junction, neurosarcoidosis (ns), sarcoidosis, seronegative myasthenia gravis, thymic hyperplasia

Introduction

Sarcoidosis is a systemic granulomatous disease of unknown cause. Its diagnosis is based on a compatible clinical and radiological presentation, the demonstration of non-necrotizing granulomatous inflammation, and the reasonable exclusion of other causes of granulomatous disease [1]. Bilateral hilar lymphadenopathy without pulmonary parenchymal involvement corresponds to radiographic stage I.

Autoimmune myasthenia gravis is a disorder of the neuromuscular junction characterized by fluctuating weakness and fatigability, which may involve the ocular, bulbar, axial, or respiratory muscles. Antibodies against the acetylcholine receptor are the most frequently identified; other patients have anti-muscle-specific kinase (MuSK) or anti-low-density lipoprotein receptor-related protein 4 (LRP4) antibodies, while a

How to cite this article

Amanahou M, Maouelainin M, Taous A, et al. (October 17, 2026) Association Between Myasthenia Gravis and Sarcoidosis: A Case Report. Cureus 18(10): e118079. DOI 10.7759/cureus.118079

proportion remain seronegative despite compatible clinical and electrophysiological findings [2,3].

The coexistence of sarcoidosis and myasthenia gravis has mainly been documented in isolated case reports and small series [4-6]. The reported phenotypes are heterogeneous, including myasthenia gravis positive for acetylcholine receptor antibodies or anti-MuSK antibodies and, in one case, thymoma [4-6]. We report a case of double-seronegative myasthenia gravis for the antibodies tested, associated with stage I thoracic sarcoidosis and thymic hyperplasia without thymoma.

Case Presentation

A 46-year-old woman was hospitalized in November 2021 for grade 3 exertional dyspnea according to the modified Medical Research Council scale, associated with initially mild, fluctuating left-sided ptosis that worsened toward the end of the day [7]. She had never received antituberculous treatment, and no history of smoking was documented. Her surgical history included tympanoplasty in 2006, excision of a vocal cord cyst in 2009, and appendectomy in 2019.

On admission, the patient was conscious, with a Glasgow Coma Scale score of 15/15, a normal heart rate, and normal blood pressure [8]. The neurological examination was characterized by fluctuating left-sided ptosis, with initially preserved limb muscle strength. Deep tendon reflexes were brisk and asymmetric in the lower limbs, without Babinski's or Hoffmann's signs. The fluctuating nature of the symptoms and their worsening with fatigue suggested a disorder of neuromuscular transmission. The main differential diagnoses included myasthenia gravis and neurosarcoidosis, the latter being considered in the context of the subsequently confirmed systemic sarcoidosis.

Pleuropulmonary examination showed peripheral oxygen saturation of 99% on room air, normally transmitted vocal fremitus, and bilaterally preserved vesicular breath sounds, with no crackles on auscultation.

A pharmacological neostigmine (Prostigmine®) test was performed at a dose of 0.5 mg by slow intravenous injection under atropine coverage. The test was positive, with transient improvement in ptosis (Figure 1). Symptomatic treatment with pyridostigmine (Mestinon®) was then initiated at a dose of 30 mg three times daily.

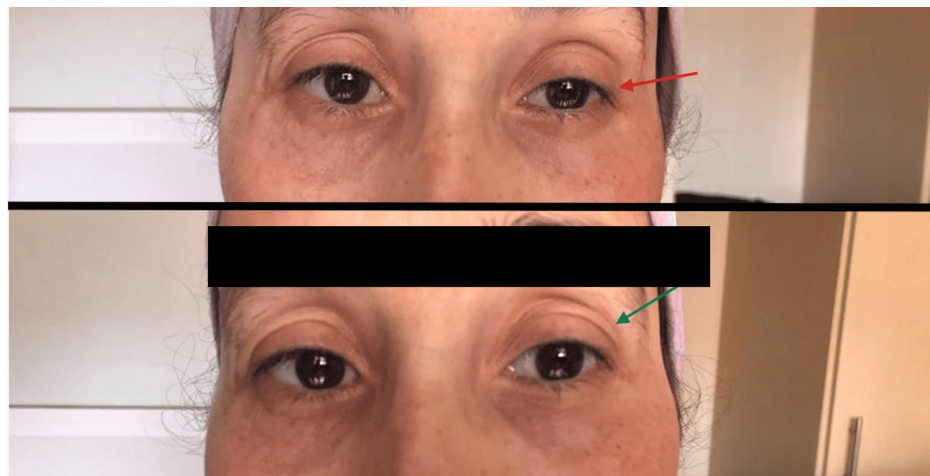


FIGURE 1: Left ptosis before and after neostigmine administration

The red arrow indicates left-sided ptosis before neostigmine administration, while the green arrow shows transient improvement in ptosis after intravenous administration of neostigmine (0.5 mg).

Neurophysiological assessment included sensory and motor nerve conduction studies in all four limbs, which showed no abnormalities. Low-frequency repetitive nerve stimulation demonstrated a decrement in compound muscle action potential amplitude during stimulation of the left ulnar nerve, compatible with a postsynaptic neuromuscular transmission disorder. Testing for antibodies against the acetylcholine receptor and anti-MuSK antibodies was negative. Anti-LRP4 antibody testing was not performed. Given the fluctuating and fatigable nature of the ptosis, the improvement after neostigmine, and the electrophysiological abnormalities, the diagnosis of myasthenia gravis, double-seronegative for anti-AChR and anti-MuSK antibodies, was retained.

At the same time, thoracic imaging showed multiple bilateral mediastinal and hilar lymphadenopathies without associated pulmonary parenchymal involvement (Figure 2). Bronchoscopy with bronchoalveolar

lavage and bronchial biopsies was performed during the same hospitalization. Histopathological examination of the bronchial biopsies showed nonspecific chronic inflammatory changes, with mild lymphocytosis in the bronchoalveolar lavage (Table 1), and the findings were not contributory to the etiological diagnosis.

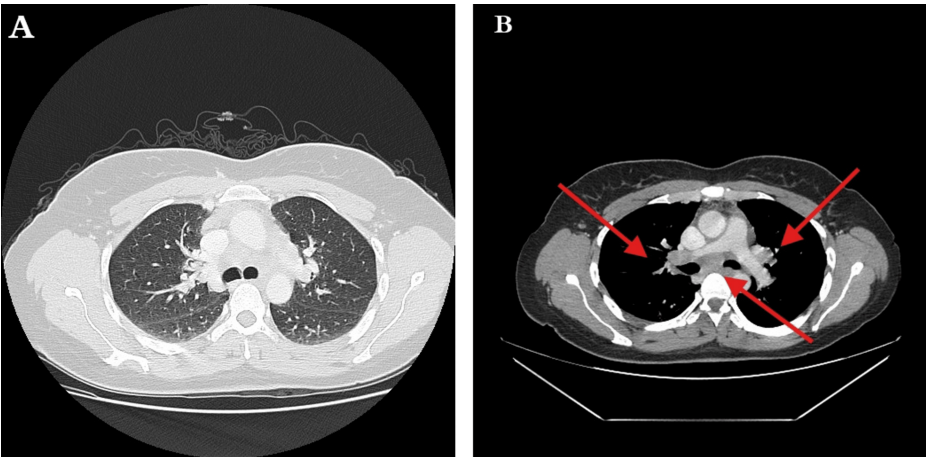


FIGURE 2: Thoracic CT showing mediastinal and hilar lymphadenopathy without pulmonary parenchymal involvement

(A) Axial CT image in the lung window showing preserved pulmonary parenchyma. (B) Contrast-enhanced axial CT image in the mediastinal window showing mediastinal and hilar lymphadenopathy (red arrows).

Cell population	Percentage
Bronchial cells	13%
Macrophages	55%
Neutrophils (PNN)	15%
Eosinophils (PNE)	2%
Lymphoplasmacytic cells	15%

TABLE 1: Cytological differential of bronchoalveolar lavage

PNN: polymorphonuclear neutrophils; PNE: polymorphonuclear eosinophils

As part of the investigation for tuberculosis, *Mycobacterium tuberculosis* was tested for by molecular methods (polymerase chain reaction (PCR)) in sputum and bronchoalveolar lavage fluid; both results were negative. Because of persistent mediastinal lymphadenopathy and the inconclusive bronchial biopsies, mediastinoscopy with lymph node biopsy was performed in December 2021. Histopathological examination revealed non-necrotizing epithelioid and giant-cell granulomatous lymphadenitis compatible with sarcoidosis. However, acid-fast bacilli staining and molecular testing of the lymph node specimen were not documented in the available medical records. The diagnosis of mediastinal and hilar lymph node sarcoidosis without pulmonary parenchymal involvement was therefore retained.

As part of the assessment of the extent of sarcoidosis, renal and liver function tests were normal. Angiotensin-converting enzyme (ACE), measured at 56 IU/L, and serum calcium, measured at 88 mg/L, were also normal according to the laboratory reference ranges. An ophthalmological consultation was also performed; the examination showed no evidence of uveitis or optic neuritis.

Because of persistent respiratory and constitutional symptoms, systemic corticosteroid therapy with prednisone was initiated in February 2022 at a dose of 40 mg/day for the treatment of sarcoidosis and was gradually tapered over a total period of 16 months.

The subsequent course was marked by the development of dysphonia and progressive worsening of the

myasthenic manifestations. The patient also reported persistent dyspnea and lower chest pain, as well as the onset of epigastric pain and polyarthralgia.

Pulmonary function tests showed a forced vital capacity (FVC) of 91% of predicted (2.78 L), a forced expiratory volume in one second (FEV1) of 94% of predicted, an FEV1/FVC ratio of 81%, a total lung capacity (TLC) of 101% of predicted (4.88 L), and a diffusing capacity for carbon monoxide (DLCO) of 86% of predicted. During the six-minute walk test, the patient walked 476 m, with a minimum oxygen saturation of 99%. These results, interpreted as normal, did not reveal any significant respiratory functional abnormality that could explain the dyspnea.

The epigastric pain prompted upper gastrointestinal endoscopy, which showed gastritis and funditis associated with *Helicobacter pylori* infection, without histological evidence of malignancy.

After approximately 12 months of corticosteroid therapy, in early 2023, while receiving prednisone at a dose of 10 mg/day, the neurological course was marked by worsening myasthenic manifestations, with increased ptosis, onset of diplopia, painful dysphagia, and predominantly left-sided weakness. The Garches myasthenic score was then 80/100 [9]. The patient was hospitalized in the neurology department and received a course of intravenous immunoglobulin, followed by marked clinical improvement.

As part of the evaluation of this neurological worsening, brain magnetic resonance imaging (MRI) was normal. Spinal MRI showed a left paramedian C3-C4 disc herniation (Table 2). In July 2023, thymectomy was performed. Histopathological examination of the surgical specimen concluded that there was thymic hyperplasia, with no evidence of thymoma. Follow-up thoracic MRI showed no residual mediastinal abnormality. Subsequently, high-dose oral corticosteroid therapy was initiated in association with continued pyridostigmine treatment. At the latest available follow-up, the patient remained under neurological follow-up, was asymptomatic, and the prednisone dose was being gradually tapered.

Investigation	Findings
Neostigmine test (0.5 mg IV)	Positive; transient improvement of left-sided ptosis
Sensory and motor nerve conduction studies	Normal in all four limbs
Low-frequency repetitive nerve stimulation	Decrement during left ulnar nerve stimulation
Anti-AChR antibodies	Negative
Anti-MuSK antibodies	Negative
Anti-LRP4 antibodies	Not performed
Brain MRI	Normal
Spinal MRI	Left paramedian C3-C4 disc herniation
Thoracic CT	Bilateral mediastinal and hilar lymphadenopathy without pulmonary parenchymal involvement
Bronchial biopsies	Nonspecific chronic inflammatory changes
MTB PCR (sputum and BAL fluid)	Negative in both specimens
Mediastinal lymph-node biopsy	Non-necrotizing epithelioid and giant-cell granulomatous lymphadenitis compatible with sarcoidosis
Thymectomy specimen	Thymic hyperplasia without thymoma
ACE	56 IU/L; within laboratory reference range
Serum calcium	88 mg/L; within laboratory reference range
Renal and liver function tests	Normal
FVC	91% predicted (2.78 L)
FEV1	94% predicted
FEV1/FVC ratio	81%
TLC	101% predicted (4.88 L)
DLCO	86% predicted
Six-minute walk test	476 m; minimum SpO ₂ 99%

TABLE 2: Summary of the main diagnostic investigations and findings

AChR: acetylcholine receptor; MuSK: muscle-specific kinase; LRP4: low-density lipoprotein receptor-related protein 4; IV: intravenous; MRI: magnetic resonance imaging; MTB: *Mycobacterium tuberculosis*; PCR: polymerase chain reaction; BAL: bronchoalveolar lavage; SpO₂: peripheral oxygen saturation; ACE: angiotensin-converting enzyme; FVC: forced vital capacity; FEV1: forced expiratory volume in one second; TLC: total lung capacity; DLCO: diffusing capacity for carbon monoxide

Discussion

The association between sarcoidosis and myasthenia gravis has rarely been described. De Bleecker et al. reported two cases in which the two conditions coexisted [4]. Spengos et al. [5] described sarcoidosis in a patient with anti-MuSK-positive myasthenia gravis, while Kurukumbi et al. reported the association of sarcoidosis, acetylcholine receptor antibody-positive myasthenia gravis, and thymoma [6]. Our case is distinguished by sarcoidosis limited to the mediastinal and hilar lymph nodes, double-seronegative myasthenia gravis for the antibodies tested, and thymic hyperplasia without thymoma.

The validity of this association rests on the independent confirmation of both diagnoses. The diagnosis of sarcoidosis was based on a compatible radiological presentation consisting of bilateral hilar and mediastinal lymphadenopathy without parenchymal involvement and on the demonstration of non-necrotizing epithelioid and giant-cell granulomas in the lymph node biopsy [1]. PCR testing for *M. tuberculosis* in sputum and bronchoalveolar lavage fluid was negative. These findings reduced the likelihood of active tuberculosis without formally excluding a paucibacillary form. However, Ziehl-Neelsen staining and molecular testing for *M. tuberculosis* on the lymph node biopsy specimen were not documented in the

available medical records. This represents a limitation of the diagnostic evaluation, as lymph node tuberculosis could not be definitively excluded on the basis of the available microbiological findings. The diagnosis of stage I thoracic sarcoidosis was therefore retained on the basis of the overall clinical, radiological, and histopathological findings, while acknowledging this diagnostic limitation.

The diagnosis of myasthenia gravis was supported by the fluctuating and fatigable nature of the ptosis, its transient improvement after neostigmine, and the decrement observed during low-frequency repetitive nerve stimulation [2,3]. Normal sensory and motor nerve conduction studies were compatible with an isolated disorder of the neuromuscular junction. Negative acetylcholine receptor and anti-MuSK antibodies supported the classification of double-seronegative myasthenia gravis for the antibodies tested. In a cohort of 250 patients with myasthenia gravis, 38 (15.2%) were double-seronegative for anti-AChR and anti-MuSK antibodies [10]. This did not exclude an autoimmune origin: low-affinity acetylcholine receptor antibodies may be detected by cell-based assays, and anti-LRP4 antibodies may be identified in some patients initially considered seronegative [3,11].

Neurosarcoidosis was the main neurological differential diagnosis. It may involve the cranial nerves, meninges, brain, spinal cord, roots, or peripheral nerves [12]. In this case, evening-predominant ptosis, diplopia, the response to neostigmine, and the postsynaptic electrophysiological abnormality were more compatible with myasthenia gravis. Normal brain MRI provided no radiological evidence of intracranial sarcoid involvement, although it could not exclude it on its own. The left paramedian C3-C4 disc herniation was a concomitant structural abnormality that could be considered in view of the left-sided predominance of weakness; however, its clinical impact could not be established in the absence of a description of radicular conflict, spinal cord compression, or intramedullary signal abnormality. It did not explain the fluctuating oculobulbar manifestations.

Dyspnea represented a second diagnostic challenge. Despite an mMRC grade of 3, the pulmonary parenchyma was preserved, and spirometry, plethysmography, and the six-minute walk test showed no significant abnormalities. Extrinsic bronchial compression by the lymphadenopathy could have contributed to the symptoms. Respiratory muscle involvement related to myasthenia gravis also remained possible because it may not be detected by routine pulmonary function tests. Targeted measurements, vital capacity in the sitting and supine positions, maximal inspiratory and expiratory pressures, or sniff nasal inspiratory pressure, would have allowed this component to be better assessed. In the absence of these data, the etiology of the dyspnea remained undetermined and was probably multifactorial.

The worsening oculobulbar manifestations led to the administration of intravenous immunoglobulin, a short-term treatment used during significant myasthenic exacerbations, particularly in the presence of bulbar manifestations [13]. Marked clinical improvement was observed.

The prolonged corticosteroid treatment in our patient also warrants consideration. The SARCORT trial, published in 2023, found no superiority of an initial prednisolone dose of 40 mg/day over 20 mg/day in terms of treatment failure or relapse in pulmonary sarcoidosis [14]. Although prolonged corticosteroid exposure may have contributed to the subsequent onset of epigastric pain, upper gastrointestinal endoscopy revealed gastritis and funditis associated with *H. pylori* infection. Therefore, a causal relationship between corticosteroid therapy and the gastrointestinal symptoms cannot be established in this patient.

The coexistence of the two diseases may be coincidental. Nevertheless, sarcoidosis is associated with an increased frequency of other immune-mediated diseases. In a cohort of 1,737 patients with sarcoidosis, 283 (16%) had at least one associated immune-mediated disease, a frequency higher than that in the general population (OR, 1.64; 95% CI, 1.44-1.86) [15]. These data support the existence of an immune-mediated background favoring autoimmune associations, but they do not demonstrate a specific or causal relationship between sarcoidosis and myasthenia gravis. The presence of thymic hyperplasia describes the histopathological phenotype in this case without establishing a common mechanism between the two conditions.

Conclusions

This case describes a rare association between stage I thoracic sarcoidosis and double-seronegative myasthenia gravis, with thymic hyperplasia without thymoma. It highlights the need to document each condition separately when neurological manifestations occur in the course of sarcoidosis. Fluctuating ptosis, diplopia, or bulbar involvement should prompt investigation for a neuromuscular transmission abnormality rather than immediate attribution to neurosarcoidosis.

Severe dyspnea associated with preserved pulmonary parenchymal imaging and routine pulmonary function tests should raise the possibility of a neuromuscular component and, when possible, justify specific assessment of the respiratory muscles. The observed association is compatible with a shared immune-mediated background, but the available data do not allow a causal relationship between sarcoidosis and myasthenia gravis to be established.

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: Maryam Amanahou, Naji Amrani Hicham, Mohamed Laghdaf Maouelainin, Abdellah Taous, Abdelfatah Zidane

Acquisition, analysis, or interpretation of data: Maryam Amanahou, Naji Amrani Hicham, Mohamed Laghdaf Maouelainin, Abdellah Taous, Abdelfatah Zidane

Drafting of the manuscript: Maryam Amanahou

Critical review of the manuscript for important intellectual content: Maryam Amanahou, Naji Amrani Hicham, Mohamed Laghdaf Maouelainin, Abdellah Taous, Abdelfatah Zidane

Supervision: Naji Amrani Hicham, Mohamed Laghdaf Maouelainin, Abdellah Taous, Abdelfatah Zidane

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Crouser ED, Maier LA, Wilson KC, et al.: Diagnosis and detection of sarcoidosis. An official American Thoracic Society clinical practice guideline. *Am J Respir Crit Care Med*. 2020, 201:e26-51. [10.1164/rccm.202002-0251ST](https://doi.org/10.1164/rccm.202002-0251ST)
2. Hehir MK II, Li Y: Diagnosis and management of myasthenia gravis . *Continuum (Minneapolis)*. 2022, 28:1615-42. [10.1212/CON.0000000000001161](https://doi.org/10.1212/CON.0000000000001161)
3. Tannemaat MR, Huijbers MG, Verschuuren JJGM: Myasthenia gravis: pathophysiology, diagnosis, and treatment. *Handb Clin Neurol*. 2024, 200:283-305. [10.1016/B978-0-12-823912-4.00026-8](https://doi.org/10.1016/B978-0-12-823912-4.00026-8)
4. De Bleecker J, van Aken L, van Landegem W, van den Abeele G, Cuvelier C: Myasthenia gravis and sarcoidosis: report of 2 cases. *Eur Neurol*. 1996, 36:326-7. [10.1159/000117284](https://doi.org/10.1159/000117284)
5. Spengos K, Vassilopoulou S, Christou Y, Manta P: Sarcoidosis in a case of MuSK-positive myasthenia gravis . *Neuromuscul Disord*. 2008, 18:890-1. [10.1016/j.nmd.2008.07.001](https://doi.org/10.1016/j.nmd.2008.07.001)
6. Kurukumbi M, Weir RL, Kalyanam J, Nasim M, Jayam-Trouth A: Rare association of thymoma, myasthenia gravis and sarcoidosis: a case report. *J Med Case Rep*. 2008, 2:245. [10.1186/1752-1947-2-245](https://doi.org/10.1186/1752-1947-2-245)
7. Bestall JC, Paul EA, Garrod R, Garnham R, Jones PW, Wedzicha JA: Usefulness of the Medical Research Council (MRC) dyspnoea scale as a measure of disability in patients with chronic obstructive pulmonary disease. *Thorax*. 1999, 54:581-6. [10.1136/thx.54.7.581](https://doi.org/10.1136/thx.54.7.581)
8. Teasdale G, Jennett B: Assessment of coma and impaired consciousness. A practical scale . *Lancet*. 1974, 2:81-4. [10.1016/S0140-6736\(74\)91639-0](https://doi.org/10.1016/S0140-6736(74)91639-0)
9. Sharshar T, Chevret S, Mazighi M, et al.: Validity and reliability of two muscle strength scores commonly used as endpoints in assessing treatment of myasthenia gravis. *J Neurol*. 2000, 247:286-90. [10.1007/s004150050585](https://doi.org/10.1007/s004150050585)
10. Cortés-Vicente E, Gallardo E, Martínez MÁ, Díaz-Manera J, Querol L, Rojas-García R, Illa I: Clinical characteristics of patients with double-seronegative myasthenia gravis and antibodies to cortactin. *JAMA Neurol*. 2016, 73:1099-104. [10.1001/jamaneurol.2016.2032](https://doi.org/10.1001/jamaneurol.2016.2032)
11. Evoli A, Palace J, Spagni G, Cheli M, Ruiter A, Verschuuren J, Maggi L: 275th ENMC International Workshop: seronegative myasthenia gravis: an update paradigm for diagnosis and management, 9-11 February 2024, Hoofddorp, the Netherlands. *Neuromuscul Disord*. 2024, 44:104468. [10.1016/j.nmd.2024.104468](https://doi.org/10.1016/j.nmd.2024.104468)
12. Stern BJ, Royal W III, Gelfand JM, et al.: Definition and consensus Diagnostic Criteria for neurosarcoidosis: from the neurosarcoidosis Consortium consensus Group. *JAMA Neurol*. 2018, 75:1546-53. [10.1001/jamaneurol.2018.2295](https://doi.org/10.1001/jamaneurol.2018.2295)
13. Narayanaswami P, Sanders DB, Wolfe G, et al.: International consensus guidance for management of myasthenia gravis: 2020 update. *Neurology*. 2021, 96:114-22. [10.1212/WNL.0000000000001124](https://doi.org/10.1212/WNL.0000000000001124)
14. Dhoria S, Sehgal IS, Agarwal R, et al.: High-dose (40 mg) versus low-dose (20 mg) prednisolone for treating sarcoidosis: a randomised trial (SARCORT trial). *Eur Respir J*. 2023, 62:2300198. [10.1183/13993003.00198-2023](https://doi.org/10.1183/13993003.00198-2023)
15. Brito-Zerón P, Pérez-Alvarez R, Feijoo-Massó C, et al.: Coexistence of immune-mediated diseases in sarcoidosis. Frequency and clinical significance in 1737 patients. *Joint Bone Spine*. 2021, 88:105236. [10.1016/j.jbspin.2021.105236](https://doi.org/10.1016/j.jbspin.2021.105236)