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Recurrent Sarcoidosis Presenting With Bilateral Leg Edema: A Case Report

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ABSTRACT

Background: Bilateral lower extremity edema may serve as an early clinical manifestation of pulmonary sarcoidosis. Identifying sarcoidosis as the underlying cause can be challenging in patients with or without a prior history of the disease, as lower extremity edema is not typically considered part of its clinical spectrum.

Case Report: A 63-year-old man presented with a complex medical history, including non-Hodgkin lymphoma, bilateral renal cell carcinoma, colon cancer, and pleomorphic sarcoma of the upper extremity. He also had a history of recurrent pulmonary sarcoidosis. He reported a 6-month history of bilateral pitting lower extremity edema that developed several months before the onset of dyspnea.

Conclusion: In patients with multiple comorbidities, recognizing sarcoidosis as a potential cause of bilateral lower extremity edema requires careful exclusion of systemic and malignant etiologies. Sarcoidosis should be considered when more common causes have been excluded.

Keywords: Differential diagnosis, edema, lymphedema, pulmonary sarcoidosis, sarcoidosis.

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INTRODUCTION

Bilateral lower extremity lymphedema is an uncommon manifestation of sarcoidosis and may even be the initial presenting symptom, sometimes occurring in the absence of pulmonary involvement. These features pose diagnostic challenges for clinicians when determining the underlying cause of lymphedema. A thorough history and physical examination are essential to exclude more common causes and identify any related symptoms or physical findings. In patients with a known history of sarcoidosis, lymphedema should be recognized as a potential manifestation and presenting feature of the disease.

CASE REPORT

A 63-year-old White man presented with a 6-month history of progressive dyspnea, initially exertional and later occurring at rest, and bilateral lower extremity edema that had begun several

months earlier. His past medical history included stage IIIB non-Hodgkin lymphoma, partial resections for bilateral papillary renal cell carcinoma, and resection of stage I colon cancer, all in remission. He had previously experienced three biopsy-confirmed episodes of pulmonary sarcoidosis in his late twenties, early thirties, and mid-fifties.

One year before the present evaluation, he underwent radiation followed by surgical excision of stage I undifferentiated pleomorphic sarcoma in the subdeltoid region of the left shoulder, with continued surveillance. A chest CT in August 2025 showed several new nodular opacities, including an 8-mm right lower lobe nodule and clustered nodules in the left upper lobe. Abdominal MRI showed no lymphadenopathy.

On physical examination, breath sounds and jugular venous pressure were normal, with no evidence of pulmonary venous congestion. His lower legs showed bilateral pitting edema that was nontender to palpation. Laboratory studies showed a normal complete blood cell count, total protein level, and albumin level. The creatinine level was 2.78 mg/dL, unchanged from his baseline. The estimated GFR was 42 mL/min/1.73 m^2. Because of the normal jugular venous pressure, absence of pulmonary venous congestion on physical examination, and normal echocardiogram, BNP was not measured. Pulmonary function tests in November 2025 demonstrated a mixed obstructive–restrictive pattern with mildly to moderately reduced gas transfer (DLCO, 11.05 mL/min/mm Hg). Duplex ultrasonography ruled out deep venous thrombosis in both legs, and echocardiography showed normal biventricular function with no evidence of elevated pulmonary pressures.

A repeat chest CT in December 2025 showed a new 10-mm solid, noncalcified nodule in the left upper lobe, additional 4-mm indeterminate nodules, and a 3-mm right upper lobe nodule. Several previously documented 5-mm nodules in the left upper lobe had regressed. No mediastinal or hilar lymphadenopathy was present. Whole-body PET imaging demonstrated linear interstitial FDG uptake and a low-grade FDG-avid 7-mm right apical nodule that had slightly increased in size since August 2025. These findings, particularly the fluctuating distribution and partial spontaneous regression, favored inflammatory granulomatous disease rather than metastatic malignancy. MRI of the left arm revealed no evidence of sarcoma recurrence.

Given the fluctuating parenchymal abnormalities, the patient's nonsmoking status, and his history of biopsy-confirmed sarcoidosis, a shared decision was made to initiate empirical corticosteroid therapy with prednisone 20 mg daily for 1 month. The decision considered the biopsy-related challenges associated with the nodule's location, the PET scan results, the

Table 1. Reported cases of sarcoidosis presenting with bilateral or distal lower-extremity edema

Study	Age	Sex	Race	Initial symptom*	Cutaneous involvement	Inguinal lymphadenopathy	Retroperitoneal lymphadenopathy	Pulmonary involvement
Silver et al.[1] (1966)**	30	Female	Black	Yes	No	Yes	Yes	No
Nathan et al.[2] (1974)	32	Female	Black	Yes	Yes	No	No	Yes
Sweeney et al.[3] (1980)	30	Male	Black	No	No	No	Yes	Yes
Cantini et al.[4] (1997) patient 1	32	Male	Not reported	Yes	No	Not reported	Not reported	Yes
Cantini et al.[4] (1997) patient 2	48	Female	Not reported	Yes	Yes	Not reported	Not reported	Yes
Cantini et al.[4] (1997) patient 3	34	Female	Not reported	Yes	No	Not reported	Not reported	Yes
Cantini et al.[4] (1997) patient 4	31	Male	Not reported	Yes	No	Not reported	Not reported	Yes
Cantini et al.[4] (1997) patient 5	28	Female	Not reported	Yes	Yes	Not reported	Not reported	Yes
Tomoda et al.[5] (1999)	55	Male	Not reported	Yes	Yes	Yes	Yes	No
Huang et al.[6] (2016)	48	Female	Asian	Yes	Yes	No	Not reported	Yes

* "Initial symptom" refers to bilateral or distal lower extremity edema being the first recognized manifestation of sarcoidosis. **Silver HM, Tsangaris NT, Eaton OM. Lymphedema and lymphography in sarcoidosis. Arch Intern Med. 1966;117:712-714.

patient's history of recurrent sarcoidosis, and the high pretest probability of the disease. He experienced marked improvement in dyspnea within 3 days and complete resolution of bilateral edema within 1 week. Follow-up chest CT after 4 weeks showed radiographic resolution of all nodular infiltrates. At the 3-month follow-up, there was no recurrence of the patient's symptoms.

DISCUSSION

Bilateral peripheral edema is a rare but recognized manifestation of sarcoidosis and may even be the presenting feature.[1–6] Published cases show that sarcoidosis-associated edema typically reflects lymphatic involvement, such as inguinal or retroperitoneal lymphadenopathy, or direct soft-tissue infiltration causing lymphatic obstruction.[1–4,6] In some patients, tenosynovitis can lead to a pitting distal edema pattern referred to as remitting extremity swelling syndrome.[4]

Among the 10 well-documented cases in the literature (Table 1), most occurred in adults aged 28–55 years, and several were associated with cutaneous sarcoid lesions, including subcutaneous nodules, plaques, erythema nodosum, or ulcerated nodules. Lymphadenopathy or pulmonary disease often accompanied these presentations. Our patient differs from prior reports in that he presented with bilateral lower extremity edema before the onset of respiratory symptoms and without radiologically detectable lymphadenopathy or cutaneous involvement.

The patient's extensive oncologic history created a diagnostic challenge, particularly in distinguishing recurrent sarcoidosis from metastatic disease. In this patient, the waxing and waning pattern of the pulmonary nodules, with some appearing while others regressed, was more compatible with an inflammatory granulomatous process than with metastatic disease. The absence of progressive lymphadenopathy and the presence of only mildly increased FDG uptake also supported sarcoidosis. Deep venous thrombosis was excluded by duplex ultrasonography, and no findings suggested anemia, hypoalbuminemia, or volume overload. In contrast, metastatic nodules arising from lymphoma or renal cell carcinoma would be expected to enlarge steadily and follow a more persistent radiologic course. The patient's rapid and complete resolution of both edema and dyspnea after the initiation of corticosteroids further supports sarcoidosis as the underlying diagnosis.

In patients with bilateral lower extremity edema in the absence of obstructive lymphadenopathy, cutaneous lesions, or tenosynovitis, alternative mechanisms must be considered to explain the symptoms. Elevated proinflammatory cytokines, such as tumor necrosis factor-alpha (TNF-alpha), interferon-gamma (IFN-γ), IL-6, and IL-18, are common in patients with sarcoidosis.[7] These cytokines increase vascular permeability, allowing protein and fluid to move from blood vessels into the interstitium. Genetic polymorphisms in these proteins may alter gene expression and contribute to lower extremity peripheral edema.

CONCLUSION

Lower extremity edema in sarcoidosis may be subtle, easily misattributed to comorbidities, or overlooked entirely, particularly in older patients and those with a history of malignancy. It is likely underreported or unrecognized as a clinical entity in sarcoidosis. Additional well-characterized reports are needed to refine the understanding of its clinical spectrum, underlying mechanisms, and diagnostic implications.

Ethics Committee Approval: This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

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