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A Rare Triad of Disseminated Sarcoidosis Involving The Heart, Lungs, And Bone Marrow: A Case Report.

Yash Ranga[1*], Cheryl Jain[2], Aayush Anand[3], Bryan Ashong[3], Riddhi Agarwal[4], Venkata Peddi[5], Abhishek Ranga[1], Bharti Chura[6]

[1]Jawahar Lal Nehru Medical College, Ajmer, India

[2]MBBS, Jawaharlal Nehru medical College, Ajmer, India

[3]University of Texas at San Antonio

[4]University of South Florida, Tampa, United States

[5]Nil Ratan Sircar Medical College, Kolkata, India

[1]Cleveland Clinic, Cleveland, United States

[6]Maharaja Ganga Singh University, Bikaner

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***Corresponding author:** Yash Ranga, Jawahar Lal Nehru Medical College, Ajmer, India

ABSTRACT

Background: Sarcoidosis is a multisystem granulomatous disorder that predominantly affects the lungs, though extrapulmonary involvement significantly increases morbidity. The simultaneous triad of pulmonary, cardiac, and bone marrow sarcoidosis is exceptionally rare, with fewer than five cases documented in medical literature. This report describes a unique case of tri-organ disseminated sarcoidosis in an elderly patient, highlighting the 'diagnostic ceiling' that often complicates the recognition of systemic disease in older populations.

Case Presentation: A 73-year-old female with known Stage I pulmonary sarcoidosis presented with hypoxic respiratory failure and pancytopenia. Diagnostic imaging revealed progressive pulmonary infiltrates and, via cardiac PET (SUV 6.7), occult cardiac involvement-a modality necessitated by MRI-incompatible hardware. Histological confirmation of disseminated disease was obtained through bone marrow biopsy, which showed non-caseating granulomas. This rare tri-organ manifestation was treated with dual-therapy prednisone and infliximab, resulting in clinical stabilization and a transition to a six-month steroid taper.

Discussion: Simultaneous tri-organ sarcoidosis is exceedingly rare and carries a high mortality risk due to cardiac and hematologic complications. This case demonstrates that when traditional imaging is contraindicated, multimodality assessment with PET and bone marrow biopsy is essential for identifying disseminated disease. Given the severity of cardiac and marrow involvement, early initiation of infliximab alongside corticosteroids may be superior to traditional steroid-sparing agents. Clinicians must maintain a high index of suspicion for systemic sarcoidosis in older patients presenting with new-onset cytopenias or cardiac symptoms.

Keywords: Sarcoidosis; Cardiac Sarcoidosis; PET-CT; Bone-Marrow Sarcoidosis; Case Report

INTRODUCTION

Sarcoidosis is a multisystem granulomatous disease of idiopathic etiology, histologically characterized by the formation of non-caseating granulomas within affected organs. In the United States, the disease most frequently affects African American women, with an estimated prevalence of 35.22 per 100,000 individuals [1]. While pulmonary involvement remains the hallmark of the condition, extrapulmonary manifestations are common and significantly drive morbidity and mortality.

Most patients present with single-organ or limited multisystem involvement. However, the simultaneous triad of pulmonary, cardiac, and bone marrow sarcoidosis is exceedingly rare, particularly when presenting in the seventh decade of life. Identifying this specific tri-organ phenotype is clinically imperative, as it often heralds an aggressive disease course and presents a 'diagnostic ceiling' in elderly patients, where systemic symptoms may be erroneously attributed to age-related comorbidities. We present a rare case of concurrent heart, lung, and marrow involvement, emphasizing the diagnostic and therapeutic complexities of disseminated disease.

CASE PRESENTATION

Initial Presentation and Clinical History: A 73-year-old African-American woman presented to the emergency department with acute-onset, left-sided pleuritic chest pain. Her medical history was significant for biopsy-confirmed Stage I pulmonary sarcoidosis (diagnosed four years prior), hypertension, and pulmonary hypertension (Groups 2 and 5). Cardiac history included atrial fibrillation, coronary artery disease, and severe mitral stenosis. Two years prior to presentation, she had an automatic implantable cardioverter-defibrillator (AICD) placed for chronic heart failure.

On physical examination, the patient exhibited signs of decompensated heart failure, including bilateral lower-extremity pitting edema and jugular venous distention. She was in acute hypoxic respiratory failure, requiring 5 L/min of supplemental oxygen via nasal cannula to maintain saturations of 94-95%.

Diagnostic Evaluation: Pulmonary and Cardiac Initial laboratory testing was notable for mild hypercalcemia (serum calcium 10.3 mg/dL). At the time of admission, she was already receiving prednisone 40 mg daily for pulmonary sarcoidosis. Computed tomography (CT) of the chest (Figure 1) revealed new-onset nodular infiltrates and confluent ground-glass opacities. Electrocardiography demonstrated a paced rhythm with no acute ST-T wave changes, and serial troponins were unremarkable. A ventilation–perfusion (V/Q) scan indicated a low probability for pulmonary embolism.

Transthoracic echocardiography (TTE) showed a left ventricular ejection fraction (LVEF) of 50% with regional wall motion abnormalities and severe mitral stenosis. Subsequent transesophageal echocardiography (TEE) identified myocardial thickening and echo-reflectivity concerning for infiltrative disease, specifically cardiac sarcoidosis. Multiple differential diagnoses were considered and ruled out systematically (Table 1).

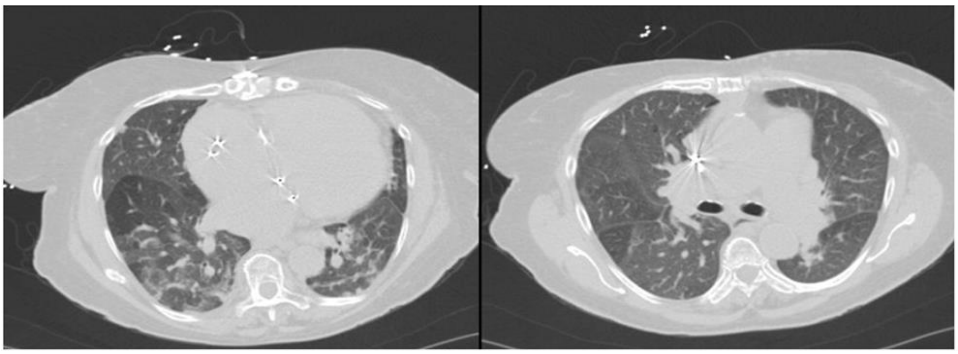

Figure 1: CT Chest revealing Ground-Glass Opacities and Nodular infiltrates (A and B)

Table 1: Differential Diagnosis

#	Condition	Rationale for Consideration	Reason Ruled Out
1	Berylliosis	Occupational beryllium exposure can mimic sarcoidosis with pulmonary and systemic granulomatous disease.	No relevant occupational exposure history.
2	Tuberculosis (TB)	TB can cause granulomas involving lung, marrow, and heart.	AFB stains, cultures, and Quantiferon testing were all negative.
3	Atypical mycobacteria (NTM – e.g., MAC, M. kansasii)	NTM infections can involve lungs and bone marrow with granulomatous inflammation.	Negative cultures and no evidence of disseminated NTM infection.
4	Fungal infections (Histoplasmosis, Coccidioidomycosis, Blastomycosis)	These fungal infections may cause lung and marrow involvement with granulomas.	Negative fungal stains and cultures; no epidemiologic exposure history.
5	Brucellosis	Can cause marrow infiltration and cardiac involvement.	No exposure risk factors; negative serologies.
6	Lymphoma (Hodgkin & Non-Hodgkin)	May cause lung and marrow infiltration.	Histology revealed granulomas without malignant cells.
7	Leukemia	Can cause marrow and systemic involvement.	Blood counts and bone marrow morphology not consistent with leukemia.
8	Metastatic carcinoma	Can involve multiple organs including marrow.	Marrow biopsy showed no primary malignancy or metastatic cells.
9	Granulomatosis with polyangiitis (GPA)	Causes pulmonary granulomas and systemic vasculitis.	ANCA negative; histology lacked vasculitic features.
10	Eosinophilic granulomatosis with polyangiitis (EGPA/Churg–Strauss)	Features asthma, eosinophilia, and cardiac involvement.	No history of asthma; no eosinophilia.
11	Connective tissue diseases (e.g., SLE, MCTD)	Can cause multi-organ inflammation.	Typically non-granulomatous; autoimmune serologies negative.

12	Langerhans cell histiocytosis	Can involve lungs and bone marrow.	Histology showed granulomas, not Langerhans cells.
13	Amyloidosis	Causes multi-organ infiltration.	Congo red staining negative; pathology not consistent with amyloid.

The "Pivot": Hematologic and Bone Marrow Findings During the hospital course, the patient reported progressive, deep-seated bone pain in the hips and thighs. Laboratory evaluation revealed new-onset pancytopenia:

- Leukopenia: WBC 2.8 × 10^3/µL (Ref: 4.6–10.2)
- Normocytic Anemia: Hemoglobin 8.6 g/dL (Ref: 12.2–16.2), Hct 28.6%, MCV 86.7 fL
- Thrombocytopenia: Platelets 102 × 10^3/µL (Ref: 142–424)
- Morphology: RBC 3.30 × 10^6/µL, MCH 26.1 pg, and MCHC 30.1 g/dL.

Given the combination of bone pain and unexplained cytopenias, a bone marrow aspiration and core biopsy were performed. Histopathological examination revealed a slightly hypocellular marrow (25–30% cellularity) with preserved trilineage hematopoiesis. Crucially, the marrow contained multifocal non-caseating epithelioid granulomas without evidence of dysplasia or malignancy (blasts <1%). Special stains, including acid-fast bacilli (AFB), Fite, and Gomori methenamine silver (GMS), were negative for infectious etiologies, confirming bone marrow involvement by sarcoidosis.

Advanced Imaging and Targeted Therapy to further characterize myocardial involvement, cardiac positron emission tomography–computed tomography (PET-CT) was performed, as cardiac magnetic resonance imaging (MRI) was contraindicated due to a non-MRI-compatible AICD. PET-CT (Figure 2) demonstrated extensive, heterogeneous fluorodeoxyglucose (FDG) uptake throughout both ventricles, with a maximum standardized uptake value (SUV) of 6.7, most prominent in the left ventricle. These findings confirmed active cardiac sarcoidosis. Retrospective review suggested that the patient's prior refractory atrial fibrillation may have been an early arrhythmic manifestation of myocardial granulomatous infiltration. A timeline of events table has also been included for ease of readers (Table 2).

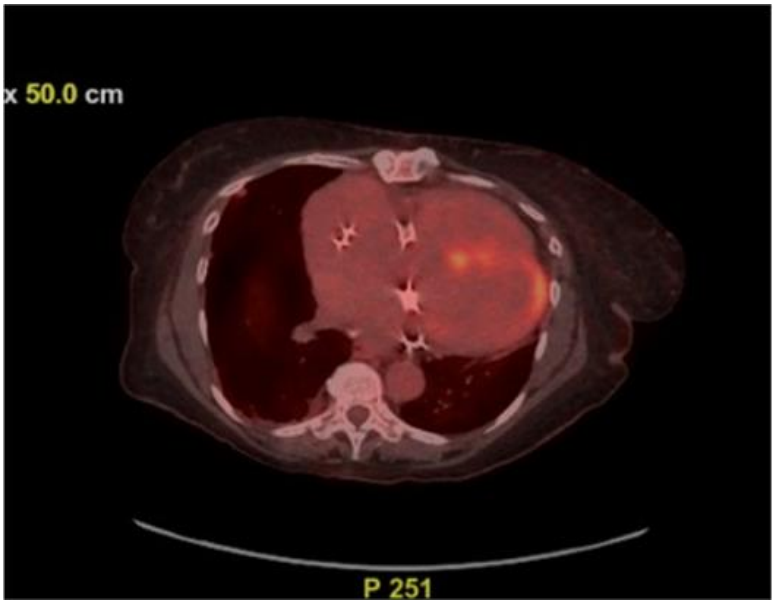

Figure 2: CT-PET showing increased uptake in the left and Right Ventricle

Table 2: Timeline of Events

Time point	Clinical Events	Diagnostic Findings	Management
Prior medical history	Known pulmonary sarcoidosis with prior atrial fibrillation requiring AICD placement	—	Chronic corticosteroid therapy
Initial presentation	Progressive dyspnea, lower-extremity edema, and worsening functional status	Clinical concern for cardiopulmonary disease	Hospital admission for evaluation
Early hospitalization	Persistent dyspnea and volume overload	Echocardiography showed reduced left ventricular systolic function suggesting possible cardiac involvement	Initiation of guideline-directed medical therapy for heart failure
Further evaluation	Progressive bone pain involving hips and thighs	Laboratory testing revealed **pancytopenia** (WBC 2.8 $\times 10^3/\mu L$, hemoglobin 8.6 g/dL, platelets 102 $\times 10^3/\mu L$)	Hematology consultation obtained
Bone marrow evaluation	Investigation of unexplained cytopenias	Bone marrow biopsy demonstrated **slightly hypocellular marrow (25–30% cellularity) with preserved trilineage hematopoiesis and multifocal non-caseating granulomas**; AFB, Fite, and GMS stains negative	Findings consistent with **bone marrow sarcoidosis**
Cardiac evaluation	Ongoing cardiopulmonary symptoms	FDG-PET demonstrated abnormal myocardial uptake consistent with **active cardiac sarcoidosis**	Multidisciplinary discussion involving cardiology, pulmonology, and hematology
Targeted treatment initiated	Management of systemic and cardiac disease	—	**Prednisone**, **sildenafil**, **ambrisentan**, and **guideline-directed heart failure therapy** (spironolactone, empagliflozin, enalapril, metoprolol); **atovaquone** for Pneumocystis prophylaxis
Hospital course	Gradual improvement in dyspnea and reduction in edema	Stable oxygenation and hemodynamics; no new arrhythmias on monitoring	Continued medical therapy
Discharge and follow-up	Clinical improvement with stable cardiopulmonary status	Ongoing surveillance for cardiac sarcoidosis and arrhythmias	Planned **multidisciplinary follow-up** with cardiology, pulmonology, and rheumatology; AICD interrogation and corticosteroid taper planned

TREATMENT

Management was directed by a multidisciplinary team including cardiology, pulmonology, and rheumatology, with treatment targeting pulmonary hypertension, systemic sarcoidosis, and heart failure related to suspected cardiac involvement.

Pulmonary Hypertension:

Therapy was initiated with sildenafil 20 mg orally three times daily and ambrisentan 10 mg orally once daily for treatment of pulmonary hypertension.

Systemic Sarcoidosis:

The patient continued prednisone 40 mg orally once daily for management of systemic sarcoidosis.

Infection Prophylaxis:

Given the anticipated need for prolonged immunosuppression, atovaquone was initiated for prophylaxis against Pneumocystis jirovecii pneumonia.

Heart Failure and Cardiac Involvement:

In the setting of suspected cardiac sarcoidosis with reduced left ventricular systolic function, clinicians-initiated guideline-directed medical therapy for heart failure, including spironolactone, empagliflozin, enalapril, and metoprolol.

FOLLOW-UP AND OUTCOMES

Following initiation of sildenafil, ambrisentan, and guideline-directed medical therapy for heart failure, the patient demonstrated progressive clinical improvement during hospitalization. Dyspnea gradually improved, accompanied by a reduction in heart failure manifestations, including decreased lower-extremity edema and improved functional status. Oxygenation remained stable on supplemental oxygen without escalation of respiratory support.

Serial physical examinations revealed improvement in cardiopulmonary findings, and follow-up chest radiography demonstrated interval improvement in pulmonary congestion and inflammatory changes. The patient remained hemodynamically stable throughout hospitalization, and continuous inpatient telemetry did not detect new arrhythmic events.

Given the high risk of cardiac involvement in sarcoidosis and the patient's prior history of atrial fibrillation with automatic implantable cardioverter-defibrillator placement, clinicians recommended close outpatient surveillance. At discharge, the patient received structured education regarding heart failure symptom recognition, arrhythmia warning signs, and strict medication adherence.

Post-discharge follow-up included monthly cardiology evaluation for monitoring of suspected cardiac sarcoidosis, with surveillance for arrhythmias, conduction abnormalities, and changes in ventricular function, in addition to periodic device interrogation of the AICD.

Further outpatient follow-up with pulmonology and rheumatology was arranged to monitor pulmonary sarcoidosis activity and guide corticosteroid tapering and immunosuppressive therapy.

DISCUSSION

The Tri-Organ Phenotype and Clinical Rarity: Sarcoidosis is a multisystem granulomatous disorder characterized by the formation of non-caseating granulomas, with pulmonary involvement occurring in approximately 90% of cases [2]. While extrapulmonary manifestations are well-documented, the simultaneous triad of pulmonary, cardiac, and bone marrow involvement represents an exceedingly rare phenotype. To our knowledge, only four previous cases of this specific triad exist in the literature. Our patient is distinct not only due to her advanced age (73 years) but also because of the confounding presence of Group 2 and 5 pulmonary hypertension, which initially masked the systemic nature of her disease. Furthermore, while historical cases have largely relied on monotherapy with high-dose corticosteroids, this case demonstrates the successful integration of infliximab to achieve clinical stability in an elderly patient with severe multiorgan dissemination.

Cardiac Sarcoidosis: Diagnostic Dilemmas in the Device Era: Cardiac sarcoidosis (CS) is a primary driver of morbidity and mortality due to its association with high-grade conduction blocks, ventricular arrhythmias, and progressive heart failure [5]. Although clinically evident CS is noted in only 5% of systemic sarcoidosis patients, autopsy and advanced imaging suggest subclinical myocardial involvement in 25-30% [3,4]. In this patient, the diagnosis was complicated by the presence of a non-MRI-compatible AICD. While endomyocardial biopsy remains the traditional "gold standard," its clinical utility is limited by low sensitivity (<20%) due to the patchy, mid-myocardial distribution of granulomas [6].

Consequently, multimodality imaging is essential. Following the Heart Rhythm Society (HRS) Expert Consensus guidelines, we utilized FDG-PET to circumvent the MRI contraindication. The intense myocardial FDG uptake (SUV 6.7) not only confirmed active inflammation but also provided a baseline for monitoring therapeutic response. This underscores the prognostic value of PET, as a higher inflammatory burden is a known predictor of adverse cardiovascular outcomes [3,7].

Bone Marrow Involvement and the Hematologic Differential: Bone marrow involvement occurs in 1-10% of sarcoidosis cases and frequently manifests as unexplained cytopenias due to granulomatous infiltration of the hematopoietic niche [8-10]. In this patient, the discovery of pancytopenia (WBC 2.8 $\times 10^3$/µL, Hb 8.6 g/dL, Platelets 102 $\times 10^3$/µL) necessitated a broad differential diagnosis. Clinicians must distinguish between direct marrow infiltration and secondary processes, such as sarcoidosis-induced immune thrombocytopenic purpura (ITP) or splenic sequestration [11].

Our histological confirmation of multifocal non-caseating granulomas, combined with negative stains for mycobacterial and fungal pathogens, was definitive. Excluding hematologic malignancies and nutritional deficiencies is paramount before attributing cytopenias to sarcoidosis, as the management strategies differ fundamentally [11].

The "Diagnostic Ceiling" of the Elderly: A central theme of this case is the "diagnostic ceiling" encountered in geriatric populations. In older patients, systemic granulomatous disease often presents as non-specific "frailty" or chronic fatigue, symptoms easily misattributed to the physiological decline of aging or pre-existing comorbidities like ischemic heart disease and chronic obstructive pulmonary disease. This "masking" effect likely contributes to the underrepresentation of elderly patients in disseminated sarcoidosis cohorts. Our case suggests that new-onset cytopenias or refractory arrhythmias in an older patient with a history of sarcoidosis, even if the disease has been stable for years, should trigger an aggressive search for extrapulmonary progression.

Multidisciplinary Management and the Role of Biologics: The management of tri-organ sarcoidosis requires a coordinated, multidisciplinary approach. While systemic corticosteroids 8remain the first-line intervention to prevent irreversible organ damage, their long-term use in the elderly is fraught with complications [12]. In this case, the severity of cardiac and marrow involvement justified the early initiation of infliximab, a tumor necrosis factor-α (TNF-α) inhibitor. Although typically reserved for refractory cases [13], the use of biologics in the acute phase of multi-organ disease may offer superior "steroid-sparing" benefits and more rapid disease suppression, particularly when cardiac stability is at risk [3,7].

CONCLUSION

This case illustrates a rare and life-threatening triad of pulmonary, cardiac, and bone marrow sarcoidosis. It serves as a clinical reminder that sarcoidosis is a dynamic disease that can transcend the "diagnostic ceiling" of aging. Early diagnosis via advanced imaging (FDG-PET) and histological confirmation, followed by the timely escalation to biologic therapy, is essential to mitigate the high mortality associated with disseminated disease.

KEY TAKE-HOME MESSAGES

- The Tri-Organ Sarcoidosis Phenotype: Simultaneous involvement of the lungs, heart, and bone marrow represents an exceedingly rare manifestation of disseminated sarcoidosis. The presence of this triad heralds an aggressive disease course and requires a high index of clinical suspicion, particularly when systemic symptoms are masked by the comorbidities of aging.
- Cardiac Sarcoidosis as a Prognostic Driver: Myocardial involvement is the primary determinant of mortality in systemic sarcoidosis. Clinicians must maintain a low threshold for cardiac evaluation in any patient presenting with new-onset arrhythmias, conduction disturbances, or heart failure, as early detection is vital to prevent sudden cardiac death.
- Hematologic "Red Flags": Unexplained pancytopenia in a patient with known sarcoidosis should not be attributed to chronic disease without investigation. Bone marrow biopsy is essential to differentiate direct granulomatous infiltration from secondary causes, such as malignancy or medication-induced toxicity, and to confirm disseminated disease.
- FDG-PET in the "Device Era": In the significant subset of patients where cardiac MRI is contraindicated due to non-MRI-compatible hardware (e.g., AICDs or older pacemakers), FDG-PET serves as a critical diagnostic and longitudinal monitoring tool to identify active myocardial inflammation and guide the escalation of immunosuppressive therapy.

PATIENT'S PERSPECTIVE

- "I have known about my sarcoidosis for years, but I never thought it could affect my heart and bone marrow."
- "The tests and biopsies were difficult to go through, but they finally gave me answers for my ongoing problems."

- "Hearing that my heart was involved was frightening because of the risk of rhythm problems and heart failure."
- "I feel comforted knowing that doctors from different specialties are working together on my care."

- "Although living with sarcoidosis is not easy, having a diagnosis and a clear treatment plan has given me hope and strength."

DECLARATIONS

Ethics approval and consent to participate: N/A

Consent to publish: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal. Availability of data and materials: Originally made by the author.

Competing interests: NIL

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Code Availability: Not Applicable

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