Case Report

An interesting case of cardiac sarcoidosis presenting as recurrent syncopal attacks

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ABSTRACT

The cardiac cause of syncope has traditionally been associated with conduction or valvular abnormalities. We present a rare case of isolated cardiac sarcoidosis in a 45-year-old Indian male, presenting with only syncope as the primary complaint. It remains a diagnosis of exclusion in such clinical presentations. Fluorodeoxyglucose positron emission tomography and cardiac magnetic resonance imaging (MRI) are newer modalities relied on for diagnosis. The patient was ultimately diagnosed based on cardiac MRI findings and correlating abdominal lymph-node biopsy. He was treated with oral steroids, and methotrexate was introduced gradually.

Key words: Cardiac magnetic resonance imaging, Cardiac sarcoidosis, Heart block, Syncope

Sarcoidosis is a multisystem granulomatous disease of unclear etiology, marked histologically by non-caseating granuloma. It commonly involves the lungs (up to 90% of cases), but extrapulmonary involvement is also not uncommon. In India, sarcoidosis remains underdiagnosed, with studies suggesting a prevalence of 10–12 cases/100,000 population [1]. Isolated cardiac sarcoidosis (CS) can have multiple varied presentations and is frequently missed due to its subtle, arrhythmogenic presentations or misdiagnosed as idiopathic conduction blocks. Sudden cardiac death can sometimes be the first and only presentation [2]. Cardiac involvement, though clinically evident in only 5% of sarcoidosis cases, is found in up to 25% of patients in autopsy studies [3,4].

We report this case to highlight isolated CS presenting solely as recurrent syncope, an extremely rare and diagnostically challenging manifestation. It underscores the importance of advanced imaging techniques such as cardiac magnetic resonance imaging (MRI) and fluorodeoxyglucose positron emission tomography (FDG-PET) [5,6], especially when systemic signs are minimal.

CASE REPORT

A 45-year-old Indian male driver with a 2-year history of diabetes mellitus and oral anti-diabetic agents experienced multiple episodes of transient syncopal attacks over 6 months.

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Table 1: Laboratory investigations on admission

Test	Value
Total leucocyte count	11.2×10^9/L
Hemoglobin	13.8 g/dL
Platelets	185×10^9/L
C-reactive protein	52 mg/L
Calcium	8.5 mg/dL
Sodium	140 mEq/L
Potassium	4.6 mEq/L
Creatinine	0.71 mg/dL
Alkaline phosphatase	354 U/L
Serum glutamic pyruvic transaminase	74 U/L

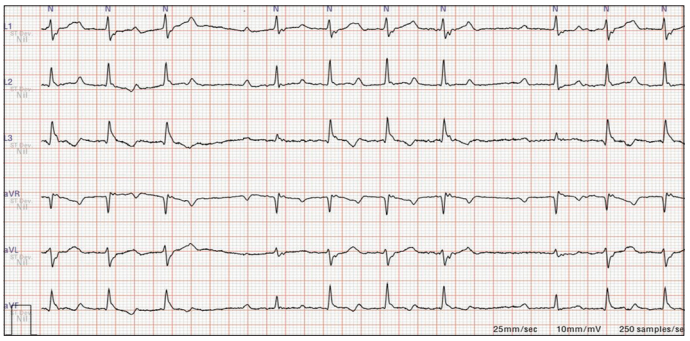

Figure 1: Electrocardiogram shows atrioventricular block changes

On presentation, he had stable vitals and a normal neurological examination. Initial differentials included hypoglycemia, atonic

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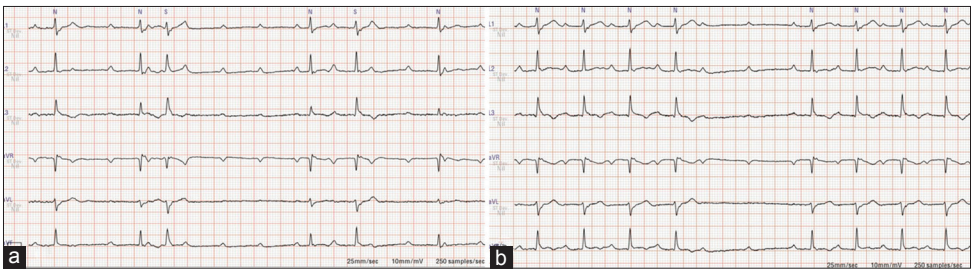

Figure 2: (a and b) 72 h Holter shows complete heart block with ventricular asystole at certain places

seizures, posterior circulation transient ischemic attack, and cardiac syncope.

Laboratory investigations on admission are shown in Table 1. Brain MRI and electroencephalogram were normal. However, initial electrocardiogram showed early right bundle branch block (RBBB) changes, which progressed to 2nd° atrioventricular block (AVB) over the course of hospitalization (Fig. 1). Holter monitoring for 72 h was done. Another episode of in-hospital syncope was noted; the Holter rhythm at that time revealed sinus pauses and brief ventricular asystole with complete heart block (Fig. 2). A permanent pacemaker was hence implanted.

Structural heart problems and valve abnormalities were first differentials, which were ruled out by a normal 2D echocardiography. A cardiac MRI (Fig. 3) revealed a patchy mid-myocardial fibrosis with lung infiltrates, and spleen involvement, suggesting chronic granulomatous disease. Differentials included CS and tuberculosis. The Mantoux test was negative. A computed tomography scan of the abdomen showed reactive lymphadenopathy. Abdominal lymph node biopsy revealed non-necrotizing granulomas. Serum angiotensin-converting enzyme levels were elevated (148 U/L), confirming systemic sarcoidosis, which presented solely with complaints related to cardiac involvement. The patient responded well to steroids, and on follow-up, low-dose methotrexate was added.

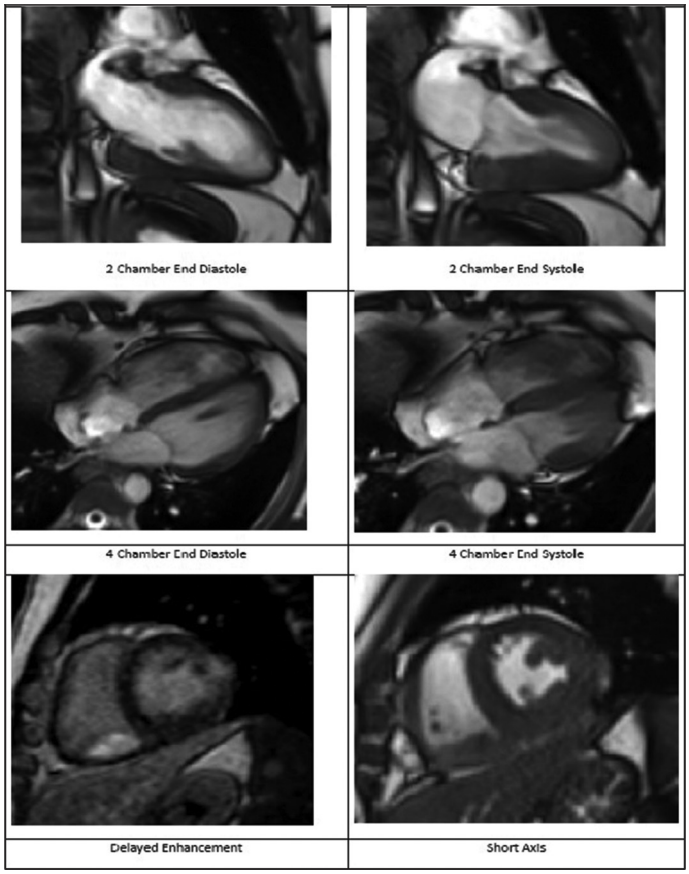

Figure 3: Cardiac magnetic resonance imaging shows myocardial fibrosis and delayed enhancement suggestive of sarcoidosis

DISCUSSION

In young patients presenting with cardiac syncope, only valvular diseases are thought of, and infiltrative disorders are usually missed. Conduction abnormalities as observed in our report are the most common electrophysiological manifestation of CS, with a prevalence up to 62% [7]. Complete heart block and bundle branch blocks have been reported in 23–30% and 12–32%, respectively, in CS patients [8]. A recent case report from Africa [9] showed similar such two cases having left bundle branch blocks, which is known to be less common compared to RBBBs [7,8]. Schuller *et al*. [10] found that among a cohort of patients with biopsy-proven pulmonary sarcoidosis, a bundle branch block pattern is associated with cardiac involvement.

Endomyocardial biopsy is considered the gold standard for diagnosing CS [11,12]. However, due to its patchy involvement, false negatives are common if the site of biopsy is not accurate. Differential diagnoses include cardiac amyloidosis, myocarditis, arrhythmogenic right ventricular cardiomyopathy, Chagas disease, and tuberculosis. Apart from only invasive approach, The heart rhythm society has two diagnostic pathways: (a) histological evidence of non-caseating granulomas in myocardium and (b) clinical diagnosis (histological evidence of extra- CS plus one or more of the following: steroid±immunosuppressant responsive cardiomyopathy or heart block, unexplained ejection fraction <40%, 2nd/3rd° AVB, patchy uptake of FDG-PET, and positive gallium uptake consistent with CS) [13,14].

Since heart rhythm disorders frequently cause death in CS patients, implantable cardiac defibrillators play a prominent role in their management [15]. It also reduces the risk of arrhythmic sudden cardiac death, which is highest in patients with lesions on MRI [16,17]. As done in our case, apart from starting therapy for sarcoidosis, urgently treating arrhythmia with pacemakers is essential. Guideline-directed medical therapy of heart failure and the use of anti-arrhythmic agents is recommended to maintain sinus rhythm in patients with atrial arrhythmia or symptomatic ventricular tachycardia [18]. The aim of therapy is to reduce myocardial inflammation to prevent fibrosis, arrhythmias, and

heart failure. Corticosteroids are first-line because of their efficacy and attainment of significant response over a relatively short period [19]. Tumor necrosis factor-alpha inhibitors (infliximab and adalimumab) have recently proven effective in steroid/non-steroidal immunosuppressant refractory cases [4,20]. In a cohort of 36 CS patients evaluated by Harper *et al*., the use of infliximab resulted in lower steroid doses and less dysrhythmia [21].

CONCLUSION

In patients with syncope and conduction abnormalities but a structurally normal heart on 2D echocardiography, infiltrative disorders such as CS should be considered. Cardiac MRI and FDG-PET help establish the diagnosis without the need for myocardial biopsy. Early immunosuppression therapy improves outcomes.

REFERENCES

1. Larry J, Dennis LK, Dan LL, Anthony SF, Stephen L. Disorders of immune-mediated injury. In: Hauser JL, editor. Harrison's Principles of Internal Medicine. 20th ed. New York: McGraw-Hill Education; 2018. p. 2600-7.
2. Hulten E, Aslam S, Osborne M, Abbasi S, Bittencourt MS, Blankstein R. Cardiac sarcoidosis-state of the art review. Cardiovasc Diagn Ther 2016;6:50-63.
3. Okada DR, Bravo PE, Vita T, Agarwal V, Osborne MT, Taqueti VR, *et al*. Isolated cardiac sarcoidosis: A focused review of an under-recognized entity. J Nucl Cardiol 2018;25:1136-46.
4. Trivieri MG, Spagnolo P, Birnie D, Liu P, Drake W, Kovacic JC, *et al*. Challenges in cardiac and pulmonary sarcoidosis: JACC state-of-the-art review. J Am Coll Cardiol 2020;76:1878-901.
5. Crouser ED, Maier LA, Wilson KC, Bonham CA, Morgenthau AS, Patterson 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.
6. Nunes H, Freynet O, Naggara N, Soussan M, Weinman P, Diebold B, *et al*. Cardiac sarcoidosis. Semin Respir Crit Care Med 2010;31:428-41.
7. Selan JC, Michaelson M, Fanburg BL, Estes NA. Evaluation and management of heart rhythm disturbances due to cardiac sarcoidosis. Heart Lung Circ 2014;23:1100-9.
8. Sekhri V, Sanal S, DeLorenzo LJ, Aronow WS, Maguire GP. Cardiac sarcoidosis: A comprehensive review. Arch Med Sci 2011;7:546-54.
9. Afriyie-Mensah JS, Awindaogo FR, Tagoe EN, Ayetey H. Cardiac sarcoidosis: Two case report. Clin Case Rep 2021;9:e04270.
10. Schuller JL, Olson MD, Zipse MM, Schneider PM, Aleong RG, Wienberger HD, *et al*. Electrocardiographic characteristics in patients with pulmonary sarcoidosis indicating cardiac involvement. J Cardiovasc Electrophysiol 2011;22:1243-8.
11. Evanchan JP, Crouser ED. Cardiac sarcoidosis: Recent advances in diagnosis and treatment and an argument for the need for a systematic multidisciplinary approach to management. J Innov Card Rhythm Manage 2013;4:1160-74.
12. Uemura A, Morimoto S, Hiramitsu S, Kato Y, Ito T, Hishida H. Histologic diagnostic rate of cardiac sarcoidosis: Evaluation of endomyocardial biopsies. Am Heart J 1999;138:299-302.
13. Liang JJ, Hebl VB, DeSimone VC, Madhavan M, Nanda S, Kapa S, *et al*. Electrogram guidance: A method to increase the precision and diagnostic yield of endomyocardial biopsy for suspected cardiac sarcoidosis and myocarditis. JACC Heart Fail 2014;2:466-73.
14. Chimenti C, Frustaci A. Contribution and risks of left ventricular endomyocardial biopsy in patients with cardiomyopathies: A retrospective study over a 28-year period. Circulation 2013;128:1531-41.
15. Birnie DH, Kandolin R, Nery PB, Kupari M. Cardiac manifestations of sarcoidosis: Diagnosis and management. Eur Heart J 2017;38:2663-70.
16. Franke KB, Marshall H, Kennewell P, Pham HD, Tully PJ, Rattanakosit T, *et al*. Risk and predictors of sudden death in cardiac sarcoidosis: A systematic review and meta-analysis. Int J Cardiol 2021;328:130-40.
17. Markatis E, Afthinos A, Antonakis E, Papanikolaou IC. Cardiac sarcoidosis: Diagnosis and management. Rev Cardiovasc Med 2020;21:321-38.
18. Kopriva P, Griva M, Tüdös Z. Management of cardiac sarcoidosis – A practical guide. Cor Vasa 2018;60:e155-64.
19. Ballul T, Borie R, Crestani B, Daugas E, Descamps V, Dieude P, *et al*. Treatment of cardiac sarcoidosis: A comparative study of steroids and steroids plus immunosuppressive drugs. Int J Cardiol 2019;276:208-11.
20. Baker MC, Sheth K, Witteles R, Genovese MC, Shoor S, Simard JF. TNF-alpha inhibition for the treatment of cardiac sarcoidosis. Semin Arthritis Rheum 2020;50:546-52.
21. Harper LJ, McCarthy M, Neto ML, Hachamovitch R, Pearson K, Bonanno B, *et al*. Infliximab for refractory cardiac sarcoidosis. Am J Cardiol 2019;124:1630-5.

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