Case Report Open Access

Surprising palpitation: An unexpected diagnosis of sarcoidosis

Hoda Raffiei Jelodar[1], Roxana Sadeghi[2, 3], Reza Hamneshin Behbahani[4,5*], Kimia Karimi Toudeshki[5]

1- Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, Iran
2- Prevention of Cardiovascular Disease Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran
3- Department of Cardiology, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran
4- Imam Hossein Educational Hospital, Shahid Madani Street, Imam Hossein Square, Tehran, Iran
5- School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran

Correspondence:

Reza Hamneshin Behbahani; Imam Hossein Educational Hospital, Shahid Madani Street, Imam Hossein Square, Tehran, Iran;
Email: rzbehbahani@gmail.com

Received: 2024.05.01
Accepted: 2025.03.12

How to cite this article:
Raffiei Jelodar H, Sadeghi R, Hamneshin Behbahani R, Karimi Toudeshki K. **Surprising palpitation: An unexpected diagnosis of sarcoidosis.** ARYA Atheroscler. 2025; 21(2): 3-9.

DOI:
https://doi.org/10.48305/arya.2024.42684.2968

Abstract

Sarcoidosis is a challenging disease characterized by the formation of granulomas in multiple organs. It presents with a variety of clinical symptoms, making diagnosis difficult for physicians. Myocardial involvement in sarcoidosis is rare, but it worsens prognosis and can lead to mortality. Physicians face challenges in diagnosing cardiac sarcoidosis due to its wide range of symptoms. Cardiac sarcoidosis that affects only the heart without involvement of other organs is extremely uncommon.

In this case, we examine the scenario of a middle-aged man who experienced palpitations and was diagnosed with ventricular tachycardia. He remained in stable condition without any other clinical signs suggestive of sarcoidosis. This case highlights the importance of considering sarcoidosis as a potential diagnosis in patients presenting with palpitations and ventricular tachycardia on electrocardiogram, particularly when these symptoms are accompanied by sudden new-onset systolic dysfunction despite normal coronary arteries.

Keywords: Case Report; Rare Presentation; Cardiac Sarcoidosis; Infiltrative Cardiomyopathy; Ventricular Tachycardia

Introduction

Sarcoidosis is a granulomatous disease affecting multiple organs, including the lungs, eyes, central nervous system, lymph nodes, and skin. An immune reaction to unidentified antigens, along with potential infectious, environmental, or occupational factors, has been proposed as the etiology of sarcoidosis[1,2].

Myocardial involvement in sarcoidosis is rare but worsens prognosis and may be fatal1. Among cardiac manifestations, conduction abnormalities, ventricular arrhythmia, and congestive heart failure have been reported more frequently in previous studies[3].

Sarcoidosis presents a diagnostic challenge for physicians due to its heterogeneous manifestations. Here, we discuss the case of a middle-aged man whose first presentation of sarcoidosis was palpitations and ventricular tachycardia (VT).

Case presentation

A 49-year-old man presented with an episode of palpitations accompanied by nausea and cold sweats, which began approximately an hour before admission. He did not report any chest pain or shortness of breath. The patient had a history of recurrent episodes over the past week preceding admission. This recurrence was confirmed by 24-hour ambulatory electrocardiogram monitoring, which recorded multiple episodes of non-sustained VT.

The patient was alert and awake, with a blood pressure of 90/65 mmHg and a regular pulse rate of 180 beats per minute. Except for tachycardia detected on cardiac auscultation, other physical examinations were unremarkable. He had a history of type 2 diabetes and was undergoing treatment with metformin.

The first electrocardiogram (EGC) in the emergency department showed ventricular tachycardia (VT) (Figure 1). Upon admission, echocardiography revealed mild left ventricular (LV) dysfunction and global hypokinesis with an ejection fraction (EF) of 45%. No left ventricular clot was observed. Additionally, mild mitral regurgitation and mild right ventricular (RV) enlargement were noted, with a systolic pulmonary artery pressure of 47 mmHg at rest. A redundant intra-atrial septum was identified, but no visible shunt flow was detected using a color Doppler study

As the patient had stable hemodynamics, an intravenous infusion of amiodarone was prescribed. However, due to a lack of response and a sudden drop in blood pressure, direct current (DC) shock was administered, successfully converting VT to sinus rhythm.

The post-conversion ECG showed T-wave inversion in leads V3–V6 and the inferior leads. Further evaluation included coronary angiography to rule out an ischemic cause of VT, which revealed normal coronary arteries. Consequently, structural alterations in the cardiac muscle were suspected.

Given the presence of VT and the development of new-onset LV dysfunction, infiltrative cardiomyopathies such as sarcoidosis, giant cell myocarditis, and lymphocytic myocarditis were considered in the differential diagnosis.

Clinical examination did not reveal any palpable lymph nodes or skin lesions. A high-resolution computed tomography (HRCT) scan of the lungs identified bilateral hilar lymphadenopathy (Figure 2). Cardiac magnetic resonance imaging (CMR) was performed to investigate potential cardiac muscle involvement (Figure 3).

CMR findings revealed mildly reduced LV and RV systolic function, as well as subepicardial to mid-wall gadolinium enhancement in the septal, inferior, and inferolateral LV segments, which was associated with diffuse myocardial edema. These findings were suggestive of active myocarditis. Given the presence of mediastinal lymphadenopathy—including bilateral hilar, right paratracheal, and subcarinal lymph nodes—further evaluation for sarcoidosis was recommended.

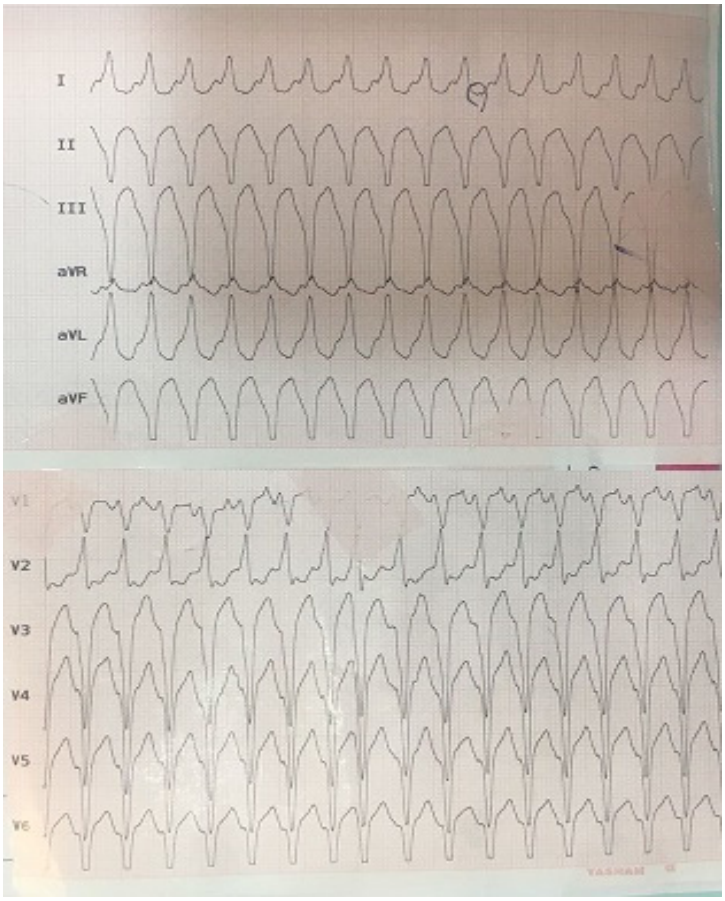

Figure 1. Electrocardiogram (ECG) shows monomorphic VT with the morphology of the left bundle branch. Discordance ECG in the emergency room showed monomorphic VT of left-bundle branch morphology indicating an origin from the right ventricle, transition at V3

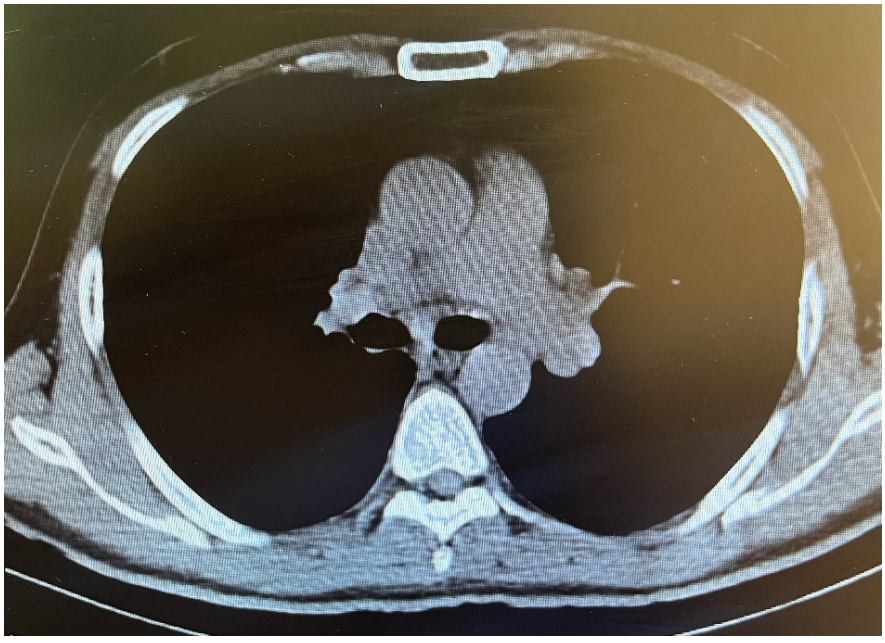

Figure 2. This CT scan shows bilateral hilar lymphadenopathy

Following CMR, a PET scan was performed. The PET scan revealed heightened metabolic activity in specific areas of the heart, suggesting an ongoing inflammatory process, most likely in the progressive stage of sarcoidosis. Several hypermetabolic lymph nodes, as well as hypermetabolic lesions in the lungs and spleen, were detected, along with a single osseous lesion in the left iliac region.

Despite these findings, the PET scan was inconclusive in differentiating between sarcoidosis, tumoral involvement, and lymphoproliferative disorders. Consequently, an endomyocardial biopsy (EMB) was performed. However, the EMB results failed to establish a specific diagnosis. As is known, EMB may be conclusive in only 30% of cases involving cardiac sarcoidosis.

After cardiac sarcoidosis was diagnosed, methylprednisolone pulse therapy was initiated. During the course of treatment, a VT episode occurred. For VT management, amiodarone was administered intravenously; however, it induced QT prolongation. As a result, amiodarone was discontinued, and Mexiletine 200 mg twice daily was prescribed.

Continuous cardiac monitoring confirmed

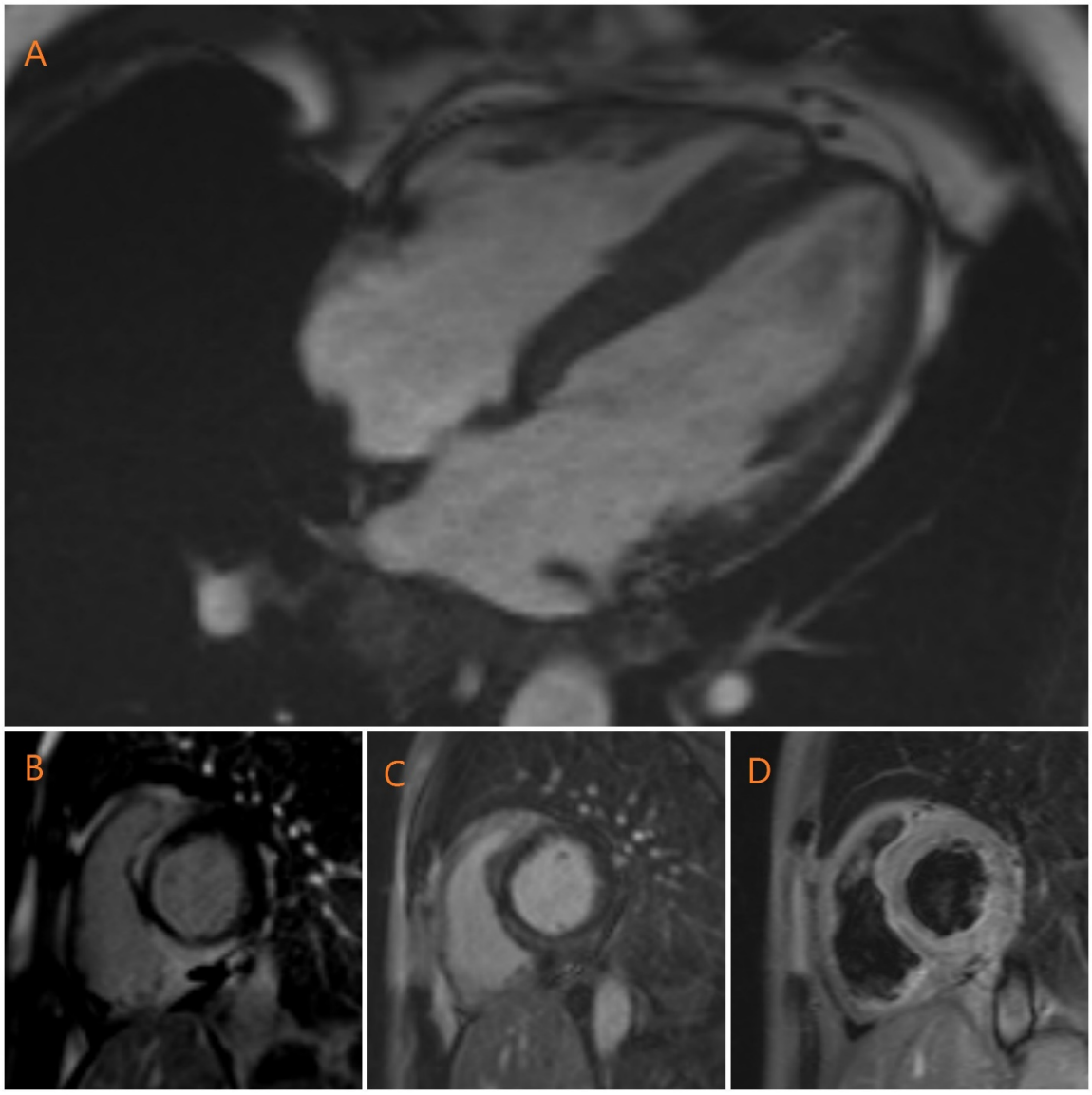

Figure 3. Cardiac Magnetic Resonance imaging suggesting sarcoidosis

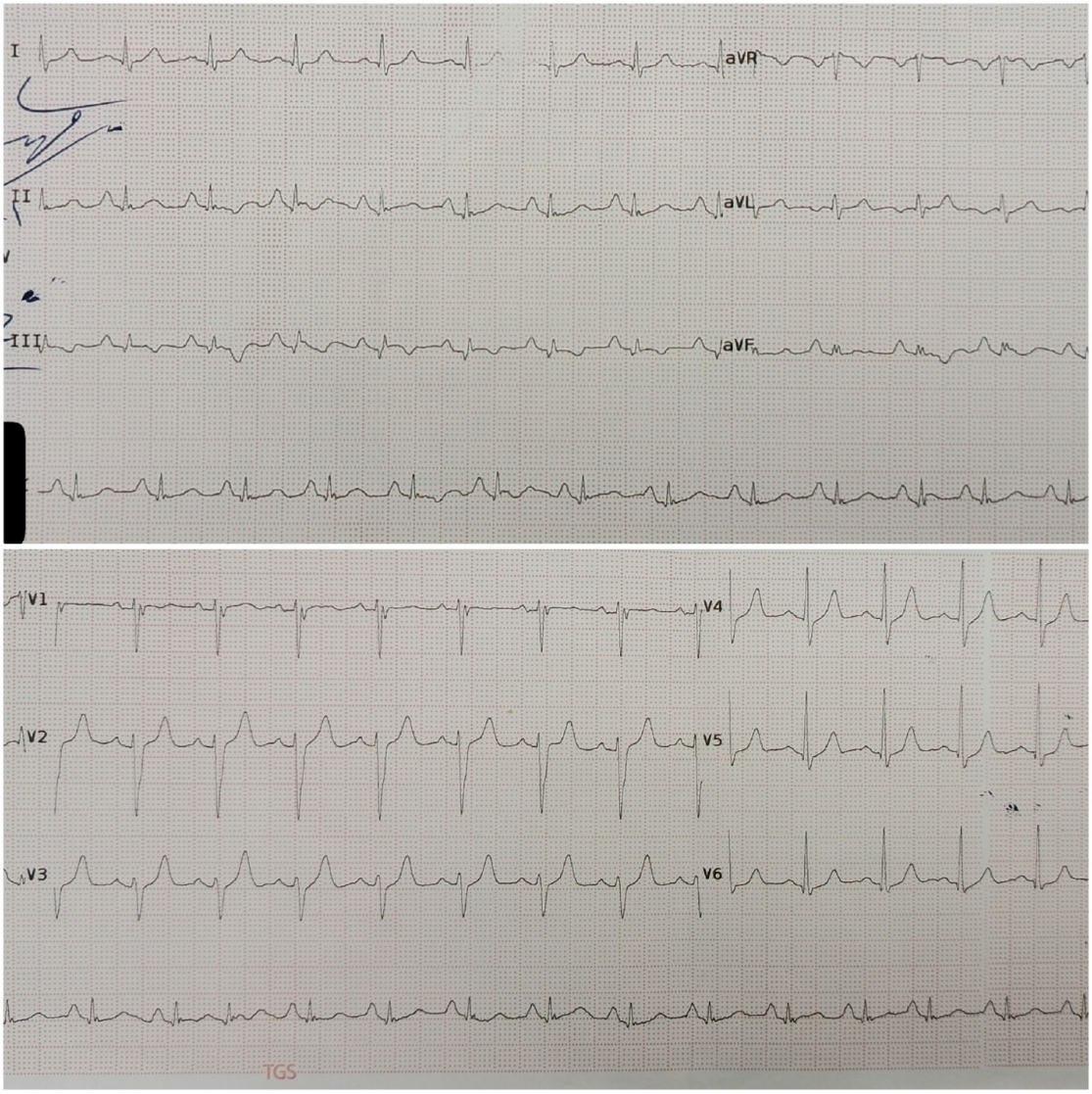

Figure 4. ECG of the patient at the time of discharge which shows normal sinus rhythm

that no additional VT episodes occurred during hospitalization. Additionally, the previously inverted T waves returned to a normal state. The ECG at the time of discharge is shown in Figure 4.

Pulmonology, neurology, ophthalmology, and rheumatology consultations were performed for further evaluation of sarcoidosis. All specialists agreed that no further tests or treatments were required.

He finally was scheduled for implantable cardioverter defibrillator (ICD) insertion to prevent future episodes of VT. Upon discharge, his ultimate EF was 45-50%. Presently, the patient is receiving azathioprine, prednisolone, empagliflozin, bisoprolol, eplerenone, and sacubitril as part of guideline-directed medical therapy (GDMT for heart failure.

Discussion

We report a middle-aged man who presented with hemodynamically stable VT without any clinical features suggestive of sarcoidosis. Sarcoidosis is a multisystem granulomatous disease with various presentations, most commonly affecting the lungs[4]. One of its least common manifestations is cardiac involvement,

with an incidence of 5% in patients, though significantly higher (20–25%) at autopsy[2,4].

Cardiac involvement presents with a wide range of signs and symptoms, from asymptomatic electrocardiographic (ECG) abnormalities to manifestations of heart failure—including dyspnea, orthopnea, peripheral edema—as well as arrhythmia- or conduction-mediated palpitations, syncope, dizziness, and even sudden death. Its clinical presentation may depend on the location and extent of granulomatous involvement, which may also be influenced by ethnicity and disease duration[5].

Cardiac sarcoidosis granulomas most frequently affect the myocardium, although any location within the heart may be involved[6]. The most commonly affected myocardial sites are the left ventricular free wall and ventricular septum, followed by the right ventricle and atrial walls[6].

Diagnosing cardiac sarcoidosis is challenging due to its broad range of clinical presentations, particularly when palpitations are the initial symptom, as they have numerous differential diagnoses. Therefore, an echocardiogram may help narrow the diagnostic possibilities by identifying supportive findings such as new-onset systolic cardiac dysfunction, regional wall motion abnormalities, ventricular septum thickness, and bright shadows suggestive of infiltrative disorders though these findings are not specific.

As a next step, cardiac MRI with gadolinium enhancement can be useful in evaluating suspected infiltrative diseases such as sarcoidosis[7,8]. Although endomyocardial biopsy has been proposed as a confirmatory test, previous studies report a sensitivity of only 30% for detecting sarcoidosis granulomas due to their patchy infiltration[9]. Consequently, this invasive diagnostic tool may not be commonly recommended.

Ventricular arrhythmias are common among cardiac involvements of Sarcoidosis and probably happen due to macro re-entry around the granuloma. As some previous studies have shown, the VT was resistant to antiarrhythmic drug (Amiodarone) used for rhythm conversion and ICD may be recommended for prevention and treatment of ventricular arrhythmias, the fatal manifestation of cardiac sarcoidosis[10]. Of course, it should be mentioned that no episodes of sustained or non-sustained VT happened following Mexiletine 200 mg BID prescription, which can suggest the idea of investigating the effect of this drug in the prevention of arrhythmia with ventricular origin in long-term follow-up in these patients.

Conclusion

This case presentation emphasizes considering sarcoidosis in patients presenting with palpitations and VT on ECG, especially if accompanied by new-onset systolic dysfunction in the presence of normal coronary arteries. Additionally, given the reported resistance to antiarrhythmic drugs, especially amiodarone, in most previous case reports—and considering the high cost, invasiveness, complications, and the impact of ICD implantation on quality of life further studies on oral antiarrhythmic drugs, including mexiletine, to control and prevent VT episodes in patients with sarcoidosis are recommended.

Acknowledgements

We acknowledge our patient for agreeing to the publication of her data in this paper to improve clinical practice. We also acknowledge the Clinical Research Development Unit of Imam Hossein Hospital, Shahid Beheshti University of Medical Sciences, for approving this paper.

Conflict of interests

The authors declare no conflict of interest.

Funding

There is no funding in this study.

Author's Contributions

Study Conception or Design: HRJ, RS
Data Acquisition: RS, RHB
Manuscript Drafting: RHB
Critical Manuscript Revision: KKT
All authors have approved the final manuscript and are responsible for all aspects of the work.

AI clarification

The authors used QuillBot for language editing. They then rechecked the content and take full responsibility for the accuracy of the publication.

References

1. Kim JS, Judson MA, Donnino R, Gold M, Cooper LT Jr, Prystowsky EN, et al. Cardiac sarcoidosis. Am Heart J. 2009 Jan;157(1):9-21. https://doi.org/10.1016/j.ahj.2008.09.009
2. Tavora F, Cresswell N, Li L, Ripple M, Solomon C, Burke A. Comparison of necropsy findings in patients with sarcoidosis dying suddenly from cardiac sarcoidosis versus dying suddenly from other causes. Am J Cardiol. 2009 Aug 15;104(4):571-7. https://doi.org/10.1016/j.amjcard.2009.03.068
3. Doughan AR, Williams BR. Cardiac sarcoidosis. Heart. 2006 Feb;92(2):282-8. https://doi.org/10.1136/hrt.2005.080481
4. Iannuzzi MC, Rybicki BA, Teirstein AS. Sarcoidosis. N Engl J Med. 2007 Nov 22;357(21):2153-65. https://doi.org/10.1056/nejmra071714
5. Rosenfeld LE, Chung MK, Harding CV, Spagnolo P, Grunewald J, Appelbaum J, et al. Arrhythmias in Cardiac Sarcoidosis Bench to Bedside: A Case-Based Review. Circ Arrhythm Electrophysiol. 2021 Feb;14(2):e009203. https://doi.org/10.1161/circep.120.009203
6. Roberts WC, McAllister HA Jr, Ferrans VJ. Sarcoidosis of the heart. A clinicopathologic study of 35 necropsy patients (group 1) and review of 78 previously described necropsy patients (group 11). Am J Med. 1977 Jul;63(1):86-108. https://doi.org/10.1016/0002-9343(77)90121-8
7. Sharma S. Cardiac imaging in myocardial sarcoidosis and other cardiomyopathies. Curr Opin Pulm Med. 2009 Sep;15(5):507-12. https://doi.org/10.1097/mcp.0b013e32832ea4c7
8. Youssef G, Beanlands RS, Birnie DH, Nery PB. Cardiac sarcoidosis: applications of imaging in diagnosis and directing treatment. Heart. 2011 Dec;97(24):2078-87. https://doi.org/10.1136/hrt.2011.226076
9. 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 Aug;138(2 Pt 1):299-302. https://doi.org/10.1016/s0002-8703(99)70115-8
10. Grimm W, Alter P, Maisch B. Arrhythmia risk stratification with regard to prophylactic implantable defibrillator therapy in patients with dilated cardiomyopathy. Results of MACAS, DEFINITE, and SCD-HeFT. Herz. 2004 May;29(3):348-52. https://doi.org/10.1007/s0005 9-004-2578-0