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CASUISTIC PAPER

Acute myocarditis mimicking ST – elevation myocardial infarction in a young adult with pharyngitis – a case report

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ABSTRACT

Introduction and aim. Acute myocarditis (AM) is a life-threatening inflammatory disease that manifests with a highly variable range of clinical symptoms, sometimes mimicking those of myocardial infarction. The aim of this report was to describe the diagnostic challenges of AM.

Description of the case. A 22-years old male previously diagnosed with pharyngitis arrived in the emergency room (ER) with retrosternal chest pain. The electrocardiogram (ECG) showed ST elevation in inferior and posterior leads and reciprocal changes with ST depression in anterolateral leads. Laboratory tests revealed elevated cardiac enzymes and bedside echocardiogram (ECHO) revealed hypokinesis of the inferior wall. Initial diagnosis of ST elevation myocardial infarction (STEMI) was made. Coronary angiogram showed normal epicardial coronary arteries and cardiac magnetic resonance imaging (CMRI) revealed subepicardial late gadolinium enhancement (LGE).

Conclusion. This case was proven challenging due to the unusual ECG and ECHO findings, mimicking inferoposterior STEMI. The need for available angiography and CMRI was mandatory for the final diagnosis of AM.

Keywords. CMRI, myocardial infarction, myocarditis

Introduction

Acute myocarditis (AM) is a life-threatening disease caused by viral, and less frequently bacterial infection, autoimmune diseases or exposure to drugs that elicit an inflammatory response in cardiac myocytes. In the emergency setting, patients with the suspicion of AM are usually evaluated based on their symptoms, electrocardiogram (ECG), echocardiogram (ECHO) and laboratory test markers. The clinical presentation is usually nonspecific and can be misinterpreted especially in rare cases which mimics ST elevation myocardial infarction (STEMI).[1] We report a young male, previously diagnosed with pharyngitis, that presented in the emergency room (ER) with a clinical presentation of inferoposterior STEMI.

Aim

The aim of this report was to describe the diagnostic challenges of AM.

Description of the case

A 22-years old male presented in the ER with continuous, non-radiating retrosternal crushing chest pain and diaphoresis lasting for three hours. Five days prior to the admission the patient was diagnosed with pharyngitis. However, one day ago, he visited a private physician with complaints of chest pain of similar character with his present symptoms. The ECG at the time showed nonspecific ST abnormalities in leads II, III, AVF, V5, V6 and his cardiac ECHO was normal. From past med-

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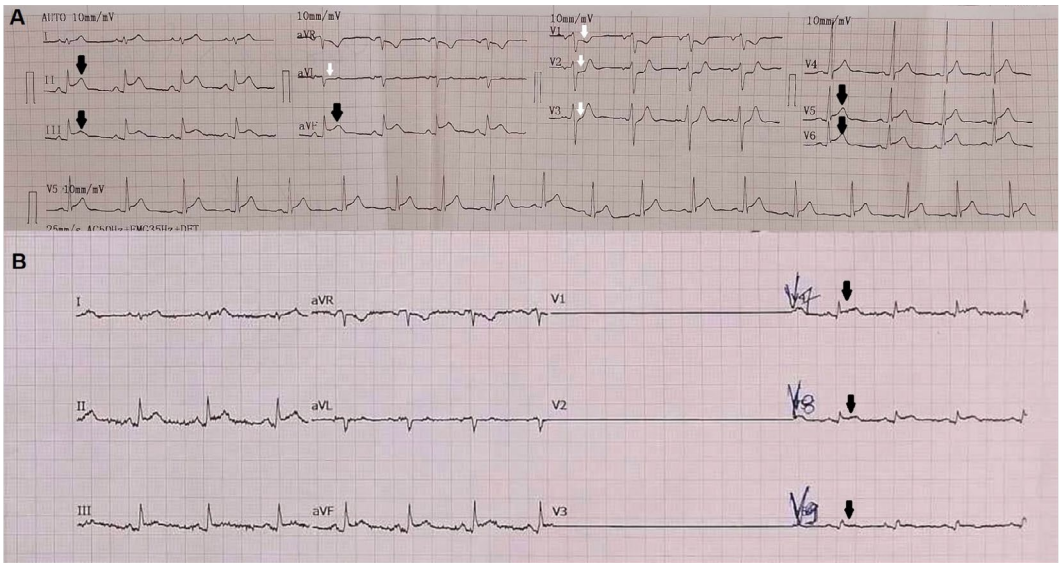

Fig. 1. A: PR depression, ST elevation in leads II, III, AVF, V5 and V6 (black arrows) and reciprocal changes with ST depression in leads V1, V2, V3, AVL (white arrows), B: ST elevation in posterior placed leads V7-V9 (black arrows)

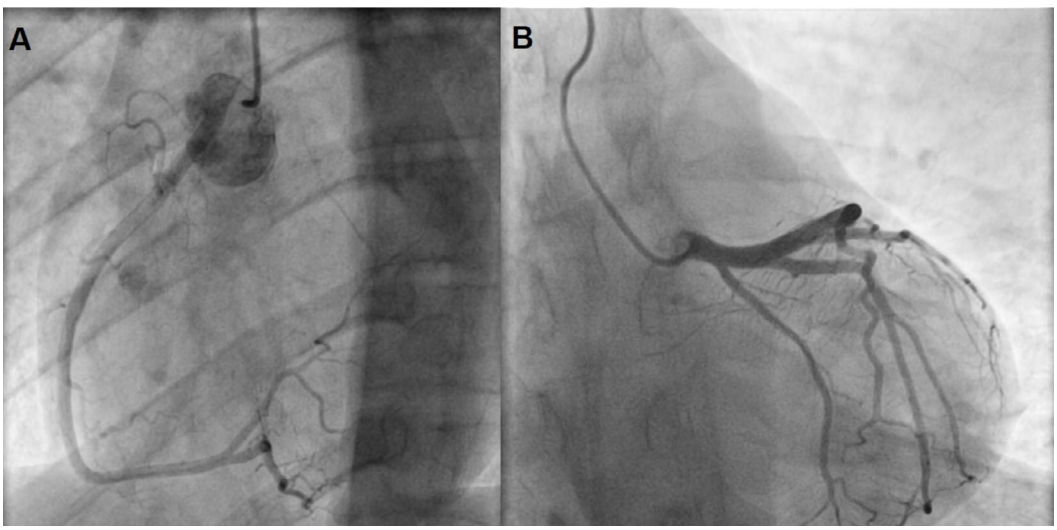

Fig. 2. A: Right coronary artery, B: Left coronary artery

ical history the patient had no allergies, no usage of recreational drugs, no chronic illnesses or cardiovascular risk factors. The family history revealed the incidence of myocarditis in his father and sister a few years prior. Additionally, due to the recent COVID- 19 pandemic, the vaccination status was acquired, and the patient proved to be fully vaccinated.

At presentation his heart rate was 83/min and his blood pressure 151/59 mmHg. The temperature was 37.3°C and SaO_2 99. The initial clinical exam showed normal heart sound S1, S2 and absence of any audible murmurs, gallops or rubs.

Laboratory test showed elevated cardiac markers, creatinine kinase (CK) of 1032 IU/l (reference range [rr]: 39–308), troponin hs of 11502 pg/mL (ng/) (rr: <72), and lactate dehydrogenase (LDH) of 395 IU/l (rr: 81-230). Further results included, white blood cell (WBC) 8,10 K/µl (rr: 4–10), hemoglobin (Hg) of 13.1 g/dL (rr: 11.7–15.7), platelets (PLT) of 295 K/µL (rr: 140–440), blood glucose (BG) of 109 mg/dL (rr: 74–106), creatinine (Cr) of 1.1 (rr: 0.6–1.3), urea (BUN) of 21 mg/dL (rr: 10–45), prothrombin time (PT) of 11.8 sec (rr: 10–13), fibrinogen (Fg) of 470 mg/dL (rr: 200–400) and C-reactive protein (CRP) of 4.3 mg/dL (rr: 0–0.5). The ECG revealed sinus rhythm 1:1, PR depression, ST elevation in leads II, III, AVF, V5 to V9 and reciprocal changes with ST depression in leads V1, V2, V3, AVL (Fig. 1A and 1B) indicating regional ischemic injury of the inferoposterior segments supplied by the posterior descending artery (PDA). A bedside ECHO was also conducted and showed hypokinesis of the inferior wall, left ventricular ejection fraction (LVEF) >60%, E>A, mild mitral regurgitation, inferior vena cava (IVC) diameter 1.6 cm with normal inspiratory collapse, and

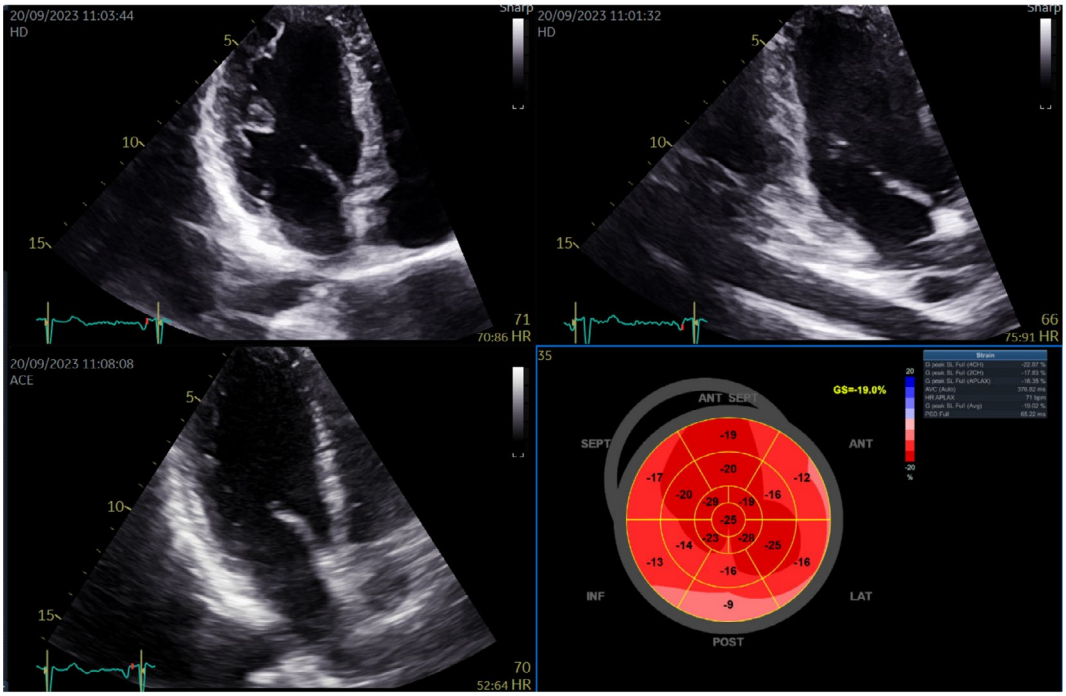

Fig. 3. Two-dimensional speckle tracking echocardiography

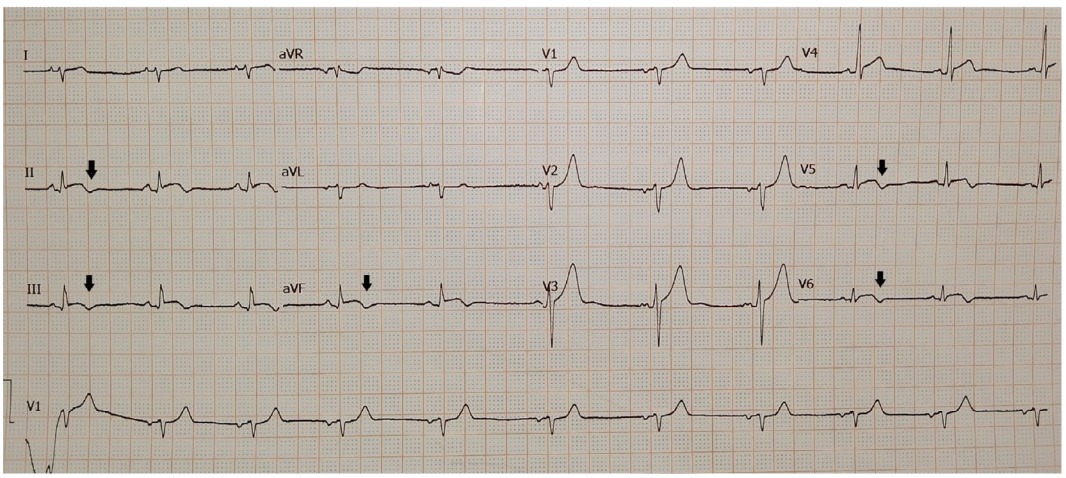

Fig. 4. ECG evolution with ST normalization and T inversion in leads II, III, AVF, V5, V6 (black arrows) 24h after the admission

no pericardial fluid buildup. The chest x-ray showed a normal size heart, clear lung fields and sharp costodiaphragmatic angles.

Based on the clinical presentation of chest pain, elevated cardiac markers, ECG and ECHO abnormalities initial diagnosis of inferoposterior myocardial infarction was made. The patient received a loading dose of 250 mg of aspirin and was transferred for primary coronary intervention (PCI). Coronary angiography was performed but revealed normal epicardial coronary arteries (Fig. 2).

In respect to the earlier results of angiography and the history of pharyngitis, acute coronary syndrome (ACS) was excluded and differential diagnosis of AM was made.

On the second day of hospitalization two-dimensional speckle tracking echocardiography was performed. Global longitudinal strain (GLS) curves using semi-automatic algorithm (GE Healthcare) reveal peak systolic strain equal to –19% (Fig. 3). Basal inferior and basal inferolateral segments were affected, areas that electrocardiographically had already been characterized by ischemic changes. Additional ECG 24 hours later showed resolving of ST changes with normalization of ST segments and inversion of T waves, mimicking the ECG evolution of an ACS after reperfusion of the culprit artery (Fig. 4).

Cardiac magnetic resonance imaging (CMRI) for confirmation of AM was performed. T2- weighted short time recovery (STIR) image showed increased subepicardial signal in inferior and posterior wall of the left ventricle (LV) suggestive of myocardial edema. In post

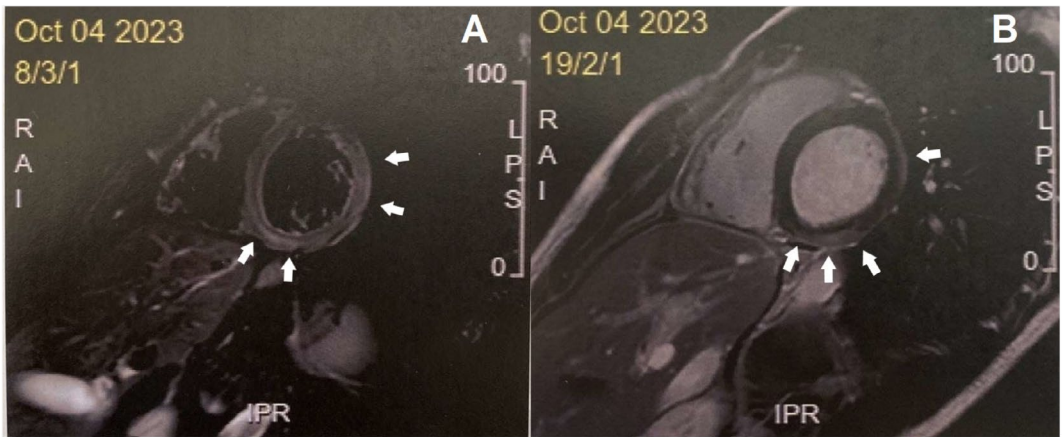

Fig. 5. A: T2-W STIR image showing intense subepicardial rim (white arrows) representing myocardial edema in the inferior and posterior wall of the LV, B: LGE image showing subepicardial scarring with intense signal in inferior and posterior wall of LV (white arrows)

contrast late gadolinium enhancement (LGE) images acquired with a T1- weighted segment inversion recovery gradient echo sequence, an increased subepicardial signal was also observed in the inferior and posterior wall of the LV indicative of myocardial scarring (Figure 5). Finally, the hypothesis was confirmed, and the final diagnosis was made.

Furthermore, etiological causes were investigated throughout the duration of hospitalization. Upon admission influenza A/B rapid diagnostic test and Covid 19 rapid antigen test were negative. Serological testing revealed negative results for cytomegalovirus (CMV) and hepatitis C virus (HCV) but positive IgM with negative IgG antibodies for Epstein Barr virus (EBV). Due to the recent diagnosis of pharyngitis antistreptolysin O titer and throat swab culture test were also conducted but both were negative.

The patient was treated with beta blockers and angiotensin converting enzyme (ACE) inhibitors. During the hospitalization a 24-hour Holter was implanted but did not reveal any abnormalities. At the follow-up one week later there was remission of the symptoms and the patient was discharged from the hospital.

Discussion

This case represents an AM, resembling a STEMI, in the setting of pre-diagnosed pharyngitis in a 22-year-old male patient. Myocarditis is an inflammatory condition, that can be caused by viral or less commonly by bacterial agents. Virus genomes that have been most identified in western countries are adenovirus, enterovirus, CMV, parvovirus B19, human immunodeficiency virus (HIV), as well as influenza and HCV.[2,3] Bacteria such as group A streptococcus (GAS) infections had been well documented as a causative agent of AM with clinical presentation of myocardial infarction, but rarely in absence of rheumatic fever.[4]

The presentation of AM is heterogeneous and overlapping with other clinical entities, ranging from asymptomatic to arrhythmias and even acute heart failure.[5] Clinical characteristics often include fever, diaphoresis, dyspnea, fatigue, cardiac arrhythmias, palpitations and chest pain being the most frequent symptom.[6-8] In general, recent infections or related symptoms should always be forewarned during history taking. Already published case series regarding cases of GAS related myocarditis found prevalence, especially in males with a mean age of 27.8 and latency period of pharyngitis to chest pain of 4.2 days.[10-12] Cardiac enzymes are elevated in most cases proving that these findings can be often misleading in these patients.[8]

Diagnostic modalities, such as ECG shows non-specific ST changes, T inversion, q waves, and even, atrioventricular (AV) blocks.[12] More specifically, in myocarditis, two ST elevation patterns have been described; a pericarditis pattern with elevation less than 5 mm, involving diffusely both limb and precordial leads, with the exception of AVR and V1, which often presents with reciprocal ST depression, and a typical myocardial infarction like pattern, characterized by J-point elevation and an flat or convex ST segment, in at least two contiguous leads, often without reciprocal ST depression.[13,14] It was found that no correlation between ST elevation and regional necrosis in CMRI was present, although in our case of myocardial infarction like type, the affected segments were related to the ECG leads that showed ischemic changes.[15,16] Regarding T wave inversion, the prevalence in AM is 9–48%, and a late manifestation in leads with previous ST elevation. T wave inversion was found to be independently related to the extent of both myocardial necrosis, as assessed later by CMRI.[17,18]

Transthoracic ECHO is generally not useful for the differentiation between myocardial infarction and myocarditis. In previous studies patients with AM showed,

regional movement abnormalities, reduced left ventricular ejection fraction and pericardial effusion.[12]

CMRI with LGE, has emerged as the cornerstone in the diagnosis of AM, especially in cases which mimics STEMI, being able to diagnose ischemic from non-ischemic pattern. In ischemic pattern, LGE always involves subendocardial layers with or without transmural extent, whereas, in non-ischemic pattern, LGE doesn't have the same characteristics, being mid-wall, subepicardial, or mixed.[11,19] The use of CMRI is of pivotal importance, but unfortunately, it is not always available in the ER. Instead, these cases are transferred for angiography, and when normal epicardial coronary arteries are found, suspicion of other etiologies are made.

Endomyocardial biopsy is considered the gold standard for diagnosis of myocarditis. Unfortunately, its invasive nature, the time needed for the results, and the likelihood of sampling errors, makes it less applicable in clinical practice.[20]

This case report was affected by three notable limitations. First, the inability to conduct serological tests for multiple viral agents limited us to establish cause- effect relationship of the AM. Second, the case report is being conducted retrospectively, recall bias might prevent us in collecting additional information from the patient, the family or other health care professionals. Finally, better understanding of this clinical presentation and the appropriate early management could be derived by studies on larger population of patients with AM mimicking STEMI.

Conclusion

AM remains a diagnostic challenge for physicians due to the range of clinical presentations. The present case, specifically shares many similarities with inferoposterior STEMI, evidenced by regional ST segment elevation with reciprocal changes and combination with echocardiographic regional hypokinesis of the corresponding segment. In cases of young patients evaluated for ACS, with absence of predisposing factors and normal coronary angiogram, CMRI is mandatory to establish the diagnosis.

Declarations

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Author contributions

Conceptualization, A.C. and A.K.; Methodology, A.K.; Validation, A.C. and N.V.; Formal Analysis, A.C., A.K. and N.V.; Investigation, A.C. and N.V.; Resources, A.C. and N.V.; Data Curation, A.K.; Writing – Original Draft Preparation, A.K.; Writing – Review & Editing, A.K.; Visualization, A.C. and A.K.; Supervision, A.C.; Project Administration, A.C.

Conflicts of interest

The authors declare no conflict of interest in preparing this article.

Data availability

The data sets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Ethics approval

Informed consent was obtained from the patient.

References

1. Lasica R, Djukanovic L, Savic L, et al. Update on Myocarditis: From Etiology and Clinical Picture to Modern Diagnostics and Methods of Treatment. *Diagnostics (Basel).* 2023;13(19):3073. doi: 10.3390/dignostics13193073
2. Yajima T, Knowlton KU. Viral myocarditis: from the perspective of the virus. *Circulation.* 2009;119(19):2615-2624. doi: 10.1161/CIRCULATIONAHA.108.766022
3. Nielsen TS, Hansen J, Nielsen LP, Baandrup UT, Banner J. The presence of enterovirus, adenovirus, and parvovirus B19 in myocardial tissue samples from autopsies: an evaluation of their frequencies in deceased individuals with myocarditis and in non-inflamed control hearts. *Forensic Sci Med Pathol.* 2014;10(3):344-350. doi: 10.1007/s12024-014-9570-7
4. Gore I, Saphir O. Myocarditis associated with acute nasopharyngitis and acute tonsillitis. *Am Heart J.* 1947;34(6):831-851. doi: 10.1016/0002-8703(47)90148-8
5. Al-Akchar M, Shams P, Kiel J. *Acute Myocarditis*. In: StatPearls. Treasure Island (FL): StatPearls Publishing; July 5, 2023.
6. Grün S, Schumm J, Greulich S, et al. Long-term follow--up of biopsy-proven viral myocarditis: predictors of mortality and incomplete recovery. *J Am Coll Cardiol.* 2012;59(18):1604-1615. doi: 10.1016/j.jacc.2012.01.007
7. Hufnagel G, Pankuweit S, Richter A, Schönian U, Maisch B. The European Study of Epidemiology and Treatment of Cardiac Inflammatory Diseases (ESETCID). First epidemiological results. *Herz.* 2000;25(3):279-285. doi:10.1007/s000590050021
8. Schmutzler L, Mirna M, Hoppe UC, Lichtenauer M. From Streptococcal Pharyngitis/Tonsillitis to Myocarditis: A Systematic Review. *J Cardiovasc Dev Dis*. 2022;9(6):170. doi: 10.3390/jcdd9060170
9. Talmon Y, Gilbey P, Fridman N, Wishniak A, Roguin N. Acute myopericarditis complicating acute tonsillitis: beware the young male patient with tonsillitis complaining of chest pain. *Ann Otol Rhinol Laryngol.* 2008;117(4):295-297. doi: 10.1177/000348940811700409
10. Mokabberi R, Shirani J, M AH, Go BD, Schiavone W. Streptococcal pharyngitis-associated myocarditis mimicking acute STEMI. *JACC Cardiovasc Imaging.* 2010;3(8):892-893. doi: 10.1016/j.jcmg.2010.05.012

11. Upadhyay GA, Gainor JF, Stamm LM, Weinberg AN, Dec GW, Ruskin JN. Acute nonrheumatic streptococcal myocarditis: STEMI mimic in young adults. *Am J Med.* 2012;125(12):1230-1233. doi: 10.1016/j.amjmed.2012.06.016
12. Caraco J, Arnon R, Raz I. Atrioventricular block complicating acute streptococcal tonsillitis. *Br Heart J.* 1988;59(3):389-390. doi: 10.1136/hrt.59.3.389
13. Hanna EB, Glancy DL. ST-segment elevation: Differential diagnosis, caveats. *Cleve Clin J Med.* 2015;82(6):373-384. doi: 10.3949/ccjm.82a.14026
14. Chen S, Hoss S, Zeniou V, et al. Electrocardiographic Predictors of Morbidity and Mortality in Patients With Acute Myocarditis: The Importance of QRS-T Angle. *J Card Fail.* 2018;24(1):3-8. doi: 10.1016/j.cardfail.2017.11.001
15. Deluigi CC, Ong P, Hill S, et al. ECG findings in comparison to cardiovascular MR imaging in viral myocarditis. *Int J Cardiol.* 2013;165(1):100-106. doi: 10.1016/j.ijcard.2011.07.090
16. Nucifora G, Miani D, Di Chiara A, et al. Infarct-like acute myocarditis: relation between electrocardiographic findings and myocardial damage as assessed by cardiac magnetic resonance imaging. *Clin Cardiol.* 2013;36(3):146-152. doi: 10.1002/clc.22088
17. De Lazzari M, Zorzi A, Baritussio A, et al. Relationship between T-wave inversion and transmural myocardial edema as evidenced by cardiac magnetic resonance in patients with clinically suspected acute myocarditis: clinical and prognostic implications. *J Electrocardiol.* 2016;49(4):587-595. doi: 10.1016/j.jelectrocard.2016.04.002
18. de Bliek EC. ST elevation: Differential diagnosis and caveats. A comprehensive review to help distinguish ST elevation myocardial infarction from nonischemic etiologies of ST elevation. *Turk J Emerg Med*. 2018;18(1):1-10. doi: 10.1016/j.tjem.2018.01.008
19. Aquaro GD, De Gori C, Faggioni L, et al. Diagnostic and prognostic role of late gadolinium enhancement in cardiomyopathies. *Eur Heart J Suppl.* 2023;25(Suppl C):C130--C136. doi: 10.1093/eurheartjsupp/suad015
20. Kang M, Chippa V, An J. *Viral Myocarditis*. In: StatPearls. Treasure Island (FL): StatPearls Publishing; November 20, 2023.