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Case Reports

Lyme Carditis and Inflammation-Driven Plaque Erosion Presenting as Sudden Cardiac Arrest

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Abstract

Lyme carditis represents a rare cardiac complication of *Borrelia burgdorferi* infection, often causing conduction disturbances but rarely causing malignant arrhythmias. Inflammatory acute coronary syndrome, driven by immune-mediated plaque erosion rather than rupture, represents a nontraditional ischemic mechanism. This case highlights their overlap. The case of a previously healthy man with Lyme disease who experienced cardiac arrest because of ventricular tachycardia is reported. Imaging showed myocardial inflammation together with coronary plaque erosion instead of plaque rupture. The patient underwent advanced diagnostic testing and received multidisciplinary medical care, which led to complete cardiac recovery and implantable cardioverter-defibrillator placement.

Keywords: Lyme disease; myocarditis; pneumonia; multimodal imaging; dynamic contrast enhanced magnetic resonance imaging

Case Report

Presentation and Physical Examination

A 72-year-old man with no relevant medical history had had a tick bite, developed an erythema migrans rash, tested positive for Lyme immunoglobulin M, and was treated with doxycycline. Twenty days later, he had just finished mowing his lawn when he experienced sudden-onset lightheadedness and shortness of breath; shortly thereafter, he collapsed. Upon arrival of emergency medical services, who witnessed the cardiac arrest, the patient's initial cardiac rhythm was ventricular tachycardia (VT). He underwent synchronized cardioversion but went into ventricular fibrillation arrest. After cardiopulmonary resuscitation, frequent shocks, and a total dose of 450 mg amiodarone, the patient regained spontaneous circulation. The patient received 11 shocks total and was brought to the nearest level 1 trauma center.

Medical History

Twenty days earlier, the patient presented at another facility with typical erythema migrans rash on a lower extremity (Fig. 1). Lyme infection was confirmed by positive titers of Lyme immunoglobulin M (and negative titers of immunoglobulin G). He did not have fever, chest pain, heart palpitations, abdominal pain, or dyspnea at that time or afterward. He was treated with doxycycline 100 mg twice daily for 10 days. He reported completing this treatment.

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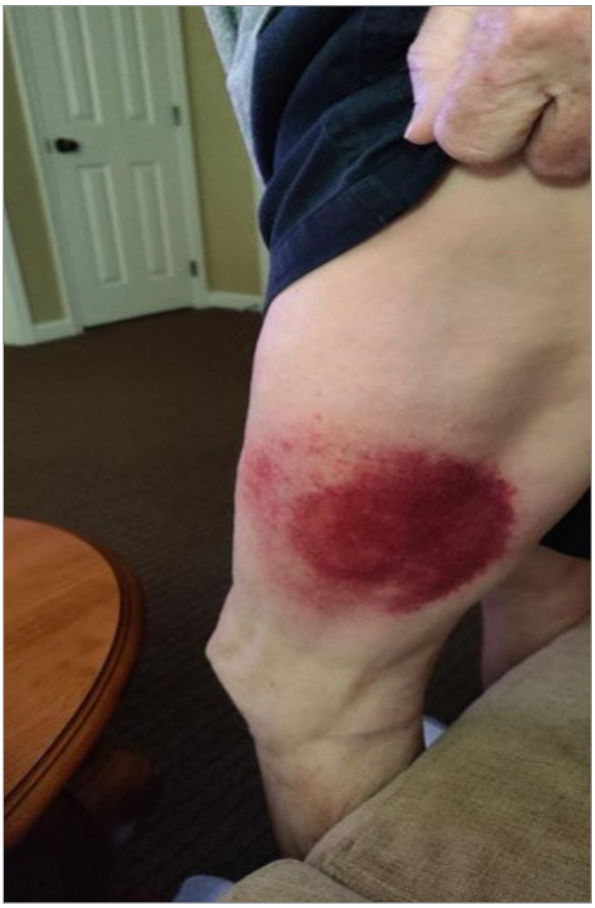

Fig. 1 Photograph shows the patient's erythema migrans rash.

Key Points

- Lyme carditis can present with malignant arrhythmias beyond atrioventricular block, including sustained VT and cardiac arrest, especially in patients with patchy myocardial inflammation and fibrosis.
- Systemic inflammation in Lyme disease may contribute to ACS by way of plaque erosion, even in the absence of traditional cardiovascular risk factors or plaque rupture.
- Advanced cardiac imaging—including cardiac MRI and intravascular ultrasonography—is essential for differentiating ischemic from inflammatory pathology and guiding appropriate therapeutic decisions, including implantable cardioverter-defibrillator placement.
- Dual pathology involving infectious myocarditis and coronary plaque instability underscores the need for broad diagnostic consideration in patients with cardiac arrest, particularly in Lyme-endemic regions and in patients without conventional atherosclerotic profiles.
- A multidisciplinary team approach is recommended for determining the best course of action in these rare cases.

Abbreviations

ACS, acute coronary syndrome
LGE, late gadolinium enhancement
LV, left ventricular
MRI, magnetic resonance imaging
VT, ventricular tachycardia
LVFWR is a rare and fatal complication of AMI.

Supplementary Materials

For supplemental materials, please see the online version of this article.

Differential Diagnosis

On arrival, after recovery of spontaneous circulation, the patient's electrocardiogram (Fig. 2) showed no conduction abnormalities but did reveal anterolateral ischemia. His high-sensitivity troponin I level was elevated at 0.048 μg/L (48 ng/L) and peaked at 1.8 μg/L (1,796 ng/L). Laboratory tests showed clinically significant leukocytosis (white blood cell count, 34×10^9/L), severe metabolic acidosis (pH, 7.0), and lactic acid at 14 mmol/L. At this time, the working diagnoses for the cause of cardiac arrest were acute coronary syndrome (ACS), stress-induced cardiomyopathy, and acute Lyme carditis.

Technique

After discussion with the multidisciplinary shock team, a decision was made not to proceed with temperature-targeted management. Inserting a Swan-Ganz catheter produced the following hemodynamic findings: central venous pressure of 10 mm Hg; pulmonary artery pressure of 37/16 mm Hg; myocardial volume of oxygen of 67%; cardiac output/input (Fick formula) of 4.15/2; and systemic vascular resistance of 1,213 dynes/s/cm^{-5}, suggesting cardiogenic shock. The patient was supported with intravenous dobutamine, lidocaine, amiodarone, and heparin. Transthoracic echocardiography revealed severely impaired left ventricular (LV) systolic function (Fig. 3), indicated by an LV ejection fraction of 15% to 19%. There was anterior, anteroseptal, and apical hypokinesis. Because of the regional wall motion abnormality, there was a high index of suspicion for ACS. The patient underwent left heart catheterization soon after recovery of spontaneous circulation, which revealed 80% stenosis of the proximal left anterior descending coronary artery and Thrombolysis in Myocardial Infarction grade 2 flow (Fig. 4).

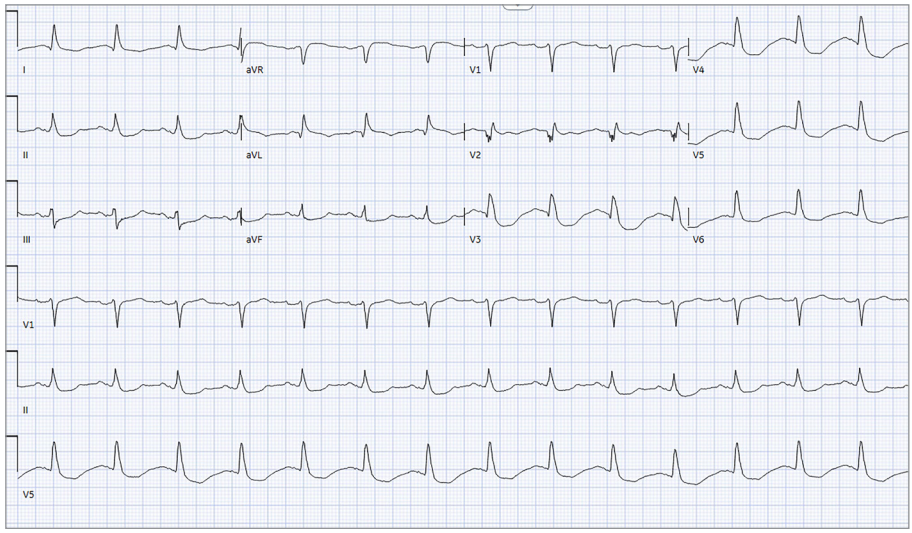

Fig. 2 Electrocardiogram obtained on presentation to the reporting center shows normal sinus rhythm and pronounced ST-segment depression in the anterolateral and inferior leads.

Interestingly, intravascular ultrasonography revealed no evidence of acute plaque rupture, but there was plaque erosion (Fig. 5). The patient underwent successful intravascular stenting that resulted in Thrombolysis in Myocardial Infarction grade 3 flow (Supplemental Figure 1, Movie 6). He was supported for 3 days with inotropes and intra-aortic balloon pump placement for Society for Cardiovascular Angiography and Interventions stage C(A) cardiogenic shock, and clinical improvement was seen after coronary revascularization.

Because the arterial lesion in the left anterior descending coronary artery did not have plaque-wall rupture and did not completely explain the patient's cardiac arrest, ACS was not believed to be the sole cause of the patient's symptoms, so cardiac magnetic resonance imaging (MRI) with late gadolinium enhancement (LGE) was performed. It revealed dense, patchy enhancement at the midanteroseptal, inferolateral, anterolateral, inferoseptal, and apical inferior segments, with associated edema on T2 imaging but no evidence of infarct (Fig. 6; Supplemental Figure 2). No thoracic lymphadenopathy was noted.

Given the patient's history of Lyme disease and the pattern of LGE, acute Lyme carditis was diagnosed. Because the patient had presented with sudden cardiac arrest, the decision was made to start intravenous steroid infusion for the Lyme carditis and to continue ceftriaxone infusion. Repeat transthoracic echocardiography showed a full recovery of LV function, with an ejection fraction of 60% (Supplemental Figure 3, Movies 7-9). Because cardiac MRI had revealed nonischemic scar tissue as a possible arrhythmogenic substrate for cardiac arrest, an implantable cardioverter-defibrillator was placed for secondary prevention.

Latest Follow-Up

One month later, to follow up on the previously found pattern of LGE, cardiac 2-[fluorine-18] fluoro-2-deoxy–d-glucose positron emission tomography was performed. The imaging was negative for cardiac sarcoidosis.

Discussion

Lyme carditis is an uncommon but clinically significant cardiac manifestation of *Borrelia burgdorferi* infection. It typically presents with conduction abnormalities, most often atrioventricular block, but its clinical spectrum extends to life-threatening arrhythmias and global myocardial dysfunction. The underlying pathophysiology involves direct myocardial invasion by the spirochete and an intense lymphocytic and macrophagic immune response.[1] In addition, molecular mimicry and antibody cross-reactivity may augment myocardial inflammation. The majority of Lyme carditis cases are associated with high-degree atrioventricular block, but this patient's presenting rhythm was a sustained monomorphic VT, a rare but critical arrhythmia that is likely caused by patchy myocardial inflammation and fibrosis. These structural changes create an arrhythmogenic substrate conducive to reentrant circuits. Ischemia, even without infarction, can also contribute to VT by increasing repolarization dispersion, particularly when

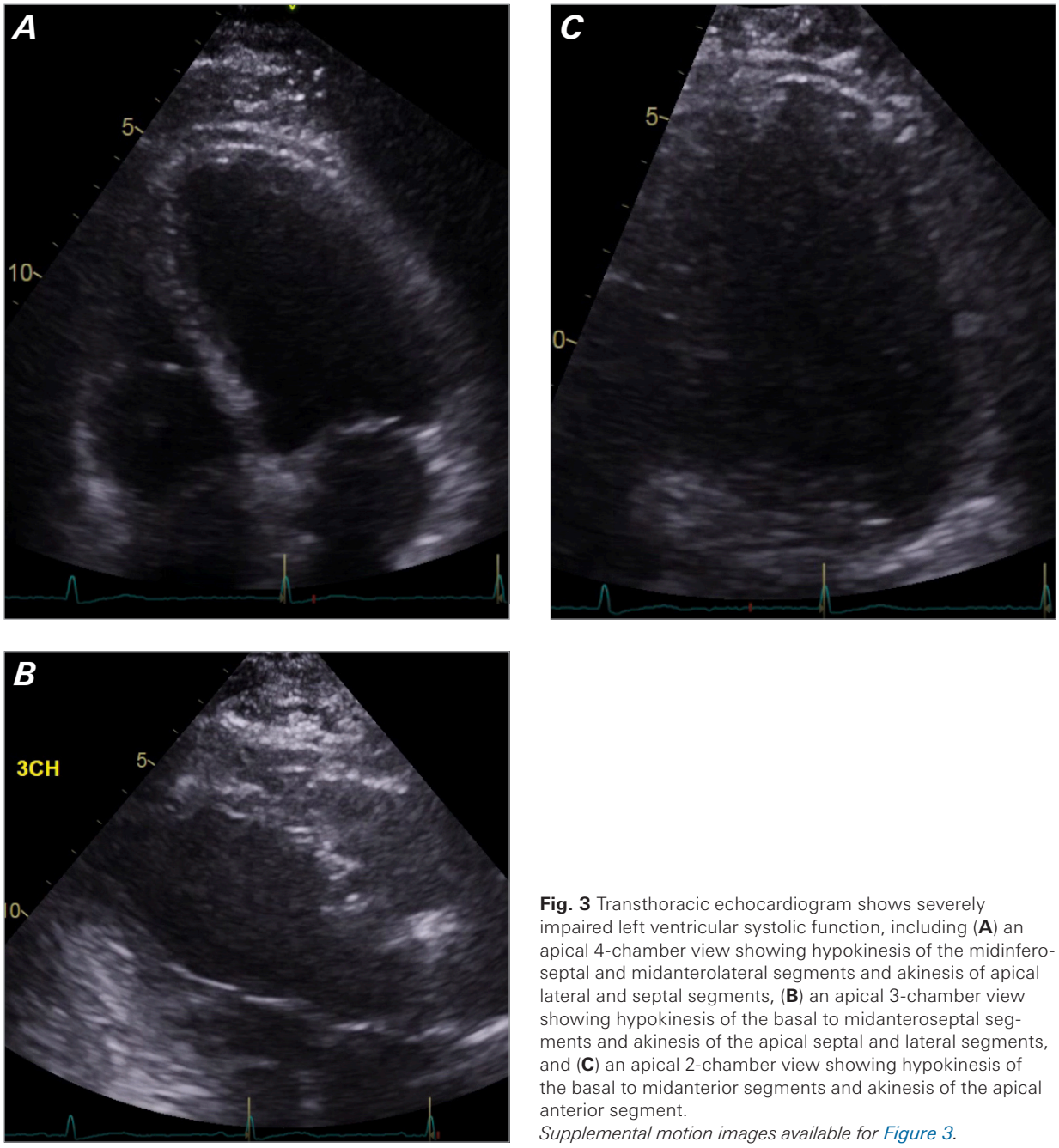

Fig. 3 Transthoracic echocardiogram shows severely impaired left ventricular systolic function, including (**A**) an apical 4-chamber view showing hypokinesis of the midinferoseptal and midanterolateral segments and akinesis of apical lateral and septal segments, (**B**) an apical 3-chamber view showing hypokinesis of the basal to midanteroseptal segments and akinesis of the apical septal and lateral segments, and (**C**) an apical 2-chamber view showing hypokinesis of the basal to midanterior segments and akinesis of the apical anterior segment.
Supplemental motion images available for Figure 3.

it is localized to the septum or the free wall of the left ventricle.[2]

Cardiac MRI was crucial in diagnosing myocarditis. Findings included midwall and patchy LGE in noncoronary distributions, which is different from ischemic injury patterns. These LGE patterns not only confirm a diagnosis of Lyme carditis but also provide prognosis because they are associated with elevated risk of arrhythmias and sudden cardiac death. Full recovery of LV function after antimicrobial and immunosuppressive therapy supports the inflammatory outcome of the injury, but persistent LGE may indicate ongoing arrhythmic risk.[3]

The patient had a proximal stenosis in the left anterior descending coronary artery that raised concerns about ACS. Intravascular ultrasonography revealed no plaque rupture but rather plaque erosion, a different pathologic mechanism that is often a result

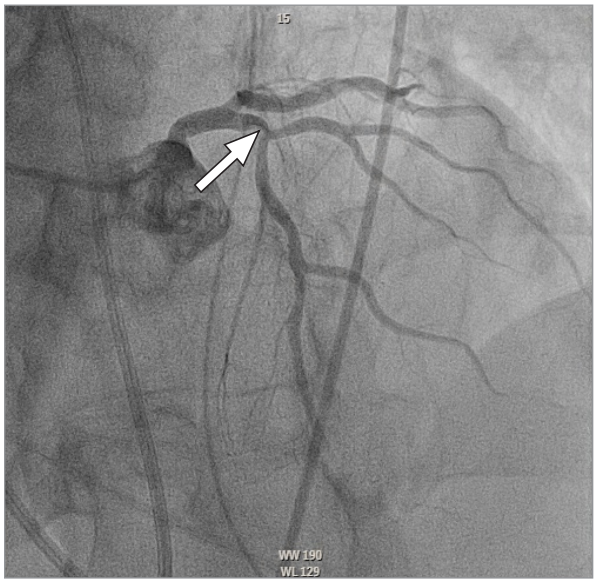

Fig. 4 Left heart angiogram shows a lesion in the proximal to mid-left anterior descending coronary artery. *Supplemental motion image available for Figure 4.*

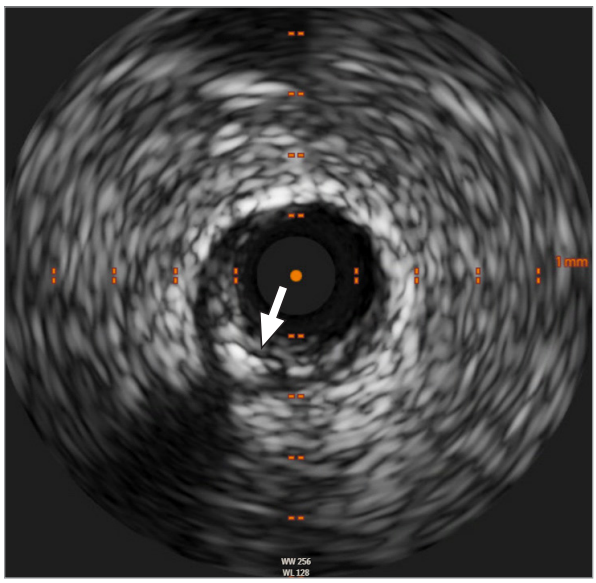

Fig. 5 Intravascular ultrasonogram shows plaque in the left anterior descending coronary artery with erosion (arrow). *Supplemental motion image available for Figure 5.*

of inflammation-induced endothelial apoptosis and dysfunction. Plaque erosion is increasingly recognized as a cause of ACS, especially in patients who do not have the typical features of rupture. It can occur in the setting of systemic inflammation, which destabilizes endothelial integrity through damage mediated by cytokines, particularly interleukin 6 and tumor necrosis factor α, promoting thrombosis without rupture.[4]

In this patient's case, systemic inflammation may have caused coronary endothelial activation, vasomotor dysfunction, and therefore plaque erosion leading to ischemia. This dual pathology—coexistent Lyme carditis and erosion-driven ACS—emphasizes how systemic infection can influence coronary pathophysiology beyond the traditionally recognized atherosclerotic mechanisms. Plaque erosion can have the angiographic features of stenosis and reduced flow without rupture features, so high-resolution modalities such as intravascular ultrasonography must be used to establish the exact pathology.

This case also indicates that inflammatory diseases such as Lyme disease can precipitate ACS through mechanisms that are not related to rupture. It expands the pathophysiologic framework within which clinicians should consider cardiac arrest in Lyme-endemic areas, particularly in patients with no traditional cardiovascular risk factors.

Because of the arrhythmic event, the recovery of the ejection fraction, and the presence of myocardial LGE, the patient was advised to undergo implantable cardioverter-defibrillator placement for secondary prevention. This case highlights the importance of advanced imaging, inflammation-targeted management, and rhythm monitoring in cases of myocarditis and ACS overlap.

Conclusion

Whereas Lyme carditis usually presents as heart block, the patient in the current report had a rare presentation of VT. This case highlights the rare and clinically complex intersection of Lyme carditis and inflammation-mediated ACS. The co-occurrence of ventricular arrhythmia with myocardial inflammation and plaque erosion in a patient without previous heart disease shows how systemic infection can disrupt coronary physiology. This disruption probably results from Lyme disease's inflammatory effects, which cause an increase in metabolic demand and generate instability in the atherosclerotic plaque, causing ACS.

The advanced techniques of cardiac MRI and intravascular ultrasonography proved essential both for diagnosing and for treating the patient because there was no evidence of plaque rupture. This case illustrates

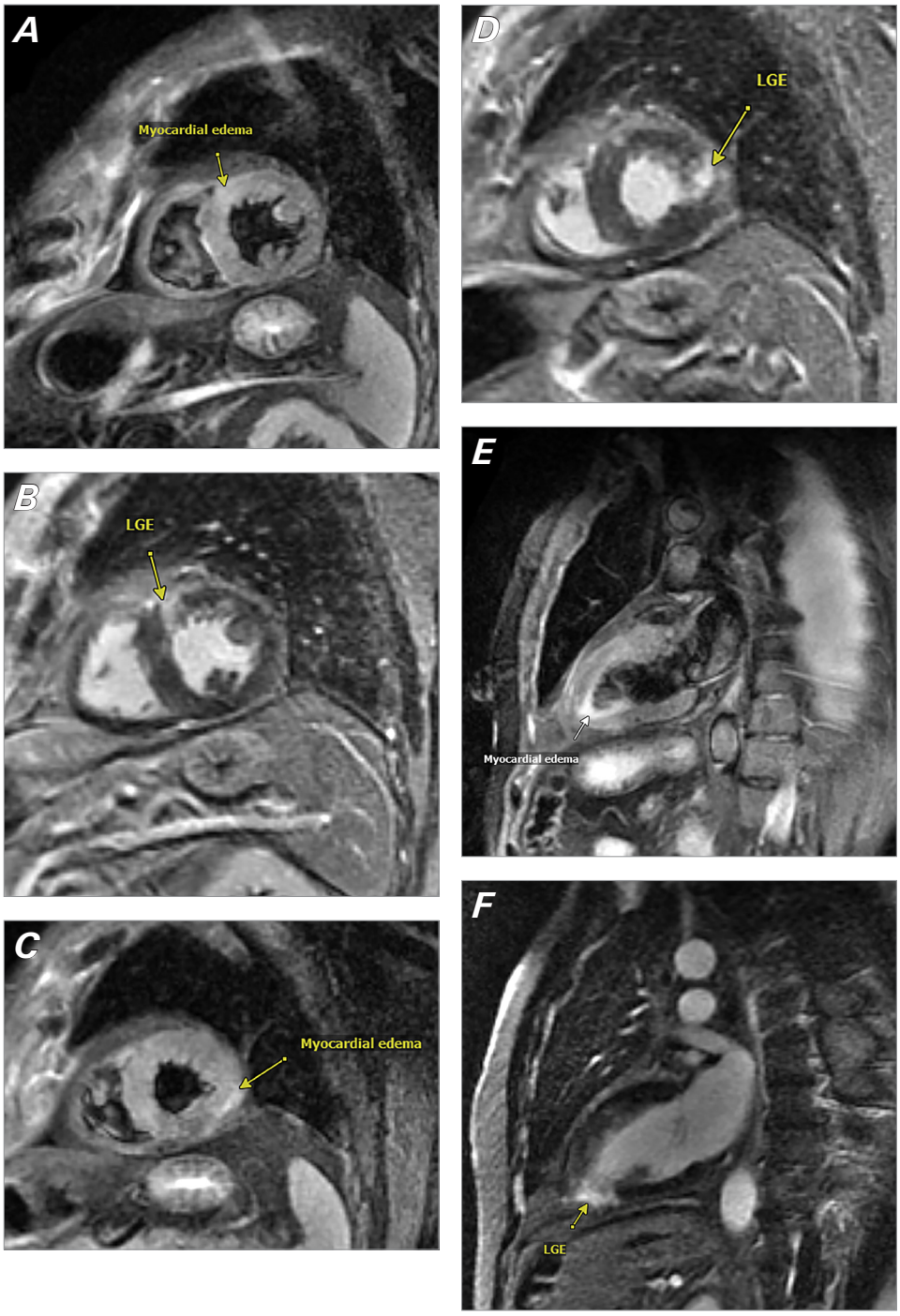

Fig. 6 Cardiac magnetic resonance images show myocardial edema in a triple inversion sequence (**A**, **C**, **E**) and its corresponding transmural LGE (**B**, **D**, **F**) in the (**A**, **B**) midanterior segment, (**C**, **D**) apical lateral segment, and (**E**, **F**) apical inferior segment.
LGE, late gadolinium enhancement.

why health care professionals must maintain high clinical alertness and work together to diagnose cardiac arrest in Lyme-endemic patients who do not have typical cardiovascular risk factors.

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