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ADVANCED

CASE REPORT: CLINICAL CASE

A Boolean Dilemma

True or False Aneurysm?

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ABSTRACT

A feared complication of acute myocardial infarction is the formation of a cardiac pseudoaneurysm. We report a case of a gargantuan, arrhythmogenic left-ventricular pseudoaneurysm with contradictory morphological characteristics. The integrative use of high-resolution 3-dimensional magnetic resonance imaging and computed tomography proved essential for the diagnostic discrimination and successful therapeutic intervention. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2021;3:112–6) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

A 59-year-old man, a heavy-smoking truck driver, was admitted to the emergency department due to a syncopal event while sitting in his truck. He reported a short period of dizziness before losing consciousness. Minutes later, he recovered spontaneously, but was unable to stand up. This event was not precipitated by fear, pain, prolonged standing, or other situational triggers. On arrival of the ambulance, the patient was alert and responsive. His heart rate was 163 beats/min and his blood pressure 69/41 mm Hg, indicative of shock. There were no signs of congestion and no cardiac murmurs were heard. The electrocardiogram (ECG) revealed a wide QRS complex tachycardia with left bundle branch block-like morphology and atrioventricular dissociation (lead II) (**Figure 1**), fitting with a ventricular tachycardia (VT) with a midseptal left-ventricular (LV) exit. The hemodynamic compromise prompted immediate electrical cardioversion. Upon restoration of sinus rhythm, the patient's hemodynamic status normalized. Pathological Q-waves, convex ST-segment elevations in leads II, III, and aVF, and T-wave negativity in leads V_5 to V_6 were noted (**Figure 2**). Cardiac necrosis markers were elevated on admission (cardiac troponin-T 364 [<14] ng/l *no rise-and-fall*, creatine kinase max 187 [<171] U/l, N-terminal pro-brain natriuretic peptide 59 [<35] pmol/l).

LEARNING OBJECTIVES

- To accurately differentiate between false and true left-ventricular aneurysms using dark-blood LGE MRI techniques.
- To recommend the use of multimodality imaging, including 3D LGE MRI, for optimal pre-procedural planning of left-ventricular pseudoaneurysmectomy and targeted anti-arrhythmic therapy using substrate-based ablation.

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PAST MEDICAL HISTORY

The patient had an unremarkable medical history and did not use medications. He never experienced palpitations, light-headedness, syncope, angina pectoris, or neurological focal signs.

DIFFERENTIAL DIAGNOSIS

A sustained monomorphic VT with cardiogenic shock and troponin rise could be the initial presentation of various structural and electrical cardiac abnormalities, including an acute coronary syndrome, nonischemic cardiomyopathy, primary electrical heart disease, and drug/toxins. The VT-QRS morphology in V_1, its fragmented appearance in the extremity leads, and the horizontal axis, point toward scar-related etiologies. The post-cardioversion ECG with persistent ST-segment elevations and pathological inferior Q-waves could allude to a silent inferior wall myocardial infarction. Secondary left-ventricular aneurysm formation should be considered, but is less likely at this point.

INVESTIGATIONS

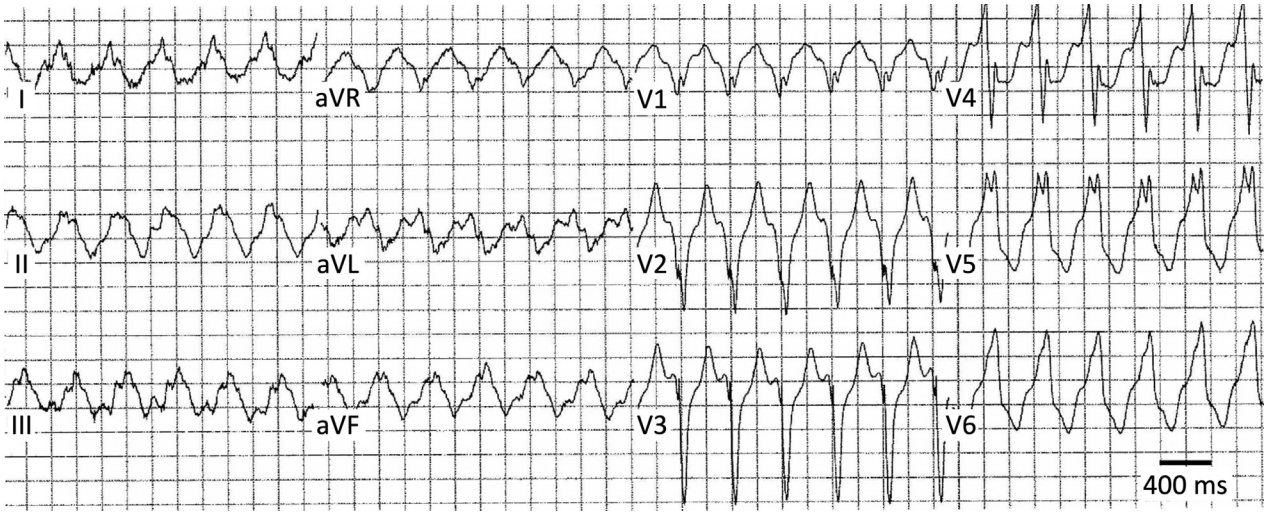
After restoration of sinus rhythm and normalization of the hemodynamic status, a transthoracic echocardiography was performed that revealed an LV ejection fraction of 25% with akinesia of the inferior and inferolateral walls. In addition, a large inferior wall aneurysm was noted, which could differential diagnostically be a false aneurysm (or pseudoaneurysm i.e., LV wall rupture contained by pericardium) or a true aneurysm (i.e., expansively outwardly remodeled necrotic myocardium) (Figure 3A). The neck of the aneurysmal sac measured 40 mm. Neither the mitral valve nor the papillary muscles appeared involved. Next, an ECG-gated contrast-enhanced computed tomography (CT) scan (90 kV, 238 mAs) was performed to noninvasively evaluate the geometry and concomitant coronary artery disease. The aneurysm measured 10.6 × 7.8 × 10.2 cm and extended from the LV base to the insertion of the posteromedial papillary muscle. The cavity was partially filled with thrombus (Figure 3B). A proximally occluded dominant right coronary artery was observed, and the other coronaries had minor wall irregularities. There were no signs of a new critical lesion.

As echocardiography and CT have low diagnostic accuracy to discriminate between a true and false aneurysm (1), 3-dimensional (3D) respiratory-navigated ECG-triggered late gadolinium enhancement (LGE) MRI with dark-blood contrast (2) and 1.6 mm isotropic resolution was performed (3). LGE of 50% transmurality was found at the aneurysmal neck, whereas the sack was characterized by wall thinning

ABBREVIATIONS AND ACRONYMS

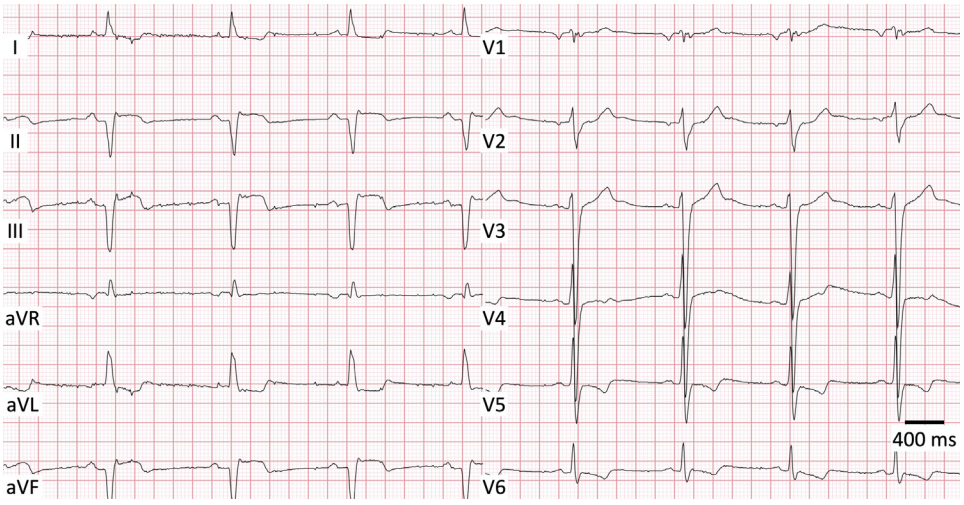
CT = computed tomography
ECG = electrocardiogram
IABP = intra-aortic balloon pump
ICD = implantable cardioverter-defibrillator
LGE = late gadolinium enhancement
LV = left ventricular
MRI = magnetic resonance imaging
V-A ECLS = veno-arterial extracorporeal life support
VT = ventricular tachycardia
3D = 3-dimensional

FIGURE 1 Electrocardiogram on Arrival



Sustained monomorphic ventricular tachycardia with left bundle branch block-like morphology (QRS width 240 ms), atrioventricular dissociation (lead II), and horizontal frontal axis suggesting a midseptal exit.

FIGURE 2 Electrocardiogram After Electrical Cardioversion



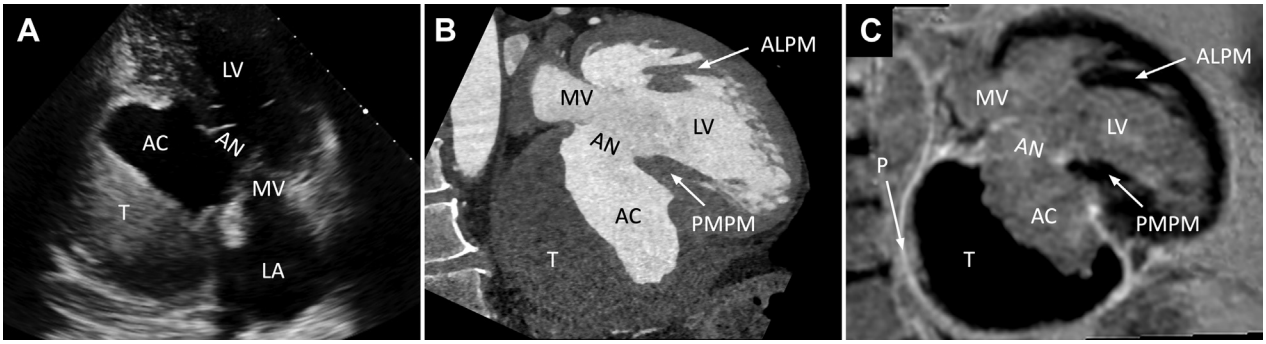
Pathological inferior Q-waves with convex ST-segment elevations and T-wave negativity in leads V_5 to V_6.

and transmural LGE until the point where the circumferential enhancement of the pericardium could not be distinguished from the wall of the aneurysmal sac (**Figure 3C**, Video 1). These MRI features are indicative of a chronic pseudoaneurysm with secondary LV remodeling. The base of the posteromedial papillary muscle was minimally involved. The MRI cine and dynamic contrast-enhanced images (Video 2) recapitulated LV functional and wall-motion abnormalities noted on echocardiography.

MANAGEMENT

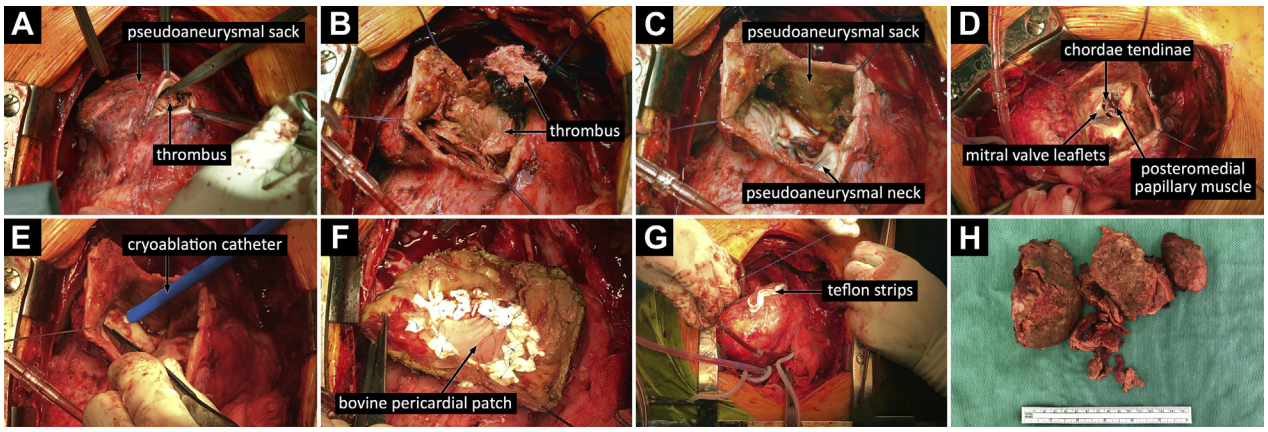
During the hospitalization, metoprolol, perindopril, and bumetanide were initiated for antiarrhythmic and heart failure treatment. To prevent rupture, restore LV geometry, and modify the arrhythmogenic substrate, an LV pseudoaneurysmectomy with intraoperative cryoablation and chamber reconstruction was performed using a bovine pericardial patch to cover the defect (**Figure 4**, Video 3). Macroscopically, the absence of discernable myocardial build-up in the

FIGURE 3 Multimodality Imaging of the LV Pseudoaneurysm



Long-axis view images of the gargantuan inferior wall pseudoaneurysm acquired using **(A)** transthoracic echocardiography, **(B)** contrast-enhanced computed tomography, and **(C)** 3-dimensional late gadolinium enhancement magnetic resonance imaging. Shown anatomic landmarks: AC = aneurysmal cavity; ALPM = anterolateral papillary muscle; AN = aneurysmal neck; LA = left atrium; LV = left ventricle; MV = mitral valve; P = pericardium; PMPM = posteromedial papillary muscle; T = thrombus.

FIGURE 4 LV Pseudoaneurysmectomy With Intraoperative Cryoablation and Chamber Reconstruction



(A) Incision of the pseudoaneurysm. **(B)** Removal of intracavitary thrombus. **(C)** Inspection of the pseudoaneurysmal cavity after thrombus removal. **(D)** Visualization of the posteromedial papillary muscle, chordae tendinae, and mitral valve leaflets. **(E)** Cryoablation of the pseudoaneurysmal neck. **(F)** Pseudoaneurysmal defect closure with bovine pericardial patch. **(G)** Residual pseudoaneurysm wall closure with Teflon strips. **(H)** Removed intracavitary thrombus.

sac, in contrast to the neck, was confirmed. Extensive pericardial adhesions were present. Because of the pre-operative severe LV dysfunction without intra-operative improvement after surgical correction, temporary mechanical circulatory support with peripheral veno-arterial extracorporeal life support (V-A ECLS) and intra-aortic balloon pump (IABP) were installed prophylactically.

DISCUSSION

From a therapeutic point of view, it is important to differentiate between true and false aneurysms (or pseudoaneurysms) (1). In the former, due to chronic outpouching of the LV wall, the aneurysmal wall consists of discernable myocardial layers. This dyskinetic and fibrotic area is generally resistant to rupture, with a 5-year survival of 71% when conservatively treated (4). In the latter, a contained rupture of the myocardium underlies pseudoaneurysm formation, with only a thin pericardial layer covering the leak, which is vulnerable to sudden expansion and rupture with high mortality (1,5).

Reliable data on the incidence of pseudoaneurysms are lacking, although the U.S. National Registry of Myocardial Infarction reported fewer than 1% of subacute myocardial infarctions to be complicated by a myocardial rupture (6). A chronic pseudoaneurysm, as in our case, is a rare phenomenon. Echocardiographic features like a neck-to-aneurysm diameter ratio of <0.4 or an inferior localization favor its diagnosis (5). The broad-based neck, rendering an intermediate ratio, and the inferior localization provide conflicting deterministic information: a "Boolean dilemma."

Historically, angiography was the preferred diagnostic tool to discriminate true from false aneurysms. Nowadays, advanced imaging modalities, such as MRI and CT, are increasingly used, providing high-resolution 3D anatomy and tissue characterization. Moreover, high-isotropic resolution 3D LGE MRI allows for multiplanar reconstruction of any desired cardiac view, in contrast to standard 2D LGE MRI with its 8- to 10-mm-thick slices. In addition, the dark-blood contrast mechanism (2) accurately distinguishes fibrotic tissue from healthy myocardium and the blood pool, important for assessment of the burden of fibrosis (7), papillary muscle involvement, pre-procedural surgical planning, and guiding substrate-based VT ablation.

Regarding the treatment strategy of pseudoaneurysms, a nonsurgical approach is associated with 30% to 45% risk of acute (1) and feared sudden late rupture leading to high mortality rates. As conservative treatment is paralleled by a 33% risk of ischemic stroke at 4 years (8), anticoagulant therapy needs to be considered. Reliable data on potential deleterious effects of anticoagulation on acute or chronic re-rupture risk, however, are lacking. Therefore, surgical resection is generally recommended, despite 10% to 23% perioperative mortality (higher if additional mitral-valve involvement) (9).

In case of true aneurysms, the 5-year survival rate is substantially favorable (71%) (4). Management generally consists of pharmacological afterload reduction (angiotensin-converting enzyme inhibitor), vascular prophylaxis, anti-anginal therapy, and, in selected cases, anticoagulation. Surgical aneurysmectomy may be considered if progressive LV dilatation, deterioration of systolic LV performance, intractable ventricular tachyarrhythmias, or when a coronary bypass graft is planned (10).

Prophylactic hemodynamic supportive measures with V-A ECLS and IABP may reduce perioperative mortality by allowing controlled LV recovery, particularly if severe ventricular failure is present preoperatively. This reduces the need for (potentially arrhythmogenic) inotropic support and provides stable hemodynamics during the first post-operative days.

FOLLOW-UP

Besides bleeding of an adhesion resection for which a rethoracotomy was performed, the patient recovered uneventfully and was successfully weaned from V-A ECLS and IABP (after 4 and 8 days, respectively). An implantable cardioverter-defibrillator (ICD) was implanted. Twelve days post-operatively, ST-segment elevations and Q-waves in the inferior leads persisted. After 3 weeks, the LV ejection fraction remained unaltered at echocardiographic evaluation and no complications were noted. No hospital admissions or ICD interventions were observed at 6-month follow-up.

CONCLUSIONS

This case of a large arrhythmogenic chronically remodeled pseudoaneurysm underscored the importance of the integrative implementation of advanced multimodality imaging tools to guide individual treatment in patients with complex electro-structural heart diseases. 3D dark-blood LGE MRI with high-isotropic resolution accurately discriminated fibrotic tissue from healthy myocardium, blood pool, and thrombus, which was crucial to solve our Boolean dilemma. With the use of advanced surgical techniques, including hemodynamic support measures, a favorable outcome was achieved.

AUTHOR DISCLOSURES

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KEY WORDS computed tomography, echocardiography, false aneurysm, magnetic resonance imaging, myocardial infarction, pseudoaneurysm

APPENDIX For supplemental videos, please see the online version of this paper.