The Heart Surgery Forum 2021-3679
24 (2), 2021 [Epub April 2021]
doi: 10.1532/hsf.3679

Online address: http://journal.hsforum.com

Rapidly Progressing Fatal Left Ventricular Pseudoaneurysm After Acute Myocardial Infarction - A Case Report of Delayed Diagnosis

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ABSTRACT

Left ventricular pseudoaneurysm (LVPA) is a rare complication of acute myocardial infarction (MI). As pseudoaneurysm is contained by the pericardium alone without involvement of myocardial tissue, LVPA are more prone to rupture and hence necessitates surgical intervention. We report a case of a 60-year-old man with acute MI due to a three-way occlusion in the coronary arteries. An emergency transthoracic echocardiogram (TTE) on the 11th day after the MI showed a small ventricular aneurysm, which was probably a late complication of the acute MI episode. A repeat TTE on the 26th day of the MI episode revealed a rapidly progressing LVPA. Emergency heart surgery was planned, but the patient died due to LVPA rupture. This case illustrates timely diagnosis and corrective surgery are key to saving patients from fatal LVPAs.

INTRODUCTION

Left ventricular pseudoaneurysms (LVPA) are rare and late complications of acute myocardial infarction (MI) with a prevalence of 0.05% [Patra 2013; Mujanovic 2014]. In many instances, rupture of the left ventricular (LV) free wall results in fatal complication of MI in approximately 2-4% of patients [Mujanovic 2014; Bisoyi 2016]. LVPA often are formed if the left ventricular wall is ruptured and contained by the pericardium along with thrombus and hematoma [Bisoyi 2016], hence contains only pericardial tissue with no myocardial tissue. In case of true aneurysm, myocardial tissue is involved with left ventricular wall out-pouching. LVPA is characterized by a narrow neck-like projection that communicates with the LV with a tendency to expand and rupture, leading to cardiac tamponade and death [Kim 2010]. Thus, LVPA remains vulnerable to rupture leading to sudden death. Prompt early diagnosis of LVPA through multimodal imaging approach is essential for preventing fatal outcomes. The differential diagnosis of LVPA includes chest wall pain, acute MI, panic disorder, pericarditis, pneumonia, pulmonary embolism, aortic dissection, LV aneurysm, and LV diverticulum. Since the symptoms and imaging features may be identical for true aneurysm, it presents a clinical challenge. In this case report, we present a patient with rapidly progressing LVPA, following an episode of acute MI.

Received February 10, 2021; received in revised form March 8, 2021; accepted March 12, 2021.

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CASE REPORT

A 60-year-old man arrived to our emergency department with severe chest pain for 10 hours; he was diagnosed with acute MI on admission. A chest radiograph revealed a normal heart (Figure 1A) and an emergency bedside two-dimensional transthoracic echocardiography (TTE) demonstrated akinesis of the left ventricular posterolateral wall, without any other clinically significant abnormalities (Figure 1B). On the second day of admission, a percutaneous coronary angioplasty (PCA) was performed. It revealed an occlusion in the left circumflex artery and right coronary artery with chronic stenosis in the left anterior descending artery. Hence, a coronary artery bypass grafting (CABG) was scheduled. On the 11th day after the acute episode of MI, a sudden onset of pulmonary edema was observed and a chest radiograph showed an enlarged thoracic cavity with mild left pleural effusion (Figure 1C). A small cavity (approximately 1.5 X 2.6 cm) in the posterolateral wall of the LV was observed after two-dimensional TTE and ventricular aneurysm was suspected (Figure 1D), which required an elective surgery. On the 26th day, follow-up chest radiograph revealed cardiomegaly with a cardiothoracic ratio of 3:4, which was accompanied by pulmonary congestion and a localized tortuous aorta (Figure 1E) with no clinical observation of chest pain, respiratory distress or febrile status. A repeat TTE confirmed a relatively larger cavity (6.4 X 5.4 cm) in the posterolateral pericardial space, which communicated with the left ventricle through a narrow cavity of 2.4 cm in size to the posterolateral left ventricular free wall, which subsequently was confirmed to be a rapidly progressed, large LVPA (Figure 1F). (Figure 1)

On the same day, a color doppler echocardiography was done to confirm the communicating passage between the left ventricle and pericardial cavity (Figure 2A). On the 27th day, electrocardiographic-gated multi-slice computed tomography (MSCT) was performed, revealing a giant pseudoaneurysm measuring 67 X 46 X 60 mm that originated from the LV posterolateral wall (Figure 2B, 2C, 2D). (Figure 2) As the pseudoaneurysm observed had progressed to a larger size than what was observed in the TTE on the previous day, emergency heart

surgery was planned for LV aneurysmoplasty. On the 28th day, an emergency surgery was performed. During surgery, the patient lost his consciousness, presented with clammy limbs, urinary and fecal incontinence, heart rate of 192 bpm, and BP of 82/60 mmHg. During surgery, his heart rate declined quickly to 48 bpm and arteriopalmus disappeared. The electrocardiogram a minute later showed a straight line, and the patient was declared has dead, due to suspected rupture of the free wall of the ventricle caused due to LVPA.

DISCUSSION

In this case report, we present a rare case of a fatal, rapidly progressing LVPA. Unlike left ventricular true aneurysms, which consist of a resistant myocardial fibrotic wall, pseudoaneurysm is confined within the less fibrous pericardium and have a strong tendency to grow rapidly with high propensity to rupture [Mujanovic 2014]. LVPA usually remains undiagnosed, as >10% of the cases presented are asymptomatic

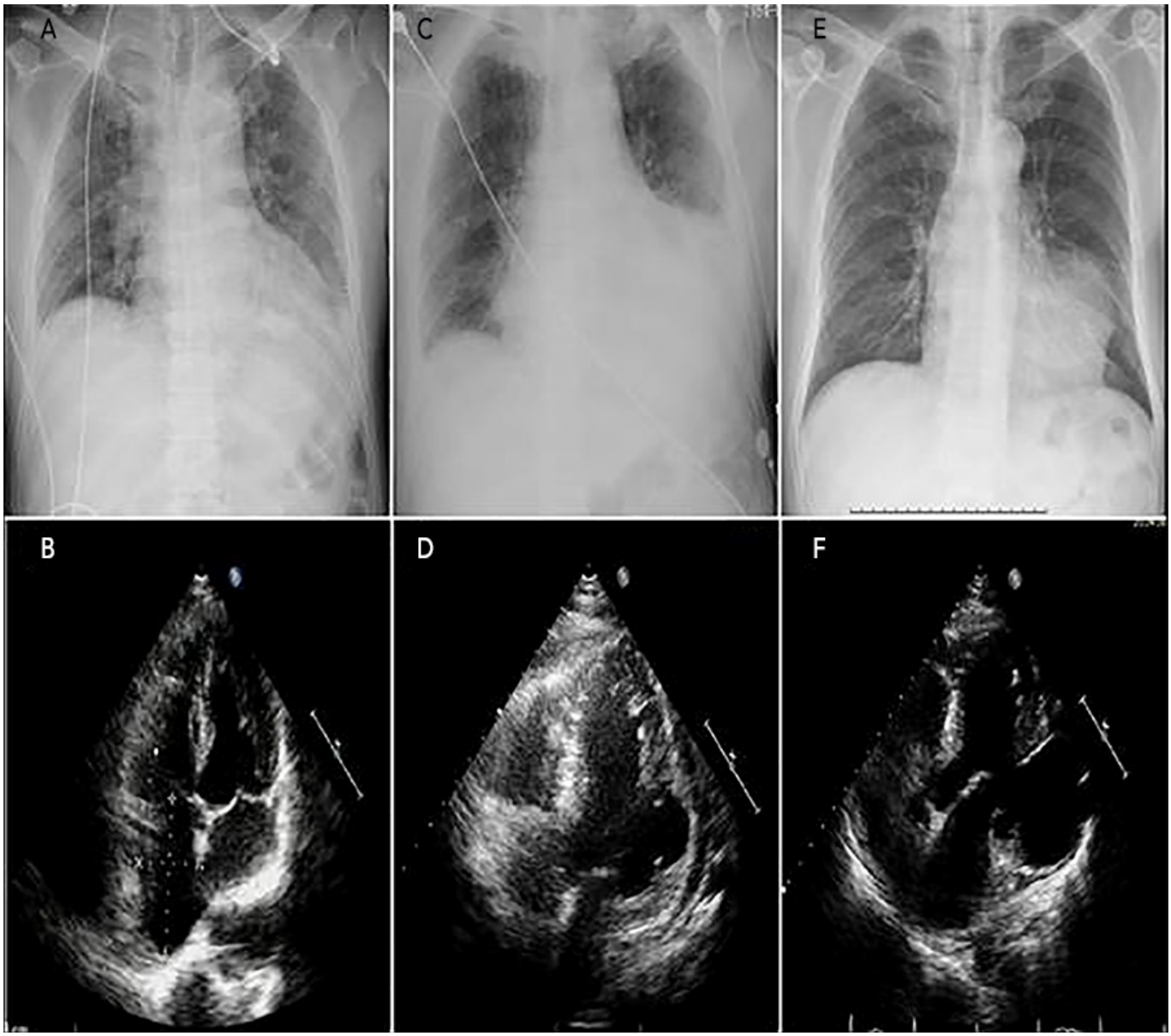

Figure 1. A) Chest X-ray revealing normal heart at admission. B) Two-dimensional transthoracic echocardiography (TTE) demonstrating akinesis in the posterolateral wall with no other clinically significant abnormalities. C) Chest X-ray revealing an enlarged heart and a mild left pleural effusion after 11 days of the acute episode of MI. D) Two-dimensional TTE showing a left ventricular aneurysm of the posterolateral wall (approximately 1.5 X 2.6 cm). E) Chest X-ray showing cardiomegaly, pulmonary congestion, and a localized tortuous aorta after 26 days of the acute episode of MI. F) Repeated TTE revealed a giant (approximately 6.4 X 5.4 cm) LV pseudoaneurysm in the posterolateral pericardial space that communicated with the left ventricle through a 2.4 cm cavity in the posterolateral left ventricular free wall.

pseudoaneurysms [Bekkers 2006]. The risk of developing LVPA is higher in the first 3 months after an acute MI episode [Mujanovic 2014]. Likewise, in our case report, asymptomatic pseudoaneurysms had occurred after few days of acute MI, and the patient died within 28 days of the acute MI episode due to rupture of an extremely unstable LV wall. Differential diagnosis of LVPA could be challenging as a patient may present with symptoms related to coronary artery disease like chest pain, dyspnea, and heart failure along with non-specific symptoms like cough, dizziness, etc., which further decreases the suspicion for LVPA [Dogan 2013].

In some instances, patient survival has been prolonged due to unruptured LVPA for years after MI [Komeda 1993]. Hurst et al. reported prolonged patient survival of 6 years

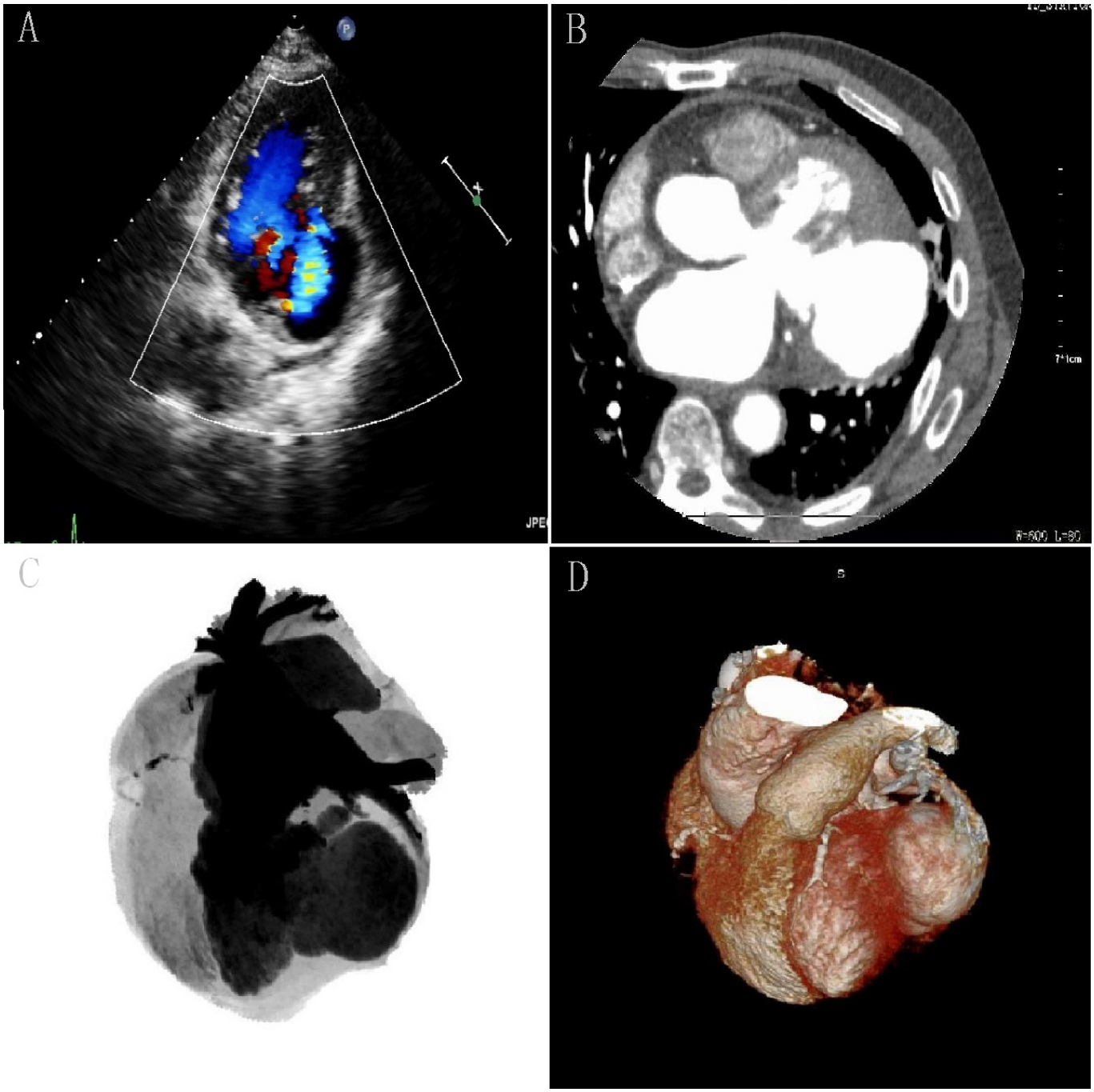

Figure 2. A) Doppler showing flow passage from the left ventricular into the pseudoaneurysm. B, C, D) Multislice computed tomography revealing a progressive pseudoaneurysm measuring 67 × 46 × 60 mm originating from the LV posterolateral wall.

after acute MI [Hurst 1963], whereas the case reported by Dogan et al. showed patient survival for 11 months before the diagnosis of LVPA [Dogan 2013]. However, in our case, LVPA was unique with rapid progression and rupture of LV wall within 28 days after acute MI, which severely hampered the clinical decision-making.

Pseudoaneurysms mostly are confined to the postero-inferior wall with the involvement of the epicardium along with the fibrous layer of the pericardium and characterized by a narrow-necked orifice leading to the ventricle via a sac-like structure containing thrombus, which is due to statis of blood flow [Kim 2010; Prifti 2017]. This could be the basis for differential diagnosis with true aneurysm as they are located at the anterior wall, and the apical segment of the ventricle involves myocardial tissue [Dogan 2013; Prifti 2017]. Thrombus formed in the LVPA could result in large pseudoaneurysm [Frances 1998] (>3cm diameter), and it may lead to systemic embolism [Ludmir 2016]. Thrombus due to statis of blood flow inside the cavity in the LVPA was also observed in our case report.

Also, in this present case report, a communicating passage was observed without a narrow neck-like orifice, which has led to misdiagnosis and delay in the treatment decision for LVPA, which later attributed for the fatality. Hence, we recommend early diagnosis with precise techniques like magnetic resonance imaging (MRI) and multi-slice computed tomography and early surgical intervention for suspected cases to overcome a lethal complication.

CONCLUSION

The differential diagnosis of left ventricular pseudoaneurysm can be difficult in initial stages as patients often present either asymptomatic or with non-specific symptoms attributed to other causes. A timely multimodal imaging diagnostic approach to rule out LVPA might help in reducing mortality.

ACKNOWLEDGEMENT

Ethical statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. Written informed consent was obtained from the patient for publication of this study and any accompanying images.

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