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MYOCARDIAL INFARCTION PRESENTING AS BOTH LEFT VENTRICULAR ANEURYSM AND VENTRICULAR SEPTAL DEFECT

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ABSTRACT

Acute myocardial infarction can result in various mechanical complications, although they have become rare with the advent of reperfusion therapies. Among these complications, ventricular septal rupture (VSR) and left ventricular aneurysm (LVA) are infrequent but life-threatening conditions associated with high morbidity and mortality. We present a rare case of a 67-year-old male with acute myocardial infarction who developed concomitant apical LVA and ventricular septal rupture.

KEYWORDS

Ventricular septal defect, left ventricular aneurysm, acute coronary syndrome

LEARNING POINTS

- Mechanical complications of myocardial infarction, such as a ventricular septal rupture (VSR) and left ventricular aneurysm (LVA), are rare but life-threatening.
- Early diagnosis is critical. A ventricular septal defect (VSD) requires immediate surgical closure, while surgery for LVA is only considered in specific cases such as chest pain or thromboembolism.
- Diagnostic tools such as echocardiography and left ventriculography play a vital role in identifying and characterising these complications, enabling timely treatment decisions.

INTRODUCTION

Acute myocardial infarction can result in lethal mechanical complications[1]. Ventricular septal defects (VSDs) are among the typical complications; however they have become rare in the reperfusion era with an incidence of 1%–3%, yet they continue to have high morbidity and mortality[1]. With a reported incidence of 3.5%–38%, left ventricular aneurysm (LVA) is relatively more common and has been associated with myocardial free-wall rupture, congestive heart failure, thrombus formation in the left ventricle (LV) and recurrent

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ventricular tachyarrhythmias[2].

Acute VSD and LVA are rare, life-threatening complications that usually occur within the first week of acute myocardial infarction, which necessitates immediate recognition and surgical correction[2]. Although the benefits of surgical reconstruction for LVA have not been clearly demonstrated, their effectiveness has recently been reported in a small number of patients[3].

Here, we report a rare case of concomitant apical LVA and ventricular septal rupture in a patient presenting with acute myocardial infarction.

CASE DESCRIPTION

A 67-year-old male with a past medical history of hypertension and diabetes was admitted for cough and haemoptysis. He also reported shortness of breath on exertion, bilateral lower extremity swelling and limited ambulation in the previous two months. The patient denied chest pain, palpitations, dizziness, orthopnoea or paroxysmal nocturnal dyspnoea; he reported a cardiac disease in the past but did not seek medical attention.

On arrival, the patient was afebrile and tachycardic with a heart rate of 105 beats per minute, blood pressure of 116/78 mmHg and saturating 96% on room air. Initial laboratory tests showed a white blood count of 18 (4.8–10.8 K/Ul), platelet 93 (130–400 K/Ul), BNP 1050 (<100 pg/ml), troponin 0.38 (<0.5 ng/ml), potassium 5.6 (3.6–5.1 Mmol/l) and an international normalised ratio of 2.2 (0.85–1.14). An initial electrocardiogram showed ST elevation in V1–V5 with Q waves. The patient received intravenous regular insulin, albuterol inhalation and furosemide intravenous 40 mg.

On examination, the patient was found to have heart failure. There were inspiratory crackles on lung auscultation and pitting oedema on the lower extremities. A holosystolic murmur at the tricuspid area and a faint S3 were present on cardiac auscultation. The echocardiogram showed a left ventricular ejection fraction of 35%–40%, a large infarct involving mild to distal antero-septal, mid-distal anterior, antero-apical and infero-apical segments. Apical aneurysmal motion and a small distal antero-septal VSD were present *(Fig. 1 and 2)*.

The gradient across VSD was 68 mmHg, suggesting acute right ventricle pressure increase without chronic pulmonary hypertension. Right ventricular size and function were normal; the left atrium was mildly dilated. The inferior vena cava was dilated with a respiratory variation of less than 50%. A CT angiogram showed segmental and subsegmental pulmonary emboli in the right lower lobe. The QuantiFERON test was negative.

The patient was started on furosemide 40 IV bid, atorvastatin 40 mg daily and IV heparin for pulmonary embolism. Left heart catheterisation showed proximal left anterior descending artery occlusion at the trifurcation of the first septal and diagonal vessel with no collateral distally. The ostial left circumflex artery showed 50% disease, the small first obtuse marginal artery with 70% disease, and the post

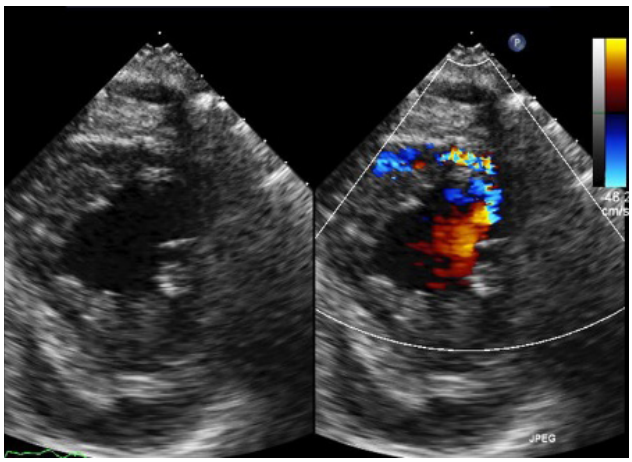

Figure 1. Short axis view of the left ventricle depicting a small distal antero-septal VSD.

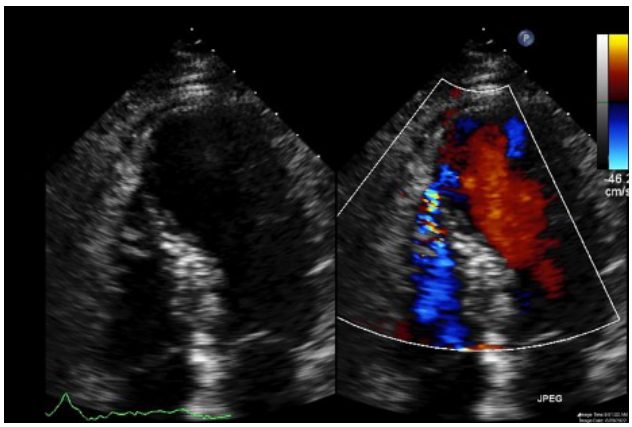

Figure 2. Four-chamber view depicting a left ventricular aneurysm with a small distal antero-septal VSD.

atrioventricular groove of the left circumflex artery having 60% disease. The right coronary artery had a 60%–70% lesion in the proximal portion, the coronary angiogram also showed a large anterior-anterolateral aneurysm and a large mid-distal VSD. The right heart catheterisation showed a shunt of at least 5 l/min.

The patient was referred to a tertiary care centre and underwent VSD surgical repair with a satisfactory outcome. He was discharged in the following days to home with follow-up as an outpatient. He visited the outpatient cardiology clinic within one week of discharge and he was stable without any complaints.

DISCUSSION

Mechanical complications are rare due to the advent of emergent reperfusion strategies for myocardial infarction. Moreover, having two mechanical complications, ventricular septal rupture and left ventricular aneurysm, in the same patient is exceptionally unusual. Prompt diagnosis and definitive surgery are critical as mortality remains high.

Ventricular septal rupture is a rare, but life-threatening complication of myocardial infarction (MI). Before the introduction of reperfusion therapies, it was estimated to occur in 1%–2% of MI patients. However, with the recent advances in reperfusion treatments over the decade, the

latest data approximates an incidence of 0.17%–0.31%[4]. Risk factors for developing VSRs after MI include single vessel disease, advanced age (>65), female gender, extensive myocardial damage, poor septal collateral circulation and delayed presentation of MI[5]. A VSR develops between the necrotic and non-necrotic myocardium regions, resulting in left-to-right shunting. VSR's pathological features are classified into three types; of the three types, Becker's type III is associated with older infarcts and involves concomitant aneurysm formation[6]. Becker's type III was seen in our patient. The diagnosis of VSD is primarily made with a transthoracic echocardiogram and on occasions, a transoesophageal echocardiogram may be utilised to determine the full extent of the abnormality[7]. If a pulmonary artery catheterisation is performed, an acute step-up in venous oxygen saturation will be noted[4]. Contrast ventriculography can be used during cardiac catheterisation as an alternative at times.

LVA is another fatal mechanical complication of MI. Similar to VSR, the incidence of LVAs has dramatically reduced with the introduction of reperfusion therapies, from a previously estimated 30%–35% to 8%–15% post-MI. Defined as a weakened area of the left ventricular wall devoid of muscle, LVA results in either akinetic or dyskinetic movement during systole[8]. The formation of LVA post-MI involves two phases: the early expansion phase, which occurs as early as 48 hours to 2 weeks post-infarction, and the late remodelling phase, which occurs within 2 to 4 weeks post-infarction[9]. The diagnosis of LVA should be suspected in patients with large anterior wall MI. While most LVAs are asymptomatic, symptoms such as shortness of breath, syncope and chest pain are concerning for large LVAs with preceding complications. Heart failure, ventricular arrhythmias and systemic embolisation secondary to stasis in blood flow in the aneurysm cavity are all possible complications[10]. Diagnostic testing often involves an initial EKG, resulting in a tall R wave in the lead AvR known as Goldberger's sign. Two-dimensional echocardiography not only helps diagnose LVA and differentiate between a true and false type of aneurysm, but it also allows the ability to calculate systolic function and identify a thrombus. However, the gold standard for diagnosing and accurately localising the site of an aneurysm is left ventriculography[11].

It is reported that without surgical repair of post-infarction VSD, 90% of patients die within two months[12]. As a result, current guidelines set by the American College of Cardiology recommend immediate surgical closure of VSDs, irrespective of the patient's haemodynamic status. Delayed surgery may be considered when there is concern regarding the ability to perform definitive repair and complex surgical anatomy. Patients who are not good surgical candidates, such as the very elderly or those with poor right ventricular function, may be considered for transcatheter septal closure (TSC). TSC is a percutaneous repair and involves advancing a sheath with a wire into the pulmonary artery to form an arteriovenous rail. While TSC has been shown to have significant overall success and low morality, disadvantages exist. The defect's size and location must be considered when selecting patients for TSC; defect sizes greater than 15 mm or inferior/posterior defects are suboptimal when considering TSC. In our patient, the defect was antero-septal and the patient was a good candidate for surgical repair of the VSD. Hence, our patient was transferred for immediate surgery.

Unlike the management of VSD, surgery is not the mainstay of LVA management. Medical management is the first concern for asymptomatic patients regardless of the size of the aneurysm. This includes optimising risk factors for ischaemia, using afterload reduction such as angiotensin-converting enzyme inhibitors and anticoagulation. Anticoagulation via warfarin is recommended for the first three months after LV infarction as the risk for mural thrombus is highest. Long-term anticoagulation should be considered for patients with large thrombi, systemic embolisation after three months or globally impaired LV function[13]. In patients with LVA, surgical repair is often not favoured due to the risk of mortality, which is around 10%. Indications for surgery include persistent chest pain, thromboembolism, refractory heart failure or life-threatening tachyarrhythmias[14]. Surgical repair of post-infarction LVA involves the reconstruction and size reduction of the dilated ventricle, thereby reducing cardiac oxygen consumption, increasing myocardial efficiency and improving contractile efficiency[14]. The endoventricular circular patch plasty technique is generally the surgical treatment of choice. This technique improves LV ejection fraction, decreases LV end-diastolic and improves overall clinic presentation, characterised by the New York Heart Association functional class status.

In the era of emergent reperfusion treatment, post-infarction complications have become rare. While rare, these complications are life-threatening and early diagnosis using techniques such as echo and left ventriculography is vital. Our patient was found to have both a VSD and an LVA, also known as a Beckers type III. In patients like ours where both a VSD and an LVA are present, immediate surgical repair of the VSD is essential given the high mortality risk without repair. Postsurgical repair of a VSD, and medical management for LVA is the treatment of choice to reduce the risk of further cardiac remodelling or thrombi formation.

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