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Left Ventricular Aneurysm Associated to Double Ventricular Septal Defect in Acute Myocardial Infarction: A Serious and Fearsome Entity

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ABSTRACT

Ventricular septal defects following myocardial infarction are a relatively infrequent complication occurring transmural myocardial infarction. The simultaneous presence of ventricular septal defects and left ventricular aneurysm in the same patient is extremely rare and usually occurs within the first week after myocardial infarction. Surgery remains the only viable treatment option. A 63-year-old man with multiple risk factors for coronary artery disease diagnosed with acute extensive anterior wall myocardial infarction, presented to our emergency room with heart failure symptoms, cardiac auscultation revealed a band-shaped systolic murmur in the left parasternal area, radiating to the whole precordium.Electrocardiography revealed sinus rhythm with persistent ST-segment elevation in leads V1–V4 with Q waves in the anterior precordial leads, and chest X-ray revealed bilateral pulmonary congestion. A Transthoracic 2D echocardiography was performed at our facility which demonstrated dilated left ventricle with severely reduced systolic function. Important regional wall motion abnormalities were present with an aneurysm of the medium and apical segments of anterior and septal wall complicated by two muscular ventricular septal defects. Colour Doppler revealed a shunt between the left and right ventricle through the aneurysmatic segment. Coronary angiogram showed a significant disease of the left anterior descending and right coronary artery. The patient underwent surgery for the simultaneous repair of the ventricular septal defect, apical aneurysm and a coronary artery bypass. Post-infarction left ventricular aneurysm associated with a double ventricular septal defect is a very unusual mechanic complication, especially five weeks after the inaugural episode with no evidence of recurrent heart attack. We present such a case that was successfully treated surgically. Rapid diagnosis and urgent surgical management significantly improves the patient's prognosis.

Introduction

The mechanical complications of a myocardial infarction (MI) are frightening, and they readily occur following a transmural MI. Ventricular septal defects (VSD) following MI are a relatively infrequent complication occurring in approximately 0.2% to 1% of patients but can cause significant morbidity and mortality. Without surgical treatment, the prognosis for patients is dismal as approximately 90% of patients die within 2 months. It is usually observed in the first 24 h and at 3 to 7 day post MI and is associated with very high morbidity and mortality.

Left ventricular aneurysms (LVA) occur in up to 40% of patients following myocardial infarction. The distribution of LVA varies: 85% are anterolateral and 5–10% are posterior. The paradoxical motion caused by the aneurysm reduces the left ventricular output and may result in intractable heart failure, hence leading to decreased survival. Hence, addressing the aneurysm and the infracted area will decrease the left ventricular volume and increases the cardiac performance.

The simultaneous presence of VSD and LVA in the same patient is extremely rare and usually occurs within the first week after MI. Surgery remains the only viable treatment option.

Case report

A 63-year-old man with multiple risk factors for coronary artery disease presented to a secondary care hospital (Laayoun) one month ago for severe retrosternal chest pain accompanied with shortness of breath.

He was diagnosed with acute extensive anterior wall MI with severely reduced systolic function with an ejection fraction (EF) of 35%, thrombolytic therapy (TT) was not given and the patient was not referred for percutaneous intervention because these services were not available at the local public hospital .The patient was discharged with medical treatment.

However, heart failure symptoms persisted, and he was referred to our centre for further management.

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On admission, pulse rate was 110/min and blood pressure of 90/60 mmHg. His physical examination revealed bilateral basal crepitations and cardiac auscultation revealed a band-shaped systolic murmur in the left parasternal area, radiating to the whole precordium. Further, his oxygen saturation was 96% on air.

Electrocardiography revealed sinus rhythm with persistent ST-segment elevation in leads V1–V4 with Q waves in the anterior precordial leads, and chest X-ray revealed bilateral pulmonary congestion.

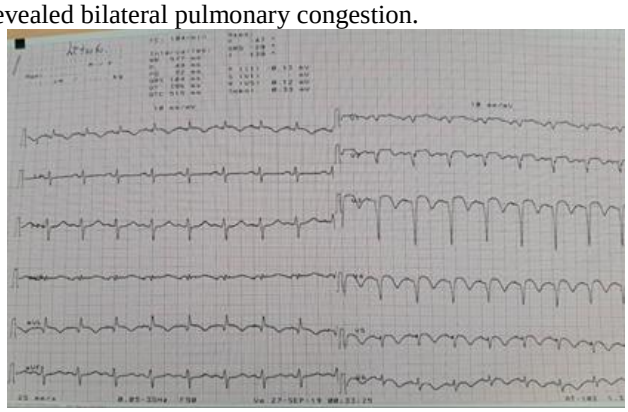

Figure 1. Electrocardiography performed the morning after admission showed sinus rhythm persistent ST elevation in leads V1-V4

A Transthoracic 2D echocardiography was performed at our facility which demonstrated dilated LV with severely reduced systolic function with an ejection fraction (EF) of 30-35%. Important regional wall motion abnormalities were present with an aneurysm of the medium and apical segments of anterior and septal wall complicated by two muscular VSD (figure A). Colour Doppler revealed a shunt between the left and right ventricle through the aneurysmatic segment (figure B).

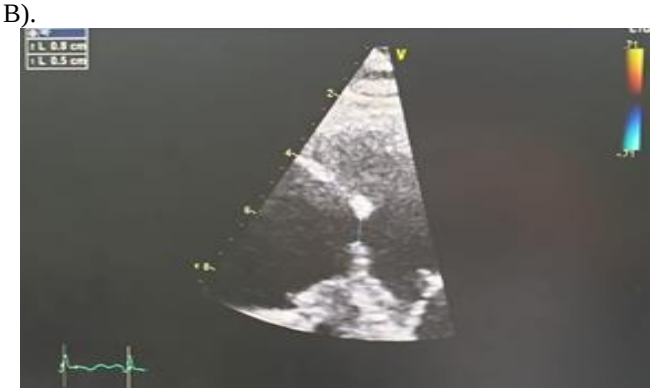

Figure 2A

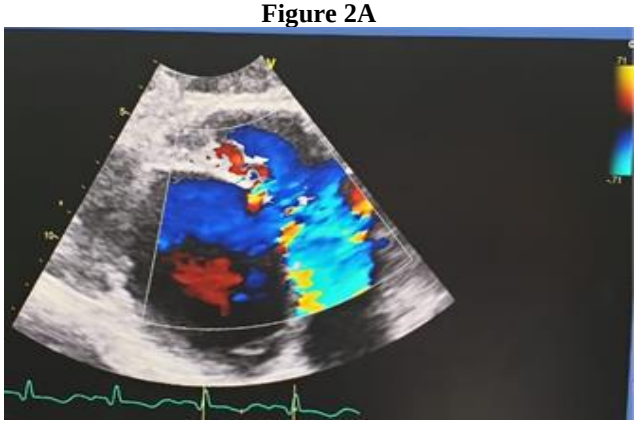

Figure 2B

Figure 2. Transthoracic echocardiography in the parasternal short-axis view showing (A) two muscular ventricular septal defect (respectively 5mm and 8 mm) with (B) a left-to-right shunt.

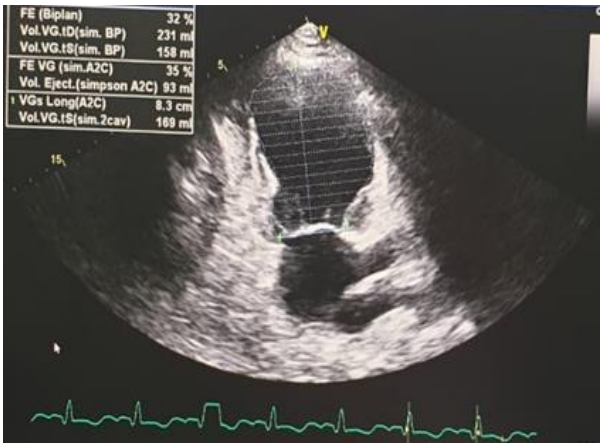

Figure 3. Transthoracic echocardiography two chamber view showing the ventricular apical aneurysm.

Coronary angiogram revealed a bitroncular damage, proximal left anterior descending (LAD) and the second portion of the right coronary artery (RCA).

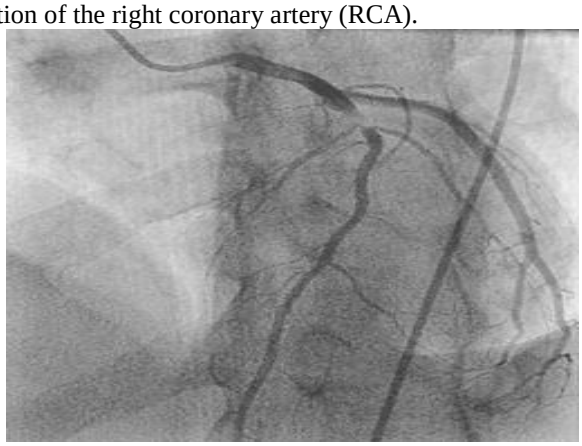

Figure 4A

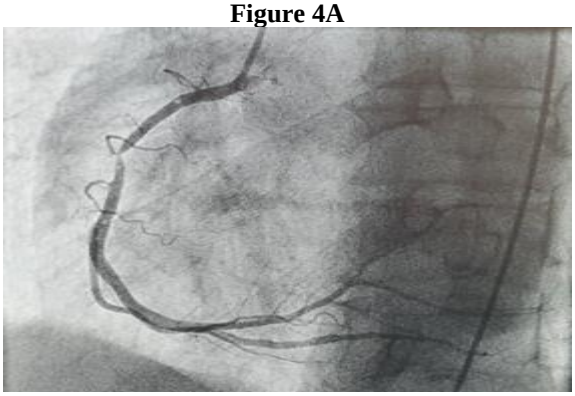

Figure 4B

Figure 4. coronary angiogram : (A) RAO view illustrates the left coronary artery with a significant occlusive lesion in proximal portion (B) LAO view showing occlusion of right coronary artery.

The patient underwent surgery for the simultaneous repair of the ventricular septal defect and a coronary artery bypass.

Under general anesthesia, after median sternotomy, the left internal mammary artery (LIMA) and Saphenous vein graft were prepared in pedicle technic and the cardiopulmonary bypass was established by cannulation of the ascending aorta and right atrium.

After aortic cross-clamp, a cold blood cardioplegia was applied. The ventricular septum was approached through a transinfarct incision, 2cm parallel to the left anterior descending artery. Ventricular septal rupture was repaired with a 2-patch technique, using a patch of bovine pericardium.

The coronary lesions in the LAD and RCA were treated using LIMA and Saphenous vein grafts, respectively. The flow measurement on the bypasses demonstrated a sufficient heart perfusion.

Discussion

Mechanical complications are uncommon but potentially lethal sequelae of acute MI and are commonly associated with early mortality without appropriate surgical intervention are more common with ST-segment elevation MI (STEMI). [1,8] Compared with the prereperfusion era, studies have suggested a reduction in the incidence of mechanical complications post-MI in the reperfusion era. [1] Studies have also demonstrated increases in mechanical complication rates with thrombolytic therapy compared with primary percutaneous coronary intervention (PCI), as well as a very grim prognosis associated with these complications. [9, 10]

Several electrocardiography abnormalities have been associated with rupture of the ventricular wall, including evidence of transmural myocardial infarction, maintained ST segment elevation, low amplitude of the QRS complex, inversion of T waves and intraventricular conduction disturbances. None of these findings are sensitive or specific for cardiac rupture and the ECG cannot be considered a useful tool for the diagnosis.

Hemodynamically, the presence of VSD gives rise to a left-to-right shunt, followed by right ventricular volume overload, increased pulmonary blood flow, and secondary volume overload of the left atrium and ventricle which in turns results to left ventricular deterioration. Cross-sectional echocardiography with a sensitivity of about 40% and Doppler ultrasound with sensitivity close to 100% are rapid and well established techniques for diagnosing VSD after myocardial infarction. [11]

The angiographic findings in our patient are similar to those of GUSTO-I trial. Specifically, in the GUSTO-I study, total occlusion of the infarct-related artery was documented in 57% of patients with VSD; as compared with 18% of those without VSD. The same study showed that the left anterior descending (LAD) artery was the most associated artery with VSD. [12]

Rupture of the free wall of the ventricle and rupture of the ventricular septum are both rare but often, fatal complications of myocardial infarction. Free wall rupture occurs up to 10 times more frequently than septal rupture, affecting up to 11% of patients after acute myocardial infarction and is almost invariably fatal, with death occurring within minutes after the development of chest pain. Mortality from post-infarct septal rupture approaches 25% in the first 24 h and up to 50% in the first week. The most important factor in determining outcome in postinfarct VSD is the development of congestive cardiac failure and cardiogenic shock, which is dependent on the magnitude of the left-to-right shunt. [13]

LVA are a common complication following transmural myocardial infarction and involve the dilatation of left ventricular wall in an abnormal fashion. LVA are often associated with total occlusion of the LAD and poor collateral supply. 75% of patients have multivessel coronary artery disease. [14] The scarred area becomes increasingly thin and dyskinetic resulting in an aneurysm, which annuls a part of the left ventricular ejection fraction, eventually leading to the cardiac failure refractory to medical therapy and necessitates surgical management.

The first LVA repair with the use of cardiopulmonary bypass was reported in 1958 by Cooley et al. [15] who used the linear closure method. In subsequent years, repair with a patch was described, which was then widely accepted with various modifications. [16,17] Vural and colleagues reported the results of patch repair were superior to those of linear repair in terms of left ventricular geometry and long-term clinical outcomes. [18] In Dor procedure, a patch is implanted inside the left ventricular, thereby excluding the akinetic portion of the left ventricular septum, thus permitting the reconstruction and the restoration of left ventricular geometry. [14] Dor plasty is effective for large anteroseptal or posterobasal aneurysms and can be used in cases of more severe left ventricular damage, where implantation of a patch avoids inadequate left ventricular dimensions after the operation, thereby facilitating better rearrangement of the myocardial fibers. [19]

Based on studies thus far, the outcomes reported on patients undergoing coronary artery bypass grafting (CABG) at the time of VSD repair have been inconsistent. Perrota et al found that patients undergoing concomitant CABG to all stenotic coronary arteries supplying blood to non infarcted areas had better 30 day mortality and long-term survival. They found that improving collateral flow to the myocardium contributed to better recovery. [20] These outcomes contrast with those of Deja et al who found that 30 day mortality and long-term survival did not improve when concomitant CABG was performed at the time of VSD repair. [21] When patients undergo concomitant procedures such as CABG at the time of VSD repair, the benefits associated with revascularization need to be weighed against the risks of prolonged cardiopulmonary bypass. Given this data, decision to revascularize should be individualized to the patient.

Choosing between (transcatheter closure) TCC and surgery will depend on the size of the VSD, the patient's hemodynamic stability, and the timing after VSD development. Maltais et al suggest that utilizing TCC for defects larger than 15mm puts the patient at risk for device embolization or recurrent VSD. [22] Omar et al recently found overall mortality was lower in the TCC group than the surgery group. [23] Horan et al indicate no survival differences between TCC and surgery. However, the authors emphasize that either TCC or surgery are superior to medical therapy alone

Current recommendations favor emergent surgical repair regardless of the patient's hemodynamic stability. However, pathophysiology supports delayed repair as wound healing provides increased strength as the infarct recovers. Following infarction, the breakdown of necrotic myocytes begins to occur with new collagen laid down between days 2 to 4. This process continues over 28 days and as new collagen is synthesized, the infarct moves from friable tissue to organized and strengthened tissue. This restructured myocardium provides better material to hold surgical suture.[2]

Conclusion

Post-infarction left ventricular aneurysm associated with a double ventricular septal defect is a very unusual mechanic complication, especially five weeks after the inaugural episode with no evidence of recurrent heart attack. We present such a case that was successfully treated surgically. Rapid diagnosis and urgent surgical management significantly improves the patient's prognosis.

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