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Case Series

Can someone die of a broken heart: cases of lethal myocardial infarctions with ventricular wall ruptures

Juan José Gómez-Piña[1]*, Vanessa Castro Luna[1], Natalia León Alfaro[1], Abraham Gilberto Medina Reyes[1], Edgar Ulises Domínguez Hernández[1], Luis Fernando Arias Velarde[2]

[1]Department of Internal Medicine, Hospital de Especialidades CMN La Raza "Antonio Fraga Mouret", Instituto Mexicano del Seguro Social, Mexico City, Mexico
[2]Department of Critical Care Coronary Unit, Hospital de Especialidades CMN La Raza "Antonio Fraga Mouret", Instituto Mexicano del Seguro Social, Mexico City, Mexico

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***Correspondence:**
Dr. Juan José Gómez Piña,
E-mail: drjgomezp@gmail.com

ABSTRACT

Ventricular septal rupture is a dreaded complication of transmural myocardial infarction, most often due to occlusion of the left anterior descending artery. The resulting interventricular shunt leads to acute heart failure and cardiogenic shock in the early days following infarction. We present three cases of patients admitted with myocardial infarction who developed ventricular septal defects within 48 hours of admission. All were diagnosed rapidly with echocardiography and underwent urgent surgical repair. However, each experienced persistent hemodynamic instability despite inotropes and intra-aortic balloon pump support. Early postoperative mortality occurred in all three patients, between 2 to 9 days after surgery. Although expedited diagnosis and surgical intervention temporarily stabilized these patients, the post-infarction septal rupture proved lethal in each case. Earlier revascularization or pre-emptive mechanical circulatory support may be needed to improve outcomes from this catastrophic mechanical complication of myocardial infarction.

Keywords: VSR, Case series, Interventricular, Rupture, Death

INTRODUCTION

The acute ST-segment elevation myocardial infarction is caused by blockage of the coronary arteries, leading to full thickness myocardial ischemia and subsequent myocardial cell death.[1] Thirty-day mortality for patients with ST-elevation myocardial infarction ranges from 2.5 to 10 percent.[2]

Ventricular septal rupture (VSR) is a major mechanical complication following myocardial infarction (MI). It results from lack of timely reperfusion after MI, usually due to complete occlusion of the left anterior descending artery or dominant right coronary artery supplying the inferior wall. The rupture creates an interventricular shunt leading to acute cardiogenic shock in the first week post-MI. VSR is a surgical emergency as the septal perforation causes rapid hemodynamic deterioration. Early diagnosis with prompt surgical repair is crucial, though outcomes remain poor despite expedited treatment. Further research into preventative strategies and mechanical circulatory support is needed to improve survival in this devastating complication of MI.[3]

The VSR is most frequently presented in patients treated with thrombolytic therapy (1.1%) compared to percutaneous intervention (0.7%) according to the Global Registry for Acute Coronary Events (GRACE) study.[4] The incidence of VSR is higher in STEMI (0.9%) compared to

non-STEMI (0.17%) and unstable angina (UA) (0.25%).[5] This entity is classified into three types: type I is an acute tear occurring within 24 hours, type II is a subacute erosion of infarcted myocardium and type III is a late rupture associated with aneurysm formation in older myocardial infarcts.[6]

Patients presents as a rapidly deterioration, with hypotension, hemodynamically instability and pulmonary edema, after 1 to 3 days, however, cardiogenic shock is the main cause of death in those patients. At auscultation a systolic murmur is heard over precordium, may also feel a thrill, loud pulmonic component of second heart sound, tricuspid regurgitation or third heart sound.[7]

Echocardiography is used to diagnose ventricular septal rupture, showing right ventricular dilatation and pulmonary hypertension due to increased right-sided blood flow. The use of echocardiography lead to determine the size and site of defect and doppler tool helps to determine the magnitude of the left-to-right shunt in the apical 4-chamber view. Electrocardiogram show elevated ST segments in patients with a ventricular aneurysm or heart block (30%).[8]

The gold standard treatment of ventricular septal rupture is the surgical repair, however, circulation must be restored prior surgical procedure, to decrease the stroke area. Contraindications for surgical procedure include absence of cardiogenic shock, pulmonary edema, congestive heart failure, good cardiac perfusion and minimal use of amines.[9]

There is a favourable prognosis if the rupture is small and the patient is hemodynamically stable, despite optimal treatment, the condition carries a mortality of 20%-50%. 10 Main electrocardiographically findings in VSR post STEMI includes atrioventricular or infranodal conduction abnormalities in approximately 40% of patients.[11]

The aim of treatment is maintained optimal blood pressure, cardiac output, hemodynamic stability. Mortality of medically treated patients with VSR turns around 24% during the first 72 hours, unfortunately increases to 75% at three weeks. Surgical mortality is high among patients with inferior MI (70%) compared to patients with anterior infarcts (30%).[12]

CASE SERIES

Case 1

A 72-years-old male with cardiovascular history of type 2 diabetes and smoking, who complains with chest pain and dyspnea with holodastolic murmur in mitral valve, presented 7 days post-infarction in the lower wall, echocardiogram with right ventricular systolic dysfunction, left ventricular ejection fraction (LVEF) of 72%, 7 mm ventricular septal defect, 18 mm aneurysm and PSAP 70 mmHg.

Case 2

A 57-years-old male with cardiovascular history of diabetes and hypertension, presents chest pain and holodiastolic murmurs in mitral valve, on first day post-infarction in inferolateral wall. Patient developed hemodynamic deterioration requiring hemodynamic assistance with aortic balloon-pump 1:2, echocardiogram with systolic dysfunction of right ventricular, LVEF of 55% and inferoseptal defect of 12 mm.

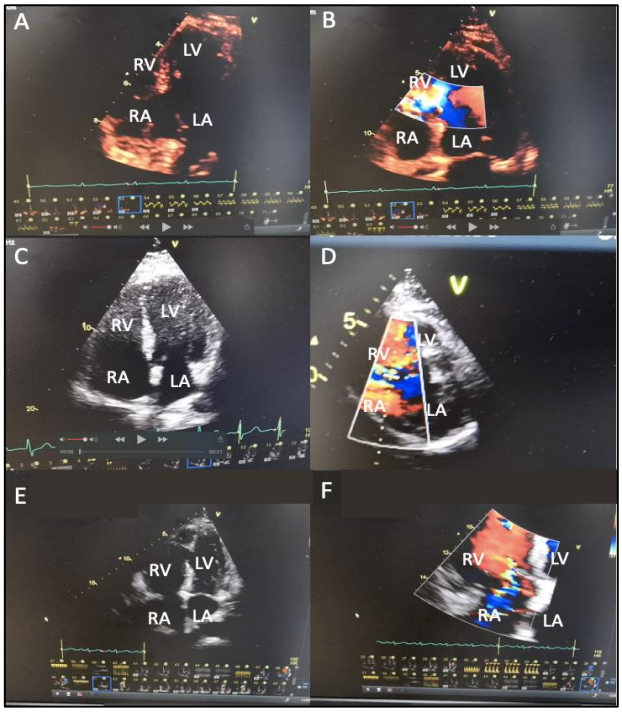

Figure 1: Echocardiogram shows a 66-year-old female with inferonasal akinesia with hypercontractility, LVEF 70%, cardiac output 8.27, (A) and a posterior interventricular septal rupture of 26 mm, maximum gradient 27 GLPS -10.4%. TAPSE 18, PCWP 28 and mild mitral regurgitation (B). A 72-year-old male with right ventricular systolic dysfunction, LVEF of 72% (C) and 7 mm ventricular septal defect, 18 mm aneurysm and PSAP 70 mmHg (D). A 57-year-old male systolic dysfunction of right ventricular, LVEF of 55% (E) and inferoseptal defect of 12 mm (F).

Case 3

A 66-years-old female with cardiovascular history of type 2 diabetes, who complains with chest pain, presenting holosystolic murmur in mitral valve due to non-reperfused inferolateral wall, 4 days later; she developed interventricular septal rupture, treated with defect closure and valve exploration mitral, tricuspid, with aortic balloon-pump 1:2.

Echocardiogram with inferobasal akinesia with hypercontractility, LVEF 70%, Cardiac Output 8.27, posterior interventricular septal rupture of 26 mm

maximum gradient 27 GLPS-10.4% is found. TAPSE 18, PCWP 28 and mild mitral regurgitation (Figure 1).

Clinical follow-up and outcomes

Case 1

The patient developed progressive hemodynamic instability secondary to acute right ventricular failure and mechanical complications of myocardial infarction (septal rupture and ventricular aneurysm).

Despite aggressive inotropic and ventilator support, he deteriorated into refractory cardiogenic shock, complicated by hospital-acquired pneumonia and subsequent septic shock. The patient succumbed to multiorgan failure on day 5 of hospitalization, with the primary cause of death attributed to septal rupture-induced acute pulmonary edema and nosocomial sepsis.

Case 2

The patient experienced acute septal rupture with cardiac tamponade, prompting emergency surgical repair. Postoperatively, he developed mesenteric ischemia secondary to septic embolism, leading to bowel perforation and septic shock. Despite surgical reintervention and maximal critical care support, the patient deteriorated into irreversible cardiogenic and septic shock, expiring on hospital day 10.

Case 3

Despite successful surgical intervention, the patient developed persistent right ventricular failure, followed by a massive pulmonary embolism. Anticoagulation therapy precipitated an intracranial haemorrhage, exacerbating her hemodynamic collapse. She suffered a cardiopulmonary arrest on postoperative day 7, with the final cause of death being multiorgan failure in the setting of irreversible cardiogenic shock.

Table 1: Comparison of patients who developed ventricular septal rupture as a complication of acute myocardial infarction.

Parameter	Case 1	Case 2	Case 3
Demographics	72-year-old male	57-year-old male	66-year-old female
Cardiovascular history	Type 2 Diabetes, smoking	Type 2 Diabetes, hypertension	Type 2 Diabetes
Presenting symptoms	Chest pain, dyspnea	Chest pain	Chest pain
Murmur	Holodiastolic (mitral valve)	Holodiastolic (mitral valve)	Holosystolic (mitral valve)
Infarction details	Lower wall MI, 7 days post-infarction	Inferolateral wall MI, 1-day post-infarction	Non-reperfused inferolateral wall MI, 4 days post-infarction
Echocardiogram findings	Right ventricular systolic dysfunction	Right ventricular systolic dysfunction	Inferonasal akinesia with hypercontractility
	LVEF 72%	LVEF 55%	LVEF 70%
	7 mm VSD	12 mm inferoseptal defect	26 mm posterior VSD (max gradient 27 mmHg)
	18 mm aneurysm		GLPS -10.4%
			TAPSE 18
	PSAP 70 mmHg		PCWP 28
			Mild mitral regurgitation
Hemodynamic support	Procedure not performed	Intra-aortic balloon pump (1:2)	Intra-aortic balloon pump (1:2)
Surgical intervention	Procedure not performed	Not detailed	Defect closure + mitral/tricuspid valve exploration
Main complications	Ventricular septal defect (VSD), aneurysm	Hemodynamic deterioration, VSD	Large VSD, mitral regurgitation

VSD: Ventricular septal defect, LVEF: Left ventricular ejection fraction, PSAP: Pulmonary artery systolic pressure, TAPSE: Tricuspid annular plane systolic excursion, PCWP: Pulmonary capillary wedge pressure, GLPS: Global longitudinal peak strain.

DISCUSSION

The ventricular septal rupture represents one of the most catastrophic mechanical complications following acute myocardial infarction, carrying exceptionally high mortality rates despite advances in cardiovascular care. This life-threatening condition typically manifests with sudden hemodynamic collapse, characterized by the abrupt development of a new harsh holosystolic murmur, electrocardiographic abnormalities and rapid progression to cardiogenic shock. While acute MI remains the predominant etiology, accounting for approximately 90%

of cases, clinicians should remain vigilant for alternative causes including penetrating chest trauma, infective endocarditis with annular abscess formation, iatrogenic injury during myocardial biopsy or complications following aortic valve replacement.[13]

The pathophysiology of post-infarction VSR involves extensive transmural necrosis leading to mechanical disruption of the ventricular septum. This creates a left-to-right shunt that precipitates acute volume overload, pulmonary hypertension and systemic hypoperfusion. The severity of clinical presentation depends on several factors, the size of the defect (ranging from punctate perforations to large communications exceeding 20 mm), the location of rupture (anterior vs inferior walls) and the patient's baseline cardiac reserve. Our case series demonstrates this spectrum, with defect sizes varying from 7 mm to 26 mm, each resulting in profound hemodynamic consequences. Current management paradigms emphasize the critical importance of timely intervention.

Both European and American guidelines strongly advocate for urgent surgical repair in cases complicated by cardiogenic shock, as highlighted by the ESC Task Force recommendations. While emergent surgery is mandatory for unstable patients, those maintaining marginal stability may benefit from 24-48 hours of preoperative stabilization using mechanical circulatory support (IABP, Impella or ECMO) to optimize end-organ perfusion. Contemporary techniques include infarct exclusion with patch repair, though debate persists regarding optimal materials (Dacron vs pericardial patches) and the role of concomitant revascularization. Percutaneous closure devices have emerged as potential bridges to surgery or definitive therapy in select high-risk patients, particularly those with posterior defects challenging to access surgically.

The dismal prognosis associated with VSR (30-days mortality exceeding 50% even with surgery) underscores the need for preventive strategies. Primary percutaneous coronary intervention within 90 minutes of STEMI presentation significantly reduces VSR incidence compared to thrombolysis or delayed revascularization. Furthermore, aggressive management of modifiable risk factors - particularly hypertension and diabetes - may mitigate the risk of myocardial rupture.[14]

CONCLUSION

Ventricular septal rupture remains as a catastrophic complication of acute myocardial infarction, characterized by high mortality despite advances in diagnostic and surgical interventions. This case series underscores the lethal nature of post-infarction VSR, as evidenced by the rapid hemodynamic deterioration and fatal outcomes in all three patients, despite expedited surgical repair and mechanical circulatory support.

The findings highlight the critical importance of early recognition, prompt echocardiographic diagnosis and emergent surgical intervention, though even these measures may not suffice to avert mortality in severe cases. The persistent challenges in managing VSR, particularly in patients with cardiogenic shock. Pre-emptive revascularization, earlier mechanical support and innovative surgical techniques may offer potential avenues to improve survival. Furthermore, the disparity between the reported incidence of VSR in global registries and its observed prevalence in this case series suggests regional or demographic variations that warrant further investigation.

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