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Ventricular septal rupture

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Abstract

Background: Ventricular septal defect (VSD) is a lethal complication of myocardial infarction.

Case presentation: A 67-year-old male who presented with a history of chest pain associated with shock... Physical examination was significant for crepitation in lower lung fields. Echo showed a left ventricular ejection fraction (LVEF) of 30-35% and muscular ventricular septal defects with left to right shunting and elevated pulmonary artery systolic pressure. In a short time, he developed respiratory failure during the hospital course. Our patient had VSD due to acute myocardial infarction (MI). The patient was transferred to another hospital on a ventilator for surgical intervention but died before any procedure.

Conclusion: Our case highlights an unfortunate patient with a VSD secondary to myocardial infarction (MI) resulting in Death.

Keywords: ventricular septal defect (VSD), a complication of myocardial infarction. Ventricular septal rupture, interventricular septum. Invasive strategy. Cardiogenic shock; Intra-aortic balloon pump; left to right shunt; IABP=intra-aortic balloon pump.

Introduction:

A ventricular septal defect (VSD) is an abnormal communication between the left and right ventricle through a weakness in the septal wall of the heart. VSD is a rare but lethal complication of myocardial infarction (MI). It is also referred to as a ventricular septal rupture (VSR). A VSR after MI is uncommon and occurs only 1-2% of the time [1]. The event occurs 2-8 days after an

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infarction, often leading to cardiogenic shock. Conservative treatment is associated with 94% mortality, while surgical treatment is associated with 47% mortality during the first 30 days [2]. Deciding to perform surgery is complicated by the critical preoperative condition of the patients and the myocardial tissue necrosis. Patients with right ventricular infarction, cardiogenic shock, and ventricular septal rupture have high in-hospital mortality rates [3]. Here, we present a VSD post-myocardial infarction case leading to multi-system organ failure.

Case Presentation

Personal data: 67-year-old male patient, Married. Smoker, Admitted to AMU hospital 7/12/2022

Complaint: chest pain 20 hours before admission, Shortness of breath 3 hours before admission

Present history: The condition started 20 hours before admission with typical retrosternal chest pain at rest, radiating to the left shoulder and back maximum from the start and increasing intensity. Then, a few hours later, the patient started to experience rapidly progressive dyspnea grade III– IV associated with orthopnea.

Negative data: No fever, no cough, no syncope, no palpitation, no lower limb edema

Medical history: Hypertension, DM on oral hypoglycaemic drugs (OHD), No History of ACS

Drug history: Bisoprolol 5 mg OD, OHD: Glimiperide 2 mg once per day (OD), Metformin 500 mg OD

Surgical history: Irrelevant

Family history: Irrelevant

Examination on presentation: General examination: The patient is alert, conscious, oriented, agitated, BMI: 23 Kg/m2, Orthopneic, No pallor, jaundice, or cyanosis

Vital signs: Blood pressure: 90/60 mmHg, equal bilateral, Pulse: 120 bpm, regular, similar on both arms, the vessel wall is not felt, peripheral pulsations are weakly felt, Respiratory rate: 30 cycle/min, O2 Saturation 94%, Temp: 37.2 ℃

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Head & neck: Trachea is central, Carotid pulsations are felt equal bilateral with no palpable thrill, Bilateral **congested normally pulsating neck veins** up to 8 cm above sternal angle, no palpable LN, Thyroid is not palpable

Cardiac examination: No precordial bulge; Apex is felt in the fifth intercostal space in MCL (midclavicular line). No visible suprasternal or epigastric pulsations

Auscultation: Aortic area: S1 heard, A2 normal, no murmurs. Mitral area:
S1 normal, S2 heard, no murmurs. Left lower parasternal area: S1 normal, S2 heard with harsh pansystolic murmur grade 4/6. Pulmonary area: S1 heard, P2 normal, no murmurs

Chest auscultation: Bilateral equal air entry, No crepitation, No wheezes

Abdominal examination: No clinically detectable organomegaly or ascites

Lower limb: Peripheral pulsations are weakly felt; no lower limb edema

Table 1: Laboratory results:

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Date	**7/12 on admission**	**9/12 After two days in CCU,**
Hgb	17	15.6
WBC	27.9	38.1
Platelets	116	152
Creatinine	2.3	4.5
Na	135	140
K	4.4	4.6
INR	1.3	1.4
CK-MB	251	62
Trop	>25	>25
CRP	106	186
ALT AST	86 220	88 268

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Course: Patient was shocked (ABG acidotic, BP 80/50, Anuric). Vasopressor and inotropes were added. (ABG improved and BP 90/60, UOP (urine output) improved). (Multiorgan hypoperfusion was still present). A cardiothoracic consultation was done. The decision was urgent surgery in a higher center with the availability of IABP. The patient was transferred for urgent surgery.

After transfer to another hospital, the patient was put on a ventilator but died before any intervention.

Electrocardiogram: Figures 1-3

Fig 1. Electrocardiogram of presentation, inferior MI.

Fig 2. Electrocardiogram after developing pulmonary edema

Fig 3. Electrocardiogram soon after deterioration of case.

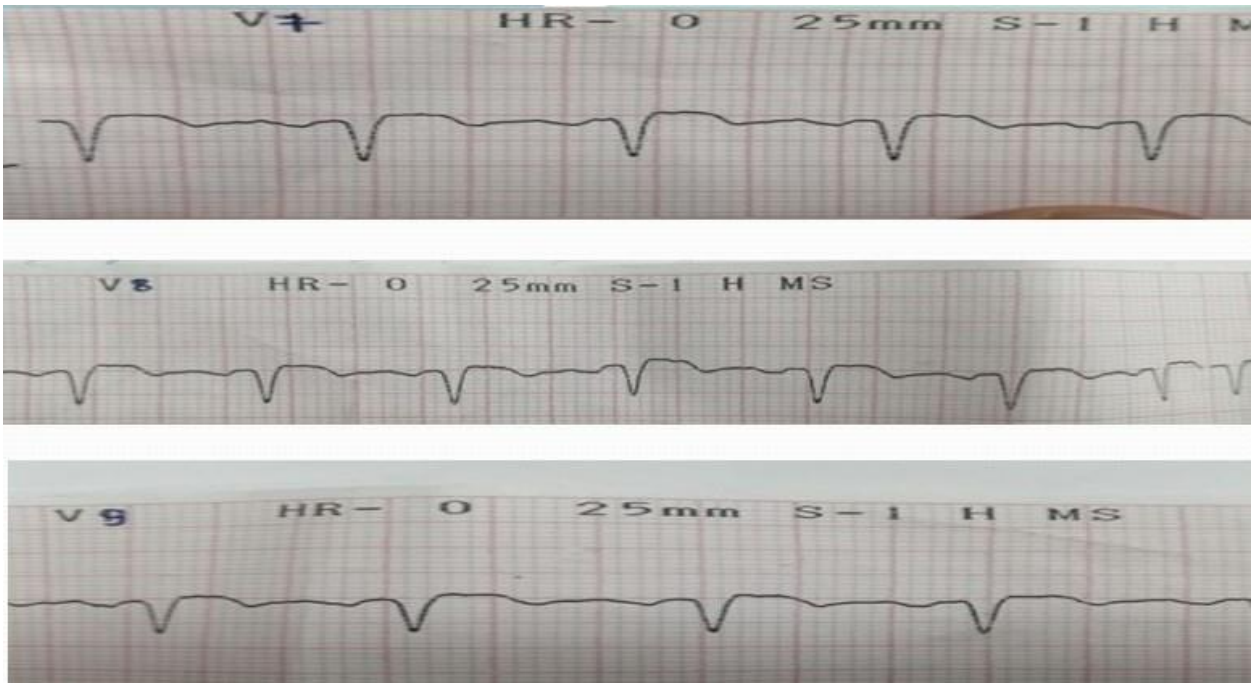

Fig 1-ECG limb leads

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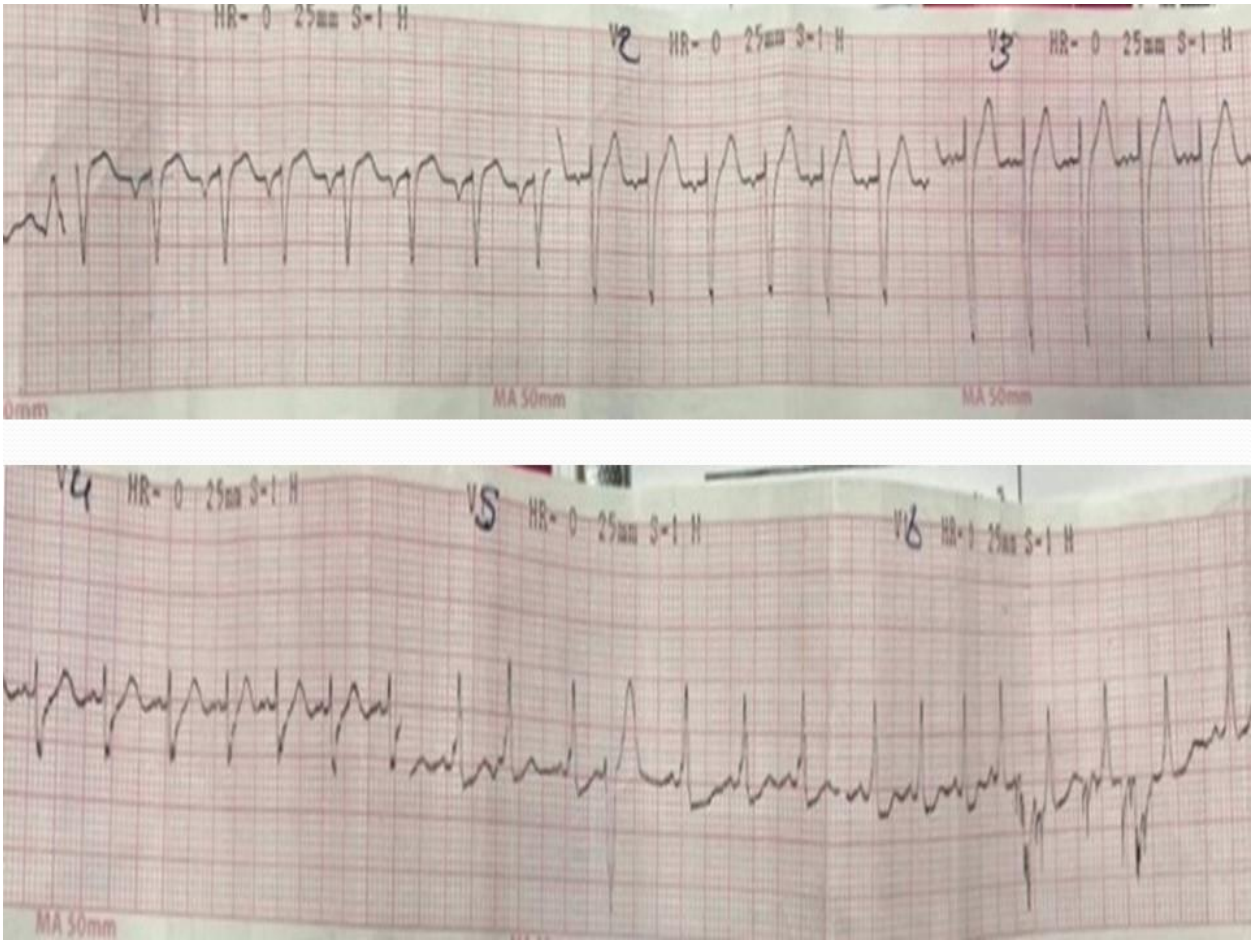

Fig 2 ECG

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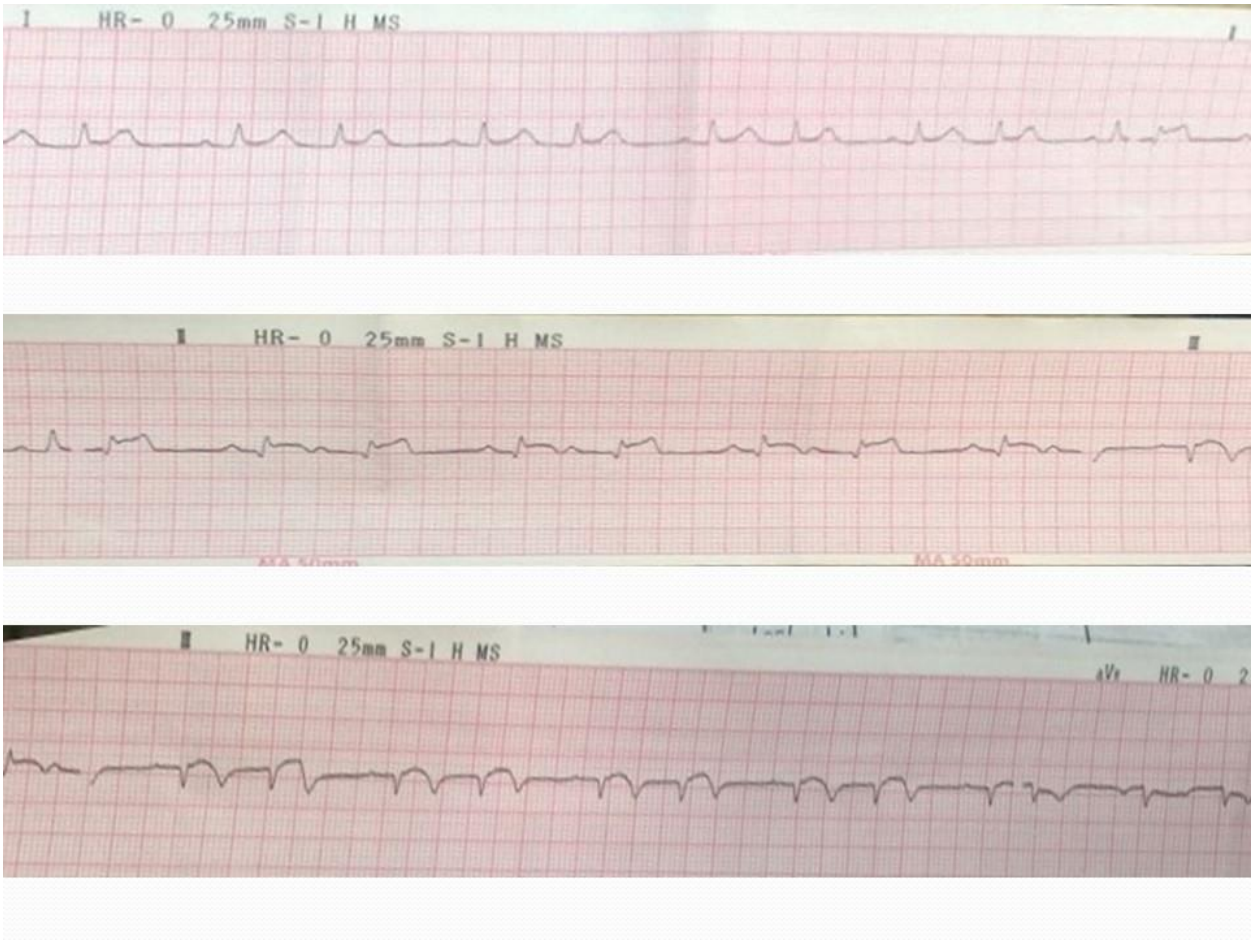

Fig 3 ECG

Echocardiography: Figures 4-7

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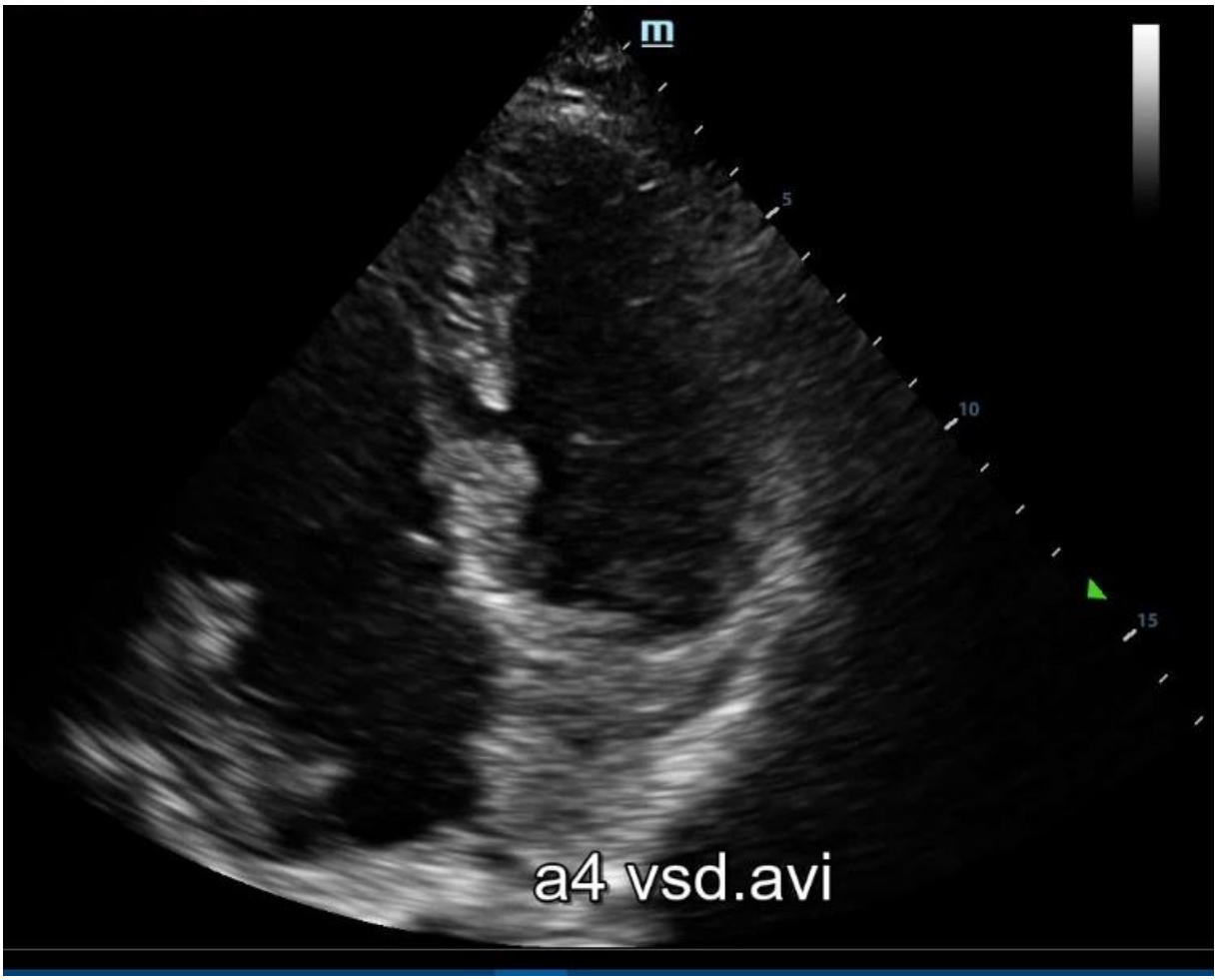

Fig 4-

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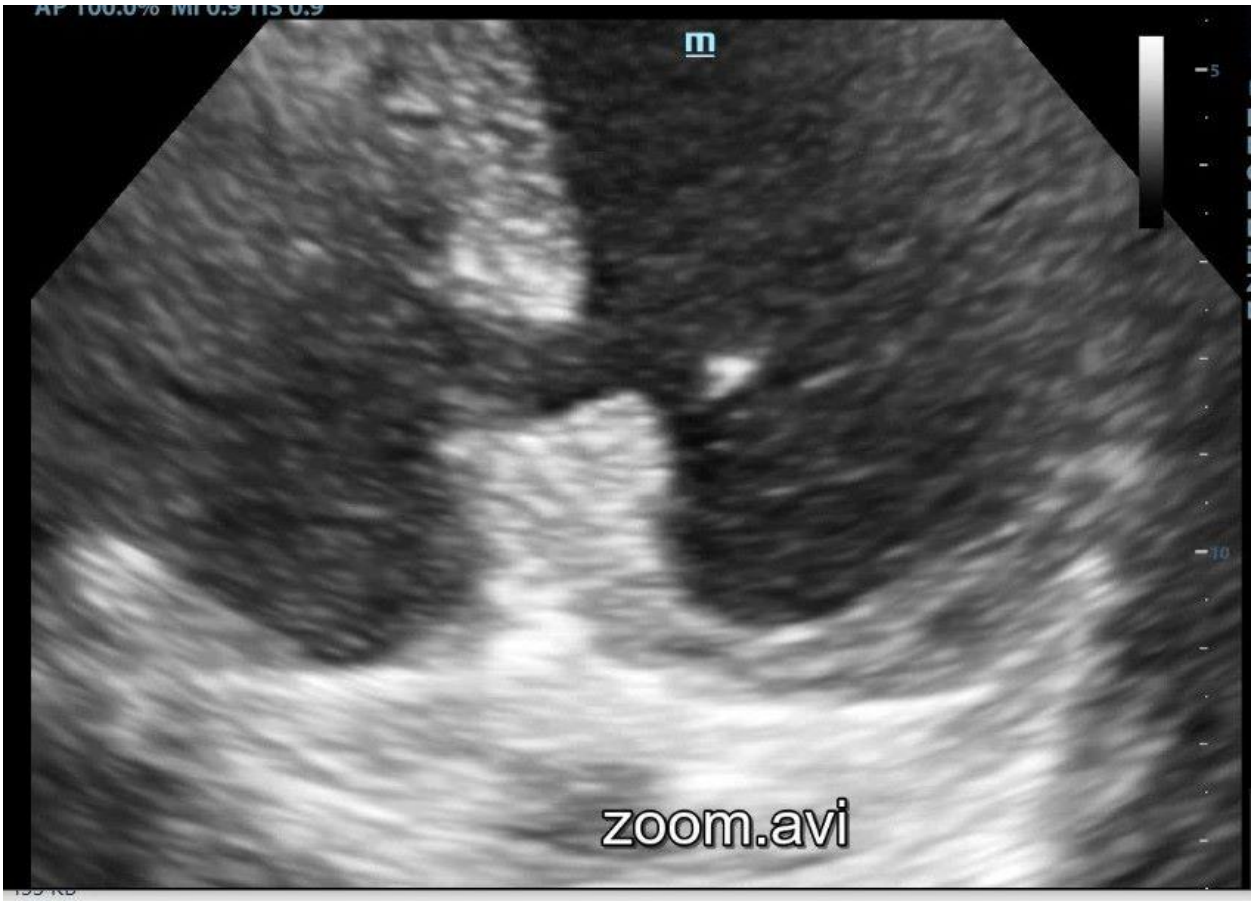

Fig 5-

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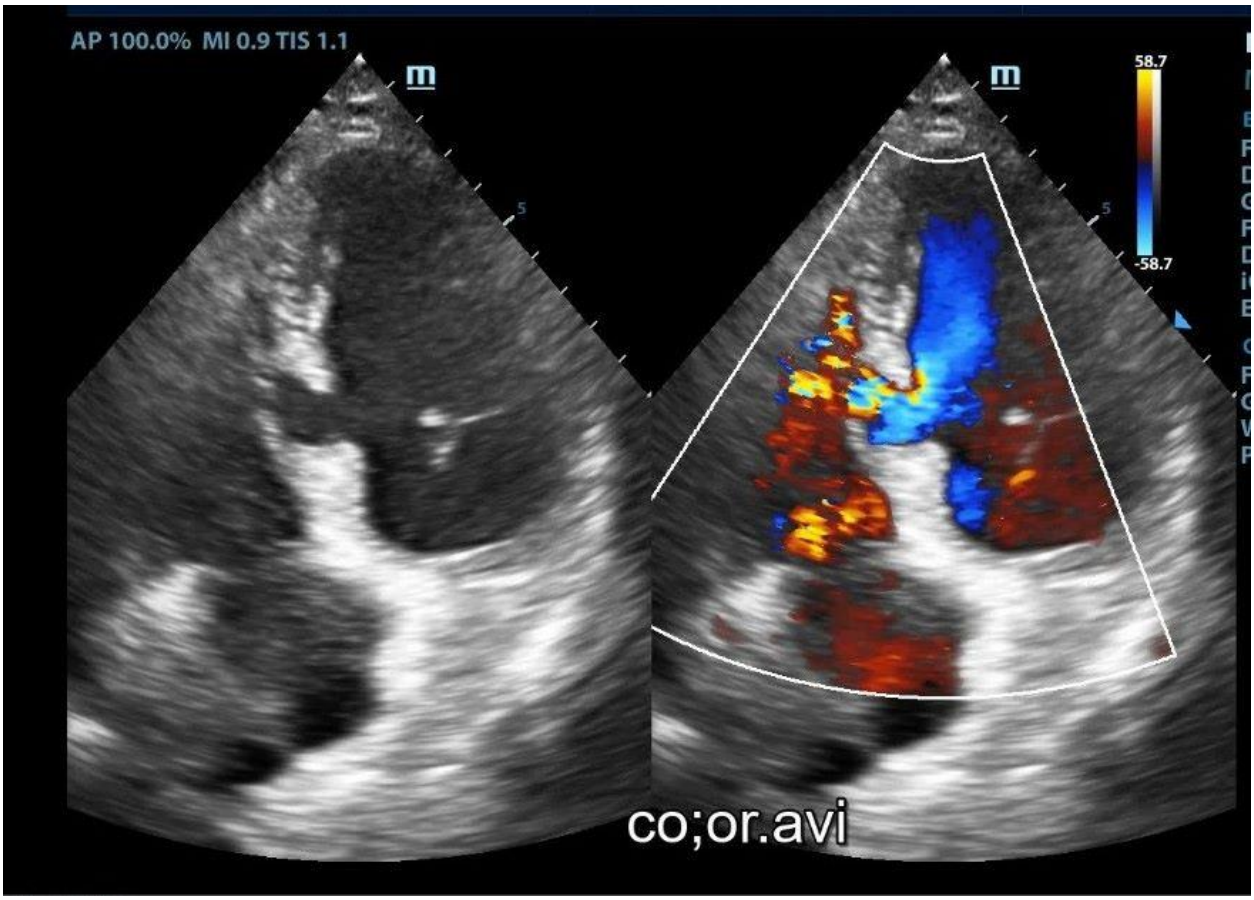

Fig 6-

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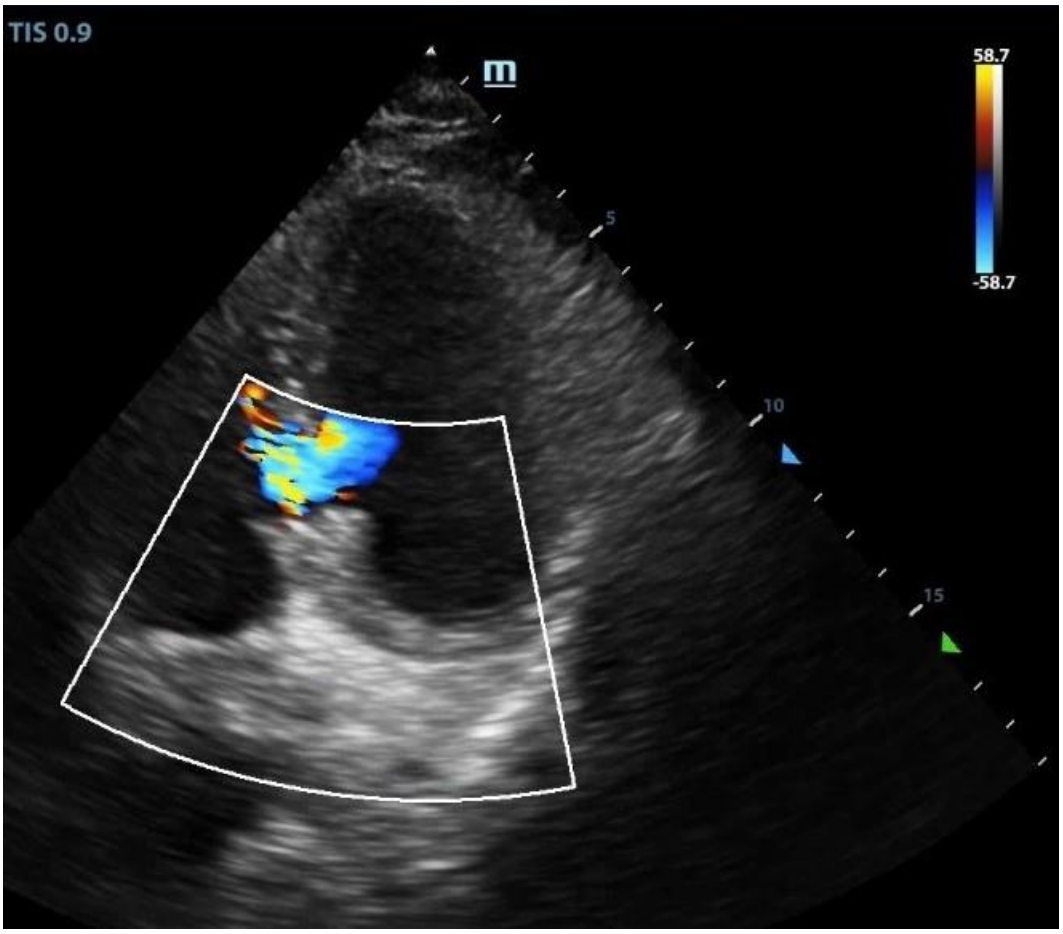

Fig 7-

Discussion

VSR: The incidence is ≈0.3%. VSRs are typically occurring 3 to 5 days after infarction. Anterior, apical or posterior VSDs More with anterior than inferior MI. Inferior infarcts are associated with "complex VSDs, those with multiple, irregular, and variable interventricular connections."

Surgical management: Conservative management is associated with 80 % mortality at 30 days. After allowing a few weeks for tissue healing, surgical Closure is the best option. Risk of rupture extension and Death while waiting for surgery. surgical repair of VSR + CABG With operative mortality 40%, risk of recurrence

Device closure: PCI+ VSD closure with high Mortality, complications, and technical challenges.

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Fig 8: Devices that can be used for VSD closure

- 53 patients with VSR
- Successful device implantation 89%
- Shunt reduction complete 23%, partial 62%
- Emergency surgery 7.5%
- Survival 58%

The 2021 ACCF/AHA guidelines for the management of patients with STEMI who develop cardiogenic shock include the following [14]:

- Immediate transfer to a percutaneous coronary intervention (PCI)-capable hospital for coronary angiography for suitable patients (class I; level of evidence, B)
- Primary PCI should be performed, irrespective of time delay from the onset of myocardial infarction (MI) (class I; level of evidence, B)

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- Urgent coronary artery bypass grafting (CABG) is indicated in patients with coronary anatomy not amenable to PCI (class I; level of evidence, B)
- Fibrinolytic therapy should be administered to patients who are unsuitable candidates for either PCI or CABG (class I; level of evidence, B)
- Beta-blockers are contraindicated (class I; level of evidence, B)
- Intra-aortic balloon pump (IABP) counterpulsation can be helpful for patients who do not quickly stabilize with pharmacologic therapy (class IIa; level of evidence, B)
- Alternative left ventricular (LV) assist devices (LVADs) for circulatory support may be considered in patients with refractory cardiogenic shock. (class IIb; level of evidence, C)

Cardiogenic shock: The incidence (7%–10%). High Mortality with 30-day Mortality approximating 40% to 45%. 1999 SHOCK trial demonstrated a survival benefit with early revascularization in myocardial infarction cardiogenic shock.

Immediate coronary angiography and PCI of the IRA (if indicated) is recommended in patients with CS complicating ACS (class 1)

Emergency CABG is recommended for ACS-related CS if the PCI of the IRA is not feasible/unsuccessful.

In cases of hemodynamic instability, emergency surgical/catheter-based repair of mechanical complications of ACS is recommended, based on the Heart Team discussion.

Fibrinolysis should be considered in STEMI patients presenting with CS if a PPCI strategy is not available within 120 min from the time of STEMI diagnosis and mechanical complications have been ruled out class Ii

In patients with ACS and severe/refractory CS, short-term mechanical circulatory support may be considered. Class IIb

The routine use of an IABP in ACS patients with CS and without mechanical complications is not recommended. Class III

Fig 8 shows the Matching of mechanical circulatory support (MCS) types with clinical presentation.

Revascularization:

Mechanical circulatory support (MCS): The IABP-Shock II trial, routine IABP using an IABP, was not associated with a reduction in 30-day all-cause mortality.

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Evaluation: The chest x-ray may reveal left ventricular enlargement and florid pulmonary edema. Two-dimensional echocardiography with Doppler is used to diagnose ventricular septal rupture, which shows blood flow across the ventricular septum.[7]

Differential Diagnosis: Acute mitral regurgitation due to papillary muscle rupture, Free wall rupture, Tricuspid regurgitation, Congenital ventricular septal defect, Atrial septal defect, Acute flash pulmonary edema

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Complications: Cardiogenic shock, Ventricular aneurysm, Thrombus formation, Ventricular arrhythmias, Free wall rupture, Death

Discussion Ventricular septal defect (VSD) is a rare mechanical complication of myocardial infarction, especially in the era of reperfusion therapy [2]. It usually occurs on days two to seven following a transmural infarction secondary to complete occlusion of any coronary vessels with septal branches supplying the interventricular septum in the absence of collaterals. Occlusion of the left anterior descending artery is the most common cause [2]. Ventricular septal rupture (VSR) can occur following anterior and inferior MI. Typically, the defect due to anterior MI is apical and straightforward. VSR secondary to inferior MI tends to be a more complex lesion with more significant tissue destruction. It has been reported as a complication of right ventricular infarction [3]. Cardiac rupture occurring 24 hours after MI results from dissecting intramural hematoma. The subacute course, which occurs within a week after myocardial infarction, is the result of a cascade of pathological events that starts with ischemia of the myocardium, then coagulative necrosis, then neutrophilic infiltration with subsequent macrophage infiltration and removal of necrotic tissue leading to the weakening of the tissue culminating in complications such as a ventricular free wall rupture, interventricular septum rupture, or a papillary muscle rupture. A much rarer type can occur for > two weeks following perforation of thinned aneurysmal myocardium [4]. Ventricular septal rupture complicates 0.2% of acute MIs, compared with 1-2% before thrombolysis was introduced [2]. Multiple observational studies identified severe risk factors that increase the risk of cardiac rupture, including rupture of the interventricular septum, first incidence of MI, ST-segment elevation, female sex, previous stroke, positive initial cardiac biomarkers, older age (>70), and higher heart rate. On the other hand, a

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history of MI with primary percutaneous coronary intervention (PCI) and low molecular weight heparin and beta-blockers during the first 24 hours were identified as protective factors for cardiac rupture [4-7]. Patients with a ruptured septum may present many symptoms and signs, ranging from mild dyspnea at exertion to severe cardiogenic shock. When the onset of hemodynamic compromise is immediate, hypotension and tachycardia are present. Biventricular heart failure with predominant right-sided failure may be present. Rupture of the septum leads to left to right shunt with subsequent right ventricular failure that can progress to pulmonary edema and biventricular failure. On physical exam, a new cardiac murmur is nearly always present. The new murmur is typically harsh, loud, and holosystolic and is heard best at the lower left and usually right sternal borders. Sometimes, the murmur is heard best at the apex and may be mistaken for acute mitral regurgitation. A thrill can be detected in up to 50% of patients; right ventricular lift and a hyperdynamic precordium may also be noted. Diagnosis is confirmed by transthoracic echocardiography, which will show disrupted ventricular septum with evidence of left-to-right shunt by color Doppler, left cardiac catheterization (evidence of left-to-right

Prognosis

Post-MI VSR carries a high mortality. The earlier the repair, the higher the Mortality, as the sutures do not hold in friable tissue. Mortality is increased in patients with cardiogenic shock. Overall, Mortality is slightly lower for patients with an anterior VSR than a posterior VSR. Other negative prognostic factors include advanced age, multiorgan failure, and advanced New York Heart Association (NYHA) class. Predictors of Mortality within 30 days include:

Shock at surgery, Renal failure, Need for emergency intervention, Significant coronary disease, mainly right coronary and circumflex disease, Duration of surgery, Prolonged cardiopulmonary bypass time, Incomplete revascularization

The prognosis is favorable if the rupture size is small and the patient is hemodynamically stable during surgical repair.

Surgical Closure

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There are two surgical techniques for repairing ventricular septal rupture, including the Daggett and David procedures.

- Daggett procedure: Patch over the defect with sutures in both ventricles (infarct inclusion technique)
- David procedure: Patch over the rupture with stitches in the left ventricle only (infarct exclusion technique)

Repairing a posterior ventricular septal rupture is more challenging than an anterior rupture due to the proximity of the papillary muscles. If the ventricular septal rupture develops within 24 hours of an MI, surgical intervention is more problematic as it is difficult to differentiate between healthy and newly infarcted tissue. Also, at this point, the muscle is weak and unable to hold the sutures. Additional procedures that may be required during the report of a VSR include mitral valve replacement, coronary artery bypass, and resection of the left ventricular aneurysm.

Percutaneous Closure: Percutaneous Closure of a VSR has been developed. Fig 9 shows Devices that can be used for VSD closure.

The Mortality of surgical intervention within 24 hours of acute myocardial infarction is over sixty percent. In contrast, the untreated ventricular septal rupture has a 40% to 80% mortality. Late surgical intervention has a good prognosis; however, this may not be an option for a patient with a hemodynamic compromise.

Surgical intervention within seven days of this complication has a mortality of 54.1%. On the other hand, surgery after seven days has a death rate of 18.4%.[9] Patients presenting with cardiogenic shock need an intra-aortic balloon counterpulsation to reduce afterload and increase cardiac output. Percutaneous intervention can repair anterior defects and ventricular septal defects less than 1.5 cm in diameter, but this technique is still in an evolutionary phase.[10]

Conclusions

A ventricular septal defect is a known, however rare, complication of myocardial infarction. Symptoms range from mild dyspnea to overt cardiogenic shock. Our case highlights an

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unfortunate patient with a VSD secondary to myocardial infarction (MI) resulting in Death MI. Awareness of this fatal complication may make healthcare providers recognize it quicker and act accordingly. More research studies should be geared towards optimal ways to detect and manage VSDs secondary to myocardial infarctions.

Abbreviations:

ACS = acute coronary syndrome;

ECMO = extracorporeal membrane oxygenation

IABP =Intra-aortic balloon counter-pulsation

MCS = Mechanical circulatory support

MI = myocardial infarction

VA = veno-arterial and VV veno-venous

VSD = ventricular septal defect

VSR = ventricular septal rupture

Declaration

- Ethical Approval and Consent to participate: The Faculty of Medicine Ethics office approved the publication of this case report.

"Written informed consent was obtained from the patient's family for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal."

- Availability of supporting data: The authors declare that data supporting the findings of this study are available within the article and in the listed references. .

- Competing interests: No competing interests

- Funding: No funds were received from any gent.\

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- Authors' contributions; Role

- SR wrote and submitted the paper, revised the grammar and plagiarism
- NA collected the data and wrote the case presentation
- RA did the echo and revised the paper
-

Acknowledgments: We thank all nursing staff and technicians who participated in managing the patient.

Data Availability: All data are available on request, the videos are all available, we sent only photos fro videos,

Consent for publication: Written informed consent was not obtained from the patient' family to publish this case report and any accompanying images; the patient died, his name is not mentioned, and no unnecessary investigations were done to benefit this publication.

Consent for publication: Consent was obtained by all participating authors in this study.

Competing interests, Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following:
Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work.
Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. Other relationships: All authors have declared that no other relationships or activities could appear to have influenced the submitted work.

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