



Ventricular Septal Defect Posterior to Acute Myocardial Infarction

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ABSTRACT: We present the case of an 84-year-old woman who arrived at our unit with chest pain of cardiac origin, a Wellens type A pattern on the electrocardiogram, and on physical examination, a holosystolic murmur at the tricuspid and mitral areas radiating to the right parasternal border—a finding suggestive of ventricular septal defect. She was admitted to the Coronary Care Unit for continuous monitoring. Transthoracic echocardiography revealed rupture of the interventricular septum, along with an image compatible with a dissecting interventricular hematoma. These findings confirmed the presence of a left-to-right shunt. This case highlights the essential role of echocardiography in the early detection of mechanical complications following acute myocardial infarction, and underscores the complexity of managing post-infarction septal rupture in elderly patients.

KEYWORDS: Echocardiography, Elderly patient, Myocardial infarction, Ventricular septal rupture, Wellens syndrome.

I. INTRODUCTION

Mechanical complications following acute myocardial infarction (AMI) are uncommon but carry significant mortality when present. These include ventricular septal rupture (VSR), free wall rupture, and papillary muscle rupture, with VSR occurring in approximately 0.2–0.3% of cases in the reperfusion era (1). Despite the availability of reperfusion therapy, these complications remain a diagnostic and therapeutic challenge, particularly in elderly patients or those with delayed presentation. VSR typically presents within the first week after infarction and results in acute hemodynamic deterioration due to a left-to-right shunt at the ventricular level. Early recognition through clinical findings and imaging is essential to guide timely surgical intervention and improve survival outcomes.

II. CASE REPORT

An 84-year-old woman with no known prior coronary disease presented to the emergency department with oppressive chest pain (10/10), radiating to the left arm, accompanied by diaphoresis and emesis. On admission, physical examination revealed a holosystolic murmur at the tricuspid and mitral areas, radiating to the right sternal border. Electrocardiogram demonstrated biphasic T waves in leads V2 to V3, consistent with a Wellens type A pattern (Figure 1). Laboratory results revealed progressively elevated CPK and CK-MB levels, however, cardiac troponin I testing was not available in the unit at the time.

She was admitted to the Coronary Care Unit, where a Swan-Ganz catheter was placed, revealing an oxygen saturation step-up at the right ventricular inflow tract, consistent with a left-to-right shunt (Figure 2). Transthoracic echocardiography showed a left-to-right shunt at the ventricular level. Coronary angiography revealed 50% proximal and 99% mid-segment stenosis in the left anterior descending artery (LAD), no lesions in the circumflex artery, and 85% ostial stenosis of the right coronary artery (Figure 3).

Further imaging with transthoracic echocardiography revealed a 28 mm serpiginous rupture of the interventricular septum, with openings of 4 mm and 3 mm on the left and right ventricular sides, respectively. Color Doppler confirmed a left-to-right shunt with a Qp:Qs ratio of 1:2 (Figure 4). A dissecting interventricular hematoma was also visualized. The patient remained hemodynamically stable (Figure 5) initially but on the final day of hospitalization, she experienced sudden-onset precordial chest pain with an intensity of 10/10. An electrocardiogram was obtained (Figure 6), after which the patient suffered a cardiorespiratory arrest. Advanced cardiopulmonary resuscitation was initiated, but there was no return of spontaneous circulation. She was pronounced dead shortly thereafter, before surgical intervention could be performed.

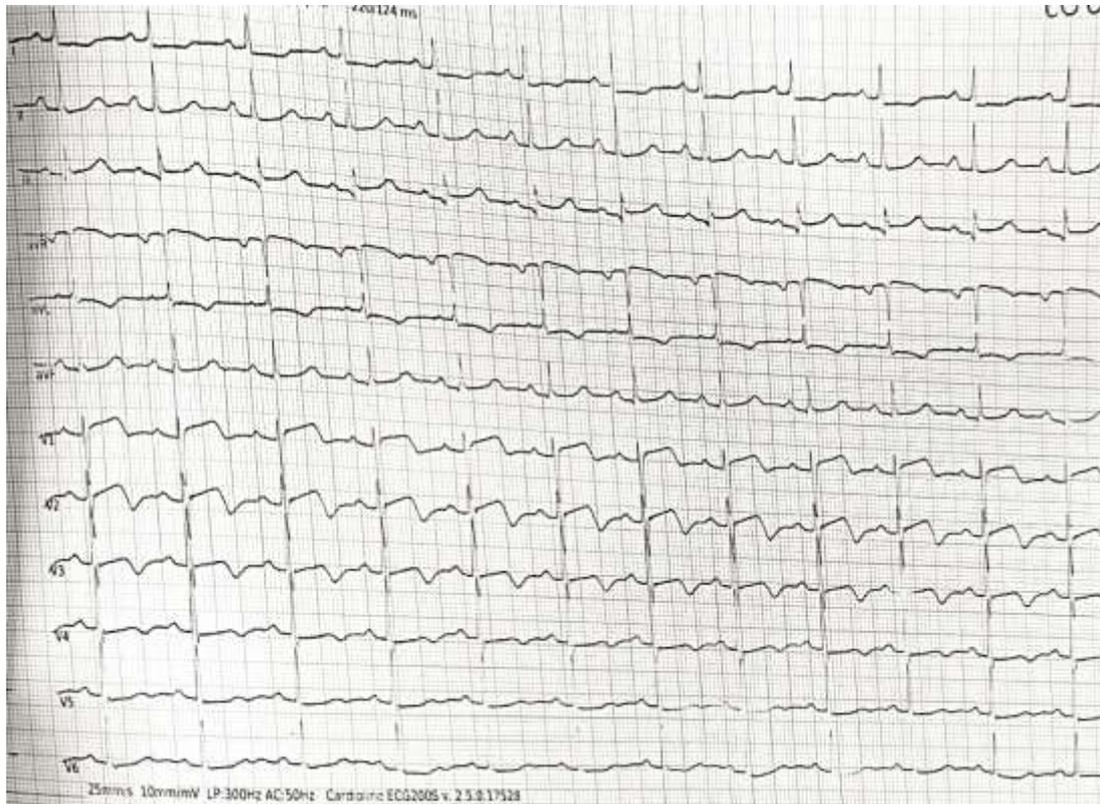


Figure 1. Electrocardiogram showing a Wellens type A pattern, indicative of critical stenosis in the left anterior descending artery.

Pres de Gases en Sangre	7.348	7.353	7.352	7.347	
pH	7.348	7.353	7.352	7.347	
pCO ₂	42.9	39.6	40.9	40.0	mmHg
pO ₂	38.5	59.8	59.5	54.6	mmHg
Pres Corregidos por Temp		Temp	Temp	Temp	
pH(T)c	7.356	7.361	7.360	7.356	
pCO ₂ (T)c	41.8	38.6	39.9	39.0	mmHg
pO ₂ (T)c	36.9	57.4	57.2	52.4	mmHg
Pres de Oximetria					
Hct _c	48.4	42.8	43.5	34.5	%
ctHb	15.8	14.0	14.2	11.3	g/dL
FCO ₂ Hb	0.9	0.6	0.7	0.9	%
FO ₂ Hb	63.8	87.3	86.0	82.2	%
FMetHb	0.9	0.8	0.8	1.3	%
FHHb	34.4	11.3	12.5	15.6	%
sO ₂	65.0	88.5	87.3	84.1	%
BO ₂	9.6	8.5	8.7	6.8	mmol/L

Figure 2. Blood gas samples obtained via Swan-Ganz catheter. Four sequential samples were collected: the first from the floor of the right atrium, the second at the right ventricular inflow tract, the third from the right ventricular outflow tract, and the fourth from the pulmonary artery. An oxygen saturation step-up of 23.5% is observed, confirming a left-to-right shunt from the left ventricle to the right ventricle.

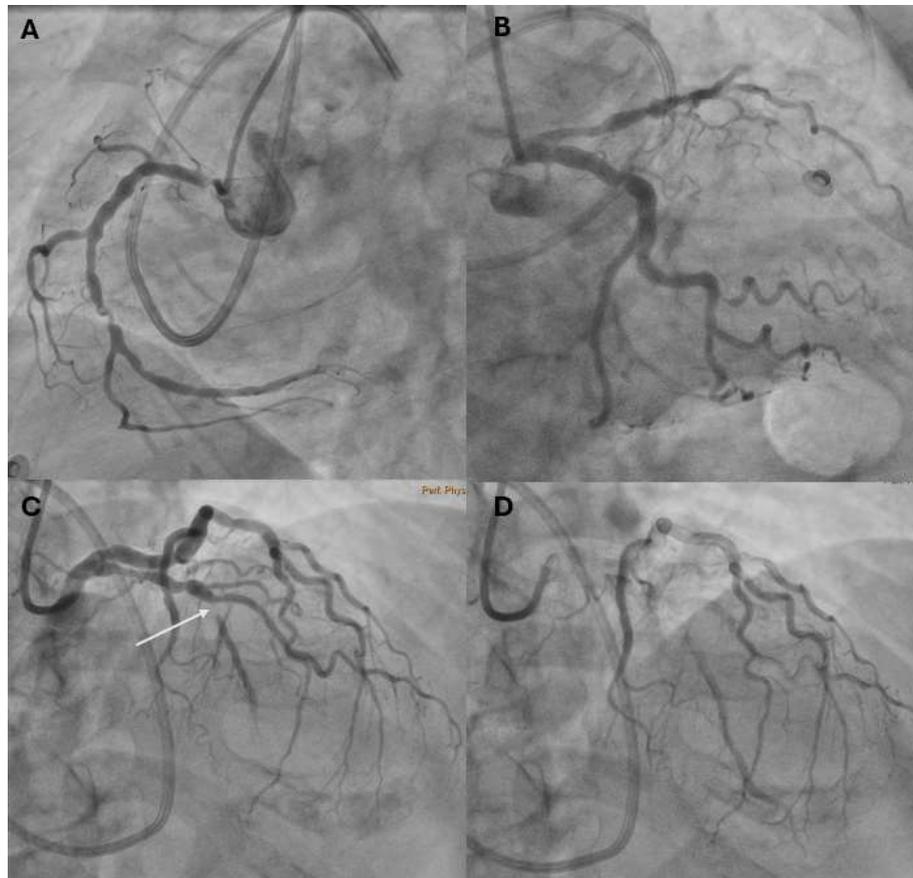


Figure 3. Bilateral coronary angiography. A: Right coronary artery (RCA) showing ostial and mid-segment lesions. B: Projection visualizing the circumflex artery (Cx) without lesions; proximal lesion is observed in the left anterior descending artery (LAD). C: LAD with proximal lesion and a critical mid-segment stenosis (highlighted by white arrow), resulting in slow distal flow. D: Distal LAD beyond the critical lesion, showing delayed flow.

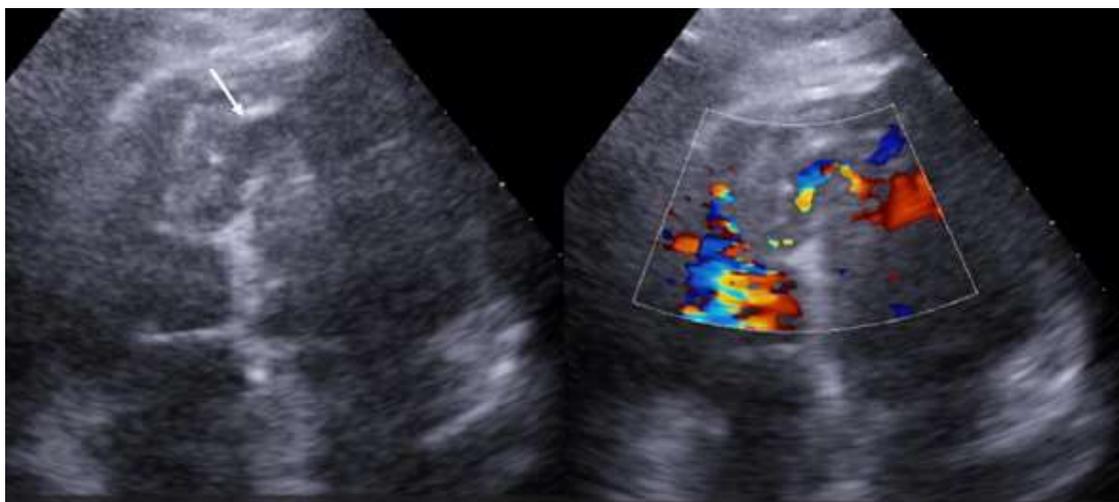


Figure 4. Transthoracic echocardiogram in apical four-chamber view showing disruption of the mid-apical interventricular septum (white arrow). Color Doppler demonstrates left-to-right flow from the left ventricle (LV) to the right ventricle (RV).

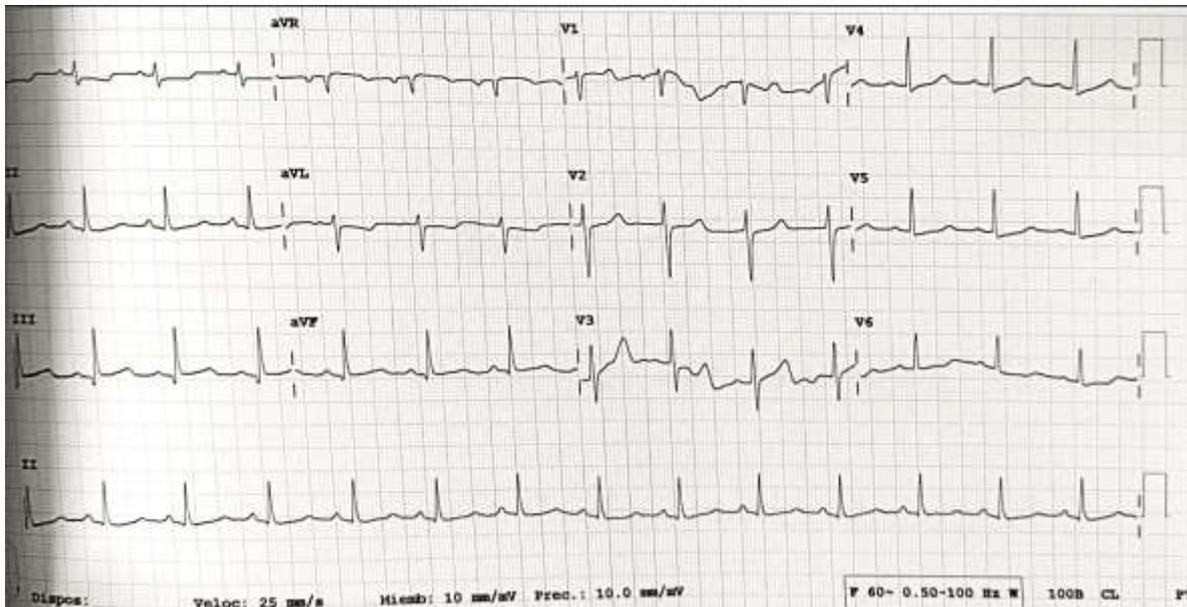


Figure 5. Electrocardiogram on the second day of stay in the coronary care unit, showing resolution of the Wellens pattern.

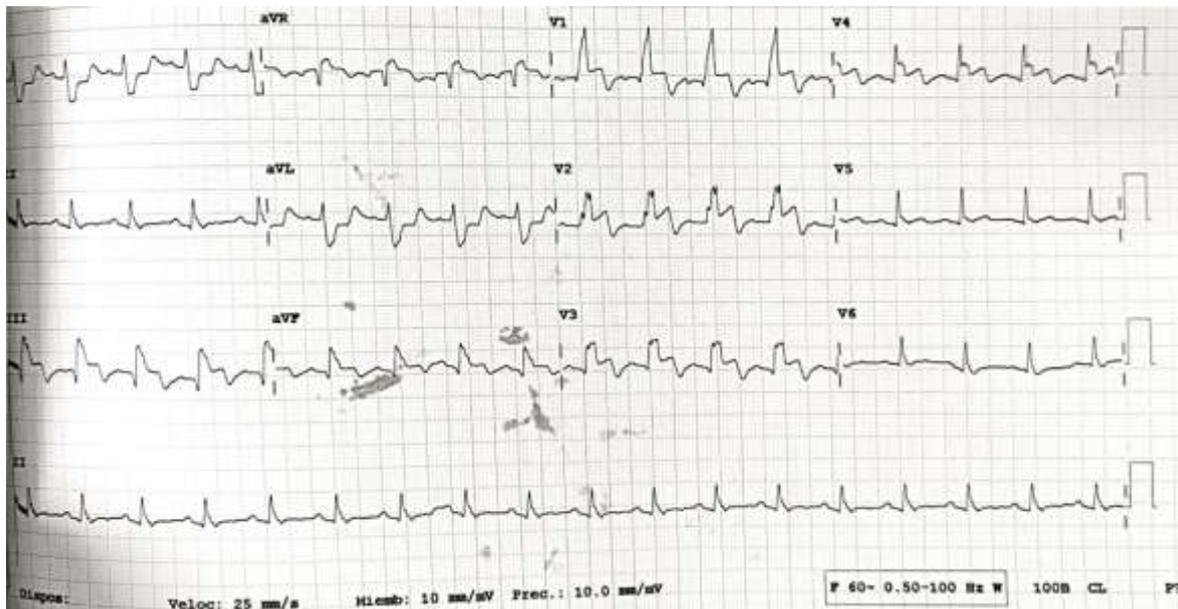


Figure 6. Last electrocardiogram showing complete right bundle branch block and subepicardial injury pattern in the inferior and anteroseptal leads.

III. DISCUSSION

Ventricular septal rupture (VSR) is a rare but catastrophic mechanical complication of acute myocardial infarction (AMI). Its incidence has decreased in the era of early reperfusion, yet its mortality remains extremely high when not treated surgically. Most cases occur within the first week after AMI, particularly in elderly patients, females, and those without timely reperfusion (1, 2). This patient presented with Wellens syndrome, a pre-infarction ECG pattern indicative of critical LAD stenosis (3). The absence of ST elevation underscores the importance of integrating clinical, electrocardiographic, and echocardiographic findings to avoid underestimating myocardial damage.



Echocardiography plays a central role in diagnosing mechanical complications, especially in cases with new murmurs, shock, or unexplained deterioration. In this case, it revealed not only a VSR but also signs of a dissecting interventricular hematoma—an extremely rare finding. This combination likely contributed to the rapid hemodynamic collapse.

Guidelines from the American Heart Association (AHA) and the European Society of Cardiology (ESC) recommend urgent surgical repair as the definitive treatment (2, 3). Surgical closure, when performed promptly, has been associated with improved outcomes; however, data from large databases such as the Society of Thoracic Surgeons (STS) report operative mortality rates exceeding 40% even in contemporary cohorts (4). Some authors suggest delaying surgery in stable patients to allow for fibrosis and reduce the risk of patch dehiscence (5). In our case, the cardiovascular surgery team opted to defer immediate repair due to the friable nature of the tissue and complexity of the defect. In our case, the cardiovascular surgery team opted to defer immediate repair due to the friable nature of the tissue and complexity of the defect. This approach remains controversial and must be individualized.

Adjunctive support with intra-aortic balloon pump or venoarterial extracorporeal membrane oxygenation (VA-ECMO) may bridge unstable patients to surgery, though such strategies are not always available or successful. The literature supports a multidisciplinary strategy combining cardiology, imaging, and cardiothoracic surgery for optimal outcomes (6).

IV. CONCLUSION

Ultimately, this case highlights how mechanical complications such as ventricular septal rupture (VSR) can present insidiously and deteriorate rapidly, even in patients who initially appear hemodynamically stable. It underscores the importance of integrating clinical vigilance with early imaging—particularly echocardiography—to promptly identify structural complications following AMI. In this context, echocardiography proved invaluable for detecting VSR. Given the high mortality associated with VSR, timely recognition, hemodynamic support, and individualized surgical planning are essential. Moreover, this case exemplifies the ongoing debate surrounding optimal timing for surgical repair, especially in elderly or high-risk patients with friable myocardial tissue. A multidisciplinary approach that incorporates cardiology, cardiovascular imaging, and surgical expertise offers the best opportunity to improve outcomes in such complex clinical scenarios.

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