Post Myocardial Infarction Ventricular Septal Rupture Revealed By Acute Liver Failure Symptoms: A Case Report

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ABSTRACT

INTRODUCTION: The mechanical complications of acute myocardial infarction (AMI) still kill despite the evolution of medicine. Early diagnosis and adequate management are necessary to improve the prognosis, and this requires first, a good clinical examination that should raise the suspicion of a mechanical complication, then the echocardiography is performed to confirm the diagnosis.

CASE PRESENTATION: We present a case of a 64-year-old patient admitted to the emergency room for jaundice with delayed ST-segment elevation myocardial infarction (STEMI). Physical examination revealed signs of right heart failure, which led us to associate jaundice with signs of acute liver failure secondary to right heart failure. Echocardiography confirmed the diagnosis of a ventricular septal rupture (VSR) with left-right shunt, and a significant dilation of the right ventricle. The patient underwent surgical closure of the VSR with fatal evolution.

DISCUSSION: VSR is a rare life-threatening mechanical complication of AMI. The clinical signs depend on the left-right shunt and the onset of heart failure, which are 2 major determinants of the therapeutic strategy and the timing of the surgery. Despite surgical closure of the VSR, the mortality remains high, but the prognosis is better in patients treated with surgery than in patients who are treated medically only.

CONCLUSION: The clinical presentation of VSR may differ from a patient to another. Good clinical sense and echocardiography are essential to set early diagnosis, and thus decide on the adequate management at the right time.

KEYWORDS: Ventricular septal rupture, acute myocardial infarction, left-right shunt, acute liver failure

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Introduction

Ventricular septal rupture (VSR) is an uncommon but life-threatening complication of acute myocardial infarction (AMI). Its clinical presentation is atypical, from an asymptomatic patient to cardiogenic shock, and must therefore be systematically sought during an AMI. Echocardiography is the gold standard to establish the diagnosis and shows the septal rupture and the left-right shunt. The existence of this complication requires urgent attention and sophisticated medical and surgical expertise.

In this article, we present the case of a patient with a VSR complicating AMI, with signs of acute liver failure.

Case Presentation

A 64-year-old male presented to the emergency department complaining of jaundice and hepatalgia. He was a smoker (18 pack-years) with a body mass index (BMI) of 24,6. His medical history included arterial hypertension diagnosed several years earlier treated by angiotensin receptor blockers, and chronic renal failure with moderately decreased function.

On admission, the vital signs included heart rate of 110 beats per minute, blood pressure 114/76 mmHg, tachypnea with an oxygen saturation above 96% on room air. The patient was afebrile. The physical examination showed muco-cutaneous jaundice, associated with signs of right heart failure (jugular vein distention and hepatomegaly). Cardiac auscultation revealed a harsh, holosystolic murmur at the lower left sternal border. Lung auscultation was otherwise unremarkable. Upon questioning further, the patient reported an episode of severe chest pain lasting several hours, dating back 5 days.

An 18-lead electrocardiogram (ECG) (Figure 1) performed on admission revealed a sinus rhythm, a right bundle branch block, with ST segment elevation in the anterior leads with necrosis Q waves, as well as reciprocal ST segment depression in the inferior and posterior leads. Transthoracic echocardiography (TTE) (Figure 2) revealed a VSR measuring 15mm, with a left-right shunt, which was demonstrated on color Doppler by the presence of flow across the interventricular septum. Left ventricular (LV) systolic function was depressed with

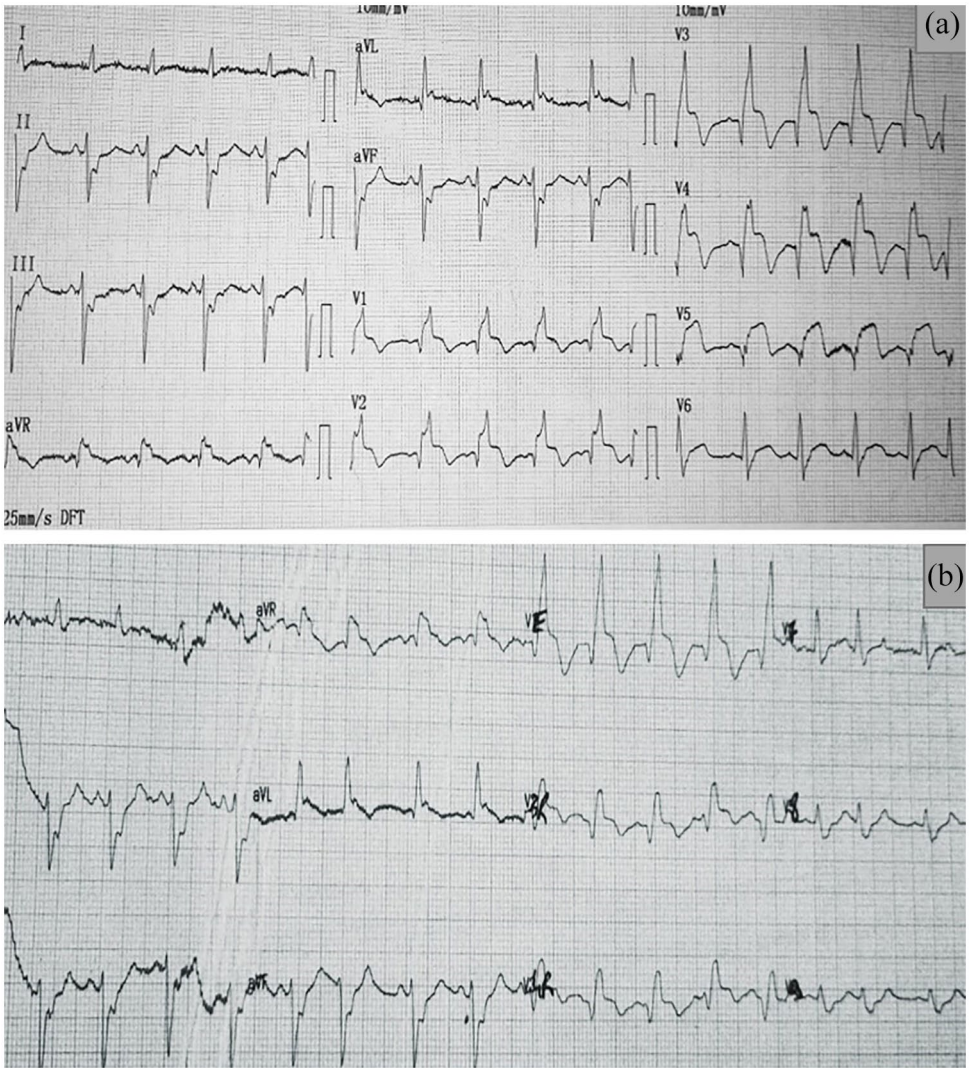

Figure 1. (a) ECG1 showing right bundle branch block, with ST segment elevation in the anterior leads with necrosis Q waves, and reciprocal ST segment depression in the inferior leads and (b) ECG2 showing reciprocal ST segment depression in leads V7 V8 V9.

an ejection fraction of 40%. The anterior wall, interventricular septum and the left ventricular apex were all akinetic. The right ventricle (RV) is dilated with systolic function in dysfunction: RV systolic annular velocity (S') at 8 cm/s, tricuspid annular plane systolic excursion (TAPSE) at 9 mm and tricuspid regurgitation with a pulmonary artery systolic pressure of 50mmHg. The right atrium is dilated, while the left atrium is normal. Laboratory analysis showed an elevated troponin T at 29 753 ng/L (normal range<50 ng/L). Additional blood tests showed a disturbed liver function. The results were as follows: total bilirubin = 35 mg/L, direct bilirubin = 24 mg/L, alkaline phosphatase = 275 U/L, AST = 7394 U/L (N: 5-34), ALT = 5355 U/L (N: 0-55), with a PT = 26% and INR = 2.78. the result of the admission arterial blood gaz analysis is normal.

Coronary angiography performed 4 hours after admission showed a total occlusion of the left anterior descending artery (LAD) with TIMI grade 0 flow. The other arteries were without significant lesion. (Figure 3).

After discussion between cardiologists and cardiovascular surgeons, given that the patient already had a secondary complication of acute right heart failure, as well as the unavailability of circulatory support, a strategy of early surgical repair was chosen, as the choice of a delayed surgery could worsen the right heart failure. The decision was then made for immediate surgical repair on 1 day of his admission and 5 days after the infarction. The decision was to perform a surgical closure of the shunt and despite the myocardial necrosis and the acute hepatic insufficiency as well as the altered general state, we wanted to take advantage of the surgical repair of the septal rupture with a revascularisation of the anterior territory, since we considered that we could have a preserved viability in this territory. For this purpose a monotruncular bypass of the LAD is performed at the same time.

After placing the patient in supine position, induction of anesthesia was performed without incident. Intraoperative transesophageal echocardiography (TEE) showed a 25 mm wide septal rupture without mitral regurgitation. The patient

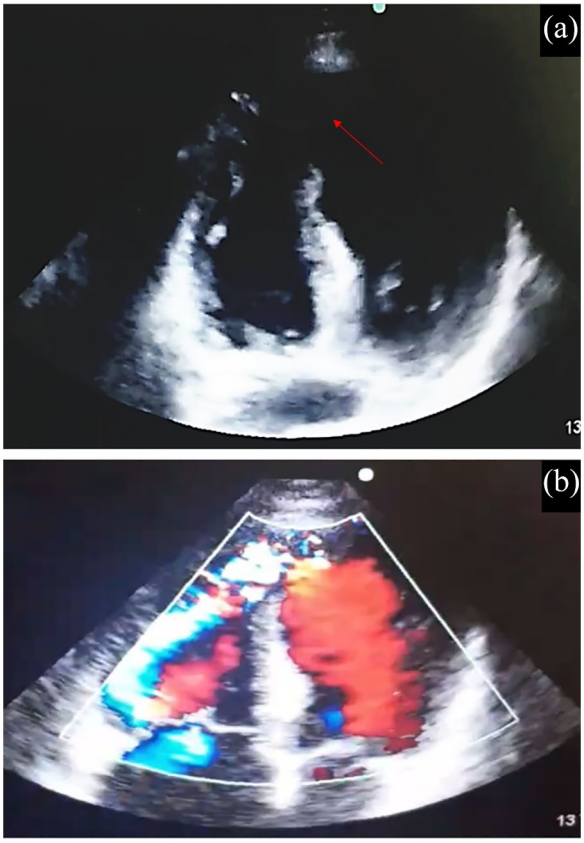

Figure 2. (a) Four chamber cardiac view TTE showing the ventricular septal ruptureand (b) four chamber cardiac view TTE color Doppler showing the left-right shunt with tricuspid regurgitation.

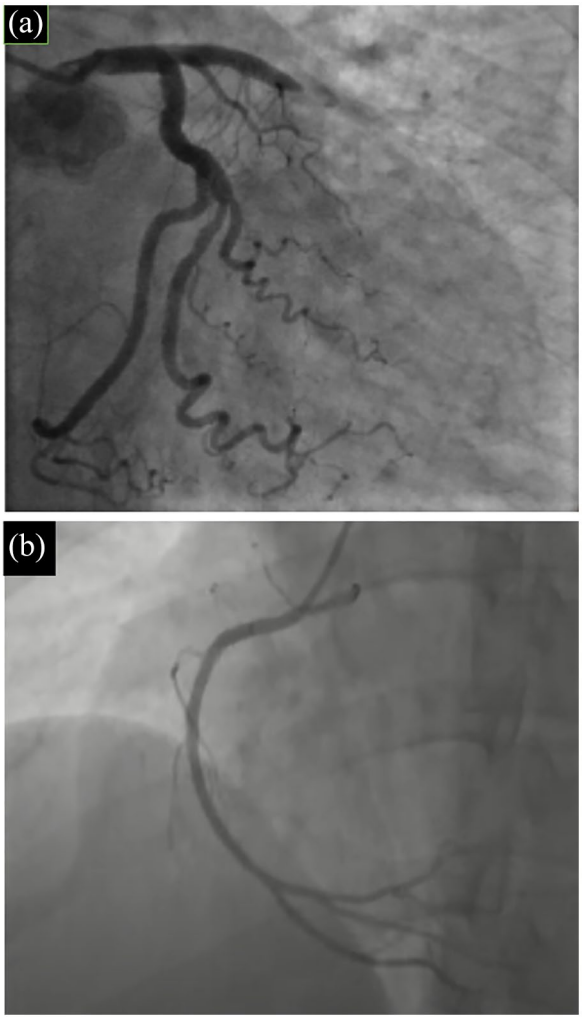

Figure 3. (a) Coronary angiography showing a total occlusion of the left anterior descending artery (LAD) with TIMI grade 0 flow with normal flow in the circumflex artery and (b) coronary angiography showing right coronary artery without anomalies.

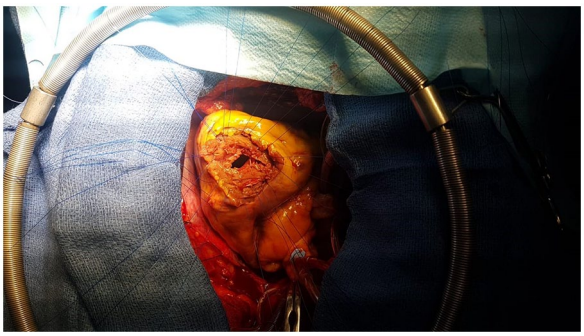

Figure 4. Surgical visualization of septal rupture after right ventriculotomy.

was approached through median sternotomy, and extra-pleural pericardiotomy is performed. Anticoagulation with unfractionated heparin was started with a dose of 3 mg/kg. Exploration showed dilated right and left cavities with ectasia of the left atrium. After establishing cardiopulmonary bypass (CPB) using aortic and bicaval cannulation, the closure of the VSR was started. The first step was to perform a vertical right anterior ventriculotomy allowing exploration of the VSR, which occupied the anterior 2/3 of the septoapical wall, which was weakened by ischemia up to the postero-external pillar of the mitral valve (Figure 4). The closure was achieved by a 0.6 mm PTFE (polytetrafluoroethylene) patch measuring 5 cm in diameter, fixed on the external side of the septal wall of the LV using 4-0 prolene, and by a second bovine pericardial patch of the same diameter which was fixed on the inner side of the septal wall of the LV (Figure 5). The ventriculotomy was closed by 2 Teflon strips reinforced by an external bovine pericardium patch. The second operative step consisted of bypassing the LAD using the left internal mammary artery, with the anastomosis performed on the LAD end-to-side.

The total duration of CPB was 150 minutes, and that of aortic clamping 103 minutes. Hemodynamics were stable under 5 µg/kg/minute of dobutamine. A postoperative TEE did not show a residual shunt. The patient was then transferred to the intensive care unit.

The day after the surgery, the patient was extubated and remained stable after successful withdrawal of dobutamine. The evolution was marked by the onset of confusion with

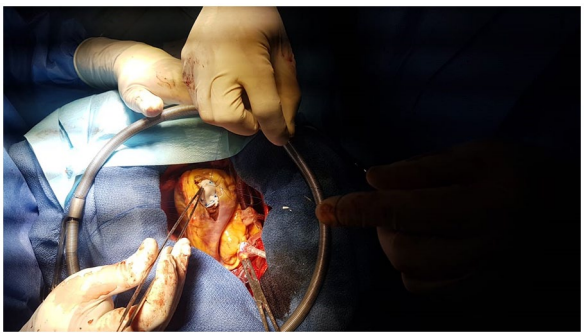

Figure 5. Fixation of the patch during the surgical repair of the ventricular septal rupture.

asterixis, and biologically worsening of liver function (PT = 17%, factor V level = 19%). The brain imaging was normal. A laboratory panel for other causes of hepatitis was performed, including viral serologies and screening of acetaminophen, and all the investigations were negative. The abdominal ultrasound and Doppler tracing showed severe dilation of the portal vein with reversed flow, without signs of vein thrombosis. We concluded to the diagnosis of hepatic encephalopathy in fulminant hepatic failure secondary to acute heart failure. The patient was put on lactulose, with no noticeable improvement. Unfortunately, the patient died 3 days later.

Discussion

VSR is a rare and life-threatening complication of AMI that should be diagnosed early.[1] With the evolution of acute coronary syndrome reperfusion strategies, the incidence of VSR has become increasingly rare.[2] Before the era of reperfusion therapies, VSR was thought to complicate 1% to 3% of AMI cases,[3] however the introduction of reperfusion therapies has decreased the incidence of VSR, which is now reported to be complicating between 0.17% and 0.31% of patients presenting with AMI.[1,4] The incidence also depends on the type of revascularization, and is lower in patients treated with primary angioplasty (0.7%) compared to patients treated with thrombolysis (1.1%).[5] The factors most associated include advanced age, high BMI, hypertension (as in our case), female sex, first episode of AMI (as in our case) and the absence of smoking.[6] VSR is more common in anterior AMIs. In these cases, the defect is typically simple, in which the entry and exit points of the communication are positioned at a similar level across the ventricular septum. Complex VSRs occur more frequently in inferior AMIs, thus there may be several channels between the left ventricle and the right ventricle across the necrotic interventricular septum.[7]

The majority of VSR occurs 3 to 5 days after the AMI.[6] In our case, the patient reported chest pain that dated back to 5 days, which suggests that the AMI occurred 5 days before admission and that the VSR developed during this time interval. The clinical presentation is nonspecific and varies from a simple asymptomatic heart murmur as in our case to a fatal cardiogenic shock. The clinical signs are mainly explained by the left-right shunt that causes right heart failure. The systolic murmur is present in 90%[2,7] which was the main guiding sign in our case, and therefore any patient presenting with AMI with a systolic murmur on auscultation should be carefully examined in order to exclude a VSR.

ECG is important for the diagnostic orientation of VSR. It shows STEMI in the majority of patients with VSR, the anterior area is more commonly affected (60%) as in our case than the inferior area (40%).[7] Echocardiography plays a key role in the diagnosis of the VSR. It allows to quickly identify the shunt, its location, and the size of the rupture as well as to eliminate other mechanical complications.[8]

Patients with a VSR should be treated immediately. The medical management is based on afterload reduction using intravenous vasodilators and intra-aortic balloon pump in order to reduce the left-right shunt. In case of pulmonary edema, diuretics should be used carefully. Vasopressors are used in case of hemodynamic instability. Circulatory support is an alternative treatment for hemodynamically unstable patients, with the aortic counterpulsation balloon being the traditional method, and the impella, a more recent method. These 2 means of circulatory assistance can help in case of cardiogenic shock after septal rupture, but to date, there is no randomized study to prove the superiority of 1 means over the other.[9]

This non-surgical treatment is only a temporary solution, since the prognosis in patients treated only medically is severe, with in-hospital mortality reaching 94% to 100%.[10] The treatment of choice is surgery despite very high morbidity and mortality.[7] European guidelines suggest that surgery should be indicated in a patient who has not responded to medical treatment, [11] however, US guidelines suggest that regardless of the patient's clinical condition, surgery should be performed as soon as the diagnosis is made.[12] The big challenge is to define the timing of surgery, which is still controversial. The fragile necrotic tissue of the heart is technically a problem for early repair. In addition, patients who underwent early surgical repair ($\leq$2 days after diagnosis) present a higher morbidity and mortality than those who underwent delayed surgery ($>$2 days after diagnosis). If the patient has refractory heart failure, with multivisceral failure, or in the extreme case of refractory cardiogenic shock, immediate surgery should be indicated[13]. Unfortunately, the limiting factor of delayed surgeries is the onset of complications and hemodynamic instability.[14,15] Percutaneous closure of VSR is an innovative treatment that can be indicated in selected cases.

In our case, the patient presented with acute hepatitis complicated by hepatic encephalopathy with a severe hemostasis disorder, in the context of acute hepatic failure of cardiac origin, which made the postoperative period very difficult, but since the liver failure was secondary to the right ventricular failure, surgical repair was necessary.

Conclusion

In conclusion, VSR is a rare and serious complication of AMI which is accompanied by a very high mortality despite the evolution of medicine. Its early diagnosis is essential to initiate the appropriate treatment for patients on a case-by-case basis, depending on the time to onset of the infarction, the complications and the factors of excess mortality. But in order to avoid this complication, the best attitude would be to act upstream by establishing an early diagnosis of AMI and treating it in time.

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Consent for Publication

Obtained.

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