

Post-infarction ventricular septal rupture; a case report: An insight to the Americas

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SOUHRN

Ruptura mezikomorového septa (ventricular septal rupture, VSR) představuje jednu z možných mechanických komplikací po akutním infarktu myokardu (AMI). I když se s ní nelze setkat často, je spojena se zvýšenou mortalitou. U většiny pacientů lze pozorovat aktivní klinické projevy, u menšího procenta z nich však místo toho dochází k dalšímu tichému rozvoji s nástupem subakutního srdečního selhání (heart failure, HF) nebo s dekompenzací. Po stanovení diagnózy je naprostě nezbytné urychleně zahájit léčbu. Nutná je léčba farmakologická (tzn. podávání anti-ischemické medikace včetně léčiv snižujících afterload) spolu s definitivní korekcí, buď intervenční, nebo chirurgickou, protože neprovedení korekce VSR vede neodvratně k úmrtí. Mnoho rozvojových zemí nicméně trpí omezeným přístupem ke zdravotní péči; výsledkem je opožděná nebo nedostatečná lékařská péče. Popisujeme případ starší ženy s dekompenzovaným HF v důsledku ruptury apikálního septa, u níž se v důsledku několika problémů – nesouvisejících se zdravotní péčí – prováděla pouze farmakologická léčba a která uvedené komplikaci nakonec podlehla.

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ABSTRACT

Ventricular septal rupture (VSR) constitutes one of the possible mechanical complications following an acute myocardial infarction (AMI), even though very infrequent, it bears an elevated high mortality rate. Although most patients develop florid clinical manifestations, a minority might have a silent evolution, experiencing a subacute heart failure (HF) onset or decompensation instead. Once identified, prompt treatment is mandatory. Management, consistent of medical therapy (i.e.; anti-ischemic and afterload reducing medications) along with definite repair, either through interventional or surgical technique, is necessary, since if uncorrected, VSR ultimately leads to death. However, many developing countries face an inadequate healthcare access, resulting in delayed and impoverished medical attention. We present the case of an elderly woman with decompensated HF due to an apical VSR, and as a result of several extra-medical issues, only medical therapy was established, and as feared, she succumbed to the disease.

Keywords:

Cardiovascular diseases in the Americas
Mechanical complication of myocardial infarction
Ventricular septal rupture

Learning points

- Every patient diagnosed with heart failure should undergo an intense evaluation, ensuring a thorough assessment of the etiology, differential diagnosis, appropriate imaging tests, and guideline-based medical therapy. This approach aims to enhance both the quality of life and overall survival.
- Although infrequent, post-myocardial infarction mechanical complications still imply a fatal evolution. Thus, their prompt diagnosis and correction become imperative.

The presence of a solid healthcare system with universal access is essential to ensure timely reperfusion and effective management of affected patients.

Introduction

Since the reperfusion era (i.e.; thrombolysis and percutaneous coronary angiography) post-AMI mechanical complications incidence has constantly declined, from 1–2% to 0.17–0.31%.^{1,2} This mortality reduction is partially ex-

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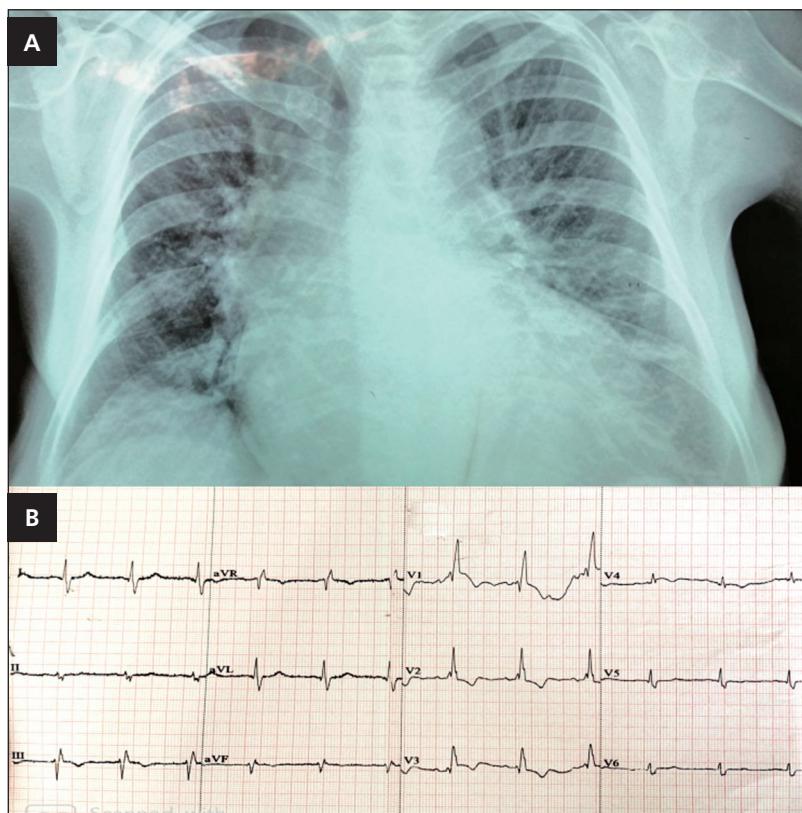


Fig. 1 – (A) Chest roentgenogram shows significant cardiac silhouette enlargement and bilateral interstitial fluid retention. **(B)** Twelve leads electrocardiogram reveals sinus rhythm and right bundle branch block along with expected repolarization alterations.

plained by a more aggressive approach towards risk factors together with more efficient anti-ischemic strategies in AMI management. When corrective treatment is neg

Case presentation

A 64-year-old woman, previously diagnosed with arterial hypertension referred progressive dyspnea, bilateral lower limb edema, non-productive cough, and a significant non-painful abdominal circumference enlarging. Physi-

cal examination confirmed symmetrical bilateral edema, painless ascites, and an outward and slightly left displacement of the apex impulse. Chest X-ray (Fig. 1A) revealed cardiomegaly and moderate congestion; the electrocardiogram (ECG) (Fig. 1B) only showed right bundle branch block. After establishing HF diagnosis, medical treatment composed of diuretics, spironolactone, enalapril, and digoxin led to clinical improvement, and was latter referred for outpatient follow-up. An initial transthoracic echocardiogram (TTE) revealed left ventricular (LV) chamber dilatation, moderate LV dysfunction (ejection fraction of

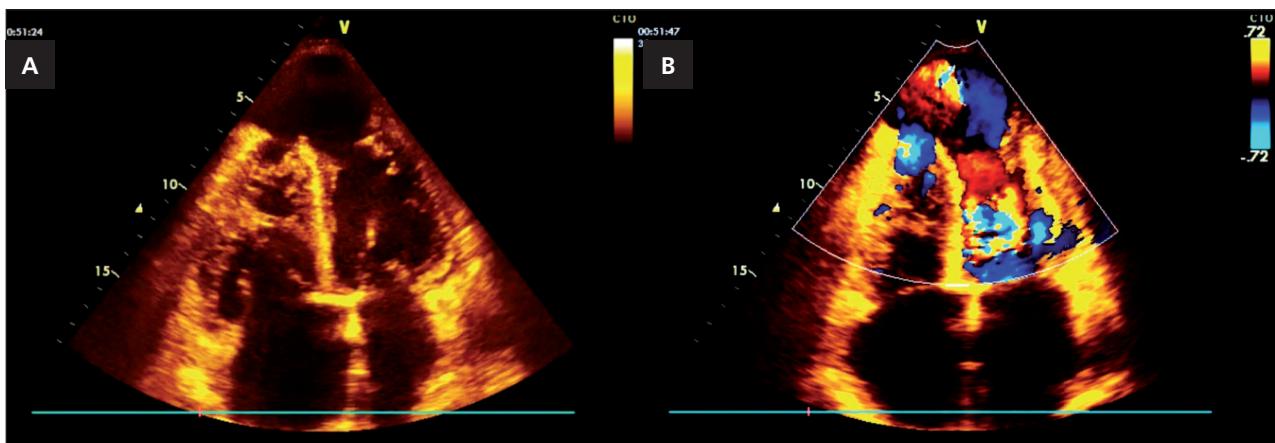


Fig. 2 – Transthoracic echocardiogram, 4-chamber apical view shows an apical septal bulging secondary to an aneurysmatic formation **(A)** along with VSR with a left to right shunt appreciated thanks to aliasing presence **(B)**.

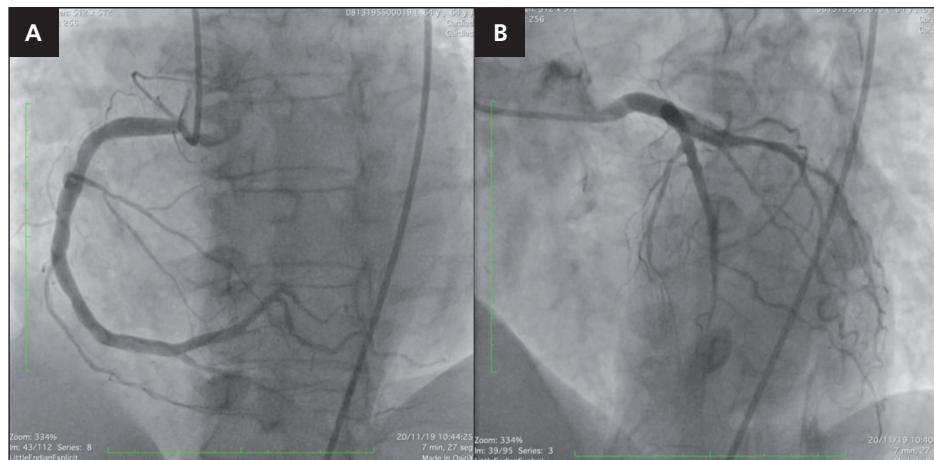


Fig. 3 – (A) Coronary angiography – anteroposterior cranial view shows multiple non-significant lesions in the right coronary artery. **(B)** Significant and diffuse left anterior descending artery disease.

35%), mild to moderate mitral regurgitation, besides anterior, septal, and apical hypokinesis. Therefore, ischemic cardiomyopathy was considered, with statin and aspirin prescription, as well as ongoing etiological and therapeutic approach. The follow-up was interrupted for eight months and she visited the emergency department again due to worsening functional class and marked systemic venous congestion. Upon evaluation, she had stable vital signs and bilateral lung crackles, and a harsh holosystolic murmur grade III/VI was noted in the mesocardium. Routine paraclinical tests did not reveal major abnormalities, but a new TTE (Fig. 2A, 2B) showed the presence of an apical septal aneurysm with 21 mm wide left-to-right shunt VSR, and a 47.6 mmHg gradient, as well as right chambers pressure overload. Coronary angiography was performed, revealing right coronary artery compromise and significant diffuse disease of the left descending anterior (LAD) coronary artery (Fig. 3A, 3B). Right heart catheterization (RHC) recorded a mean pulmonary arterial pressure of 40 mmHg. VSR was classified as Type III secondary to its association with an aneurysm along an unknown evolution (the patient never recalled any acute chest pain). Due to these findings, surgical closure of the VSR was proposed; however, the patient declined the treatment due to the high costs and perceived surgical risk. Unfortunately, three months later, the patient passed away due to decompensated HF.

Discussion

VSR along free wall rupture, aneurysmatic (a severely scarred and non-mobile portion of the myocardium) formation and acute mitral regurgitation, are post-AMI mechanical complications. Risk factors include older age, female sex, prior stroke, chronic kidney disease and HF, as well as ST segment elevation AMI, cardiogenic shock (CS), and longer reperfusion time.^{1,2} It is well established that VSR is less likely to occur in patients with previous diabetes, hypertension, MI or smoking history.^{1,3} Usually it develops in the first three to five days after AMI and rarely can be presented two weeks later. Apical location

(LAD disease) is the more frequent presentation, up to 70% of cases in opposition with inferolateral (inferior or lateral infarcts).^{2,4} Regardless of location, a left-to-right shunt appears, leading to myriad of manifestations, ranging from a minimal symptomatic syndrome to a frank circulatory collapse. Clinical manifestations include congestive HF, hypoperfusion signs along ongoing anterior or inferior ischemia on the ECG; at least half of patients will debut with CS.^{1,4} According to Becker and Mantgem, VSR can be classified onto three different subtypes, where Type III is usually related to concomitant anterior or inferior aneurysms, associated to older infarcts. Further classification divides VSR in simple (through-and-through defect) or complex (serpiginous trajectory). Diagnosis relies on echocardiography assessment, whether 2-D or through Doppler techniques, other imaging methods (cardiac magnetic resonance, cardiac tomography and ventriculography) are quite useful along with RHC. Optimal medical therapy in combination with an adequate reperfusion strategy is mandatory, and reparation/closure should always be pursued, since if untreated, VSR mortality might reach up to 94% at 30 days¹ which is similar to the natural history of VSR itself.⁵ Afterload reduction might lead to an increased effective LV stroke volume and also increases intracoronary perfusion, henceforth the use of vasodilatory medications along with mechanical support is necessary. LV assist devices have not shown any survival augmentation,² however, their use is generally recommended. During the acute phase, is difficult to distinguish upon bare-eye inspection, viable from necrosed myocardium; early surgery (<7 days) mortality reaches up to 54% and decreases up to 18% if delayed (7 days). Therefore, emergency surgery is reserved to patients with CS and refractory pulmonary edema. Percutaneous device closure may represent a valuable alternative to surgical repair, with the advantage of immediate shunt reduction to prevent hemodynamic deterioration and its minimally invasive nature, with 80–100% success rate, which depends on CS presence and delaying time. A defect is considered suitable for percutaneous closure when it has less than 15 mm diameter, along an anterior and simple configuration.

Conclusion

Cardiovascular disease is one of the leading causes of death worldwide, and mortality due to AMI has fallen substantially during the last decades. However, in many developing countries, universal health care access is simply unreachable. Our case reflects the sorrowful evolution of many patients in the Americas, and remind us the need to constantly improve our health politics in order to ensure a more human and integral medical assistance.

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Conflict of interest

The authors declare that there is no conflict of interest.

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Ethical statement

Approval for the publication was granted by the Ethical Committee of the hospital where the patient underwent evaluation.

Informed consent

We obtained the patient's relative consent in order to publish the article. Any information concerning the patient's identity has been omitted.

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