

Title page

Concomitant mechanical complications of true silent inferior myocardial infarction; interventricular septal aneurysm, and ventricular septal rupture mimicking congenital ventricular septal defect (VSD): A case report

Running Title: Concomitant mechanical complications of MI

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Key clinical message

In any patient with ventricular septal defect (VSD), attention should be paid to the regional wall motion abnormality of left ventricle as the VSD may not be congenital and due to mechanical complications of silent myocardial infarction.

Abstract

This case report demonstrate concurrent of two rare complications of true silent inferior STEMI in the poorly controlled diabetic patient with de-novo heart failure, mimicking congenital VSD. This complication of myocardial infarction often have catastrophic manifestations and the presence of this complications of MI with smooth and gradual course is very scarce.

Keywords

myocardial infarction, mechanical complication, interventricular septal aneurysm, ventricular septal rupture, ventricular septal defect

INTRODUCTION

It has been noted that 4% of patients with acute myocardial infarction (MI) develop myocardial rupture, including free ventricular wall rupture and intra-cardiac component rupture comprising ventricular septum and/or papillary muscles. Although timely reperfusion therapy has decreased the incidence of these complications dramatically, the occurrence of these complications is associated with poor clinical outcomes ¹⁻⁴. It is now known that some factors increase the risk of cardiac ruptures, such as female gender, advanced age, ST-segment elevation myocardial infarction (STEMI), higher levels of cardiac enzymes, tachycardia, and low blood pressure at the time of admission ^{5,6}.

The simultaneous presence of the two complications mentioned earlier is scarce ². In the current case report, we aim to describe an acute inferior MI patient with a concomitant interventricular septal aneurysm and ventricular septal rupture.

CASE PRESENTATION

A 62-year old man was referred to the Emergency Department as a case of congenital VSD with clinical manifestation of de-novo heart failure. He had a past history of uncontrolled diabetes mellitus, hypertension, dyslipidemia, a family history of premature coronary artery disease in his younger brother, and paroxysmal atrial fibrillation. Heart failure symptoms began one month ago. He had no complaint of chest pain, clear history of myocardial infarction, and coronary artery disease.

The patient was admitted to another hospital due to COVID-19 infection three months prior to his current hospitalization. In the medical workup performed in that center, congenital VSD was diagnosed as an incidental finding during the evaluation of cardiac complications of COVID-19 infection.

During the physical examination, he had stable vital signs and normal oxygen saturation. A holosystolic murmur (grade 4/6) in his left sternal border and bibasilar lung crackles were auscultated, and the lower extremities were symmetrically edematous.

The laboratory data were unremarkable other than an increased level of NT-proB-type Natriuretic Peptide (3622 pg/ml) and a high level of HbA1c (11.3%). The troponin level was within a normal limit. Electrocardiogram (ECG) at presentation revealed sinus rhythm with normal axis, Q wave and mild ST-segment elevation, T-wave inversion in inferior leads, and mild ST-depression in precordial leads (**Figure 1**).

Spiral chest computed tomography scan showed cardiomegaly, congested lungs, moderate right-sided pleural effusion, and ascites.

A transthoracic echocardiogram (TTE) was then conducted, which demonstrated moderate LV enlargement and moderate LV systolic dysfunction (EF: 40%) with significant regional wall motion abnormality in the inferior and inferoseptal wall, aneurysmal formation in base and mid inferoseptal segments of LV, which created intraventricular pulsatile globular structure (56*49mm) at the mid-part of right ventricular (RV) chamber with paradoxical systolic expansion. This structure was connected to the LV cavity with a narrow neck (21mm) as well as to the RV cavity through four small perforations with a 60 mmHg systolic pressure gradient. The RV was enlarged with moderate-to-severe systolic dysfunction with moderate-to-severe tricuspid regurgitation (TR), and the estimated systolic pulmonary artery pressure (PAP) was 70 mmHg. There was a unidirectional left to right shunt with $QP/QS = 1.95$ through small perforations of this pulsatile structure into the RV chamber. For better evaluation of this pathological finding, TEE was performed, which confirmed the above findings.

Since the patient had diabetes mellitus and regarding ECG abnormalities, the presence of regional wall motion abnormalities in echocardiography, de-novo heart failure, and reduced left ventricular systolic function, the patient underwent coronary angiography, which revealed significant left main (LM) stenosis and three-vessel disease (3VD). Notably, the right coronary artery (RCA) was totally cut off at the mid part without an antegrade and retrograde runoff (figure3). In left ventricular (LV) angiography, LV injection showed LVEF: 40% as well as opacification of the LV followed by septal aneurysm and then right ventricle (figure and video 4). According to this finding, we were confronted with two rare mechanical complications in the true silent inferior MI in a poorly controlled diabetic patient who was misdiagnosed with congenital VSD. These two

mechanical complications was septal aneurysm followed by post-MI VSR. Thus, we recommended the patient undergo surgery for coronary artery bypass graft surgery (CABGS) and aneurysmectomy and ventricular septal repair. Unfortunately, the patient did not come for surgery.

DISCUSSION

Many patients with severe ischemic heart disease (IHD) never experience angina pectoris. The episodes of silent ischemia are higher prevalent in diabetic patients. Early reperfusion strategies have significantly reduced the rate of cardiac ruptures, including ventricular septal rupture (VSR) following acute MI during the recent years ^{2,3}. Concurrent interventricular septal aneurysm and VSR following the acute MI have been rarely reported. Although recently reperfusion therapy has dramatically decreased the incidence of these complications, the occurrence of these complications is still associated with poor outcomes and high mortality rates. For instance, the mortality of post-MI ventricular septal rupture (VSR) alone, despite surgical treatment, is approximately 40-75%. The diagnosis of these complications is very important since they significantly change the management of patients from medical to surgical treatment. In the current case, the interesting point was the presence of two rare mechanical complications of MI, including VSR and interventricular septal aneurysm, which were simultaneously identified in a diabetic patient with no obvious symptoms of the acute coronary syndrome (true silent MI) that mimic congenital VSD. The mechanical complication of ST-elevation myocardial infarction (STEMI) often have very fulminant and catastrophic manifestations in patients, and the existence of two concurrent mechanical complications of MI with smooth and gradual course is very scarce, which was erroneously misdiagnosed as congenital VSD. We believe the cause of this smooth disease course in the present case stems from the presence of an IVS aneurysm that prevented the rapid raise in

pulmonary blood flow, which resulted in a slow clinical course. In this case, VSR was originated from multiple perforations at the wall of IVS aneurysm.

In 2015, Davila et al. ¹ reported a case of non-STEMI who was afflicted with the concurrent left ventricular pseudoaneurysm and VSR.

Negi et al. in 2016 ⁷ described a 78-year-old woman with inferior STEMI who developed ruptured intraseptal pseudoaneurysm two weeks later in the acute phase of MI.

In a case report published by Zhang et al. in 2018 ², they reported a 65-year-old woman with a history of inferior STEMI two months ago without reperfusion therapy that presented with de-novo heart failure. The diagnostic workup revealed the concurrent true infroposterior LV aneurysm and VSR.

In 2020, Fisicaro et al. ⁸ suggested a novel entity of pseudoaneurysm of IVS and reported a 79-year-old woman with unknown history of coronary artery disease who was referred for symptoms of de-novo heart failure. In past medical history, she had a history of Bentall operation due to aortic dissection. They reported a post-ischemic IVS pseudoaneurysm with multiple perforations inside the RV cavity in the background of neglected anterior STEMI. Coronary angiography was performed and showed the total occlusion at the mid part of LAD.

The last case report was very similar to our patient, who had two rare mechanical complications in anterior STEMI, except our patient had the same complications in the background of inferior STEMI. Silent true MI and no reperfusion therapy due to the asymptomatic nature of MI in these patients made them susceptible to mechanical complications of MI.

Post-MI pseudoaneurysm leads to approximately 50% risk of rupture and resulting from the subacute rupture of LV free wall and rarely IVS that constitute the organized thromboses and

pericardium. However, in these cases, they comprised only a thin external layer of RV side of IVS. Although the gold standard method for distinguishing the true from pseudoaneurysm is at the time of surgery; however, the echocardiographic findings were in favor of pseudoaneurysm.

The absence of a continuous layer of the myocardium, an overhanging shelf like border of this structure wall, globular appearance, neck diameter to maximum cavity diameter of less than 50%, and paradoxical systolic pulsation were in favor of pseudoaneurysm that would dramatically change the management of patients from medical to surgical treatment since there is a significantly higher risk of rupture in pseudoaneurysm in comparison with a true aneurysm.

CONCLUSION

In any patient with ventricular septal defect (VSD), attention should be paid to the regional wall motion abnormality of left ventricle as the VSD may not be congenital and due to mechanical complications of silent myocardial infarction. We reported the concurrent of two rare complications of true silent inferior STEMI in the elderly poorly controlled diabetic patient with de-novo heart failure, mimicking congenital VSD. . The final medical plan for the patients was coronary artery bypass graft (CABG) surgery, aneurysmectomy, and VSR repair.

ETHICAL STATEMENT

Informed consent was obtained by patient for publication this case report.

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Figure legends

Figure 1: Electrocardiography of the patient

Figure2: The schematic view of the interventricular septal aneurysm and multiple perforations and ventricular septal rupture. VSR; ventricular septal rupture

Figure 3: Coronary angiography of the patient demonstrates that the right coronary artery is cut off in the mid part (arrowhead)

Figure 4: Left ventricular angiogram showing opacification of the LV (red arrow) followed by septal pseudoaneurysm (blue arrow) and then of the RV (green arrow)