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Images in Cardiology

CT imaging of post-myocardial infarction ventricular septal defect with a contained rupture/pseudoaneurysm

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ABSTRACT

This is a CT imaging study of a 63-year-old female who presented to our center with ST segment elevation MI and was found to have life threatening post-MI ventricular septal defect with associated pseudoaneurysm, which was detected on cardiac CTA. The patient refused surgical management and had a successful percutaneous VSD repair.

1. Case summary with imaging description

A 63-year-old female with past medical history of cerebral palsy and hyperlipidemia presented with 2 h of dyspnea and substernal chest pain. On arrival, the patient was found to be tachycardiac (HR-120 bpm), diaphoretic, and hypotensive (Blood pressure of 80/56 mm of Hg) with a grade 2/6 holosystolic murmur noted on physical examination. A 12 lead electrocardiogram showed ST-segment elevation in leads II, aVF, V5, and V6. The patient was given aspirin and clopidogrel and taken for urgent cardiac catheterization, which demonstrated complete occlusion of the proximal right coronary artery (RCA) and a ventricular septal defect (VSD), considered being infarct related. A drug eluting stent was successfully deployed in proximal RCA. A transthoracic echocardiogram was obtained later, confirming the presence of VSD in an aneurysmal basal and mid septum.

The patient was offered an emergent surgical repair of VSD and pseudoaneurysm, which she refused; therefore, a percutaneous VSD repair was planned. Cardiac CTA was performed prior to the procedure, which demonstrated a contained rupture/pseudoaneurysm at the basal inferior and inferoseptal segments with a 1.9 × 1.2 cm sac (Panel A). The rupture extended anteriorly into the basal interventricular septum, dissecting it approximately 5.5 cm, with subsequent communication to the mid-apical right ventricle (Panel B and Panel C). A percutaneous VSD repair was done via simultaneous right and left heart catheterization and a 28-mm

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Amplatzer Septal Occluder was successfully deployed with mild residual shunting (Panel D). The patient's post-operative course was complicated by refractory hypotension, bradycardia, and later by circulatory arrest which eventually led to her demise.

2. Comment

Ventricular septal defect and left ventricular free wall rupture are fatal mechanical complications of acute MI with high in-hospital mortality rates despite optimal medical and surgical treatment.[1] The incidence of Post-MI VSDs has declined from 1% to 3% in pre-thrombolytic era to 0.2%–0.34% currently because of improvements in reperfusion and myocardial salvage.[2] The risk factors for septal rupture include a female sex, age >65 years, anterior wall infarct, single-vessel disease, extensive MI, and poor septal collateral circulation.[2] Medical therapies are intended only for temporary stabilization before surgery, as the condition of most patients deteriorates rapidly. The key to the management of VSD is a prompt diagnosis with an aggressive approach to hemodynamic stabilization and immediate surgical repair (Fig. 1).

Left ventricular free wall rupture is another deadly complication of MI, and in most cases, patients expire before arrival to the hospital due to severe intra-pericardial bleeding which rapidly results in cardiac tamponade and death.[3] On rare occasions, the rupture is contained by pericardial and fibrous tissue, creating a left ventricular pseudoaneurysm. Characteristically, the orifice of the pseudoaneurysm is narrow, with a characteristic to and fro blood flow, from the left ventricle into the pseudoaneurysm during systole, and from the pseudoaneurysm into the left ventricle during diastole. The pseudoaneurysm wall is made of adherent pericardial or fibrous tissue, without any myocardial or

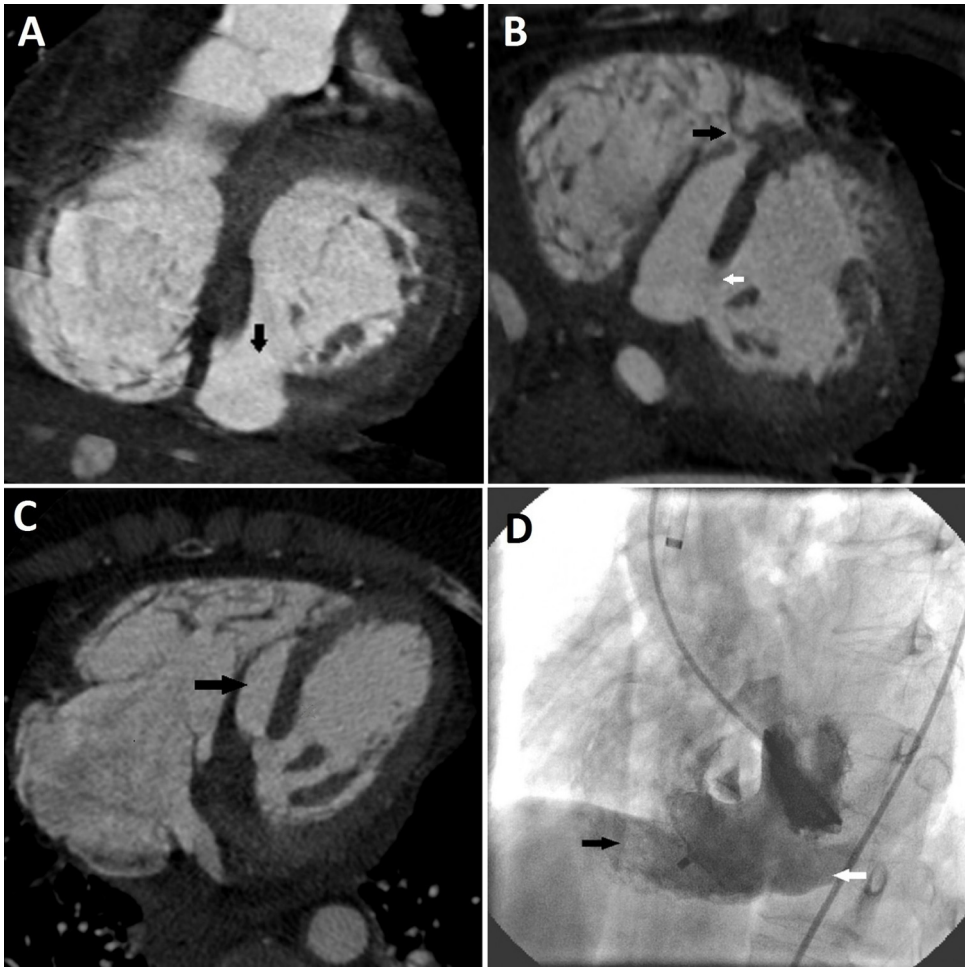

Fig. 1 – (Panel A) Cardiac CT short axis view demonstrating a rupture in the basal inferior segment of the interventricular septum communicating to a contrast-filled sac (arrow). (Panel B) Cardiac CTA in the short axis plane demonstrates the post-infarct myocardial rupture in the basal infero-septal segment of the interventricular septum (white arrow). The rupture dissects through the interventricular septum creating a VSD into the right ventricle (black arrow). (Panel C) Cardiac CTA in the axial plane demonstrates the large post-infarct VSD (black arrow). (Panel D) Fluoroscopic image after contrast administration into the left ventricle (ventriculogram) demonstrating the Amplatzer Septal Occluder within the VSD and persistent filling of a portion of the pseudoaneurysm (white arrow) with mild residual shunting into the right ventricle (black arrow).

endocardial layers. Thus, this wall is thin and may easily rupture and cause bleeding into the chest cavity and death. It demonstrated that approximately two-thirds of all left ventricular pseudoaneurysms occur after myocardial infarction.[4] Surgery is considered the treatment of choice; however, it is frequently unsuccessful. Suturing into the necrotic myocardium may fall apart. The quoted surgical mortality is 23%. Medical therapy has higher mortality (30–45%).[4] Transcatheter repair of pseudoaneurysm is now another treatment option.[5]

The patient described in this case had a very early onset of Post-MI VSD with a concomitant pseudoaneurysm. The patient refused surgical correction, which is the treatment of choice and was managed via primary percutaneous VSD repair, which has shown promising results[6] but was futile as the patient expired shortly after.

Conflicts of interest

The authors have none to declare.

REFERENCES

1. Kutty RS, Jones N, Moorjani N. Mechanical complications of acute myocardial infarction. *Cardiol Clin*. 2013;31:519–531.
2. Crenshaw BS, Granger CB, Birnbaum Y, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction, GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) Trial Investigators. *Circulation*. 2000;101:27–32.
3. Reddy SG, Roberts WC. Frequency of rupture of the left ventricular free wall or ventricular septum among necropsy cases of fatal acute myocardial infarction since the introduction of coronary care units. *Am J Cardiol*. 1989;63:906–911.
4. Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. *J Am Coll Cardiol*. 1998;32:557–561.
5. Dudiy Y, Jelnin V, Einhorn BN, Kronzon I, Cohen HA, Ruiz CE. Percutaneous closure of left ventricular pseudoaneurysm. *Circ Cardiovasc Interv*. 2011;4:322–326.
6. Thiele H, Kaulfersch C, Daehnert I, et al. Immediate primary transcatheter closure of postinfarction ventricular septal defect. *Eur Heart J*. 2009;30:81–88.