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CASE REPORT

Ventricular septal defect: early against late surgical repair

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Ventricular septal defect (VSD) is a rare complication of right ventricular infarction (RVI) which is associated with significant mortality, if not treated appropriately. It typically occurs within the first 10–14 days after myocardial infarction. Surgical repair has been shown to reduce in-hospital mortality from 90% to 33–45%. Early surgical VSD repair has also been associated with high 30-day operative mortality of 34–37%. Furthermore, after an acute MI the friable myocardium enhances the risk of recurrent VSD with early surgical repair. We present a case of a middle-aged woman who developed VSD after an RVI. Her surgical repair was delayed by 2 weeks due to development of *Staphylococcus aureus* bacteremia. During this period, she was managed medically and later on underwent percutaneous repair with an amplatzer VSD occluder device. Keeping this patient encounter in mind, we would like to emphasize on the limited recommendations available for early against late surgical repair of VSD.

Keywords: *right ventricular myocardial infarction*; *ventricular septal rupture*; *early against late repair*

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Right ventricular infarctions (RVIs) are seen in nearly 50% of acute inferior wall myocardial infarctions. The complications of RVI can be either electrical, which includes ventricular fibrillation during RV pacing, AV block or mechanical-like RV dysfunction, and shock (1). It is associated with significant mortality (91%) secondary to increased incidence of cardiogenic shock. The occurrence of ventricular septal rupture has declined over the recent times due to the advent of advanced percutaneous interventions (2).

We present a case of a 69-year-old woman who developed ventricular septal defect (VSD) with left-to-right shunt and hemodynamic instability after an RVI. Her VSD repair was delayed by 2 weeks due to *Staphylococcus aureus* bacteremia. We present this case to review benefits and risks of early VSD repair.

Case presentation

A 69-year-old Caucasian woman presented to outside hospital with complaints of chest pain and shortness of breath. Chest pain started approximately a week ago and lasted for about 5 days but then subsided for a day. Next day, the patient was experiencing continuous chest tightness both on right and left side. She was also having dyspnea on exertion. The patient had a past medical history of hypertension, hyperlipidemia, obesity, and severe degenerative joint disease. Past surgical history was significant for hysterectomy. She denied any history of smoking cigarettes or using illicit drugs. The patient had a history of occasional alcohol intake. Medications at home included amlodipine/benazepril 10/20 mg daily, raloxifene 60 mg daily, celecoxib 200 mg BID, clonazepam 1 mg daily, fenofibrate 145 mg daily, a multivitamin tablet daily, a calcium tablet 600 mg daily, glucosamine 1500 mg daily, tramadol/acetaminophen 37.5/325 mg BID, and hydrocodone/acetaminophen 10/300 mg as needed for pain.

On arrival to the emergency department, the patient was tachycardic with a heart rate of 114/min, tachypneic with a respiratory rate of 24/min. She was normotensive at that time. Within the next few minutes, the patient became significantly hypotensive. Vitals at that time were blood pressure 78/52, heart rate 109/min, respiratory rate 22/min, and saturation of 96% on 3 L of oxygen via nasal cannula. On physical examination, the patient appeared to be in acute distress. On cardiovascular assessment, S1 and S2 were audible with no added murmur or gallop.

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She had bilateral pitting lower extremity edema. On lung examination, the patient had bilateral rhonchi and increased respiratory effort.

EKG showed sinus tachycardia with q waves and ST segment elevations of almost 2 mm in leads III and aVF. There was also left atrial enlargement (Fig. 1). Radiograph of chest showed interstitial edema. Laboratory results from admission included a basal metabolic panel that showed blood urea nitrogen of 40 mg/dL (6–20 mg/dL), creatinine of 1.2 mg/dL (0.7–1.2 mg/dL), and potassium of 4.2 mEq/L (3.5–5.0 mEq/L). First set of troponin levels were found to be 1.60 ng/mL (0.0–0.3 ng/mL) and first set of CK was 118 U/L (29–200 U/L). A Doppler echocardiogram was done in the emergency room, which showed normal ejection fraction of 60–65% with RV hypokinesis, which was consistent with RVI.

The patient was started on vasopressors and emergently transferred to our facility to undergo cardiac catheterization. Left heart catheterization revealed 99% stenosis to mid-right coronary artery and subsequently the patient underwent percutaneous coronary intervention (PCI) to the right coronary artery. An intra-aortic balloon pump was placed during cardiac catheterization as the patient was in cardiogenic shock. After successful PCI, the intra-aortic balloon pump was removed as her vitals seemed to have stabilized. Two days later, again the patient developed hypotension and worsening shortness of breath. Urgent bedside Doppler echocardiogram showed a new left-to-right shunt in the ventricle, which was noted to be a muscular VSD. The patient was transferred to a tertiary care medical center for surgical repair. At the tertiary care hospital, transesophageal echocardiogram (TEE) was done, which revealed large VSD with a dimension of 1.38×0.9 cm^2 and an area of 0.9 cm^2 located in the posterior wall of ventricle (Fig. 2). TEE also showed mildly reduced RV systolic function and moderate to severe tricuspid regurgitation (Figs. 3 and 4).

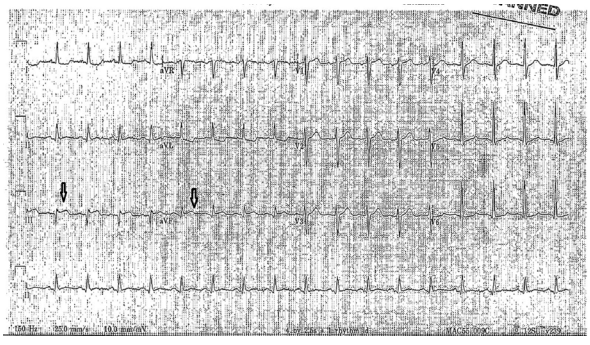

Fig. 1. EKG showing sinus tachycardia with q waves and ST segment elevations of almost 2 mm in leads III and aVF (arrow) along with ST segment depressions in I and aVL leads. This implies that there is possible inferior wall infarction. Left atrial enlargement also noted.

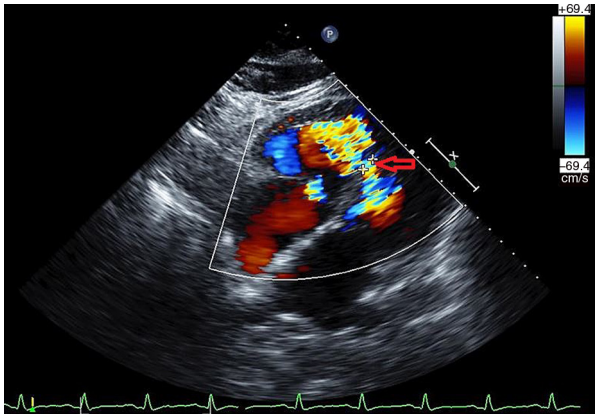

Fig. 2. Echocardiogram with color Doppler displaying a ventricular septal defect postmyocardial infarction (arrow).

The patient subsequently developed *S. aureus* bacteremia and *Clostridium difficile* colitis, which were successfully treated with intravenous antibiotics. She underwent cardiothoracic surgery for possible surgical versus percutaneous repair of the VSD. Based on the location of the VSD, the patient was considered to be an excellent candidate for percutaneous repair with amplatzer post-infarct VSD occluder device. After placement of the amplatzer VSD device, significant reduction in left-to-right shunt was noted on TEE. Also, hemodynamically the patient improved and her hypotension resolved, so the intra-aortic balloon pump was discontinued. Eventually, the patient developed sepsis secondary to *C. difficile* colitis, which resulted in development of disseminated intravascular coagulation, hemodynamic collapse, and death.

Discussion

Ventricular septal rupture (VSR) is a rare complication of RVI with decreasing incidence due to the advent of reperfusion therapy. Risk factors for septal rupture include hypertension, advanced age, female gender, smoking, history of angina or MI, extensive MI, RVI, and delayed presentation after onset of MI (3). The risk factors for

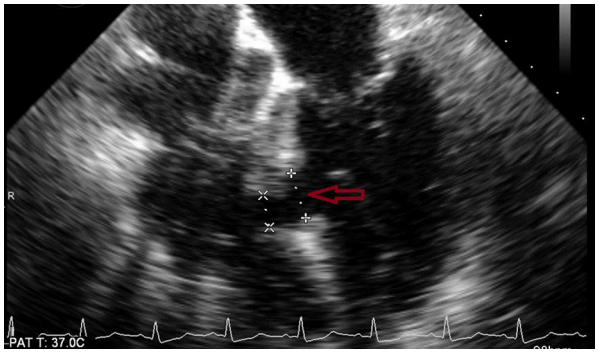

Fig. 3. Transesophageal echocardiogram showing ventricular septal defect (arrow) with size.

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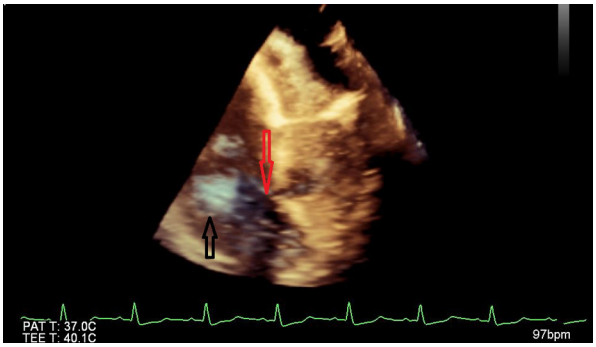

Fig. 4. 3D echocardiogram showing ventricular septal defect (red arrow) and necrosis (black arrow).

septal rupture in this patient were female gender, delayed presentation (as she was having chest pain since 7 days with normal CPK and q waves in inferior leads) and RVI.

The above case represents a middle-aged woman with a VSD whose surgical repair was delayed by 2 weeks due to development of *S. aureus* bacteremia. The management of this patient's VSD was challenging as current guidelines suggest that unstable patient's should be considered for emergent repair (4). In contrast to this, recent literature demonstrates that early surgical VSD repair has been associated with high 30-day operative mortality of 34–37% (5). Furthermore, after an acute MI the friable myocardium enhances the risk of recurrent VSD with early surgical repair (5). The development of RV dysfunction in patients with cardiogenic shock is not associated with favorable outcomes after early surgical repair as evidenced in recent studies (5). In conjunction with above-mentioned facts, we decided to stabilize the patient medically and opted for late repair to which the patient responded adequately.

Keeping this patient encounter in mind, we would like to emphasize on the limited recommendations available for early against late surgical repair of VSD. A discussion can be initiated whether patients with hemodynamically unstable VSD could be managed at first by medical therapy or intra-aortic balloon pump or is emergent surgical intervention necessary in such patients. Unfortunately, we do not have enough literature to corroborate the decision made in this patient's management. More studies are required to evaluate the benefits of early repair in such patients. In addition, location of VSD is an important determinant of percutaneous versus surgical repair. In our patient, posterior VSD made it anatomically difficult to place a surgical patch thus making percutaneous repair with amplatzer device more desirable.

After our extensive literature search, we came across only one case of RVI complicated by VSD. According to Sharif et al. RVIs have been reported to be associated with right-to-left shunt usually located at the atrial level (6). The novelty of our case lies in the development of left-to-right shunt at the level of muscular portion of interventricular septum. Sadaniantz et al. (7) noted that the VSD could complicate an RVI even if left ventricular function is preserved.

Authors' contributions

All authors reviewed and approved the final version of the manuscript. The manuscript has not been published and is not being considered for publication elsewhere in whole or in part in any language.

Conflict of interest and funding

The authors reported no conflict of interest and no funding was received for this work.

References

1. Manno BV, Bemis CE, Carver J, Mintz GS. Right ventricular infarction complicated by right to left shunt. J Am Coll Cardiol 1983; 1: 554–57.
2. Moore CA, Nygaard TW, Kaiser DL, Cooper AA, Gibson RS. Postinfarction ventricular septal rupture: the importance of location of infarction and right ventricular function in determining survival. Circulation 1986; 74: 45.
3. Crenshaw BS, Granger CB, Birnbaum Y, Pieper KS, Morris DC, Kleiman NS, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. Circulation 2000; 101: 27–32.
4. Coskun KO, Coskun ST, Popov AF, Hinz J, Schmitto JD, Bockhorst K, et al. Experiences with surgical treatment of ventricle septal defect as a post infarction complication. J Cardiothorac Surg 2009; 4: 3.
5. Pang PY, Sin YK, Lim CH, Tan TE, Lim SL, Chao VT, et al. Outcome and survival analysis of surgical repair of post-infarction ventricular septal rupture. J Cardiothorac Surg 2013; 8: 44.
6. Sharif H, Pezzella AT, Farah HH, Baljepally R, Maki D. Intracardiac right-to-left shunt complicating right ventricular failure. Asian Cardiovasc Thorac Ann 1999; 7: 287–91.
7. Sadaniantz A, Aitken P, Katz A. Isolated right ventricular infarction complicated by ventricular septal defect. Int J Cardiol. 1996; 53: 25–28.