Muscular ventricular septal defect after mitral and aortic valve replacement

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Summary

We describe a case series of five patients who were referred to our cardiac rehabilitation department after mitral or aortic valve replacement, and whose transthoracic echocardiographic studies showed postoperative muscular septal defects.

Introduction to case series

From September 2007 to December 2008, five patients aged 54–69 years were admitted to our institution for cardiac rehabilitation after surgical cardiac valve replacement (table 1). Successful replacement of the aortic valve was performed in four of the patients and mitral valve replacement in the other. All patients received mechanical prosthetic valves (ATS medical) and had a history of dyspnoea, fatigue and exercise limitation. Physical examination on admission did not reveal signs of congestion in any of the patients. The admission electrocardiogram showed sinus rhythm or atrial fibrillation (patient 4) and signs of left ventricular hypertrophy in 3 of 5 patients. Doppler echocardiography revealed a restrictive muscular ventricular septal defect (fig. 1, 3, 4, 5 and 6) with Qp/Qs <1.4 in all of the patients. The function of the prosthetic valves was normal and in one patient (patient 5) systolic pump function was moderately reduced.

Echocardiographic follow-up revealed spontaneous closure of the septal perforation in four of the five patients (fig. 2). In one patient the septal defect persisted for one and a half years after surgery, but he remained asymptomatic without deterioration of left ventricular function or increased pulmonary artery systolic pressure.

Discussion

Ventricular septal defect (VSD) may be of congenital origin, but also acquired as a result of myocardial infarction, penetrating trauma, blunt trauma, post-surgery or iatrogenic. VSD is the most common congenital cardiac abnormality in infants and children. It is estimated that 25–40% of such defects have closed spontaneously by the time the child is two years old;

Table 1
Patients' characteristics.

Age	Gender	Diagnosis	Valve prosthesis	Size of prosthesis	NYHA class at admission	VSD location	LVDD, mm	LAD, mm
54	Male	Combined aortic stenosis with regurgitation	ATS	23 mm	II	Mid-septal	46	40
59	Male	Severe aortic regurgitation	ATS	23 mm	II	Anteroseptal basal	50	46
59	Male	Severe mitral regurgitation due to chordal rupture	ATS	31 mm	I	Mid-basal septum	48	45
69	Female	Combined aortic stenosis with regurgitation	ATS	20 mm	II	Mid-septobasal	50	54
61	Male	Severe aortic stenosis	ATS	22 mm	II	Anteroseptal	55	60

LVDD = left ventricular diastolic diameter; LAD = left atrial diameter; VSD = ventricular septal defect.

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Figure 1
Patient 1, at admission. Four-chamber view: muscular VSD mid-septal.
LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle; VSD = ventricular septal defect.

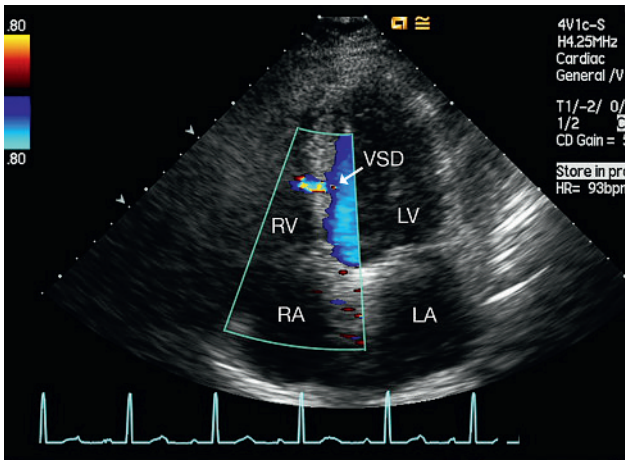


Figure 2
Patient 1, five months later. Four-chamber view: no evidence of residual VSD.
LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.

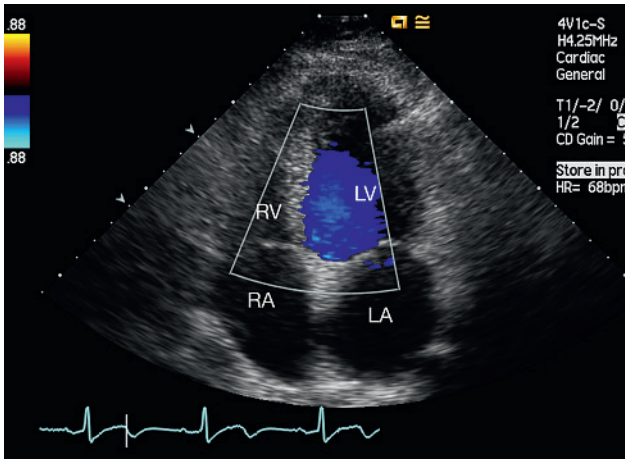


Figure 3
Patient 2, at admission. Parasternal long-axis view (LV outflow): muscular VSD anteroseptal basal.
AO = aorta; LA = left atrium; LV = left ventricle; RV = right ventricle; VSD = ventricular septal defect.

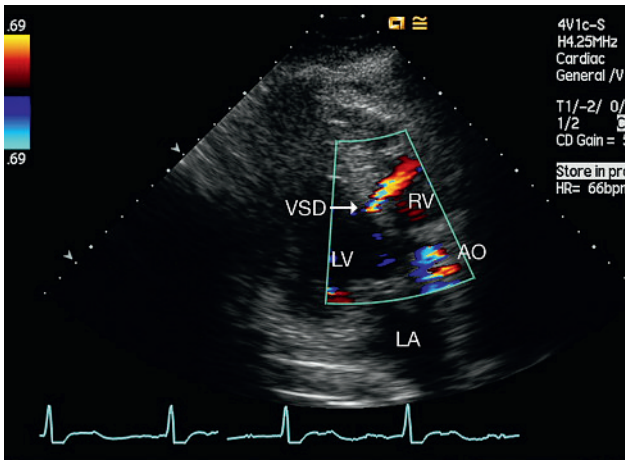


Figure 4
Patient 3, at admission. Four-chamber view: muscular VSD mid-septal.
LV = left ventricle; RA = right atrium; RV = right ventricle; TR = tricuspid regurgitation; VSD = ventricular septal defect.

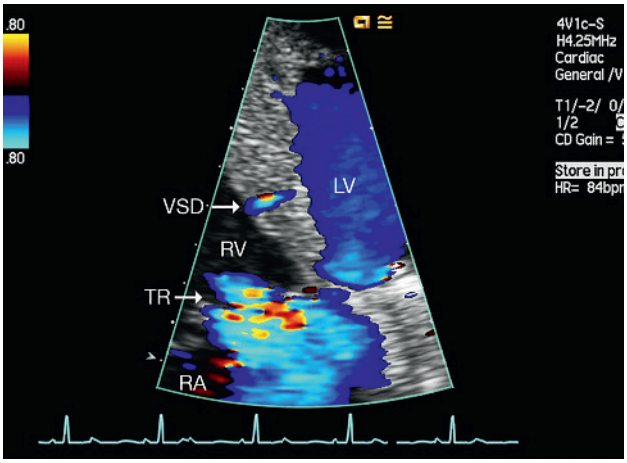


Figure 5
Patient 4, at admission. Four-chamber view: muscular VSD mid-septobasal.
LV = left ventricle; RA = right atrium; RV = right ventricle; TR = tricuspid regurgitation; VSD = ventricular septal defect.

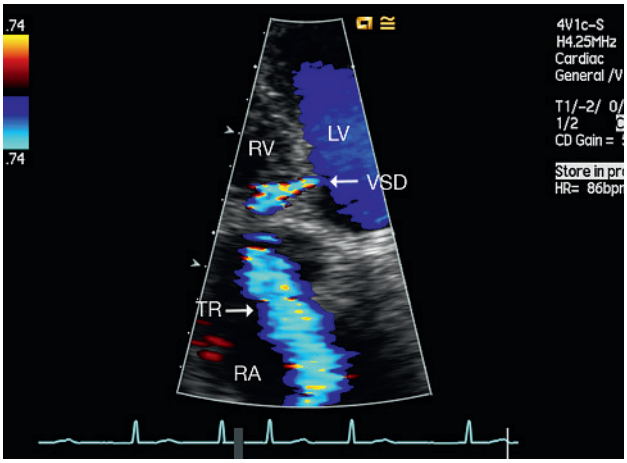


Figure 6
Patient 5, at admission. Parasternal long-axis view (LV outflow): muscular VSD mid-anteroseptal.
AO = aorta; LV = left ventricle; RV = right ventricle; VSD = ventricular septal defect.

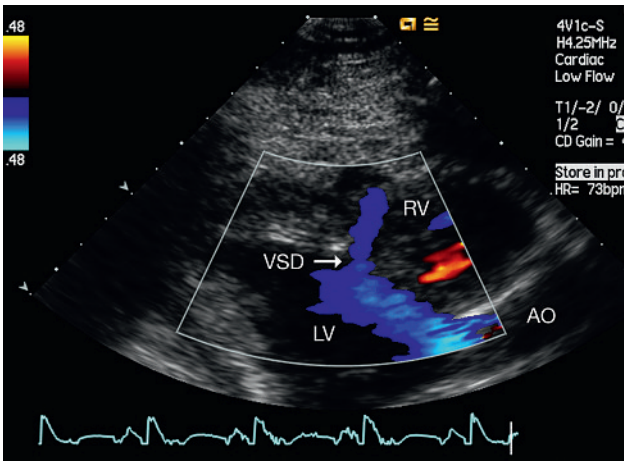


Figure 7
Long-axis view: venting cannula in position. Cannula dimensions: lenght 70 mm, diameter 3.5 mm.

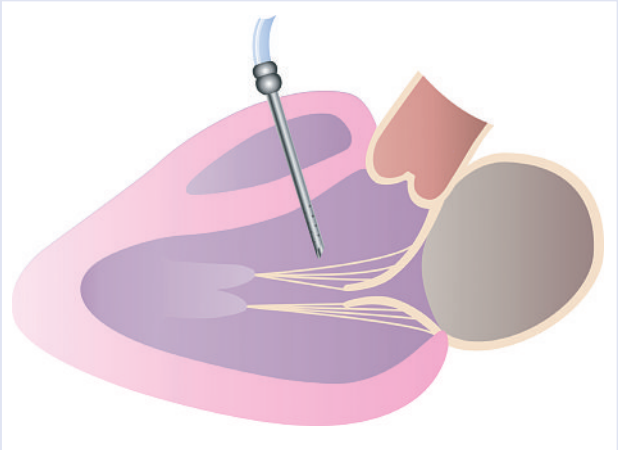
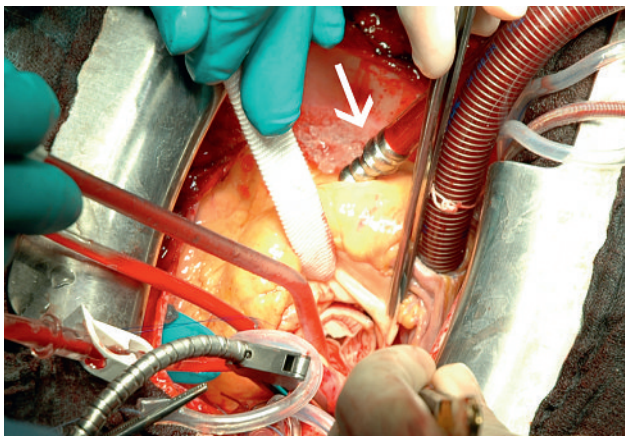


Figure 8
Venting cannula (arrow) in position during aortic valve replacement.



90% of those that eventually close do so by the time the child is ten years old [1–2].

Ventricular septal defect complicating acute myocardial infarction is uncommon, with a reported incidence of 0.2% of all myocardial infarctions. The outcome remains extremely poor, with mortality of 50% in patients undergoing surgical repair and 95% in those treated medically [3]. This is not unexpected, given the advanced age, severity of coronary artery disease, haemodynamic instability and technical challenges associated with the surgical procedure.

Posttraumatic VSD is infrequent, with clinical sequelae ranging from imminent death to complete spontaneous resolution. It is unclear what the most appropriate management strategy is. Careful observation has been advocated in the management of asymptomatic patients with small, restrictive shunts. Percutaneous or surgical VSD closure is required for large defects or symptomatic patients [4–6].

Septal perforation has also been described as a rare complication of some cardiac interventions, such as transvenous pacemaker-electrode implantation [7], and after percutaneous coronary interventions [8], hypertrophic obstructive cardiomyopathy treated by left ventricular myotomy and myectomy, or alcohol-induced septal ablation [9].

Development of an iatrogenic VSD following mechanical aortic or mitral valve replacement is a rare complication. Such VSDs are typically membranous in location and small in size, with little haemodynamic significance. These defects result not only from technical surgical complications but also from postoperative endocarditis [10–12].

In the case of our patients the muscular septal defects were probably secondary to left ventricular transseptal venting during surgical valve replacement (fig. 7 and 8). Left ventricular venting has been used since the early years of open-heart surgery with a heart-lung machine [13–14], and is frequently used to prevent air embolism and ventricular distension during systemic cooling [15].

There are several techniques for venting during open-heart surgery: cannulation of the left ventricle apex, the aortic root, the left atrium, the pulmonary artery or through direct puncture of the free wall of the right ventricle and ventricular septum by a metal cannula with multiple perforations near the tip (fig. 7). This method was used in all the patients of our series.

Complications after ventricular venting are uncommon, but e.g. bleeding and left ventricular [16] or aortic false aneurysms [17] have been documented. To the best of our knowledge left ventricular venting complicated by iatrogenic muscular septal defect has not so far been described as a complication after surgical valve replacement.

The natural history of iatrogenic VSD may be variable and include spontaneous closure, as in the case of four of our five patients. An observational follow-up should therefore be recommended for asymptomatic patients with small defects. All patients in the present report had valve replacement with mechanical valves (ATS) requiring antibiotic prophylaxis for endocarditis [18]. The ACC/AHA 2008 Guidelines for the Management of Adults with Congenital Heart Disease recommend surgical closure of a ventricular septal defect if there is a Qp/Qs (pulmonary-to-systemic blood flow ratio) of 2.0 or more and clinical evidence of LV volume overload (Class I Indication, Level of Evidence: B), and also if the patient has a history of infective endocarditis (Class I Indication, Level of Evidence: C). Percutaneous closure of VSD (Class IIb, Level of Evidence: C) offers an attractive alternative to surgical management in patients with increased surgical risk factors, multiple previous cardiac surgical interventions, poorly accessible muscular VSDs, or "Swiss cheese"-type VSDs. Indications for catheter device closure of VSDs include iatrogenic artefacts after surgical replacement of the aortic valve and residual defects after prior attempts at surgical closure or trauma [19].

Conclusion

In this report we describe a case series of five patients who developed muscular VSDs after left ventricular venting during the mitral or aortic valve replacement procedure. To our knowledge this rare complication has not been reported previously. In our series most of these small VSDs closed spontaneously, and therefore an observational approach may be appropriate. Percutaneous or surgical closure is indicated for symptomatic patients or patients with haemodynamically relevant defects. In addition we recommend the use of other venting routes to avoid such complications.

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