INTRODUCTION

Congenital heart disease (CHD) is one of the most common congenital abnormalities, affecting between 0.8 and 1.2 percent of all live births worldwide. The most common congenital cardiac anomaly in children and the second most common congenital abnormality in adults is a ventricular septal defect (VSD).1,2 Ventricular septal defect and atrial septal defect were the most common subtype of CHD with an incidence of 5.29/1000 and accounted for about 29.6% of all cases of CHD.3

CHD was common in developing countries in Africa and Asia but uncommon in most developed countries. Due to the various cardiac events that large VSDs cause, most large VSDs are surgically repaired early in infancy. As a result, discovering an asymptomatic large VSD in an elderly patient is quite rare.4,5 This study aims to show the surgical results in individuals who had VSD and CAD repaired simultaneously. Here, we report a surgical treatment experience in a 46-year-old patient who suffered from heart failure due to myocardial infarction with VSD and missed diagnosed as VSR. She underwent VSD patch repair concomitant with coronary artery bypass graft (CABG).

CASE PRESENTATION

A 46-year-old woman was referred to our hospital with a Ventricular septal rupture and a previous myocardial infarction. The patient had not experienced a heart murmur since he was a toddler, which had been identified as a Ventricular septal rupture and later as VSD. Until this episode, she had been working without experiencing any heart symptoms. He had been receiving uncontrolled medical therapy for type 2 diabetes mellitus for the last ten years. When she was referred to our hospital, her condition was the holosystolic murmur grade III/V, which was mainly audible in the left parasternal to apex lesion and was accompanied by a palpable thrill. Moist rales were heard in the bilateral lung field.

Chest X-ray examination found significant pulmonary congestion and reduced bilateral lung permeability due to pleural effusion (cardiothoracic rate of 60%) (Figure 1). The electrocardiogram result was Normal sinus rhythm, 100 bpm. Q5 pattern in V3, V4; Incomplete RBBB. Left to right shunt signal and interventricular septal defect were seen in Echocardiography and Continuous-Wave Doppler (Figure 2). The muscular septal part identified the 5 mm of IVS closure and the bilateral lung field.

Keywords: VSD, CAD, CABG, Adult Congenital Heart Disease.


Background: Ventricular septal defect (VSD) is the most common congenital cardiac anomaly in children and the second most common in adults. It often coexists with other cardiovascular conditions, including hypertension, coronary artery disease (CAD), and heart failure. This presents unique challenges in diagnosis and treatment.

Case Presentation: We present the case of a 46-year-old woman with a previously undiagnosed VSD complicated by a ventricular septal rupture and prior myocardial infarction. Coronary artery bypass grafting (CABG) and VSD repair were performed. Unexpectedly, the VSD turned out to be an atrial septal defect (ASD) of the muscular type. The repair was successful using mattress sutures and a 0.6mm PTFE patch. Two-coronary-artery bypass was also performed. The patient’s recovery was uneventful, with no residual VSD.

Conclusions: Discovering a large, asymptomatic VSD in an elderly patient is uncommon. Simultaneous repair of VSD and CAD yields favorable surgical outcomes, highlighting the importance of thorough preoperative evaluation in such cases.
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Figure 1. Significant pulmonary congestion and reduced bilateral lung permeability due to pleural effusion were seen (cardiothoracic rate of 60%).

Figure 2. Left to right shunt signal and interventricular septal defect were seen.

Figure 3. Aneurysmatic scar in the anterior-posterior left ventricle due to myocardial infarction.

Figure 4. Multiple muscular VSD with an orifice diameter of 15 mm. The VSD was closed using a PTFE patch.

coronary artery (RCA) and 99% stenosis in mid left anterior descending artery (LAD), total occlusion in distal LAD, 50% in the left circumflex artery. So, the final preoperative diagnoses were as follows: (1) old myocardial infarction (anteroseptal) due to three-vessel disease; (2) Ventricular septal rupture; (3) mild MR; (4) mild TR; (5) type 2 diabetes mellitus. We considered that VSR should be repaired simultaneously with coronary disease. Thus, after medical control for congestive heart failure, we performed, as an urgent operation, coronary artery bypass grafting (CABG) and VSR repair.

The saphenous vein graft (SVG) and the left internal mammary artery (LIMA) were harvested. In the median sternotomy approach, the pericardium was opened, and we found that the MPA size is bigger than the Aorta, Persistent Left Superior Vena Cava (PLSVC) and coronary sinus is bigger than normal size. As is standard practice, a cardiopulmonary bypass was established with superior and inferior vena cava drainage and ascending aorta perfusion. We decided to cannulate the PLSVC because there was a small innominate vein and Persistent Left Superior Vena Cava. Due to myocardial infarction, there was an aneurysmatism scar in the anterior-posterior left ventricle.

We performed left ventriculotomy after ascending aorta cross-clamp and antegrade cardioplegia administration (Figure 3). Instead of numerous VSRs, we discovered no VSRs but interventricular defects with thick margins (not a rupture-like defect but muscular VSD with two defects close to each other). The VSD was repaired with mattress sutures and a PTFE patch (round shape, thickness 0.6mm) and the ventriculotomy was closed with teflon.

We performed anastomosis for a two-coronary-artery bypass: SVG to RPDA and LIMA to LAD (Figure 4). Following declamping of the ascending aorta, proximal SVG anastomosis was performed. The patient was weaned from cardiopulmonary bypass uneventfully. The
aortic cross-clamp time is 150 minutes; the cardiopulmonary bypass time is 187 minutes. The patient's postoperative course was uneventful. No residual VSD.

DISCUSSION

The occurrence of myocardial infarction (MI) ventricular septal rupture (MIVSR) is a fatal complication that can be avoided by prompt referral, echocardiography examination, cardiac catheterization, and immediate operation. The use of inotropes and mechanical assistance before surgery has been demonstrated to improve the outcome in these individuals. In this patient, we decided to use IABP and inotropes to support hemodynamic flow to coronary vessels due to old myocardial infarction.

According to expert opinion and professional guidelines, early closure of myocardial infarction (MI) ventricular septal rupture (MIVSR) should be considered to reduce the duration of poor systemic perfusion caused by left-to-right shunting, pulmonary overcirculation, and systemic hypoperfusion, which can lead to resistant multiple organ failure and death. In this case we decided to urgent repair of MIVSR because the patient is in an acute heart failure state.

The exact time of MIVSR closure and perioperative treatment is still up for debate. Most percutaneous device closures were performed in prior research during the subacute and chronic (2-week) periods after the initial identification of MIVSR. The death rate related to delayed closure (two weeks after MIVSR detection) has been reported to be 6.1–10.0%. In comparison, the mortality rate linked with early closure (two weeks after MIVSR incidence) has been reported to be as high as 66 percent. The differential diagnoses of MIVSR were Acute mitral regurgitation due to papillary muscle rupture, Free wall rupture, Tricuspid regurgitation, Congenital ventricular septal defect, Atrial septal defect, and Acute flash pulmonary edema. In this case, during operation, we found no MIVSRs instead of multiple muscular VSDs with the diameter of the orifice was 15mm. There were no significant differences in signs and symptoms between MIVSR and VSD with myocardial infarction.

Over the last three decades, significant advancements in heart surgery, intensive care, and noninvasive diagnosis have resulted in a massive increase in the number of adults with CHD, and it was quite common in developing countries in Africa and Asia. Ventricular septal defect (VSD) is a common congenital heart defect in children, but it is less common in adults due to spontaneous and surgical closure. Due to the late onset of symptoms in some people with uncomplicated VSD, the diagnosis is sometimes postponed until adulthood.

In this particular case, congestive heart failure due to coronary disease triggered an opportunity to clarify MR, TR, and a large defect in the ventricular wall. We diagnosed it as a myocardial infarction (MI) ventricular septal rupture (MIVSR) because there was no history of VSD, and the incidence of ACHD (VSD) with CAD rarely occurred.

Patients with VSD can acquire other cardiovascular diseases such as hypertension, atherosclerotic coronary artery disease, vascular disease, stroke, and heart failure. Because intracardiac anomalies and CAD coexist, these people have unique clinical characteristics when it comes to diagnosis, treatment, and recovery. Only a few occurrences of surgery in CHD and CAD patients have been documented. As the lifespan of people with ACHD extends, the impact of acquired heart disease grows. For late-surviving people with a cyanotic CHD, myocardial infarction is one of the primary causes of death.

Generally, the surgical indications for VSD in adults are large defects with pulmonary to systemic output Qp/Qs > 1.5/1, pulmonary hypertension >50mmHg, PA systolic pressure is less than 50% systemic and pulmonary vascular resistance is less than one-third systemic, progressive dilatation of the left atrial or the LV, reduced LV function, aortic regurgitation with perimembranous VSD, and a history of endocarditis, especially recurrent.

The surgical indication for elderly VSD patients, on the other hand, is less apparent. Unless significant heart failure is identified, a conservative follow-up is often recommended. However, it is important to note that as systemic vascular resistance increases with age, the degree of shunt might rise, making VSDs more hemodynamically significant later in life.

The interventricular septum, which separates the two chambers of the heart, is a curved structure that is not symmetrical due to differences in pressure between the chambers. It comprises five parts: the membranous, muscular (also called trabecular), infundibular, atrioventricular, and the inlet. If one of these components fails to develop or fuse properly during the heart’s early formation, it can result in a VSD (ventricular septal defect) specific to that component. Different locations and variations in tissue types of VSDs have led to various classification systems. However, a new unified classification has simplified and categorized VSDs into four major groups:

1. Type 1 (infundibular outlet): This VSD is below the aortic and pulmonary valves in the right ventricle’s outlet septum. It’s relatively rare, accounting for only 6% of VSDs, except in the Asian population, where it makes up about 30%. This type often leads to issues with the aortic valve, including prolapse and regurgitation, and typically does not close independently.
2. Type 2 (membranous): This is the most common type, making up 80% of all VSDs. It’s located in the membranous septum below a specific structure. It may also involve the muscular septum, which is known as perimembranous. Sometimes, the tricuspid valve forms a pouch that can reduce the shunt and lead to spontaneous closure.
3. Type 3 (inlet or atrioventricular canal): This VSD is positioned within the right ventricular septum just below the inlet valves. It’s relatively uncommon, accounting for only 8% of VSDs, and is often seen in patients with Down syndrome.
4. Type 4 (muscular, trabecular): This VSD is located in the muscular septum and is bordered by muscle, typically in certain areas of the interventricular septum. They can be multiple and have a Swiss cheese-like appearance. They represent up to 20% of VSDs in infants but are less common in adults due to a tendency to close on their own.
The primary problem with VSDs is the creation of a shunt (passageway) between the right and left ventricles. The significance of the VSD is determined by factors like its size, location, and resistance in the pulmonary blood vessels. Aside from location, VSDs can also be classified by size. They’re considered small if they’re 25% or less of the diameter of the aortic annulus, medium if they’re more than 25% but less than 75%, and large if they’re greater than 75% of the aortic annulus diameter.14-16

In cases where there is a long-standing large left-to-right shunt, the blood vessels in the lungs undergo irreversible changes, leading to a condition known as persistent pulmonary arterial hypertension (PAH). When the pressure in the lung circulation surpasses that in the body’s circulation, the shunt direction reverses, resulting in a right-to-left shunt. This is called Eisenmenger syndrome and occurs in 10% to 15% of patients with VSD.14,17

Approximately 37% of congenital heart conditions in children are attributed to isolated VSD. Among newborns, the occurrence of isolated VSD is around 0.3%. However, this rate is notably lower in adults due to the fact that up to 90% of these defects may naturally close over time. Gender does not appear to influence the likelihood of VSD. The distribution of each type is detailed in the section discussing the underlying mechanisms of the condition.15,18-19

VSD is the most frequently occurring congenital abnormality present from birth. While small defects typically close on their own within the first year of life, larger ones can lead to significant complications. The primary interventions for substantial defects involve either surgical closure or the use of a medical device.20-22

The special aspect of this case is that two different heart problems are treated simultaneously. A Ventricular Septal Defect (VSD) is a hole in the heart’s lower chamber wall. Coronary Artery Bypass Grafting (CABG) is a surgery to improve blood flow around blocked heart arteries. When a patient has both a VSD and needs CABG, it’s a complex situation. The innovation here is the ability to address both issues in one surgery, which requires careful planning and expertise. This advancement in cardiac surgery has the potential to improve overall patient outcomes.

Patients with VSD can acquire other cardiovascular diseases such as hypertension, atherosclerotic coronary artery disease, vascular disease, stroke, and HF. Discovering an asymptomatic large VSD in an elderly patient is quite rare. Surgical results in individuals who had VSD and CAD repaired at the same time have a good result. Because intracardiac anomalies and CAD coexist, these people have unique clinical characteristics when it comes to diagnosis, treatment, and recovery. Only a few occurrences of surgery in CHD and CAD patients have been documented.18-20

CONCLUSION

We can conclude that the patient, in this case, recovered well after surgery, with no residual VSD observed. The procedure successfully addressed the multiple cardiac issues, representing a positive patient health outcome. This paper still has limitations, as this case is rare, and there is still limited literature discussing similar matters. It is hoped that future researchers can increase the number of reported cases similar to this one, thus expanding the existing analysis.

CONFLICTS OF INTEREST

The authors certify no conflict of interest with any financial organization regarding the material discussed in the manuscript.

ETHICS CONSIDERATIONS

This review of published literature did not require an ethical review.

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AUTHOR’S CONTRIBUTION

Firman Al Faruq was primarily responsible for writing the manuscript and co-coordinating the study design, data analysis, data interpretation, and data collection. Heroe Soebroto contributed to data analysis, data interpretation, critical revision of the article for intellectual purposes and final approval. Oky Revianto Sediono Pribadi contributed to the provision of the article’s material, expertise and writing of the article for important intellectual content. All authors have reviewed and approved the final version of the manuscript for submission.

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REFERENCES

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