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Surgical Treatment of Ventricular Septal Defect Following Myocardial Infarction: A Case Report

Eglė Ereminienė[1], Rūta Jurgaitienė[1], Rimantas Benetis[2], Giedrė Bakšytė[1], Giedrė Stanaitienė[1]

[1]Department of Cardiology, Medical Academy, Lithuanian University of Health Sciences, [2]Department of Cardiac, Thoracic, and Vascular Surgery, Medical Academy, Lithuanian University of Health Sciences, Lithuania

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Summary. *Ventricular septal defect after myocardial infarction is a rare but often life-threatening mechanical complication. The keys of management are a prompt diagnosis of ventricular septal defect and an aggressive approach to stabilize patient's hemodynamics. Invasive monitoring, judicious use of inotropes and vasodilators, and an intra-aortic balloon pump are recommended for the optimal support of patient's hemodynamics. The best results are achieved if optimally medically managed patients survive at least 4 weeks before elective surgery necessary for scar formation in a friable infarcted tissue.*

We report a case of acute myocardial infarction complicated by the rupture of ventricular septum. Instead of attempting an immediate surgical closure of ventricular septal defect, the postponed surgery was successfully performed 3 weeks after the occurrence of ventricular septal defect. Preoperatively, clinical and hemodynamic conditions of the patient were maintained stable with the support of an intra-aortic balloon pump and inotropes.

Introduction

In 1847, Latham was first to describe ventricular septal defect (VSD) at autopsy, but he did not associate its etiology with coronary artery disease. Brunn made the first antemortem diagnosis of acquired VSD in 1923, and in 1934, Sager established the clinical association between myocardial infarction (MI) and VSD. Surgical VSD treatment became available in 1957.

Ventricular septal defect is a life-threatening mechanical complication of acute MI. Despite optimal medical and surgical treatment, patients with VSDs have a high in-hospital mortality rate. During the prethrombolytic era, VSDs occurred in 1%–3% of individuals with MI (1). The incidence of VSDs declined with thrombolytic therapy to 0.2%–0.34% because of improvements in reperfusion and myocardial salvage (2, 3). Pathological and clinical studies have indicated that the mean time from MI to VSD is 3 to 5 days, but if the patient receives thrombolytic therapy, VSD may occur within the first 24 hours after MI (3, 4).

VSD occurs in a necrotic zone of the infarcted myocardial tissue and results in a left-to-right shunt. The degree of shunting is determined by the size of the septal rupture, the level of pulmonary and systemic vascular resistance, and the function of left and right ventricles. As left ventricular systolic function deteriorates and forward flow declines, compensatory vasoconstriction leads to increasing systemic vascular resistance, which, in turn, increases the magnitude of the left-to-right shunt. The size of the VSD can also abruptly expand because of exposition to shear forces and processes of necrotic tissue removal by macrophages, resulting in a sudden hemodynamic collapse even in patients who appear to be clinically stable with the normal LV function (5). Superimposed ischemic mitral valve regurgitation or ventricular aneurysm may be present, which further compromises heart function. As the left ventricle fails and the systolic pressure declines, left-to-right shunting decreases and the fraction of the shunt diminishes. The depressed left ventricular function commonly leads to impaired peripheral organ perfusion and death in most patients.

In about 60% of VSD cases, rupture occurs in the anteroapical septum secondary to the occlusion of the left anterior descending artery, and in 40%, posterior septal rupture occurs due to the occlusion of a dominant right coronary artery or a dominant left circumflex artery. Posterior VSD is often accompanied by mitral valve insufficiency secondary to posterior papillary muscle infarction.

It is important to quickly distinguish VSD from clinical symptoms, such as recurrent chest pain (50% of cases), shortness of breath, hypotension, and symptoms of biventricular failure. The main auscultatory feature is a loud holosystolic murmur

Correspondence to R. Jurgaitienė, Department of Cardiology, Medical Academy, Lithuanian University of Health Sciences, Eivenių 2, 50028 Kaunas, Lithuania
E-mail: ruta.jurgaitiene@lsmuni.lt

audible over a large area, including the left sternal border and the apical area. The patient's clinical course undergoes a sudden deterioration within hours or days, which rapidly leads to the worsening of the hemodynamic state with the development of congestive heart failure and, often, cardiogenic shock. Mortality in patients with cardiogenic shock is particularly high, and according to the SHOCK Trial Registry, it is 87% (6, 7).

The risk factors for septal rupture include advanced age (>65 years), female sex, single-vessel disease, anterior infarct location, extensive MI, no current smoking, and poor septal collateral circulation (2, 8, 9).

The differential diagnosis of postinfarction cardiogenic shock should exclude free ventricular wall rupture, mitral insufficiency secondary to papillary muscle rupture, or left ventricular dilatation.

Echocardiography is the mainstay for the diagnosis and evaluation of VSD; its sensitivity and specificity have been reported to be as high as 100%. Echocardiography reveals the location and size of the ventricular septal defect by color Doppler, shows left and right ventricular functions, and estimates the right ventricular systolic pressure; the left-to-right shunt is quantified by the pulsed Doppler technique. Repeated echocardiographies should be done to assess the mechanical heart function and the progress of VSD.

Medical therapy is intended only for temporary stabilization before surgery, as the condition of most patients deteriorates rapidly, and they die before the surgical intervention. The key to the management of VSD is a prompt diagnosis and an aggressive approach to hemodynamic stabilization, angiography, and surgery. The optimal approach includes hemodynamic stabilization with the administration of oxygen and mechanical support. Drug therapy is aimed to reduce afterload on the heart and to increase forward cardiac output with the help of vasodilators. Intravenous nitroglycerin can be used as a vasodilator and may provide an improved myocardial blood flow in patients with a significant ischemic cardiac disease. The profound level of cardiogenic shock in some patients precludes vasodilator treatment, often necessitating vasopressor support. Inotropic agents used alone may increase cardiac output and organ perfusion, but do not change the ratio of pulmonary to systemic flow (Qp-to-Qs); moreover, due to a marked increase of left ventricular work, myocardial oxygen consumption increases undesirably. Intraaortic balloon counterpulsation (IABCP) offers the most important means of temporary hemodynamic support. IABCP reduces left ventricular afterload, thus increasing systemic cardiac output and decreasing the Qp-to-Qs ratio. IABCP also facilitates diastolic augmentation with an increase in the coronary blood flow, resulting in improved oxygen supply. In patients with cardiogenic shock, IABCP should be followed by immediate surgery. Patients with VSDs die because of end-organ failure due to cardiac failure. The high risk of mortality can be reduced only by shortening the duration of shock.

Patients with VSDs who underwent surgical repair in the GUSTO-I trial showed lower 30-day and 1-year mortality rates than patients who were treated medically (47% vs. 94% and 53% vs. 97%, respectively) (3). Lemery et al. reported a 30-day survival rate of 24% in patients treated medically compared with 47% in those treated surgically (10). The operative mortality rate is directly related to the interval between infarction and surgical repair: if repair is performed 3 weeks or more after an infarction, the rate is approximately 20%; if surgery is performed before this time; the mortality rate approaches 50% (11). Achieving hemodynamic stability before surgery is very beneficial, because 2–3 weeks or more after perforation allow the VSD edges to become firmer and fibrotic resulting in VSD repair that is more secure and easily accomplished. The criteria for a surgery delay include adequate cardiac output, no evidence of cardiogenic shock, an absence of signs and symptoms of congestive heart failure or minimal use of vasopressor agents to control initial symptoms, an absence of fluid retention, and good renal function.

Case Report

A 70-year-old woman with a history of recurrent chest pain manifesting after 1 week following right coronary artery angioplasty and stent implantation was admitted to our hospital. On admission, the patient's electrocardiogram showed anterior myocardial infarction: ST-segment elevation and Q waves in the anterior precordial leads. A coronary angiogram revealed a total proximal occlusion of the anterior interventricular branch of the left coronary artery. Angioplasty and stent implantation were performed, but the restored flow of anterior interventricular branch of the left coronary artery was of TIMI (thrombolysis in myocardial infarction) grade 1. Chest pain, shortness of breath, and general malaise disappeared after the procedure. Three days after angioplasty and stenting, the patient suddenly experienced fatigue, dyspnea, and tachycardia, which occurred after physical exertion. Auscultation revealed a band-shaped systolic murmur in the left parasternal area. Transthoracic 2D echocardiography confirmed a 7-mm VSD in the anterior-apical ventricular septum (Fig. 1), subsequently causing pulmonary artery hypertension with a gradient of 60 mm Hg and relative tricuspid valve insufficiency (grade 3) (Table). A left ventricular apical aneurysm and decreased left ventricular ejection fraction (from 35% to 20%) were documented (Table,

Table. Preoperative and Postoperative Echocardiographic Parameters

	3 Weeks Before Operation	2 Weeks Before Operation	1 Week Before Operation	1 Week After Operation	4 Months After Operation
LVEDD, mm	54	54	50	56	57
MVR	I–II	I–II	II	II	II
EF, %	25	20	20	20	20
RVEDD, mm	35	45	45	30	30
TR	III	IV	IV	II–III	II–III
PAP sist., mm Hg	60	70	70	60	46
Defect size, mm	7	13	20	No defect	No defect

EF, ejection fraction; LVEDD, left ventricular end diastolic diameter; MVR, mitral valve regurgitation;
PAP sist., systolic pulmonary artery pressure; RVEDD, right ventricular end diastolic diameter; TR, tricuspid regurgitation.

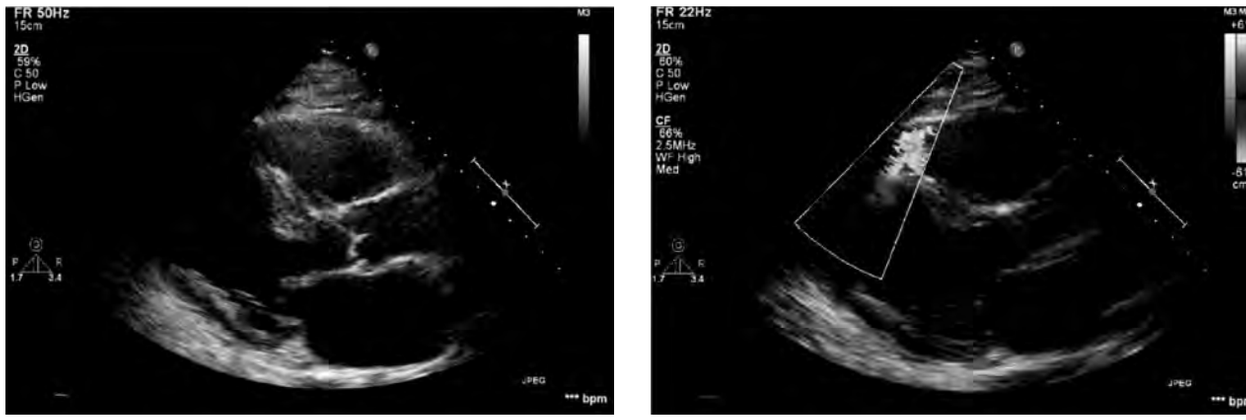

Fig. 1. Ventricular septal defect and shunt from the left to the right (a parasternal long-axis view)

Fig. 2). The symptoms of congestive heart failure progressed due to low cardiac output. The patient was transferred to the coronary care unit to treat heart failure and prevent the progression of cardiogenic shock. Despite medical treatment, the size of the VSD, pulmonary hypertension, and pulmonary congestion were gradually increasing (Table). On day 7 following the VSD appearance because of an increasing dose of dopamine, an intra-aortic balloon pump (IABP) was inserted with the aim to reduce systemic vascular resistance and left ventricular afterload, decrease the magnitude of the left-to-right shunt, enrich coronary circulation, and improve the diastolic function. As the IABP improved the diastolic arterial blood pressure, dopamine was temporarily discontinued. The patient's condition was stabilized, and the surgical VSD repair could be postponed. On day 12 after the IABP was inserted, the platelet count decreased to 67×10^9/L, and the IABP was discontinued to avoid a further decrease in the platelet count. Further medical management of the patient's hemodynamics without the IABP failed, i.e., the blood pressure dropped, the pulmonary venous pressure increased, and the clinical signs of pulmonary edema appeared. Therefore, urgent surgery was performed using cardiopulmonary bypass under moderate hypothermia. The VSD was identified (Fig. 3) and closed directly via left parFR 50Hz
15cm
0:00:00
M3
2D
58%
C 50
P Low
HGen
***bpm

Fig. 2. Ventricular septal defect and left ventricular apical aneurysm (a parasternal long-axis view)

aseptal ventriculotomy. A pericardial patch was tailored matching the shape of left ventricular infarction and sutured to the noninfarcted endocardium of the interventricular septum and the anterolateral ventricular wall using a double-layer, continuous 3/0 Prolene suture in order to exclude the left ventricular cavity from the infarcted myocardium (Fig. 4). Left ventricular aneurysmectomy along with tricuspid valve annuloplasty and single bypass using saphenous vein grafting to the right coronary artery were performed. The postoperative period

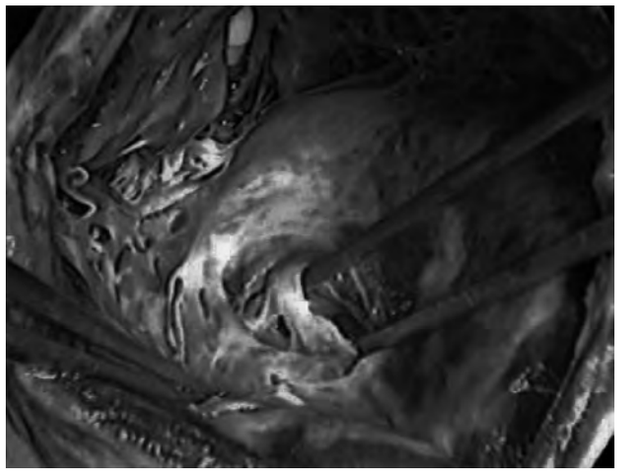

Fig. 3. Postinfarction ventricular septal defect: a surgical view

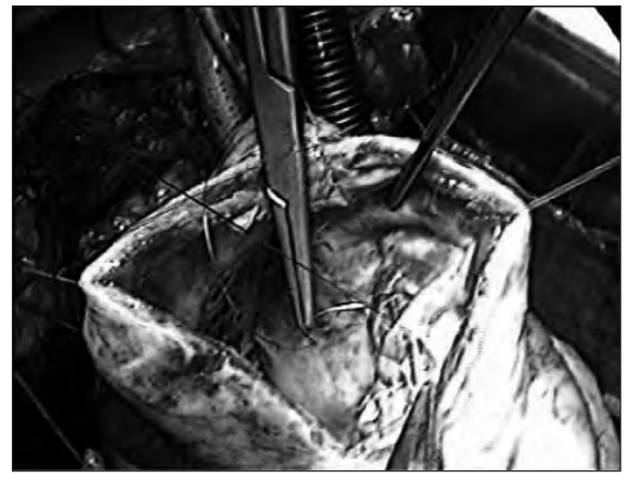

Fig. 4. Closure of ventricular septal defect with a pericardial patch: a surgical view

was complicated with prolonged ventilation due to respiratory distress syndrome. The postoperative medical treatment included intravenous diuretics and a continuous infusion of intravenous heparin, which was later replaced by aspirin. An angiotensin-converting enzyme inhibitor and small doses of a beta-blocker were prescribed to diminish LV wall stress and decrease LV afterload. An echocardiographic examination confirmed no residual shunt, reversal remodeling of the right ventricle, and reduction of the pulmonary artery systolic pressure down to 46 mm Hg (Table). The patient's condition remained stable for 4 months after operation with the functional improvement from NYHA class IV to II-III.

Discussion

Despite progression in medical and surgical management, the early mortality rate of operated patients still is 24%–33% (12, 13). At present, the strongest early mortality predictors are deterioration in the patient's condition before surgery and cardiogenic shock on admission. Other important factors associated with 30-day mortality are time from MI to VSD diagnosis and time from VSD diagnosis to surgical or percutaneous treatment (13).

Attempting to reduce a high early mortality rate associated with VSD closure surgery, surgeons have developed new operative techniques. A decreased incidence of the residual VSD was achieved using the infarct exclusion technique and the suturing of a pericardial patch to the healthy endocardial tissue, covering the infarcted muscle of the ventricular septum and the ventricular wall. Coronary artery bypass grafting performed simultaneously to all the stenotic coronary arteries, supplying the non-infarcted area, improves the collateral flow to the myocardium, contributes to its better recovery, and significantly improves survival (14–16).

Despite the operation technique, an important question, namely, the timing of surgery, remains controversial because medical treatment alone is generally inadequate to save patients and is associated with an extremely high mortality rate of 94% to 100% (12). However, early surgical repair is difficult and complicated because of the friable infarcted and necrotic tissue. Early mortality outcomes may be significantly better if patients with VSDs survive for at least 4 weeks before operation, which may be necessary for the significant scar formation of the margins of VSD. In the study by Coskun et al., the mortality rate after urgent VSD surgery within 3 days due to intractable cardiogenic shock was 100%, while all the patients who underwent surgical repair later than after 36 days survived (17). The criteria for delayed surgery include the maintenance of adequate cardiac output, no evidence of cardiogenic shock, the absence of congestive heart failure and severe pulmonary hypertension accomponied with concomitant tricuspid insufficiency, which predict higher morbidity and mortality rates. However, still only approximately 10%–15% of patients can be treated conservatively for a period of 2–4 weeks, after which surgery can be performed with a lower risk. Because all ventricular septal perforations are exposed to shear forces and processes of necrotic tissue removal by macrophages, the rupture site can expand abruptly. This may cause a sudden hemodynamic collapse, and even in patients who appeared to be clinically stable and with the normal left ventricular function, early death may occur before surgery. It is important to remember that sometimes a delay in VSD surgical repair may result in the deepening of cardiogenic shock, multiple organ failure, and early death. Since emergency surgery carries high mortality, it is the last resort for a large postin-

farction VSD with cardiogenic shock because the persistence of class IV cardiogenic shock in VSD, if not operated, is associated with a 100% mortality rate.

Another important question is how to manage patients with VSDs and keep a stable condition while waiting for elective surgery. The goals of immediate management include the reduction of systemic vascular resistance with the aim of left-to-right shunt reduction, maintaining cardiac output and the mean arterial blood pressure to ensure adequate end-organ perfusion, and maintaining coronary artery blood flow (18). The independent predictors of long-term mortality – tricuspid regurgitation causing increased pulmonary hypertension, heart failure, and increased heart rate – may be managed medically. Medical management has an important role in stabilizing the patient's condition and improving the cardiac function preoperatively and survival after a surgical VSD operation. Despite its benefits, pharmacological therapy alone may not be adequate to maintain systemic perfusion.

Mechanical circulatory support is frequently necessary as a temporary measure prior to surgical VSD repair. Although no definite data exist that an IABP improves survival, its use is widely accepted. The preoperative IABP was used from 40% to 95% of cases in different studies as a favorable support in the treatment of VSD (12, 17). The IABP decreases left ventricular afterload; thus, by increasing systemic cardiac output and decreasing the Qp-to-Qs ratio, it reduces the magnitude of the left-to-right shunt, facilitates diastolic augmentation, and increases the coronary and renal blood flow. Improved oxygen supply by the IABP and temporary hemodynamic support remains one of the most effective methods, providing circulatory support while waiting for optimal time for surgery. Preoperative patients successfully managed with the IABP are not subjected to cardiogenic shock, and the absence of cardiogenic shock before operation is associated with a significantly lower risk of hospital mortality (19).

Our case also demonstrated that the IABP was a corner stone for maintaining the stable patient's condition; after the IABP was discontinued, further medical management of the patient's hemodynamics failed. The problem is that an IABP can be contraindicated or have to be discontinued due to IABP-related complications. It was noticed that the transient benefit of the IABP peaks at 24 hours, with no further benefit derived from prolonged use (18).

In the acute setting where surgery is deemed, another opportunity is a percutaneous closure of smaller VSDs with catheter-based devices. Transcatheter closure may now offer a different treatment option for critically ill patients and provide a bridge to stabilize patients' conditions with a shunt reduction in order to allow time for myocardial fibrosis, thus, facilitating a subsequent elective surgical correction (13). Ventricular septal occluders are offered to treat only small or medium ventricular septal defects, which are less than 15 mm (13, 20).

A novel strategy in surgical management may be a staged approach of an initial left ventricular assist device to stabilize the hemodynamic situation of the patient, thereby averting end-organ failure followed by secondary surgical repair to avoid surgery on the freshly infarcted myocardium (21).

For patients who successfully survive the preoperative period and surgery, a long-term prognosis is relatively good with a 10-year survival rate of 51% (19).

Conclusions

The keys of management of patients with ventricular septal defect are a prompt diagnosis of ventricular septal defect and an aggressive approach to stabilize hemodynamics. Invasive monitoring of hemodynamics, oxygen therapy, an intra-aortic balloon pump, and judicious use of inotropes and vasodilators help maintain optimal hemodynamics. Medical management is intended only for the temporary stabilization of the patient's status before surgery is carried out. The main task is to prolong the preoperative period in a stable condition, preferably for at least 4 weeks. The opportunity to postpone ventricular septal defect closure surgery is related with a better prognosis for a patient.

Statement of Conflict of Interest

The authors state no conflict of interest.

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