Iatrogenic ventricular septal defect: A rare complication of surgical reconstruction of mitral paravalvular dehiscence

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ABSTRACT

INTRODUCTION: Iatrogenic ventricular septal defect is a rare complication after the surgical replacement of cardiac valves. Small defects may have no hemodynamic significance or remain unremarked at the end of the surgical procedure. Understanding of the valvular anatomy alone is not always enough to avoid such complications, especially in the hands of young surgeons.

PRESENTATION OF CASE: We present a case of iatrogenic ventricular septal defect that developed early after the surgical closure of a hemodynamically significant mitral paravalvular leak. Although the patient’s critical state did not allow surgical intervention and he died, we think the lessons drawn from this case could be helpful to avoid such horrible complications in the future.

DISCUSSION: This case documents a rare disastrous complication after imperfect surgical closure of a mitral paravalvular leak. Despite the unfortunate end, in reporting this case we try to direct the light to the possible mechanisms that led to the development of this injury focusing on the embryological and anatomical background.

CONCLUSION: Understanding the anatomical and embryological structure of the cardiac fibrotic skeleton should keep cardiac surgeons more vigilant in detecting iatrogenic ventricular septal defects before the development of a devastating hemodynamic state.

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1. Introduction

Iatrogenic ventricular septal defect is a rare complication after the surgical replacement of cardiac valves. Small defects may have no hemodynamic significance or remain unremarked at the end of the surgical procedure. Late detection of these defects may lead to serious hemodynamic deterioration, which may render the clinical course irreversible. We present a case of iatrogenic ventricular septal defect that, developed early after the surgical closure of a hemodynamically significant mitral paravalvular leak. Although the patient’s critical state did not allow surgical intervention and he died, we think the lessons drawn from this case could be helpful to avoid such horrible complications in the future. In this paper we attempt to study the physioanatomic and embryologic bases that, might have led to the development of this complication.

2. Presentation of the case

A 61-year-old man was admitted to our cardiac surgical department in December 2015 due to a significant mitral prosthetic paravalvular leak. The symptoms of congestive heart failure dominated the clinical course. In 2008, the patient was planned to undergo aortic valve replacement due to a calcified stenotic aortic valve. During that procedure, the mitral valve was accidentally injured and the left fibrotic trigone was ruptured, so the mitral valve was replaced too. The patient’s past medical history was significant for chronic obstructive pulmonary disease, chronic atrial fibrillation, and previous pacemaker implantation due to third-degree atrioventricular block. The patient was in good health condition until the summer of 2015, when he was admitted to the hospital due to symptoms of dyspnea, fatigue, and weight loss. Transthoracic and transesophageal echocardiography were performed. A significant mitral paravalvular leak was revealed. The hemodynamic function of the aortic mechanic valve was normal. Echocardiography showed diffused hypokinesis and depressed left ventricle function (ejection fraction: 45%) without segmental wall motion disturbance. Based on the tricuspid annular plane systolic excursion (TAPSE) right ventricular dysfunction was also observed (TAPSE: 11 mm). No atrial or ventricle septal flow abnormalities were detected. Coronarography showed no significant coronary stenosis. Laboratory studies performed one week prior to and upon admission showed normal hepatic function, normal renal function, and normal inflammatory biomarkers. An urgent operation was performed. Median resternotomy and a transatrial septal approach were selected. The exposure of the entire mitral valvular ring and

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annulus was only partially feasible. An approximately 1 × 1 cm paravalvular dehiscence was observed in the region near the tricuspid septal annulus. The mitral annulus could not be explored perfectly, due to the pulling effect of the aortic prosthetic ring. In an attempt to reconstruct the dehiscence between the mitral prosthetic ring and the annulus, the surgeon covered the defect by pulling down the lower part of the atrial septum towards the prosthetic ring. Four 2/0 Ethibond-pledgeted sutures were used. Postoperative transesophageal echocardiography showed no paravalvular leak or other abnormal flow patterns. The patient was weaned from the cardiopulmonary bypass. Due to sustaining right ventricle dysfunction epinephrine and norepinephrine was initiated, but gradually stopped by the second postoperative day. Afterward, the patient was weaned from the respirator and extubated. In the fourth postoperative day, he became oliguric, then hypotensive. Central venous pressure (CVP) increased from 12 to 18 cmH2O. Circulatory support with high doses of epinephrine, norepinephrine and dopamine was restarted with only minimal beneficial effects. Transthoracic echocardiography showed a 2-cm wide pericardial effusion around the right ventricle, which was drained out through a mini subxiphoid incision. Thereafter, the blood pressure risen from 60 to 90 mmHg and CVP declined to 14 cmH2O. Three hours later, as the patient’s clinical state showed no further improvement, another echocardiographic examination was performed, which showed signs of severe right ventricle failure (TAPSE: 5 mm) and a 10-mm ventricle septal defect in the region of the membranous septum (Figs. 1 and 2). At this stage, the PCT was 56 mM/L, and systemic vascular resistance was critically low, mimicking the course of severe sepsis. Further surgical reoperation was considered, but not performed due to the severely critical state of the patient.

Postmortem examination (Fig. 3), in accordance with preoperative echocardiographic images, revealed a ventricle septal defect that was 1 cm in diameter. The defect was identified between the area below the aortic annulus and under the tricuspid septal leaflet. No necrotic signs were seen at the borders of the defect. Postinfarct VSD was precluded as no postmortem signs of early myocardial infarct or coronary occlusion were observed. No signs of active endocarditis were identified.

3. Discussion

This case documents a rare disastrous complication after imperfect surgical closure of a mitral paravalvular leak. During the primary operation, the intertrigonal region was accidently injured and the subsequent mitral valve regurgitation was addressed by the implantation of a mechanical prosthesis. This area was severely scarred, as documented by postmortem examination. Paravalvular dehiscence might be the result of the earlier trigosal injury, which might have weakened the structure of the fibrotic skeleton in this area. Although iatrogenic ventricular septal defects (VSDs) have been reported after aortic and mitral valve replacement [1], to our knowledge this complication has never been yet described in the English literature. Most of the reported cases dealt with successful surgical or percutaneous closure based on the enormous experience gained in treating congenital VSD [2]. Despite the unfortunate end, in reporting this case we try to direct the light to the possible mechanisms that led to the development of this injury focusing on the embryological and anatomical background. This may make us more vigilant in the future in avoiding complications that originate from structural rupture of the fibrotic skeleton.

3.1. Embryological and anatomophysiological aspects

During the cardiac cycle the movement of the fibrotic skeleton is stationary against the dynamic movement of the atrial and ventricle septum. The implantation of a stented bioprosthesis or mechanical valves in the cardiac skeleton may increase the rigidity of the fibrous zones at the boarders of the muscular septum, which may make the septum more vulnerable to stress forces during its dynamic motion. In our case, the Teflon-pledgeted sutures were introduced first to the lower part of the atrial septum, near to the mitral annulus, then through the rigid ring of the mitral mechanical prosthesis. In the setting of high-dose epinephrine-induced tachycardia, the repetitive stress forces on the membranous septum...
may profoundly increase, as it is the weakest point of the septum-
fibrotic skeleton attachment.

During fetal development, the membranous ventricular septum
develops independently from the muscular septum; therefore the
membranous portion is not actually a part of the muscular ven-
tricle septum, but rather is part of the aortico-pulmonary septum,
which fuses during its development with the muscular septum [3].
At the base of the heart, the membranous septum is in relation
with the mitral, tricuspid, and aortic annulus through the right fibrotic
trigonum, to which the primary atrial septum is attached superiorly
[4].

The right fibrotic trigonum is the center of complex anatomical
structures, with different embryological origins that, attach and function in relation to each other. These structures are: the
primary atrial septum, membranous ventricular septum, aortic
annulus (base of non- and right coronary sinus), tricuspid septal
annulus, and anteromedial mitral annulus. After aortic and mitral
mechanical valve replacement, the dynamics of the right fibrotic
trigonum and the related structures may change to adapt to the
new stress forces exerted on the structures with dynamic move-
ments around the cardiac cycle. The anterior mitral annulus is not
attached to the ventricular musculature, but rather is suspended
between the right and left trigone. Therefore, the shape of this part
of the annulus dynamically changes through the cardiac cycle. This
feature would be lost after implantation of a prosthetic valve in the
mitral orifice and the annulus would become immobile and round
in shape.

In this setting, the portion of the trigonum underlying the tri-
cuspid septal annulus may be the most vulnerable against tearing
forces. The effect of these tearing forces may be more signifi-
cant in catecholamine-induced tachycardia. This surgical solution,
selected to reconstruct the mitral paravalvular leak developed near
the right fibrotic trigone, could have been successful in the setting
of a native aortic valve, as this may distribute the tearing force
exerted through the other two sides of the trigonum, the aortic
native annulus, and the tricuspid septal annulus.

3.2. Diagnostic considerations

This defect could have been a post-infarction rupture of the
ventricular septum. This was precluded due to a negative coro-
narogram, and the absence of coronary occlusion or evidence of
a postinfarct septal defect pathology pattern during postmortem
examination [5,6].

Although VSD has been reported as a complication of perian-
ular distribution of prosthetic valve endocarditis [7], in our case
endocarditis was not diagnosed. No bacteria or fungi was cultured
during the microbiological examination of blood cultures. No ve-
egatations were observed during echocardiography and postmortem
examination showed no signs of endocarditis all over the cardiac
endocardium.

4. Conclusion

Understanding the anatomical and embryological structure of
the cardiac fibrotic should keep cardiac surgeons more vigilant in detecting iatrogenic ventricle septal defects before the
development of a devastating hemodynamic state. Other pathological
states should be considered and excluded.

Conflicts of interest

Authors have no conflicts of interest.

Sources of funding

Authors have no sponsor or any other external funding to declare.

Ethical approval

The patient in this case report had, unfortunately, deceased. Due
to extreme anonymity, no ethical approval was approved.

Consent

The patient in this case report had, unfortunately, deceased. His
only next of kin is his life’s partner whom he was living for
twenty years. No other relatives were found alive. Written informed
consent was obtained from her for publication of this case and
accompanying images. A copy of the written consent is enclosed.

Author contribution

Aref Rashed: He decided to summarize the clinical course in a
case report for publication. He edited the anatomical and embry-
ological background.

Karoly Gombocz: He prepared the figures and contributed in editing.

Janos Fulop: He edited the surgical part in the report. Nasri
Alotti: He revised the report and contributed in editing the conclusion.

Registration of research studies

One case report, not listed in the research register.

Guarantor

Aref Rashed.

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