Case Report



Occlusion of Ventricular Defect Post-Myocardial Infarction with Prothesis Cera

Henrique Barbosa Ribeiro, Luciano Moreira Baracioli, Luiz Junya Kajita, Martina Battistini Pinheiro, Expedito E. Ribeiro, José Carlos Nicolau

Instituto do Coração (InCor) do Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo - Brazil

Introduction

Ventricular septal defect (VSD) that occurs after acute myocardial infarction (AMI) is a rare event, with an incidence reported in the literature from 0.2% to 0.34% of the cases¹. The treatment of choice is surgical correction, and if not performed earlier, with due support (especially the implantation of intraaortic balloon in cases of cardiogenic shock²-³), the disease has a mortality rate above 90%¹-³-⁴.

In turn, the appropriate surgical treatment of VSD still results in high mortality rates, which vary in current series 20% to $87\%^4$.

More recently it has been proposed as an alternative method, less invasive than surgery, closure of VSD after AMI with percutaneous technique by occlusion devices^{3,5}. In most reports and case series in the literature evaluating its use in the context of AMI, we used the Amplatzer® septal occluder (AGA Medical Corporation, Plymouth, MN, USA)^{3,5}. Currently, other prostheses are available, such as prostheses CERA made of nitinol with ceramic coating layers and ePTFE mesh. In this sense, we report the first case of percutaneous closure of VSD after AMI conducted in our country with the use of this prosthesis.

Case Report

Male patient, 55 years, white, with previous hypertension, dyslipidemia, and smoker (40 years. pack). He developed chest pain at rest and exertion, of recent onset (five weeks), associated with dyspnea. Chest radiography showed an enlarged cardiac silhouette, and it was sent for echocardiography, which showed an ejection fraction (LVEF) = 65%, although akinesia of the anteroseptal wall. The clinical treatment was optimized and it was requested myocardial perfusion scintilography with pharmacological stress, which showed persistent uptake in the

Keywords

Myocardial infarction; heart septal defects, ventricular / mortality; heart septal defects, ventricular / surgery; septal occluder device.

Mailing Address: Henrique Barbosa Ribeiro •

Rua Cônego Eugenio Leite, 866 / 43, Cerqueira César. Postal Code 05414-001, São Paulo, SP - Brazil

E-mail: hbribeiro@cardiol.br, henrique37@terra.com.br Manuscript received July 06, 2011; manuscript revised August 17, 2011; accepted August 22, 2011. inferior wall (apical portion) and the anteroseptal wall with mild transient component, and LVEF of 40%.

Consequently, coronary angiography was performed, which demonstrated the Left Anterior Descending artery (LAD) occluded in the medium third, receiving collateral circulation grade I of the Right Coronary Artery (RCA), circumflex artery with obstruction of 70% in its distal third; RCA without obstructive injuries. On the left ventriculography, in addition to moderate anterior hypocontractility and anteroseptal akinesia, it was observed muscular VSD near the ventricular apex (Figure 1). That same day, a new echocardiogram confirmed the muscular VSD with about 5 mm and L/R shunt.

The patient was then referred to our hospital, two weeks after diagnosis of VSD, maintaining some degree of dyspnea (NYHA class II). We promoted a discussion (clinician, interventionalist and cardiac surgeon) and due to the not indication for CABC (very narrow distal bed of LAD) and anatomy of VSD favorable for percutaneous closure (muscular VSD near the cardiac apex, about 5 mm edges diameter and > 5 mm), we opted for the correction of the VSD with the use of percutaneous prosthesis.

The procedure for closure of the VSD was successful, through the implantation of prosthesis CERA muscular type No. 10 driven by transthoracic echocardiography and angiography. This device for muscular VSD is made of a mesh of ultra-fine wires of nitinol with polyester patches in the inner portion (ePTFE)⁶, composed of two discs connected by a central waist length of 7 mm (Figure 2). In addition, the prosthesis mesh, screw and hub are coated with nanostructures of ceramic, which prevent direct contact of nitinol (nickel-containing) in the blood. This prosthesis is self-expansible and self-centered, being implanted by low-profile system that allows rescuing it and reposition it at any time before its final release from the delivery cable (Figure 2). All these devices are registered on the National Health Surveillance Agency (ANVISA).

There were no complications during the procedure, and control ventriculography showed no residual shunt (Figure 1). The patient remained asymptomatic after the procedure and was discharged within 48 hours. In the follow-up of nine months, the patient was asymptomatic, with significant improvement in functional class (NYHA class I), and control echocardiogram showed normal LVEF without evidence of residual shunt.

Discussion

In our case of post-AMI VSD closed percutaneously with prosthesis CERA, the patient had shown good immediate

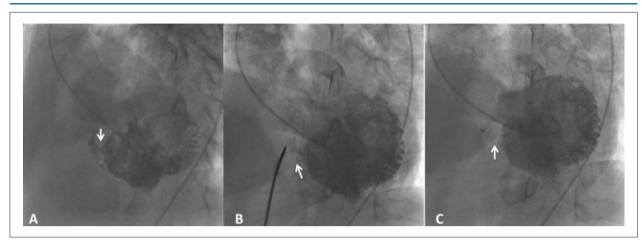


Figure 1 - Left ventriculography in hepatoclavicular projection showing: (A) ventricular septal defect (VSD)-type single muscle in the middle of the septum with a minimum diameter of 5 mm and a significant left-right flow (white arrow) (B) Percutaneous implantation of prosthesis CERA (white arrow) for closure of the VSD, (C) Control ventriculography demonstrating device well positioned with a minimum residual flow by the mesh of the prosthesis (white arrow).



Figure 2 - Prosthesis CERA of muscular ventricular septal defect. Prosthesis made of fine mesh of nitinol with two discs connected by a central waist of 7 mm in length. Internally, we note the patches of ultrathin polyester (ePTFE). A steel release cable connects to the right disk by a screw mechanism.

clinical improvement and in the follow-up of nine months. The VSD occurring after AMI is an event with high morbidity and mortality, especially in patients who develop cardiogenic shock and those not subject to surgical closure of the deffect^{1,3,4}. However, this type of treatment still presents success rates of less than ideal, especially when performed in patients with high operative risk and / or unfavorable anatomy⁴.

Although surgical treatment is still considered the treatment of choice for correction of post-AMI VSD², we have the fact that the septal branches are exposed to shear stress and the removal of necrotic tissue, soon after the occurrence of VSD,

which may result in subsequent abrupt expansion of the defect, with subsequent residual shunt. Obviously, the surgical results are better when you expect the healing of myocardial infarction (four to six weeks)^{3,5,7}, but these findings only feature a selection bias (more severe cases die earlier), and the official recommendation of all current guidelines on the subject is the correction of the defect as soon as possible after diagnosis².

Since the introduction of percutaneous closure of VSD⁷, several series have been reported, being initially used only for patients with VSD in sub-acute or chronic stage, or for patients with residual shunt after operation⁵. Later, Thiele et al³

Case Report

published the first experience with the procedure performed in acute patients including cardiogenic shock, the survival rate at 30 days was 35%, reaching 12% in those with cardiogenic shock (p <0.001). In addition, 41% of patients had residual shunt, left ventricular rupture or embolization of the device in follow-up.

Most case and series reports in the literature evaluating the percutaneous closure of VSD in the context of AMI used the Amplatzer® septal occluder (AGA Medical Corporation, Plymouth, MN, USA)^{3,5}. However, today many other prostheses have been tested as alternatives to Amplatzer® prostheses for septal defect closure, including the prosthesis CERA, whose characteristics were already described. Important to emphasize that these characteristics prevent direct contact of nitinol with the blood, promote tissue growth and reduce the risk of formation of thrombus⁶.

In this case, the defect was small (5 mm) and located in the muscle, with description of spontaneous closure of VSD after AMI with such characteristics⁸. However, our patient presented about five to six weeks of development of AMI and had an unfavorable clinical evolution, with progressive worsening of

symptoms, the option for the correction of the defect resulted in complete remission of symptoms.

Conclusion

It is concluded that, despite surgical treatment remains the treatment of choice in patients with VSD after AMI, the percutaneous procedure may be an option, especially in patients with high operative risk and/or unfavorable anatomy for surgery.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

There were no external funding sources for this study.

Study Association

This study is not associated with any post-graduation program.

References

- 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. GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) Trial Investigators. Circulation. 2000;101(1):27-32.
- IV Guidelines of Brazilian Society of Cardiology for treatment of acute myocardial infarction with ST-segment elevation. Arq Bras Cardiol. 2009;93(6 Suppl. 2):e179-264.
- Thiele H, Kaulfersch C, Daehnert I, Schoenauer M, Eitel I, Borger M, et al. Immediate primary transcatheter closure of postinfarction ventricular septal defects. Eur Heart J. 2009;30(1):81-8.
- Menon V, Webb JG, Hillis LD, Sleeper LA, Abboud R, Dzavik V, et al.
 Outcome and profile of ventricular septal rupture with cardiogenic shock
 after myocardial infarction: a report from the SHOCK Trial Registry. SHould

- we emergently revascularize Occluded Coronaries in cardiogenic shocK? J Am Coll Cardiol. 2000;36(3 Suppl. A):1110-6.
- Demkow M, Ruzyllo W, Kepka C, Chmielak Z, Konka M, Dzielinska Z, et al. Primary transcatheter closure of postinfarction ventricular septal defects with the Amplatzer septal occluder- immediate results and up-to 5 years follow-up. EuroIntervention. 2005;1(1):43-7.
- Zhang D, Zhang Z, Zi Z, Zhang Y, Zeng W, Chu PK. Fabrication of graded TiN coatings on nitinol occluders and effects on in vivo nickel release. Biomed Mater Eng. 2008;18(6):387-93.
- Lock JE, Block PC, McKay RG, Baim DS, Keane JF. Transcatheter closure of ventricular septal defects. Circulation. 1988;78(2):361-8.
- Mansur AP, Rossi EG, Ramires JAF. Spontaneous closure of ventricular septum rupture after acute myocardial infarction. Arq Bras Cardiol. 1990;55(6):379-80.