Current Cardiology Reports (2019) 21: 59
https://doi.org/10.1007/s11886-019-1142-8

STRUCTURAL HEART DISEASE (RJ SIEGEL AND NC WUNDERLICH, SECTION EDITORS)

Techniques, Timing, and Prognosis of Transcatheter Post Myocardial Infarction Ventricular Septal Defect Repair

Alessia Faccini [1] · Gianfranco Butera [2,3]

Published online: 20 May 2019

Abstract

Purpose of Review This review aims to delineate the actual role of percutaneous intervention in the closure of post-myocardial infarction ventricular septal defect (post-MI VSD) and to briefly summarize the main steps of this procedure.

Recent Findings Most of the published studies report experiences using Amplatzer devices for post-MI VSD closure. In the acute phase, morbidity and mortality are quite high up to 70%, with a mean success rate of 90%, with 95% confidence intervals from 60 to 100%, and a 30-day mortality of 40%, with 95% confidence intervals from 0 to 55%. In the chronic phase, that is 14 days after myocardial infarction, results are very encouraging, with lower morbidity and mortality (23% at 30 days) and a higher rate of complete closure. A multimodality imaging approach has been proposed in order to increase the success rate of this procedure.

Summary Percutaneous closure is a safe and effective procedure in highly specialized centers and an appropriate patient selection is of paramount importance to the success of the procedure. Device closure of post-MI VSD can be considered a true alternative to the standard surgical approach. However, many problems still exist for percutaneous post-MI VSD treatment.

Keywords Post-myocardial infarction ventricular septal defect · Percutaneous intervention · Ventricular septal defect occluder

Introduction

Post-myocardial infarction (MI) ventricular septal defects (VSDs) are ruptures within a necrotic tissue. Compared with congenital VSDs, they are not discrete holes, and one defect may tear into multiple defects. In the era of reperfusion therapy, rupture of the ventricular septum may occur in around 0.2% of MI [1]. It is usually observed in the first 24 h and at 3 to 7-day post-MI and is associated with very high morbidity and mortality [1]. Risk factors for post-MI VSDs include advanced age, female gender, and left anterior descending coronary artery infarction. Medical therapy alone has a survival rate of less than 8% at 30 days [2] and of less than 3% at 1 year [1–4]. On the contrary, surgical and interventional procedures have a 30-day mortality that ranges between 30 and 60% [1–6]. Post-MI VSDs can present with hemodynamic deterioration and signs and symptoms of acute heart failure but, as reported in recent European guidelines, there is no consensus on the optimal timing for surgery, as an early intervention seems to have a 20–40% mortality rate and a high risk of recurrent ventricular rupture, while a late one bears the risk of rupture extension and death while waiting for surgery [7••]. Recent reviews describe a lower mortality in patients who undergo either surgical or interventional post-MI VSD repair at least 2 weeks after MI diagnosis [2], and the Society of Thoracic Surgeons National Databases seems to confirm that a longer interval between diagnosis of MI and surgical repair (> 21 days) is associated with lower odds of operative mortality [8]. Efforts have been done in order to stratify the prognosis of patients with post-MI VSDs and to guide their management, with low mean blood pressure with intra-aortic balloon pump, higher EuroSCORE II, higher Killip class, and shorter intervals between MI and VSD development as well as between VSD formation and

This article is part of the Topical Collection on *Structural Heart Disease*

✉ Gianfranco Butera
gianfrancobutera@libero.it; Gianfranco.butera@gstt.nhs.uk

Alessia Faccini
alessiafaccini@hotmail.com

[1] Department of Congenital Cardiology and Cardiac Surgery, IRCCS Policlinico San Donato, San Donato Milanese, Italy

[2] Department of Pediatric Cardiology and Adults with Congenital Heart Disease, Evelina Children's Hospital- St Thomas Hospital, 249 Westminster Bridge Road, London SE1 7EH, UK

[3] King's College London, 57 Waterloo Road, London SE1 8WA, UK

surgical intervention being strong predictors of mortality [9]. There are evidences showing that concomitant revascularization by CABG to all the stenotic arteries, supplying the non-infarcted area, improves 30-day mortality [10, 11].

Surgical repair of acutely ruptured myocardium is challenging, and different techniques have been used [4–6]. Moreover, in the current era, the use of extracorporeal membrane oxygenation (ECMO) has been proposed as a bridge to surgical intervention in patients with post-MI VSD and cardiogenic shock [12•]. Furthermore, an up to 40% risk of post-operative residual shunting has been described, with patch dehiscence, development of a new VSD, or an overlooked second VSD being among the most important causes for recurrence. Mortality associated with a redo surgery to repair a recurrent post myocardial infarction VSD has been reported to be up to 31% [4–6].

Since the first report in 1988 by Lock et al. [13], percutaneous post-MI VSD closure has become an option and the current European guidelines state that it may soon become an alternative to surgery [7••], useful also to close residual shunts following surgical post-MI VSD closure [14]. Main contraindications for transcatheter therapy are defect size > 35 mm, basal VSD rim near the mitral or aortic valves, and apical VSD without sufficient margins [15].

Devices, Procedure, and Technique of Device Implantation

The use of a variety of devices has been reported in the literature. In particular, most of the studies describe the use of Amplatzer™ (Abbott Laboratories, Abbott Park, IL, USA) tools such as atrial septal defect occluder, muscular VSD occluder, and post-infarct muscular VSD occluder [16–18]. The first one seems to be the worst option especially in the acute phase [19], while the second and the third one have better results [20]. The post-infarct muscular VSD occluder has been described to have a greater success than the devices used previously [16]. It is similar to the muscular device, with two important differences: the connecting waist is longer with a 10-mm length, and the discs are 10 mm larger than the connecting waist. The device is available in sizes that range from 16 to 24 mm in 2-mm increments and can be delivered through 9 to 10-Fr-long sheaths. The size of the device is usually chosen as having a waist at least 4 mm larger than the stretched diameter of the VSD found on the transesophageal echocardiography.

The principal steps of the procedure are similar to those used for closure of muscular defects. There are two major differences. First, patients are usually in critical clinical conditions, often requiring inotropic and/or mechanical support with intra-aortic balloon pumping, ECMO, or Impella. Second, when possible, it is advisable to perform percutaneous coronary arterial revascularization before attempting closure of the ventricular septal defect.

The vascular venous access is usually obtained from the jugular vein, while the femoral vein access is used for anterior defects. An arterial access is also gained through the femoral artery. The defect is commonly crossed from the left ventricle by using a right coronary artery catheter (Cordis Corporation, Miami Lakes, FL, USA) and an exchange angled torque wire (0.035-in. Terumo wire, Terumo Corporation, Tokyo, Japan). The wire is often directed towards the pulmonary artery where it is snared and pulled out from the venous access in order to create an arterio-venous circuit (Fig. 1a–c). The Terumo wire can be exchanged for an AGA exchange rope wire. The defect can be balloon-sized to be sure that the guide wire is not trapped around a cord or that the wire is not crossing the largest hole. Finally, balloon sizing may be useful in order to rule out multiple defects (Fig. 1d).

Sometimes, in particular in the presence of apical defects, it may be easier to enter the defect from the right ventricle directing the guide wire in the left ventricle and the aorta where it is snared.

Echocardiography (usually transesophageal) is very useful in order to visualize the maximum left ventricular, septal, and right ventricular orifices of the defect itself and to look after the guide wire crossing these openings. Sometimes, in order to improve the visualization of apical defects, transthoracic imaging may be more useful.

The next steps include the progression of the long sheath over the artero-venous circuit, the opening of the left ventricular disc (Fig. 1e), the approximation of the left ventricular disc to the left ventricular aspect of the septum, the opening of the right ventricular disc (Fig. 1f), and finally the release of the device.

Sometimes, some special techniques could be useful to succeed, such as the over-the-wire device implantation [21] or the goose-neck snare-assisted techniques [22].

Successful use of the Gore septal occluder (GSO) (W. L. Gore and Associates, Flagstaff, AZ) has also been described in case reports, adopting the standard percutaneous technique [23].

Results

Various complications can occur due to the percutaneous intervention: tamponade subsequent to perforation of the ventricular wall [24], partial or total dislocation of a successfully deployed device into the right ventricle, persistent shunting from device dysfunction, or partial dislocation of deployed occluders that can result in infective endocarditis in the future

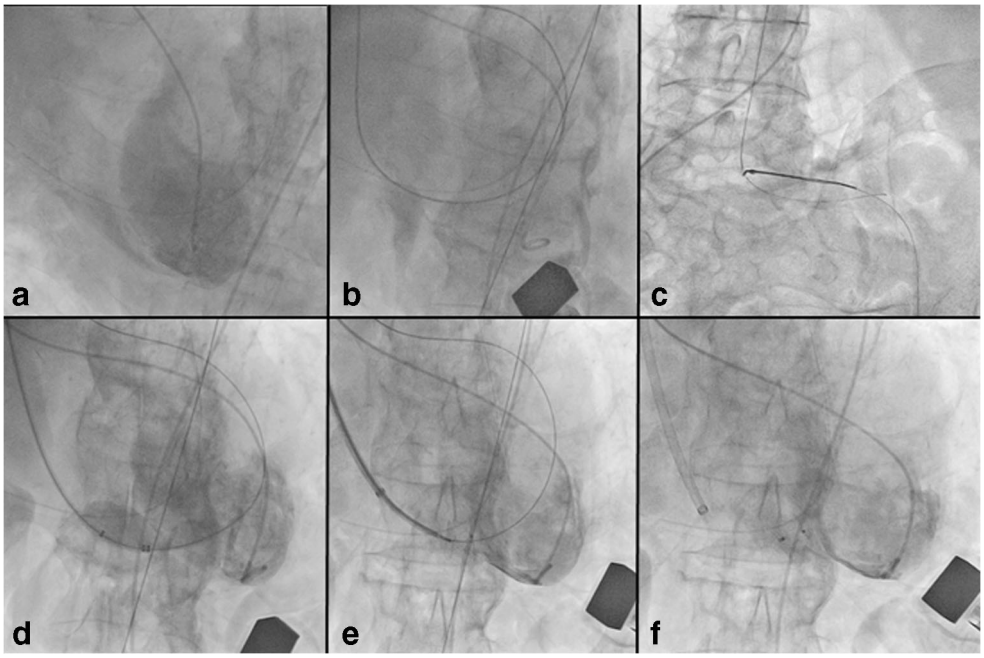

Fig. 1 **a** Left ventricular angiography in the left anterior oblique view showing a left-to-right shunting through a post-MI VSD. **b** Fluoroscopy in the left anterior oblique view showing the guide wire through the VSD. **c** Fluoroscopy in the anterior-posterior view showing the guide wire caught by a goose-neck snare to create the arterio-venous circuit. **d** Left ventricular angiography in anterior-posterior view with a sizing balloon across the VSD. No further defects are shown. **e** Left ventricular angiography in the anteroposterior view showing a device properly placed. The device is still attached to the delivery cable. A transthoracic echocardiographic monitoring is performed. **f** Guide wire is removed. Left ventricular angiography shows trivial intra-prosthetic shunting

[25], arrhythmias [26], hemolysis [27], and tricuspid leaflet flail due to chordal rupture [28].

Several studies have reported the experiences using Amplatzer devices in these subjects [16–20, 24, 29–34]. These papers show that, in the acute phase, morbidity and mortality are quite high up to 70%, and the rate of successful closure is low. In fact, pooling all data from the literature, we calculate a mean success rate of 90%, with 95% confidence intervals from 60 to 100%, and a 30-day mortality of 40%, with 95% confidence intervals from 0 to 55%.

In the chronic phase, that is 14 days after myocardial infarction, results are very encouraging, with lower morbidity and mortality (23% at 30 days) and a higher rate of complete closure [24].

There are several limitations to the currently available options. Devices should be available in larger sizes, should be softer, and endothelialized quickly. Furthermore, improvements in imaging should help in selecting subjects who could beneficiate from a percutaneous treatment. A detailed in vivo description of the dynamic anatomy of post-MI VSDs has been published, highlighting the complex characteristics of these defects with VSD edges that can be thin and akinetic and with some defects that can get larger and some smaller in systole, meaning that single-phase imaging may not adequately assess size [35•]. Therefore, a multimodality imaging approach is actually emphasized in the literature [36].

Place in Clinical Management

Treatment of a post-myocardial infarction VSD remains a major challenge in particular in the acute phase. In several centers, only subjects surviving more than 3–4 weeks are considered for closure (either surgical or percutaneous). This improves the procedural results; however, an earlier aggressive approach may salvage more subjects. Recurrent post-infarction VSD following surgical patch repair may beneficiate from transcatheter closure as the treatment of choice.

Conclusions

In summary, the currently available data shows that, in particular after the introduction of the VSD Amplatzer devices, percutaneous closure is a safe and effective procedure in highly specialized centers. Appropriate patient selection is of paramount importance to the success of the procedure. Device closure of post-MI VSD can be considered a true alternative to the standard surgical approach, both in the acute and subacute phase post MI, bearing in mind that an acute approach, usually reserved for hemodynamically unstable patients, has a higher mortality. The percutaneous approach can be regarded as the first

treatment for residual post-surgical defects. However, many problems still exist for percutaneous post-MI VSD treatment. Therefore, further improvements in technology are needed in order to overcome present limitations and risk of complication.

Compliance with Ethical Standards

Conflict of Interest Alessia Faccini declares no conflict of interest. Gianfranco Butera is a proctor for Abbott.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1. Crenshaw BS, Granger CB, Birnbaum Y, for the GUSTO-I Trial Investigators. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. Circulation 2000; 101: 27–32.
2. Omar S, Morgan GL, Panchal HB, Thourani V, Rihal CS, Patel R, et al. Management of post-myocardial infarction ventricular septal defects: a critical assessment. J Interv Cardiol. 2018;31(6):939–48.
3. 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;36:1110–6.
4. Labrousse L, Choukroun E, Chevalier JM, Madonna F, Robertie F, Merlico F, et al. Surgery for post infarction ventricular septal defect: risk factors for hospital death and long term results. Eur J Cardiothorac Surg. 2002;21:725–31.
5. Papadopoulos N, Moritz A, Dzemali O, Zierer A, Rouhollapour A, A Ackermann H, Bakhtiary F. Long-term results after surgical repair of postinfarction ventricular septal rupture by infarct exclusion technique. Ann Thorac Surg 2009; 87:1421–1425.
6. Cerin G, Di Donato M, Dimulescu D, Montericcio V, Menicanti L, Frigiola A, et al. Surgical treatment of ventricular septal defects complicating acute myocardial infarction. Experience of a north Italian referral hospital. Cardiovascular Surg. 2003;11:149–54.
7. •• ¨ Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al; ESC Scientific Document Group. 2017 ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the task force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018;39(2):119–177. **ESC guidelines assess that there is no consensus on the optimal timing of post-MI VSD surgical closure.**
8. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the society of thoracic surgeons national database. Ann Thorac Surg. 2012;94(2):436–44.
9. Malhotra A, Patel K, Sharma P, Wadhawa V, Madan T, Khandeparkar J, et al. Techniques, timing & prognosis of post infarct ventricular septal repair: a re-look at old dogmas. Braz J Cardiovasc Surg. 2017;32(3):147–55.
10. Barker TA, Ramnarine IR, Woo EB, Grayson AD, Au J, Fabri BM, et al. Repair of post-infarct ventricular septal defect with or without coronary artery bypass grafting in the northwest of England: a 5-year multi-institutional experience. Europ J Cardio-thorac Surgery. 2003;24:940–6.
11. Perrotta S, Lentini S. In patients undergoing surgical repair of post-infarction ventricular septal defect, does concomitant revascularization improve prognosis? Interactive Cardiovascular Thoracic Surg. 2009;9:979–87.
12. • McLaughlin A, McGiffin D, Winearls J, Tesar P, Cole C, Vallely M, et al. Veno-arterial ECMO in the setting of post-infarct ventricular septal defect: a bridge to surgical repair. Heart Lung Circ. 2016;25(11):1063–6 **This paper highlights the emerging role of ECMO in post-MI VSD management**.
13. Lock JE, Block PC, McKay RG, Baim DS, Keane JF. Transcatheter closure of ventricular septal defects. Circulation. 1988;78:361–8.
14. Elasfar AA, Soofi MA, Kashour TS, Koudieh M, Galal MO. Transcatheter closure of residual postinfarction ventricular septal defect after dehiscence of surgical patch repair. Ann Saudi Med. 2014;34(2):171–4.
15. Barik R. Transcatheter closure of post-myocardial infarction ventricular defect: where are we? Indian Heart J. 2016;68(1):99–101.
16. Holzer R, Balzer D, Amin Z, Balzer D, Amin Z, Ruiz CE, et al. Transcatheter closure of postinfarction ventricular septal defects using the new Amplatzer muscular ventricular septal defect occluder: results of a US registry. Cathet Cardiovasc Interv. 2004;61:196–201.
17. Szkutnik M, Bialkowski J, Kusa J, Banaszak P, Baranowski J, Gasior M, et al. Postinfarction ventricular septal defect closure with Amplatzer occluders. Eur J Cardiothorac Surg. 2003;23:323–7.
18. Chessa M, Carminati M, Cao QL, Butera G, Giusti S, Bini RM, et al. Transcatheter closure of congenital and acquired muscular ventricular septal defects using the Amplatzer device. J Invasive Cardiol. 2002;14(6):322–7.
19. Bialkowski J, Szkutnik M, Kusa J, Kalarus Z, Gasior M, Przybylski R, et al. Transcatheter closure of postinfarction ventricular septal defects using Amplatzer devices. Rev Esp Cardiol. 2007;60:548–51.
20. Shabestari MM, Ghaderi F, Hamedanchi A. Transcatheter closure of postinfarction ventricular septal defect: a case report and review of literature. J Cardiovasc Thorac Res. 2015;7(2):75–7.
21. Butera G, Castaldi B, McDonald ST. Over the wire technique device implantation. Catheter Cardiovasc Interv. 2012;80(3):485–92.
22. Butera G, Lovin N, Basile DP, Carminati M. Goose-neck snare-assisted transcatheter ASD closure: a safety procedure for large and complex ASDs. Catheter Cardiovasc Interv. 2016;87(5):926–30.
23. Arias EA, Bhan A, Lim ZY, Mullen M. Utility of the Gore septal occluder in transcatheter closure of post-myocardial infarct ventricular septal defect. JACC Cardiovasc Interv. 2016;9(21):2259–61.
24. Sabiniewicz R, Huczek Z, Zbroński K, Scisło P, Rymuza B, Kochman J, et al. Percutaneous closure of post-infarction ventricular septal defects—an over decade-long experience. J Interv Cardiol. 2017;30(1):63–71.
25. Shahreyar M, Akinseye O, Nayyar M, Ashraf U, Ibebuogu UN. Post-myocardial infarction ventricular septal defect: a comprehensive review. Cardiovasc Revasc Med. 2018.
26. Schlotter F, de Waha S, Eitel I, Desch S, Fuernau G, Thiele H. Interventional post-myocardial infarction ventricular septal defect closure: a systematic review of current evidence. EuroIntervention. 2016;12(1):94–102.

27. Rao DS, Barik R, Siva Prasad A. Hemolysis induced by PMIVSD occluder. Indian Heart J. 2016;68(Suppl 2):S60–3.
28. Kafes H, Ozeke O, Demirkan B, Acar B, Aysenur Ekizler F, Karabulut O, et al. Flail tricuspid leaflet during the percutaneous closure of post-myocardial infarction ventricular septal defect. CASE (Phila). 2017;1(5):207–9.
29. Goldstein JA, Casserly IP, Balzer DT, Lee R, Lasala JM. Transcatheter closure of recurrent postmyocardial infarction ventricular septal defects utilizing the Amplatzer postinfarction ventricular septal defect device: a case series. Catheter Cardiovasc Interv. 2003;59:238–43.
30. Aggarwal M, Natarajan K, Vijayakumar M, Chandrasekhar R, Mathew N, Vijan V, et al. Primary transcatheter closure of post-myocardial infarction ventricular septal rupture using Amplatzer atrial septal occlusion device: a study from tertiary care in South India. Indian Heart J. 2018;70(4):519–27.
31. Thiele H, Kaulfersch C, Daenhert I, Schoenauer M, Eitel I, Borger M, et al. Immediate primary transcatheter closure of postinfarction ventricular septal defects. Eur Heart J. 2009;30:81–8.
32. Martinez MW, Mookadam F, Sun Y, Hagler DJ. Transcatheter closure of ischemic and post-traumatic ventricular septal ruptures. Catheter Cardiovasc Interv. 2007;69:403–7.
33. Ahmed J, Ruygrok PN, Wilson NJ, Webster MWI, Greaves S, Gerber I. Percutaneous closure of post-myocardial infarction ventricular septal defects: a single centre experience. Heart, Lung Cir. 2008;17:119–23.
34. Egbe AC, Poterucha JT, Rihal CS, Taggart NW, Cetta F, Cabalka AK, et al. Transcatheter closure of postmyocardial infarction, iatrogenic, and postoperative ventricular septal defects: the mayo clinic experience. Catheter Cardiovasc Interv. 2015;86:1264–70.
35.• Hamilton MCK, Rodrigues JCL, Martin RP, Manghat NE, Turner MS. The in vivo morphology of post-infarct ventricular septal defect and the implications for closure. JACC Cardiovasc Interv. 2017;10(12):1233–43 **This paper describes the post-MI VSD morphology and explains possible difficulties in VSD closure related to such different shapes**.
36. Iyer S, Bauer T, Yeung M, Ramm C, Kiser AC, Caranasos TG, et al. A heart team and multi-modality imaging approach to percutaneous closure of a post-myocardial infarction ventricular septal defect. Cardiovasc Diagn Ther. 2016;6(2):180–4.