

# Three weeks after coronary angioplasty, device closure of post-myocardial infarction ventricular septal defect

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## ABSTRACT

Nach der ersten surgical closure scheint die percutaneous device closure safe and effective in patients treated for a residual shunt, as well as after two to three weeks of index myocardial infarction. In the second of an acute myocardial infarction thrombolysed with streptokinase, the index case demonstrated a ventricular septal defect. Die allgemeine Gesundheit des Patienten war vergleichsweise stabil. There was a 90 % proximal stenosis of the left anterior descending coronary artery, as demonstrated by cardiac catheterization and coronary angiography. Anderen koronaren Arterien waren normal. In order to treat the coronary artery lesion, an angioplasty and stenting was performed using a drug eluting stent (DES) with a very good angiographic result. Die Patientin wurde nach vier Tagen in einer stabilen Situation entlassen. His ventricular septal defect was closed three weeks later. mittels eines kardio-O-Fix-Geräts mit einem kleinen residualen Shunt perkutan. Die Vorgehensweise war unauffällig und kurz. Following five days of the procedure, he was released in a very stable state with a minimal residual shunt. If the patient's condition allows strengthening the ventricular septal defect border, a staged procedure is a better option.

**Key words:** Cardio-Fix-Occluder, myocardial infarction, ventricular septal defect

## INTRODUCTION

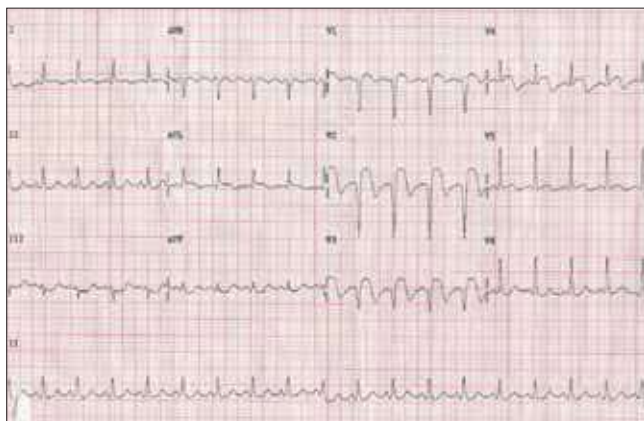
0.2 % of patients in this thrombolytic era have a ventricular septal defect (VSD) that complicates an acute myocardial infarction (AMI). In der ersten Woche beträgt die Häufigkeit etwa 1-2 %. In Bezug auf Medizinmanagement sterben 94 % innerhalb von 30 Tagen.[2,1] Die übliche Behandlung war die surgical closure of post infarction VSD with or without appropriate revascularization, regardless of the clinical state.[3] Die Sterblichkeitsraten bei surgical closure remain high at 20–87% in the majority of series.[3,4,5] In order to allow scarring of the surrounding tissue, which allows better anchoring of sutures, many surgeons prefer surgical closure after a 3–4 week delay. An interventionelle Methode mit einem Gerät ist weniger

invasive option, and allows immediate complete closure after initial hemodynamic stabilization. Immediate reduction of the left-to-right shunt, even if the VSD is not completely closed, may stabilize the patient enough to function as a bridge to surgery.[6] Current interventional reports are mainly restricted to VSD closure in the sub acute or chronic setting, or for residual shunts after initial surgical closure.[7-9] We report a case of anterior myocardial infarction (MI) which was subjected to per-cutaneous transluminal coronary angioplasty (PTCA) of the culprit lesion on fourth day of myocardial infarction, and closure of VSD with a device 3 weeks later, to allow reasonable stabilization of patient and the rims of the defect.

## CASE REPORT

A 60-jähriger Mann ohne Vorgeschichte von Hypertension, Diabetes mellitus oder Tabakkonsum erlitt innerhalb von sechs Stunden eine acute anterior wall myocardial infarction und wurde mit streptokinase thrombolysiert. He wurde in unser Krankenhaus verlegt.

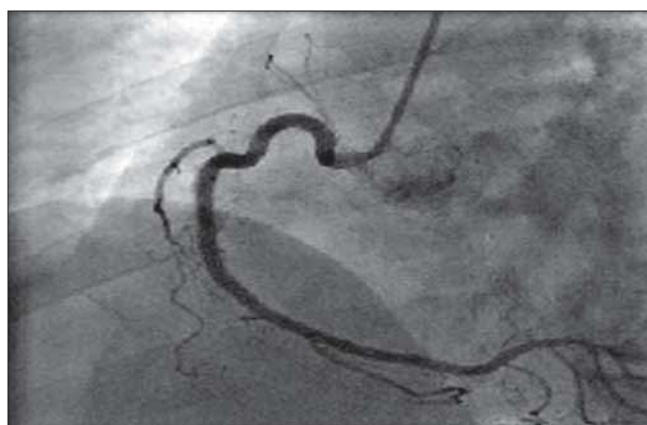
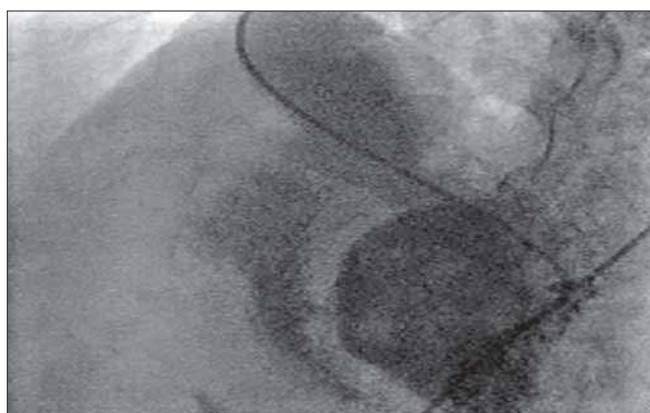
on detection of a ventricular septal defect, ongoing angina and breathlessness at rest next day. On admission, he was in congestive heart failure of Killip II with a pulse rate of 109/minute, blood pressure of 100/70 mm Hg and a grade IV pansystolic murmur at left lower parasternal area. His 12 lead surface electrocardiogram (ECG) was consistent with evolved acute anterior wall myocardial infarction [Figure 1]. Two-dimensional and 3-dimensional echocardiogram showed akinetic mid and distal interventricular septum and apex, moderate left ventricular dysfunction and a muscular ventricular septal defect of 10 mm size in the anterior-middle part of the septum [Figure 2]. Cardiac catheterization done on the same day revealed a single muscular ventricular septal defect of size 10 mm [Figure 3], with a significant step up of oxygen saturation at right ventricular level by 23% and the ratio of pulmonary blood flow (QP) to the systemic blood flow (Qs) depicted as QP/QS equal to 1.8:1. Pulmonary artery pressure (40/12/15 mm Hg) and pulmonary wedge pressure (mean 17 mmHg) was mildly elevated. Left Anterior Descending artery was the only affected vessel with proximal 90% stenosis (Type B;



**Figure 1:** 12 lead electrocardiography shows persistent ST segment elevation in V2-V4 even after thrombolysis as an evidence of septal aneurysm

Society of Coronary Angiography and Interventions (SCAI) [Figures 4 and 5]. Coronary angioplasty using a drug eluting (Endeavour Resolute 3 mm × 15 mm) stent was done with good angiographic result [Figure 6]. He was pain free and asymptomatic since then. The device closure was planned after 4 weeks; however had to be done earlier as patient was unwilling to stay longer than 3 weeks in hospital due to some domestic problem.

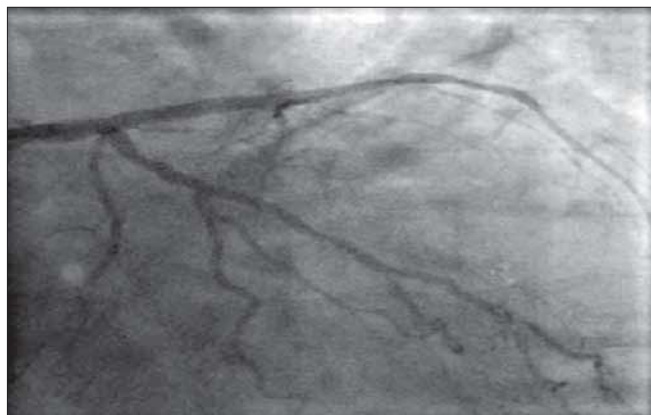
**Figure 3:** Left ventricular angiogram in left anterior oblique view shows single discrete muscular VSD of size 10mm



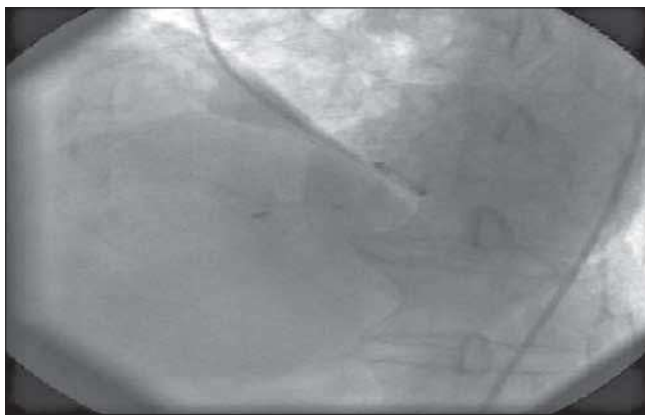
**Figure 4:** Selective coronary hooking of right coronary artery reveals normal right coronary artery

**Figure 2:** Echocardiography in apical four chamber view single and discrete muscular septal defect

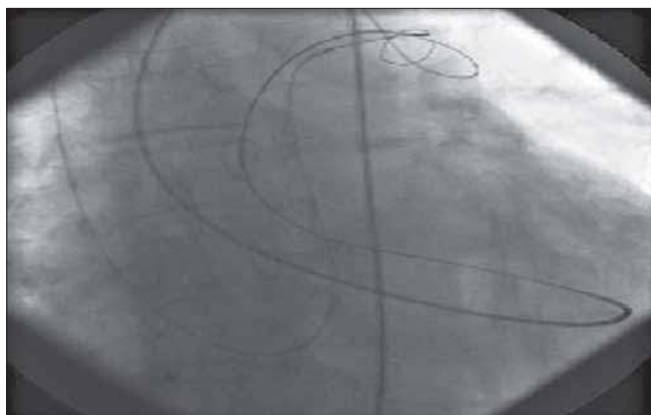
**Figure 5:** Selective left coronary injection shows type B proximal significant stenosis in left anterior descending artery



**Figure 6:** Successful angioplasty and stenting of left anterior descending artery with very good angiographic result



**Figure 8:** Appropriate placement of Cardio-Fix septal occluder across ventricular septal defect with tiny residual shunt is evident from left ventricular angiogram in left anterior oblique view



**Figure 7:** Successful snaring of regular guide wire from left pulmonary artery from right internal jugular venous approach



On day-23, the right femoral artery and right internal jugular vein were cannulated under local anesthesia. After a left ventricular angiogram, the ventricular septal defect was crossed using a 5 French right Judkin's (JR) catheter and a 0.035" × 260 cm angled tip Terumo wire (Terumo, Tokyo, Japan) using a retrograde arterial approach. It was passed into the right ventricle, to the main pulmonary artery and then into the left pulmonary artery. The right Judkin's catheter was advanced a little more into the right ventricular outflow tract (RVOT) and the Terumo wire was exchanged with a 0.035" × 260 cm Teflon e[change length wire (Medtronic). The distal end of that wire was then caught with a 10 mm Amplatzer goose-neck snare (Microvena, MN) in the left pulmonary artery [Figure 7], and extracted outside via the right internal jugular sheath. A 12 Fr 180 degree curve device delivery sheath with its dilator (Star Way Medical Technology, Inc) was advanced from the jugular vein into the left ventricle (LV) across the VSD. Then the dilator and the guide wire were removed. A 14 mm Cardio-O-Fix septal occluder was screwed onto the delivery cable, and introduced into the sheath. The distal disc (left ventricle) was initially opened and pulled back onto the LV side of

**Figure 9:** Two dimensional color Doppler showing septal occluder exactly across the ventricular septal defect after four days of procedure

the septum under trans-thoracic echocardiography (TTE) and fluoroscopic guidance. After confirming the septal alignment, the proximal disc (right ventricle) was also opened. Once adequate placement was ascertained by TTE and left ventricular angiogram, the device was released [Figure 8]. TTE and left ventricular angiography showed proper positioning of the device with only minimal shunting through the device. Patient remained hemodynamically stable after the procedure. The duration of the procedure was 35 minutes. TTE on the next day showed only a trivial residual shunt [Figure 9]. The patient was discharged on day 4 in a stable condition. At 4 month follow-up, he remained symptom free and had fair left ventricular function on echocardiogram.

## DISCUSSION

Ventricular septal defect (VSD) complicates acute myocardial infarction in about 1-2% cases in the first week.<sup>[1]</sup> Surgical closure had been the gold standard of this fatal complication.<sup>[2]</sup> As the mortality rates of surgical

closure remains high at 20 – 87% in acute stage, surgeons recommend a 3 – 4 week delay to allow scarring of the surrounding tissue to occur, which allows for better anchoring of sutures. Device closure of post infarction VSD is more studied in sub-acute and chronic stages and for residual shunts after surgical closure. Till today there are no guidelines available for device closure of ventricular septal defect after myocardial infarction. The small series and anecdotal reports highlighted that device closure in acute settings has a high mortality and more complications like device embolization, major shunting, left ventricular rupture and malignant arrhythmias.<sup>[7-10]</sup> The reported number of interventional post infarction VSD procedures till Nov 2009 were less than 200 in literature.<sup>[11]</sup> In a recent study, Demkow and colleagues described their 5 year experience with Amplatzer device with satisfactory results. For very severely ill patients, surgery may be a better option.<sup>[12-14]</sup>

The possible difficulties faced by intervention cardiologist

are I) lack of expertise due to the rarity of the procedure,

II) the rigid delivery sheath that has to cross the ventricular septal defect (VSD) and needs to be advanced into the direction of the left ventricular outflow tract, might tear the borders of the VSD, resulting in an increased VSD size or, in the worst case, left ventricular rupture III) The requirement of guide wire removal after insertion of the delivery sheath may result in kinking of sheath or jumping of device into right ventricle and IV) The currently available device sizes of the muscular VSD occluder not sufficient to close the large and complex VSD. Furthermore, healing of the infarcted myocardium over time may increase the size of the VSD leading to device malposition and embolization. This requires the use of devices that are larger than the measured VSD size at the time of implantation. The higher transventricular pressure for a VSD leads to persistent shunting until thrombus formation and endothelialization of the device has occurred. In contrast to surgical VSD closure, inferior and basal VSDs are more easily closed with an interventional technique due to the more favorable access from the upper jugular vein.

We critically analyzed all four variables: The hemodynamic status, coronary artery disease burden, VSD profile and financial aspects before adopting this strategy for ventricular defect closure after myocardial infarction. The timing of the procedure was critical. As the patient had angina and we found a significant critical lesion, we did angioplasty of the coronary artery lesion on fourth day. As the acute setting has potential for more complications, we staged the VSD closure to a later date. We had to take up the device closure a few days earlier than a desirable gap of

4 four weeks as the patient gave only conditional consent. Literature review showed only few anecdotal reports for device closure with Cardio-O-Fix VSD occluder which is used by us. This device is less expensive than commonly used ones. The use of this device in a case of post-operative residual VSD was recently presented in.<sup>[15]</sup> The Starway Cardio-O-Fix VSD Occluder is a self-expandable, double disc implant device made from a Nitinol wire mesh. The two discs are linked together by a short connecting waist corresponding to the size of the VSD. In order to increase its closing ability, the discs and the waist are filled with polyester fabric. The polyester fabric is securely sewn to each disc by surgical sutures. In our experience it is as user friendly as Amplatzer VSD occluder.

## CONCLUSION

In reasonably hemodynamically stable patient, with a favorable coronary anatomy and ventricular septal defect anatomy, an initial percutaneous revascularization of culprit lesion and elective device closure of VSD at a later date may make a safer strategy as alternative to surgery. This strategy also helps using local anesthesia instead of general anesthesia, TTE in place of trans-esophageal echocardiogram and Cardio-O-fix device as an used alternative to Amplatzer VSD occluder, hence adding to the safety and reduced cost of the whole procedure.

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