PRIMARY PERCUTANEOUS CLOSURE OF ACUTE VENTRICULAR SEPTAL RUPTURE COMPLICATING ACUTE MYOCARDIAL INFARCTION WITH SEVERE LV DYSFUNCTION

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ABSTRACT

Post myocardial infarct ventricular septal rupture is a devastating complication and leads to high mortality and in some situation may be a factor for inoperability. We are reporting a 57 years old patient who had acute anterior infarction and developed ventricular septal rupture. She had severe left ventricular dysfunction and primary surgical treatment was not possible due to high mortality. In due course her condition further deteriorated and developed multi organ failure. She was discussed in multi disciplinary meeting and decided to do the device closure of VSD as first stage and then later on CABG surgery. She underwent transcatheter closure of VSD and was managed medically. There was gradual improvement in her LV function as well as renal functions after the procedure. After two weeks of procedure she was discharged home with view to undergo CABG in next 6-8 weeks. This is first ever device closure of ischemic VSD at our institution as well as in Pakistan.

Keywords: Coronary artery bypass grafting, Myocardial infarction, Ventricular septal rupture.

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INTRODUCTION

Acute rupture of ventricular septal wall is devastating complication of acute myocardial infarction. The frequency of septal rupture is about 50% of ventricular wall rupture and occur mostly 3-5 days after acute infarction. The acute septal rupture carries significantly high mortality¹. The septal rupture occurs equally in anterior or non-anterior infarction2. In anterior infarction the site of rupture is apical septum³ as in our patient, the VSD was in apical area. The ischemic VSD may further deteriorate the condition patient characterized of by hypotension, biventricular failure and new onset murmur4.

CASE REPORT

A 57 year old woman was admitted with chest pain and angina CCS II-III and shortness of breath NYHA III along with dizziness. She was diagnosed as a case of large anterior myocardial infarction. She received thrombolytic and supportive treatment but the condition did not improve. On 5th day of admission she developed marked tachycardia,

Correspondence: Dr Nadeem Sadiq, Paeds Cardiology Department AFIC/NIHD, Rawalpini, Pakistan Email: drnadeemsadiq@yahoo.com hypotension and pansystolic murmur.

2-D Echocardiography was performed which showed dilated LV anteroseptal hypokinesia with apical half of akinesia along with aneurysmal formation of LV apical area. Left Ventricular ejection fraction was 25% and there was 5 mm muscular VSD at the junction of apical and middle third of inter-ventricular septum. Coronary angiography showed a critical proximal left anterior descending and moderate RCA artery stenosis. She was discussed in MDT (multidisciplinary team) meeting and as she not improving with any supportive medicines and was having severe LV dysfunction (25%), so decided not suitable for CABG and surgical VSD closure at present. The transcatheter VSD device closure was planned but her condition deteriorated and developed multi-organ failure with deranged function. Transoesophageal echocardiography (TOE) was performed which showed a 4.5 mm VSD. The inferior and posterior left ventricular walls contracted moderately but septum and apex were akinetic

Transcatheter ischemic VSD closure was planned under general anaesthesia. The left femoral artery was entered with 6 Fr Femoral sheath and right internal jugular vein with 6 Fr

Radial sheath. LV angiogram was done with Pigtail and VSD was identified measured 4.5 mm. Then VSD was crossed using a retrograde arterial approach with a 6 Fr Judkin Right catheter. Then terumo wire crossed the VSD and advanced to RV then to LPA and the end of this wire was then snared in the pulmonary artery with a goose-neck snare and then extruded from RIJV and thus creating an arterovenous guide wire loop. Intravenous heparin and ceftriaxone were administered. A 8 Fr long delivery sheath was advanced from the jugular vein into the left ventricle cavity. A 6 mm Shshma ventricular septal occluder was screwed onto the delivery cable, compressed into the loader, and introduced into the long sheath. The LV disc was extruded and pulled back onto the left ventricular side of the septum under TOE guidance. Once TOE confirmed septal alignment, the proximal ventricular) disc was deployed. The device was then released by counterclockwise rotation of the delivery wire. TOE and LV angiogram showed no residual leak through the device. Her symptom improved and tachycardia settled down and there was no murmur. She was discharged home after two weeks of procedure with improved LV functions. There was improvement in her symptoms (NYHA -II) and LV functions (EF 30%) after two months. She was now suitable for CABG surgery.

DISCUSSION

Early surgery improves the survival following acute rupture of ventricular septum but long term benefits depend on residual shunt and left ventricular function. However patients with inferior VSD and severe LV or RV dysfunction have high mortality with poor prognosis and surgery should be offered under exceptional cases⁵. Without surgery, the mortality reaches fifty percent during first week and less than 20% survive after one month. The early operative mortality for VSD repair remained high than surgery done after six weeks of acute MI6. The surgical management of Ischemic vsd including intra-Aortic balloon pumps (IABP) may be necessary in most cases. Anthanassiadi K et al during 10 year experience managed post infarct Ventricular Septum reconstruction with synthetic patch and infarctectomy, the mortality remained 50% due to heart failure and multi organ failure7. The recurrence of VSD after successful surgical repair in patients is 10-20% because of patch dehiscence, development of new VSD or overlooked second VSD8. The residual leaks following surgical repair of ischemic VSD may necessitates reoperation or sometimes may close spontaneously9. Transcatheter device closure of ischemic VSD is a well known modality of treatment and may help in hemodynamic stabilization of patient before urgent surgery, as an interim measure for the development of scar and strengthening the myocardium before definite surgery or a permanent alternate to primary or redo surgery^{10,12}. Primary transcatheter VSD closure in emergency may offer advantages over surgery or may be the only option as in our patient. She was discussed in multi disciplinary team meeting as she was having severe LV dysfunction with EF 25% and large aneurysmal akinesia of apex. The transcatheter device closure may be associated with a number of procedure related complications including major residual shunting, LV rupture and device embolization, as occurred in 41% in Thiele H et al who did primary transcatheter VSD closure in 29 patients over a period of 4.5 years¹³. Our case is of interest in that transcatheter device closure was the only option due to very high surgical mortality and unwillingness of surgical treatment by the patient as well. Apart from all risks this was our first ischemic Vsd device closure so was challenging. A number of device used for ischemic VSDs include Rashkind umbrella¹², clamshell double occluder¹¹, Amplazer occluders¹³ but we used a Shshma (Shangai shaped memory alloy- Chinese made) VSD occluder consisting of self expanding double disc made of nitinol mesh, containing three polyester patches. The two disc are connected by a waist corresponding to the size of septal defect.

Device migration remained the main complication of transcatheter closure and main causes include inexperience operator, inaccurate sizing/deployment, and inadequate rim and may result in device embolism or

encroachment on adjacent structures liketrapping of mitral or tricuspid chordae and encroachment upon the aortic valve.

Transcatheter closure is an established method of treating selected congenital defects¹¹. More experience is needed to assess its value as a primary closure technique or bridge to surgery in acute ventricular septal rupture.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

REFERENCES

- Figueras J, Alcalde O, Barrabés JA, Serra V, Alguersuari J, Cortadellas J et al. Changes in hospital mortality rates in 425 patients with acute STelevation myocardial infarction and cardiac rupture over a 30-year period. Circulation 2008 Dec 16;118(25):2783-9.
- Batts KP, Ackermann DM, Edwards WD. Postinfarction rupture of the left ventricular free wall: clinicopathologic correlates in 100 consecutive autopsy cases. Hum Pathol. 1990 May;21(5):530-5.
- 3. Pasternak, RC, Braunwald, E, Sobel, BE. Acute myocardial infarction. In: Heart Disease, 4th ed, Braunwald, EB (Ed), Saunders, Philadelphia 1992.

p.200.

- Sibal AK, Prasad S, Alison P, Nand P, Haydock D. Acute ischaemic ventricular septal defect--a formidable surgical challenge. Heart Lung Circ. 2010 Feb;19(2):71-4.
- Reeder GS. Identification and treatment of complications of myocardial infarction. Mayo Clin Proc. 1995 Sep;70(9):880-4.
- Daggett WM, Guyton RA, Mundth ED, Buckley MJ, McEnany MT, Gold HK et al. Surgery for post-myocardial infarct ventricular septal defect. Ann Surg. 1977 Sep;186(3):260-71.
- Athanassiadi K, Apostolakis E, Kalavrouziotis G, Charitos C, Karamichali E, Kakavos D et al. Surgical repair of postinfarction ventricular septal defect: 10-year experience. World J Surg. 1999 Jan;23(1):64-7.
- Caputo M, Wilde P, Angelini GD. Management of postinfarction ventricular septal defect. Br J Hosp Med. 1995 Dec 13-1996 Jan 16;54(11):562-6.
- Vogel JH, McFadden RB, Fisher GU, Jahnke EJ. Spontaneous closure of residual ventricular septal defect following surgical repair of ventricular septal defect complicating acute myocardial infarction. Adv Cardiol. 1980:71:343-5.
- Hachida M, Nakano H, Hirai M, Shi CY. Percutaneous transaortic closure of postinfarctional ventricular septal rupture. Ann Thorac Surg. 1991 Apr;51(4):655.
- 11.Landzberg MJ, Lock JE. Interventional catheter procedures used in congenital heart disease. Cardiol Clin. 1993 Nov;11(4):569-87.
- 12. Lock JE, Block PC, McKay RG, Baim DS, Keane JF. Transcatheter closure of ventricular septal defects. Circulation. 1988 Aug;78(2):361-8.
- Thiele H, Kaulfersch C, Daehnert I, Schoenauer M, Eitel I, Borger M, Schuler G. Immediate primary transcatheter closure of postinfarction ventricular septal defects. Eur Heart J. 2009 Jan;30(1):81-8.