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Management of Post Infarction Ventricular Septal Defect

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ABSTRACT

Ventricular septal rupture remains an infrequent but a serious, devastating and life threating complication of acute myocardial infarction. We report a case of 74 years old man with post infarction anterior ventricular septal rupture who was successfully managed. Ventricular septal rupture was closed by artificial (gortex) patch; concomitant coronary artery bypass grafting was done. Postoperative complications included reopening for excessive bleeding, low cardiac output with high inotropic support. A rapid diagnosis, aggressive medical management and prompt surgical intervention are required to optimize survival and recovery in patients who present with septal rupture complicating myocardial infarction.

Key Words: Ventricular septal rupture, Coronary artery bypass graf.

INTRODUCTION

In the pre-thrombolytic therapy era, ventricular septal rupture complicated approximately 1-3% of cases of acute myocardial infarctions.[1,2] Left ventricular wall rupture is the cause of death in approximately one third of patients who have a fatal acute myocardial infarction.[3,4] Most ventricular ruptures occur in the LV free wall, and these are usually fatal. In 15%-20% of cases, the rupture occurs in the interventricular septum, and this is also often fatal unless surgically treated.[5] The first successful repair was reported by Cooley and Colleagues in 1957.[6] Post myocardial infarction ventricular septal defect (VSD) occur usually within 2 weeks after infarction.[7] They results from perforation of the ventricular septum secondary to ischemic injury following acute myocardial infarction (AMI)[8] and in 60% of the cases are the results of completely occluded coronary artery supplying anterior wall of left ventricule and subsequent transmural myocardial infarction (MI).[7] When ventricular septal rupture complicates AMI, the mortality is high. Reperfusion therapy has reduced the incidence of septal rupture. A recent report suggests that coronary bypass at the time of septal repair does improve long term survival especially in patients with multivessel disease.[9] This case report describes a patient who underwent an operation for ventricular septal rupture (VSR) with coronary artery bypass grafting and successful management of multiple post operative complications.

CASE REPORT

A 74 years old normotensive, normoglycemic ex-smoker gentleman was admitted in our hospital with the complaints of right sided chest pain radiating to right shoulder and neck with generalized weakness for last 15 hours. He was referred from a local hospital.

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On admission his pulse rate was 64/min regular, respiratory rate 24/min; SpO_2 was 95%, and blood pressure 80/60 mm of Hg with Glasgo coma scale (GCS) 14. On ECG acute myocardial infarction (anterior & inferior) and biochemical investigation of blood revealed serum Troponin I 45.47 ng/l and CKMB 197 U/L. Echocardiography showed apex was dyskinetic to aneurysmatic, distal septum was akinetic and ejection fraction (EF) 35% with moderate pericardial effusion. Serum creatinine 1.83mg/dl, Serum urea 29 mg/dl. Liver function test includ ALT 200U/L and AST 629U/L, serum electrolytes were within normal limit. On chest examination bilateral crepitation was present in both lung fields. He was treated with dopamine and frusemide intravenously and clopidegrol, atorvastatin and glyceryl trinitrate orally. Central Venous Pressure (CVP) line and urinary catheter was introduced.

On the next day there was a rise of body temperature with the complaints of chest pain. Inotropic support (dopamine infusion) withdrawn, inj. low molecular weight heparin started and planned for coronary angiogram (CAG). At evening still there was rise of body temperature, urinary catheter was taken out and sample from CVP line was sent for culture and sensitivity (C/S). CVP line was removed and CAG was postponed due to high temperature. One unit of packed cell was transfused due to low haemoglobin concentration. Blood pressure lowers down and dopamine support was started through peripheral intravenous (IV) line again.

On fifth day of admission under the ionotropic support through peripheral intravenous line (inj. dopamine 5 µgm/kg/min) repeat Echo showed moderate pericardial effusion with regional wall motion abnormality, ejection fraction was 42% and colour mapping showed ventricular septal rupture (VSR) close to apical region with left to right shunt. All valves were normal in morphology and function. Coronary angiogram was done on same day and revealed left main stem tapering with mild plaque (Fig: 1), 100% occlusion of left anterior descending (LAD) artery (Fig:2), 80% lesion in left circumflex artery and 100% occlusion of right coronary artery (RCA) (Fig:3).

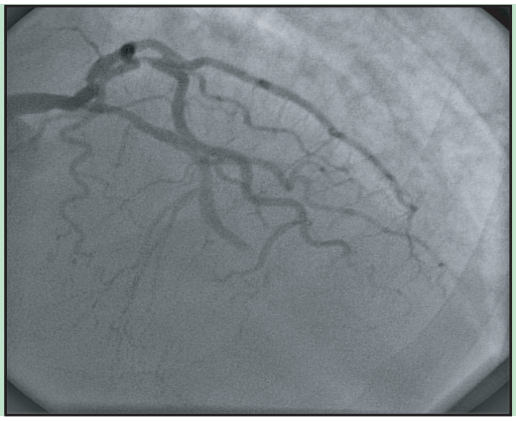

FIGURE 1 : *LMS (Left Main Stem) tapering with mild plaque*

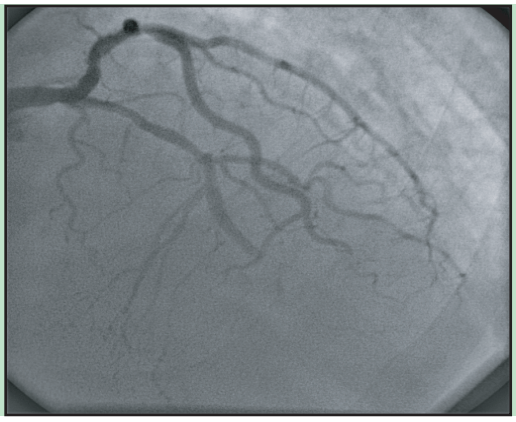

FIGURE 2 : *LAD (Left Anterior Descending) artery 100% occlusion*

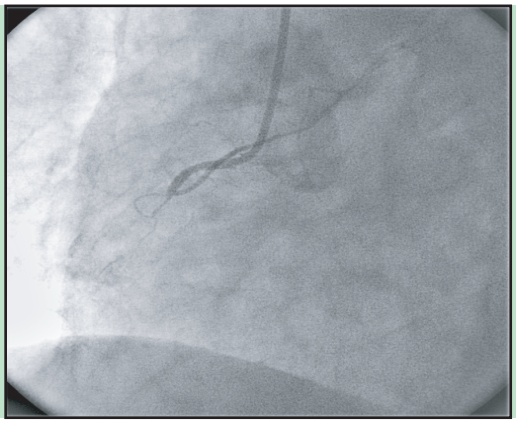

FIGURE 3 : *RCA (Right Coronary Artery) 100% occlusion*

On seventh day after admission this patient underwent coronary artery bypass surgery (CABG) and surgical closure of VSD. After median sternotomy haemopericardium was found. Apex and anterolateral wall were infracted and akinetic. Standard cardiopulmonary bypass (CPB) was established with aortic and bicaval venous cannulation. Body cooled down to 30°C, aorta cross clamped and heart was arrested by antegrade cold blood cardioplegia with topical cooling. An incision was started near the left ventricle (LV) apex 1-2 cm from and parallel to the left anterior descending coronary artery and carried right through the infracted muscle. The VSD and the margins of the infracted muscle were identified. A gortex patch was tailored according to the LV infarction and sutured to the lower part of the non-infracted endocardium of the interventriculum septum with a continuous 3/0 polyprolene suture. The patch was sutured to the non infracted endocardium of the anterolateral ventricular wall. Once the patch was completely secured to the LV endocardium, the infracted myocardium became largely excluded from the LV cavity. The ventriculotomy was simply closed over two strips of Teflon felt.

Coronary artery bypass grafting was done to LAD (left anterior descending), OM1 (obtuse marginal), OM2 and RCA (right coronary artery) was non graftable. Patient was weaned from CPB slowly and smoothly without any event. The patient shifted in ICU in stable condition with minimum inotropic support. Reopening was done after three and half hours due to excessive bleeding. Total 850 ml blood came out from the chest cavity. Still there was oozing of blood and no specific bleeding point was found. Chest was kept open by covering with ioban and patient shifted to ICU with high inotropic support. Two units of fresh whole blood and four units of fresh frozen plasma (FFP) were transfused on 1st postoperative day (POD) day and patient's chest was still kept open and remained on mechanical ventilation. On 2nd POD he was stable haemodyamically with moderate inotropic support. Patient's chest was closed on 3rd POD and extubated on the same day. His drain tubes were removed also on same day. On 8th POD he was shifted to general ward from ICU step down. His recovery in the ward was uneventful and he was discharged from hospital on 14th POD.

During post operative follow up (two weeks after discharge) patient was stable haemodynamically, there was no added sound at pericardium, ECG revealed sinus rhythm and on echocardiography there was no VSD.

DISCUSSION

Though ventricular septum rupture is a complicated case and mortality high after surgery; it is the only realistic option of treatment we have because non surgical treatment is generally accepted to be inadequate.[10,11] Despite advances in surgical care, operative mortality is still considerable, approaching 50% with the major risk factors being cardiogenic shock, renal failure, right ventricular and or left ventricular failure, posterior inferior location and residual VSD.[12]

We successfully managed our case though initially patient needed ionotropic support to over come cardiogenic shock. The question of whether to perform bypass grafts at the same time as the VSD closure has remained largely unresolved over recent years. The safety of coronary angiography in these unstable patients has been a concern. It has been shown that up to 4.5% of these patients can deteriorate haemodynamically during cathetarizatoin.[13] In contrast, Labrousse et al. demonstrated angiography to be safe in their cohort[12] study. In our experience coronary angiography did not adversely affect the clinical state of the patients.

The traditional operative techniques for patients with post MI-VSD consist of infarctectomy and reconstruction of the LV and RV walls with a Dacron fabric graft. These procedures were associated with increased LV and RV dysfunction and consequently high operative mortality.[14] We used here gortex patch with infarct exclusion which is physiologically sound and many authors

reported enhanced operative survival because it leaves the right ventricle undisturbed and restores LV geometry and operative mortality is 13.5%.[15]

Patients with post infarction septal rupture should be operated immediately if conservative attempts failed to stabilize haemodynamics. Decision on terms of surgical intervention should be made individually for each patient. Patch closure of the ventricular septal rupture or exclusion of myocardial infarction, reconstruction of the left ventricle and selective myocardial revascularization provide acceptable results.

REFERENCES

1. Topaz O, Taylor AL. Interventricularseptal rupture complicating acute myocardial infarction: from pathophysiologic features to the role of invasive and non invasive diagnostic modalities in current management. *Am J Med* 1992;93:683-8.
2. Moor CA, Nygaard TW, Kaiser DL, Cooper AA, Gibson RS. Postinfarction ventricular septal rupture: the importance of location of infarction and right ventricular function in determining survival. *Circulation* 1986;74:45-55.
3. Feneley MP, Chang VP, O'Rourke MF. Myocardial rupture after acute myocardial infarction. Ten year review. *Br heart J* 1983;49:550-6.
4. Honan MB, Harrell FE Jr, Reimer KA,Califf RM, Mark DB,Pryor DB, Hlatky .Cardiac rupture, mortality and timing of thrombolytic therapy; a meta-analysis. *J Am Coll Cardiol* 1990;16:359-67.
5. Radford M J, Johnson RA, Daggett WM Jr, Fallon JT, Buckley MJ, Gold HK, Leinbach RC. Ventricular septal rupture: a review of clinical and physiological features and analysis of survival. *Circulation* 1981;64:545-53.
6. Cooley DA, Belmonte BA, Zeis LB, Schnur S. Surgical repair of ruptured interventricular septum following acute myocardial infarction. *Surgery* 1957;41:930-37.
7. Juraszynski ZJ, Kotlarska W, Drewniak K, Kotlinski K, Hunka I, Borys M, Dabrowski M. Surgical treatment of interventricular septum rupture in an 87-year-old female with acute antero-lateral myocardial infarction and 97% risk according to the Euroscore scale. Kardiol. *Pol* 2006;64(3):297-99.
8. Yonga GO, Munene JC, Ogendo SW. Post-infarction ventricular septal defect in Nairobi: Case report. *East Afr Med J* 2005;82(12):660-62.
9. Muehrcke DD, Daggett WM Jr, Buckley MJ, Akins CW, Hilgenberg AD, Austen WG.Postinfarct ventricular septal defect-can we do better? *Ann Thorac Surg* 1992;54:876-82.
10. Crenshaw BS, Granger CB, Birnbaum Y, Pieper KS, Morris DC, Kleiman NS, Vahanian A, Califf RM, Topol EJ.Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. *Circulation* 2000; 101:27-32.
11. Held AC, Cole PL, Lipton B, Gore JM, Antman EM, Hockman JS, Corrao J, Goldberg RJ, Alpert JS. Rupture of the interventricular septum complicating acute myocardial infarction: a multicentre analysis of clinical findings and out come. *Am Heart J* 1988;110:1330-36.
12. Labrousse L, Choukroun E, Chevalier JM, Madonna F, Robertie F, Merlico F, Coste P, Deville C. Surgery for post infarction ventricular septal defect (VSD): risk factors for hospital death and long term results. *Eur J Cardiothorac Surg* 2002;21:725-31.
13. Cox FF, Plokker HW, Morshuis WJ, Kelder JC, Vermeulen FE. Importance of coronary revascularization for late survival after postinfarction ventricular septal rupture. *Eur Heart J* 1996;17:1841-45.
14. Giuliani ER, Danielson GK, Pluth JR, Odyniec NA, Wallace RB.Postinfarction ventricular septal rupture: surgical considerations and results. *Circulation* 1974;49:455-59.
15. David TE, Dale l, Sun Z. Post infarction ventricular septal rupture: repair by endocardial patch with infarct exclusion. *J Thorac Cardiovasc Surg* 1995; 110:1315-22.