POST MYOCARDIAL INFARCTION VENTRICULAR SEPTAL RUPTURE REPAIR VIA TRICUSPID VALVE

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ABSTRACT
We report a case of post myocardial infarction ventricular septal defect in anteroinferior interventricular septum, which was repaired via tricuspid valve. We believe this approach saves incision in left ventricle which helps preserve the function of left ventricle after the surgery and it should be adopted in suitable patients.

KEY WORDS: Post myocardial infarction; ventricular septal defect; and repair.

INTRODUCTION
After the use of thrombolytic and percutaneous coronary intervention the incidence of post myocardial infarction ventricular septal rupture (VSR) has reduced to 0.17-0.31% which was as high as 1-3%.1-4 Regardless of all these improvements the mortality rate of VSR still remains drastically high 45-80%.6-9 According to the studies done by GUSTO-I and AMI6-8,10 easy access to echo and the effect of reperfusion injury and fibrinolysis on tissue pathology can be adding factors for early detection of VSR. No matter how much improvement in non-surgical treatment, surgical repair of Post MI VSR remains the gold standard. Percutaneous intervention is effective option in selected cases. According to the current guidelines of American College of Cardiology Foundation and American Heart Association (ACCF/AHA) surgical repair should be done at time of diagnosis. The exact timing, approach and perioperative therapeutic management of VSR is still a moot point.

CASE REPORT
We report a case of patient presented to us with diagnosis of post myocardial infarction ventricular septal rupture (Post MI-VSR). He had myocardial infarction a week before and he was managed conservatively in cardiology department. His echo showed 1cm ventricular septal rupture (VSR) located over anteroinferior segment of interventricular septum. Angiography showed left anterior descending artery having 80% stenosis. After appropriate investigation, blood arrangement and optimization of inflammatory markers he was operated upon. After general anesthesia arterial and venous lines were inserted. Patient was cleaned and drapped, median sternotomy was done and pericardium opened, after full heparinization. Ascending aorta was cannulated, superior vena cava and inferior vena cava were cannulated and snugged. Antegrade cardioplegia was given and cold saline poured in pericardium. Vent was placed in left atrium. Right atrium was opened and tricuspid valve located. VSR was identified in the anterior inferior position in interventricular septum. After size and anatomy of VSR was defined a Dacron patch was secured over VSR with pludgeted 3/0 prolene, taking healthy margins of the septum. Right atrium was then closed and patient was taken off bypass in conventional pattern. Hemostasis was then secured and wound was closed after placing mediastinal drain and pacing wire. Patient was shifted to ICU where he was kept on ventilator for 4 hours. Invasive BP, ABG, Urine output and ECG were monitored. After optimizing the parameters, he was extubated. Mediastinal drain, after being dry for 5 consecutive hours was removed on next day. He was mobilized, orally allowed and shifted to ward on second post operative day. He was discharged home on 6th day. He had post operative visit at seventh day and one month. ECHO showed no residual VSR at both visits.
RESTAURANT

In 1934 Sager described the association of post myocardial infarction with VSR. Historically, up to 5% of myocardial infarction were associated with mechanical complication such as free-wall rupture, papillary muscle rupture and Post MI-VSR which have now reduced to 0.2% due to thrombolytic therapy and early PCI. If these interventions are delayed then resulting increase in myocardial damage rises up to 2%. In 2008 Poulsen explained the 5% of early death (usually 2-4 days but varying from a few hours to several weeks) after AMI are directly related to Post MI-VSR. Risk factors include current smoking history, increasing age and male gender (3:2). The mean age of presentation in GUSTO was 62.5 years and ranged from 44 to 81 years.

Patients often present with complain of recurrent chest pain which recovers from initial management of an uncomplicated AMI. With an acute clinical deterioration a rapid assessment of etiology is critical. To reach the final diagnosis following investigations need to be done. Echocardiography when done along with Doppler is 100% sensitive and specific in the diagnosis of Post MI-VSR. In cardiac catheterization left anterior descending artery is most likely involved leading to anteroapical septal VSD in 60% of the cases. Adding to single vessel disease found in 64% of the cases, 7% has concomitant double vessel disease while remaining 29% has TVD.

In general 25% of patients with Post MI-VSR die in first 24 hours, for those who survive the acute event, 1, 2 and 4 week survival is 50%, 35% and 20% respectively. Prolonged untreated survival has been reported with up to 7% of patients surviving to 1 year. In ACSD study, early repair occurring less than 7 days post MI had much greater mortality than delayed repair occurring more than 7 days post MI.

The initial attempt at repairing Post MI-VSR was through a ventriculotomy in the right ventricular outflow tract. Than due to animal studies anterior defects were approached through anterior wall while posterior defects through the inferior. Repair of apical defects involves excising the apical defect and bringing together the residual edges of the left and right ventricular walls using a primary repair reinforced with pledges. In anterior defect most anterior septal infarcts require repair with a patch excluding the necrotic septum from the higher pressure left ventricular cavity. Repair of the posterior defect is similar to anterior repair but the incision is along the distal right coronary and parallel to posterior descending artery through the infarcted basal muscle. The overall predictors of post operatives mortality includes: shock at time of surgery, clinical deterioration while awaiting surgery, need for concomitant CABG and pre operative renal failure (as a marker for shock and organ failure).

REFERENCES
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Authors declare no conflict of interest.

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