

CASE REPORT

Transcatheter Closure of Ventricular Septal Rupture with Swan Ganz Balloon

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INTRODUCTION

Sudden occlusion of the coronary artery results in ischemia, myocardial cell death and myocardial infarction(MI). The most common cause is atherosclerotic plaque rupture. MI can be complicated by arrhythmia such as ventricular tachycardia, heart block, heart failure, ventricular wall rupture and death.

CASE REPORT

A 47 year old lady had an acute severe episode of retrosternal chest pain, radiating to left arm and was diagnosed to have anteroseptal MI in a district hospital. The only cardiovascular risk factor was hypertension. Her vital signs were stable, Killips Class I. Thrombolysis with streptokinase and double antiplatelets acetylsalicylate 300mg and clopidogrel 300mg stat were administered. As her chest pain and ST elevation in the ECG persisted after the completion of the thrombolysis, she was transferred to a nearby tertiary hospital with cardiac facility the next morning with the presumption of failed thrombolysis.

At the emergency department of the tertiary hospital, her blood pressure(BP) was 96/75mmHg, heart rate 93/min, she was in acute pulmonary edema and Killips Class IV. She was started on inotrope Dobutamine and vasopressor Dopamine. A new systolic murmur was noted with a presumptive diagnosis of acute MI complicated by VSR.

ECHO revealed a muscular site of the VSR, left to right shunt by colour flow doppler, moderate size with no mitral regurgitation. The apical and septal segments were hypokinetic/akinetic with ejection fraction about 25-35%. The maximum cardiac enzymes measurements on day 2 of MI were CK-MB 204(U/L), CPK 3210(U/L), LDH 655(U/L) and AST 381(U/L).

She was electively intubated and ventilated to facilitate invasive catheterisation procedures. A coronary angiogram(COROS) was done which showed triple vessels disease with normal left main stem, 100% occluded proximal left anterior descending artery(LAD), 80% stenosis at mid circumflex artery and 70% stenosis mid right coronary artery(RCA). Left ventricular gram using 5FR pigtail showed the presence of VSR. Intra-aortic balloon pump(IABP) was inserted for hemodynamic support.

The in house surgeon was not keen to operate immediately in this unstable high risk patient. Thus, we embarked on the

transcatheter intervention options. Initially, an Amplatzer closure device was used to occlude the VSR but failed. Then, we attempted to engage a 7FR Swan Ganz catheter via the femoral vein. When the inflated Swan Ganz balloon occluded the VSR spot, systolic blood pressure(SBP) improved from 60 to 95 mmHg.

Post catheterisation, IV heparin was started for the in-situ IABP. Her BP remained relatively constant about SBP 90mmHg on inotrope, vasopressor and IABP support. However, in the early hours of day 3 post MI, she developed supraventricular tachycardia, bradycardic episodes and eventually periods of asystole which didn't respond to cardiopulmonary resuscitation efforts and she died.

DISCUSSION

Acute MI is a result of sudden significant thrombotic occlusion of the coronary artery system. Survival rate of MI has increased with the use of thrombolytic agents. If a patient deteriorates suddenly and goes into cardiogenic shock, the suspicion of free ventricular wall rupture, VSR and papillary muscles rupture must be considered.

It is found that female gender and age more than 60 years predispose to VSR occurrence. Most commonly VSR occurs on day 3 to day 5 of MI but it can also occur early on day 1 to day 3 of MI. Thus, the clinician should be alert to the possibility of VSR in the presence of hemodynamic instability and new precordial systolic murmur. This will prompt immediate appropriate investigation and treatment. Chest XRay may show pulmonary edema and the cardiac size may remain the same in acute setting. Transthoracic ECHO(TTE) is useful to look for anatomical defect, shunts, regional wall motion abnormality, ejection fraction, Qp:Qs, mitral regurgitation and pericardial effusion. This patient's TTE could clearly demonstrate the VSR with left to right shunt and obviated the need for transesophageal ECHO. Qp:Qs parameters were not obtained in this case due to time constrain and patient's unstable condition.

There would be imbalances in the pulmonary to systemic blood flow (Qp-to-Qs) which exclude the use of vasodilators but necessitate the use of inotrope and vasopressor at the expense of more myocardial work and oxygen consumption. Despite commencing dopamine and dobutamine, our patient's hemodynamic status didn't improve much. IABP normally improves the coronary blood flow during the diastolic phase augmentation, decreases afterload and

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increases the blood pressure. Usually IABP is inserted prior to any coronary catheterisation procedure in unstable patient.

The main culprit vessel was the occluded LAD with no collaterals, leading to necrosis of its distribution area, the ventricular septum in this case. As the necrotic muscles present a challenge to the surgeon to place a patch with good suture anchoring, some surgeons prefer to wait 1-2 weeks for more scar tissue to form before surgery is contemplated as emergency surgery carries high perioperative mortality risk of 19-58%^{1,2}.

Hence, the dilemma. Faced with a reluctant surgeon amidst a very grim clinical scenario, we needed some form of transcatheter intervention to augment and mitigate her precarious physiologic parameters.

Certain centers have attempted to use closure devices such as the Amplatzer devices commonly used in congenital septal defects closure and the Swan Ganz balloon for permanent or temporary occlusion of the VSR^{1,3}. We initially tried to use an Amplatzer closure device to close it without success. We then improvised with a Swan Ganz catheter by placing the balloon at the VSR site and successfully deployed the inflated balloon. The SBP improved almost immediately. The placement of the Swan Ganz balloon was intended as a bridge to definitive surgery and buy more time while the patient was being medically optimised.

When the patient was becoming more stable hemodynamically for VSR surgery, the surgeon would normally perform concomitant coronary artery bypass

grafting(CABG). Unfortunately our patient passed away before the operation. We can only postulate that the necrotic ventricular septal area had expanded, hence loosening the Swan Ganz balloon or fatal arrhythmias had led to her death. Unfortunately, the placement of the Swan Ganz balloon at the VSR site couldn't buy her more time in a high risk situation for her surgery.

CONCLUSION

There must be high level of clinical suspicion for VSR to allow early detection and prompt management. Beside the conventional surgical repair of VSR, there's a trend towards non-surgical usage of transcatheter devices such as Amplatzer to close it percutaneously⁴. Here we expand the option of temporary closure of VSR using Swan Ganz balloon as a bridge to surgery in high risk category of patients although early surgery is still the best option.

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