HISTORY AND PHYSICAL
This is a case of a 64 years old female, newly diagnosed with diabetes mellitus, who came in due to chest pain. Two months prior to admission, patient had sudden onset of severe chest pain. She was subsequently admitted at the National Kidney and Transplant Institute and managed as a case of Acute Coronary Syndrome. During this admission, the patient had episode shortness of breath associated with orthopnea and exertional dyspnea. Two-dimensional echocardiography was done which revealed ejection function of 62 – 69 %, concentric left ventricular hypertrophy with hypokinesia of the left ventricular apex with suspicious flow across the muscular interventricular septum indicating a ventricular septal rupture shunt. The patient was stabilized and was discharged improved.
Five days prior to admission, she was noted to have progressive bipedal edema. She was then readmitted at the NKTI. One day prior to admission, she was noted to have shortness of breath along with epigastric pain and vomiting. The patient was transferred to our institution for intervention.
On physical examination, the patient had stable vital signs, not in distress and ambulatory. Neck veins were distended. On chest examination, there was no lagging and retractions but with crackles on bilateral mid to lower lung field. The patient had adynamic precordium, point of maximal impulse at the 5th intercostal space left mid clavicular line, with thrill at the left parasternal area, no heave, S1 normal, S2 split, normal rate, regular rhythm, grade 4/6 holosystolic murmur over left parasternal and apical area. The abdomen was soft, not distended, no ascites but with palpable liver edge 3 cm below subcostal margin. The lower extremities showed grade II pitting bipedal edema with full pulses.
The assessment on admission was Atherosclerotic Heart Disease, Coronary artery disease, s/p Acute coronary syndrome (March 2016), Ventricular septal rupture, Congestive Heart failure, NYHA Functional classification II-III, Diabetes Mellitus type 2.
On admission, the patient was worked-up. She underwent coronary angiography with noted one vessel disease (LAD). On LV angiogram, contrast injection showed passage of dye from LV to RV through the muscular part of the interventricular septum. On the 6th day, the patient underwent ventricular septal rupture device closure using VSD occluder size 17/10. The patient was able to tolerate the procedure well. On the 18th hospital day, the patient then underwent PCI.
IMAGING
INDICATION OF INTERVENTION

The indication for closure of an interventricular septal defect after acute myocardial infarction causing hemodynamic compromise with evidence of loud holosystolic murmur and left ventricular dysfunction with lower extremities edema is warranted in our patient. The prognosis of post-AMI VSD is very poor, with mortality rates as high as 50% at 1 week and 90% at 2 months with conservative medical management. The patient had a history of AMI 2 months prior to admission and surgery is technically difficult owing to the myocardial tissue being soft and friable. Percutaneous closure device closure is a viable option in chronic period in patients with comorbidities and whose septal anatomy is favourable to device placement. The patient presented with ventricular dysfunction, with history of diabetes which put her to a high risk candidate for surgery. The septal anatomy of the ventricular rupture of the patient was at the muscular area, which is the only area recommended by the American Heart Association for device closure.

INTERVENTION

The patient underwent device closure of the muscular ventricular septal rupture on the 6th hospital day of admission. Left heart catheterization was performed via the right femoral artery percutaneous puncture and a French 6 sheath was inserted. A French 6 pigtail catheter was manipulated under fluoroscopic guidance into the descending aorta, ascending aorta and to the LV. LV angiography at LAO 35, cranial 35 showed a muscular ventricular septal defect. Right heart catheterization was performed through a right femoral vein percutaneous puncture. A French 6 sheath was inserted via the right femoral vein. A French 6 multi-snare catheter was then inserted and manipulated under fluoroscopic guidance into the IVC, RA and RV. An arteriovenous guide wire splint was then created. The long Terumo guide wire 032 x 260 mm from the LV was manipulated thru the VSD to the RV, RA and to the IVC. The sheath was carefully advanced until its tip was placed in the ascending aorta. As soon as the sheath reached the ascending aorta, the arterial catheter was replaced via
the guide wire with a pigtail catheter. The terumo guide wire was then pulled out by the snare to the right femoral vein. The introducer set was attached to the guide wire and pulled back to the IVC, RA, and RV thru the VSD and to the LV. A 6/8 mm VSD device occluder was then placed to occlude the VSD. Cineangiography post occlusion of VSD showed the device positioned within the VSD, with minimal shunting of contrast in the center of the device.

**LEARNING POINTS OF THE PROCEDURE**

This is a novel case of percutaneous device closure of ventricular septal rupture post-MI in our institution. In a study done by Demkow *et al*, transcatheter closure has improved survival rates in selected patients in suitable anatomy. One of the challenges among interventional cardiologist is the margins of defect wherein the borders on may be necrotic and the poor clinical condition of the patient on presentation. According to Bialkowski *et al*, procedure failures were observed in acute post-MI VSD and satisfactory in subacute and chronic phase cases which was seen in our case. Therefore, proper patient selection should be done in order to have a favorable outcome.