HISTORY AND PHYSICAL
This is a case of a 45 year old female who came in due to easy fatigability. Patient was a diagnosed case of rheumatic heart disease since 1980 but lost to follow up. She had a history of repeated admission for one year prior to admission due to exertional dyspnea and congestion. Two months prior to admission patient was seen at the outpatient department and was noted to have difficulty of breathing, easy fatigability and bibasal rales hence she was admitted and started on medical management. She was discharged in an improved state with plan for interventional procedure. Patient was discharged as a case of rheumatic heart disease, mitral stenosis, atrial fibrillation in chronic ventricular response, NY functional classification II – III. Patient was re-admitted as a case of Lutembacher Syndrome, Atrial Septal Defect secundum type and Rheumatic Heart Disease with severe Mitral Stenosis two months after for interventional procedure.

On physical examination, the patient had stable vital signs with blood pressure of 110/60 mmHg, cardiac rate of 76 beats per minute, not in distress and ambulatory. No neck vein distention nor palpable masses noted. On chest examination, there was a symmetrical expansion with no crackles and wheezing. The patient had adynamic precordium, point of maximal impulse at the 5th intercostal space left intercostal space, no thrill, no heave, S1 normal, S2 split, normal rate, regular rhythm, grade 3/6 diastolic murmur at the apex. The abdomen was soft, not distended, no ascites. There were no cyanosis, no edema with good capillary refill time at < 3 seconds.
**INDICATION OF INTERVENTION**

The incidence of ASD in patients with mitral stenosis is 0.6-0.7% and the incidence of MS in patients with ASD is 4%. Lutembacher's syndrome is defined as the rare combination of congenital atrial septal defect and acquired mitral stenosis. The haemodynamic effects of this syndrome are a result of the interplay between the relative effects of the atrial septal defect and mitral stenosis. Mitral stenosis augments the left to right shunt through the atrial septal defect. Because the mitral stenosis was, in fact, rheumatic in aetiology, the syndrome was defined eventually as a combination of congenital atrial septal defect and acquired, almost always rheumatic, mitral stenosis. Percutaneous transcatheter therapy has become the most widely accepted therapy, using balloon mitral valvuloplasty for mitral stenosis and the amplatz atrial septal occluder for closure of an atrial septal defect. In a study of Uguro SU, et al, percutaneous correction is preferred to surgical correction as there is decreased morbidity compared to open-heart surgery. There is also faster recovery with decreased
length of hospital stay. Indications for percutaneous intervention in these patients include severe mitral stenosis causing hemodynamic instability that causes recurrent admission for the patients.

INTERVENTION

The patient underwent pre - percutaneous mitral commissurotomy transesophageal echocardiogram which showed a mitral valve area of 0.41 cm² by planimetry with mild mitral regurgitation. Left heart catheterization was done percutaneously using a 6 french pigtail catheter through a left femoral arterial sheath. The catheter was retrogradely advanced to the ascending aorta, aortic valve, and left ventricle. Pre-percutaneous mitral commissurotomy and atrial septal defect closure pressure recordings showed a low systemic arterial pressure of 89/60 mmHg and elevated LVEDP (17 mmHg). The PA pressure was elevated at 77/28 mmHg with no systolic pressure gradient across the pulmonic valve during pullback. The transmitral mean gradient is 10 mmHg. The Mullins catheter was directly inserted into the LA thru the interatrial septal defect and the coiled guidewire was inserted through the catheter into the left atrium. A size 26 mm Mitrapath balloon catheter was inserted over the guidewire and maneuvered sequentially across the interatrial septum and the mitral valve with the aid of a stylet. The balloon was inflated thrice at 23, 24 and 26 mm. The final gradient across the mitral valve was at 6.3 mmHg. Right heart catheterization was done percutaneously using a 7 french Counard catheter through a right transfemoral venous sheath and was advanced anterogradely into the RA, RV, PA and LA through the ASD. An Amplatz stiff wire was introduced as an exchange wire through the Counard catheter and advanced into the right atrium and through the atrial septal defect into the left atrium with the tip extending beyond the level of the left upper pulmonary vein. The femoral vein sheath was then removed. The introducing sheath with the dilator was advanced over the exchanged wire into the left atrium and was positioned at the left atrium border while the dilator was removed. The delivery cable was passed through the loader and An ASD occluder was screwed clockwise onto the tip of the delivery cable. The device and the loader were then immersed in a saline solution and the occluder was pulled into the loader. The loader was introduced into the delivery sheath and was advanced into the left atrium. The retention skirt was deployed and pulled firmly against the interatrial defect. The position of the device was confirmed through transesophageal echocardiography. The device was adjusted until the retention skirt was well seated in the interatrial septum. The device was then released from the delivery cable and the system removed. Post PTMC intra-procedural 2D echocardiogram showed a mitral valve area of 1.0 cm² by planimetry.

LEARNING POINTS OF THE PROCEDURE
Percutaneous transcatheter closure of ASD and mitral valvotomy is the treatment of choice for Lutembacher syndrome until and unless the lesions are incongruous for the procedures. The patient was symptomatic with moderate to severe mitral stenosis with valve morphology favorable for PBMV.