History

• A 19-year-old woman presented with dyspnea and fatigue.
• There was mild cyanosis with physical signs consistent with mitral stenosis and pulmonary hypertension.
• History suggestive of rheumatic fever.
• Echocardiography was done and confirmed severe mitral stenosis (mitral valve area was 1.02 cm²).
• Mean pressure gradient was 13.9 mmHg.
• Grade I mitral regurgitation.
• Mitral valve score was 7/16.

MVA = 1.02 cm²
• Percutaneous balloon mitral valvuloplasty (PBMV) was planned after complete transesophageal echocardiographic (TEE) assessment (clear both left atrium and left atrial appendage).
• PBMV was successfully performed utilizing a multitrack balloon technique with a post-procedure MV area of 2.4 cm² and mean PG of 6.2 mmHg.

• Two years after successful percutaneous balloon mitral valvuloplasty, the patient developed symptoms of pulmonary congestion and palpitation.
• A prominent continuous murmur was auscultated, and echocardiography demonstrated a sizable PDA (4.9 mm) with left-to-right shunting.
• The patient was taken to the cardiac catheterization laboratory where hemodynamic evaluation revealed moderate pulmonary hypertension (pulmonary arterial pressure = 69/21 m = 39 mmHg).
• A significant L-R shunt was observed across a sizable PDA (5.1mm).
We decided to close the PDA percutaneously

• The PDA was crossed from an antegrade approach with a 0.035" Amplatz extra-stiff interventional guide wire (Meditech, Watertown, Mass.) positioned with the tip in the descending aorta.
• An 8F Mullins sheath was advanced over the wire and positioned across the PDA into the descending aorta.
• A 10/8 Amplatzer device was implanted through the sheath and retained on the delivery catheter until injection of a contrast confirming proper positioning with cessation of PDA flow.

• The patient was discharged within 24 h.
• Symptoms improved rapidly, and at 1-year follow-up repeat physical examination and echocardiography confirmed complete ductal occlusion with no residual shunt.
• Although there was no definitive evidence of prior streptococcal infection, the clinical profile and the echocardiographic findings suggest an acquired rheumatic etiology affecting mitral valve in our patient.

• Congenital mitral stenosis can present with some leaflet thickening and commissural fusion; and can occur in association with other congenital heart diseases.

• 5% of symptomatic infants with isolated congenital MS are known to die within the first six months of life.

• The late survival of our patient in the presence of a major associated intra-cardiac lesion makes congenital MS unlikely.

• About 50% of patients with rheumatic heart disease may not have a prior history of rheumatic fever.

• Recurrent subclinical active carditis leading to late mitral stenosis may occur in the natural history.
These arguments favor a rheumatic etiology for the mitral stenosis in our case

PDA was missed... Why?
• PDA is a relatively common lesion in females and may be particularly difficult to diagnose in the setting of increased pulmonary arterial resistance when a continuous murmur and/or typical color Doppler flow findings may be absent.

• This was our case where PAP increased as a result of MS & when the PAP decreased after PBMV, the PDA murmur was re-auscultated.

• In such patients device closure is an effective tool for non-surgical closure of moderate-sized PDA and should be considered in the therapeutic decision making for any patient with this lesion, regardless of age.
• This report draws attention to an interesting association between rheumatic mitral stenosis and PDA.
• We should be aware of complex association between congenital and acquired structural heart diseases.
Eisenmenger syndrome could be also a presentation of severe rheumatic mitral stenosis when it is associated with congenital shunt lesion like PDA in our case.