ASD with MS and Stroke

A 10 year boy was brought to us in Aug 2003 with c/o mild exertional dyspnoea since childhood. There was an h/o recurrent respiratory tract infections in childhood, with failure to thrive and a small delay in developmental milestones. No h/o cyanosis. On examination, he had RV type of apex, left parasternal heave, wide, fixed splitting of S2, ESM in pulmonary area, and a Mid-diastolic murmur in tricuspid area. He was clinically diagnosed to have ASD. 2D Echo revealed a large ostium secondum type of ASD with L→R shunt, with e/o Right sided volume overload and mild pulmonary hypertension. The defect size was 18-20 mm and there was a good rim of tissue all around the defect.
In addition, there was mild thickening of mitral valve near the tips (? myxomatous,? rheumatic) but no significant diastolic gradient. The patient was subjected to ASD device closure using Amplatz device. The patient was fine for following two years with remarkable improvement in symptoms. Subsequently, he was lost to follow-up.

Since last two years he again started having progressive effort dyspnoea, initially NYHA class II but which gradually progressed over next several months to NYHA class III. He came to us for the follow up in October 2007. On examination, he had raised JVP, RV heave, diastolic shock, loud 1st heart sound in the mitral area, loud P2 in pulmonary area, O.S. 60-80 m.sec. after S2, and a low pitched rumbling mid diastolic murmur in apical region with presystolic accentuation. 2D Echo revealed rheumatic involvement of mitral valve. Anterior mitral leaflet was thickened, not calcified. Posterior mitral leaflet was almost fixed. MVA by P½ t was 0.6 sq.cm. and by planimetry, it wa 0.8 sq.cm. Peak and mean diastolic gradient were 28 and 18 mm Hg respectively. Wilkin score – T2, C0, P2, S2 = 6/12. No AS/AR. Dilated LA. LA appendage well seen and empty. Severe pulmonary hypertension was present.

Since the patient had ASD device in situ, septal puncture was not possible. Hence despite the valve being suitable for BMV, the patient was subjected to CMC.

Post procedure, the patient developed high grade fever spikes. Clinical examination and lab investigations were unremarkable. 2DEcho revealed nicely opened lateral commisure with MVA around 1.8 to 2.0 sq. m. No vegetations, no clot.

He was treated empirically with antibiotics after which the fever subsided. However, 12 days after CMC, while he was being planned for discharge, the patient developed acute onset left hemiparesis with left UMN facial paresis. Immediately his CT scan was obtained which showed no bleed. Repeat 2DEcho screening revealed a large LA thrombus. The power on left side rapidly deteriorated from grade 3 to grade 1 within 45 minutes. In view of acute onset of ischemic stroke with significant neuro-deficit, it was decided to subject him to intra-arterial thrombolysis. Intra Venous thrombolysis was not given in view of present of LA clot and recent surgery. Cerebral angiography revealed acute embolic occlusion of Right ICA in its intra-cavernous segment with no distal flow. A soft 0.014 mm wire was passed across the embolus. Progreat (Terumo Japan) was introduced and placed within the embolus. Export Catheter was used to aspirate the embolus. White cheesy material was aspirated. Further, thrombolysis was carried out. Total of 12 mg of rTPA was injected. The time interval between onset of symptoms and 1st injection of rTPA was about 2 hours. There was moderate improvement of intro-cranial flow with residual thrombosis. Post procedure, he was started on heparin drip and overlapped with Tab warfarin. 10 hours later, patient showed remarkable improvement and power in the left upper and lower limb improved. Within 24 hours the patient was mobilized, with power becoming almost grade 5. He was discharged on both Warfarin and Aspirin. He is now doing fine with no residual neurodeficit.