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CASE REPORT

CRITICAL MITRAL STENOSIS: A RARE PRESENTATION OF PEDIATRIC RHEUMATIC HEART DISEASE

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ABSTRACT: Rheumatic fever (RF) and rheumatic heart disease (RHD) continue to be a major health hazard in most developing countries. Paediatric and juvenile mitral stenosis (MS), upto the age of 12 and 20yr respectively, severe enough to require operative treatment was documented. These negate the belief that patients of RHD become symptomatic ≥20 years after RF as well as the fact that congestive cardiac failure in childhood indicates active carditis and RF.
KEYWORDS: Rheumatic fever, Rheumatic heart disease, Juvenile mitral stenosis.

INTRODUCTION: Although in developed countries it has declined, it is still a major public health problem in developing world.[1] The overall prevalence estimated to be about 1.5-2/1000 in all age groups, in India.[2] The disease actually has its roots in childhood (5-15 yr).[3] Mitral stenosis is highly prevalent in developing countries because of its association with the prevalence of rheumatic fever. Here, patients tend to be young with a pliable valve, whereas in developed countries patients are of increased age with several co-morbidities.[4] Though Progression of mitral stenosis in developing countries is malignant and intervention is often necessary during teenage years. Here is such a case of isolated critical Mitral Stenosis that developed within a short time after an attack of Rheumatic fever and became significantly symptomatic within the pediatric age group.

CASE REPORT: A 8 year old boy presented with moderate to high grade continuous fever for 15 days duration with headache, and myalgia. History of Cough with frothy sputum and gradual onset progressively increasing respiratory difficulty was present for same duration. Mother also gave the history of gradual onset exertional dyspnoea for last 2-3 years which was non-seasonal and slowly affected outdoor activities to daily activities of the patient. The child also experienced acute attack of severe respiratory difficulty at night at frequent interval for which he awakened from sleep with much distress. Mother also noticed that child was not gaining weight for this same period.

There was no history of chest pain, squatting, pain in legs during walking or history that suggestive of any neuro deficit. Mother stated that at age of five and half years the child experienced prolonged low grade fever, breathlessness and chest pain for which he undergone some special investigation outside, and received some oral medication on Out Patient Department basis. There was no history of joint pain, skin lesion or any abnormal movement at that time. Since then child is suffering from repeated attacks of low grade fever that was irregularly treated outside. On examination at the time of admission Child was restless, continuously maintained sitting posture, pallor and oedema was positive. Pulse examination showed tachycardia (140/min) with low volume pulse, high respiratory rate (36/min). Blood pressure was 80/60 mm of Hg. There was engorged and pulsatile neck vein. CVS examination showed bulging of precordium at left side, Apex in normal

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position and of tapping character. A diastolic thrill was palpable at apex. On auscultation first heart sound was sharp and loud, and pulmonary component of second heart sound also loud at pulmonary region. A low pitched localised mid diastolic murmur of grade IV was audible at apical region. Apart from this there was crepitation especially in basal chest and tender hepatomegaly.

Investigation showed Hb-9gm%, WBC- 8300, normal DC, Platelet Count- 2.5 lakh, ESR- 38 mm/hr, CRP- 51.48 mg. ASO titre- 554 U/ dl. ECG showed sinus arrhythmia.

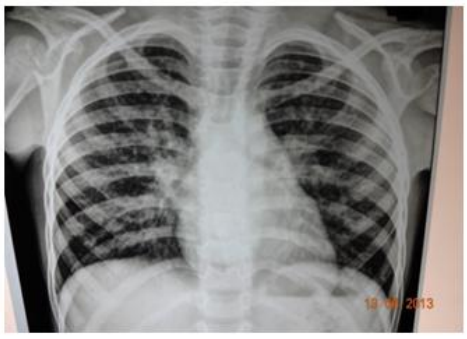

Figure 1

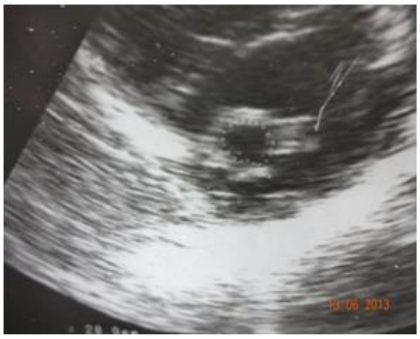

Figure 2

Chest X ray P-A showing pulmonary congestion Echocardiography showing critical stenosis.

In Echocardiography there was Critical Mitral stenosis (< 0.78 sqm) with pressure gradient 28/20 mm of Hg (Peak/mean), trivial aortic regurgitation, and severe pulmonary hypertension with good left ventricular systolic function.

After consultation with cardiologist diagnosis was confirmed as recurrence of rheumatic fever in a case of established rheumatic mitral stenosis and oral prednisolone was started with 2 mg/kg along with anti- congestive therapy and anti-streptococcal antibiotic, but despite this continued therapy for 2 months congestive cardiac failure was not controlled probably due to development of indolent carditis. The child was given azithromycin (12.5 mg/kg) along with other anti-congestive measure as per advice from cardiologist. The child improved symptomatically while waiting for surgery (Ballon dilatation of mitral valve). Now the child is on anti-congestive measure, penicillin prophylaxis (3 week interval) along with azithromycin (12.5 mg/kg for 1 year).

DISCUSSION: In developing countries, the main cardiovascular causes of death in those aged between 5 and 35 years are rheumatic fever and rheumatic heart disease. Among the RHDs Mitral stenosis remains an important cause of morbidity despite the ease with which it can be diagnosed and treated. The normal mitral valve orifice area is approximately 4-6 sq.cm. Critical mitral stenosis occurs with a valve area of less than 1 sq.cm.[5,6] In India, critical MS may be found in children as young as 6 to 12 years old. (Up to 20%). In developing countries, rheumatic MS manifests 10-30 years after the initial rheumatic insult to the mitral valve. In developed countries, this latent period may be as long as 50 years. In developing countries like India, early evidence of MS may develop as early as the teenage years, presumably because of a more aggressive initial attack and/or recurrent bouts of rheumatic fever (Consequences of suboptimal or absent antibiotic prophylaxis).[6] Out of 1408 patients with rheumatic heart disease seen in a hospital at New Delhi between 1967 to 1973, 51% having

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mitral stenosis, among them 2.8% patients were below 10 years of age[7].In many cases past history of RF is not obvious. Studies from different parts of India showed that about 43-50 per cent developed significant mitral obstruction without a history to suggest RF indicating that acute RF is not being recognized, possibly because RF is occurring with subclinical carditis but without arthritis, arthralgia, subcutaneous nodules, and chorea. Here our case is such where symptomatic Mitral stenosis has been developed very rapidly within 2 year of the probable first attack of rheumatic fever. So our aim is to consider rheumatic origin of severe mitral stenosis while considering differential diagnosis of juvenile mitral stenosis.

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