A Case Study on Chronic Rheumatic Heart Disease with Mitral Valve Stenosis

Keywords: Rheumatic heart disease, Mitral valve stenosis, atrial fibrillation, Shortness of breath, Mitral regurgitation, Inferior vena cava filter, DVT

ABSTRACT

A female patient of age 62 years admitted in hospital 5 months back with shortness of breath and chest discomfort and she was diagnosed as chronic rheumatic heart disease, moderate mitral valve stenosis with moderate mitral valve regurgitation, history of deep vein thrombosis 2 years back and treated with IVC Filter Implantation. Then treated with anticoagulants, antihypertensive agents. She complained 4 days back with shortness of breath admitted in ICU department and treated with furosemide infusion, T.Acenocoumain 1mg, T.Metoprolol 25 mg, T.torsemide10 mg and spironolactone50mg, T. Digoxin 0.25 mg and she improved symptomatically in two days and vitals are normal and stable.
INTRODUCTION

Chronic rheumatic heart disease, representing the permanent lesions of the cardiac valves, is the most serious consequence of rheumatic fever. It accounts for a significant number of repeated hospitalizations and deaths. Recurrences of rheumatic fever play an important role in the worsening of the valve lesions, but the damage, as a result of the process of cicatrisation can be progressive even in the absence of subsequent acute episodes. The clinical presentation, the mortality, as well as the frequency and speed of development of an established valve disease after the acute phase, vary considerably geographically, influenced primarily by the socioeconomic and medical backgrounds of the populations involved. In developed countries, severe rheumatic valve disease is now uncommon in children and adolescents, and treatment for the advanced form of the disease is usually limited to adults. In contrast, in many developing countries, chronic rheumatic heart disease remains the most important cause of acquired cardiac disease among patients ages aged between 5 and 30. It is a systemic immune process that is sequelae to a beta hemolytic streptococcal infection of pharynx. It is the most common in developing countries. However, it is responsible for 250,000 deaths in young people worldwide each year. Over 15 million people have evidence of rheumatic heart disease.

Epidemiology:

Rheumatic Heart Disease is the most critical form of acquired heart disease in children and young adults living in developing countries. RHD accounts for approximately 15 to 20 percent of all patients with heart failure in endemic countries.

Etiology:

Rheumatic Heart Disease results from either single or repeated attacks of rheumatic fever that results in rigidity and deformity of valve cusps, the fusion of the commissures, or shortening and fusion of the chordate tendineae. Over 2 to 3 decades valvular stenosis and regurgitation results. In chronic rheumatic heart disease, the mitral valve alone is the most commonly affected valve in an estimated 50 to 60% of cases. Combined lesions of both the aortic and mitral valves occur in 20% of cases. Involvement of the tricuspid valve occurs in about 10% of cases in association with mitral or aortic disease. Tricuspid valve cases are thought to be more common when recurrent infections have occurred. The pulmonary valves are rarely affected.
Pathophysiology:

Rheumatic Heart Disease is the result of valvular damage caused by an abnormal immune response to streptococcus pyogenes infection, which is classified as a group a streptococcus that causes acute rheumatic fever. Acute rheumatic fever occurs around three weeks after group a streptococcal pharyngitis that can affect joints, skin, brain, and heart. After multiple episodes of rheumatic fever, progressive fibrosis of heart valves can occur, this can lead to rheumatic valvular heart disease.

Clinical features:

The most common presenting features of Acute Rheumatic Fever includes fever (>90% of patients) and arthritis (75% of patients). The most serious manifestations in carditis (>50% of patients) because it can lead to chronic rheumatic heart disease. There are four other clinical features are arthritis, carditis, skin manifestations, chorea.

Management:

Medical management for chronic RHD is largely on the presence or absence of cardiovascular symptoms. Most patients with mild to moderate valvular involvement will remain asymptomatic for years. Strict adherence to secondary prophylaxis should be emphasized, as poor adherence and reoccurrence of ARF have independently been associated with an increased risk of RHD complications and death. Heart failure should be considered a surgical disease, with no long term role for medical management, except in cases where surgery is unavailable or contraindicated. Exercise restrictions are based both on the severity of valvular disease and the intensity of the desired activity, and should be guided by the 2005 Bethesda Guidelines. Emerging data suggests that statins may slow the progression of rheumatic mitral and aortic stenosis, but prospective adult trials have shown mixed results. Statins are not currently recommended in patients with chronic RHD.

Investigations:

- Hb: 9.8g/dl
- Serum creatinine: 1.75mg/dl
- Blood Urea: 42mg/dl
• Serum Uric Acid: 3.8mg/dl

• PTINR: 13sec

• INR: 09sec

• Serum sodium: 136mmol/lit

• Serum Potassium: 5.1mmol/lit

• Serum chlorides: 107mmol/lit

**Differential Diagnosis:**

ECG: Sinus tachycardia, Atrial Flutter, Incomplete right bundle branch block.

2D ECHO: CRHD

Moderate Mitral valve stenosis with Moderate Mitral valve regurgitation

Mitral Valve Area by Planimetry: 1.5cm², MV (Gr-16/7 mmHg)

Normal Left Ventricular Function

Ejection Factor: 60%

**Methodology:**

A prospective Observational study was conducted in tertiary care teaching Hospital.

**Treatment:**

She Implanted IVC Filter for Deep Vein Thrombosis and treated with Tab.Acenocoumarol 1 mg, Tab. Metoprolol 50 mg, Tab.Diltiazem 90 mg, Tab. Torsemide 10mg+Spironolactone 50 mg, Tab.Ranitidine 150 mg 5 months back, Now she treated with Injection Furosemide 20mg Infusion QID, Injection Heparin 4000 Iu 6th Hourly, Injection Ceftriaxone, Tab. Acenocoumarol 1 mg, Tab. Metoprolol 25 mg, Tab.Digoxin 0.25 mg, Tab. Pantoprazole 40 mg, Tab. Spironolactone 25 mg for two days only then stopped. Tab. Torsemide 10 mg+spironolactone 50 mg.
RESULTS AND CONCLUSION

She was symptomatically improved in two days and Vitals are normal and stable on Discharge condition. She counseled with non pharmacological remedies to maintain normal conditions because there is no medications can correct the mitral valve stenosis.

<table>
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<th>Test value</th>
<th>Normal Range</th>
</tr>
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<tbody>
<tr>
<td>UREA</td>
<td>46 mg/dl</td>
<td>15-40mg/dl</td>
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<tr>
<td>Serum creatinine</td>
<td>1.35mg/dl</td>
<td>0.6-1.4 mg/dl</td>
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<tr>
<td>Uric acid</td>
<td>4.5mg/dl</td>
<td>3.5-7.0 mg/dl</td>
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<tr>
<td>Serum Sodium</td>
<td>136mmol/lit</td>
<td>135-155mmol/lit</td>
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<tr>
<td>Serum potassium</td>
<td>5.1mmol/lit</td>
<td>3.5-5.5 mmol/lit</td>
</tr>
<tr>
<td>Serum chlorides</td>
<td>107mmol/lit</td>
<td>90-110mmol/lit</td>
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(PT)PROTHROMBIN TEST with INR

<table>
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<th>Test value</th>
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<tr>
<td>Mean control value</td>
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<tr>
<td>INR</td>
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</tr>
</tbody>
</table>

Limit salt intake in diet, Maintain healthy weight, Do regular exercise, Avoid caffeine (or) coffee it can worsen the irregular heartbeats, Avoid Fatty food consumption, Regular Medication Adherence to the Patient.

DISCUSSION

Chronic Rheumatic Heart Disease is much more common in females than males. She suffered with shortness of breath and chest discomfort due to accumulation fluid and increasing the volume of blood it can leads to increases the workload on the heart so she treated with diuretics to reduce the blood accumulation in lungs, blood thinners to prevent the blood clots, Beta-blockers to slower the heart rate and Antiarrhythmics are used to treat atrial fibrillation (or) other rhythm disturbances due to mitral valve stenosis.

REFERENCES


Citation: M.Swapna et al. Ijppr.Human, 2019; Vol. 16 (4): 296-301.
5. Sika-Paotonu D, Beaton A, Steer A, Carapetis J. Acute Rheumatic Fever and Rheumatic Heart.