

ORIGINAL ARTICLE

FREQUENCY OF SEVERE MITRAL STENOSIS IN YOUNG FEMALE PATIENTS HAVING PURE MITRAL STENOSIS SECONDARY TO RHEUMATIC HEART DISEASE

Shakeel Ahmad, Umar Hayat, Humera Naz

Department of Cardiology, Ayub Medical College, Abbottabad, Pakistan

Background: High morbidity and mortality due to Rheumatic heart disease (RHD) associated with females is mainly because of late diagnosis on one hand and socioeconomic reasons on the other hand. Poor referral to tertiary care centres leads to delayed diagnosis which results in complications. The objectives of this cross-sectional descriptive study was to assess the frequency of severe mitral stenosis in woman of child bearing age, having pure mitral stenosis (MS) secondary to rheumatic heart disease. **Methods:** Two hundred and fifty women of child bearing age with RHD were enrolled in the study using consecutive non-probability sampling technique. Out of these 250 patients, cases of pure MS were selected. Patients with associated mitral regurgitation and aortic valve disease were excluded. After admission, assessment of mitral valve stenosis was done with 2D colour Doppler echocardiography. **Results:** Out of 250 consecutive patients of rheumatic carditis, 110 (44%) patients had pure mitral valve stenosis, 85 (34%) had stenosis with mitral regurgitation and 55 (22%) patients had both mitral and aortic valve problem of varying severity. Among 110 patients with pure mitral valve stenosis, 48 (43.6%) had severe mitral valve stenosis. Severe mitral valve gradient (MVG) and high pulmonary artery pressure (PAP) was observed in 66 (60%) and 49 (44.5%) of the patients respectively. **Conclusion:** This high frequency can be linked to lack of early detection of the disease at primary level, poor management of throat infections and poor rheumatic fever prophylaxis at community level.

Keywords: Rheumatic heart disease, mitral stenosis, mitral valve gradient, pulmonary artery pressure, primary health centre

INTRODUCTION

The incidence of rheumatic fever is on decline in the western world^{1,2} but is still a common problem in developing countries like Pakistan with a prevalence rate of 22/1,000 population. This high prevalence rate put Pakistan among the highest in the world.³ Rheumatic fever may present with arthritis, chorea and carditis, however carditis is the commonest. In carditis all the three layers of the heart are involved. Endocardium is mostly affected, the common of which is the mitral valve involvement⁴, which predominately affects 2/3 of the female having rheumatic carditis.^{5,6} In mitral valve, stenotic lesions are more common in females and regurgitant lesions are more common in males. Aortic valve problems are more in male than in female⁷ resulting in high morbidity and mortality.⁸ These complications result from late diagnosis and late referral to the centres specialized in the management of this problem and sometimes because of shortage of available resources. Improper and late management of rheumatic heart disease and associated complications leads to high morbidity and mortality in women of child bearing age.

Severity of mitral stenosis can be assessed clinically as well as with different investigations. Clinically the severity is assessed by dyspnoea as

classified by NYHA classification, history of palpitation on exertion, cough and haemoptysis, frequent respiratory tract infections, poor growth, frequent admissions to hospital with pulmonary oedema, and previous thromboembolic phenomenon.

Although blood complete picture, ESR, ECG, X-ray chest help in assessing the mitral valve severity; echocardiogram is the most important tool for evaluating the severity of mitral stenosis.^{9,10} The mitral valve severity is assessed by left atrium (LA) size, mitral valve area, mitral valve gradient, and pulmonary artery pressure.¹¹

Normal mitral valve area is 4–6 Cm. Stenosis is mild when the area is more than 1.6 Cm; it is moderate when the area is between 1.0–1.6 Cm and it is severe when the area is less than 1.0 Cm.¹¹ Normal valve gradient is 0–4 mm Hg, it is mild when it is less than 5, moderate when it is 5–10 and severe when more than 10 mm Hg.^{11,12}

Normal pulmonary artery pressure is between 15–25 mm Hg. It is mild when below 30, moderate when between 30 and 50, and severe above 50 mm Hg.¹³

We can also determine the presence, severity and haemodynamic consequences of mitral stenosis. In mitral stenosis two dimensional echocardiographic imaging allows definition of leaflet anatomy and dynamics, sub-valvular disease,

ventricular functions and involvement of other valves. Spectral and colour Doppler echo-cardiographic techniques permits accurate measurement of transvalvular gradient, determination of functional orifice area, evaluation of associated valvular regurgitation and assessment of pulmonary artery pressure.¹¹⁻¹³ This enables us to decide for the future plan of management other than medical therapy like commissurotomy or mitral valve replacement before end stage disease results.

MATERIAL AND METHODS

Two hundred and fifty female patients of reproductive age suffering from rheumatic heart disease were enrolled in the Department of Cardiology Ayub Teaching Hospital Abbottabad. These patients were selected from cardiology outpatient department, referral cases from gynaecology and obstetrics and medical units. Out of these, cases of pure mitral stenosis were selected for the study after their initial work up like blood complete examination, ECG, X-ray chest and echocardiography.

Assessment of mitral stenosis was done with 2D colour Doppler echocardiography. Mitral valve area was calculated with trace method in parasternal short axis view using 2D echo. Left and right atrial and ventricular sizes were taken in all patients. Left atrium (LA) was searched for any clots in apical 4 chamber view.

Every patient was subjected to Continuous Wave (CW) and Pulse Wave (PW) Doppler studies in apical 4 chambers view. Mitral valve area was calculated with pressure half time method and mitral valve gradient was observed in left ventricular inflow in each case. Pulmonary artery pressure was assessed by pressure gradient in tricuspid valve in apical 4 chamber view, and the relationship or mitral valve area with left atrium size, mitral valve gradient and pulmonary artery pressure was made.

Blood complete with ESR examination was done in each case to see the level of haemoglobin and to see the activity rheumatic process by looking the ESR of the patient. ECG was performed to evaluate the state of heart rate and arrhythmia like atrial fibrillation. X-ray chest in PA view helped us in the assessment of pulmonary artery pressure and to detect any lung pathology like lung infection or infarction. Blood culture was taken in few patients who presented with fever.

RESULTS

Out of 250 consecutive patients of rheumatic carditis, 110 (44%) patients had pure mitral valve stenosis, 85 (34%) had stenosis with mitral regurgitation and 55

(22%) patients had both mitral and aortic valve problem of different severity.

One hundred and ten patients diagnosed with pure mitral stenosis were selected for the study. Each patient was subjected to ECG and echocardiography at the same time to see the relationship between the heart rate and mitral valve gradient.

Mitral valve area was observed both with 2D and CW Doppler system. Severe mitral valve stenosis (area less than 1.0 cm) was observed in 48 (43.64%) patients. Mild (1.8 cm and above) to moderate (1.0–1.8 cm) stenosis was observed in 26 (23.64%) and 36 (32.73%) patients respectively. CW Doppler showed almost the same results.

Left atrium was enlarged in almost all cases but had different relationship with mitral valve gradient and mitral valve area. In severe mitral stenosis with the area of less than 1.0 Cm, 49 (45.5%) of patients had left atrial size of more than 5 Cm but in 2 patients the sizes were relatively small for the same degrees of stenosis and in 5 patients the sizes were beyond 6.5 Cm with relatively less stenosis.

Severe mitral valve gradient (more than 10 mm Hg) was observed in 66 (60%) patients, 31 (28.2%) patients had moderate Mitral Valve Gradient (MVG) between 5 and 10, and 13 (11.8%) had mild MVG (less than 5 mm Hg) respectively.

High Pulmonary artery pressure (PAP) (above 50 mm Hg) was observed in 49 (44.5%) patients, moderate in 40 (36.4%) patients and mild in 21 (19.1%) patient pulmonary pressure was high in patients of severe mitral stenosis. With in the mitral stenosis patients group it was higher in those having severe mitral stenosis. The patients having small left atrium had a higher PAP for the same degree of mitral stenosis. PAP was also higher in patients of high mitral valve gradient for the same degree of mitral stenosis. PAP was found to be high in patient with atria fibrillation then in sinus arrhythmia.

DISCUSSION

Rheumatic heart disease is the most frequent cause of abnormal valvular function. Acquired mitral stenosis (MS), or mitral valve stenosis, is virtually synonymous with rheumatic heart disease. Current estimates indicate that the prevalence of rheumatic fever in the United States is less than 1 case per 100,000 people. A steady decline has been observed in incidence of rheumatic fever and, thus, in acquired MS. In some developing countries, such as India, the prevalence of rheumatic fever is 100–150 cases per 100,000 people. Following development of rheumatic heart disease, 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). In some developing countries, the prevalence of rheumatic heart disease in children is 5–15 cases per 1,000 people.^{12,13}

Our study was a hospital based study and we calculated the prevalence of MS in patients of Referral centre (RC). The results showed that among the RC patients around half of them developed pure mitral stenosis and the mean age of those having pure MS was 25 years.

Both the frequency of MS observed in our study and the mean age of the patients was higher as compared to other studies. The frequency of severe mitral stenosis was above 48% in our study. The main reasons for this high frequency may be late diagnosis and Poor rheumatic fever prophylaxis which can be attributed to the failure to diagnose and treat at the primary or secondary health centres.^{6,14}

Most of the patients in our study was diagnosed for MS incidentally when they got admitted in hospital for some other cause mostly during pregnancy, which may be attributed to the high age of diagnosis of MS as compared to other studies which were mostly done in developed countries having better diagnostic facilities.^{13,15}

LA size and its relationship with mitral valve gradient and mitral valve area has been reported in different studies and the results varied across the studies.^{16–19} LA size depends on how much time is taken for that stenosis to develop. It would take less time if the load of infection is high or when the virulence of organism is high when the patient is immune-compromised, but LA may not enlarge to the same degree which may result in high pressure in left atrium and any clinical condition causing tachycardia may result in pulmonary oedema.^{16,20} Contrary to that if the organisms are of low virulence or when the patients are immuno-competent, stenosis may develop slowly and left atrium get enough time to dilate which can accommodate large volume of blood without any increase in LA pressure, such patients become symptomatic very late in their course of disease.^{21,22}

Mitral valve showed a linear relationship with heart rate, the higher the rate the higher was the gradient across the mitral valve. Mitral valve gradient has reverse relationship with mitral valve area, the lesser the mitral valve area the greater was the gradient. Mitral valve gradient was high when LA size was small for the same degree of mitral valve area. Mitral valve gradient was low in patient with enlarged left atrial size for the same degree of mitral valve area the findings were consistent with other studies.^{10,12}

Mitral valve gradient and pulmonary artery pressure was higher in patients having severe MS. The same observation was reported by Lue in a study to assess the Long term outcome of patients with rheumatic fever.¹⁴

CONCLUSION

Rheumatic carditis is a deadly disease, especially when it involves the young females in their child bearing age having very high morbidity and mortality. Measures should be taken to avoid its complication:

RECOMMENDATIONS

1. Facilities should be present at least at the secondary health centres for prevention, proper diagnosis and management of rheumatic fever and RHD.¹⁵
2. Health authorities should bar range refreshing courses for the health personal working in primary and secondary health centre every 2–3 years and should be given certificate for practice only when they attend such courses.
3. Medicine for such patients should be available at the primary health centre free of cost.
4. Intervention when required should be available at each tertiary care centre free of cost or at least in affordable cost to a common man.
5. Policy interventions to control this cardiovascular epidemic and raising public awareness about various cardiovascular diseases.

REFERENCES

1. Carrilho-Ferreira P, Pedro MM, Varela MG, Diogo AN. Severe rheumatic mitral stenosis: a 21st century medusa. *Arch Intern Med* 2011;171:1498–9.
2. Bisno AL, Shulman St, Dajan A. The rise and fall of rheumatic. *JAMA* 1988;259:728–9.
3. Akhtar N, Sadiq M, Chagani H. Guidelines for prevention of rheumatic fever and rheumatic heart disease. *Pak J Cardio* 2004;15:136–48.
4. Rose AG. Etiology of acquired valvular disease in adults. A survey of 18132 autopsies and 100 consecutive valve replacement operations. *Arch Pathol Lab Med* 1986;110:385–9.
5. Mitral stenosis, valvular heart disease In: Braunwald, Heart Disease: A textbook of cardiovascular medicine. (6th edition) Philadelphia: WB Saunders; 2001. p. 1643
6. Burger W, Brinkies C, Illert S, Teupe C, Kneissl GD, Schröder R. Right ventricular failure before and after percutaneous balloon mitral valvuloplasty. *Int J Cardiol* 1997;58:7–15.
7. Movahed MR, Ahmad Kashani M, Kasravi B, Saito Y. Increase prevalence of mitral stenosis in women. *J Am Soc Echocardiogr* 2006;19:911–3.
8. Ozer O, Davutoglu V, Sari I, Akkoyun DC, Suco M. The spectrum of rheumatic heart disease in the south eastern entolia endemic region. Results from 1900 patients. *J Heart Valve Dis* 2009;18:68–72.
9. Nichol PM, Gibert BW, Kisslo JA. Two dimensional echocardiographic assessment of mitral stenosis. *Circulation* 1977;55:120–8.

10. Akhtar N, Rehman F, Anam K, Begum N, Naher S, Fatima N, *et al.* Valvular heart disease in pregnancy. Maternal and foetal outcome. *Mymensingh Med J* 2011;20:436–40.
11. Binder TM, Rosenhek R, Porenta G, Maurer G, Baumgartner H. Improved assessment of mitral valve stenosis by lumetric real-time three-dimensional echocardiography. *J Am Coll Cardiol* 2000; 36:1355–61.
12. Saxena A. Strategies for the improvement of cardiac care services in developing countries: What does the future hold. *Future Cardiol* 2012;8:29–38.
13. Leavitt JL, Coats MH, Falk RH. Effects of exercise on transmitral gradient and pulmonary artery pressure in patients with mitral stenosis or prosthetic mitral valve Doppler echocardiographic study *J Am Coll Cardiol* 1991;17:1520–6.
14. Lue HC, Wv MH, Wang JR, Wufi Wu YN. Long term outcome of patients with rheumatic fever receiving penicillin G prophylaxis every three weeks doses ever 4 week. *J Padiat* 1994;125:812–6.
15. Lock JE, Kalilula M, Shrivastavas BJ and Kearne JF. Percutaneous catheter commissurotomy in rheumatic mitral stenosis. *N Eng J Med* 1985;313:1515–8.
16. Selcuk MT, Selcuk H, Maden O, Temizhan A, Aksu T, Dogan M, *et al.* Relationship between inflammation and atrial fibrillation in patients with isolated rheumatic mitral stenosis. *J Heart Valve Dis* 2007;16:468–74.
17. Movahed MR, Ahmadi-Kashani M, Kasravi B, Saito Y. Increased prevalence of mitral stenosis in women. *J Am Soc Echocardiogr* 2006;19:911–3.
18. Supino PG, Borer JS, Preibisz J, Bornstein A. The epidemiology of valvular heart disease: a growing public health problem. *Heart Fail Clin* 2006;2::379–93.
19. Glancy DL. Mitral stenosis: I. Anatomical, physiological, and clinical considerations. *J La State Med Soc* 2003;155(2):91–5, quiz 96, 119.
20. Roberts-Thomson KC, Stevenson IH, Kistler PM, Haqqani HM, Goldblatt JC, Sanders P, *et al.* Anatomically determined functional conduction delay in the posterior left atrium relationship to structural heart disease. *J Am Coll Cardiol* 2008;51:856–62.
21. Naveed Akhtar, Sabeen Razaque. Penicillin clinic; a model program for prevention of Rheumatic fever & Rheumatic heart disease in Pakistan. *Pak J Cardiol* 2007;18(3-4):66–72.
22. Messika-Zeitoun D, Serfaty JM, Laissy JP, Berhili M, Brochet E, Iung B, *et al.* Assessment of the mitral valve area in patients with mitral stenosis by multislice computed tomography. *J Am Coll Cardiol* 2006;48:411–3.
23. Rashid SZ, Rajput IA, Samad A. Echocardiographic profile in mitral stenosis. *Pak J Cardiol* 2007;18:18–24.

Address for Correspondence:

Dr. Shakeel Ahmed, Department of Cardiology, Ayub Medical College, Abbottabad, Pakistan. **Cell:** +92-333-9887159

Email: omerhayat@hotmail.com