CLINICAL VIGNETTE

Palpitations as an Early Symptom of Mitral Stenosis

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A 67-year-old Indian female presented for cardiac evaluation for palpitations. The patient's medical history was significant for type 2 diabetes, hyperlipidemia and stage 3 ovarian cancer, status post resection and chemotherapy, now in remission. The palpitations began approximately 2 years prior and were described as "a racing heart", but not irregular, occurring 1-2 times per week and lasting for up to 30 minutes. The symptoms occur mainly at rest as she lives a mostly sedentary lifestyle. The patient denied associated symptoms including chest pain, shortness of breath, dizziness, or syncope. She described no significant family history of cardiac disease. She is a non-smoker and denied alcohol use.

She previously sought care for these symptoms from her primary care doctor. She was told her EKG was normal and that her symptoms were related to anxiety. She was offered a trial of sertraline but declined. Since then, she has engaged in mindfulness and meditation to help alleviate symptoms, but these measures have worked inconsistently. Recently, she describes an increase in frequency and duration of her symptoms; therefore, she presented to cardiology for further evaluation.

On presentation, she was afebrile with a blood pressure of 155/70 and pulse 115/min. Respiratory rate was 12/min and oxygen saturation was 97% on room air. Physical exam revealed a regular rate and rhythm with a normal S1/S2. No murmur, rubs, or gallops were present. JVP was normal at 7 cm. Lungs were clear to auscultation. Distal pulses were 2+ and she did not have lower extremity edema. EKG done showed sinus tachycardia at a rate of 117 bpm.

Based on her presentation, the patient was referred for an echocardiogram and event monitor. On the day of her echo, the blood pressure was 133/72 and heart rate averaged to 100 beats per minute. The LV systolic function was normal with an ejection fraction of 60%. She had mild left ventricular hypertrophy and impaired left ventricular relaxation. Visually the mitral valve appeared thickened, calcified, and restricted. The hemodynamic measurements were most consistent with moderate mitral stenosis (valve area 2.6 cm^2 by pressure half-time, 1.3 cm^2 by the continuity equation with a mean gradient of 13 mmHg). At least moderate mitral regurgitation was noted. The left atrium was severely dilated, and the pulmonary artery pressure was moderately elevated at 50-55 mmHg.

A 14-day event monitor showed that her predominant rhythm was sinus with an average rate of 84 beats per minute. Four short paroxysms of supraventricular tachycardia were noted with the longest and fastest run lasting 7 beats at 150 beats per minute. These non-sustained atrial runs were too short to clearly define but in this particular clinical setting were concerning for atrial fibrillation (AF).

Discussion

Rheumatic fever still remains the most common cause of mitral stenosis (MS) worldwide but has become increasingly less frequent in the developed world.[1] To understand the impact that rheumatic fever has on the mitral valve, it is important to understand how the infectious process affects cardiac structure and ultimately hemodynamic function. Rheumatic fever is caused by streptococcal infection and is based upon a cross reaction between group A streptococci antigens and glycoproteins found on heart valves. This reaction causes inflammation of the heart, primarily in the endocardium causing thickening and scarring of the cardiac valves, which eventually leads to narrowing of the valvular orifice.[1] The infectious process is acute. The progression of MS is most commonly gradual with the natural history of disease encompassing a long latent phase where the architecture of the mitral valve is damaged, but the patient remains asymptomatic mainly due to compensation of cardiac function. Recent studies have suggested this latent phase can range from 7-20 years.[2]

With time, the heart becomes less able to compensate for the stenotic valve, mainly due to increasing pressure gradients between the left atrium and left ventricle and structural changes occur. The left atrium enlarges and pulmonary congestion ensues eventually leading to pulmonary hypertension. Narrowing of the mitral valve orifice diminishes cardiac output. The reduction of cardiac output and pulmonary congestion combine to cause heart failure in patients with mitral stenosis.[2]

Atrial fibrillation is a common complication of mitral stenosis affecting approximately 40% of all patients.[2] It mainly occurs due to enlargement of the left atrium and ensuing disruption of the conduction system. Early on, patients tend to have paroxysms of the arrhythmia but as the disease progresses more sustained arrhythmia is typical. For patients with severe mitral stenosis, the complications of atrial fibrillation can be serious as the arrhythmia tends to further diminish cardiac output especially in the setting of rapid ventricular rates. Additionally, the enlarged thrombus prone left atrium found in progressive mitral stenosis increases the incidence of systemic embolism, which is exacerbated by atrial fibrillation due to

impairment of left atrial contraction.[2] These patients are at high risk for stroke. In general, atrial fibrillation is poorly tolerated in patients with significant mitral stenosis and can be an indication for intervention.[3]

Management and Intervention

The indication for intervention is based primarily on the severity of symptoms and the type of intervention depends on the degree of narrowing and the architecture of the valve.[4] The Abascal echocardiographic score (also known as the Wilkins score) was first described in 1990 as a means to predict the success of percutaneous balloon mitral valvotomy (PBMV) in rheumatic mitral valve stenosis. The score takes into account mitral leaflet mobility, leaflet thickening, leaflet calcification, and subvalvular thickening.[4]

PBMV is recommended for symptomatic patients with severe mitral stenosis (MVA ≤ 1.5cm2) and favorable valve morphology (Abascal score ≤ 8) in the absence of contraindications such as left atrial thrombus (Class I A).[5] Mitral valve surgery is indicated in those patients with severe MS who are not candidates for PMBV or have failed prior PMBV (Class I B).[5] In those who are asymptomatic, PMBV is reasonable in those with severe MS who develop new onset Afib or severe MS with exercise (IIB, LOE C).[5]

The mean pressure gradient is another widely used parameter to determine the severity of MS and is dependent on the transvalvular flow and the diastolic filling period and will vary greatly with changes in heart rate. The diastolic pressure half-time is even more sensitive to variables such as compliance of the left ventricle and left atrium. Other measures of mitral valve stenosis such as the continuity equation or the proximal isovelocity surface area may be used if discrepancies exist.

Conclusion

Rheumatic mitral stenosis remains a relevant clinical entity though not encountered as often in the United States as in other parts of the developing world. Accurate diagnosis and appropriate medical and interventional treatment are critical to optimizing the clinical care and outcome of patients with MS. Our patient reported having pharyngitis as a young girl in India, which likely was the pathophysiologic trigger for her development of mitral stenosis. She does not currently meet criteria for intervention with percutaneous or surgical intervention due to the lack of severe narrowing of her valve and absence of symptoms. However, the clinical progression of her case does warrant monitoring with routine echocardiography and in her case further evaluation of atrial fibrillation, so her stroke risk can be more accurately defined. If she were to develop atrial fibrillation, anticoagulation and avoidance of tachyarrhythmias would be an imperative first step. A loop recorder is planned for next month.

Figures

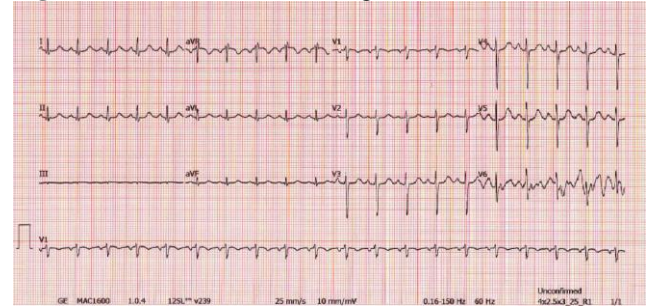**Figure 1:** 12 Lead Electrocardiogram.

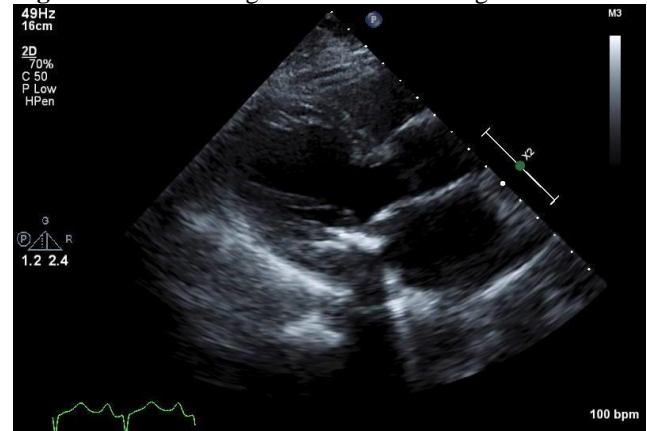**Figure 2:** Echocardiogram- Parasternal long axis view

Figure 3: Echocardiogram- Color Doppler through the mitral valve. Note color aliasing in the left ventricle suggestive of turbulence of flow distal to the stenosis (red arrow).
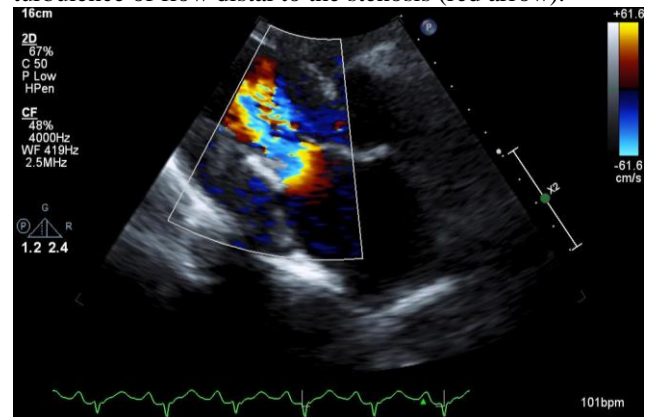
Figure 4: Echocardiogram- Transmitral pressure halftime and mean gradient.
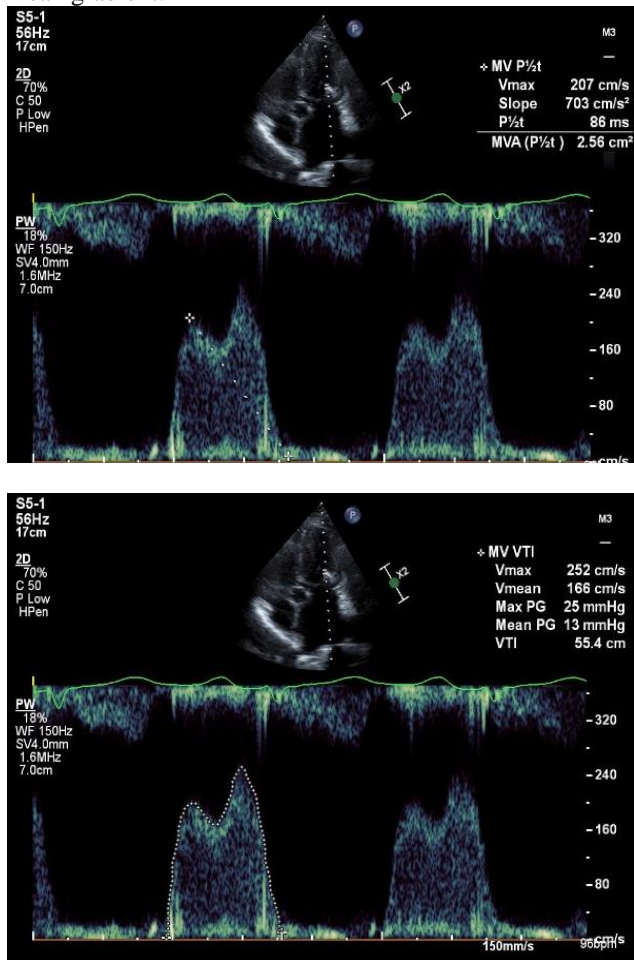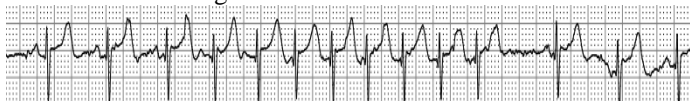

Figure 5: Event monitor rhythm strip showing a non-sustained atrial run concerning for atrial fibrillation.

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