A 75-Year-Old Woman with Fatigue and a Longstanding Heart Murmur

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What's the Take Home?

Volume 65 - Issue 12 - December 2025

Introduction. A 75-year-old woman presents with subacute (1-2 months) onset of easy fatigue and shortness of breath with exercise.

Patient history. There have been no chest pains of any kind, no palpitations, no paroxysmal nocturnal dyspnea or pedal edema. The patient noted that she has no history of smoking or prior cough, hemoptysis or history of chronic obstructive pulmonary disease. However, she is known to have a heart murmur since her 40s, which was initially termed "floppy mitral valve syndrome" but in recent times, was more accurately evaluated by echocardiography as mitral valve prolapse with regurgitation. Following this diagnosis, she has had periodic examinations and echocardiograms. The most recent was about 6 months ago, which showed the mitral valve regurgitant volume approaching 50% of ejection fraction (EF).

She otherwise has always been very healthy and extremely active. She is now a semi-retired dancing schoolteacher. She has 2 grown children and no history of rheumatic fever. She takes no prescription medicines.

Physical examination. The patient's physical examination reveals a thin woman with BMI 19-20. (Table 1)

Table 1. Physical examination findings

Physical examination	Findings
General appearance	Thin woman; BMI 19–20
Vital signs	Afebrile; pulse 88/min, regular; blood pressure 110/70 mm Hg; respirations 12/min
Lungs	Clear to auscultation; no wheezes or rales
Cardiac examination	Strong Point of Maximal Impulse; Grade III holosystolic murmur loudest at the apex with radiation toward the base
Extremities	No pedal edema
Laboratory results	Routine blood testing within normal limits

Correct Answer. D. Prompt surgical intervention with mitral valve repair

Discussion. Mitral regurgitation (MR) is the most common valvular disease encountered in clinical medicine reaching a 10% prevalence in older adults (older than 75 years). (1). There are several etiologies initially grouped into two major domains: primary MR and secondary MR. The former is due to disease of the mitral valve itself and is caused most by degenerative changes in the valve resulting in ballooning and prolapse of the valve with eventual flail and regurgitant leakage. A less common primary modality in modern times is rheumatic fever-related scarring and deforming of the valve apparatus resulting in stiff, fixed-open valve or scarring with shrinkage of valve cusps, leaving an unclosed valve with regurgitant leakage. Secondary MR is actually a disease of the left ventricle (LV) caused by either (or both) ischemic heart disease leaving a non-functional portion of the LV unable to participate in normal systolic closure of the valve or significant congestive heart failure (CHF) of any cause with an LV so dilated and large that the valve cusps cannot close with systole (1,2). These categories are usually easily differentiated on clinical grounds with histories of lifelong heart murmur in an otherwise cardiologic normal patient, history or no of rheumatic fever (which often caused multiple valve lesions, stenotic and/or regurgitant), history and cardiac findings of coronary disease, myocardial infarction and LV scarring or the presence of significant CHF.

The presented patient is known to have a long history of MR, is female of thin body habitus (the so called "floppy mitral valve syndrome" in previous times) and in fact has had excellent echocardiography follow up of her valve imaging, gradients, EF evolution and changes and regurgitant volumes. Thus, this is clearly primary mitral valve disease.

There was a time when wise, stethoscope wielding senior physicians would spend much time in very quiet rooms auscultating the patient's heart and announcing their findings and diagnoses to a rapt audience of younger doctors and students. It remains true that there are strong clues to a murmur being mitral in origin: 1) holosystolic rather than "diamond shaped" as with aortic stenosis; 2) a musical "cooing" characteristic rather than the rasping, coarse nature of aortic murmurs and 3) apical location prominence rather than at the base. But the diagnostic standard is now Doppler and cardiac echo, which clearly demonstrate the mitral source, the regurgitant volume, specific valve cusp involved, and the extent of compensatory LV dilatation. All of these are vital to the evaluation of MR severity, in determining need/timing of surgical correction, and how to perform such correction.

Regarding patient presentation, there will be the earlier diagnosis heart murmur and usually a quite extended, even decades, of asymptomatic heath. Eventually the volume overload becomes too much for the heart to sustain and symptoms become evident, namely heart failure findings of easy fatigue, dyspnea with effort/exercise.

Doppler and echocardiogram finding and evaluation. Two dimensional and Doppler echocardiography are the standards of care in diagnosis and evaluation of MR. (1). Although capable of elegant, multicolor time lapse demonstrations of regurgitant jets, valve co-adaptation and myriad other technological numbers and marvels, for our WTTH purposes, these are the following key metrics:

1. The diagnosis of severe MR is made the 50% or greater of LV stroke volume is regurgitant into the left atrium

2. Criteria for surgical intervention include onset of LV dysfunction defined by EF less than 60% and /or end systolic LV dimension greater than 40 mm.

3. In patients with severe MR (more than 50% of LV stroke volume is regurgitant) and onset of ANY clinical symptoms is an independent criterion for surgical intervention (1).

Regarding therapy, quite simply for MR, it is totally dependent on whether the MR is primary or secondary as previously discussed. It will once again be emphasized that primary MR is due to disease of the valve itself whereas secondary MR is a disease of the LV (either coronary disease with lost/scarred LV in the region of the valve and chordae tendinea or LV failure of other cause with dilation enough to separate the valve leaflets to the point of non-approximation and closure). (1,3). As these pathophysiologies differ, so does their management. There is no effective medical therapy for primary MR plain and simple (1). As discussed, when the clinical and/or echocardiographic findings reach the thresholds of symptom onset of any kind and/or the Echo criteria is listed, then surgical intervention to repair the leaking valvular pump function is required (1,2). The traditional surgical modality is mitral valve repair via sternotomy. However, technology marches on, especially in cardiac surgery ORs, such that far less invasive mini-right thoracotomy techniques are rapidly gaining traction as a methodology of choice with equivalent results yet much less morbidity of post-op pain as well as shorted hospital stays (1). In fact, even newer and less invasive techniques have been studied and do not require thoracotomy at all. These are the transvalvular procedures and transcatheter mitral valve replacement procedures where the disease mitral valve is approached via a transfemoral artery canulization into and across the mitral valve where the surgeon then repairs, replaces or places chips of the mitral valve (4). For now, this method is usually reserved for patients with significant risk for any thoracotomy type surgery, but it seems only a matter of time and experience before this safer technique is more broadly applied.1,4

A point that must be mentioned is that despite the use of words like "safer” and "easier", MR surgery is a difficult procedure with one study noting that "the most important factor of long-term success for mitral valve repair is the experience and expertise of the surgeon" (1). In fact, there are numerical definitions in place to define "experience" and "expertise": surgical

centers results of successful repairs in more than 95% of cases; operative mortality less than 1%; center having performed more than 50 cases/year and individual surgeons more than 5 cases/year. (1).

Regarding the management of secondary MR, these patients often have significant coronary disease and LV disease, which results in non-closure of the mitral valve. They are often very poor surgical candidates and do poorly even if the survive the surgical procedure. Initial management is aggressive medical means to improve LV function with CHF therapeutics of ACE inhibitor, beta blockers, anti-arrhythmic maneuvers to name a few. Surgery is only considered after a 3-month course fails (3). Whether to move forward with a surgical procedure, as well as the type of the procedure to perform, is determined by cardiologists and cardiac surgeons. These are difficult and risky decisions to be sure (3,5,6). The presented patient had primary mitral valve disease causing her MR, not LV disease, and the depth of discussion on surgical candidacy and procedure was much clearer cut.

Patient follow-up. Several consultations (internist, cardiologist and cardiac surgeon) confirmed the new onset of easy fatigue and dyspnea on exertion.

A new set of echocardiography and Doppler studies were essentially unchanged with EF hovering around 60% and LV systolic dimension in the high 30 mm ranges. The previous workup already had confirmed the presence of severe MR.

Surgical intervention and repair were recommended and the patient agreed. At a large referral center hospital regarding cardiac valvular surgery, she underwent a mini-right thoracotomy approach mitral valve repair procedure with resection of the flail and prolapsing posterior leaflet with tightening of the annular ring. At the end of the procedure, transesophageal echocardiogram showed, excellent results with essentially no residual MR.

Following the procedure, she had several episodes of transient atrial fibrillation, which reverted to sinus with cardioversion. She was discharged on Day 5 in sinus rhythm but was placed on apixaban for now as a precaution. At 6 weeks post-op, she remains well with ever-diminishing thoracotomy pain, in sinus rhythm. Although still in cardiac physical therapy, and still somewhat limited regarding intensity of activity, she has no exertional dyspnea of other cardiac sign or symptoms.

What's the take home? Mitral regurgitation currently the most common valvular heart disease in the United States with a prevalence approaching 10% in adults older than 75 years (1). These numbers can be expected to increase with the aging of the population.

Mitral regurgitation is divided into two pathophysiologies: primary MR and secondary MR. Primary MR is due to intrinsic degenerative changes in the valve itself resulting in prolapse leaflets with flail or less commonly in modern times valve damage with dysfunction from rheumatic fever scarring. Secondary MR is due to CHF heart disease of whatever cause, although usually ischemic coronary disease, causing cardiac dilatation of the LV with

papillary muscle failure and non-coarctation/closure of the mitral valve leaflets. Of equal importance is the difference in therapies wherein primary MR is addressed with surgical repair or replacement while secondary MR will have initial medical management aimed to improve LV function since they do very poorly with surgery. Firm echocardiographic numbers are utilized to determine the presence on severe MR (regurgitant volume greater than 50% of stroke volume) and as criteria for indication for surgery in primary cases (EF less than 60% and or LV volume greater than 40 mm).

As is so common in cardiology today, superb technological advances are driving less invasive and safer methods of valve repair from sternotomy to mini thoracotomy to transvalvular repairs via transfemoral catheters. In the hands of reference center teams with experience and expertise in mitral valve surgery success rates more than 95% with surgical mortality less than 1% can be achieved.

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CITATION
Rubin RN. A 75-year-old woman with fatigue and a longstanding heart murmur. *Consultant*. 2025;65(12):DOI:10.25270/con.2025.12.000001

DISCLOSURES
The author reports no relevant financial relationships.

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