

Acute Mitral Regurgitation- An Easy-to-Miss but Critical-to-Diagnose Condition

CLINICAL VIGNETTE

Acute Mitral Regurgitation- An Easy-to-Miss but Critical-to-Diagnose Condition

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Case Report

A 71-year-old male with history of mitral valve prolapse with mild mitral regurgitation (MR) seen on echocardiogram 4 years prior, presented to the emergency room with rapidly progressive dyspnea on exertion the night prior to admission. He developed shortness of breath at rest with orthopnea and paroxysmal nocturnal dyspnea. He was free of exertional chest pain, denied palpitations, light-headedness, presyncope, or syncope. He had no lower extremity edema, recent fevers, chills, night sweats, or dental work.

Prior to the day of admission, he was able to walk 30 minutes daily without exertional chest pain or dyspnea. His past medical history otherwise included benign prostatic hypertrophy, with prior TURP. He was a lifelong non-smoker, rarely drank alcohol, and denied drug use. Family history was negative for premature coronary disease or sudden cardiac death.

Upon arrival to the emergency room, he was tachypneic and hypoxic at 88% on room air. His blood pressure was 101/62 with heart rate of 107, and respiratory rate 34 breaths per minute. Physical exam was remarkable for respiratory distress, tachypnea, tachycardia, elevated jugular venous pulsation to the angle of the jaw, and a loud blowing 4/6 holosystolic murmur heard throughout the precordium but loudest at apex radiating to axilla and back. Diffuse crackles were present throughout his lung fields.

Laboratory values showed mildly elevated troponin peaking at 0.36, white blood cell count 15.7, hemoglobin 13.3, hematocrit 39.1, platelet count 148, unremarkable chemistry panel,

abnormality. Chest x-ray revealed pulmonary vascular congestion and interstitial edema.

Echocardiogram revealed normal left ventricular size with hyperdynamic function, mild left atrial enlargement, mitral valve prolapse and likely flail

portion of the posterior leaflet with severe anteriorly directed MR. There was moderate to severe tricuspid regurgitation with severe pulmonary hypertension.

Flail posterior mitral valve leaflet

He was diagnosed with acute-on-chronic MR suspected to be due to ruptured chordae in the setting of mitral valve prolapse. He was treated with IV nitroprusside and IV furosemide drips. The patient continued to decompensate and required intubation. Cardiac Catheterization found no significant coronary artery disease and an intraaortic balloon pump was placed. Right sided hemodynamics showed a right atrial pressure of 18, right ventricular pressure of 65/11, PA pressure of 53/18 with a mean of 40, and a pulmonary capillary wedge pressure of 24. His mixed venous saturation was 88% on 100% FiO₂.

After further diuresis and stabilization, he underwent complex mitral valve repair with quadrangular resection of the P3 segment, triangular resection of P2 segment and a 30mm ring annuloplasty. He also underwent MAZE procedure as he developed atrial fibrillation during the hospitalization.

Discussion of Acute Mitral regurgitation (AMR):

There are two basic mechanisms for acute mitral regurgitation (AMR)- ruptured chordae tendinae or papillary muscle rupture¹. Ruptured chordae tendinae can result from mitral valve prolapse (myxomatous disease)², infective endocarditis, trauma, rheumatic valvular disease, or spontaneous rupture. Rupture of the papillary muscle can be due to trauma, acute myocardial infarction, or papillary muscle displacement from myocardial ischemia/infarction.

The hemodynamic changes in AMR are more severe than those in chronic MR, primarily related to the lack of time for the left atrium and ventricle to adapt. In AMR, the left atrium is usually not compliant (like it is in chronic MR) and therefore the sudden and dramatic increase in left atrial volume leads to an abrupt pressure elevation which is immediately reflected into the pulmonary circulation, leading to pulmonary edema. Additionally, because the left ventricle is not dilated (as it is in chronic MR), most of the blood pumped by the ventricle goes backward across the mitral valve, limiting the effective forward flow and decreasing cardiac output. The acute drop in cardiac output may lead to cardiogenic shock. The neurohumoral response to the reduction in cardiac output is an increase in vascular resistance, which further worsens the mitral regurgitation.³

AMR usually presents as an emergency with the acute onset and rapid progression of dyspnea

dizziness, and altered mental status. Occasionally, the elevated pulmonary artery pressures lead to acute right-sided heart failure. In clinical practice, the clinical presentation of AMR often mimics an acute pulmonary process (such as infection or acute respiratory distress syndrome) and the physical examination findings often are subtle. Therefore it is imperative to include acute valvular regurgitation in the differential diagnosis of any patient presenting with pulmonary decompensation and early echocardiography to make this diagnosis.⁴

On physical exam, the patients are often in pulmonary edema, appear diaphoretic, and have a rapid thready pulse (due to reduction in forward output). The neck veins may be distended if there is an increase in right-sided pressure and may demonstrate a “v” wave if the elevated right ventricular pressure leads to tricuspid regurgitation. The left ventricular impulse is hyperdynamic but not displaced (unless AMR is superimposed on chronic MR). Up to 50% of patients with AMR (particularly ischemic mitral regurgitation) may not have an appreciable murmur.⁵ When present, the murmur may be early systolic, midsystolic, or holosystolic. Since the left atrial pressure rises so rapidly and the pressure gradient between the left ventricle and left atrium diminishes quickly, the murmur is often soft, low pitched, and decrescendo, ending before the A2 heart sound. An S3 is commonly present but may be difficult to appreciate if tachycardia is present.

Acute mitral regurgitation (MR) is a medical and surgical emergency. Generally, the definitive treatment is prompt surgical intervention. However, medical therapy may be needed to support the patient while the diagnosis is made and until surgery can be performed.⁶

Medical stabilization options include intravenous nitroprusside which reduces mitral regurgitation by reducing systemic vascular resistance and improving mitral valve competence as the left ventricular size falls^{7,8}. The decrease in MR severity increases forward cardiac output and diminishes pulmonary congestion⁶. Inotropes such as dobutamine may also be helpful in improving cardiac output.

Another temporizing option to stabilize the patient prior to surgery is placement of an intra-aortic balloon pump to reduce left ventricular afterload, which decreases regurgitant volume and increases forward cardiac output, while simultaneously increasing diastolic and mean aortic pressure.

Prompt mitral valve surgery is recommended for AMR. The risk of surgery is high, with mortality rates as high as 50 percent.³

Acute MR caused by chordal rupture, as in this patient example, often can be treated early with mitral valve repair⁹. Mitral valve repair is associated with a lower operative mortality, improved preservation of left ventricular function, and better long-term survival when compared to mitral valve replacement. Furthermore, the risks of a prosthetic valve and anticoagulation are avoided³.

Conclusions and points to remember about acute mitral regurgitation:

Acute severe valvular regurgitation is a surgical emergency, but accurate and timely diagnosis can be difficult. Although cardiovascular collapse is a common presentation, examination findings to suggest acute regurgitation may be subtle as approximately 50 percent of patients with moderate to severe MR have no audible murmur. A high index of suspicion and early echocardiography is essential to making the diagnosis.

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