Acute Mitral Regurgitation: The Dreaded Masquerader

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INTRODUCTION

Unilateral pulmonary edema in patients with hypoxemic respiratory failure is usually secondary to infectious etiology. However, cardiogenic unilateral pulmonary edema is a rare yet important entity that is often misdiagnosed initially, leading to significant delays in treatment. Cardiogenic unilateral pulmonary edema is often secondary to acute mitral regurgitation (MR), a life-threatening hemodynamic abnormality that requires urgent medical attention and usually surgical intervention. Acute posterior leaflet flail has been associated with right-sided unilateral pulmonary edema. However, anterior leaflet flail leading to right upper lobe opacity is very rare.

CASE PRESENTATION

A 73-year-old woman presented to the emergency department with progressively worsening shortness of breath over 5 days, associated with nonproductive cough, fever, and chills. Her medical history was significant for poorly controlled hypertension, diabetes, nonobstructive coronary artery disease, and complete heart block with wide QRS complex escape rhythm, for which she declined cardiology referral. The patient collapsed on her way from the hospital's parking lot to the emergency department, where she was found to be in severe respiratory distress. Initial vital signs were a respiratory rate of 28 breaths/min, a heart rate of 52 beats/min, blood pressure of 70/50 mm Hg, and temperature of 98.1°F. Her oxygen saturation was 79% on room air. Physical examination revealed bilateral crackles and normal heart sounds with no appreciable murmur or gallop. Chest radiography demonstrated a large right upper lobe opacity and bilateral interstitial infiltrates (Figure 1).

Because of worsening respiratory failure, the patient was intubated and initiated on mechanical ventilation. Initial investigations demonstrated leukocytosis (15,300/μL), elevated lactate (13.5 mmol/L), mildly elevated cardiac troponin I (0.188 ng/dL), and markedly elevated B-type natriuretic peptide (2,002 pg/mL) prior to intubation. The patient remained afebrile despite subjective sensation of fever and chills. Blood, sputum, and urine cultures were obtained on admission and did not demonstrate any growth. Electrocardiography revealed sinus bradycardia with complete heart block and wide QRS complex escape rhythm. In the medical intensive care unit, the patient received antibiotic therapy and intravenous fluids for presumed diagnosis of multilobar pneumonia. After an initial hemodynamic improvement, the patient's course rapidly deteriorated, with development of hypotension requiring initiation of norepinephrine and transcutaneous pacing.

Subsequently, transthoracic echocardiography demonstrated normal left ventricular ejection fraction, estimated at 55%–65%, with evidence of restrictive filling pattern (Video 1). Left ventricular end-diastolic diameter and end-systolic diameter were measured at 50 and 35 mm, respectively. The mitral valve leaflets appeared thickened. There was severe flail motion of the anterior mitral leaflet, with evidence of ruptured chordae tendineae (Figure 2, Videos 2 and 3). There was a severe eccentric MR directed toward the posterior wall of the left atrium (Figures 3 and 4, Video 4). The left atrium was markedly dilated (Video 1). The right ventricle was dilated, with reduced systolic function (Video 1). Pulmonary artery systolic pressure was estimated in the range of 60–65 mm Hg (Figure 5). Transesophageal echocardiography was performed to better differentiate between infective endocarditis and myxomatous degeneration, and to exclude the possibility of acute ischemic MR considering the recent diagnosis of complete heart block. Transesophageal echocardiography confirmed anterior mitral leaflet flail with evidence of ruptured chordae tendineae and intact papillary muscles (Figure 6, Videos 5 and 6). The valve morphology was consistent with myxomatous proliferation. The MR jet was eccentric, directed toward the posterior wall of the left atrium and the right upper pulmonary vein, with systolic reversal of flow in the pulmonary vein (Figures 7 and 8, Video 7). Applying the volumetric flow equation method at the mitral valve annulus and left ventricular outflow tract, the regurgitant volume and regurgitant fraction were calculated to be 66 mL and 62%, respectively.[1] The effective regurgitant orifice area by the continuity method was calculated to be 0.6 cm^2.[1] There was no evidence of valvular vegetation. Subsequently, the working diagnosis was changed from pneumonia to cardiogenic shock secondary to acute MR caused by ruptured chordae tendineae. The etiology of mitral valve disease was presumed to be myxomatous degeneration.

The patient was urgently taken to the cardiac catheterization laboratory for further hemodynamic evaluation and stabilization. An intraaortic balloon pump and temporary transvenous pacemaker were placed for hemodynamic support. Right-heart catheterization revealed a mean pulmonary artery pressure of 44 mm Hg and pulmonary capillary wedge pressure of 34 mm Hg. Using the assumed Fick method, the cardiac index was calculated at 2.1 L/min/m^2.[2] Following a discussion with the cardiovascular surgery team, coronary angiography was not performed, as only emergent mitral valve replacement was contemplated in this critically ill patient. Intraoperatively, the patient was found to have ruptured chords of the middle segment of the anterior mitral valve leaflet (A2). The mitral valve was replaced with a 27-mm Mosaic bioprosthetic porcine pericardial valve prosthesis (Medtronic, Minneapolis, MN). Pathologic examination of the mitral

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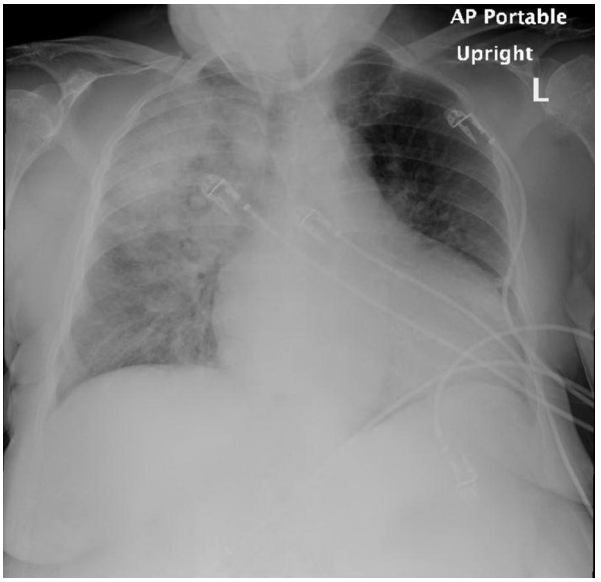

Figure 1 Chest radiograph showing right upper lobe opacity with bilateral interstitial infiltrates.

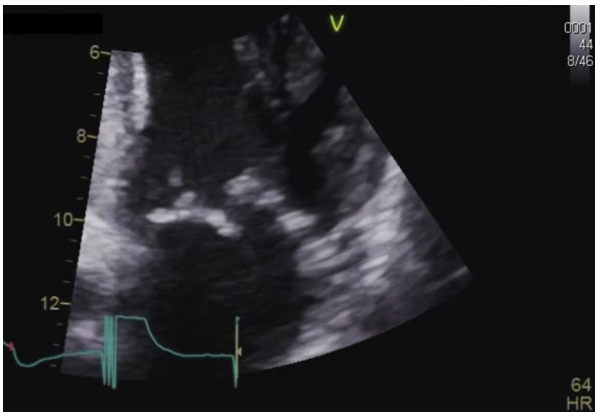

Figure 2 Transthoracic echocardiography, apical four-chamber view, demonstrating flail motion of the anterior leaflet of the mitral valve.

valve specimen revealed fibrocalcific valve tissue, myxoid changes, and no evidence of infection, thus confirming myxomatous degeneration as the etiology of mitral valve disease.

The patient was extubated and weaned off vasopressors on the fourth postoperative day. Postoperative transthoracic echocardiography demonstrated a normally functioning bioprosthesis in the mitral position and normal left ventricular systolic function (Video 8). The patient also underwent dual-chamber permanent pacemaker placement and was discharged home in a stable condition and continues to receive outpatient clinic follow-up.

DISCUSSION

We describe the case of an elderly woman who presented with right upper lobe opacity, constitutional symptoms, and hypotension, masquerading as right upper lobe pneumonia. Transthoracic and transesophageal echocardiography were crucial to arriving at the correct diagnosis of myxomatous degeneration and flail anterior mitral

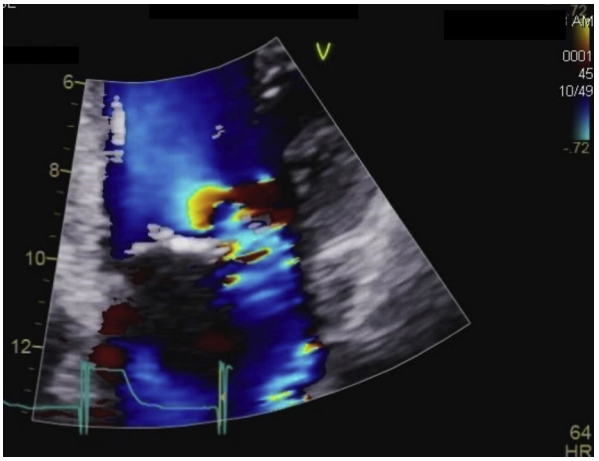

Figure 3 Transthoracic echocardiography, apical four-chamber view, with color Doppler demonstrating an eccentric, posteriorly directed MR jet.

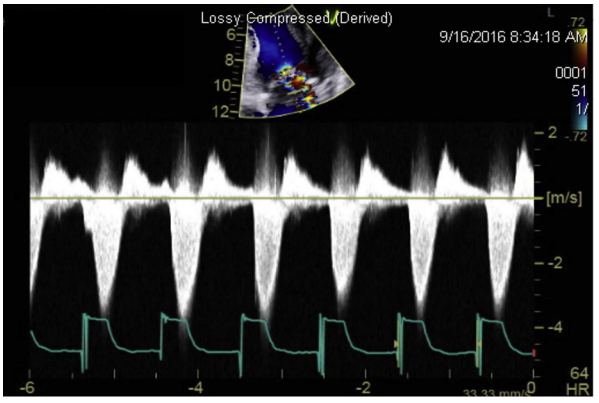

Figure 4 Transthoracic echocardiography, apical four-chamber view with continuous-wave and color Doppler across the mitral valve, demonstrating a dense, triangular, early peaking MR jet.

valve leaflet leading to severe acute MR, localized pulmonary edema, and cardiogenic shock.

In our patient, the diagnosis of acute MR was delayed by >36 hours from the initial presentation. This case highlights the importance of considering cardiogenic pulmonary edema as a potential etiology of unilateral pulmonary opacity.[3,4] In a study of 131 patients with severe MR, Schnyder *et al.*[5] reported that the prevalence of right upper lobe opacity was 9.2% (12 patients). Moreover, in a study of 869 consecutive patients admitted with cardiogenic pulmonary edema, Attias *et al.*[4] found that 18 patients (2.1%) had unilateral pulmonary edema on chest radiography, of whom 16 (89%) had right-sided unilateral pulmonary edema, with the right upper segment being the most commonly affected. Alternatively, left-sided unilateral pulmonary edema was seen only in two patients (11%). In this study, all 18 patients with unilateral pulmonary edema had severe MR. Among these patients, 16 (89%) had posterior leaflet prolapse, and only two (11%) had anterior leaflet prolapse. There were 10 patients with organic MR, among whom eight had right-sided pulmonary edema on chest radiography from posterior leaflet prolapse, one patient had right-sided pulmonary edema from anterior leaflet prolapse, and one patient had left-sided pulmonary edema from anterior leaflet prolapse. Among the eight patients with functional MR, seven had right-sided