Acute Mitral Regurgitation Presenting with Right Upper Lobe Opacification

John Wallis, MD and Mark Decaro, MD

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ABSTRACT

We describe a rare entity in which acute mitral regurgitation causes asymmetric findings on chest radiograph. The patient presented with rapid-onset respiratory failure from flash pulmonary edema. She had unilateral infiltrates on chest radiograph, which evoked infectious etiology. However, we identified a flail mitral valve leaflet, for which the patient received an emergent mitral valve replacement. Fortunately, she made a full recovery. We discuss the mechanism of the asymmetric chest radiograph findings, which we were able to confirm using a transesophageal echocardiogram.

CASE PRESENTATION

A 62-year-old female presented to the emergency department in acute respiratory failure. Her past medical history included obesity, asthma, hypertension, diastolic heart failure, and mild-moderate mitral regurgitation. She arrived unresponsive via EMS with an oxygen saturation of 23% for which she was immediately intubated. She required maximal ventilator settings (FiO2 100%, PEEP 16) and neuromuscular blockade to achieve adequate oxygenation.

Initial chest radiograph demonstrated diffuse opacities with dense consolidation in the right upper lobe (Figure 1). Subsequent chest radiographs showed near-complete opacification of the right hemithorax (Figure 2).

These asymmetric lung findings were suspicious for infectious etiology, including Covid-19 or bacterial pneumonia causing ARDS. The patient’s family informed us that she had felt normal upon awakening, and developed sudden shortness of breath when ambulating to the bathroom. This history of rapid progression is less consistent with infectious causes. She was afebrile and had no respiratory secretions. She tested negative for Covid-19, including from a tracheal aspirate. She had a mild leukocytosis of 12.4 with only 52% neutrophils, normal procalcitonin (0.05 ng/mL), and an unimpressive C-reactive protein (0.90 mg/dL). These results were inconsistent with severe infection, so we investigated cardiac etiologies.

Initial ECG showed sinus tachycardia at 132 bpm with no ischemic changes. High-sensitivity troponin was 66 ng/L initially, increased to 144 ng/L, and then peaked at 151 ng/L before downtrending. She was treated for acute coronary syndrome with aspirin, statin, and an unfractionated heparin drip, although her troponin elevation was most likely a result of profound hypoxia. Her pro-BNP
was 246 pg/mL. A transthoracic echocardiogram showed normal biventricular function, but detected a flail anterior mitral valve leaflet with significant mitral regurgitation with an eccentric jet. The patient was growing more hypotensive requiring vasopressors, her extremities were becoming cool, and she was anuric despite diuretics, consistent with cardiogenic shock. Her respiratory status had somewhat stabilized.

The patient was emergently transferred to our main campus where she had an intra-aortic balloon pump placed for afterload reduction. A transesophageal echocardiogram confirmed severe mitral regurgitation and showed a ruptured anterior papillary muscle (Figure 3). This TEE showed severe systolic flow reversal in the right upper pulmonary vein, with only mild systolic flow reversal in the three other pulmonary veins. She underwent coronary angiography showing no obstructive coronary artery disease. She was taken to the OR for an emergent bioprosthetic mitral valve replacement. Afterwards, her clinical status improved, she began making urine, her pulmonary artery pressures corrected, and she was extubated 72 hours after presentation. Subsequent chest x-rays showed rapid resolution of the asymmetric infiltrates (Figure 4).

### DISCUSSION

This was a dramatic presentation of spontaneous, non-ischemic papillary muscle rupture causing severe acute mitral regurgitation, with respiratory failure from resultant flash pulmonary edema. She was critically ill within minutes of symptom onset. The patient is fortunate to have a positive outcome. Of particular interest is the initial chest radiograph showing asymmetric opacification with dense consolidation of the right upper lobe. This appearance sent us down an infectious diagnostic pathway, which could have delayed the diagnosis.

There is previous literature describing this phenomenon. Focal or unilateral pulmonary edema has been described in patients with mitral regurgitation from numerous causes, including spontaneous valve perforation, valve perforation due to infectious endocarditis, transient papillary muscle dysfunction due to myocardial ischemia, or spontaneous papillary muscle or chordae tendineae rupture. Case reports show that this finding is often initially mistaken for pneumonia or other respiratory illnesses.

Schnyder et al. found that 9% of patients with severe mitral regurgitation had chest radiograph findings that were “localized or predominant” in the right upper lobe. Attias et al. studied 869 patients admitted with cardiogenic pulmonary edema, and found that 2.1% had unilateral pulmonary edema (UPE). Notably, all of the patients with UPE were found to have severe mitral regurgitation. Of patients with cardiogenic pulmonary edema and severe MR, 75% had bilateral findings and 25% had unilateral findings.
Only 6% of patients with bilateral pulmonary edema received antibiotics, whereas 61% of patients with unilateral pulmonary edema received antibiotics. This shows that focal chest radiograph findings often invoke infectious etiology among clinicians.

Outcomes are widely divergent depending on initial imaging findings. Patients with UPE were found to have significantly lower blood pressure on presentation, higher use of NIPPV/IPPV, and more frequent use of vasopressors/inotropes. Patients with severe MR had an in-hospital mortality of 39% when presenting with unilateral findings, compared to 6% when presenting with bilateral findings. Therefore, presentation with focal radiographic findings not only delays the diagnosis, but has been shown to correlate with worse outcomes and death.⁸

There is a proposed mechanism for localized pulmonary edema due to MR. Typically, systolic or diastolic left ventricular failure leads to increased pressure within the left atrium, which is transmitted symmetrically to each of the pulmonary veins. This leads to increased hydrostatic pressure within pulmonary capillaries, causing a uniform degree of pulmonary edema throughout the lungs.

It is thought that asymmetric pulmonary edema is due to a mitral regurgitant jet that propels blood selectively towards the orifice of a particular pulmonary vein within the left atrium. If a regurgitant jet causes increased pressure within that pulmonary vein, it would transmit increased hydrostatic pressure selectively to the pulmonary capillaries that drain into that pulmonary vein, causing focal edema.

This mechanism is supported by Kashiura et. al, who described two cases of unilateral pulmonary edema from severe acute MR. The first case had an eccentric jet blowing towards the right side of the left atrium and presented with right-sided opacities. The second case had a jet blowing towards the left side of the left atrium, and that patient presented with left-sided opacities.² This indicates that the direction of the regurgitant jet affects which pulmonary veins are selectively pressurized, which correlates with focal edema. In our case, the regurgitant jet was directed towards the right upper pulmonary vein, which drains the right upper and middle lobes, causing right upper lung opacification. The patient’s TEE confirmed severe systolic flow reversal within the right upper pulmonary vein, which provides evidence supporting this mechanism.

As in our case, acute mitral regurgitation can present with sudden life-threatening respiratory failure and cardiogenic shock, so prompt diagnosis is critical. It is often misdiagnosed as pneumonia or other respiratory illness. Patients with this finding have worse outcomes and delays in diagnosis and treatment compared to similar patients with bilateral pulmonary edema.⁹ In our case, the key to the diagnosis was the reported history of sudden-onset dyspnea that progressed to respiratory failure within minutes.

In conclusion, acute mitral regurgitation should be considered in the differential diagnosis for patients with focal right upper lobe opacities in the appropriate clinical context – such as sudden-onset hypoxic respiratory failure, especially if vitals, exam, and laboratory biomarkers are inconsistent with severe infection. Awareness of this entity could be life-saving. Our findings support the previously proposed mechanism of a regurgitant jet selectively pressurizing the right upper pulmonary vein, leading to right upper lobe edema.

REFERENCES