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Acute Mitral Regurgitation Presenting as Unilateral Pulmonary Edema

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Abstract

Acute mitral regurgitation caused by papillary muscle rupture is a rare and life-threatening complication of myocardial infarction. This devastating complication can present as unilateral cardiogenic pulmonary edema which can be easily mistaken for other cardiopulmonary pathology, resulting in delay of care. Proper evaluation and diagnosis is key for obtaining timely surgical evaluation. Here we present a case of an 87-year-old male with a past medical history of coronary artery disease status post percutaneous intervention with one stent, cerebrovascular accident status post left carotid stenting, and Alzheimer's dementia that presented to the emergency department with a chief complaint of exertional dyspnea for three days. In the emergency department, he required 5L O2 by nasal cannula to maintain an oxygen saturation of 93%. He never required supplemental oxygen previously. Physical exam was significant for rales in the right lower lobe. No murmur, jugular venous distention, or pitting edema were noted. Chest imaging revealed right-sided infiltrate versus edema with a small right lower lobe effusion. The left lung was grossly normal on imaging. The patient was admitted to the medicine service and treated with IV antibiotics. Echocardiogram was later performed which showed an LV ejection fraction of 55-60% with new onset severe mitral regurgitation. The patient was urgently taken for cardiac catheterization and found to have critical coronary artery disease. An intra-aortic balloon pump was placed, and surgical evaluation was planned for possible CABG/MVR. However, patient was deemed to not be an appropriate surgical candidate and was subsequently transferred for placement of ventricular assist device and eventual Mitra Clip. Ultimately, the patient decompensated, and was transitioned to hospice care before passing away 22 days after initial presentation.

Keywords

Mitral Regurgitation, Unilateral Pulmonary Edema

Conflict of Interest Statement

None of the listed authors have conflicts of interest to disclose in regard to this case report.

CASE REPORT

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Abstract

Acute mitral regurgitation caused by papillary muscle rupture is a rare and life-threatening complication of myocardial infarction. This devastating complication can present as unilateral cardiogenic pulmonary edema which can be easily mistaken for other cardiopulmonary pathology, resulting in delay of care. Proper evaluation and diagnosis is key for obtaining timely surgical evaluation. Here we present a case of an 87-year-old male with a past medical history of coronary artery disease status post percutaneous intervention with one stent, cerebrovascular accident status post left carotid stenting, and Alzheimer's dementia that presented to the emergency department with a chief complaint of exertional dyspnea for three days. In the emergency department, he required 5L O2 by nasal cannula to maintain an oxygen saturation of 93%. He never required supplemental oxygen previously. Physical exam was significant for rales in the right lower lobe. No murmur, jugular venous distention, or pitting edema were noted. Chest imaging revealed right-sided infiltrate versus edema with a small right lower lobe effusion. The left lung was grossly normal on imaging. The patient was admitted to the medicine service and treated with IV antibiotics. Echocardiogram was later performed which showed an LV ejection fraction of 55−60% with new onset severe mitral regurgitation. The patient was urgently taken for cardiac catheterization and found to have critical coronary artery disease. An intra-aortic balloon pump was placed, and surgical evaluation was planned for possible CABG/MVR. However, patient was deemed to not be an appropriate surgical candidate and was subsequently transferred for placement of ventricular assist device and eventual Mitra Clip. Ultimately, the patient decompensated, and was transitioned to hospice care before passing away 22 days after initial presentation.

Keywords: Mitral regurgitation, Unilateral pulmonary edema

An 87-year-old male with a past medical history of coronary artery disease status post percutaneous intervention with one stent, cerebrovascular accident status post left carotid stenting, hypertension, hyperlipidemia, mild Alzheimer's dementia, gout, and chronic kidney disease stage IV presented to the emergency department with a chief complaint of exertional dyspnea for three days. The patient denied any known trigger or precipitating event. Prior to this, he had been rather active, playing golf and going on walks several times per week, which he had not been able to do since symptom onset. Since that time, he had endorsed a cough productive of frank blood, which had subsided into pink-tinged phlegm by presentation. He also complained of a stomach pain three days prior that was precipitated by eating a meal and had not recurred. He denied any other chest pain, palpitations, diaphoresis, or swelling. He reported that he was a previous pack-per-day smoker but quit twenty years ago. The patient denied any history of known congestive heart disease.

On the day of presentation, the patient felt that his symptoms were unbearable and he scheduled and appointment with his outpatient physician who then recommended that the patient present to a hospital. During the initial encounter the patient mentioned that three days prior he had experienced, what he described, as nonspecific upper abdominal discomfort that ultimately subsided within a few hours. He

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could not identify any apparent triggers to this pain and stated it was vague. He then stated that he felt slightly short of breath in the days following when became increasing more noticeable, prompting to contact his primary care physician. In the emergency department, the patient was hypertensive, tachypneic, and saturating at 93% on 5L nasal cannula. He had never required supplemental oxygen prior. Physical exam was significant for rales in the right lower lobe. No murmur, jugular venous distention, or pitting edema were noted. Laboratory studies were significant for an elevated BUN/creatinine at 76/4.93, elevated high-sensitivity troponin at 1918, and elevated BNP at 2461. Viral studies, procalcitonin, and d-dimer results were within normal limits. Electrocardiogram showed right bundle branch block which was a present in a historical EKG as well as QT prolongation. Chest imaging revealed right-sided infiltrate versus edema with a small right lower lobe effusion (Fig. 1), and grossly clear left lung. The patient was admitted to the medicine service, started on IV Rocephin 1g and azithromycin 500 mg. His home antihypertensives were resumed.

The following morning, patient was met at bedside lying down comfortably. He had no acute complaints and was able to pull himself up unassisted to sit upright in bed without difficulty. He endorses continued shortness of breath with exertion but stated his breathing had not worsened. Overnight troponins had continued to decrease to 1421, down from 1647 to 1918. He continued to require 5L O2 throughout the day to maintain a saturation of 92%. The patient experienced acute worsening shortness of breath with oxygen desaturations to 80% on 5L NC. Repeat chest imaging showed worsening edema of the right lung (Fig. 2). Echocardiogram was performed, and showed an LV ejection fraction of

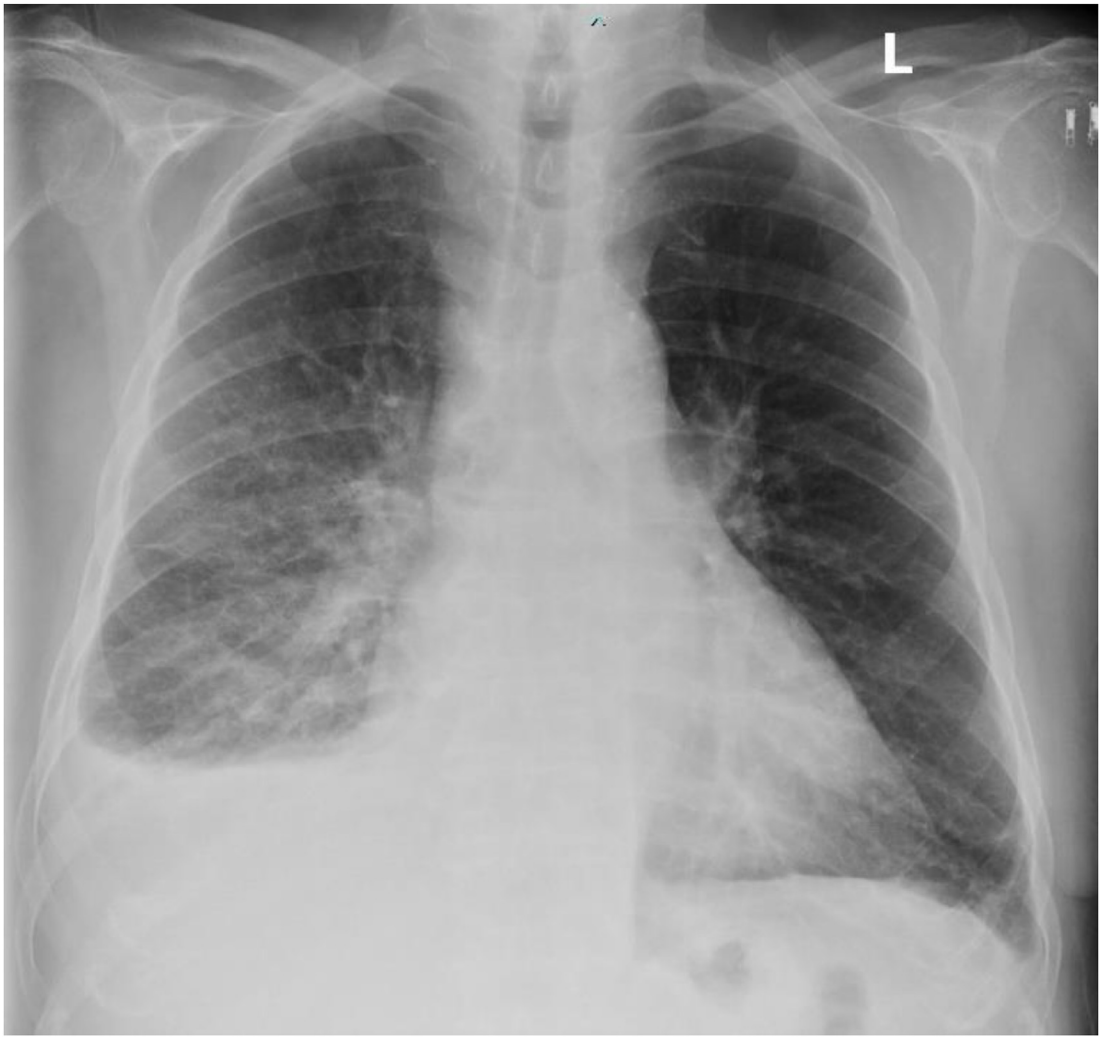

Fig. 1. Portable CXR (Admission).

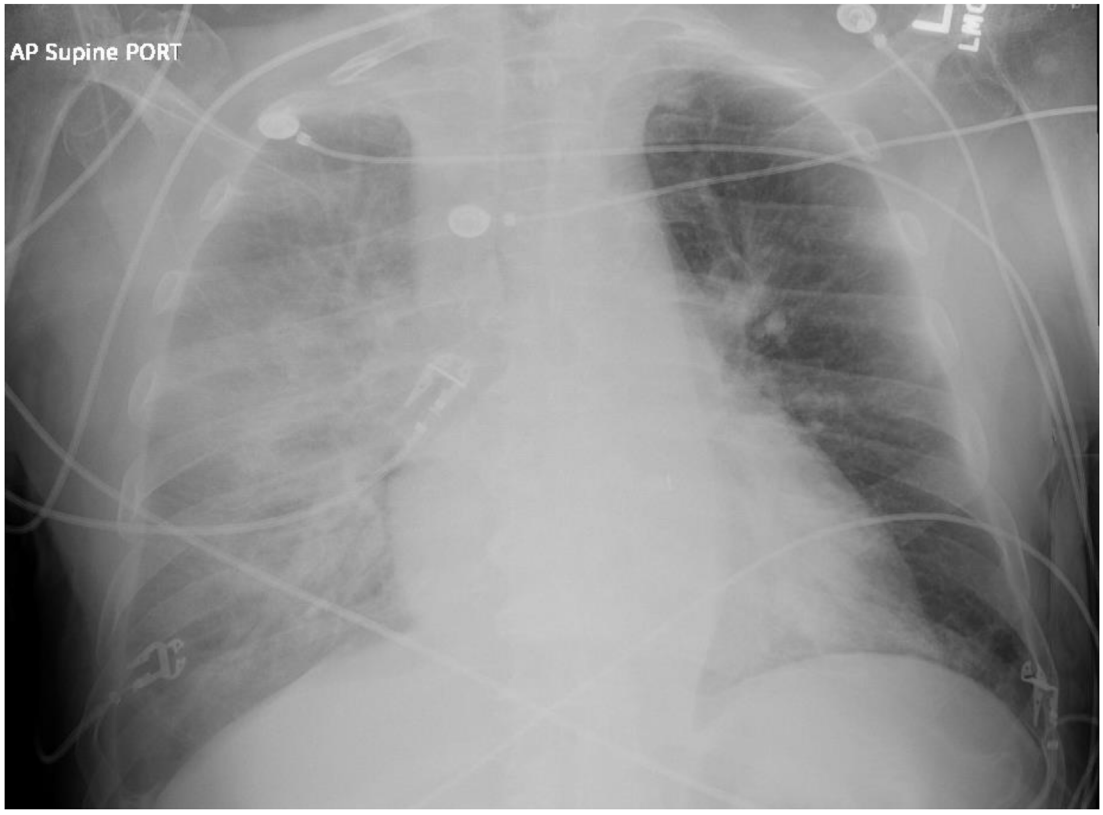

Fig. 2. Portable CXR (Hospital Day 1).

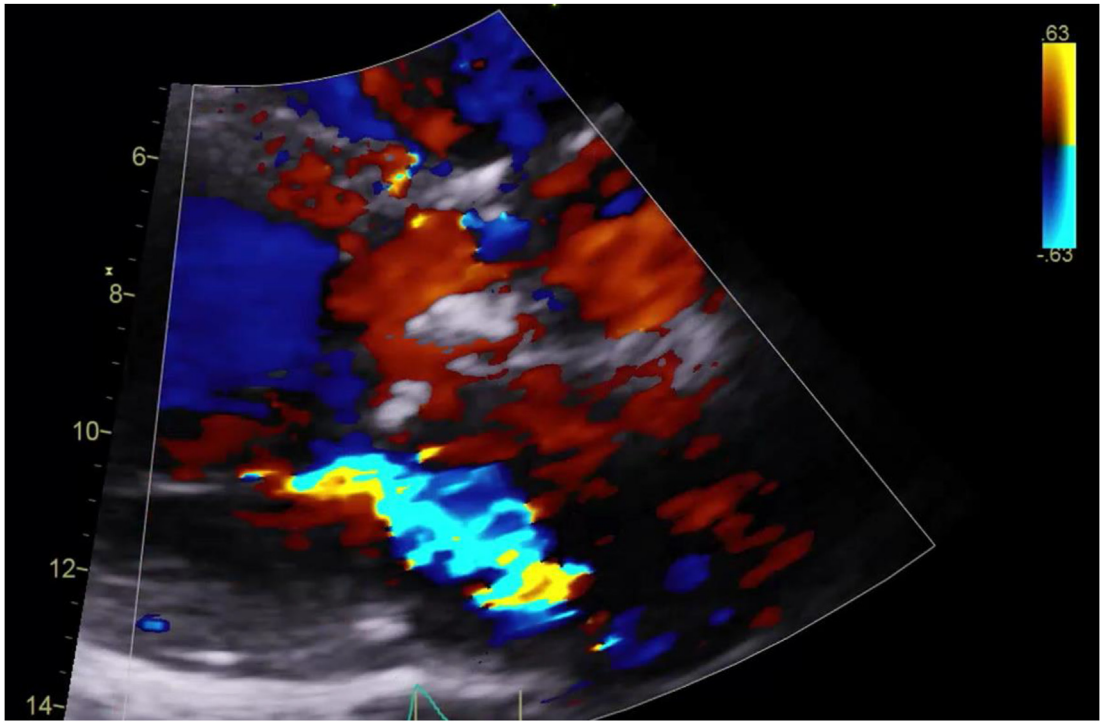

Fig. 3. Echocardiogram—Parasternal long axis showing turbulent flow through mitral valve.

55−60% with new onset severe mitral regurgitation (Figs. 3 and 4).

The patient was urgently taken for cardiac catheterization which revealed 100% occlusion of the right coronary artery, 95% occlusion of the left anterior descending artery, and elevated left ventricular end diastolic pressure of 40 mm Hg. Critical coronary artery disease involving the mid LAD at the diagonal bifurcation was found, as well as a chronic total occlusion of the RCA receiving collaterals from the LAD (Fig. 5). This completely obstructed right coronary was the culprit lesion inciting papillary muscled rupture with the sequalae of cardiogenic shock. An intra-aortic balloon pump was placed via the right common femoral artery. Following the procedure, surgical evaluation was planned for possible CABG/MVR. However, patient was deemed to not be an appropriate surgical candidate due to his kidney function and cognitive status, and was subsequently transferred for placement of ventricular assist device and eventual Mitra Clip. Ultimately, however, the patient decompensated and was transitioned to hospice care before passing away 22 days after initial presentation.

Discussion

Acute mitral regurgitation caused by papillary muscle rupture is a rare and life-threatening complication of myocardial infarction. Papillary muscle rupture occurs in 1%−5% of individuals with acute myocardial infarction and generally occurs within one week of the insult, although there have been documented cases of rupture after up to 3 months.[1] The mitral valve is composed of two papillary muscles, the posteromedial and the anterolateral. The anterolateral papillary muscle is supplied by marginal branches of the left circumflex and the left coronary arteries while the posterolateral is supplied by the posterior descending artery only. Due to its single blood supply, the posteromedial papillary muscle is thus more vulnerable to insult. Given that 70%−80% of the population are right-dominant, occlusion of the right coronary is responsible for sequelae from infarct to papillary rupture to regurgitation.[2]

The right superior pulmonary vein drains the right upper and middle lobes, while the right inferior pulmonary vein drains the right lower lobe. Mitral regurgitation results in backflow through these pulmonary veins, generally resulting in bilateral cardiogenic pulmonary edema due to an increase in hydrostatic pressure. Unilateral cardiogenic pulmonary edema comprises only 2% of cases of pulmonary edema and can be easily mistaken other for pathology such as pneumonia.[3] Multiple studies have shown that 9%−25% of individuals who presented with radiographic signs of unilateral pulmonary edema were mainly affected in the right upper

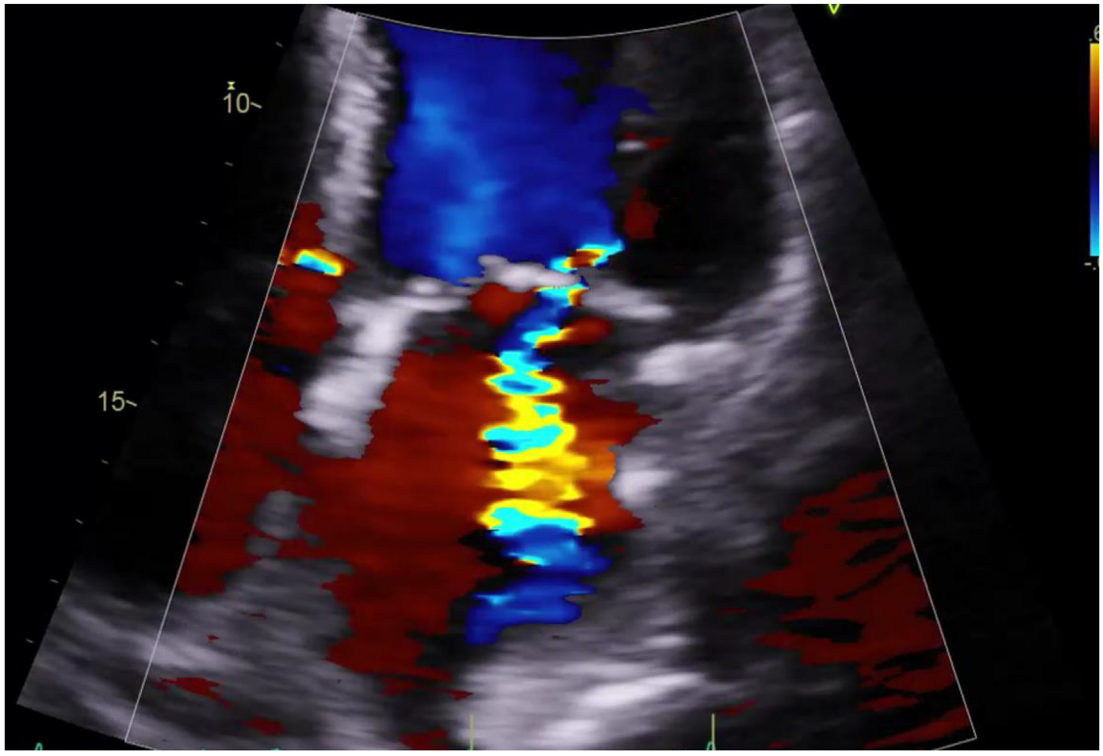

Fig. 4. Echocardiogram−Apical four chamber showing turbulent flow through mitral valve.

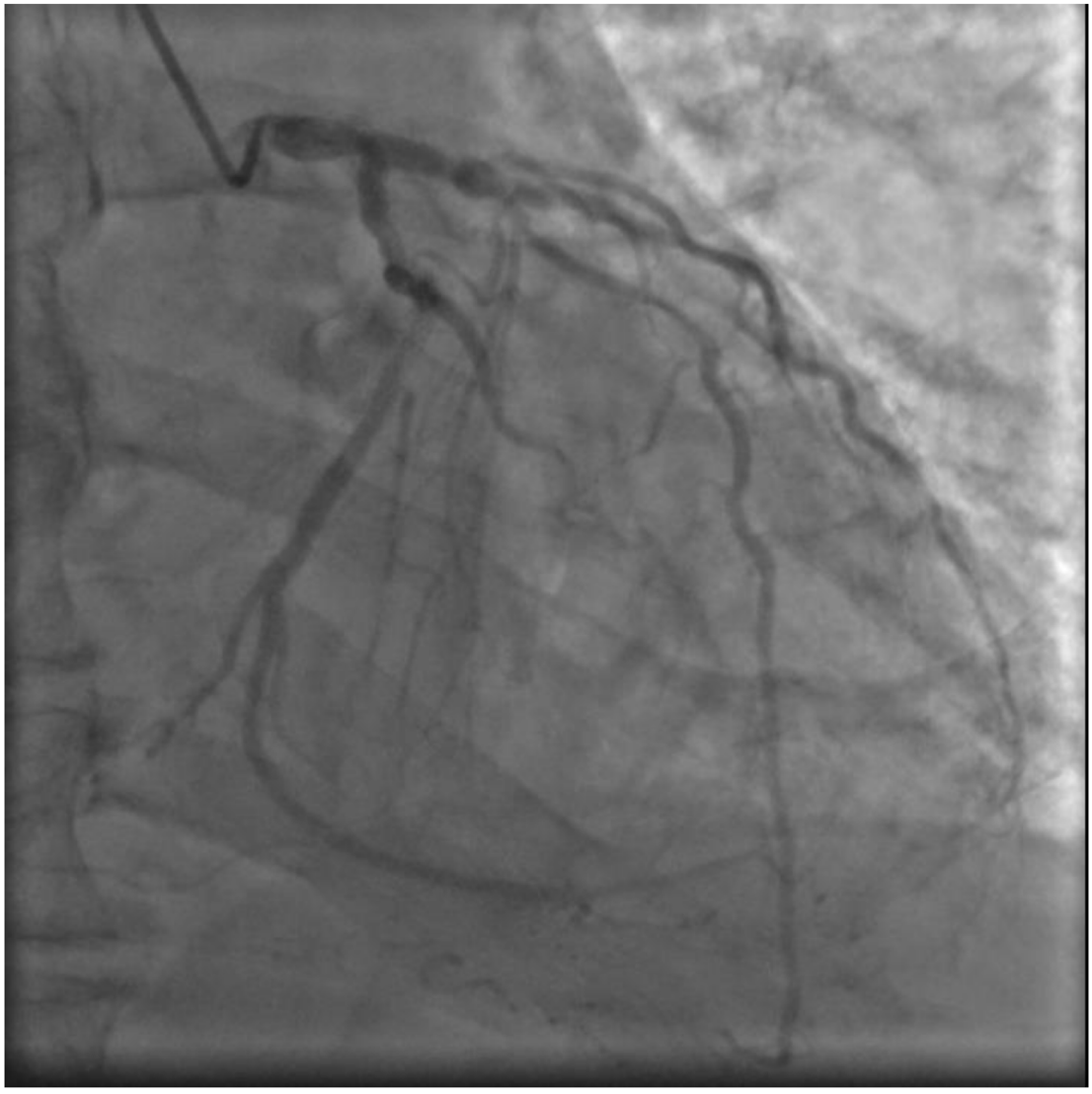

Fig. 5. Cardiac cath with RCA CTO.

lobe.[3–5] This predilection is likely anatomical due to the path of least resistance as blood refluxing through the mitral valve preferentially moves rightwards and posteriorly.[5]

The posteromedial papillary muscle is relatively more vulnerable to injury given its single blood supply. Rupture of this papillary muscle is a known consequence of myocardial infarction, which then results in an acute ischemic mitral regurgitation. Regurgitant flow is directed at the left atrial wall, causing increased hydrostatic pressure in the pulmonary veins, that ultimately results in pulmonary edema. There are fewer than twenty cases in the previous thirty years in which acute mitral regurgitation is described presenting as a unilateral pulmonary edema. The majority of the cases manifest in the upper lobe edema which makes our case unique. Although unilateral cardiac pulmonary edema is a rare presentation comprising just 2% of the cases, there should be a high index of suspicion for mitral valve pathology, as prompt surgical evaluation is key for successful potential valve replacement.

Conclusion

Unilateral pulmonary edema is a rare radiographic presentation of mitral valve disease, with a potentially devastating and life-threatening course. Seemingly innocuous preliminary findings should prompt further investigation, especially in patient with a known history of cardiac disease. A diagnostic stratagem that includes chest radiography, cardiac echocardiography, and timely cardiac catheterization should prove vital to uncovering the

etiology behind unilateral cardiogenic pulmonary edema, so that the appropriate surgical evaluation may be performed.

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Authors contribution

JSP, AK, WO – Manuscript and literature review. KV – Attending physician.

Conflict of interest

None of the listed authors have conflicts of interest to disclose in regard to this case report.

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