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When Unilateral Pulmonary Edema Is Not Pneumonia: Fulminant Acute-on-Chronic Mitral Regurgitation Due to Spontaneous Chordal Rupture in Barlow's Disease

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Nadia Loudiyi [1], Hafsa Erregui [1], Mohamed Malki [1], Zouhair Lakhal [1], Aatif Benyass [1]

1. Cardiology Center, Mohammed V Military Instruction Hospital of Rabat, Mohammed V University, Rabat, MAR

Corresponding author: Hafsa Erregui, hafsaerregui17@gmail.com

Abstract

Unilateral pulmonary edema (UPE) is an uncommon and frequently overlooked manifestation of cardiogenic pulmonary edema that may mimic infectious pneumonia and is strongly associated with severe eccentric mitral regurgitation (MR), particularly in the setting of posterior leaflet prolapse or chordal rupture. We report the case of a 50-year-old woman with no previously known valvular heart disease who presented with abrupt-onset dyspnea at rest, rapidly progressing to severe respiratory distress and orthopnea within 24 hours. On admission, she was hypoxemic, with asymmetric crackles predominantly involving the right lung fields. Chest radiography demonstrated extensive right-sided alveolar infiltrates, initially raising suspicion of pneumonia. However, the absence of fever, systemic inflammatory response, and elevated infectious biomarkers prompted urgent cardiac evaluation. Transthoracic and transesophageal echocardiography revealed severe degenerative MR due to spontaneous chordal rupture, resulting in a flail P2 scallop extending into P3 on a background of Barlow's disease, with a severe eccentric regurgitant jet. Despite initial stabilization with oxygen therapy, intravenous diuretics, and nitrates, the patient rapidly progressed to refractory cardiogenic shock requiring vasoactive and inotropic support. Urgent mitral valve surgery was indicated but could not be performed because of fulminant hemodynamic deterioration, and the patient died approximately two hours after admission to the cardiac intensive care unit. This case highlights a rare and catastrophic presentation of previously unrecognized degenerative MR in which spontaneous chordal rupture precipitated right-sided UPE and rapid hemodynamic collapse despite an apparently preserved left ventricular ejection fraction. It emphasizes that acute severe MR should be promptly considered in patients presenting with unilateral pulmonary infiltrates and acute respiratory failure when infectious features are absent. Early echocardiographic recognition and immediate surgical evaluation are crucial, as the therapeutic window before irreversible hemodynamic collapse may be extremely narrow.

Categories: Cardiology
Keywords: acute severe mitral regurgitation, barlow's disease, mitral valve prolapse, pneumonia mimic, spontaneous chordal rupture, unilateral pulmonary edema

Introduction

Acute severe mitral regurgitation (MR) is a life-threatening condition that may rapidly progress to pulmonary edema, cardiogenic shock, and death if not promptly recognized and treated [1]. Unlike chronic MR, acute MR is poorly tolerated because the relatively noncompliant left atrium cannot accommodate the sudden regurgitant volume, resulting in abrupt elevations in left atrial and pulmonary venous pressures [1]. Although cardiogenic pulmonary edema typically presents with bilateral alveolar infiltrates, unilateral pulmonary edema (UPE) is an uncommon and frequently overlooked manifestation that may closely mimic infectious pneumonia, potentially delaying appropriate diagnosis and treatment [2,3]. Right-sided involvement predominates and is strongly associated with severe eccentric MR, particularly in the setting of posterior mitral leaflet prolapse or chordal rupture [2,4]. Nishimura et al. reported UPE in 50% of patients with acute severe MR caused by chordal rupture, with all cases involving the right lung, supporting the role of eccentric regurgitant jets in producing asymmetric pulmonary venous hypertension [3].

Barlow's disease is characterized by diffuse myxomatous degeneration, excessive leaflet tissue, and multisegmental prolapse and may be complicated by spontaneous chordal rupture with abrupt hemodynamic deterioration [1]. We report a case of previously unrecognized Barlow's disease complicated by spontaneous chordal rupture resulting in a flail P2 scallop extending into P3, severe eccentric MR, and right-sided UPE mimicking pneumonia, followed by rapid progression to refractory cardiogenic shock before definitive surgical intervention could be performed. This case highlights the importance of considering acute severe MR in patients presenting with unilateral pulmonary infiltrates and acute respiratory failure and emphasizes the critical role of early echocardiographic evaluation in identifying the underlying mechanical lesion.

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Case Presentation

A 50-year-old woman with no modifiable cardiovascular risk factors and no significant past medical history, including no prior history of acute rheumatic fever or known valvular heart disease, presented to the emergency department with a 24-hour history of abrupt-onset resting dyspnea. The symptoms progressed dramatically over a few hours, evolving into severe respiratory distress and incapacitating orthopnea, prompting emergency admission. She denied chest pain, palpitations, syncope, or presyncope. Furthermore, she reported no cough, fever, or other symptoms suggestive of an infectious process, and there was no history of recent trauma.

On admission, she was hemodynamically stable, with a blood pressure of 120/80 mmHg and a heart rate of 130 beats/min. She was afebrile and markedly hypoxemic, with an oxygen saturation of 86% on room air. Pulmonary examination revealed asymmetric crackles, predominantly involving the right lung fields. There were no signs of right-sided heart failure, including jugular venous distension, peripheral edema, or hepatomegaly. The calves and thighs were soft and non-tender. Cardiac auscultation disclosed a soft, high-pitched, blowing holosystolic murmur of MR, best heard at the apex and radiating toward the left sternal border.

The admission electrocardiogram demonstrated sinus tachycardia (133 beats/min) with normal PR and QTc intervals and occasional premature supraventricular beats, without atrial fibrillation, conduction abnormalities, or ischemic changes (Figure *1*).

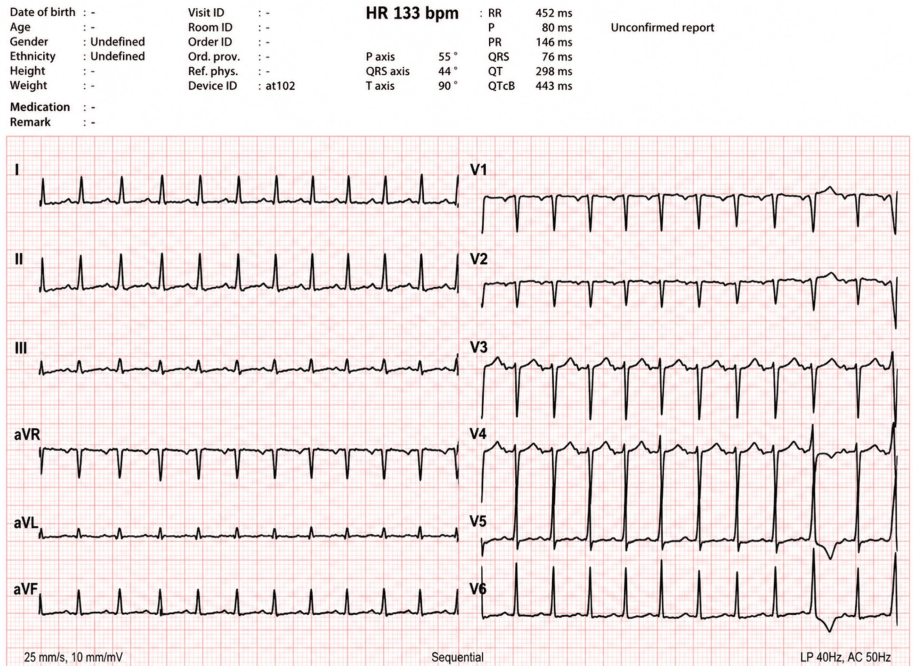


FIGURE 1: Twelve-lead electrocardiogram on admission.

The 12-lead electrocardiogram shows sinus tachycardia at 133 beats/min with an isolated premature ventricular complex (PVC). The QRS complexes are narrow (76 ms), with a normal frontal QRS axis.

Chest radiography revealed extensive patchy alveolar opacities predominantly involving the right lung, particularly the upper and middle lung zones, with relative sparing of the left lung and no pleural effusion (Figure *2*). The marked asymmetry of the infiltrates initially raised the suspicion of right-sided pneumonia; however, given the abrupt onset of symptoms and the clinical findings, unilateral right pulmonary edema secondary to acute severe MR was strongly suspected.

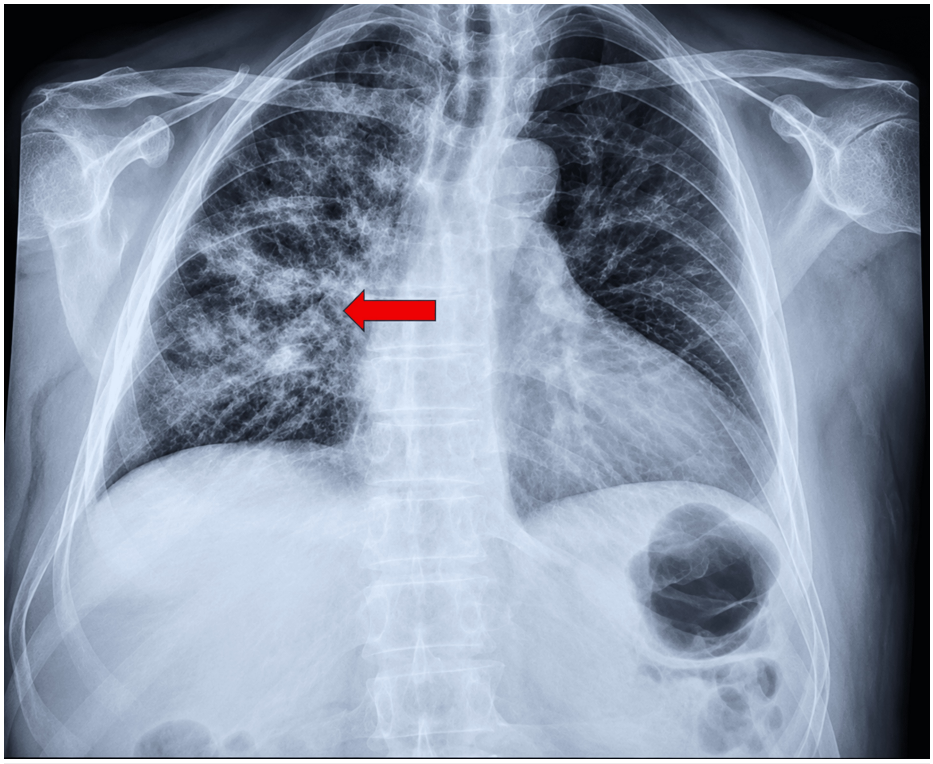


FIGURE 2: Frontal chest radiograph at admission.

Frontal chest radiograph showing extensive right-sided patchy confluent air-space opacities, predominantly involving the perihilar and mid-to-lower lung zones (red arrow), with relative sparing of the left lung. No pleural effusion or cardiomegaly is present. The unilateral distribution closely mimics an infectious pulmonary process.

Laboratory investigations were largely unremarkable (Table *1*). The complete blood count, coagulation profile, renal and hepatic function tests, and serum electrolytes were within normal limits. Inflammatory markers were also unremarkable, with a C-reactive protein level of 1 mg/L and a procalcitonin level of 0.01 ng/mL, providing no laboratory evidence of an ongoing bacterial infection. High-sensitivity cardiac troponin I was within the normal range (1 ng/L; upper reference limit, 35 ng/L), with no evidence of acute myocardial injury. In contrast, N-terminal pro-B-type natriuretic peptide (NT-proBNP) was elevated at 1,000 pg/mL, consistent with increased cardiac wall stress in the context of acute heart failure.

Laboratory parameter	Patient value	Reference range	Interpretation
Complete blood count			
White blood cell count	6,000/mm³	4,000–10,000/mm³	Normal
Hemoglobin	14 g/dL	13–17 g/dL	Normal
Platelet count	200,000/mm³	150,000–400,000/mm³	Normal
Coagulation profile			
Prothrombin time (PT)	100%	70–100%	Normal
International normalized ratio (INR)	1.0	0.8–1.2	Normal
Renal function and electrolytes			
Urea	0.30 g/L	0.15–0.45 g/L	Normal
Creatinine	7 mg/L	6–12 mg/L	Normal
Estimated glomerular filtration rate (eGFR)	90 mL/min/1.73 m²	≥90 mL/min/1.73 m²	Normal
Sodium (Na^+)	135 mmol/L	135–145 mmol/L	Normal
Potassium (K^+)	4.0 mmol/L	3.5–5.0 mmol/L	Normal
Liver function			
Aspartate aminotransferase (AST)	30 U/L	10–40 U/L	Normal
Alanine aminotransferase (ALT)	30 U/L	7–40 U/L	Normal
Inflammatory markers			
C-reactive protein (CRP)	1 mg/L	<5 mg/L	Normal
Procalcitonin (PCT)	0.01 ng/mL	<0.05 ng/mL	Normal
Cardiac biomarkers			
High-sensitivity cardiac troponin I (hs-cTnI)	1 ng/L	<35 ng/L	Normal
N-terminal pro-B-type natriuretic peptide (NT-proBNP)	1,000 pg/mL	<125 pg/mL	Elevated

TABLE 1: Laboratory findings on admission.

ALT: alanine aminotransferase; AST: aspartate aminotransferase; CRP: C-reactive protein; eGFR: estimated glomerular filtration rate; hs-cTnI: high-sensitivity cardiac troponin I; INR: international normalized ratio; K^+: potassium; Na^+: sodium; NT-proBNP: N-terminal pro-B-type natriuretic peptide; PCT: procalcitonin; PT: prothrombin time

Transthoracic echocardiography revealed severe degenerative MR secondary to spontaneous chordae tendineae rupture, resulting in a flail P2 scallop (Figure *3*). The mitral valve exhibited marked leaflet redundancy and excessive tissue, highly suggestive of Barlow's disease. Color Doppler demonstrated a highly eccentric anteriorly directed regurgitant jet. Quantitative assessment confirmed severe MR, with an effective regurgitant orifice area of 0.64 cm² and a regurgitant volume of 80.7 mL (Figure *4*).

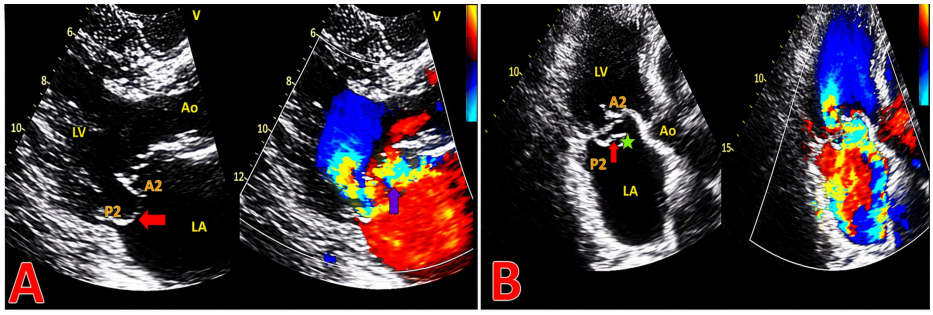


FIGURE 3: Transthoracic echocardiographic findings of degenerative mitral valve disease with flail P2 scallop and severe eccentric mitral regurgitation.

A: Parasternal long-axis view demonstrating a flail P2 scallop secondary to chordal rupture (red arrow), associated with marked leaflet redundancy and excess valvular tissue suggestive of Barlow's disease. Color Doppler shows a severe eccentric anteriorly directed mitral regurgitation jet (purple arrow). B: Apical four-chamber view demonstrating a flail P2 scallop secondary to spontaneous chordae tendineae rupture (red arrow). The green star highlights the flail segment protruding into the left atrium during systole. Associated excess leaflet tissue is consistent with degenerative myxomatous mitral valve disease (Barlow's disease).

A2: anterior mitral leaflet segment 2; Ao: aorta; LA: left atrium; LV: left ventricle; P2: posterior mitral leaflet segment 2

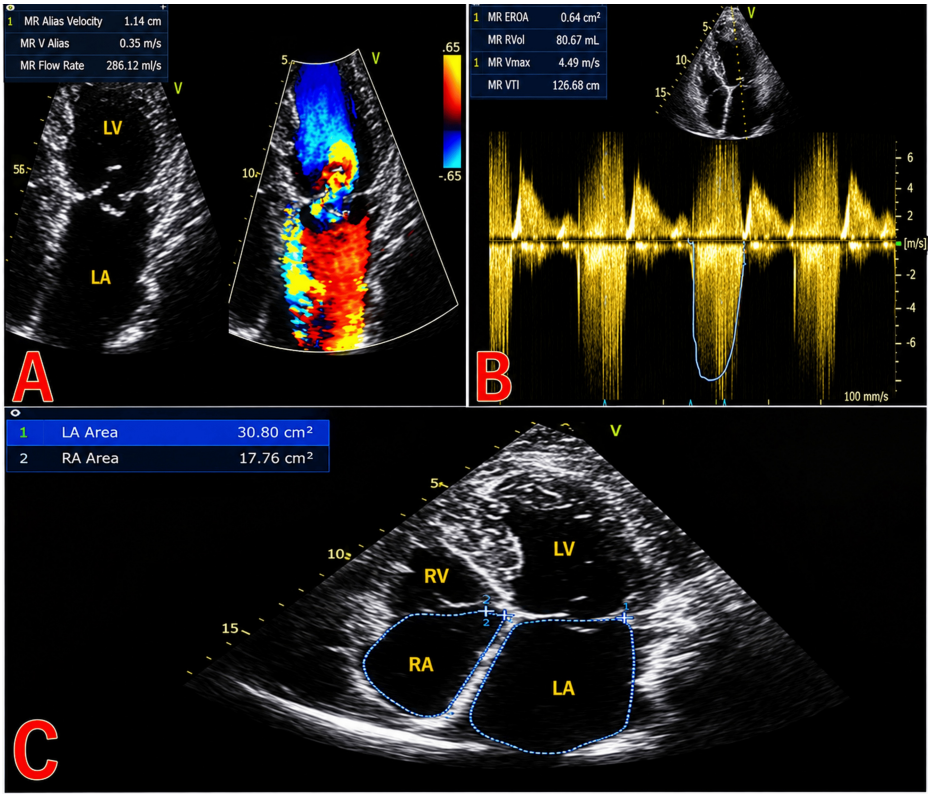


FIGURE 4: Quantitative echocardiographic assessment of severe mitral regurgitation.

A: Color Doppler and proximal isovelocity surface area (PISA) analysis demonstrating a large flow convergence zone, consistent with severe mitral regurgitation. B: Continuous-wave Doppler of the mitral regurgitant jet showing a dense, triangular, early-peaking systolic envelope. Quantitative assessment confirmed severe mitral regurgitation, with an effective regurgitant orifice area of 0.64 cm² and a regurgitant volume of 80.7 mL. C: Apical four-chamber view demonstrating marked left atrial enlargement.

LA: left atrium; RA: right atrium; PISA: proximal isovelocity surface area; EROA: effective regurgitant orifice area

The left ventricle was non-dilated and non-hypertrophied, with an apparently preserved left ventricular ejection fraction of 69%. The right ventricle was of normal size and exhibited preserved systolic function. The left atrium was markedly enlarged (left atrial area: 30.8 cm^2), suggesting underlying chronic degenerative mitral valve disease. Mild-to-moderate pulmonary hypertension was present, with an estimated pulmonary artery systolic pressure of 42 mmHg. The inferior vena cava was thin and collapsible, suggesting normal right atrial pressure. Despite the marked left atrial enlargement, the absence of left ventricular remodeling was compatible with a recent spontaneous chordal rupture leading to acute hemodynamic decompensation of previously unrecognized degenerative MR.

The patient was initially stabilized with supplemental oxygen delivered via a non-rebreather mask, intravenous loop diuretics (furosemide), and intravenous nitrate therapy, resulting in partial improvement of respiratory distress and oxygenation. Continuous cardiac and hemodynamic monitoring was instituted, and the patient was subsequently admitted to the intensive cardiac care unit. Once her respiratory status had partially stabilized, transesophageal echocardiography was performed to further delineate the mechanism of mitral valve dysfunction and guide therapeutic decision-making.

TEE demonstrated marked thickening and redundancy of both mitral leaflets, with excessive leaflet tissue and multisegmental billowing, findings highly suggestive of Barlow's disease (Figure *5*). Mid-esophageal views revealed spontaneous rupture of the chordae tendineae, identified by two freely mobile ruptured chordal remnants (green stars), resulting in a flail posterior leaflet involving the P2 scallop with extension into P3 (Figure *5A*, *5C*, *5D*). The prolapsing segment was seen protruding into the left atrium during systole (red arrows).

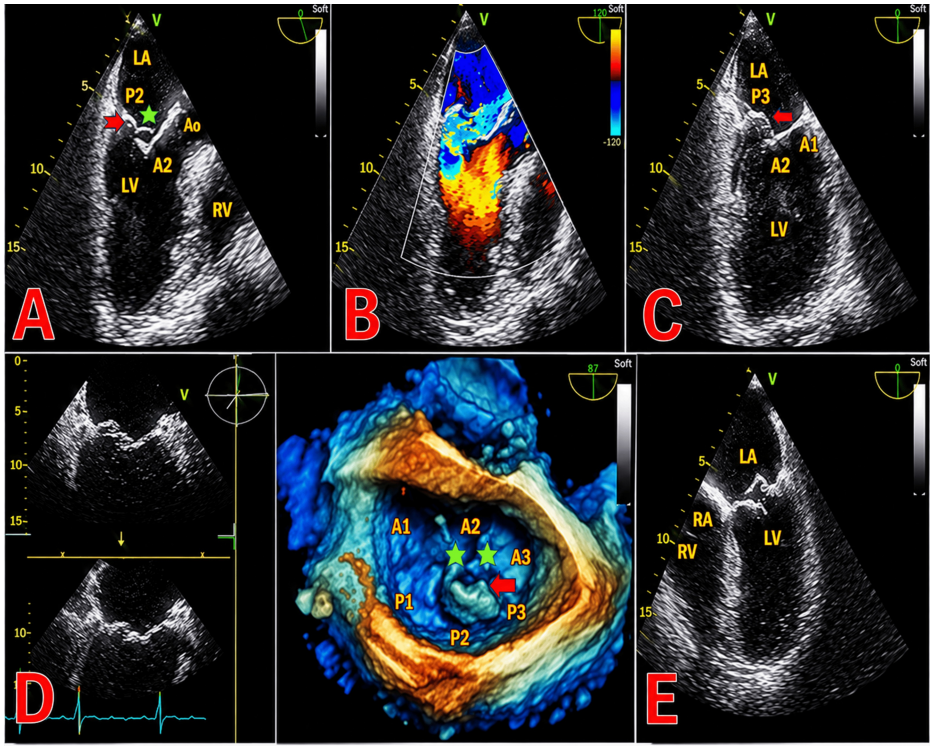


FIGURE 5: Transesophageal echocardiographic characterization of Barlow's disease with ruptured chordae causing a flail P2 scallop extending into P3 and severe eccentric mitral regurgitation.

A: Mid-esophageal long-axis view (120°) demonstrating marked leaflet thickening, redundancy, and excess valvular tissue consistent with myxomatous degeneration and Barlow's disease. The red arrow indicates systolic prolapse of the posterior mitral leaflet due to a flail P2 scallop, while the green star denotes ruptured chordae tendineae. B: Mid-esophageal long-axis color Doppler view (120°) demonstrating a severe eccentric anteriorly directed mitral regurgitation jet occupying a large portion of the left atrium. C: Mid-esophageal two-chamber view (90°) showing prolapse of the posterior mitral leaflet involving the P2 scallop with extension into P3 (red arrow), protruding into the left atrium during systole. D: Biplane transesophageal imaging (0° and 90°) confirming the flail P2 scallop with systolic displacement of the prolapsing segment into the left atrium (yellow arrow). The central three-dimensional en-face surgical view from the left atrial perspective demonstrates a flail P2 scallop extending into P3 (red arrow) associated with ruptured chordae tendineae (green stars), on a background of diffuse myxomatous degeneration characteristic of Barlow's disease. E: Mid-esophageal four-chamber view (0°) showing marked left atrial enlargement. Left atrial enlargement and the overall chamber anatomy.

Color Doppler imaging demonstrated a severe eccentric anteriorly directed MR jet occupying a large portion of the left atrium (Figure *5B*). Three-dimensional en-face surgical views further confirmed the anatomical complexity of the lesion, demonstrating a flail P2 scallop extending into P3 associated with ruptured chordae tendineae and diffuse myxomatous degeneration, characteristic of Barlow's disease (Figure *5D*).

No evidence of valvular vegetations, leaflet perforation, papillary muscle rupture, intracardiac thrombus, or associated structural abnormalities was identified.

Overall, TEE established the diagnosis of acute severe degenerative MR caused by spontaneous chordal rupture complicating Barlow's disease, resulting in a flail P2 scallop extending into P3 and severe eccentric MR.

Based on the transesophageal echocardiographic findings, the patient was diagnosed with acute severe degenerative MR secondary to spontaneous chordal rupture complicating Barlow's disease. Given the severity of the regurgitation and the presence of acute heart failure, urgent surgical intervention was considered mandatory and represented a Class I indication according to current valvular heart disease guidelines.

The patient was managed in the cardiac intensive care unit with high-flow oxygen supplementation, intravenous furosemide (40 mg followed by repeated doses according to clinical response), and intravenous nitrate therapy titrated to blood pressure in an attempt to reduce left ventricular filling pressures and alleviate pulmonary congestion. Continuous electrocardiographic and invasive hemodynamic monitoring was instituted.

Given the favorable anatomy of the lesion, mitral valve repair was considered the preferred surgical strategy, consisting of posterior leaflet repair with reconstruction of the ruptured chordae using expanded polytetrafluoroethylene (ePTFE) neochordae and implantation of an annuloplasty ring.

However, shortly after completion of the diagnostic work-up, the patient's clinical condition rapidly deteriorated. She developed cardiogenic shock, characterized by profound hypotension (blood pressure, 80/60 mmHg), cold extremities, mottled skin, and signs of peripheral hypoperfusion. Intravenous vasoactive support with norepinephrine and inotropic therapy with dobutamine were promptly initiated.

Despite aggressive medical management, the patient developed refractory cardiogenic shock and died approximately two hours after admission to the cardiac intensive care unit, before definitive surgical intervention could be undertaken.

This catastrophic evolution highlights the fulminant nature of acute severe MR and the narrow therapeutic window available before irreversible hemodynamic collapse occurs.

Discussion

UPE is an uncommon manifestation of cardiogenic pulmonary edema and represents an important diagnostic pitfall because its radiographic appearance may closely mimic unilateral pneumonia [2]. Right-sided involvement is markedly predominant, and severe eccentric MR is one of its most characteristic cardiac associations [2,3]. Delayed recognition may postpone echocardiographic assessment and definitive treatment in a condition in which hemodynamic deterioration can be rapid [2,3].

Our patient presented with abrupt-onset dyspnea, severe hypoxemia, and extensive right-sided alveolar infiltrates initially suggestive of pneumonia. However, the absence of fever, cough, leukocytosis, elevated inflammatory markers, and increased procalcitonin made an infectious etiology unlikely. In this setting, the association of acute respiratory failure, unilateral pulmonary infiltrates, and a newly detected systolic murmur should raise suspicion for acute severe MR and prompt urgent echocardiographic evaluation [2,3].

The asymmetric distribution of pulmonary edema in severe MR is largely related to the direction of the eccentric regurgitant jet and its preferential impact on pulmonary venous flow [2,3]. Posterior leaflet prolapse or flail generally produces an anteriorly directed eccentric jet, which may preferentially increase pressure within right-sided pulmonary veins and promote right-sided pulmonary congestion [3,4]. This mechanism is strongly supported by Nishimura et al., who studied 38 patients with acute severe MR caused by chordal rupture and found UPE in 19 patients (50%); all cases were right-sided, and 18 of the 19 patients with UPE had posterior leaflet chordal rupture [3]. This mechanism closely parallels our patient, in whom spontaneous chordal rupture resulted in a flail P2 scallop extending into P3 and a severe eccentric anteriorly directed regurgitant jet.

Echocardiography was central to establishing both the severity and mechanism of MR. Current recommendations emphasize a multiparametric echocardiographic assessment of MR severity together with precise characterization of mitral valve anatomy and mechanism, with three-dimensional transesophageal echocardiography providing additional anatomical information when required [5]. In our patient,

transthoracic echocardiography rapidly identified severe MR, whereas transesophageal echocardiography demonstrated marked leaflet redundancy, excess valvular tissue, and multisegmental billowing consistent with Barlow's disease, together with ruptured chordae and a flail P2 scallop extending into P3. TEE also helped exclude alternative mechanisms of acute MR, particularly infective endocarditis, leaflet perforation, and papillary muscle rupture. These findings illustrate the pivotal role of comprehensive echocardiography not only in diagnosing severe MR but also in defining the underlying mechanical lesion and planning intervention [5].

An important consideration in our case is the distinction between purely acute MR and acute decompensation of previously unrecognized chronic degenerative MR. The markedly enlarged left atrium suggests that degenerative MR had probably been present before admission, whereas the abrupt onset of symptoms and the absence of substantial left ventricular remodeling support a recent mechanical event. Taken together, these findings favor acute decompensation of previously silent chronic degenerative MR precipitated by spontaneous chordal rupture in the setting of Barlow's disease [1,5].

Another striking feature was the rapid transition from initial hemodynamic stability to refractory cardiogenic shock despite an apparently preserved left ventricular ejection fraction of 69%. In severe MR, left ventricular ejection fraction may overestimate effective left ventricular systolic performance because part of the total stroke volume is ejected backward into the relatively low-pressure left atrium rather than forward into the systemic circulation [1,5]. Consequently, an apparently preserved or hyperdynamic left ventricular ejection fraction does not necessarily imply adequate forward cardiac output. In our patient, the abrupt rise in left atrial and pulmonary venous pressures, severe pulmonary congestion, profound hypoxemia, and reduction in effective forward output likely contributed to the rapid development of refractory cardiogenic shock. Thus, an apparently preserved left ventricular ejection fraction should not be interpreted as evidence of hemodynamic stability in acute severe MR [1,5].

Several differential diagnoses should be considered in this setting. Infectious pneumonia represents the principal initial diagnostic challenge in patients with unilateral alveolar infiltrates [2,3]. Ischemic acute MR, particularly due to papillary muscle rupture following myocardial infarction, is another critical differential diagnosis because it may similarly present with pulmonary edema and cardiogenic shock [6,7]. In our patient, the absence of chest pain, negative serial troponins, absence of ischemic electrocardiographic changes, an apparently preserved left ventricular ejection fraction of 69%, and direct visualization of chordal rupture without papillary muscle rupture strongly favored a primary degenerative mechanism. Infective endocarditis was also considered but was unsupported by the clinical, biological, and echocardiographic findings.

Management of acute severe MR is time-sensitive and requires immediate stabilization followed by definitive correction of the underlying mechanical lesion [6,7]. Medical therapy may include oxygen or ventilatory support, intravenous diuretics, and afterload reduction when blood pressure permits, while vasoactive and inotropic support may be necessary in patients progressing to cardiogenic shock [6-8]. These measures should generally be regarded as a bridge rather than definitive treatment when severe MR results from a correctable structural lesion.

Urgent surgery is indicated in patients with acute severe MR [6]. In primary degenerative MR, mitral valve repair is preferred whenever a durable repair is technically feasible, particularly when the lesion is amenable to reconstructive surgery [5,6]. In our patient, urgent mitral valve surgery was therefore indicated, with posterior leaflet reconstruction, neochordal implantation, and annuloplasty representing a potential repair strategy. Unfortunately, the extremely rapid progression to refractory cardiogenic shock prevented definitive surgical intervention.

Temporary mechanical circulatory support may represent a bridge to definitive surgery in selected patients with refractory cardiogenic shock when immediate operative intervention cannot safely be performed [8,9]. Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) has been successfully used to provide temporary hemodynamic and respiratory stabilization in critically ill patients with acute severe MR, although its potential to increase left ventricular afterload must be considered and left ventricular unloading may be required in selected cases [8,9]. In our patient, however, the interval between hemodynamic deterioration and death was too short to allow implementation of advanced mechanical circulatory support.

Previously published reports and series are summarized in Table *2*. Of particular relevance, Nishimura et al. demonstrated a strong relationship between posterior chordal rupture, acute severe MR, and right-sided UPE [3]. Compared with these reports, the particularity of our case lies in the combination of previously unrecognized Barlow's disease, spontaneous chordal rupture involving P2-P3, a severe eccentric anteriorly directed MR jet, right-sided UPE initially mimicking pneumonia, an apparently preserved left ventricular ejection fraction of 69%, and extremely rapid progression to refractory cardiogenic shock resulting in death before definitive surgical intervention.

Study	Patient(s)	MR mechanism	Presentation / Echocardiographic findings	Management & outcome
Attias et al., 2010 [2]	18-patient UPE series	Severe MR frequently associated with UPE	Predominantly right-sided UPE (89%); frequent diagnostic confusion with pneumonia	Medical stabilization ± definitive treatment; higher mortality than bilateral pulmonary edema
Nishimura et al., 2022 [3]	38 patients with acute severe MR due to chordal rupture	Chordal rupture, predominantly involving the posterior mitral leaflet	UPE in 19/38 (50%); all UPE cases were right-sided; 18/19 had posterior leaflet chordal rupture	Medical stabilization followed by definitive intervention according to clinical status; variable outcomes
Jiang et al., 2021 [4]	67-year-old woman	Posterior mitral leaflet prolapse with severe eccentric MR	Right-sided UPE mimicking pneumonia; severe eccentric MR	Medical stabilization followed by mitral valve intervention; survived

TABLE 2: Previously published cases of unilateral pulmonary edema secondary to severe mitral regurgitation and their management strategies.

Summary of previously published cases of unilateral pulmonary edema secondary to severe mitral regurgitation, highlighting the underlying mechanisms, initial misdiagnoses, management strategies, and clinical outcomes. Most reported cases involved severe eccentric mitral regurgitation affecting the posterior mitral leaflet and initially mimicked infectious pneumonia. Early echocardiographic diagnosis and prompt surgical correction were associated with favorable outcomes in the majority of cases

MR: mitral regurgitation; UPE: unilateral pulmonary edema

Overall, this case conveys three important clinical messages. First, right-sided UPE should not automatically be attributed to pneumonia, particularly when respiratory symptoms are abrupt and infectious findings are absent [2,3]. Second, urgent echocardiography is essential because it can rapidly establish the diagnosis of severe MR and identify its underlying mechanical mechanism [5]. Finally, acute chordal rupture complicating previously silent degenerative MR may lead to profound hemodynamic deterioration despite an apparently preserved left ventricular ejection fraction, emphasizing the narrow therapeutic window between diagnosis and definitive intervention [1,6-9].

Conclusions

This case highlights an uncommon and potentially fatal presentation of acute decompensation of previously unrecognized degenerative MR due to spontaneous chordal rupture in Barlow's disease. Right-sided UPE should raise suspicion for acute severe MR rather than being automatically attributed to pneumonia, particularly when symptom onset is abrupt, a systolic murmur is present, and inflammatory findings are absent. Prompt echocardiographic evaluation is crucial for establishing the diagnosis, defining the underlying valvular mechanism, and guiding urgent management. Importantly, an apparently preserved left ventricular ejection fraction should not provide false reassurance, as acute severe MR may rapidly progress from apparent hemodynamic stability to refractory cardiogenic shock. Early recognition and immediate referral for definitive intervention are therefore essential, as the therapeutic window before irreversible hemodynamic collapse may be extremely narrow.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Hafsa Erregui, Nadia Loudiyi, Mohamed Malki, Zouhair Lakhal, Aatif Benyass

Acquisition, analysis, or interpretation of data: Hafsa Erregui, Nadia Loudiyi, Mohamed Malki, Zouhair Lakhal, Aatif Benyass

Drafting of the manuscript: Hafsa Erregui, Nadia Loudiyi, Mohamed Malki, Zouhair Lakhal, Aatif Benyass

Critical review of the manuscript for important intellectual content: Hafsa Erregui, Nadia Loudiyi, Mohamed Malki, Zouhair Lakhal, Aatif Benyass

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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