Valvular Heart Disease

Prevalence, Characteristics, and Outcomes of Patients Presenting With Cardiogenic Unilateral Pulmonary Edema

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Background—Cardiogenic unilateral pulmonary edema (UPE) is a rare entity, frequently leading to initial misdiagnosis. We sought to assess the prevalence of UPE and to determine its impact on prognosis compared with bilateral pulmonary edema.

Methods and Results—We studied the characteristics and outcomes of patients admitted to our institution for cardiogenic pulmonary edema during an 8-year period. The study population included 869 consecutive patients. The prevalence of UPE was 2.1%: 16 right-sided UPE (89%) and 2 left-sided UPE (11%). In patients with UPE, blood pressure was significantly lower \( (P \leq 0.01) \), whereas noninvasive or invasive ventilation and catecholamines were used more frequently \( (P=0.0004 \text{ and } P<0.0001) \), respectively. The prevalence of severe mitral regurgitation in patients with bilateral pulmonary edema and UPE was 6% and 100%, respectively \( (P<0.0001) \). In patients with UPE, use of antibiotic therapy and delay in treatment were significantly higher \( (P<0.0001 \text{ and } P=0.003) \). In-hospital mortality was 9%: 39% for UPE versus 8% for bilateral pulmonary edema (odds ratio, 6.9; 95% confidence interval, 2.6 to 18; \( P<0.0001 \)). In multivariate analysis, unilateral location of pulmonary edema was independently related to death whatever the model used (adjusted odds ratio, 6.5; 95% confidence interval, 1.3 to 32; \( P=0.021 \) for model A; and adjusted odds ratio, 6.8; 95% confidence interval, 1.1 to 41; \( P=0.037 \) for model B).

Conclusions—Unilateral pulmonary edema represented 2.1% of cardiogenic pulmonary edema in our study, usually appeared as an opacity involving the right lung, and was always associated with severe mitral regurgitation. Unilateral pulmonary edema is related to an independent increased risk of mortality and should be promptly recognized to avoid delays in treatment. (Circulation. 2010;122:1109-1115.)

Key Words: diagnosis ■ heart failure ■ mitral valve ■ prognosis

The clinical presentation of heart failure may be cardiogenic pulmonary edema.\(^1\) History and physical examination, as well as laboratory tests, echocardiography, and chest radiography, may help in making the correct diagnosis.\(^4\) The usual pattern of acute cardiogenic pulmonary edema on the chest radiograph is a bilateral symmetrical opacity occupying the central zones of the lungs, resulting in the classic “butterfly shadow.” Diagnosis of bilateral pulmonary edema (BPE) remains the most obvious, and appropriate treatment must be initiated promptly to ensure accurate medical management.\(^1,2\)

Clinical Perspective on p 1115

The unilateral location of cardiogenic pulmonary edema is unusual\(^3-8\) and has rarely been described and mainly reported in association with severe mitral regurgitation (MR).\(^9,10\) Little is known about the characteristics of unilateral pulmonary edema (UPE), and its prevalence has never been assessed in a large series of pulmonary edema. Furthermore, initiation of appropriate treatment may be delayed because the clinical presentation of UPE is often atypical, and this diagnostic trap is underestimated. The aims of the study were to assess the prevalence of UPE in a large series of consecutive patients with acute cardiogenic pulmonary edema, to describe the clinical and echocardiographic features of these patients, and to determine the impact of UPE on prognosis compared with BPE.

Methods

Over an 8-year period (from January 2000 to May 2008), we reviewed all patients \((n=869)\) referred to our intensive care unit and coronary care unit for acute cardiogenic pulmonary edema. Criteria for selection included age >18 years and a final diagnosis of acute cardiogenic pulmonary edema.\(^1\) Patients were excluded if the acute dyspnea was secondary to acute respiratory distress syndrome.

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sepsis, pneumonia, alveolar hemorrhage, or neoplasia. Noncardiogenic pulmonary edema related to septic shock or in association with acute respiratory distress syndrome was excluded. If a patient was admitted more than once for cardiogenic pulmonary edema during the study period, only the data for the first admission were analyzed. All the clinical records were studied, including demographics, cardiovascular risk factors, medical history, prior history of heart failure, previous treatment before admission, and clinical examination at admission, as well as ECG, chest x-ray, echocardiographic and laboratory data, management during hospitalization, and in-hospital outcomes (death, urgent mitral surgery). Leukocytosis was defined as white blood cell count >10,000/mm³. Adequate treatment for pulmonary edema (nitrates, diuretics) was considered to have been delayed if it was started ≥8 hours after the first medical contact. The final diagnosis of acute cardiogenic pulmonary edema was established according to different parameters: clinical examination, medical history, ECG, chest x-ray, echocardiographic examination, brain natriuretic peptide results, and response to therapy (diuretics).

Anterior-posterior supine chest radiographs were performed during the acute phase of pulmonary edema at admission and were reviewed by an experienced observer blinded to clinical data. The location of pulmonary infiltrates was systematically assessed, allowing BPE to be distinguished from UPE. All chest radiographs with a suspicion of asymmetrical pulmonary infiltrates were reviewed by 3 experts to confirm the final diagnosis of UPE.

Transesophageal echocardiography was performed during hospitalization (within 48 hours after admission) with the Toshiba Powervision 8000 or Corevision systems (Toshiba, Otawara-Shi, Japan) or a Siemens/Sequoia Acuson system (Acuson, Mountain View, Calif) equipped with multifrequency transducers. Transesophageal echocardiography was performed only if necessary. We systematically reviewed the following echocardiographic parameters according to guidelines: end-diastolic left ventricular (LV) diameter, end-systolic LV diameter, and left atrial diameter in the parasternal long-axis view; LV ejection fraction (LVEF) using 2-dimensional Simpson rules; and systolic pulmonary artery pressure using peak velocity of tricuspid flow regurgitation with continuous-wave Doppler. The severity of valvular heart disease was estimated according to the guidelines. Severe MR was defined by a grade ≥3/4. Organic MR was defined by regurgitation resulting from rheumatic heart disease, endocarditis, or leaflet prolapse secondary to chordae rupture (fibroelastic degeneration, Barlow disease) or from papillary disease, endocarditis, or leaflet prolapse secondary to chordae rupture (fibroelastic degeneration, Barlow disease) or from papillary muscle rupture. Functional MR was diagnosed if MR was associated with coronary artery disease or dilated cardiomyopathy and there was no other pathogenesis.

**Statistical Analysis**

Continuous variables are presented as mean±SD and as median (25th to 75th percentiles) and compared by use of the Wilcoxon rank-sum test. The Wilcoxon rank-sum test was chosen because of the small sample size of patients presenting with UPE. Qualitative variables were presented as absolute values and percentages and were compared by use of the χ² test or Fischer exact test as appropriate. Mortality rates were compared between patients with UPE and BPE using univariate and multivariate logistic regression models, which allow the calculation of the odds ratio (OR) and adjusted OR (aOR) with a 95% confidence interval (CI). Despite the small sample size of patients with UPE, we have used a logistic regression. The multivariate model (model A) was adjusted for the following first set of dichotomous variables: unilateral or bilateral location of pulmonary edema on chest x-ray; age (<79 years, ≥79 years); presence of severe MR; LVEF (<30%, ≥30%); and history of heart failure, coronary artery disease, chronic obstructive pulmonary disease, renal insufficiency, and diabetes mellitus. Other covariates were selected by stepwise forward analysis among the following second set of additional dichotomous variables: sex, hypertension, history of atrial fibrillation, neoplasia, mitral valve disease, dyslipidemia, smoking, treatment before admission (β-blockers, angiotensin-converting enzyme inhibitors, angiotensin II receptor antagonists, aspirin, clopidogrel, statin, vitamin K antagonists, diuretics), presence of fever (temperature >38°C), tachycardia (>120 bpm) or shock (systolic arterial pressure <90 mm Hg) at admission, pulmonary hypertension (systolic pulmonary artery pressure >40 mm Hg), and administration of antibiotic treatment for management. A second multivariate model (model B) was also used with the same variables plus 2 additional variables (hospitalization in coronary-care unit or intensive care unit and invasive mechanical ventilation). A value of P<0.05 was considered statistically significant. Statistical analyses were performed with SPSS version 8.0 (SPSS Inc., Chicago, Ill).

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

**Results**

**Population Characteristics**

The study population included 869 consecutive patients (475 men and 394 women; mean age, 75.4±13 years; range, 21 to 101 years). Clinical records, ECG, chest x-ray, and routine laboratory data were available in all patients; transthoracic and/or transesophageal echocardiography was performed in 96.1% of patients in the first 48 hours after admission.

The population was divided according to the location of pulmonary edema in 851 patients (97.9%) presenting with BPE versus 18 patients with UPE (Table 1). Severe MR was diagnosed in 71 patients (8%).

**Patients Presenting With UPE**

The prevalence of UPE among our patients presenting with acute cardiogenic pulmonary edema was 2.1%. The baseline characteristics of these 18 patients were as follows: 10 men and 8 women; mean age, 76.4±12.9 years; and range, 46 to 94 years. All these patients presented with negative blood cultures. UPE was right-sided in 16 cases (89%): upper segment in 6 patients (37.5%), upper and middle lobes in 2 patients (12.5%), middle and inferior lobes in 4 patients (25%), and the entire right lung in 4 patients (25%). In 2 patients (11%), the location of UPE was the left lung: entire lung in 1 patient and left upper lobe in the other.

Systolic and diastolic blood pressures were significantly lower in patients with UPE (P=0.01 and P=0.002, respectively). Plasma B-natriuretic peptide level was 1250±1157 ng/L in patients with UPE versus 1036±991 in patients with BPE (P=0.37). Noninvasive or invasive ventilation and catecholamine use were more frequent in patients with UPE than in patients with BPE (P=0.0004 and P<0.0001, respectively). There was no significant difference in the 2 groups concerning the mean time from symptom onset to admission: 29±27 hours (range, 10 minutes to 96 hours; median, 24 hours) in patients with UPE versus 21±23 hours (range, 1 to 96 hours; median, 12 hours) in patients with BPE (P=0.27).

All patients with UPE presented with severe MR: an organic MR in 10 patients and a functional MR in 8 patients. In 90% of patients with organic MR, the radiological location of UPE was related to the lateralized direction of MR: 8 patients with posterior leaflet prolapse had right-sided UPE (the Figure, A), and 1 patient with anterior leaflet prolapse presented with left-sided UPE (the Figure, B). One patient with anterior leaflet prolapse had right-sided UPE. Among the 8 patients with UPE and functional MR, 7 presented with right-sided UPE and 1 with left-sided UPE.
Characteristics of Patients With Cardiogenic Pulmonary Edema and Severe MR

Among the 71 patients with cardiogenic pulmonary edema and severe MR, 53 patients (75%) presented with BPE, whereas 18 patients had UPE (25%). Clinical characteristics (sex ratio, age, heart rate, temperature, cardiovascular risk factors, previous medical history, and previous treatment before admission) were similar between patients with UPE and severe MR and patients with BPE and severe MR (all P>0.05). At admission, diastolic blood pressure was lower in the group of patients with UPE (64±10 mm Hg [median, 64 mm Hg] versus 79±20 mm Hg [median, 78 mm Hg]) in patients with BPE and severe MR; P=0.003, whereas there was a tendency toward decreased systolic blood pressure in patients with UPE (119±21 mm Hg [median, 123 mm Hg]) versus 133±31 mmHg [median, 130 mm Hg] in patients with

### Table 1. Characteristics of Patients Presenting With Pulmonary Edema: Entire Population and BPE Versus UPE

<table>
<thead>
<tr>
<th></th>
<th>All Pulmonary Edema (n=869)</th>
<th>BPE (n=851)</th>
<th>UPE (n=18)</th>
<th>P, BPE vs UPE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Women, n (%)</strong></td>
<td>394 (45)</td>
<td>386 (45)</td>
<td>8 (44)</td>
<td>0.94</td>
</tr>
<tr>
<td><strong>Age, y</strong></td>
<td>75.3±13.2</td>
<td>75.3±13.2</td>
<td>76.4±12.9</td>
<td>0.73</td>
</tr>
<tr>
<td><strong>Medical history, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior heart failure</td>
<td>164 (19)</td>
<td>160 (19)</td>
<td>4 (22)</td>
<td>0.55</td>
</tr>
<tr>
<td>Current smoker</td>
<td>265 (33)</td>
<td>278 (33)</td>
<td>7 (39)</td>
<td>0.58</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>209 (24)</td>
<td>205 (24)</td>
<td>4 (22.2)</td>
<td>0.85</td>
</tr>
<tr>
<td>Hypertension</td>
<td>524 (60)</td>
<td>514 (60)</td>
<td>10 (56)</td>
<td>0.68</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>252 (29)</td>
<td>249 (29)</td>
<td>3 (17)</td>
<td>0.3</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>350 (40)</td>
<td>339 (40)</td>
<td>11 (61)</td>
<td>0.07</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>188 (22)</td>
<td>182 (21)</td>
<td>6 (33)</td>
<td>0.22</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>101 (12)</td>
<td>98 (12)</td>
<td>3 (17)</td>
<td>0.25</td>
</tr>
<tr>
<td>COPD</td>
<td>87 (10)</td>
<td>84 (10)</td>
<td>3 (17)</td>
<td>0.24</td>
</tr>
<tr>
<td>Chronic renal failure*</td>
<td>99 (11)</td>
<td>95 (11)</td>
<td>4 (22)</td>
<td>0.04</td>
</tr>
<tr>
<td><strong>Treatment, n (%)†</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-blockers</td>
<td>231 (27)</td>
<td>227 (27)</td>
<td>4 (22)</td>
<td>0.79</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>250 (29)</td>
<td>246 (29)</td>
<td>4 (22)</td>
<td>0.79</td>
</tr>
<tr>
<td>ARA</td>
<td>97 (11)</td>
<td>95 (11)</td>
<td>2 (11)</td>
<td>1.0</td>
</tr>
<tr>
<td>Aspirin</td>
<td>239 (28)</td>
<td>231 (27)</td>
<td>8 (44)</td>
<td>0.1</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>65 (7)</td>
<td>64 (8)</td>
<td>1 (6)</td>
<td>1.0</td>
</tr>
<tr>
<td>Statins</td>
<td>184 (21)</td>
<td>183 (22)</td>
<td>1 (6)</td>
<td>0.14</td>
</tr>
<tr>
<td>Vitamin K antagonists</td>
<td>145 (17)</td>
<td>143 (17)</td>
<td>2 (11)</td>
<td>0.75</td>
</tr>
<tr>
<td>Diuretics</td>
<td>301 (35)</td>
<td>292 (34)</td>
<td>9 (50)</td>
<td>0.17</td>
</tr>
<tr>
<td><strong>Clinical examination‡</strong></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>139.5±34.8</td>
<td>140±34.9</td>
<td>118.7±21</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Median 135 (116–160)</td>
<td>135 (117–160)</td>
<td>123 (100–136)</td>
<td>0.01</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>78.0±18.7</td>
<td>78.3±18.8</td>
<td>64.3±10.3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Median 79 (65–90)</td>
<td>80 (65–90)</td>
<td>64 (55–71)</td>
<td>0.002</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>93.6±25.3</td>
<td>93.7±25.4</td>
<td>87.8±20.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Median 90 (75–108)</td>
<td>90 (75–108)</td>
<td>92 (69–108)</td>
<td>0.33</td>
</tr>
<tr>
<td>Temperature, °C</td>
<td>37.3±0.8</td>
<td>37.1±0.8</td>
<td>37.3±0.9</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Median 37 (37–37.6)</td>
<td>37 (37–37.5)</td>
<td>37.5 (36.8–38)</td>
<td>0.93</td>
</tr>
<tr>
<td><strong>Electrocardiogram, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sinus rhythm</td>
<td>605 (70)</td>
<td>593 (70)</td>
<td>12 (67)</td>
<td>0.78</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>207 (24)</td>
<td>203 (24)</td>
<td>4 (22)</td>
<td>1.0</td>
</tr>
<tr>
<td>Permanent pacing</td>
<td>52 (6)</td>
<td>50 (6)</td>
<td>2 (11)</td>
<td>0.29</td>
</tr>
<tr>
<td>AVB or SAB</td>
<td>5 (1)</td>
<td>5 (1)</td>
<td>0</td>
<td>1.0</td>
</tr>
</tbody>
</table>

*Defined as creatinine clearance <30 mL/min.
†Before admission.
‡At admission.
BPE and severe MR; \( P=0.14 \). The prevalence of severe MR among patients with BPE and UPE was 6\% and 100\%, respectively (\( P<0.0001 \)).

MR murmur was detected in similar proportion in patients with UPE and BPE (56\% versus 66\%, respectively; \( P=0.43 \)) and was significantly more perceptible in patients with organic MR than in patients with functional MR (83\% versus 43\%, respectively; \( P=0.0004 \)). In patients with UPE, plasma B-natriuretic peptide level was 1250 \( \pm \) 1157 versus 1150 \( \pm \) 828 ng/L in patients with BPE (\( P=0.82 \)). Leukocytosis was found more frequently in patients with UPE (72\% versus 40\% in BPE; \( P=0.02 \)), but fever was found in only 11\% of patients with UPE.

Echocardiographic characteristics of patients with severe MR are presented in Table 2. No significant echocardiographic difference was observed between UPE and BPE, except for the prevalence of grade 4/4 MR (56\% in patients with UPE versus 28\% in those with BPE; \( P=0.04 \)).

In patients with severe MR, noninvasive or invasive ventilation and catecholamine infusion were more frequent in UPE than in BPE: 61\% versus 26\% for ventilation (\( P=0.008 \)) and 44\% versus 11\% for catecholamine infusion (\( P=0.005 \)). In patients with UPE, use of antibiotic therapy was significantly higher than in patients with BPE associated with severe MR (61\% versus 6\%; \( P<0.0001 \)). Delay in treatment was significantly more frequent in patients with UPE than in patients with BPE and severe MR (33\% versus 4\%; \( P=0.003 \)). Mean time to initiation of diuretic treatment in patients with UPE was 12.6 hours (range, 0.5 to 72 hours) after the first medical contact.

In patients with UPE and organic MR (n=10), urgent mitral surgery was performed in 5 patients (2 valve repairs and 3 valve replacements), and in the 5 remaining patients, surgery was not performed because of age (82.4 \( \pm \) 6.8 years), comorbidities (n=2), low LVEF (LVEF <20\% in 1 patient), and/or multiorgan failure (n=3). All patients who underwent surgery survived, whereas only 1 patient survived among the 5 patients who were not referred for emergency surgery. In patients with BPE, emergency mitral surgery was performed in 12 patients (23\%): 8 mitral valve repairs and 4 mitral valve replacements.

**Management According to the Care Unit**

Patients admitted to the intensive care unit (n=179) were younger than patients (n=690) admitted in coronary care unit (70 \( \pm \) 13 versus 77 \( \pm \) 13 years; \( P<0.0001 \)) and had a more severe clinical presentation (significantly lower systolic and diastolic blood pressures; \( P=0.03 \) and \( P=0.0001 \), respectively). Noninvasive or invasive ventilation and catecholamine use were significantly more frequent in patients admitted to the intensive care unit (71\% versus 14\%, \( P<0.0001 \); and 41\% versus 6\%, \( P<0.0001 \), respectively). The administration of antibiotic therapy was significantly higher in the intensive care unit than in the coronary care unit (39\% versus 11\%; \( P<0.0001 \)). Antibiotic therapy was prescribed in 36\% (49 of 88) of patients needing catecholamine use or invasive ventilation versus 13\% (96 of 636) of patients with pulmonary edema who did not require catecholamine use or invasive ventilation (\( P<0.0001 \)). The rate of mortality was significantly higher in patients hospitalized in an intensive care unit (22\% versus 6\%; \( P<0.0001 \)).

**Prognosis**

Among the global population of patients with pulmonary edema, in-hospital mortality was 9\% (79 patients); 39\% in
patients with UPE versus 8% in patients with BPE (OR, 6.9; 95% CI, 2.6 to 18; P=0.001). Table 3 summarizes the multivariate analysis for identifying variables independently associated with death. Whatever the multivariate model used, unilateral location of pulmonary edema was independently related to death (aOR, 6.5; 95% CI, 1.3 to 32; P=0.021 for model A; and aOR, 6.8; 95% CI, 1.1 to 41; P=0.037 for model B).

Among the population of patients presenting with severe MR and pulmonary edema (n=71), in-hospital mortality was significantly higher in patients with UPE: 39% versus 6% in patients with BPE (OR, 10.6; 95% CI, 2.4 to 48; P=0.0021). The association was also significant in multivariate analysis when controlling for variables from the first set (aOR, 9.0; 95% CI, 1.5 to 54; P=0.016 using model A; and aOR, 7.8; 95% CI, 1.0 to 62; P=0.05 using model B).

Discussion
The main results of the present study are as follows: The prevalence of UPE was 2.1% among a large population of patients with cardiogenic pulmonary edema; UPE was always associated with severe MR (organic or functional MR); a delay in the initiation of appropriate treatment was frequent; and UPE is related to an increased risk of mortality.

UPE is an old and unusual medical entity with a prevalence that has not been studied recently. In our population of patients with cardiogenic pulmonary edema, we found an estimated UPE prevalence of 2.1%, showing that UPE is not as unusual as presumed. Vascular or bronchial obstruction, congenital heart disease, and prolonged rest on one side may induce UPE, but severe MR remains the main cause of UPE.

In our study, UPE was always associated with severe MR and represented 25% of pulmonary edema with severe MR. Two previous studies found that 9% and 22% of patients presenting with severe MR and signs of congestive heart failure had radiological signs of UPE, mainly in the right upper lobe. Mitral valve anatomy may explain the mechanism of UPE, the regurgitant flow being directed toward the right pulmonary veins, particularly the superior right pulmonary vein. Severe organic MR consisting of posterior leaflet prolapse is particularly involved in UPE, as demonstrated in our study. Interestingly, Gurney and Goodman found that the back pressure V wave and the mean pulmonary capillary wedge pressure were higher in the right upper lobe than in the left lung. These focal increased pressures may thus promote the unilateral presentation. In our series, UPE was right-sided in 89% of cases, and left-sided UPE was infrequent and represented only 0.2% of all cardiogenic pulmonary edema. These different chest x-ray patterns may be explained by many factors, including direction and severity of MR but also anatomic position of pulmonary veins in the left atrium and anatomic drainage of pulmonary veins. The main mechanism of MR in UPE is mitral leaflet prolapse, but functional MR may also be involved, as demonstrated in our study and in previous case reports.

The unilateral x-ray pattern may lead to false diagnosis of pneumonia and delayed management. In 18 pregnant women presenting with pulmonary edema, Choi et al have shown that patients with UPE had initial misdiagnosis (pneumonia), resulting in delays in treatment and resolution (4 to 5 days). In our study, a high rate of antibiotic therapy (61%) and
leukocytosis (72%) was observed in patients with UPE. This leukocytosis was rarely associated with fever (11%), and all the bacteriologic samples were sterile. Leukocytosis has previously been reported in patients with UPE resulting from severe MR in the absence of proven infection.23,24 The association of unilateral pulmonary infiltrates and/or leukocytosis and/or acute respiratory distress led to the initiation of antibiotic therapy, usually in the absence of fever. This may delay therapeutic management of UPE; initiation of appropriate treatment was delayed in 33% of our UPE cases versus 4% in patients without UPE \( (P=0.003) \), highlighting the difficulty of immediately making the correct diagnosis of UPE. Furthermore, patients with UPE presented with a high risk of mortality (39%), with a risk of death 6.9-fold higher than that of patients with BPE, and delay in adequate treatment of UPE could be one of the explanations for this increased mortality. As expected, we found that the clinical presentation of patients admitted to the intensive care unit was more severe with increased use of catecholamine and mechanical ventilation and that the prognosis of these patients was worse. Use of antibiotic therapy was also more frequent and thus was related to the severity of clinical condition at admission. The clinical presentation of UPE may be misleading, with an initial misdiagnosis of pneumonia. In multivariate analysis including the location of hospitalization (model B), the unilateral location of pulmonary edema remained associated with an independent risk of mortality \( (\text{aOR}, 6.8; 95\% \ CI, 1.1 \text{ to } 4.1; \ P=0.03) \), illustrating the severe prognosis of this particular pattern of pulmonary edema.

Absence of fever, sudden onset of dyspnea, history, and level of B-natriuretic peptide may help to differentiate UPE from other diagnoses.24 A murmur on examination can be useful, especially for organic MR. However, the intensity of the murmur is lower and correlates poorly with the degree of regurgitation in ischemic MR.25 Given that the sensitivity in detecting MR at bedside examination is highly variable, especially for a patient presenting with respiratory distress, the key examination remains bedside transthoracic echocardiography. Transesophageal echocardiography can also be useful in determining the severity and mechanism of MR and in documenting the differential pressure between the right and left pulmonary veins.26 If early recognition is crucial, prompt management is also important. In patients with UPE, 4 deaths were observed in patients with organic MR who did not undergo emergency mitral surgery. In the Euro Heart Survey, Mirabel et al27 found that 49% of patients with symptomatic severe MR were denied surgery despite the recommendations of current guidelines.26,28 In our study and the study by Mirabel et al,27 the main reasons for not performing surgery were age, presence of comorbidities, and low LVEF. Prompt surgical intervention should be discussed and could improve prognosis.30,31

**Study Limitations**

The main limitation of our report is that this is a retrospective study of a rare condition. UPE has been described mainly in case reports; no large prospective studies have been performed. In our study, we systematically reviewed all chest x-rays of a large population of patients with acute cardiogenic pulmonary edema \( (n=869) \), and despite a low number of patients with UPE, we were able to obtain statistically significant results.

**Conclusions**

UPE represented 2.1% of all cardiogenic pulmonary edema in our study. It usually appeared as an opacity involving the right upper lobe and was always associated with severe MR (organic or functional MR). UPE should be promptly recognized to avoid delays in treatment that affect prognosis. Finally, UPE is related to an independent increased risk of mortality.

**Disclosures**

None.

**References**

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Cardiogenic bilateral pulmonary edema usually appears on chest x-rays as bilateral and symmetrical opacities, resulting in the classic “butterfly shadow.” Cardiogenic unilateral pulmonary edema (UPE) is an unusual entity. Little is known about the characteristics of UPE, and its prevalence has never been assessed in a large series of pulmonary edema. Among a large population with cardiogenic pulmonary edema (n=869), we found that the prevalence of the unilateral pattern of cardiogenic pulmonary edema was 2.1%; that UPE was right-sided in 89%, involving mainly the right upper lobe; and that UPE was always associated with severe organic or functional mitral regurgitation. In-hospital mortality of UPE was significantly higher (39% versus 8% in patients with bilateral pulmonary edema). In multivariate analysis, UPE was independently related to death, with a risk of death 6.5-fold higher than in patients with bilateral pulmonary edema. Because of initial misdiagnosis caused by the unilateral location of opacities on chest x-rays, a delay in diagnosis and adequate treatment was observed in 33% of cases. These results have implications in routine practice to best recognize this clinical entity and thus to avoid delay in treatment that may affect prognosis.
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Prevalence, Characteristics, and Outcomes of Patients Presenting With Cardiogenic

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