Unilateral pulmonary edema is a distinctly unusual clinical entity, often misdiagnosed initially as one of the more common causes of focal lung disease. Predominantly lobar pulmonary edema is rarer still. We report a case of right upper lobe pulmonary edema caused by the acute onset of severe mitral regurgitation. In addition, we briefly review the other causes of unilateral pulmonary edema, focusing on the cases that have been reported in association with heart failure and valvular heart disease. The majority of cases of right upper lobe pulmonary edema have been associated with mitral regurgitation. In addition to confirming the presence of mitral regurgitation, transesophageal echocardiography proved useful in delineating the mechanism for edema formation. It detected differential gradients between the right and left pulmonary venous systems and documented the direction of the regurgitant flow.

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Right Upper Lobe Pulmonary Edema Caused by Acute Mitral Regurgitation*

Diagnosis by Transesophageal Echocardiography

James M. Roach, M.D.; Karl C. Stajduhar, M.D.; and Kenneth G. Torrington, M.D., F.C.C.P.

Pulmonary edema can be defined as a pathologic state in which extravascular liquid accumulates in the lungs. This generally occurs because liquid is being filtered across a semipermeable membrane (the pulmonary microvascular endothelial surface) faster than it can be removed. The factors that determine the rate of liquid and solute exchange across this type of membrane have been well described by the Starling equation and are largely dependent on the differences in hydrostatic pressure and protein osmotic pressure between the pulmonary microvasculature and the perimicrovascular interstitial space. Edema occurs when alteration of one or more of these factors favors movement of fluid out of capillaries to the interstitial spaces (as in this case); furthermore, fluid may eventually move into the alveolar spaces. In the great majority of instances, the edema will occur bilaterally; rarely unilateral or even lobar edema will develop when focal imbalance of these forces occurs. We report the case of a patient with right upper lobe pulmonary edema associated with severe mitral regurgitation, and we review similar cases reported in the literature.

In our patient, transesophageal echocardiography was very useful in diagnosing the cause of as well as determining the mechanism of edema formation.

CASE REPORT

A 73-year-old white man presented with the complaints of the acute onset of chest pain, "achey" muscles and fevers to 39.3°C. He sought medical attention and a new grade 4/6 holosystolic murmur was noted; he was admitted to the hospital to evaluate the possibility of subacute bacterial endocarditis. Medical history was significant only for mitral valve prolapse documented by echocardiography in 1986. Physical examination revealed a well-developed, well-nourished white man in no distress. Vital signs were normal. Lung examination revealed mild basilar rales on the right, and cardiovascular examination revealed an S3 and a grade 4/6 holosystolic murmur that radiated to the axilla. There were no Janeway lesions, Roth spots, or splinter hemorrhages.

Laboratory evaluation revealed a hemoglobin value of 12.4 g/dl and WBC of 8,300 cu/mm. ESR was 52 mm/h. Abnormalities on screening chemistries included a total protein level of 5.8 g/dl, an

*From the Pulmonary and Critical Care Medicine Service, Department of Medicine, Walter Reed Army Medical Center, Washington, DC, and the Uniformed Services University of the Health Sciences, Bethesda, Md.

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LA = left atrium; LPV = left pulmonary vein; LV = left ventricle; MR = mitral regurgitation; RPV = right pulmonary vein; UPE = unilateral pulmonary edema

**Figure 1.** Posteroanterior and lateral chest roentgenograms revealing the presence of right-sided (largely upper lobe) unilateral pulmonary edema.
angiography, pleural infiltrate. A higher venous raphy confirmed the presence of a unilateral interstitial infiltrate, sparing the cortex of the lung. There is also a right pleural effusion, which is an incidental finding along the posterior chest wall.

albumin level of 3.1 g/dl, and an elevated SGOT (88 IU/L), SGPT (99 IU/L), and LDH (325 IU/l). Multiple sets of blood cultures were negative, as were surveys for "culture negative" endocarditis. Chest roentgenogram (Fig 1) revealed an enlarged cardiac silhouette, a right pleural effusion, and an interstitial infiltrate located predominantly in the right upper lobe. Chest computed tomogram (CT) (Fig 2) confirmed this finding and noted relative sparing of the periphery. Transthoracic echocardiography revealed a myxomatous mitral valve with a flail posterior leaflet and severe mitral regurgitation (MR). Right heart cardiac catheterization showed an elevated mean right main pulmonary artery pressure of 27 mm Hg, and a mean pulmonary capillary wedge pressure of 21 mm Hg. Coronary angiography was normal. Left ventriculography showed severe mitral valve prolapse and 4+ MR. Transesophageal echocardiography by pulse wave Doppler demonstrated reversal of pulmonary venous inflow in both the left and right pulmonary veins consistent with severe MR. Pulmonary venous inflow velocity sampled at the right pulmonary vein (RPV) orifice was significantly greater than that sampled at the left pulmonary vein (LPV), indicating a much higher pressure gradient from the RPV to the right atrium (LA) as compared with the pressure gradient from the LPV to the LA. Color Doppler noted a "mosaic of flow" or a high-velocity regurgitant jet directed at the RPV. The patient was placed on a regimen of afterload reduction and diuretics and his infiltrate and symptoms slowly resolved. We attributed his fever to pulmonary edema or perhaps a small myocardial infarction as no other source was identified. He has not required surgical repair of the flail mitral leaflet to date.

**Discussion**

Though uncommon, unilateral pulmonary edema (UPE) has been reported in association with several different clinical situations (Table 1). Focal imbalance of Starling forces is the common thread that unites this wide variety of etiologies. "Reexpansion" pulmonary edema, which occasionally occurs following thoracentesis or treatment of a pneumothorax, accounts for the majority of reported cases. Several cases have also been reported in the recent literature in association with left ventricular failure. In 1971, Bahl et al. reported the case of a man with "an unusual presentation of left heart failure, in that UPE, due to mitral insufficiency [confirmed by cardiac catheterization], was seen radiographically to be localized largely to the right upper lobe." Gamsu et al. in 1981, reported two cases of isolated right upper lobe pulmonary edema; one of these patients had acute MR. In this patient, Swan-Ganz catheter in the right upper lobe pulmonary artery confirmed a pressure of 78/40 mm Hg and a pulmonary capillary wedge pressure of 44 mm Hg; left-sided pressure measurements were not reported. He was placed on a regimen of nitroprusside and the infiltrate resolved in 24 h. Kusumi et al. in 1984 discussed two patients with UPE associated with LV failure, one of whom had MR confirmed by cardiac catheterization. In 1986, Keren et al. described a man who developed UPE associated with acute myocardial infarction; he had "signs of mitral regurgitation by auscultation."

Recently, Gurney and Goodman reported four cases of "pulmonary edema localized in the right upper lobe accompanying mitral regurgitation." In one of their patients, cardiac catheterization revealed a mean left pulmonary artery pressure of 33 mm Hg and a mean right upper lobe pulmonary artery pressure of 50 mm Hg. They speculated that the edema accumulated in the right upper lobe because the vector of blood flow from the left ventricle (LV) to the LA was targeted preferentially at the right superior pulmonary vein (thus increasing hydrostatic pressure locally). As we were not contemplating this etiology at the time of cardiac catheterization, differential right- and left-sided pressures were not obtained. Transesophageal echocardiography, however, confirmed that the regurgitant jet was directed at the right pulmonary venous system; further pulsed Doppler demonstrated that the velocity of flow, and therefore the pressure gradient, was much greater at the orifice of the right pulmonary venous system as compared with the left. To our knowledge, this is the first report of transesophageal echocardiography being useful in delineating the mechanism of edema formation in a patient with UPE.

In summary, 12 cases of UPE associated with LV failure and have been reported in the English literature in the last 20 years; interestingly, all patients had right-sided edema, eight had documented MR, and six of these eight had edema

**Table 1—Reports of Unilateral Pulmonary Edema**

<table>
<thead>
<tr>
<th>Association</th>
<th>Reference(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reexpansion after thoracentesis or pneumothorax</td>
<td>3-9, 21</td>
</tr>
<tr>
<td>Left ventricular failure (including mitral regurgitation)</td>
<td>10-16, 21</td>
</tr>
<tr>
<td>Pulmonary arterial compression</td>
<td>17-18</td>
</tr>
<tr>
<td>Creation of systemic to pulmonary artery shunts</td>
<td>19-21</td>
</tr>
<tr>
<td>Lateral decubitus position</td>
<td>22-24</td>
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<tr>
<td>Misplacement of central venous pressure catheter</td>
<td>25</td>
</tr>
<tr>
<td>Swyer-James syndrome with left ventricular failure</td>
<td>26</td>
</tr>
<tr>
<td>Postoperative pulmonary vein thrombosis</td>
<td>27, 28</td>
</tr>
<tr>
<td>Following removal of a pleural tumor</td>
<td>29</td>
</tr>
<tr>
<td>Epilepsy</td>
<td>30</td>
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<tr>
<td>Associated with old poliomyelitis</td>
<td>31</td>
</tr>
<tr>
<td>Intubation of right main-stem bronchus</td>
<td>32</td>
</tr>
<tr>
<td>Acute bronchial obstruction by foreign body/ tumor</td>
<td>33, 34</td>
</tr>
<tr>
<td>Intrapleural nitrogen mustard therapy</td>
<td>35</td>
</tr>
</tbody>
</table>
largely in the right upper lobe. In the majority of these cases, the problem was initially misdiagnosed as a more common cause of focal lung disease (eg, pneumonia). Our case and literature review should alert physicians to the possibility of UPE developing in patients with acute MR. Further, transthoracic echocardiography may be a useful modality in documenting differential pressure gradients between the right and left pulmonary venous systems and in documenting the direction of regurgitant flow.

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Fatal Obstruction of the Left Ventricular Outflow Tract Caused by Low-Profile Bioprostheses in the Mitral Valve Position*

Uberto Bortolotti, M.D., F.C.C.P.; Aldo Milano, M.D.; Vincenzo Tursi, M.D.; Marco Minarinti, B.S.; Gaeto Thieme, M.D.; and Alessandro Mazzucco, M.D.

Two cases of fatal left ventricular outflow tract obstruction following mitral valve replacement with low-profile bioprosthesis valves are described. This unfortunate experience highlights the importance of bioprosthesis valves in patients with a small left ventricular cavity.

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*From the Departments of Cardiovascular Surgery and Pathology, University of Padova Medical School, Padova, Italy.
†Department of Pathology.
Reprint requests: Dr. Bortolotti, Istituto di Chirurgia Cardiovascolare, Universita di Padova, Padova 35129, Italy

Fatal Obstruction of LV Outflow Tract (Bortolotti et al)