Hyperlucent Lung in a Patient With Mitral Valve Disease*

Abdullah F. Mobeireek, MBBS, FCCP; Ismail Joharjy, MD; and Abdulrah F. Mobeireek, MD

A 15-year-old boy with rheumatic valvular heart disease was referred for cardiac surgery. His chief complaints were increasing shortness of breath (New York Heart Association class III) and palpitations. He was well otherwise previously. He specifically denied any significant respiratory troubles. He was receiving digoxin, furosemide, and warfarin sodium. On physical examination, he had an irregular pulse (80 beats per minute) and BP of 100/70 mm Hg. Cardiovascular examination showed an increased jugular venous pressure (9 cm above sternal angle), displaced apex to the sixth intercostal space and anterior axillary line, sustained parasternal lift, decreased intensity of S₁, a loud pulmonary component of S₂, an opening snap, a mid-diastolic murmur and a loud pansystolic murmur at the apex, and an enlarged liver (4 cm below the costal margin). Chest examination showed reduced expansion on the left side, a resonant percussion note bilaterally, markedly reduced breath sounds on the left side, and fine basal crackles on the right side.

*From the Departments of Medicine, Pulmonary, Radiology, and Cardiology Divisions, College of Medicine, King Saud University, Riyadh, Saudi Arabia.

Manuscript received July 31, 1997; revision accepted January 6, 1998.

Correspondence to: Abdullah F. Mobeireek, MBBS, FCCP, Dept of Medicine (38), College of Medicine, King Saud University, PO Box 2925, Riyadh 11462, Saudi Arabia

Figure 1. The frontal and lateral chest radiograph on presentation.
A chest radiograph is shown in Figure 1. The ECG showed atrial fibrillation at a rate of 120 beats/min and right axis deviation. Echocardiography showed an enlarged left atrium (LA) to 70 mm, a mitral valve area of 1.4 cm², and a dilated left ventricle with decreased ejection fraction to 40%. Doppler echocardiography showed severe mitral regurgitation, moderate mitral stenosis, moderate tricuspid regurgitation and stenosis, moderate aortic regurgitation, and moderate pulmonary hypertension. Arterial blood gas values were within normal limits.

Just before surgery, the pulmonologist was consulted for a left hyperlucent lung. He suggested postponing the surgery for further tests, including a CT scan, a ventilation-perfusion scan, and possibly bronchoscopy. The patient’s father refused to let him have these tests and insisted on his son’s having the surgical operation at the scheduled time. The surgical operation (annuloplasty and commissurotomy of the mitral and tricuspid valves) was performed uneventfully. Postoperatively, breath sounds could be heard on both sides, and there were no added sounds. The chest radiograph is shown in Figure 2. The size of the LA decreased from 70 to 45 mm on a follow-up echocardiography.

What is the Diagnosis?
Diagnosis: Right-sided unilateral pulmonary edema secondary to mitral regurgitation and a left hyperlucent lung resulting from partial obstruction of the left main bronchus by a large LA.

Several conditions can predispose a patient to unilateral pulmonary edema. The post-lung reexpansion situation is the most commonly reported, but there are several others that were reviewed in detail by Calenof et al. and more recently by Roach et al. Unilateral pulmonary edema secondary to congestive heart failure is a rare but well-recognized entity. In 1993, Roach et al found 12 cases in the English-language medical literature, and since then, only one more case has been reported to the authors’ knowledge. Similar to the patient reported here, all except the last, had right-sided pulmonary edema and eight had mitral regurgitation. In the latter situation, the jet of mitral regurgitation is eccentric and directed to the right upper and lower pulmonary veins, causing a rise in the pulmonary venous pressure in the right lung. This leads to transudation of fluid into the interstitium and alveoli, producing pulmonary edema by changing the balance of the Starling forces.

Does this alone provide the answer for the chest roentgenogram abnormality? In other words, was the surgeon deceived by the visual illusion by calling this a hyperlucent lung while it was in fact a right hypoluculent lung? No, there was also a true hyperlucency evident from the decrease in both the number and caliber of the vascular markings on the left side. This is because of compression of the grossly enlarged LA on the left main bronchus and the lobal bronchi. Clues for this include the striking reduction of breath sounds on the left side and the suggestive appearance on the lateral chest roentgenogram (Fig 1). Also, left atrial enlargement is known to cause splaying of the carina and occasionally atelectasis of the left lower lobe or the entire lung. In this case, hyperlucency occurred rather than atelectasis because obstruction was partial.

The mechanism of hyperlucency was nicely explained by Gaensler with the use of the West diagram. He proposed that when there is obstruction, the extra-alveolar pressure is higher than atmospheric pressure. This leads to collapse of the intrathoracic arteries, thereby resulting in hyperlucency. Arteriolar vasoconstriction because of local hypoxia also was suggested as another possible mechanism, but Gaensler considered this a less likely one since the larger arteries are believed to account for the hypolucency. In either case, the balance of the Starling forces will change to protect the ipsilateral lung from pulmonary edema, while the situation will be aggravated in the contralateral lung because of the increased flow.

Unilateral pulmonary edema and contralateral hyperlucent lung were described before in association with the Swyer-James or MacLeod syndrome and pulmonary artery agenesis. The former is an unlikely diagnosis in this patient because of the lack of a medical history of previous respiratory complaints and infections and the presence of proximal airway obstruction on a chest radiograph. Also, the patient had immediate improvement after surgery although more recovery may take a longer time. Pulmonary artery agenesis also is ruled out because in this condition the hilum is absent and the hyperlucency is usually in the contralateral lung.

In summary, this case shows right-sided unilateral pulmonary edema resulting from mitral regurgitation and simultaneous presence of a left hyperlucent lung because of compression of bronchi by a large LA. No similar case appears to have been described in the English-language medical literature to the authors’ knowledge. Cardiomegaly should not be overlooked as a cause of bronchial obstruction since this may be a contributing factor in dyspnea in the cardiac patient.

References