Chronic Pulmonary Emboli*
Sparing of Affected Regions of Lung from Noncardiogenic Pulmonary Edema

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In chronic pulmonary emboli, lung perfusion may be maintained by either recanalization of occluded pulmonary artery segments or collateral bronchial circulation. We present a case of noncardiogenic pulmonary edema superimposed on chronic pulmonary emboli, in which the occluded segments of lung were spared from pulmonary edema, but not from neutrophil infiltration. This case demonstrates that chronic emboli may lead to roentgenographic sparing in noncardiogenic pulmonary edema, similar to that previously reported in acute pulmonary emboli superimposed on noncardiogenic edema.

Several studies have suggested that acute pulmonary emboli prevent development of pulmonary edema in occluded lung segments. The mechanism appears to involve a local decrease in hydrostatic perfusion pressure secondary to acute occlusion of the pulmonary vascular supply. Since patients with chronic pulmonary emboli may develop collateral circulation from the bronchial arteries or recanalize the occluded segments of pulmonary artery, it is unclear whether chronic pulmonary emboli may similarly prevent pulmonary edema in lung segments. We report a case of chronic pulmonary emboli with superimposed pulmonary edema. Pulmonary edema did not occur in lung segments with chronically occluded pulmonary arterial vascular supplies. Therefore, chronic pulmonary emboli can be as effective as acute pulmonary emboli in preventing pulmonary edema in affected regions of the lung. The development of characteristic roentgenographic findings in a patient with pre-existing pulmonary hypertension should lead the clinician to suspect this situation and may provide a clue to the etiology of the pulmonary hypertension.

Case Report

A 61-year-old white man was admitted to the hospital for evaluation of new onset seizures. Past medical history was significant for a long history of deep venous thrombosis complicated by recurrent pulmonary emboli, pulmonary hypertension, and cor pulmonale. An umbrella had been inserted into the inferior vena cava many years prior to admission and the patient was receiving long-term coumarin therapy. Significantly, the patient had a two-week history of post-tussive syncope, and on admission, was noted to have several generalized tonic clonic seizures that were also immediately preceded by coughing. Neurologic examination was unremarkable, and remaining physical examination was significant only for signs consistent with pulmonary hypertension and cor pulmonale. Admission laboratory values included a normal glucose value, normal elec-

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trolyte levels, Ca++, Mg++, a protime of 23, and arterial blood gas values as follow: pH, 7.48; Pco2, 22, and Po2, 58 on 2L/min of O2. Chest roentgenogram (Fig 1) revealed cardiomegaly and pulmonary hypertension. Head CT scan demonstrated cortical atrophy and an old left temporal infarct, but was otherwise unremarkable. Syncope workup (including Holter, serial ECGs, and cardiac enzymes) was negative. The patient was treated with phenytoin and initially did well without further seizure activity. Two days following admission, he complained of nausea and recurrent vomiting. He subsequently developed a temperature of 103°F, marked respiratory distress, and progressive hypoxemia. Repeat chest roentgenogram (Fig 2) demonstrated bilateral alveolar infiltrates with significant sparing of the right and left lung bases. Sputum Gram stain revealed numerous polys and mixed bacterial flora. A clinical diagnosis of aspiration pneumonitis and adult respiratory distress syndrome was made. The patient was intubated, mechanically ventilated with 100 percent O2, and was treated with chloramphenicol, amikacin, and high dose methylprednisolone. Myocardial infarction was ruled out by serial enzyme studies and ECGs. Despite these interventions, the patient developed progressive hypoxemia, hypotension, and metabolic acidosis. Three days following admission, he suffered a cardiac arrest and could not be resuscitated.

Pertinent autopsy findings included pulmonary artery atherosclerosis and right ventricular and right atrial enlargement. No evidence of myocardial infarction was found. In addition, a large thrombus was noted in the auricle of the right atrium. No clots were noted at the umbrella site. Examination of the lungs revealed old emboli which occluded most of the pulmonary artery supply to the right lower lobe (Fig 3) and a large portion of the pulmonary artery supply to the left lower lobe. No acute pulmonary emboli were found, and the embolized segments of lung demonstrated no evidence of acute infarction. The lungs were also noted to have patchy areas of pneumonia and edema. Specifically, alveolar edema did not occur in segments of the right and left lower lobes with occluded pulmonary artery vascular supplies. These findings were consistent with the picture seen on the chest roentgenogram (Fig 2). Of note, however, was the observation that all segments of the lung (with the exception of a small area in the left upper lobe which revealed neither neutrophil infiltration nor edema) demonstrated marked endobronchial and intraalveolar neutrophil infiltration. Furthermore, neutrophil infiltration appeared to be comparable in lung segments with occluded and nonoccluded pulmonary artery vascular supplies. The lack of evidence of myocardial infarction and presence of neutrophil infiltration were most consistent with a diagnosis of aspiration pneumonitis and noncardiogenic pulmonary edema.

**Discussion**

Several case reports have suggested that acute pulmonary emboli appear to prevent nonperfused segments of lung from developing pulmonary edema. Specifically, Hyers et al and Williams et al reported two cases of pulmonary emboli. In both cases, pulmonary edema appeared to be confined to nonembolized segments of lung. In addition, Bedard et al reported two cases of ARDS and angiographically-proven

**Figure 1.** Admission chest x-ray film, demonstrating enlarged pulmonary arteries.

**Figure 2.** Subsequent chest x-ray film, demonstrating diffuse alveolar infiltrates with sparing of both bases.

**Figure 3.** Post-mortem specimen, demonstrating occlusion of pulmonary artery to right lower lobe by fibrotic organized thrombus.
acute pulmonary emboli. The distinctive feature of these cases was that ARDS preceded the development of pulmonary emboli. However, despite the pre-existence of ARDS, embolized segments of lung were subsequently demonstrated to exhibit roentgenographic clearing of edema. While these reports support the concept that acute pulmonary emboli can prevent pulmonary edema in nonperfused segments of lung, it was not clear whether chronic pulmonary emboli could afford the same effect, since patients with chronic pulmonary emboli may have more effective collateral circulation (or recanalize their previously occluded segments). This case demonstrates that chronic pulmonary emboli can cause sparing from pulmonary edema.

The exact mechanism by which pulmonary emboli spare nonperfused lung segments from noncardiogenic pulmonary edema is not known. In this case, it would appear that chronic emboli did not cause a decreased inflammatory stimulus since we observed equal numbers of neutrophils in embolized and nonembolized segments of lung. Furthermore, the fact that neutrophils were able to accumulate in embolized segments of lung suggests that perfusion of these segments was of sufficient quantity to allow an inflammatory response. One could speculate, therefore, that chronic pulmonary emboli do not prevent pulmonary edema by altering underlying inflammatory mechanisms or preventing increased alveolar capillary membrane permeability. Rather, it is likely that chronic pulmonary emboli merely prevent the edematous expression of this increased permeability by decreasing hydrostatic perfusion pressures. This concept is supported by the studies of Tate et al., who demonstrated that isolated lungs perfused with chemically generated oxygen metabolites developed noncardiogenic pulmonary edema which could be prevented by using a vasodilator to prevent associated increases in pulmonary artery perfusion pressures. Furthermore, it was clear that the vasodilator prevented pulmonary edema by decreasing hydrostatic perfusion pressures (rather than altering permeability) since a modest mechanical pressure challenge caused severe pulmonary edema in oxygen metabolite and vasodilator perfused lungs, but did not cause edema in normal lungs. Since Bedard’s cases demonstrated pre-existence of increased permeability, it seems likely that acute pulmonary emboli spare nonperfused lung segments from pulmonary edema through a similar hydrostatic pressure mechanism.

Iannuzzi et al. reported that patients with diffuse alveolar infiltrates secondary to noncardiogenic pulmonary edema often demonstrate a relative sparing of the lung bases on chest roentgenogram. While this pattern of basilar sparing is certainly well recognized, it must be noted that the lung bases are also the most frequent location of pulmonary emboli. Since patients with noncardiogenic pulmonary edema are often at risk for the development of pulmonary emboli, the possibility of underlying or coexisting pulmonary embolism should be considered in patients with noncardiogenic pulmonary edema who demonstrate significant sparing of a lung zone on chest roentgenogram.

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Pulmonary Edema following Conversion of Tachyarrhythmia*
A Case following Burst Atrial Pacing
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Restoration of normal sinus rhythm is usually followed by improved hemodynamics. By contrast, pulmonary edema and cardiovascular collapse have been reported following successful electrical reversion of various tachyarrhythmias to normal sinus rhythm. The mechanism for this adverse reaction is not clear but has been thought to relate, at least in part, to electrical myocardial damage from the counter-shock. This report describes a patient in whom this complication occurred on two occasions, first following external countershock and subsequently following burst atrial pacing. Thus, conversion to sinus rhythm may be responsible for this phenomenon independent of the method of conversion.

Although not recently a subject for review in the literature, pulmonary edema with and without cardiovascular collapse following restoration of sinus rhythm has previously been reported in 1 to 3 percent of patients following electrical reversion of atrial flutter, atrial fibrillation, and ventricular tachycardia.12 This report describes a patient in whom atrial flutter was treated once with cardioversion and subsequently with burst atrial pacing; each time conversion to sinus rhythm was accompanied by pulmonary edema and cardiovascular collapse. We are unaware of a previous report of this complication following overdrive atrial pacing, an observation which appears to exclude electrical injury to the myocardium as the only cause for this syndrome.

Case Report
A 62-year-old man underwent coronary artery bypass surgery in April 1984. Preoperatively, radionuclide ventriculography demonstrated a resting ejection fraction of 43 percent. One month following an unremarkable recovery from surgery, he developed atrial flutter with a ventricular rate of 150. After digitalization, he was cardioverted to sinus rhythm with a single shock of 20 joules. In

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