Bilateral Reexpansion Pulmonary Edema Following Unilateral Pleurocentesis*

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Acute ipsilateral pulmonary edema following reexpansion of the lung after pleurocentesis or pneumothorax is a well described entity. We report the unusual occurrence of bilateral pulmonary edema following unilateral pleurocentesis in a young male without heart disease. Various hypotheses regarding the mechanism of reexpansion pulmonary edema include increased capillary permeability due to hypoxic injury, decreased surfactant production, altered pulmonary perfusion and mechanical stretching of membranes. This case suggests that forces leading to ipsilateral reexpansion pulmonary edema also affect the contralateral lung.

(Acute) partial pressure of O₂; (pCO₂) partial pressure of CO₂; pH = negative logarithm of hydrogen ion activity

Acute ipsilateral pulmonary edema following lung reexpansion after pleurocentesis or treatment of pneumothorax is a well described clinical phenomenon that may have serious consequences. However, there have only been three reported cases of either bilateral or contralateral pulmonary edema after lung reexpansion in the 20th century. All three have occurred in patients with significant underlying heart or renal disease. We report the occurrence of bilateral acute pulmonary edema following unilateral pleurocentesis in a young man.

CASE REPORT

A 36-year-old male, nonsmoker, former alcoholic without other prior medical or surgical problems experienced progressive low-back pain, weight loss and intermittent vomiting over a two-year period. Evaluation including a chest x-ray film in November 1984 revealed no abnormalities. An extensive radiologic evaluation in December 1984 for right upper extremity weakness revealed herniation of C5/C6 disc toward the right, a large retroperitoneal mass and a small left pleural effusion. The herniated disc was removed surgically. Biopsy of the retroperitoneal mass revealed poorly differentiated sarcoma. One month later, after he experienced progressive dyspnea for one week, a chest x-ray film revealed a massive left pleural effusion with deviation of the mediastinum toward the right.

The patient was transferred to the hospital for evaluation of progressive dyspnea. Blood pressure at arrival was 122/68 mm Hg, the heart rate was 140 beats per minute and the respiratory rate was 30 breaths per minute. Relevant physical findings at admission were absent breath sounds and dullness to percussion on the left, clear right lung to auscultation, elevated left and normal right jugular venous pressures, a 2-cm anterior cervical lymph node, a palpable mass on the left side of the abdomen, grade 2/6 systolic ejection murmur at the left sternal border without S₂ or S₃ sounds and absence of dependent edema. On admission, a left decubitus radiograph demonstrated a massive left pleural effusion, plate-like atelectasis at the right base and deviation of the mediastinum toward the right.

Left pleurocentesis was performed using a sterile technique with a 14-gauge catheter attached to liter-sized vacuum collection bottles at −53 mm Hg of vacuum; 4,150 ml of blood-tinged exudate was drained in less than 30 min with relief of symptoms and return of left jugular venous pressure to normal. Cultures and cytology of the fluid were later reported as normal.

On completion of the pleurocentesis, the radiograph demonstrated a residual left hydropneumothorax and perihilar opacification of the left lung (Fig 1). The right lung was clear other than minimal plate-like atelectasis. The mediastinum returned to normal position and the heart was normal in size. During the next few days

Figure 1. Chest radiograph on completion of the pleurocentesis demonstrates residual left hydropneumothorax and perihilar opacification of the left lung with minimal plate-like atelectasis at right base. The midline has returned to normal position.

Figure 2. Chest radiograph 4½ h after pleurocentesis shows increased opacification of the left lung and perihilar opacification of the right lung.

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The acute appearance of bilateral interstitial and air space disease in a perihilar distribution is characteristic of acute pulmonary edema. The rapid clearing of the consolidation over several days without administration of antibiotics and the absence of a history of aspiration, fever or hemoptysis are also suggestive of acute pulmonary edema. Several factors suggest that the patient suffered from bilateral reexpansion pulmonary edema. The patient's young age; normal heart size; absence of a history of cardiac disease or cardiac risk factors; absence of electrocardiographic changes of ischemia, infarction or arrhythmia argue against cardiac dysfunction. There was no history, physical findings or radiographic evidence to suggest volume overload, drug abuse or toxic gas exposures. Unfortunately, the data that would have been useful in further excluding cardiac causes of edema, ie, pulmonary capillary wedge pressure, cardiac output and serial cardiac enzyme measurements, were not obtained because they were not considered clinically warranted.

For these reasons and the temporal relationship with pleurocentesis, we conclude that the bilateral pulmonary edema resulted from unilateral pleurocentesis. We were unable to find any report of bilateral or contralateral pulmonary edema following pleurocentesis in this century. There have been three reported cases of contralateral pulmonary edema following reexpansion of pneumothorax. 18 Pneumothorax occurred in one patient following closed chest massage after cardiac arrest at home. Pneumothorax occurred in the second patient with myeloma and renal failure during placement of a central venous catheter for resuscitation of respiratory arrest due to iatrogenic volume overload. There is an additional reported case of alternating contralateral and ipsilateral pulmonary edema following treatment of a pneumothorax due to central venous catheter placement in a patient with an acute anerterioseptal infarction and cardiopulmonary arrest. 19 The complex physiologic setting of these three cases suggests numerous etiologies for the radiographic observations. At the turn of the century, bilateral reexpansion pulmonary edema was reported following pleurocentesis using the Potain apparatus that employed -760 mm Hg of intrathoracic suction. Bilateral pulmonary edema following removal of unilateral pneumothorax or pleural effusion has been reported experimentally. 20

Three questions are raised by this case: (1) Did the diffuse bilateral opacities have the same etiology? (2) Did the opacities represent pulmonary edema? (3) If edema was present, did it result from reexpansion of the lung following pleurocentesis? Several hypotheses have been proposed to explain unilateral pulmonary edema following ipsilateral evacuation of pleural effusion or pneumothorax. Clinical and experimental data suggest altered capillary permeability, 7,9,13,15,18 decreased surfactant production, 6,8,9 or hypoxic injury. 5,5,6,20,81

Opacification of the contralateral lung could result from spillage of edema fluid from the reexpanded ipsilateral lung; however, this should produce radiographic changes in the contralateral lung of air space disease without any interstitial component. Various tissue factors could conceivably be released from the ipsilateral lung resulting in both local and systemic alteration of capillary permeability. 80 Other variables affecting pulmonary fluid exchange include pulmonary

**Figure 3.** Chest radiograph immediately after placement of the chest tube (11 h after pleurocentesis) demonstrates progressive opacification of both lungs with thickening of the fissures on the right, right air bronchograms and a right pleural effusion. The left hydropneumothorax decreased in volume.

hours the patient became progressively more dyspneic. Arterial blood gas value analysis on room air was as follows: \( P_{aO_2} \), 41 mm Hg; \( P_{aCO_2} \), 35 mm Hg; and pH, 7.46. A radiograph 4½ h after pleurocentesis showed increased opacification of the left lung and a new, perihilar opacification of the right lung consistent with interstitial edema (Fig 2). The left hydropneumothorax was unchanged. Jugular venous pressure continued to be normal, the electrocardiogram showed sinus tachycardia without other abnormality, and cardiac auscultation was unchanged.

One hundred percent oxygen was administered by face mask and a chest tube was inserted into left hemithorax with 20 cm H\( _2 \)O suction. Immediately after placement of the chest tube (11 h after pleurocentesis) a radiograph demonstrated further increase in the opacification of both lungs (Fig 3). There was new thickening of the fissures on the right, new right air bronchograms and a new right pleural effusion. The left hydropneumothorax decreased in volume. Twenty-two and one half hours after pleurocentesis, a chest radiograph showed a decrease in right lung consolidation. There was no change in left hydropneumothorax or left lung opacification. There was gradual spontaneous clinical and radiographic improvement over the next seven days.

There was never radiographic evidence of cardiac enlargement, azygous vein distension or other systemic venous distension to suggest cardiac failure, volume overload or tamponade. The patient subsequently underwent sclerotherapy for the persistent left hydro pneumothorax.

Over the next three months, the left pleural effusion reaccumulated and the right pleural space remained free of fluid. At no time were tumor cells detected in the pleural fluid. The patient began adriamycin therapy for the retroperitoneal neoplasm without response and the patient died without postmortem examination three months after admisison.

**Discussion**

Several factors indicate that pulmonary edema was the cause of the acute bilateral consolidations in this patient after rapid drainage of a massive unilateral pleural effusion.
capillary and interstitial hydrostatic pressures, oncotic pressures of plasma and interstitium and lymphatic flow.

We hypothesize that the mechanisms leading to unilateral re-expansion pulmonary edema can involve the opposite lung when there is significant contralateral lung compression. The marked right mediastinal displacement and subsegmental atelectasis evident in this patient indicate that compression of the contralateral lung was present. Removal of the large volume of fluid in less than 30 min resulted in acute re-expansion of both lungs. Thus, the potential mechanisms that alter capillary permeability were present bilaterally.

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Regression of the Left Main Trunk Lesion by Steroid Administration in Takayasu’s Aortitis*
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A 62-year-old man with unstable angina due to severe narrowing of the left main trunk (LMT) was examined. Emergency bypass surgery was performed with an internal mammary artery graft, instead of a saphenous vein graft, because of the thickened, edematous ascending aorta. Postoperative coronary angiography showed the lesion of the LMT markedly regressing. Presumably, this stenotic lesion of the LMT was caused by active aortitis and was partially reversible by steroid administration both during and after surgery. Steroid therapy can be added to the list of treatments for cases of LMT disease associated with Takayasu’s aortitis, if signs of active inflammation are present.

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LMT = left main trunk; CRP = C-reactive protein; LAD = left anterior descending artery; CX = circumflex artery; IMA = internal mammary artery

A severe LMT lesion is an absolute surgical indication for aortocoronary bypass. In the present case, severe stenosis of the LMT due to Takayasu’s aortitis regressed considerably after steroid administration.

CASE REPORT

A 62-year-old man had noticed an oppressive sensation in his chest upon exertion since March 1989. On May 23, 1989, the sensation had lasted for ten hours, and he was transferred to this hospital. The peak creatine phosphokinase level was 796 IU, but no abnormal Q waves developed on electrocardiography. After admission, he remained free from angina pectoris with nitrates, calcium antagonists, and β-adrenergic blockers. The level of CRP was 5.5 mg/dl, and normocytic and normochromic anemia was present. Five days after admission, as the oppressive sensation in his chest became again associated with congestive heart failure and ST-segment depression in all chest leads, an emergency coronary angiography was performed after insertion of an intra-aortic balloon pump. A long segment of the LMT was found to be severely stenotic (Fig 1A). The right coronary artery gave poor collaterals to the LAD and the CX.

The patient’s condition could not be controlled completely, regardless of the therapy; emergency bypass surgery was therefore

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