Re-expansion pulmonary edema: evidence for increased capillary permeability

GEORGE B. BUCZKO, MD
RONALD F. GROSSMAN, MD, FRCP(C)
MELVYN GOLDBERG, MD, FRCS(C)

Re-expansion pulmonary edema is a well defined complication of procedures that involve rapid drainage of large pleural effusions or evacuation of large pneumothoraces. Not only is the pathogenesis of the condition unknown, but also the entity has yet to be characterized as either hydrostatic or due to increased capillary permeability, although this can be inferred from a comparison of the protein concentration in the pulmonary edema fluid and the serum. In patients with hydrostatic pulmonary edema the protein concentration in the edema fluid is less than 60% of that in simultaneously obtained serum, whereas in patients with increased capillary permeability the two concentrations are similar. While the protein concentration in the edema fluid has been measured in patients with re-expansion pulmonary edema, there has not, to our knowledge, been a report comparing the protein concentrations in the two fluids. We have made this comparison in a patient and have found that re-expansion pulmonary edema has features of the type of pulmonary edema that is due to increased capillary permeability.

Case report

The patient, a 59-year-old woman, came to hospital with a history of increasing shortness of breath for 2 weeks and a dry cough for 1 week. Three years earlier a simple left mastectomy for ductal carcinoma of the breast had been performed, and a year later she had undergone radiotherapy because of recurrence in a left supraclavicular node.

The patient was dyspneic. Her blood pressure was 130/90 mm Hg, pulse rate 100 beats/min and oral temperature 36.5°C. There was decreased movement of the left hemithorax, with dullness to percussion, decreased tactile fremitus and diminished air entry in this area. The remainder of the physical examination gave normal results.

A chest roentgenogram revealed a large left-sided pleural effusion (Fig. 1). A left-sided thoracentesis revealed a bloody exudate with a protein concentration of 46 g/l, and a closed-chest pleural biopsy showed metastatic adenocarcinoma.

The next day 3 litres of fluid was drained from the left pleural space and talc poudrage performed; 1 litre drained spontaneously and 2 litres drained following the application of 20 cm H2O of negative pressure. Within 5 minutes of the application of suction the patient became markedly dyspneic and coughed up large volumes of frothy, straw-coloured fluid. Within 30 minutes her blood pressure fell to 90/60 mm Hg and she required artificial ventilation for profound respiratory distress. There was no clinical evidence of congestive heart failure.

Arterial blood obtained while the patient was breathing room air showed a pH of 7.45, an oxygen tension of 45 mm Hg and a carbon dioxide tension of 30 mm Hg. An electrocardiogram was normal. A chest roentgenogram revealed a left-sided parenchymal infiltrate but no cardiomegaly and a normal right lung (Fig. 2).

Normal saline and two units of albumin were given intravenously, and 5 cm H2O of positive end-expiratory pressure was applied. Within 2½ hours the patient was symptomatically improved and the endotracheal tube was removed. Over the next 48 hours improvement continued, though she required supplemental oxygen to

FIG. 1—Massive left-sided pleural effusion.

maintain adequate arterial oxygenation.

The fluid collected by sputum-trap suction of the endotracheal effluent moments after the onset of symptoms had a protein concentration of 40 g/l, with an albumin level of 22 g/l. At that time the corresponding serum levels were 54 and 33 g/l. No bacteria were isolated from the endotracheal effluent.

Three days after a chest roentgenogram revealed only atelectasis at the base of the left lung (Fig. 3). Five days after talc poudrage the chest tubes were removed and the patient was sent home.

Discussion

The protein concentration in the edema fluid was 74% of that in a simultaneously drawn blood sample in our patient with re-expansion pulmonary edema. Fein and coworkers recently demonstrated that in all the patients in their study with noncardiogenic pulmonary edema the ratio of the protein levels in edema fluid and blood was equal to or exceeded 0.60, whereas in patients with cardiogenic pulmonary edema it was less than 0.56, with a mean of 0.46. The ratio in our patient was similar to that found in patients with adult respiratory distress syndrome of other causes.1-4,6

Although the clinical presentation of unilateral expansion pulmonary edema has been well described,7-9 its mechanism has not yet been elucidated. Possible explanations include loss of tissue surfactant,10 mechanical trauma to the membrane due to a shift in blood flow from the systemic to the pulmonary circulation11 and a high negative intrapleural pressure.12 This report demonstrates that an increase in the lung's capillary permeability to protein is at least part of the explanation; theories relying solely on changes in pressure gradients are too simplistic.

References


BOOKS

This list is an acknowledgement of books received. It does not preclude review at a later date.


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