tended to include pleural effusion and infiltration. Furthermore, amyloidosis should be added to the list of causes of pleural effusion.

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REFERENCES


To the Editor:

We thank Doctors Graham and Ahmad for their constructive comments. They wish to extend the classification for pulmonary amyloid disease to patients with pleural infiltration. We do not agree that pleural amyloid disease represents a unique presentation of amyloidosis. Although their patient had pleural effusion and uncommonly extensive pleural amyloid deposits, AL (immunoglobulin-related amyloid) was clearly systemic at the outset. The primary manifestation in their patient was cardiac disease. Angina due to cardiac amyloid disease is well recognized.13 Their patient had cardiac, pulmonary parenchymal, as well as pleural amyloid, deposits. The patient seen at Massachusetts General Hospital also had cardiac amyloidosis (AL) evidenced by a monoclonal IgM protein, an EKG showing atrial fibrillation and low voltage, and a chest x-ray film revealing pulmonary vascular congestion and cardiomegaly. Parenchymal (diffuse interstitial) as well as pleural amyloid disease was proven by biopsy. In neither instance, in view of associated congestive heart failure and biopsy-proven pulmonary parenchymal amyloid disease, can one conclude with certainty that the pleural effusions originated from the pleural amyloid deposits. In the Swedish case cited,4 a long-standing parenchymal lung mass was recognized with additional pleural involvement. In none of the cases was there isolated pleural involvement.

The first two cases would best fit the pattern of diffuse parenchymal (interstitial) amyloid disease which, as noted in our editorial,4 usually manifests symptomatic cardiac involvement as the major symptom. These patients survive longer than one year. The final case would fit the more favorable form of nodular parenchymal amyloid disease. Since pleural involvement was not shown to be the sole presentation, it should not be considered a separate basis for classification. In all cases, the pleura was extensively but incidentally involved. The small fraction of cases with extensive deposition does not justify recognition of pleural amyloid as a distinct clinicopathologic entity.

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Air Leak in Chest Tubes

To the Editor:

The article by Bishop et al (Chest 1987; 91:400-02) points out that the air suctioned through the chest tube in patients with ARDS and bronchopleural fistulas contain CO2 in levels similar to that present in expired air and therefore is not wasted ventilation. This, however, is not unique to patients with ARDS and bronchopleural fistulas but occurs in most situations in which there is persistent leakage of air through a chest tube. In 1978, I measured the Pco2 in the chest tube of 11 patients with persistent air leaks and compared it with the end expiratory Pco2.

One patient had severe bullous emphysema and developed a pneumothorax while on a respirator. A chest tube was inserted and out of each tidal volume of 750 ml, on average 400 ml was suctioned through the chest tube for several days. Despite the extensive air leak through the chest tube, he maintained Pco2 at the same level as before the pneumothorax. The patient's end expiratory Pco2 was identical to that present in the air that was suctioned through the chest tube. Another patient who had undergone a decortication for pleural abscess did not have difficulty maintaining Pco2 at 30 mm Hg on a respirator despite the fact that 50 percent of the delivered minute ventilation was suctioned through the chest tube. The Pco2 of the chest tube air was 17 mm Hg.

The other nine patients included one patient with a pneumothorax following a transbronchial lung biopsy (the ratio of chest tube Pco2 to end expiratory Pco2 was 0.8,) four patients with persistent air leaks following resection of lung carcinomas (ratio range 0.5 to 1.2) two patients with sarcoidosis and resections of aspergillosis (ratio 0.5 to 0.9,) one patient with cystic fibrosis who developed a pneumothorax (ratio 1.0) and one patient with chronic tuberculous bronchopleural fistula (ratio 1.0).

In all these patients, gas excreted in the chest tube participated in gas exchange and, in fact, these patients could be considered to have an alternative system of ventilation, ie a flow-through system of ventilation in addition to the tidal system that nature devised. The point to bear in mind is that, except in the unusual patient with necrotizing pneumonia and a large bronchopleural fistula, persistent air leak via a chest tube is usually well tolerated, even when the air leak constitutes a significant fraction of the patient's minute ventilation.

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Hypersensitivity Pneumonitis in a Hairdresser

To the Editor:

We read with interest the paper by Weiss and Baur on hypersen-
Communications

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ARDS following Equine ATG Therapy

To the Editor:

We wish to draw attention to the recent case report by Dean et al (Chest 1987; 91:619-20) in which they described the onset of adult respiratory distress syndrome (ARDS) within hours of intravenous equine antithymocyte globulin (ALG) for rejection prophylaxis in a renal transplant recipient. We wish to confirm and extend their observation by reporting the development of ARDS in a cardiac transplant recipient receiving prophylactic equine antithymocyte globulin (HATG).

A 51-year-old man underwent uncomplicated orthotopic cardiac transplantation for dilated cardiomyopathy. Immediately prior to transplant, the patient received prophylaxis with 500 mg HATG, 8 mg/kg cyclosporine (CSA), 2 mg/kg azathioprine and 1 mg methyprednisolone. Postoperatively, he received daily HATG, CSA, azathioprine, and prednisone therapy. His immediate postoperative course was complicated by an increased A-a gradient necessitating 36 hours of ventilatory support. Chest x-ray examination demonstrated increased interstitial markings. He was extubated on the second postoperative day but continued to require supplemental O2 to maintain an arterial saturation greater than 90 percent. On the evening of the second postoperative day, the patient developed a gradual onset of increasing hypoxia and respiratory distress necessitating re-intubation on the morning of the third postoperative day. Chest x-ray films were now consistent with pulmonary edema. In the belief that the patient had volume overload pulmonary edema, the patient was treated aggressively with diuretic therapy. After a 5 L diuresis, the patient's pulmonary edema worsened and a pulmonary arterial line was inserted which revealed pulmonary artery pressure of 30/15 and pulmonary capillary wedge pressure of 12 to 14 mm Hg. Cardiac output was 5.5 L/min. A 2-D echocardiogram demonstrated normal left ventricular chamber size and vigorous contractile function. Thus, noncardiogenic pulmonary edema was suspected secondary to HATG and therapy was discontinued after four doses. Over the next ten days, a gradual resolution of pulmonary edema was observed.

As with the case report by Dean et al, it is impossible for us to definitely prove that pulmonary edema was secondary to HATG. However, several factors suggest this as the causal agent. Noncardiogenic pulmonary edema is suggested by the near-normal pulmonary capillary wedge pressure, worsening pulmonary edema despite a 5 L diuresis, and slow resolution (ten days). Since all medications other than HATG were continued, it is unlikely that these medications produced the ARDS. Rather, acute lung injury most likely resulted from the administration of cytotoxic antibodies (HATG). It is noteworthy that pulmonary edema has also been observed following OKT3 antibody. Although pulmonary edema in these situations has been attributed to volume overload, we would not be surprised if pulmonary edema in some of these cases was also secondary to direct lung injury. Perhaps clinicians should be aware of the potential for lung injury following the use of cytotoxic antibodies in general.

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REFERENCE