component(s) of the "urea formaldehyde resin system," rather than "urea formaldehyde" per se, might be implicated in the pathogenesis.

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References

Death from ARDS and Cardiovascular Collapse following Lidocaine Administration

To the Editor:

We read with interest the selected report of ARDS following administration of lidocaine by Howard and associates (Chest 1982; 81:644-45). A similar experience occurred at our institution and merits description.

A 19-year-old woman had fiberoptic bronchoscopy performed to evaluate new, bilateral interstitial infiltrates. A diagnosis of histiocytosis X was made three years earlier when a pathologic, long-bone fracture was noted. She had received cyclophosphamide and methotrexate in the past for this condition. At the time of bronchoscopy, she was receiving no chronic medications. The patient was pre-medicated with 1 mg of hydromorphone and 0.5 mg of atropine intramuscularly. Less than 30 ml of 1% lidocaine solution was employed for topical anesthesia. Bronchoalveolar lavage of the right middle lobe and four transbronchial biopsies of the right middle and lower lobe were performed without incident. The patient tolerated the procedure well, and postbronchoscopy, the chest film was unchanged.

Approximately 20 minutes after returning to her room, the patient was noted to develop hypotension and tachycardia and she became obtunded. An intravenous line was placed and saline solution and naxalone were administered without clear improvement. A respiratory arrest was observed, and she was promptly intubated. The vocal cords were widely patent and placement of an endotracheal tube yielded large amount of foamy pulmonary edema. She was placed on 100% oxygen with 5 cm PEEP and transferred to the Respiratory Intensive Care Unit. Corticosteroids, antithrombin, and a vasopressor were started.

Arterial blood gases on 100% oxygen with 5 cm PEEP revealed a PaO₂ of 58 mm Hg. A chest roentgenogram demonstrated extensive bilateral alveolar infiltrates; the frothy pulmonary edema fluid continued to be suctioned out of the endotracheal tube. Hypotension and refractory bradycardia occurred. The patient subsequently died five hours after bronchoscopy. Autopsy revealed pathology consistent with the adult respiratory distress syndrome and changes consistent with histiocytosis.

We feel our patient developed an anaphylactic, or delayed anaphylactoid, reaction to lidocaine with cardiovascular collapse and the adult respiratory distress syndrome. Although this case was similar to that of Howard and associates, the pulmonary deterioration in our patient was refractory to all forms of therapy. Since lidocaine is so frequently used in bronchoscopic procedures, this reaction should be looked for and an extensive history for lidocaine allergy should be sought before it is administered.

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Endobronchial Mucormycosis

To the Editor:

The recent case report of Schwartz and co-workers (Chest 1981; 81:653-54) of a young diabetic presenting with isolated tracheal mucormycosis and acute upper airway obstruction is noteworthy. Particularly gratifying is the successful combination of amphotericin B therapy with surgical resection, followed by primary reanastomosis. We have encountered three similar patients in our institution who presented with acute obstruction of major bronchi due to endobronchial mucormycosis. The patients are similar to those reported by Schwartz et al in that the disease was well-localized to major bronchi without distal invasion and combination therapy may have been appropriate.

Two adult patients with underlying diabetes mellitus presented with radiographic signs of central obstructing lesions. Bronchoscopy in both revealed granulation tissue and grey-white mucoid material blocking main-stem bronchi, with characteristic hyphae present on brushings. Both patients died of sudden massive hemoptysis while receiving amphotericin B, one after refusing pneumonectomy. The third patient, a five-month old child with recurrent urinary tract infection, presented with rapidly developing hyperinfarction of the left lung due to a broncho-stenotic lesion in the left main-stem bronchus. Mucor was found in the pneumonectomy specimen. The child was treated with amphotericin B and has done well for three years.

Examination of the pathologic specimens from these three cases revealed isolated mucormycosis in the central bronchi, locally invading through the wall into adjacent pulmonary arteries and lymph nodes. Mucor was identified only in the local sites.

A small number of additional patients with prominent disease in lobar or main-stem bronchi were identified in the literature. These cases, plus Schwartz's, may represent a specific type of pulmonary mucormycosis. The clinical features of this group include underlying diabetes mellitus or chronic renal failure, symptoms and signs suggesting an obstructing lesion in the major airway, serious local complications attributable to this lesion (distal atelectasis, fistula to the adjoining arteries), with confinement of the pathology to one well-localized area. The illness can be insidious and the diagnosis is made at bronchoscopy. The treatment appears to be combined surgical resection plus amphotericin B.

In contrast, three patients with mucormycosis and acute leukemia presented with a fulminating disease diffusely involving multiple lobes, but difficult to diagnose because the spumten was negative, as was the bronchoscopy. Indeed, open lung biopsy or transbronchial biopsy was needed to establish diagnosis. These patients died, and postmortem examination revealed widespread angiocentric disease.

Although the possibility that endotracheal/endobronchial mucormycosis is a distinct clinical entity in the spectrum of lower respiratory fungal disease is still conjectural, we do want to alert physicians encountering lesions in the central airways of diabetics to consider mucor. Furthermore, consideration must be given to the possibility of early resection along with therapy with amphotericin B in those patients with mucor in the central airways.

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References