inhibition. In addition, the effects of this drug may not be uniform in patients with cirrhosis of the liver, and cycloxygenase inhibition may have greater effects on pulmonary hemodynamics and gas exchange in patients with hepatogenic pulmonary angiodysplasia than in their counterparts. Although the effects of PGF\(_2\alpha\) and indomethacin on the pulmonary circulation were thought to be significant in this case, the hypoxemia remained. Thus, the predominance of vasodilatative PCs may not be the only etiologic factor linked to hepatogenic pulmonary angiodysplasia.

In summary, hepatogenic pulmonary angiodysplasia seems to be a state of reversible intrapulmonary vascular dilatation, and the predominance of vasodilatative PGs or other eicosanoids (or both) may contribute to the dilatation of intrapulmonary vascular systems.

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REFERENCES


Fatal Pulmonary Aspergillosis
Presenting as Acute Eosinophilic Pneumonia in a Previously Healthy Child*

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A previously healthy boy presented with cough and diffuse pulmonary interstitial infiltrates. Acute eosinophilic pneumonia was diagnosed by bronchoalveolar lavage in the absence of a demonstrable infectious etiologic agent. Corticosteroid therapy resulted in immediate improvement but was followed by respiratory distress and death from invasive aspergillosis and Pseudomonas cepacia sepsis.

(CHEST 1991; 100:875-77)

Eosinophilic pneumonia and pulmonary aspergillosis each make up a clinical spectrum that may overlap and be difficult to differentiate. Corticosteroids are the mainstay of treatment in noninvasive disease. We describe a fatal case of invasive pulmonary aspergillosis presenting as an eosinophilic pneumonia treated with corticosteroids.

CASE REPORT

An 11-year-old boy was seen by his physician because of dyspnea, fever, and cough. A chest roentgenogram showed diffuse infiltrates. He was hospitalized and treated with intravenous erythromycin, but was transferred to Children's Hospital of Pittsburgh because of progressive dyspnea.

There was no history of wheezing or asthma in the patient or family members. The patient had played with his two siblings in a compost pile 8 h before his symptoms developed.

He was a white boy in respiratory distress with suprasternal and intercostal retractions. The temperature was 38.5°C; the respiratory rate, 60/min; the heart rate, 129/min; and the blood pressure, 110/56 mm Hg. On auscultation of the chest, there were no adventitious sounds.

The white blood cell count was 15,300/cu mm with 87 percent neutrophils, 3 percent band forms, 2 percent lymphocytes, 1 percent monocytes, and 8 percent eosinophils. Arterial blood gas values with the patient receiving 3 L O\(_2\)/min by nasal cannula showed a pH of 7.46, P\(_{CO_2}\) of 17 mm Hg, and a P\(_O_2\) of 111 mm Hg. The admission chest roentgenogram showed diffuse pulmonary interstitial infiltrates (Fig 1). The IgG, IgM, IgA, and IgE levels were normal. The C3, C4, and CH100 (total complement) were normal.

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\(C=p\) complement; Ig=immunoglobulin; NK=natural killer; A=aspergillus; BAL=bronchoalveolar lavage; NBT=nitroblue tetrazolium; ABPA=allergic bronchopulmonary aspergillosis
Multiple clinical syndromes are associated with pulmonary aspergillosis. Immunocompetent hosts are affected by hypersensitivity lung diseases including allergic broncho-pulmonary aspergillosis and hypersensitivity pneumonitis; the features of these entities have been reviewed. In both conditions, pulmonary eosinophilia may be present, and corticosteroids are effective treatment. Invasive aspergillosis typically occurs in immunocompromised patients and is frequently fatal. We have found only one report of invasive disease in immunocompetent children, although several adults have been affected. Exposure to a large inoculum, as occurred in our patient, has been suggested as a cause of invasive disease in normal hosts.

The absence of wheezing and a history of asthma made a diagnosis of ABPA unlikely in our patient. His presentation was more consistent with a form of hypersensitivity pneumonitis, but the absence of cutaneous reactivity and initially, of precipitating antibodies, is atypical. Given the lack of evidence of active infection, eosinophilic pneumonia is a diagnosis of exclusion.

BAL may aid in the diagnosis of eosinophilic pneumonia and has been advocated as an alternative to lung biopsy. This approach is supported by Allen et al. in their description of four patients with acute eosinophilic pneumonia who were successfully treated with corticosteroids. In the light of the present case, however, we would recommend open lung biopsy in addition to BAL in all cases of acute eosinophilic pneumonia.

Pseudomonas cepacia is an unusual cause of sepsis in pediatric patients, but may be accounted for on the basis of the immunosuppressive effects of corticosteroids. Corticosteroids may increase the incidence of pulmonary aspergillosis in laboratory animals, but the role of antibacterials in predisposing to invasive fungal disease is unclear.

Although local invasion in the presence of a hypersensitivity response has been described, it is impossible to determine whether our patient's invasive pulmonary aspergillosis resulted from the inhalation of a large amount of Aspergillus spores followed by corticosteroid therapy, or...
whether therapy only aided the advancement of a primary invasive process. Although corticosteroids are helpful in eosinophilic lung diseases, immunosuppression may occur and allow for progression of illness, with potentially fatal consequences.

REFERENCES
8 Zimmermann RA, Miller WT. Pulmonary aspergillosis. AJR 1970; 109:505-15

Prolonged Reversible Quadriplegia in Mechanically Ventilated Patients Who Received Long-term Infusions of Vecuronium*

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The use of neuromuscular blocking agents, particularly pancuronium, in patients receiving mechanical ventilation has been reported to cause prolonged paralysis and atrophy. We describe two mechanically ventilated patients with asthma who developed prolonged muscular weakness and atrophy after receiving the shorter-acting agent vecuronium. These cases illustrate the potential of any neuromuscular blocking agent to cause these complications, especially in patients who are immobile, have decreased renal or liver function, or receive concomitant myotoxic agents.

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Neuromuscular blocking agents, such as pancuronium bromide, are used frequently to obtain total muscle relaxation in patients who are being mechanically ventilated. Long-term use of pancuronium has been reported to produce neuromuscular complications such as prolonged paralysis and atrophy. Two of these reports suggested that vecuronium, with a shorter duration of action and shorter recovery index than pancuronium, and which is excreted predominantly by the liver, would be less likely to result in these complications. We report two cases of disuse atrophy in patients with status asthmaticus who received prolonged infusions of vecuronium.

CASE REPORTS

Case 1

A 20-year-old female patient who presented in status asthmaticus required intubation and mechanical ventilation. She was treated with metaproterenol, aminophylline, and methylprednisolone. A lobar pneumonia was treated empirically with erythromycin and tobramycin. Eight days after hospital admission sputum stain was positive for Legionella, and the tobramycin therapy was discontinued.

Because of high peak ventilatory pressure and agitation, a vecuronium drip at 4 to 6 mg/h was started on the day of hospital admission, with fentanyl added for sedation. Severe refractory bronchospasm was treated unsuccessfully with intravenous isoproterenol and halothane anesthesia. Bronchoalveolar lavage was subsequently performed with removal of thick mucus plugs from all airways, resulting in reduction of the peak airway pressure. Eight days after hospital admission the vecuronium therapy was discontinued, and 3 days later the patient was extubated. Tobramycin and vancomycin were given from day 16 to day 23 for suspected endocarditis.

Immediately after extubation, the patient was noted to be confused and to have diffuse muscle weakness: grade 1 to 2/5 proximally, and grade 2 to 3/5 distally. She had bilateral foot and wrist drop. Cranial nerves and sensation were normal. The deep tendon reflexes were 1+ bilaterally. The creatinine kinase concentration was 778 U/L, and the erythrocyte sedimentation rate was 93 mm/h. Computed head tomography and a lumbar puncture were normal. Electromyelography (EMG) and nerve conduction studies were compatible with an inflammatory myopathy or an acute denervation disease. A muscle biopsy specimen showed areas devoid of any myofibrillar components and vacuoles filled with ground glass material probably representing glycogen.

Over the next 3 weeks, the patient's muscle strength improved to 3 to 4/5, although her deep tendon reflexes remained 1+ in all extremities. One month after the vecuronium therapy was discontinued, the patient was ambulating with a walker, and after 6 months the patient had regained her normal neuromuscular function.

Case 2

A 62-year-old woman with an acute exacerbation of asthma required intubation and mechanical ventilation for impending