Unilateral Atelectasis in Asthma*  

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Complete atelectasis of the right lung occurred in a 19-year-old man, with a protracted history of asthma. In addition to hydration, antimicrobials, intermittent positive pressure breathing, chest percussion, and bronchodilators, the patient had bronchoscopy on four occasions during the hospitalization, with progressive improvement. The early and repeated use of bronchoscopy, in addition to appropriate medical treatment, facilitated the clinical course of massive atelectasis occurring with asthma and was largely responsible for the reversibility of the lesion.

Minimal atelectasis as a complication of asthma is relatively common. Most cases of atelectasis reported in the English literature are segmental or lobular, with a tendency for the middle lobe to be more frequently involved. Leeks and co-workers reported a 7.4 percent incidence of segmental atelectasis in 530 hospitalized asthmatic children. Massive pulmonary atelectasis, described as involvement of more than one lobe, is a rare complication of asthma. Treatment for atelectasis is primarily administration of antimicrobials, relief of bronchospasm, and supportive care. The purpose of this communication is to describe a case of complete atelectasis of the right lung associated with asthma and to suggest an approach to treatment.

CASE REPORT

A 19-year-old man was hospitalized with a long history of asthma. Between ages one and seven years, the asthma was severe, and he received a series of desensitization injections at age six. The asthma remitted at age seven and recurred when he was 13. The asthma, present all year, was usually worse in the spring and was exacerbated by upper respiratory infections. Multiple allergies included dust, trees, pollen, animal dandruff, fish, peas and beans. Beginning in October, 1967, desensitization injections were given every two weeks, except weekly during August and September, and were continued until the present illness.

Events leading to the present hospitalization began in September, 1971, when the patient noted progressive dyspnea, wheezing and cough that did not respond to the ephedrine-phenobarbital-theophylline combination he had been using for three or four years. His condition improved with antibiotic therapy, but in early October, 1971, he was again treated for cough, dyspnea and wheezing, with some improvement. In late October, with recurrent dyspnea, cough and fever, a sharp substernal chest pain developed. Roentgenogram (Fig 1) demonstrated right lower lobe atelectasis, and he was hospitalized elsewhere. Bronchoscopy was performed and copious amounts of secretions were aspirated, but the lung remained collapsed. The aspirate was negative for malignant cells, and cultures were negative for acid-fast bacteria and other pathogens. The leukocyte count was 20,000 per cu mm, with 16 percent eosinophils. Results of skin tests for histoplasmosis and tuberculosis were negative. Treatment included intermittent positive pressure, postural drainage, antibiotics, and an ephedrine-phenobarbital-
Theophylline preparation. He was transferred to Indiana University Medical Center Nov. 17, 1971, with the complaint of "collapsed lung."

Examination on admission revealed a young man in no acute distress. The blood pressure was 114/70 mm Hg, heart rate 124 beats per minute, respiratory rate 16 per minute, and temperature 98.6° (37°C). Pertinent clinical findings included decreased breath sounds and dullness over the right posterior hemithorax. The left lung was slightly hyperresonant, but otherwise clear. No cardiac gallop or enlargement was detected, and the remainder of the physical examination was not remarkable. An admission chest roentgenogram (Fig 2) demonstrated collapse of the right lung, shift of mediastinal structures and elevation of the right hemidiaphragm. Initial arterial blood gases breathing room air demonstrated an oxygen saturation of 92 percent, oxygen tension of 85 mm Hg, carbon dioxide tension of 35 mm Hg, and pH of 7.46. Other laboratory studies revealed: hemoglobin, 16 gm percent and leukocytes 16,000 per cu mm, with 27 percent eosinophils.

Intermittent positive pressure, chest percussion and postural drainage were initiated. Aminophylline therapy, one gram in 4 liters of fluid daily, was started and bronchoscopy was performed November 19, with aspiration of mucous plugs from the right lower lobe bronchi. Bronchoscopy was terminated due to sudden and severe bronchospasm that responded to treatment with epinephrine and aminophylline. A postbronchoscopy roentgenogram demonstrated no improvement. Bronchoscopy was performed again November 24. The right main bronchus was partially occluded by mucopus, and mucous plugs were aspirated from the right upper lobe bronchi. Washings from both bronchoscopies demonstrated no malignant cells, and culture findings for fungi and bacteria were negative. A postbronchoscopy roentgenogram revealed some clearing and aeration of the right upper lung, with return of the heart and mediastinal structures toward a normal position. Gram stain of the sputum revealed a few positive diplococci, identified as usual throat flora, and the patient was started on erythromycin therapy. Since the right lower lobe remained collapsed (Fig 3), a third bronchoscopy was performed November 30, with aspiration of approximately 5 ml of mucoid material from the right lower lobe bronchus. A roentgenogram then demonstrated complete expansion of the right lower and upper lobes, with continuing atelectasis of the middle lobe. Intravenously administered hydrocortisone sodium succinate was begun to reduce bronchial edema and congestion. Because of persistent right middle lobe atelectasis (Fig 4), bronchoscopy was performed again December 2, with aspiration of secretions, and the postbronchoscopy roentgenogram showed improvement. Subsequent roentgenograms revealed gradual clearing of the middle lobe. The patient was discharged December 17, with a small residual density in the right middle lobe. One day before discharge, repeated arterial blood gas determinations demonstrated oxygen saturation of 97 percent, oxygen tension of 91 mm Hg, carbon dioxide tension of 38 mm Hg, pH of 7.47, and a lung scan demonstrated perfusion to all lobes of the right lung, with small defects in the right upper lobe, posterior right lower lobe and left upper lobe. Pulmonary function tests were also performed a day before discharge. Residual volume, functional residual capacity, vital capacity, and total lung capacity were normal. Maximum voluntary ventilation (MVV) was 151 liters per minute, with forced expiratory volume (FEV1)/forced vital capacity, (FVC) 83 percent, FEV1/FVC 92 percent, and FEV1/FVC 97 percent. Peak expiratory flow was 522 liters per minute and airway resistance was normal at 1.4 cm H2O per liter per second. Therapy was continued with orally administered bronchodilators and decreasing amounts of prednisone. A subsequent chest roentgenogram January 4, 1972, was normal.

**Comment**

The etiology of atelectasis in asthma has been attributed to inspissated mucoid or purulent plugs, bron-
chospasm and edema of the bronchial wall. The problem of plugs is compounded by the invariably present tachy-pnea, resulting in respiratory loss of fluids. Dehydration produces tenacious, inspissated plugs, which are difficult to remove via the cough mechanism. Messer et al., in a large autopsy series, reported a 97 percent incidence of abnormal bronchial contents (mucous, purulent exudate, or both) in 35 patients dying in status asthmaticus. Mucous plugs in the bronchi and focal areas of collapse were prominent features in another 20 cases of fatal status asthmaticus. In the present case, mucoid material was found at the four bronchoscopies.

The degree of atelectasis is of interest in the present case, since reports of complete atelectasis of a lung are rare in the literature. However, there is no reason to believe that the mechanism responsible for the massive collapse in the present case is qualitatively different from the more frequent lobar or segmental atelectasis.

Treatment in the present case was directed primarily to re-expansion of the right lung. In cases of segmental or lobar asthmatic atelectasis, conservative management with fluids, chest percussion, administration of bronchodilators and antimicrobials, and intermittent positive pressure breathing, is usually successful. When these measures fail, bronchoscopy with removal of obstructing secretions is indicated. In this patient, bronchoscopy was performed at an early point in the hospitalization because of extensive atelectasis and was repeated until satisfactory expansion of the lung was appreciated.

Careful and persistent suctioning and irrigation of the bronchi were performed during bronchoscopy, with improvement in the postbronchoscopy roentgenogram and clinical condition. As judged by pulmonary function studies, chest roentgenogram, lung scan, arterial blood gas determinations, and clinical status, this patient essentially made a complete recovery. Early and multiple bronchoscopies are indicated in patients with massive asthmatic atelectasis to re-expand the lung and prevent lung abscess and bronchiectasis. Multiple bronchoscopies in the present case preserved a functioning lung that otherwise might have remained collapsed, with resultant tissue destruction.

References

Wenckebach Periods in Posterior Inferior Division of Left Bundle

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A case of right bundle branch and left anterior superior division block-bifascicular block, developing into intermittent complete heart block-trifascicular block is described. Atrial stimulation produced Wenckebach periods, Mobitz type II conduction defect, with block below the His-bundle area and at the AV node and complete heart block. His-bundle electrography documented the Wenckebach's phenomenon as occurring below the His bundle in the only conducting fascicle, post inferior division of the left bundle. During atrial stimulation a phenomenon of concealed conduction into the infra-His conduction system was also noted. Implantation of a permanent cardiac pacemaker abolished symptoms of syncope.

Wenckebach's phenomenon was thought to be exclusively a property of AV functional tissue. Its occurrence at the AV node has been confirmed by the use of His bundle electrographic studies. Bundle branches, on the other hand, have not been commonly known to show Wenckebach's phenomenon. Scattered reports describe such an occurrence, and support it by clinical electrocardiograms only. Although experimentally produced, and confirmed by direct recordings by Scherf and Shookhoff in animals in 1925, Wenckebach's phenomenon has been directly demonstrated to occur in bundle branches only in recent rare reports.10,11 This communication will describe a patient in whom His bundle electrography confirmed that Wenckebach's phenomenon was occurring in the posterior inferior division of the left bundle, and further that the right bundle branch and left anterior superior division block progressed to complete heart block, necessitating the implantation of a permanent pacemaker.

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