Massive Asthmatic Atelectasis*

JOSEPH RAKOWER, M.D., F.C.C.P., PINKHAS WAYL, M.D., F.C.C.P.
and HERMAN HALBERSTADT, M.D., F.C.C.P.

Jerusalem, Israel

Atelectasis as a complication of bronchial asthma occurs more commonly than is diagnosed or suspected. It is usually of the lobular type and patchy in distribution. This type was first described by Huber and Koesler in 1920.1 Many authors have suggested that status asthmaticus and even death occurring during an attack of asthma are related to lobular atelectasis.2, 8

Massive atelectasis, on the other hand, is much less common than the lobular type. Since Clarke9 first described massive asthmatic atelectasis in 1930, there have been six reports published5, 6, 7, 8, 9, 10 describing a total of 15 cases. The purpose of this communication is to present two more cases and to review the clinical aspects of this uncommon complication of a common disease.

Case 1: An eight year old boy was admitted to the Hadassah Hospital on February 28, 1952 for an acute attack of asthma. His father and one sister were reported to suffer also from asthma. The patient's first attack occurred in 1948 during hospitalisation for acute nephritis. X-ray films of the chest at that time were normal. There followed five major attacks at varying intervals which necessitated hospitalization. These were usually marked by paroxysmal dyspnea, severe nonproductive cough, chest pain, wheezing and fever up to 40° C. Examination usually revealed cyanosis and tachycardia. Dullness, absence of breath sounds, and rhonchi were heard over various areas of the chest during the different admissions.

During the second admission for an asthmatic attack in Nov. 1949, x-ray film of the chest showed irregular opacities scattered chiefly over the right middle lung field. Following treatment with penicillin and streptomycin in this as well as in subsequent attacks dyspnea, fever, and x-ray findings cleared completely. During an attack in Jan. 1950 x-ray films showed a homogeneous opacity of the right lower lobe and scattered opacities of the right upper lobe (Fig. 1).

In December 1951 he was admitted for stomatitis and treated with penicillin. There was rapid improvement of the oral lesions, but five days later a severe attack occurred, and x-ray films showed extensive opacity of the right upper and middle lobes and tracheal deviation to the right (Figs. 2 and 3). While experiencing another attack in April 1952, x-ray films showed again a similar picture (Figs. 4 and 5).

In June, 1952 he experienced still another attack, x-ray film at this time revealed an extensive, homogeneous opacity of the entire left lung with marked mediastinal shift to the left, as well as appearance of a mediastinal hernia. (Fig. 6).

All these bouts of atelectasis were followed by complete clearing of clinical signs, symptoms and x-ray findings within four to 10 days following institution of therapy, usually penicillin. Between acute attacks the patient was fairly well except for symptoms of chronic bronchitis. Bronchoscopy and bronchography could not be performed because attempts at laryngeal anesthesia stimulated recurrence of asthmatic attacks.

Leucocytosis on admission varied from 11,800 to 20,400 and eosinophiles from 0 to 31 per cent of the total white count. The sedimentation rate was usually elevated up to 60/98 Westergren. No sputum could be obtained. Urine and feces were normal.

The Mantoux test was negative on three occasions but twice injections of tuberculin were followed by asthmatic attacks, without abnormal x-ray findings. Histamine injection was uneventful. The Middlebrook-Dubos test was positive in 1:128 dilution but 14 cultures of the gastric contents were negative for tubercle bacilli.

This case is remarkable for the recurrent episodes of atelectasis which was reserved on five different occasions in four years. The first time there occurred the lobular type, patchy in distribution, while in the other four times there was massive atelectasis. The highly positive Middlebrook-Dubos test and the allergic response to tuberculin with precipitation of asthmatic attacks were probably non-specific reactions.

*From the Department of Chest Diseases of Hadassah University Hospital. Jerusalem, Israel.
Figure 1 (Case 1): January, 1960. Atelectasis of right upper lobe. Figure 2 (Case 1): December, 1960. Atelectasis of right lower lobe. Figure 3 (Case 1): December, 1960. Lateral roentgenogram.
Case 2: A 15 year old girl had had asthmatic attacks which were generally preceded by rhino-pharyngitis from the age of three. Tonsillectomy was performed in 1947 at the age of nine. The tuberculin test was negative and in 1952 she received BCG. The asthma improved after the initiation of a diet consisting of vegetables and milk as well as the institution of psychotherapy. However in January 1953 she developed an unusually severe attack of asthma that lasted three days marked by cough, dyspnea and low grade fever.

Physical examination showed dullness over the right upper chest and musical rhonchi. Fluoroscopy revealed a sharply defined shadow involving the anterior segment of the right upper lobe. X-ray films (Fig. 7) showed extensive opacity of the right upper lobe with mediastinal and cardiac shift to the right. Treatment included expectorants and aminophylline. After six days, following expectoration of thick mucus, there was complete clearing of the signs of atelectasis on both, physical and x-ray examination.

Discussion

Bronchial obstruction in asthmatic attacks has been variously ascribed to bronchospasm, edema of the bronchial walls and to dyscrinia, i.e. excessive secretion of mucus of a peculiar character.

Bronchospasm is the oldest of these three theories with Laennec among its first proponents. However, it is particularly difficult to explain massive atelectasis on this basis because spastic closure of the larger bronchi is prevented by their cartilaginous rings. Most authors, on the basis of autopsy findings2,3 or bronchoscopy4, have concluded that the two factors of edema of the bronchial mucosa and increased mucus secretion are the main causes of bronchial obstruction occurring in asthma. On histological examination there is found an increase in mucus—producing goblet cells, with decrease in the number of normal columnar ciliated cells. Thus, secretion of mucus is increased and at the same time there is a decrease in ciliary action thereby promoting stasis of the secretions. Dehydration resulting from the loss of fluid during increased respiratory activity tends to promote drying of the mucus into inspissated tenacious plugs that obstruct the bronchi. Such plugs are sometimes seen on bronchoscopy, and their removal is followed by prompt, clinical improvement. This process of hypersecretion of mucus and its subsequent inspissation may be so marked as to lead to laminar impaction of the mucus in second order bronchi. The bronchi become dilated and the resulting picture may simulate neoplasm, abscess formation, or tuberculosis.12

Massive asthmatic atelectasis occurs more commonly in childhood than in adult life because the smaller calibre of the bronchi in the child predisposes to bronchial obstruction. Of the 15 cases previously reported in the literature, five were in adults and 10 in children. This type of atelectasis is almost always unilateral and in only one of the reported cases4 was it bilateral. Both sides are involved with equal frequency. In two cases atelectasis was segmental, in 10 the distribution was lobar, while in five an entire lung was involved. In five there was a second attack of atelectasis, while in one of the cases reported here there was a total of four attacks of massive atelectasis as well as one of the lobular type. In the other 12 cases there was only a single attack.

Massive asthmatic atelectasis is almost always accompanied by obstructive infection and fever, leucocytosis and an elevated sedimentation rate are usually found, streptococci and pneumococci occur in the sputum and
may be cultured from the mucus plugs. These organisms are usually regarded as secondary invaders. The classical roentgenological signs of atelectasis are usually present. The most helpful of these signs are those produced by shift of the mediastinum accompanied sometimes by mediastinal herniation. Elevation of the diaphragm on the affected side and contralateral compensatory emphysema are also frequently found.

On bronchoscopy obstructing plugs of tenacious amorphous material can be seen.

As is usual in allergic individuals, patients whose asthma leads to atelectasis show considerable variations in eosinophile counts. On the other hand, the presence of eosinophiles in the sputum, possibly reflecting their concentration in the lung itself, is a much more constant finding.

The fact that atelectasis is almost always accompanied by infection makes it necessary to rule out pneumonia in the differential diagnosis of massive asthmatic atelectasis. Asthmatic type of breathing with expiratory dyspnea, history of recurrent attacks of this type, eosinophilia in the blood or sputum, and X-ray findings help in differentiating between the two conditions. Fibrinous bronchitis may also cause massive atelectasis due to hyposection of a thick mucus. However in this condition a long history of previous asthmatic attacks is lacking. Furthermore, the bronchial casts appear as grayish root-like formations distinctly different from the mucus plugs or the amorphous mucus in the cases of massive atelectasis of the asthmatic type.

The transient irregular, mottled, migrating eosinophilic infiltrations may tend to resemble the findings in the lobular type of atelectasis, but these conditions however do not produce such severe clinical symptoms as does asthmatic atelectasis.

Massive asthmatic atelectasis usually clears on treatment. Improvement occurs within three to five days, and within one to six weeks there

FIGURE 7 (Case 2): Atelectasis of the anterior segment of right upper lobe.
is usually complete reexpansion of the lung. The only fatal case reported
was one with massive atelectasis of both lower lobes.4 In lobular asthmatic
atelectasis, on the other hand, there are many more fatal cases reported.2,3
Treatment is directed at removal of the mucus plugs obstructing the
bronchi, combating infection and relieving the attack.
Postural drainage, expectorants, trypsin and aerosol inhalation are help-
ful in relieving obstruction. Penicillin or aureomycin is used to combat
secondary infection due to pneumococci and staphylococci usually found in
this condition.
In the treatment of the asthmatic seizure per se, ACTH or cortisone are
the drugs of choice. Aminophylline and epinephrine may be used however,
and are particularly helpful in tiding the patient over until hormonal
treatment takes effect. It may be possible to prevent further attacks which
predispose to atelectasis by identification and removal of allergens.

SUMMARY
Massive atelectasis is a rare complication of bronchial asthma. It is
caused by obstruction of the bronchi by the secretion of large amounts of
thick tenacious mucus by an altered bronchial mucosa. The clinical aspects
of this condition are discussed and two cases are presented.

RESUMEN
La atelectasia masiva es una rara complicación del asmabronquial. Es
causada por obstrucción de los bronquios por grandes volúmenes de secre-
ción mucosa espesa adherente producida por una mucosa bronquial
alterada. Los aspectos clínicos de esta condición se discuten y se presentan
dos casos.

RESUME
L'atélectasie massive est une complication rare de l'asthme. Elle est
due à l'obstruction de la bronche, par une importante quantité de mucus
épais et solide provenant de la muqueuse bronchique altérée. Les auteurs
mettent en discussion les aspects cliniques de cet accident et en présentent
deux exemples.

REFERENCES
80:325, 1950.
4 Clarke, J. A.: "Pulmonary Atelectasis as a Complication of Bronchial Asthma,"
5 Tucker, G.: "Bronchoscopic Observations on Obstructive Pulmonary Atelectasis,"
7 Kahn, J. S.: Cited by Friedman, T. B. and Molony, C. J.
8 Friedman, T. B. and Molony, C. J.: "Role of Allergy in Atelectasis in Children,"
10 Rubin, E. H.: " travellers."
11 Bases, L. and Curtin, A., "Prevention of Death in Status Asthmaticus: Value of
13 Rakower, J.: "Atelectasis Massive au Cours de la Bronchite Pseudo-Membraneuse

Downloaded From: http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21263/ on 04/01/2017