

Unilateral pulmonary collapse in asthmatics

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Hopkirk, J. A. C., and Stark, J. E. (1978). *Thorax*, 33, 207–210. **Unilateral pulmonary collapse in asthmatics.** Five asthmatic patients developed collapse of one lung. Three of the patients were children and three of the five had repeated episodes of atelectasis. Episodes of atelectasis were usually associated with localised chest pain, which was not pleuritic in character, and with breathlessness, but without wheezing. They were not related to clinically apparent respiratory infections or to deterioration of the underlying asthma. The cause is obscure, but re-expansion seems to be hastened by oral corticosteroid therapy.

Atelectasis is a well-recognised complication of asthma. It is usually segmental or lobar and is said to be more frequent in children than in adults (Clarke, 1930; Hughes and Reisman, 1969; Crofton and Douglas, 1975). Lecks *et al.* (1966) reported atelectasis in 39 out of 530 children treated in hospital for asthma. The middle lobe is most frequently involved (Dees and Spock, 1966; Wittig and Chang, 1967). Massive atelectasis involving more than one lobe (Brashear *et al.*, 1973) is rare (Aronsohn and Pressman, 1958; Rakower *et al.*, 1955) and collapse of an entire lung has been reported very infrequently.

Case reports

CASE 1

This girl had mild asthma from infancy until the age of 9 years but was then free of symptoms for three years. At the age of 12 she suddenly developed a fever, cough, and left-sided chest pain with no preceding wheeze or upper respiratory symptoms. Although she was breathless, no wheeze was heard, and the signs were those of collapse of the left lung. The radiograph showed collapse of the left lung with a prominent air bronchogram. There was no excess of eosinophils in the peripheral blood, and haemolytic streptococci were grown from mucopurulent sputum. The radiograph became normal after two weeks of treatment with penicillin and physiotherapy, and she remained symptom free for three months.

She then developed wheeze and a cough, and a radiograph showed collapse of the lingula. Three days later there was also collapse of the left lower

lobe. Prick tests against common allergens including *Aspergillus fumigatus* were negative, a sweat test was normal, and serum precipitins were not detected against *A. fumigatus*. At bronchoscopy the only abnormality seen was thick 'mucopus' in the left main bronchus. The aspirate contained no excess of eosinophils, and no fungi or bacteria were isolated on culture. The radiograph became normal after 10 days' treatment with physiotherapy and inhaled bronchodilators. She remained free of symptoms, but a chest radiograph one month later unexpectedly showed collapse of the left lower lobe, although she had no respiratory symptoms at that time. It was decided to treat this episode with corticotrophin (40 units twice weekly) and the radiograph returned to normal in three days. The dose of corticotrophin was slowly reduced, but two months later, while still on a small dose of corticotrophin, she had another episode of cough and chest pain with further collapse of the left lower lobe. The dose of corticotrophin was increased and the radiograph again returned to normal within three days. In the three years since this episode she has regularly inhaled corticosteroids and has had no further symptoms and no further recognised episodes of atelectasis.

CASE 2

This girl, who had recurrent episodes of cough and wheeze from age 2 years, became wheezy at the age of 6, and a radiograph showed collapse of the middle lobe. There was no history of inhalation of a foreign body. Middle lobe collapse persisted in spite of physiotherapy, and bronchoscopy four months later showed thick 'mucopus' in the right

main bronchus. The secretions were aspirated, and a bronchogram showed normal filling of the middle lobe bronchus with no peripheral abnormality. The aspirate contained no excess of eosinophils and was sterile on culture. She was seen again at the age of 8 with severe asthma, which was poorly controlled by inhaled bronchodilators and cromoglycate and over the next two years required two courses of oral corticosteroids. Serial radiographs were normal during this period. At the age of 10 she suddenly developed fever, breathlessness, and a sharp right-sided pain in the chest, which was not aggravated by respiratory movements. She said that these symptoms were quite unlike those of her previous asthma. A radiograph showed collapse of the right lung. There was no blood eosinophilia, prick tests against five common allergens, including *A. fumigatus*, were negative, and Aspergillus precipitins were not detected in the blood. A sweat test was normal. She was treated with physiotherapy, bronchodilators, and antibiotics, and the lung re-expanded within two weeks. Treatment was continued with regular inhaled corticosteroids, and she has since been well for two years with normal spirometry and radiograph.

CASE 3

This man had been wheezy as a child but became symptom-free in early adult life. At the age of 39 his asthma recurred but improved on regular treatment with inhaled bronchodilators and sodium cromoglycate. While on this treatment he had only occasional slight breathlessness until he developed sudden onset of shortness of breath with fever and discomfort in the right chest. There had been no upper respiratory symptoms and he had had no wheezing or breathlessness for some months before this episode. A chest radiograph showed collapse of the right lung. There was no blood eosinophilia, prick tests produced 4 mm weals to house dust, house dust mite, and grass pollen, but no reaction to *A. fumigatus*, and Aspergillus precipitins were not detected in the blood. The sputum contained no eosinophils, and *Haemophilus influenzae* was isolated on culture. He improved slowly on treatment with oral ampicillin, inhaled bronchodilators, and physiotherapy. The chest radiograph was normal by the 14th day. He has remained well since while regularly inhaling corticosteroids and a bronchodilator.

CASE 4

After recurrent episodes of cough and wheeze since the age of 3 years, this boy started treatment with sodium cromoglycate at the age of 7 and re-

mained virtually free of symptoms until the age of 10. He then developed mild wheeze, persisting for about 10 days, followed by sudden sharp left chest pain, breathlessness, and fever. A radiograph showed collapse of the left lung with a proximal air bronchogram. There was no blood eosinophilia and only one out of eight prick tests against common allergens was positive. A skin test against *A. fumigatus* was negative and Aspergillus precipitins were not detected in the blood. At bronchoscopy thick 'mucopus' was aspirated from the left main bronchus. This contained no eosinophils or visible fungal elements and was sterile on culture. He was treated with physiotherapy and bronchodilators, and the left lung re-expanded within 10 days. He has remained well since on regular cromoglycate and bronchodilator and has had no recurrence of radiological abnormalities.

CASE 5

This young woman has had seven documented episodes of atelectasis. She has had asthma since childhood but there is no family history of atopic disease. At the age of 9 she developed left chest pain with collapse of the left lower lobe. It was noted in the records that her asthma had not worsened before that admission. She was treated with physiotherapy and antibiotics, and three weeks later the lobe had re-expanded. At the age of 19 her asthma deteriorated and she received regular oral corticosteroids at low dosage for two years. She then suddenly developed left chest pain and fever, and a radiograph showed collapse of the left lung with a proximal air bronchogram. This episode had not been preceded by deterioration of asthma or by upper respiratory symptoms. At bronchoscopy thick 'mucopus' was aspirated from the left main bronchus; it contained no excess of eosinophils and was sterile on culture. The dose of corticosteroids was increased and the lung re-expanded within a few days, after which corticosteroid therapy was stopped.

During the next two years she had three further episodes of collapse of the left lower lobe with no respiratory symptoms at other times. None of these episodes was associated with increased wheezing, and on no occasion did she have a blood eosinophilia. Bronchoscopy twice showed no abnormality other than secretions in the left lower lobe bronchus. On each occasion the lobe re-expanded during a course of oral corticosteroid therapy. A bronchogram and a cine-bronchogram during an asymptomatic period was normal. Prick tests were positive to 4 out of 18 common allergens but negative to *A. fumigatus*, and Aspergillus precipitins were not detected.

Since the last of these episodes she has regularly inhaled corticosteroids and has remained free of symptoms between further episodes of atelectasis. In the last year she has had episodes of collapse of both right and left lower lobes on separate occasions, and re-expansion has occurred within a few days of starting treatment with oral corticosteroids. On each occasion spirometry showed a reduced FEV₁ and vital capacity but normal FEV₁%.

During the most recent episode of left lower lobe collapse fiberoptic bronchoscopy was performed under local anaesthesia. In contrast to the findings when this and our other patients were bronchoscoped under general anaesthesia, fiberoptic bronchoscopy revealed a patent lower lobe bronchus with normal looking mucosa and patent segmental bronchi, which were free of secretions. As soon as suction was applied, however, large quantities of yellow secretions appeared in the peripheral bronchi. In all other respects this episode of lobar collapse behaved like the previous ones, and re-expansion occurred within a few days of starting oral corticosteroid therapy.

Discussion

The five patients described have between them suffered 14 separate episodes of massive atelectasis and each has collapsed an entire lung on one occasion. The clinical features of these episodes have been remarkably similar. All patients had been asthmatic in the past, but in none had the asthma been severe enough to require hospital admission or regular systemic corticosteroid therapy. Each episode of atelectasis, with the exception of one asymptomatic episode, presented with a short period of breathlessness, without wheezing, but with discomfort in the chest of a non-pleuritic character often described as 'pulling'. Several episodes were associated with fever and one patient developed rigors. Two patients have themselves recognised a recurrence of atelectasis by a return of identical symptoms and have been able to distinguish these symptoms from those of their previous asthma. There was no apparent association with preceding upper or lower respiratory tract infections, although virological studies were not carried out. Although excess secretions were seen in the bronchi of the affected lobe, none of the patients had noticed increase of cough or sputum before atelectasis was recognised. Spirometry, when recorded during episodes of atelectasis, usually showed a restrictive pattern.

The radiological appearances were typical of collapse of one or more lobes, and an air bronchogram was prominent within the area of collapse in

most cases. Recurrent episodes of collapse affected either the same or different lobes, and the same patients sometimes developed atelectasis on different sides on different occasions. Bronchograms carried out in two patients showed no residual abnormality after resolution of pulmonary shadowing, and a cine bronchogram in one patient who had had repeated episodes of collapse of the left lower lobe showed no abnormal collapse of the left lower lobe bronchus. In only one of our five patients was an eosinophilia present in peripheral blood at the time of massive atelectasis, and in no case was there an excess of eosinophils in stained smears of sputum or of bronchial aspirate. Rigid bronchoscopy under general anaesthesia always showed excessive secretions, often described as 'mucopurulent' in the bronchi of the affected lobe or lung. However, a potentially pathogenic bacterium was isolated from only one bronchial aspirate and no fungi were seen in or isolated from any specimens of sputum or aspirate. Allergic bronchopulmonary aspergillosis was considered and excluded in all cases. Cystic fibrosis was excluded in most cases by demonstration of normal concentrations of sodium and chloride in sweat, and in none of the others has a subsequent course suggested cystic fibrosis.

The pathogenesis of massive atelectasis in these patients remains obscure. Dunnill (1960) described plugging of airways with mucus in the lungs of patients who died of asthma, but this predominantly affected smaller airways. The lack of association of atelectasis with deterioration of asthma was notable in the episodes we observed. Felson (1973) postulates suction of mucus distally into small airways, a mechanism which could account for the presence of lobar atelectasis with air-filled bronchi visible on plain radiographs as an air bronchogram. The nature of the secretions and the reason for their accumulation in mild asthmatics, who were not producing sputum, remains unclear.

Although secretions were usually aspirated without difficulty at bronchoscopy, expansion of the affected lobe was slow thereafter, and it is our impression that expansion occurred as rapidly, if not more so, in episodes treated with corticosteroids. Three of our five patients had recurrent episodes of lobar atelectasis but, in the absence of severe asthma or evidence of permanent bronchial or pulmonary damage, it was not considered justifiable to prescribe continuous oral corticosteroids in an attempt to prevent subsequent attacks. It is our impression that several patients have had reduced frequency of episodes of atelectasis while regularly inhaling corticosteroids.

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