Clinical Problems

Asthma in the Elderly

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Summary

Fifteen patients who developed asthma after the age of 60 years are reported. Attention is drawn to apparent difficulties of diagnosis in this age group. A history of chronic bronchitis is common, and a change in symptoms, especially the abrupt onset of increased breathlessness, wheezing, and paroxysmal nocturnal dyspnoea, should arouse suspicion of the development of asthma. A past or family history of allergy is confirmatory evidence, as is the presence of blood or sputum eosinophilia. Retrosternal pain is not uncommon, and angina pectoris or left ventricular failure must be excluded. Chest radiographs showed changes consistent with old quiescent tuberculosis in five patients; careful follow-up is therefore essential as asthma in this age group often requires steroid therapy.

Introduction

Asthma often begins in childhood,[1–4] and young children who have frequent episodes of "wheezy bronchitis" may well be constitutionally asthmatic.[5] There appears, however, to be less general appreciation that bronchial asthma may occur for the first time in the elderly,[3] in whom there is real risk of a mistaken or partial diagnosis such as chronic bronchitis or "obstructive airways disease." We here present data on a group of adults who developed asthma after the age of 60, and discuss some of the problems common to them.

Present Study

During the past 18 months 15 adults whose asthmatic symptoms began after the age of 60 have been seen in the respiratory clinic at Manchester Royal Infirmary (Tables I and II).

Cough was an important symptom in 14 of the 15 patients; in most this produced sputum which was typically mucoid and clear though in some it was intermittently yellow. Six patients gave a history of chronic productive cough, consistent with a diagnosis of chronic bronchitis, for periods of up to 22 years before a change in symptoms suggested that new factors were operative. Dyspnoea was the dominant symptom in all patients, and was accompanied by tightness in the chest and usually but not always by frank wheezing. In all but one patient paroxysms of breathlessness occurred during the night. These characteristically woke them in the early hours of the morning with tightness in the chest, cough, and wheezing. One or two patients had more than one such episode through the night, each lasting ½-1 hour. Most thought that the sensation of tightness was not painful, though one patient (Case 13, below) described it as a retrosternal constriction, which we could not confidently distinguish from angina pectoris, and three (Cases 2, 4, and 15) had quite definite attacks of central chest pain.

A family history of allergic disease was obtained from three patients (Cases 1, 10, and 13) and a past history of symptoms consistent with allergic rhinitis was elicited from four (Cases 1, 3, 8, and 13). In some cases such aspects were reliably established only after detailed questioning because many years had elapsed since the relevant event, and the significance was not apparent to the patient. Only three patients smoked tobacco at the time they were referred to us. One (Case 4) smoked two cigarettes daily, and two (Cases 9 and 12) smoked about 30 g and 20 g of pipe tobacco a week respectively. Four patients had never smoked and the other eight had stopped 1-15 years previously.

The blood showed eosinophilia in about half of the patients, and sputum smears contained eosinophils ++ (possible gradings: 0, +, ++) in 12 patients; only one patient was unable to produce a suitable sample of sputum. Serum was tested by Dr. G. Taylor for the presence of precipitins to *Aspergillus fumigatus* and other common saprophytic fungi, and to *Haemophilus influenzae*. The former were all negative while precipitins to *H. influenzae* were positive in three patients (Cases 2, 4, and 11). Prick skin tests with a range of common allergens including house dust, the house dust mite, feathers, various pollens, and moulds were done routinely. Four patients (Cases 1, 10, 11, and 13) gave positive weal and flare reactions to several reagents, but not to *A. fumigatus*. All the other patients had negative responses.

Pulmonary function tests confirmed the presence of diffuse airways obstruction in all patients with the exception of one (Case 7) who was initially seen by us at a time when her symptoms were in remission. During a subsequent relapse she was treated at home by her own practitioner with prednisolone, to which she responded well. Provocative inhalation tests with nebulized solutions of histamine and studies of the comparative bronchodilator effectiveness of atropine methonitrate and isoprenaline were done in a few patients but the results contributed no data of practical value. Only five patients showed an appreciable bronchodilator response to isoprenaline aerosol, as judged by a 10% or greater increase in forced expiratory volume in one second. All patients had normal overall alveolar ventilation with arterial P_{CO_2} values ranging from 37 to 44 mm Hg (estimated by the rebreathing method of Campbell and Howell.[6]).

Chest radiographs showed apical opacities consistent with old, quiescent pulmonary tuberculosis in five instances (Cases 3, 8, 9, 13, and 14). Otherwise the radiographs appeared normal and in particular showed no evidence of left heart failure. Electrocardiograms were abnormal in six patients (Cases 2, 4, 9, 11, 12, and 14). One (Case 9) had transient "P pulmonale" waves and clockwise rotation of the heart during an exacerbation; flattened T waves and S-T depression were seen in leads facing the left ventricle in the other five. In addition Case 4 had an abnormal

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TABLE I—*Clinical Data*

Case No.	Sex	Age at Onset	Dominant Features
1	M.	60	Nocturnal cough, dyspnoea, wheezing
2	F.	60	Chronic bronchitis.* Sudden onset of episodic dyspnoea, wheezing, P.N.D., and precordial pain
3	M.	61	Simple bronchitis 10 years. Abrupt change of symptoms with wheezing, dyspnoea, and P.N.D.
4	M.	61	Chronic bronchitis 10 years. Sputum usually purulent. Sudden development of P.N.D. and episodic chest pain
5	M.	61	Dyspnoea, wheezing, and P.N.D.
6	M.	62	Chronic bronchitis 22 years. Sudden onset of wheezing and P.N.D.
7	F.	62	Episodic wheezing, especially in mornings. P.N.D.
8	M.	63	Chronic bronchitis* with mucopurulent sputum. Sudden onset of continuous wheezing
9	M.	63	Chronic bronchitis.* Sudden deterioration with orthopnoea and P.N.D.
10	M.	65	Gradual onset of wheezing and P.N.D.
11	M.	66	Acute development of P.N.D.
12	M.	67	Dyspnoea and P.N.D.
13	M.	68	P.N.D. with retrosternal tightness
14	F.	68	Ischaemic heart disease. Acute onset of dyspnoea, orthopnoea, and P.N.D.
15	F.	69	Acute episodic dyspnoea, wheezing, P.N.D., and chest pain

* Duration uncertain, but greater than five years.
P.N.D. = Paroxysmal nocturnal dyspnoea.

ABLE II—*Laboratory Data*

Case No.	Eosinophils		Skin Tests	Predicted Values*		Measured Lung Volumes†			
	Sputum	Blood (/mm³)				Before Steroids		On Steroids	
				FEV1	VC	FEV1	VC	FEV1	VC
1	++	200	Pos.	2·85	3·85	0·87	1·84	2·38	3·94
2	++	780	Neg.	1·90	2·30	0·49	1·16	1·79	2·45
3	++	270	Neg.	2·94	4·05	1·46	2·96	2·65	3·84
4	++	480	Neg.	3·03	4·15	0·76	1·69	1·68	2·63
5	++	1,390	Neg.	3·00	4·10	1·52	2·74	2·66	3·69
6	++	390	Neg.	2·75	3·80	0·83	1·81	1·68	2·50
7	++	630	Neg.	1·71	2·00	1·51	2·17	—	—
8	+	280	Neg.	3·02	4·25	0·92	2·17	2·34	3·56
9	++	240	Neg.	2·50	3·50	0·70	1·87	2·01	3·14
10	++	880	Pos.	3·33	4·60	1·36	3·47	3·47	5·10
11	++	460	Pos.	2·70	3·92	0·98	1·57	2·65	3·49
12	No sputum	0	Neg.	2·78	3·95	0·87	1·45	2·40	3·70
13	++	930	Pos.	2·80	4·00	1·30	2·39	2·17	3·50
14	++	210	Neg.	2·05	2·70	0·92	1·36	1·67	2·00
15	+	100	Neg.	1·85	2·40	0·37	0·38	1·68	2·17

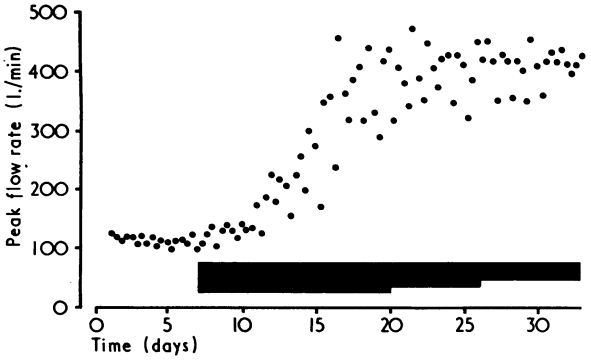
* Predicted values (1 B.T.P.S.) from Cotes.[16]
† Lung volumes were measured with a Vitalograph and have been corrected to B.T.P.S.
FEV1 = Forced expiratory volume in one second. VC = Vital capacity. B.T.P.S. = Body temperature, pressure, and saturation.

P-wave axis due to an ectopic pacemaker. No patient had electrocardiographic evidence of right ventricular hypertrophy.

Eight patients were treated with disodium cromoglycate. Five (Cases 1, 3, 6, 12, and 13) derived no benefit and the others (Cases 7, 10, and 11) showed slight but unsustained improvement. Fourteen patients required corticosteroid therapy. Striking improvement occurred in all instances, and side effects have so far proved no more conspicuous or troublesome than in other age groups of asthmatic patients. Weight gain varied from 1·8 to 9 kg, and one patient (Case 8) developed diabetes mellitus.

CASE 1

A 60-year-old man developed increasingly troublesome nocturnal cough associated with dyspnoea, wheezing, and the production of white sputum. On direct questioning he admitted to having unexplained attacks of rhinitis in the preceding 12 months, and his daughter had symptoms suggestive of hay fever. He had smoked 20 cigarettes a day until 15 months previously. Apart from signs of airways obstruction no other abnormality was found on physical examination. His atopic status was confirmed by skin testing, when weal and flare reactions were produced in response to a range of common allergens. Clumps of eosinophils were found in his sputum although the blood eosinophil count was only 200/mm³. As treatment with bronchodilator drugs and disodium cromoglycate had been tried without benefit, a trial of corticosteroids was advised starting at 30 mg of prednisolone a day. Changes in airways obstruction were monitored with a Wright peak flow meter (see Chart). Each point on the Chart represents the average of three readings, each set of readings being recorded at the same time every morning (7 a.m.), afternoon (1-2 p.m.), and night (9-10 p.m.). Clinical improvement corresponded to the peak flow results. Corticosteroid therapy has since been discontinued without any untoward effect.



Response to prednisolone therapy in Case 1. Initial dose 30 mg daily, reducing by 5-mg steps.

CASE 13

The patient was well until 66 years of age, when he had a sudden unexplained syncopal attack after which he experienced dyspnoea especially at night, cough productive of thick mucoid sputum, and gripping retrosternal pains indistinguishable from angina pectoris. At first he was thought to have had a "heart attack" with residual angina and left ventricular failure. Later his persistent cough was attributed to chronic bronchitis. He was referred for assessment of his "chronic obstructive bronchitis" six years after the onset of symptoms, at the age of 72 years.

On questioning, he admitted to occasional sneezing bouts, and gave a family history of asthma (his father and one niece had died from asthma). He was overweight, his B.P. was 180/110 mm Hg, without cardiomegaly, and there were signs of airways obstruction. Prick skin tests produced weal and flare responses to a range of common allergens, and he had both blood (930/mm³) and sputum (++) eosinophilia. There was no evidence of allergic aspergillosis. The electrocardiogram was normal. A chest radiograph showed

fibrotic changes at both lung apices; there were no signs of pulmonary venous congestion.

Treatment with disodium cromoglycate was accompanied by limited success. A subsequent trial with prednisolone produced marked improvement, both symptomatically and on objective testing.

Discussion

The term "asthma" still awaits a comprehensive and generally acceptable definition.[7] We use it here to imply diffuse airways obstruction which varies in severity either spontaneously or as a result of therapy. When patients such as ours are fully documented the diagnosis of asthma seems immediately apparent but such cases may readily masquerade under various guises such as chronic bronchitis, emphysema, or left heart failure. Indeed, some patients may have had chronic bronchitis[8] for several years before a change in symptoms occurs, suggestive of the additional development of asthma. An abrupt onset of breathlessness, episodic wheezing, and paroxysmal nocturnal dyspnoea appear to be important symptoms. A past history of sudden bouts of rhinorrhoea or sneezing strengthens the suspicion of asthma, as does a family history of allergy. Nevertheless, recollection of such potentially relevant features may be poor in the elderly and in our experience is usually lacking.

It is not surprising that a history consistent with chronic bronchitis is common in a group in their seventh decade, most of whom had smoked at some time and who lived in an industrial conurbation. Of six patients with such a history two had circulating precipitins to *H. influenzae*, which have been shown to occur more commonly in association with chronic bronchitis than asthma.[9] Precipitins to *H. influenzae* were also found in a third patient who gave no antecedent history of bronchitis. But neither a history of chronic bronchitis nor finding serum precipitins to *H. influenzae* excludes the possibility that the real cause of a patient's disability is asthma, and we believe that the risk of this possibility being overlooked is increasingly likely in older patients.

Paroxysmal nocturnal dyspnoea was a major symptom in most of our patients (Table I). It is most commonly seen in left heart failure, asthma, and psychogenic breathlessness.[10] Left ventricular failure may be particularly difficult to differentiate from bronchial asthma when it produces airways obstruction and when other characteristic signs are lacking. Radiographic evidence of pulmonary venous congestion is then invaluable, and on occasions we have found the arterial response to the Valsalva manoeuvre[11] of help. Rarely, a therapeutic trial with diuretics and digoxin may be required. Electrocardiographic abnormalities were seen in six of our patients and the E.C.G. therefore has little discriminatory value. Similarly, a history of retrosternal pain may be misleading: three patients gave such a history and a fourth had a sensation of tightness in the chest which we could not distinguish from angina pectoris. And in this age group angina could well be precipitated by the hypoxia of an asthmatic attack. Despite these difficulties, however, paroxysmal nocturnal dyspnoea appears to be a valuable symptom which should arouse a suspicion of asthma if only because it is so often a dominant feature of the condition.

Sputum eosinophilia is of considerable value in assessing asthmatic patients, and sometimes provides the single most useful clue to the diagnosis. Obviously, one does not rigidly demand the finding of sputum eosinophilia before diagnosing asthma, and sometimes (especially in children) where the clinical features, natural history, and therapeutic response are typical of asthma, their presence can be reasonably assumed. Also, in patients presenting with status asthmaticus treatment takes precedence over investigation, and an occasional patient fails to produce sputum—especially if corticosteroid therapy has been initiated. In others, where the aetiology of airways obstruction is uncertain, sputum eosinophilia is an invaluable diagnostic pointer towards asthma. Sputum eosinophilia has been found in patients with emphysema[12] and chronic bronchitis[13] but these appear to be rare associations. We would support the view that sputum eosinophilia equates well with steroid responsiveness.[14] Corticosteroid therapy reduces not only sputum volume but also sputum eosinophil content; hence the sputum should be examined before starting such treatment.

Corticosteroid therapy was required in all but one of the patients to control their symptoms and permit them to return to a normal life. Because of the hazards of corticosteroid drugs, and especially in the elderly, treatment should ideally be preceded and followed by frequent objective measurements of airflow obstruction.[15] The response in peak flow rate that can be achieved is shown in the Chart. Side effects of steroids which might be expected in the elderly, such as osteoporosis, diabetes, and reactivation of tuberculosis, were not outstanding problems. Diabetes developed in one of the patients and was controlled with an appropriate diet and an oral sulphonylurea drug. Five patients had radiographic changes consistent with old, quiescent pulmonary tuberculosis, and to one we gave antituberculous chemotherapy concurrently with steroids. Similar changes could be the consequence of allergic aspergillosis though we found no evidence of this. No patient showed any sign of recrudescence of tuberculosis during follow-up periods of 6 to 18 months. Perhaps the risk of reactivation of tuberculosis is greatest when least expected, and such patients must be kept under regular surveillance for this complication.

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