Recurrence of asthma following removal of a noradrenaline-secreting phaeochromocytoma

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Summary

A patient with asthma and a phaeochromocytoma is described. At about the time she was first noted to be hypertensive her asthma resolved spontaneously but bronchospasm returned with some severity when the tumour was removed. The phaeochromocytoma was of the noradrenaline secreting variety. Possible mechanisms through which this catecholamine might have produced the observed alleviation of asthma are considered.

KEY WORDS: hypertension, α-adrenoceptor, noradrenaline.

Introduction

Asthma may improve spontaneously with age particularly as children mature. Complete disappearance for several years followed by severe recurrence is unusual. We report such a relapse of asthma following the removal of a noradrenaline (NA) secreting phaeochromocytoma. The increased plasma level of circulating NA in this patient may have temporarily alleviated the tendency to bronchoconstriction. Such a relationship has not been previously reported.

Case report

A 49-year-old secretary presented in 1982 with a 6-month history of increasing depression. She had felt generally unwell, with a poor appetite, weight loss and occasional nausea but no paroxysmal flushing or palpitation. There had been two episodes of fainting and on the second occasion her blood pressure had been found raised. The patient also described an intermittent pain in the left anterior thigh and groin.

She had first been found to be hypertensive in 1974 and was treated with amiloride and hydrochlorothia-
Discussion

The recurrence of this patient's asthma following removal of the phaeochromocytoma suggests that the rapid decline in the level of circulating noradrenaline (NA) led to bronchoconstriction. NA is not usually associated with the production of bronchodilatation but we felt that the time course of her symptoms was such as to suggest that the catecholamine might have caused the disappearance of her asthma. Numerous studies have been performed to investigate the effect of various catecholamines on different preparations of lung tissue. Strip preparations of human peripheral lung contract when NA is applied (Black, Turner and Shaw, 1981) probably due to the vascular element which this preparation contains. When isolated strips of human bronchi (removed at surgery for cancer or immediately post-mortem) have been tested NA has produced a net relaxant effect (Mathe, Astrom and Persson, 1971; Goldie, Paterson and Wale, 1982) presumably due to the ability of NA in high concentration to stimulate the β-mediated effect when the population of α-receptors is sparse. Cabezas, Graf and Nadel (1971) have provided evidence in vivo that there are no functionally significant α-adrenergic constrictor mechanisms in the bronchi by stimulating the thoracic sympathetic nerves of dogs and measuring the airways dimensions directly from bronchograms; after pharmacologic β-blockade, sympathetic stimulation did not constrict the airways. There has been considerable debate over the role of α-receptors in airways muscle especially in the smaller airways. Some have argued that they do not exist (Foster, 1966; Guirgis and McNeill, 1969) but it seems more likely that they are present in very sparse numbers (Mathe et al., 1971) and that α-adrenergic constrictor function can only be demonstrated after β-blockade.

An alternative explanation for the disappearance of this patient's asthma rests on the discovery that adrenergic fibres of sympathetic origin enter peripheral parasympathetic ganglia where they inhibit transmission from the cholinergic parasympathetic preganglionic fibres to the neurones of the ganglion (Skok, 1980). This arrangement is best documented in the gut but catecholamine-containing elements have been demonstrated by histofluorescence in the cholinergic ganglia of calf lung (Jacobowitz et al., 1973). The innervation of the bronchi is predominantly parasympathetic constrictor (Mann, 1971) and any reduction in cholinergic traffic would be effectively bronchodilator. High levels of circulating NA might therefore be expected to mimic the inhibitory effect of the adrenergic neurones reducing acetylcholine secretion at the neuromuscular junction with consequent bronchodilatation.

Some sympathetic fibres terminate on bronchial smooth muscle producing bronchodilatation by stimulation of the β-receptor. It has been suggested (Henderson et al., 1979; Barnes, Dollery and MacDermott, 1980) that in asthmatics the number of α-receptors are increased and β-receptors decreased; the fact that the asthma of our patient improved rather than deteriorated in the presence of high concentrations of circulating NA militates against this suggestion. It seems that if asthma disappears as hypertension develops then the possibility of a catecholamine secreting tumour should be considered.

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References


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