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Nasal polyps in identical twins

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Abstract
Nasal polyps are multifactorial in aetiology but are associated with respiratory diseases, particularly late onset asthma. Several members of a family may be affected with nasal polyps but there is little evidence for a genetic basis for this. Some evidence to support a genetic predisposition comes from the development of polyps in identical twins.

Introduction

Although nasal polyps are a common clinical finding and occur in between one and two per cent of the caucasian population, little is known of their aetiology. Some conditions are known to be associated with their development: cystic fibrosis, Kartagener's syndrome and primary immune deficiency. These cases are rare and the majority of polyps arise either with the development of late onset asthma, which may be associated with aspirin intolerance, or without any chest disease.

While they are not a disease as such, it would be helpful to look at other factors that predispose to their development including a genetic predisposition. Some families have several members in different generations with nasal polyps but while such groupings suggest a genetic predisposition, the evidence is anecdotal. Identical twins who developed nasal polyps are presented here to support this hypothesis.

Case report

Identical male twins aged sixty developed nasal polyps.

The first patient presented in 1987 with a long history of nasal symptoms and was found on examination to have nasal polyps. These responded to beclomethasone diproprionate initially but recurred and were removed surgically in 1989. The patient had a recurrence of nasal symptoms and required a further polypectomy in 1991.

His brother had a long history of rhinitis with blockage, attacks of sneezing and anterior rhinorrhoea and a post nasal discharge which had exacerbated over the proceeding nine months. Nasal polyps were removed in 1991.

Neither patient had asthma nor seasonal allergic rhinitis. Both had different occupations and lived in different parts of the West Midlands.

Discussion

Cystic fibrosis (CF) is an autosomal recessive disease which is associated with a gene defect on the seventh chromosome in three quarters of the cases. A number of different phenotypes exist but there is no information at present to determine whether nasal polyps are associated with any particular one. Polyps are found in patients who have the respiratory manifestations of the disease and who have less colonization with *staphylococcus aureus* and a lower incidence of meconium ileus and gastrointestinal symptoms in the first year of life (Drake-Lee and Pitcher Willmott, 1982). This study suggested that this finding might be due to a variation in the host genome with which the CF gene interacts.

CF gene probes have been used to look at the genetic material in seven patients with nasal polyps without CF (Burger *et al.*, 1991). G551D mutation was found to be higher than expected in this small study so this work suggests that nasal polyps are associated with a higher incidence of this gene mutation.

Molony and Oliver (1980) looked at the HL-A classification of 29 patients with nasal polyps and found that there was a higher incidence of A1/B8 in patients who had nasal polyps, asthma and aspirin hypersensitivity and that there was a tendency for this to be found in those who had asthma and polyps.

Asthma can be subgrouped clinically into two groups; those who have an allergic basis to their disease and those who do not. The role of allergy has been studied more extensively and it has long been recognized that there is a familial tendency to asthma which is modified by environmental exposure to allergens. This point has been recently re-explored by Cookson and Hopkin (1988) and Cookson *et al.* (1989) who suggested that the tendency to produce IgE was situated on the eleventh chromosome. Studies in identical twins in Australia confirm that there was a liability to reported disease amongst twins and that these were higher in homozygous twins (Duffy *et al.*, 1990).

Late onset asthma or intrinsic asthma rather than allergic asthma is associated with nasal polyps (Drake-Lee *et al.*, 1984). The evidence for HLA subgrouping in intrinsic asthma is conflicting but there does appear to be a genetic predisposition to late onset asthma in families (Pirson *et al.*, 1991).

This report demonstrating nasal polyps arising in identical twins who live apart, supports further the hypothesis that a genetic predisposition may occur in some cases in the development of nasal polyps. Additional studies are required to confirm the validity of this conclusion.

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