REVIEW

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Chronic inferior turbinate enlargement and the implications for surgical intervention*

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SUMMARY

Nasal obstruction due to chronic enlargement of the inferior turbinate is a common problem for the ENT surgeon. This review will discuss the pathology of 'bilateral' and 'unilateral' turbinate enlargement associated with chronic rhinitis and nasal septal deviation, and focus on the structural changes in the turbinates. Cellular hyperplasia, tissue oedema and vascular congestion all contribute to turbinate enlargement, but there is some evidence that bony enlargement is associated with unilateral turbinate enlargement. There is no evidence for cellular hypertrophy despite the common use of the term 'turbinate hypertrophy' and this term should be replaced with the more correct term of 'turbinate enlargement'. The underlying pathology of turbinate enlargement has important implications for the surgical management of nasal obstruction.

Key words: turbinate hypertrophy, turbinate enlargement, surgery, mucosa, bone

INTRODUCTION

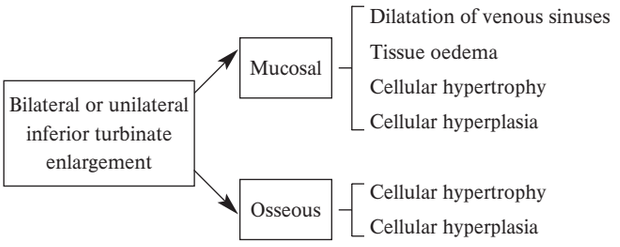
Chronic nasal obstruction is a common symptom of nasal disease. Enlargement of the inferior turbinates is one of the most frequent underlying mechanisms [1]. Turbinate enlargement can be bilateral or unilateral. Bilateral turbinate enlargement is caused by nasal inflammation as a result of allergic and non-allergic rhinitis, other environmental triggers, such as dust and tobacco and medical causes, including pregnancy [2]. Unilateral turbinate enlargement occurs in association with a congenital, or acquired anatomical deviation of the nasal septum into the contralateral nasal passage. It has been proposed that unilateral turbinate enlargement occurs to protect the more patent nasal passage from the drying and crusting caused by excess airflow [3,4], but the underlying mechanism for this is unknown.

Turbinate surgery is commonly performed in an attempt to relieve nasal obstruction, despite a lack of evidence regarding the underlying pathology of turbinate enlargement. In the literature [3,4] and in rhinology textbooks [1], the mucosal layer of the inferior turbinate is frequently implicated as the structure responsible for turbinate enlargement. In particular it is commonly claimed that dilatation of the venous sinuses, due to engorgement with blood, is the underlying mechanism [1,5]. Pollock [6] suggests that turbinate enlargement is simply due to "mucosal thickening". It has also been suggested that turbinate enlargement is caused by mucosal oedema with eosinophils and mast cells present in an inflammatory response [1]. Saunders [7] describes three ways in which the inferior turbinate causes nasal obstruction. In addition to mucosal engorgement he also describes an undue prominence of the turbinate bone and redundant hyperplastic turbinate mucosa as potential mechanisms for turbinate enlargement [7]. Fairbanks and Kaliner [8] claim that turbinate enlargement involves both bony and mucosal elements.

The term 'turbinate hypertrophy' was first coined in the late 1800s [9,10] to describe enlargement of the inferior turbinate and it remains in common use today. Hypertrophy is defined as "the enlargement of an organ or tissue resulting from an increase in size of its cells" [11]. In contrast, cellular hyperplasia is enlargement due to an increase in the number of cells. Hyperplasia and hypertrophy of the mucosal or osseous layers of the inferior turbinate also provide potential explanations for the mechanism of inferior turbinate enlargement (Figure 1).

An understanding of the structural mechanisms of turbinate enlargement is important to the rhinologist who is frequently

Figure 1. Possible mechanisms of inferior turbinate enlargement.



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faced with managing this common problem. In particular, it is helpful to know whether there is mucosal or bony enlargement as this will impact on the choice of surgery.

In this review the structure of the normal inferior turbinate and the pathology of turbinate enlargement will be examined. The review will focus on the evidence for mucosal and bony changes observed in turbinate enlargement with reference to bilateral and unilateral disease. The implications of the pathology of turbinate enlargement on the surgical management of patients with nasal obstruction will be discussed and it is proposed that the term turbinate enlargement is a more accurate description of the pathology than turbinate hypertrophy.

STRUCTURE OF THE NORMAL INFERIOR TURBINATE

The inferior turbinate is an elongated, scroll-like paired structure of the lateral nasal wall. It consists of a central layer of bone and a mucosal layer on either side [12]. The normal thickness of the mucosal and bony layers has been studied with computed tomography (CT) [13] and histology [3,12]. CT scans showed the mean anterior dimensions of the medial and lateral mucosa were 3.33 mm and 2.06 mm respectively [13]. However the patients studied are unlikely to represent 'normality' because all had septal deviations, and the measurements were taken from the nasal passage with the smaller turbinate. The mean thickness of the medial mucosal and lateral mucosal layers was 1.76 mm and 1.03 mm respectively in a study examining cadaveric and post-operative inferior turbinate specimens [3]. In a similar study the values were 1.59 mm and 0.94 mm [12]. These values seem very thin, and it is likely that the vascular component of the mucosal width is underestimated by examining histological specimens because of collapse of the erectile venous sinuses.

The turbinate is mainly covered with pseudostratified columnar epithelium with smaller islands of squamous epithelium, basal cells, ciliated and non-ciliated cells and comprises 10% goblet cells [12]. The epithelium is separated from the lamina propria by a basement layer. The lamina propria is thicker in the medial layer than the lateral layer and extends to the periosteum of the bony layer. It consists of connective tissue containing a few lymphocytes and other immunocompetent cells, seromucous glands, a rich network of thin walled venous sinuses and a few arteries. The osseous layer consists of cancellous bone. The mean thickness of the bone is 1.2 mm [12] in a histological study and 1.6 mm when measured with CT [13]. The mean height of the inferior turbinate is 7.75 mm [3]. Age and gender are reported to have no significant effect on total turbinate thickness or the thickness of individual turbinate layers. However, ageing is associated with a reduction in the number of submucosal glands and increased venous sinuses [12].

The structure of the inferior turbinate can be studied by several different methods; histology, CT and decongestion. Histology can provide information on the amounts of soft and bony tissues and information about the relative amounts of different tissues such as glands and venous sinuses. Histological examination can determine whether turbinate enlargement is due to cellular hypertrophy or hyperplasia. CT can give measures of soft and bony tissue components in the turbinates. The relative degree of shrinkage of the turbinate on application of a topical decongestant can provide information about the amount of venous sinuses in the turbinates.

1. STRUCTURE OF THE ENLARGED INFERIOR TURBINATE IN UNILATERAL DISEASE

Histology

Turbinate tissue from patients with septal deviations and compensatory unilateral turbinate enlargement has been examined in histological studies [3]. The proportion of submucosal glands, connective tissue, epithelium, arteries and venous sinuses in the inferior turbinates from these patients has not been shown to be significantly different when compared with cadaveric controls [3]. The width of the medial mucosa and lateral mucosa in both groups were not significantly different despite the fact that the enlarged inferior turbinates were significantly wider [3]. Qualitative assessment of mucosal architecture has shown that 42.1% of turbinates from patients with compensatory enlargement exhibit pathological changes. These include dilated, engorged thin-walled venous sinuses, fibrosis of the lamina propria, subepithelial infiltration of lymphocytes, plasma cells and eosinophils and dilatation of excretory glandular ducts [3].

Histological examination of inferior turbinates from patients with septal deviations and compensatory enlargement of the contralateral inferior turbinate has shown significant bone expansion when compared with cadaveric controls [3]. A two-fold increase in the thickness of the osseous layer of the inferior turbinate was observed [3]. Enlargement of the bony layer accounted for 3/4 of the entire growth of the inferior turbinate. The number or size of individual cells was not examined.

Computed tomography

The relative enlargement of bony and soft tissue components of inferior turbinates in patients with unilateral compensatory turbinate enlargement associated with nasal septal deviation has been evaluated by CT in several studies [4,13]. These studies have the advantage of examining the inferior turbinate in life.

The mean width of the anterior medial mucosa in unilateral turbinate enlargement is significantly wider at 5.33mm, when compared with the contralateral turbinate as a control. The lateral mucosal layer has also been shown to be significantly wider in patients with unilateral compensatory enlargement [13].

A study of 99 patients with moderate to severe septal deviation used CT to calculate intraturbinate ratios at three different levels to assess mucosal contribution to turbinate enlargement [4]. The intraturbinate ratio is the ratio of overall turbinate cross sectional area to bony turbinate cross sectional area. In enlarged turbinates associated with nasal septal deviations, the intraturbinate ratio was highest in the posterior segment, implying that the mucosal component is predominantly responsible for turbinate mass in the posterior segment [4].

The interturbinate ratio is the CT calculated ratio of the cross sectional area of the inferior turbinate bone on either side of the septum. The interturbinate ratio for patients with severe septal deviations has been shown to be significantly higher compared with patients with normal and mild deviations in the anterior and middle segments of inferior turbinate bone [4]. This suggests that in compensatory enlargement of the inferior turbinate associated with severe septal deviation there is prominent enlargement of the inferior turbinate bone in the anterior and middle thirds when compared with the contralateral side.

Decongestant studies

Several studies have examined the vascular contribution to compensatory unilateral turbinate hypertrophy by evaluating response to decongestants using acoustic rhinometry [14-16]. Hilberg [14] found no significant difference in the effect of decongestion on minimal cross-sectional area (MCA), cross-sectional area at 3.3 cm from the nostril, and cross-sectional area 4 cm from the nostril, when comparing a group of patients with turbinate enlargement with normal controls. Grymer [15] defines 'mucosal hypertrophy' as present if decongestion increases cross sectional area by more than 100% relative to the non-decongested value. This is based on the values of a 63% area increase in a normal reference group. In patients with compensatory unilateral turbinate enlargement due to septal deviations of varying severity 62% of patients met this criterion for 'mucosal hypertrophy' [15]. This led the authors to conclude that 'mucosal hypertrophy' is common to patients with nasal obstruction regardless of the degree of septal deviation.

2. THE STRUCTURE OF THE ENLARGED INFERIOR TURBINATE IN BILATERAL DISEASE

Histology

Punch biopsies taken from the inferior turbinates of patients with chronic hypertrophic allergic and non-allergic rhinitis have shown characteristic features [17]. An increase in goblet cell population with thickening of the basement membrane has been documented. There is also an increase in the number of blood vessels observed with associated congestion and dilatation and connective tissue stromal oedema. In addition patients with allergic rhinitis have increased numbers of eosinophils and patients with non-allergic rhinitis exhibit a marked predominance of the mucous acini of the glands [17].

A scanning electron microscopy study of inferior turbinate biopsies taken from patients suffering with perennial rhinitis and normal controls has shown qualitative differences in the ultrastructure of the mucosa. Patients with perennial rhinitis have more pseudo-stratified columnar epithelium and more goblet cells than controls, supporting the idea that cellular hyperplasia is responsible for turbinate hypertrophy [14].

Berger compared inferior turbinate specimens from patients with bilateral enlargement with specimens from a control group undergoing septal surgery [19]. Although there are serious issues concerning the nature of the control group, it is the only study that examines both the mucosal and bony components in bilateral turbinate enlargement. The enlarged turbinates were significantly wider than the controls and the medial mucosal layer made the greatest contribution to the total increase in inferior turbinate width [19]. There was no significant difference in the width of the lateral mucosal layer or the bony layer between groups. Furthermore, the increase in the width of the medial mucosa was predominantly due to an increase in the thickness of the lamina propria that houses subepithelial inflammatory cells, venous sinuses and submucosal glands [19]. The relative proportion of venous sinuses in enlarged turbinates was significantly greater than in controls. 26.8% of the medial mucosal layer of enlarged inferior turbinates comprised venous sinuses compared to 19.2% of control inferior turbinates [19]. Qualitative assessment of the enlarged inferior turbinates showed metaplastic squamous epithelium in 10% of specimens, fibrosis of the lamina propria in 90% of specimens, dilated and engorged thin-walled venous sinuses in 15% of specimens and a marked subepithelial inflammatory cell infiltrate in 65% of specimens. Qualitative assessment of control inferior turbinates revealed the only pathology was dilated venous sinuses, which occurred in 5% of specimens [19].

Decongestant studies

Corey used acoustic rhinometry to demonstrate a significant difference between controls and patients with allergic rhinitis in their response to a topical decongestant at the minimal cross-sectional area (MCA) [16]. The average total percentage area change at the MCA for controls was 15.6% compared to 24.6% for patients with allergic rhinitis [16]. This infers that patients with allergic rhinitis have an exaggerated response to decongestants because there is a greater change in the volume of the venous sinuses than in controls. However, these patients were not labelled as having turbinate hypertrophy, although their symptom scores for nasal congestion were high. Decongestant studies may underestimate the vascular contribution to mucosal enlargement in some patients with chronic inflammation where there is mucosal infiltration of fibrous tissue. This can render engorged venous sinuses incapable of decongestion [21,22].

DISCUSSION

This is the first review to examine the evidence for the mechanism of turbinate enlargement. Despite the common occurrence of turbinate enlargement there is little evidence to explain which turbinate structures are responsible for the enlargement. The limited evidence available appears to suggest that there may be two types of turbinate enlargement with underlying differences in the pathology.

In unilateral turbinate enlargement associated with nasal septal deviation of the contralateral turbinate there is limited evidence for bony expansion as a major contributing factor. There is evidence from CT studies to suggest that an element of mucosal enlargement is also important. Although there is some evidence for an increase in size of the bony component of the turbinate it is not clear if this is merely due to an increase in the overall size of the turbinate. It is generally assumed that the unilateral turbinate enlargement associated with septal deviation occurs to protect the more patent side from the drying and crusting caused by excess airflow [3,4]. A deviated nasal septum may be congenital, the product of growth asymmetry, or the result of trauma. However, it is unclear whether cellular hyperplasia or hypertrophy is the underlying mechanism for bone expansion. The underlying cause of unilateral turbinate enlargement is also unknown. The turbinate enlarges to occupy the increased volume of the nasal passage, but factors that trigger and control the increase in the size of the turbinate are unknown.

In bilateral turbinate enlargement there is evidence for a mucosal contribution to enlargement. Histological studies suggest that a combination of mechanisms may be responsible for mucosal enlargement, including cellular hyperplasia, tissue oedema and vascular congestion. There is no evidence for cellular hypertrophy. Therefore there is no evidence to substantiate use of the term 'turbinate hypertrophy' and it is proposed that the term 'turbinate enlargement' is a more accurate description of the condition. There is no evidence to support bony enlargement in bilateral disease. With cases of acute rhinitis it appears that the main cause of turbinate enlargement is filling of venous sinuses since the size of the turbinate can be reduced by application of a topical decongestant [22]. In cases of chronic rhinitis there may be some tissue fibrosis due to a chronic inflammatory response and this may make it difficult to reverse the increase in turbinate size by medical intervention, such as treatment with topical corticosteroids. In these cases surgery to reduce the size of the turbinate may be the only option for treatment of nasal obstruction.

Many surgical techniques have been described to reduce the size of enlarged turbinates. Today, these can be broadly divided into two main categories: turbinate electrosurgery or soft tissue reduction (including submucosal diathermy and surface diathermy) and turbinate bone resection/reduction (including crushing and trimming of the inferior turbinate bone and total turbinectomy). An appreciation of the relative enlargement of the bony and soft tissue constituents of the inferior turbinate is important when deciding on which type of surgical intervention is most appropriate.

Electrosurgical techniques result in heating of intracellular contents and vaporization of cells. Heat causes coagulation and obliteration of the venous sinuses leading to submucosal fibrosis and scarring [23] which is thought to anchor the mucosa to the periosteum. Therefore electrosurgery would be most suited for the treatment of mucosal turbinate enlargement due to vascular congestion and oedema, and avoids the additional risks associated with surgery involving the inferior turbinate bone. Inferior turbinate enlargement due to predominantly bony changes, as occurs in unilateral compensatory enlargement associated with a septal deviation, would be best treated by reduction or resection of the inferior turbinate bone.

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