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RHINOLOGY: NASAL OBSTRUCTION (JV NAYAK, SECTION EDITOR)

The Inferior Turbinate: Role in Normal Respiration and Airway Obstruction

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Abstract

Purpose of Review The understanding of the inferior turbinate (IT) function, role in nasal obstruction, and its management for symptomatic relief has evolved. We aim to provide a comprehensive review of the anatomy, contribution to nasal function and response of the IT to sinonasal disease.

Recent Findings Techniques to study the nasal physiology are expanding; these include the computational flow dynamics that has allowed a better understanding of the function and role of the inferior turbinate in normal respiration and airway obstruction.

Summary The IT function includes warming, moisturizing, and filtering the air prior to the entrance to the lower airways. More sophisticated techniques have been developed to study the nasal physiology, though not widely used in clinical practice.

Keywords Inferior turbinate · Nasal obstruction · Airway obstruction · Normal respiration · Hypertrophy of the inferior turbinate

Introduction

The approach in the management of the inferior turbinate has evolved and continues to evolve as we continue to understand the importance of this structure in the sinonasal cavity. Otolaryngologists recognize the middle and superior turbinates as important structures in the nasal cavity. The complex anatomy of the middle turbinate, and its proximity to the sinus cavities, requires surgeons to thoroughly understand its embryology, anatomy, and function. The role of the superior turbinate, particularly with respect to olfaction, requires surgeons to better comprehend its anatomy to preserve its structure and function during surgery. Some may argue it is the most important turbinate in the nasal cavity. A better understanding of the anatomy, function, and response to various sinonasal conditions will provide surgeons a better understanding on how to approach the inferior turbinate in its management.

Anatomy

The embryology of the inferior turbinate is very similar to that of the middle and superior turbinates. Two studies have shown the inferior turbinate originates directly from the lateral portion of the cartilaginous nasal capsule [1, 2]. The inferior turbinate is cartilaginous in nature during the 14th week of gestation, but during the 17th or 18th week, ossification of cartilage takes place along the lateral half of the inferior turbinate. The free edge of the inferior turbinate remains cartilaginous until the 36th week [2]. The upper margin of the cartilage prior to ossification is attached to the maxillary bone and is likely the fracture point during intraoperative out-fracturing [2].

The vascular supply of the inferior turbinate is given by the inferior turbinate artery that arises in 54.2% of the cases exclusively or in 16.6% partly off the descending

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palatine artery [3]. Most commonly, 3 arteries branch within the inferior turbinate (IT): one superomedially, inferomedially, and inferolaterally [3]. Surprisingly, the caliber of the artery gets larger as it courses toward the inferior turbinate anteriorly, and it is believed to occur due to anastomosis with facial artery at the pyriform aperture [4]. Some surgeons may experience troublesome bleeding along the anterior face of the inferior turbinate following inferior turbinate procedures.

The innervation of the inferior turbinate primarily comes from the posterior inferior nasal nerve (PINN) as well as branches of the superior inferior nasal nerve. The PINN has been studied well due to its roll in non-allergic rhinitis. Cadaveric dissections demonstrate that the PINN is located just posterior to the descending branch of the sphenopalatine artery [5]. However, there are a number of accessory branches directly from the pterygopalatine ganglion or greater palatine nerve that innervate the inferior turbinate [6]. Consequently, it is not surprising that patients who undergo posterior inferior nasal neurectomy for non-allergic rhinitis may not have a complete resolution of their symptoms due to the number of accessory nerves innervating the inferior turbinate.

The mucosa of the inferior turbinate is the primary target in inferior turbinate hypertrophy. The mucosa is similar to respiratory epithelium in that it is comprised of pseudostratified ciliated columnar epithelium. Stratified squamous epithelium can be found on the face on the inferior turbinate. The mucosa of the inferior turbinate can be divided into two components: medial mucosal layer and lateral mucosal layer. The medial mucosal layer is significantly thicker than the lateral mucosal layer [7]. The significant difference is largely due to the asymmetric lamina propria, found between the basement membrane and the periosteum. The lamina propria houses serous, mucous, and seromucous glands, in addition to venous sinusoids, which are also significantly more prominent on the medial side compared to the lateral side [7]. The expansile capabilities of the venous sinusoids allow for significant mucosal growth during the normal nasal cycle and in pathologic states [8]. Given the mucosal architecture, it is understandable that submucosal resection of inferior turbinates with a microdebrider is advantageous when addressing inferior turbinate hypertrophy [9, 10]. By elevating the periosteum off the inferior turbinate, the microdebrider is able to resect the thick lamina propria, glands and venous sinusoids that play a significant role in the hypertrophic component of the turbinate.

The bone of the inferior turbinate has been classified as lamellar, compact, and bullous [11]. The bone is termed lamellar when thin, compact when bulky, combined when bulky with a prominent spongy osseus layer, and bullous when pneumatized. Compact and combined types have the greatest cross-sectional area, which could theoretically lead to more prominent obstruction in pathological states [11]. In one study, lamellar was found to be most common and bullous least [11]. It is known that pneumatization of the turbinates most commonly occurs in the middle turbinate, followed by the superior, then inferior turbinates [12]. An appreciation that inferior turbinate hypertrophy can be associated to the underlying bone is important in the management. On physical exam, poor response of inferior turbinate hypertrophy to topical nasal decongestant indicates high likelihood of hypertrophic bone, which will require surgical resection for optimal results. Imaging of the sinonasal cavity can also provide evidence of hypertrophic bone as the underlying cause of the generalized inferior turbinate hypertrophy but should not be the primary investigation in patients presenting with nasal obstruction.

Contribution to Nasal Function

The sinonasal cavity functions to warm, moisturize, and filter the air prior to entering the lower respiratory tract. Many components of the nasal cavity contribute to these tasks, but the inferior turbinate plays a major role in these functions. Computational flow dynamics (CFD) has provided a better understanding of the role and function of the inferior turbinate. CFD studies have shown slight variability between patients with respect to airflow, but the bulk of the airflow is described to go through the common meatus, followed by the middle meatus [13–15]. The size of the inferior turbinate can have a major impact on the size of the common meatus and consequently affect the bulk of the nasal airflow. However, a few studies refute the claim that the major airflow occurs through the common meatus, arguing instead for greater airflow through the middle meatus instead [16, 17••). CFD studies have also shown that a large majority of the warming of inspired air occurs at the head of the inferior turbinate [18–20], which provides the respiratory tract with warm air. The air warms up by the surrounding mucosal tissue after the airflow hits the head of the inferior turbinate and turbulent flow allows the air to slow down. When the warm air is expired through the nasal cavity, it interacts with the cool nasal mucosa resulting in condensation and moisturization of the nasal cavity. The inferior turbinate is believed to provide 16% of the air conditioning, whereas the middle turbinate provides 12%. The rest of the air conditioning occurs along the septum and lateral wall [21•].

Nasal Cycle

Kayser first described the "nasal cycle" phenomena in 1895 (Kayser, 1895). The underlying physiology is not fully clear but the central nervous system is believed to play a role in alternating the congestion and decongestion of the nasal mucosa. The central nervous system, in particular the hypothalamus, along with the sympathetic and parasympathetic nerves seem to control the cycle [22, 23]. The increase in congestion is due to engorgement of venous sinusoids along the anterior end of the inferior turbinate and septum [24]. The classic pattern of the nasal cycle is reciprocal congestion and decongestion of the two sinonasal cavities but changes can occur only on one side and is termed hemicyclic or to both sides at the same time and is termed parallel cycle. Many patients will complain of the nasal cycle particularly at night. The length of nasal cycle can be quite variable [25] but the cycle is known to occur significantly longer and reverse less frequently when patients are asleep [26, 27].

Nasal cycle has been postulated to help with the "respiratory defense" [25]. It is believed that during the congestive phase, the distension of the venous sinusoids effects the cellular tight junctions and the hydrostatic pressure of the sinusoids facilitates plasma exudate onto the surface of nasal epithelium. Plasma exudate contains immunoglobulins, which bind to viruses and bacteria, as well as containing a number of inflammatory mediators [25, 28].

Pathologic Response of the Inferior Turbinates

The aforementioned anatomical differences within the different regions of the inferior turbinate are critical to the pathogenesis of inferior turbinate hypertrophy, regardless of underlying etiology. The location, size, and vasoactive capabilities of the inferior turbinate relegate it a key player in airway resistance [29]. Multiple pathologic processes may alter the gross and microscopic structure of the inferior turbinates, including septal deviation and inflammatory disorders such as allergic rhinitis, nonallergic rhinitis, and chronic rhinosinusitis [30]. These disorders in turn lead to histological differences in terms of hypertrophy and hyperplasia, distorted cilia, inflammatory cell infiltrates, and mucosal thickening with subsequent macroscopic changes and symptomatic obstruction [30, 31]. The internal nasal valve, at the level of the anterior tip of the inferior turbinate, provides up to two-thirds of upper airway resistance during inspiration [32]. As flow is proportional to the cross-sectional area, a decrease in cross-sectional area due to mucosal or bone hypertrophy can significantly reduce airflow and lead to significant symptomatic obstruction [11, 33].

Response to Allergic Rhinitis

Allergic rhinitis (AR) is an unremitting disorder affecting up to 30% of adults and 45% of children with increasing prevalence worldwide [34–37]. The immunologic response and key inflammatory mediators seen in this type-1 mediated hypersensitivity reaction dictate the macroscopic response of the inferior turbinates. Following initial sensitization, subsequent IgE cross-linking on mast cells leads to degranulation and release of mediators that characterize the early-phase reaction [34, 38]. Histamine promotes vasodilation, increased vascular permeability, glandular hypersecretion, and sensory stimulation. Arachidonic acid derivatives contribute as well, including cysteinyl leukotrienes that act as eosinophil chemoattractants and promoters of maturation and adhesion, prostaglandin D2 (PGD2) that recruits Th2 lymphocytes and eosinophils, and thromboxane A2 (TXA2) that increases vascular permeability [34, 38]. The late-phase reaction is characterized by the release of cytokines and mediators including IL-4 IL-13, cysteinyl leukotrienes, and tumor necrosis alpha (TNF-alpha) that lead to increased vascular permeability and recruitment of neutrophils, eosinophils, basophils, mast cells, and lymphocytes [38–40]. Increased integrin expression promotes leukocyte adhesion and transepithelial migration [38]. The resultant edema and cellular infiltration from the early and late-phase reactions contribute significantly to the bilateral turbinate hypertrophy seen in AR [34, 38].

Histologic analysis has verified the presence of elevated levels of inflammatory cells within the mucosa of the inferior turbinates in AR [41]. Compared to the maxillary and ethmoid sinuses, the inferior turbinates also host greater numbers of mast cells and IL-4 in the setting of AR. In combination, these changes lead to neural hyperresponsiveness, venous dilatation, and tissue edema that define the pathophysiologic findings associated with AR. Structural analysis has shown that, in decreasing order, the mucosal constituents that comprised the greatest dimensions in the inferior turbinates are the connective tissue, venous sinusoids, submucosal glands, epithelium, and arteries. While the volume of loose connective tissue and inflammatory cell infiltrate is similar between the medial and lateral mucosal layers in a normal turbinate, venous sinusoids are at baseline more prevalent in the medial and inferior mucosal layers than the lateral mucosa [42]. In AR, the more prevalent venous sinusoids in the medial and inferior mucosal layers exhibit the greatest increase in dimensions and contribute significantly to mucosal thickening [35, 41]. This is largely due to the response to inflammatory mediators, which increases the fluid and cellular in the inferior, medial, and lateral lamina propria [43]. In contrast to normal turbinates, those with

hypertrophy have a greater incidence of fibrotic lamina propria, most consistently in the inferior mucosal layer, along with a subepithelial inflammatory cell infiltrate [43]. The relative dimension of the serous glands in these turbinates also increases while the epithelium decreases [43]. These histological changes in turn lead to objective and subjective evidence of turbinate enlargement and obstruction seen in AR.

Response to Chronic Rhinosinusitis

Chronic rhinosinusitis (CRS) encompasses a similarly debilitating group of heterogeneous inflammatory disorders with prevalence estimates ranging from 2 to 27% worldwide [44–46]. It is an inflammatory disorder of the nose and paranasal sinuses that has noteworthy macroscopic and histological sequelae, including endoscopic findings of purulence, mucosal thickening, polyposis, and turbinate enlargement. CRS, as with allergic rhinitis, is frequently associated with bilaterally enlarged turbinates that contribute to airway obstruction and exacerbate symptoms of sinusitis.

Inflammatory cells and molecules again mediate the turbinate response in this disorder. Levels of neutrophils and T-cells increase in the lamina propria and epithelium, in addition to eosinophilic pro-inflammatory mediators such as LTC4, LTD4, LTE4, and ECP [35]. Nasal secretions typically show elevated levels of TNF-alpha, IL-1beta, IL-6, and IL-8 [35]. Kinins increase vascular permeability and venous engorgement, and IL-1beta promotes neutrophil adherence to the vascular endothelium, allowing for infiltration of the sinuses and progression of sinus inflammation [35, 47]. Airway inflammation activates membrane-associated tissue factor on the mucosa of the inferior turbinates and sinuses, leading to the downstream activation of the coagulation cascade. Thrombin, a byproduct of the coagulation cascade induces fibrin deposition and the secretion of vascular endothelial growth factor (VEGF) and platelet-derived growth factor (PDGF), is elevated in the in the epithelial cells of the inferior turbinate mucosa [34, 48]. Along with the eosinophil infiltration prevalence in the mucosa, these mediators lead to tissue remodeling with characteristic findings of epithelial soughing, subepithelial fibrosis, basement membrane thickening, goblet cell metaplasia, and mucosal cell hyperplasia [48]. Interestingly, inferior turbinates have demonstrated a significantly greater distribution of glands and fewer eosinophils than nasal polyps, suggesting a unique pathogenesis for each, despite the coexistence of both in CRS [49].

Mucus is normally secreted from epithelial goblet cells and submucosal acinar cells [50]. The density of acinar cells in the nose is at baseline significantly higher than in the maxillary or ethmoid sinuses, suggesting a disproportionate contribution of nasal secretions from the inferior turbinates. In CRS, the hyperplasia and hypertrophy of acinar, but not goblet, cells in the lamina propria indicate a source for the viscous hypersecretion seen in CRS [50]. Mucus from the maxillary sinus travels toward the ostium, then past the medial surface of the inferior turbinate to the nasopharynx. Thus, the medial surface of the inferior turbinate, like the middle turbinate and septum, is frequently exposed to inflammatory proteins and cells that alter its structure, cellular architecture, and gene expression [50]. Chronic inflammation additionally leads to poorly organized, dense, and elongated cilia with impaired architecture and function that may increase the risk of persistent disease or recurrence despite maximal medical therapy [31]. Together with increased mucus viscosity, the altered cilia contribute to impaired mucociliary clearance seen in CRS [51].

Response to Septal Deviation

Septal deviation is estimated to occur in up to 20% of school-aged children [52]. A straight septum permits laminar flow of inspired air over the turbinates to be cleaned, warmed, and humidified [52]. While asymptomatic to many, septal deviation may affect nasal respiration and lead to symptomatic obstruction, requiring septoplasty. Objective findings are often visible on anterior rhinoscopy, endoscopy, and radiologic imaging [53]. Unlike the response seen with AR or CRS, the structural, rather than inflammatory, etiology of septal deviations minimizes the effect on the mucosa, cells, and inflammatory mediators, and there is no impairment of mucociliary clearance [51]. Most strikingly, septal deviations are associated with unilateral, rather than bilateral, turbinate enlargement, and may be present regardless of the degree of septal deviation [53].

Because the narrowest portion of the nose spans lies anteriorly from the nostrils to the aperture pyriformis, any septal deviation at this location has a significant effect on airflow [54]. The contralateral nasal passageway consequently experiences an increase in airflow that leads to drying and crusting [54]. While some suggest that contralateral turbinate hypertrophy is a compensatory response to limit excessive airflow, others argue for a congenital etiology [55, 56]. Aslan et al. found that while the cross-sectional area of the IT bone in adults with septal deviation was greater than that in adults without septal deviation, no difference in the IT cross-sectional area was noted between children with and without septal deviation [56]. Furthermore, the cross-sectional area of the IT bone was greater in adults than in children. Taken together, these findings favor a compensatory mechanism for both bony and mucosal turbinate hypertrophy [56]. Septoplasty has also been shown to increase

ipsilateral and decrease contralateral IT mucosal thickness, suggesting both a compensatory etiology of the hypertrophy and its natural reversibility [57].

In normal inferior turbinates, the mean thickness of the medial mucosal layer is greatest, followed by the thickness of the bone, then the lateral mucosal layer [42]. CT and histological analyses have verified that both bone and mucosa enlarge with septal deviations, unlike the primarily mucosal thickening seen in allergic rhinitis and CRS, both bone and mucosa enlarge with septal deviations [8, 55, 58]. The bony dimensions have been shown to increase significantly, at a rate of twofold in one study [43]. While there is fairly widespread agreement that the changes seen in the bone and mucosa vary throughout the inferior turbinate, controversy exists as to which segments of the turbinate experience the greatest increase in bony and mucosal dimensions. Estomba et al. found evidence of bone and mucosa enlargement anteriorly, mucosa medially, and bone posteriorly [59]. Uzun et al. showed evidence of bony hypertrophy in the anterior and middle segments, and mucosal hypertrophy posteriorly [60]. Adding to the complexity and confusion, changes in the bone and mucosa also differ depending on the cause of septal deviation, with bone hypertrophy more common in congenital deviations and mucosal thickening in traumatic deviations [30]. These differences have important clinical implications as the sites of bone and mucosal enlargement dictate the most appropriate and precise surgical intervention for each pathologic turbinate.

Conclusion

The inferior turbinate plays a major role in the function in the nose. Overall, physicians who manage the inferior turbinate are starting to appreciate these roles and are balancing the risks and benefits with respect to the management of the inferior turbinate.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
• Of importance
•• Of major importance

1. Bingham B, Wang RG, Hawke M, Kwok P. The embryonic development of the lateral nasal wall from 8 to 24 weeks. Laryngoscope. 1991;101(9):992–7.
2. Arredondo de Arreola G, López Serna N, de Hoyos Parra R, Arreola Salinas MA. Morphogenesis of the lateral nasal wall from 6 to 36 weeks. Otolaryngol Head Neck Surg. 1996;114(1):54–60.
3. Scott JR, Psaltis AJ, Wormald PJ. Vascular anatomy of the inferior turbinate and its clinical implications. Am J Rhinol Allergy. 2020;34(5):604–9.
4. Padgham N, Vaughan-Jones R. Cadaver studies of the anatomy of arterial supply to the inferior turbinates. J R Soc Med. 1991;84(12):728–30.
5. Eren E, Zeybek G, Ecevit C, Arslanoğlu S, Ergur I, Kiray A. A new method of identifying the posterior inferior nasal nerve: implications for posterior nasal neurectomy. J Craniofac Surg. 2015;26(3):930–2.
6. Bleier BS, Schlosser RJ. Endoscopic anatomy of the postganglionic pterygopalatine innervation of the posterolateral nasal mucosa. Int Forum Allergy Rhinol. 2011;1(2):113–7.
7. Berger G, Balum-Azim M, Ophir D. The normal inferior turbinate: histomorphometric analysis and clinical implications. Laryngoscope. 2003;113(7):1192–8.
8. Egeli E, Demirci L, Yazýcý B, Harputluoglu U. Evaluation of the inferior turbinate in patients with deviated nasal septum by using computed tomography. Laryngoscope. 2004;114(1):113–7.
9. Brunworth J, Holmes J, Sindwani R. Inferior turbinate hypertrophy: review and graduated approach to surgical management. Am J Rhinol Allergy. 2013;27(5):411–5.
10. Stewart MG, Smith TL, Weaver EM, Witsell DL, Yueh B, Hannley MT, et al. Outcomes after nasal septoplasty: results from the Nasal Obstruction Septoplasty Effectiveness (NOSE) study. Otolaryngol Head Neck Surg. 2004;130(3):283–90.
11. Uzun L, Ugur MB, Savranlar A, Mahmutyazicioglu K, Ozdemir H, Beder LB. Classification of the inferior turbinate bones: a computed tomography study. Eur J Radiol. 2004;51(3):241–5.
12. Yang BT, Chong VF, Wang ZC, Xian JF, Chen QH. CT appearance of pneumatized inferior turbinate. Clin Radiol. 2008;63(8):901–5.
13. Segal RA, Kepler GM, Kimbell JS. Effects of differences in nasal anatomy on airflow distribution: a comparison of four individuals at rest. Ann Biomed Eng. 2008;36(11):1870–82.
14. Yu S, Liu Y, Sun X, Li S. Influence of nasal structure on the distribution of airflow in nasal cavity. Rhinology. 2008;46(2):137–43.
15. Xiong GX, Zhan JM, Jiang HY, Li JF, Rong LW, Xu G. Computational fluid dynamics simulation of airflow in the normal nasal cavity and paranasal sinuses. Am J Rhinol. 2008;22(5):477–82.
16. Chung SK, Son YR, Shin SJ, Kim SK. Nasal airflow during respiratory cycle. Am J Rhinol. 2006;20(4):379–84.
17. •• Zhao K, Jiang J. What is normal nasal airflow? A computational study of 22 healthy adults. Int Forum Allergy Rhinol. 2014;4(6):435–46. **Findings from this study describe the variations of nasal airflow in healthy subjects. On average, the flow is higher in the middle meatus region than the inferior meatus.**
18. Lindemann J, Keck T, Wiesmiller K, Sander B, Brambs HJ, Rettinger G, et al. A numerical simulation of intranasal air temperature during inspiration. Laryngoscope. 2004;114(6):1037–41.
19. Lindemann J, Keck T, Wiesmiller K, Sander B, Brambs HJ, Rettinger G, et al. Nasal air temperature and airflow during respiration in numerical simulation based on multislice computed tomography scan. Am J Rhinol. 2006;20(2):219–23.
20. Chen XB, Leong SC, Lee HP, Chong VF, Wang DY. Aerodynamic effects of inferior turbinate surgery on nasal airflow—a computational fluid dynamics model. Rhinology. 2010;48(4):394–400.
21. • Naftali S, Rosenfeld M, Wolf M, Elad D. The air-conditioning capacity of the human nose. Ann Biomed Eng. 2005;33(4):545–53. **This study provides a detailed analysis of the relative contribution of endonasal structural components to the air-conditioning process.**

22. Stoksted P. Rhinometric measurements for determination of the nasal cycle. Acta Otolaryngol Suppl. 1953;109:159–75.
23. Williams M, Eccles R. A model for the central control of airflow patterns within the human nasal cycle. J Laryngol Otol. 2016;130(1):82–8.
24. Costa DJ, Sanford T, Janney C, Cooper M, Sindwani R. Radiographic and anatomic characterization of the nasal septal swell body. Arch Otolaryngol Head Neck Surg. 2010;136(11):1107–10.
25. Eccles R. A role for the nasal cycle in respiratory defence. Eur Respir J. 1996;9(2):371–6.
26. Rohrmeier C, Schittek S, Ettl T, Herzog M, Kuehnel TS. The nasal cycle during wakefulness and sleep and its relation to body position. Laryngoscope. 2014;124(6):1492–7.
27. Kimura A, Chiba S, Capasso R, Yagi T, Ando Y, Watanabe S, et al. Phase of nasal cycle during sleep tends to be associated with sleep stage. Laryngoscope. 2013;123(8):2050–5.
28. Persson CG, Erjefält I, Alkner U, Baumgarten C, Greiff L, Gustafsson B, et al. Plasma exudation as a first line respiratory mucosal defence. Clin Exp Allergy. 1991;21(1):17–24.
29. Rohrich RJ, Krueger JK, Adams WP, Jr., Marple BF. Rationale for submucous resection of hypertrophied inferior turbinates in rhinoplasty: an evolution. Plast Reconstr Surg. 2001;108(2):536–44; discussion 45–6.
30. Korkut AY, Islim F, Gulseven Ciftci S, Dogan R, Gedikli O, Kahya V, et al. Evaluation of inferior turbinate hypertrophy in patients with congenital and traumatic nasal septum deviation. J Laryngol Otol. 2012;126(8):784–8.
31. Li YY, Li CW, Chao SS, Yu FG, Yu XM, Liu J, et al. Impairment of cilia architecture and ciliogenesis in hyperplastic nasal epithelium from nasal polyps. J Allergy Clin Immunol. 2014;134(6):1282–92.
32. Lunder M, Baricevic J, Lunder M, Planinsic I. Our experiences with immunosuppressive therapy. Med Arh. 1975;29(2):101–5.
33. Hilberg O, Grymer LF, Pedersen OF, Elbrønd O. Turbinate hypertrophy. Evaluation of the nasal cavity by acoustic rhinometry. Arch Otolaryngol Head Neck Surg. 1990;116(3):283–9.
34. Bousquet J, Khaltaev N, Cruz AA, Denburg J, Fokkens WJ, Togias A, et al. Allergic rhinitis and its impact on asthma (ARIA) 2008 update (in collaboration with the World Health Organization, GA[2]LEN and AllerGen). Allergy. 2008;63(Suppl 86):8–160.
35. Farmer SE, Eccles R. Chronic inferior turbinate enlargement and the implications for surgical intervention. Rhinology. 2006;44(4):234–8.
36. Bousquet PJ, Leynaert B, Neukirch F, Sunyer J, Janson CM, Anto J, et al. Geographical distribution of atopic rhinitis in the European Community Respiratory Health Survey I. Allergy. 2008;63(10):1301–9.
37. Asher MI, Montefort S, Björkstén B, Lai CK, Strachan DP, Weiland SK, et al. Worldwide time trends in the prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and eczema in childhood: ISAAC Phases One and Three repeat multicountry cross-sectional surveys. Lancet. 2006;368(9537):733–43.
38. Ricciotti E, FitzGerald GA. Prostaglandins and inflammation. Arterioscler Thromb Vasc Biol. 2011;31(5):986–1000.
39. Borish L. Allergic rhinitis: systemic inflammation and implications for management. J Allergy Clin Immunol. 2003;112(6):1021–31.
40. Christodoulopoulos P, Cameron L, Durham S, Hamid Q. Molecular pathology of allergic disease. II: Upper airway disease. J Allergy Clin Immunol. 2000;105(2 Pt 1):211–23.
41. Ardehali MM, Amali A, Bakhshaee M, Madani Z. Histopathologic characteristics of inferior turbinate vs ethmoidal polypin chronic rhinosinusitis. Ann Diagn Pathol. 2011;15(4):233–6.
42. Mangla JC, Desbaillets LG. Endoscopic removal of chicken gizzards by polypectomy snare in a patient with Nissen's fundoplication. Am J Gastroenterol. 1975;64(2):133–6.
43. Berger G, Gass S, Ophir D. The histopathology of the hypertrophic inferior turbinate. Arch Otolaryngol Head Neck Surg. 2006;132(6):588–94.
44. Shashy RG, Moore EJ, Weaver A. Prevalence of the chronic sinusitis diagnosis in Olmsted County. Minnesota Arch Otolaryngol Head Neck Surg. 2004;130(3):320–3.
45. Blackwell DL, Collins JG, Coles R. Summary health statistics for U.S. adults: National Health Interview Survey, 1997. Vital Health Stat 10. 2002(205):1–109.
46. Hastan D, Fokkens WJ, Bachert C, Newson RB, Bislimovska J, Bockelbrink A, et al. Chronic rhinosinusitis in Europe—an underestimated disease. A GA^{2}LEN study. Allergy. 2011;66(9):1216–23.
47. Itoh K, Katahira S, Matsuzaki T, Ohyama M, Fukuda K. Retention fluids of chronic sinusitis induce neutrophil adherence to microvascular endothelial cells. Acta Otolaryngol. 1992;112(5):882–9.
48. Shimizu S, Ogawa T, Takezawa K, Tojima I, Kouzaki H, Shimizu T. Tissue factor and tissue factor pathway inhibitor in nasal mucosa and nasal secretions of chronic rhinosinusitis with nasal polyp. Am J Rhinol Allergy. 2015;29(4):235–42.
49. Nguyen KH, Suzuki H, Wakasugi T, Hohchi N, Hashida K, Ohbuchi T. Different expressions of erbB1/2 and tight junction proteins in hypertrophic inferior turbinates and nasal polyps. Eur Arch Otorhinolaryngol. 2013;270(3):945–51.
50. Majima Y, Masuda S, Sakakura Y. Quantitative study of nasal secretory cells in normal subjects and patients with chronic sinusitis. Laryngoscope. 1997;107(11 Pt 1):1515–8.
51. Passàli D, Ferri R, Becchini G, Passàli GC, Bellussi L. Alterations of nasal mucociliary transport in patients with hypertrophy of the inferior turbinates, deviations of the nasal septum and chronic sinusitis. Eur Arch Otorhinolaryngol. 1999;256(7):335–7.
52. Aziz T, Biron VL, Ansari K, Flores-Mir C. Measurement tools for the diagnosis of nasal septal deviation: a systematic review. J Otolaryngol Head Neck Surg. 2014;43(1):11.
53. Grymer LF, Illum P, Hilberg O. Septoplasty and compensatory inferior turbinate hypertrophy: a randomized study evaluated by acoustic rhinometry. J Laryngol Otol. 1993;107(5):413–7.
54. Grymer LF, Hilberg O, Elbrønd O, Pedersen OF. Acoustic rhinometry: evaluation of the nasal cavity with septal deviations, before and after septoplasty. Laryngoscope. 1989;99(11):1180–7.
55. Akoğlu E, Karazincir S, Balci A, Okuyucu S, Sumbas H, Dağli AS. Evaluation of the turbinate hypertrophy by computed tomography in patients with deviated nasal septum. Otolaryngol Head Neck Surg. 2007;136(3):380–4.
56. Aslan G, Uzun L, Ugur MB, Uckurt YS, Sagun OF, Yazicilar O. Unilateral inferior turbinate bone hypertrophy: is it compensatory or congenital? Am J Rhinol Allergy. 2013;27(4):255–9.
57. Kim DH, Park HY, Kim HS, Kang SO, Park JS, Han NS, et al. Effect of septoplasty on inferior turbinate hypertrophy. Arch Otolaryngol Head Neck Surg. 2008;134(4):419–23.
58. Orhan I, Aydın S, Ormeci T, Yılmaz F. A radiological analysis of inferior turbinate in patients with deviated nasal septum by using computed tomography. Am J Rhinol Allergy. 2014;28(1):e68-72.
59. Chiesa Estomba C, Rivera Schmitz T, Ossa Echeverri CC, Betances Reinoso FA, Osorio Velasquez A, Santidrian HC. Compensatory hypertrophy of the contralateral inferior turbinate in patients with unilateral nasal septal deviation. A computed tomography study Otolaryngol Pol. 2015;69(2):14–20.
60. Uzun L, Savranlar A, Beder LB, Ugur MB, Cinar F, Ozdemir H, et al. Enlargement of the bone component in different parts of compensatorily hypertrophied inferior turbinate. Am J Rhinol. 2004;18(6):405–10.