The Role of Rhinitis in Chronic Otitis Media

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Non-infectious rhinitis is a chronic inflammatory disease of the nasal mucosa, characterized by obstruction, sneezing, pruritus and hypersecretion. These clinical findings are due to hypersensitivity of the immune system and nervous phenomena occurring in the mucous membrane. The etiology may be allergic or non-allergic, depending whether the individual has a type I immunologic response or not.

Allergic rhinitis affects a large amount of the population. Recent studies show that 10 to 30% of the American population has allergic rhinitis. According to Vining, 40 to 50 million of Americans have rhinitis. In São Paulo, Brazil, these numbers are even higher, due to its unique atmospheric conditions and pollution.

In allergic rhinitis, after the contact of the nasal mucosa to a specific antigen, mediators like histamine, leukotriens, kinines and cytokines are released by mucosal mast cells increasing vascular permeability, mucosal blood flow and mucous production. They also contribute to the attraction and activation of eosinophils, which leads to an inflammatory response (allergic inflammation). These aspects are responsible for the clinical findings of the disease.

Regarding non-allergic rhinitis, this entity is considered a diagnosis of exclusion, due to the various etiologies of non-allergic inflammatory mucosa, like non-allergic rhinitis eosinophilia (NARES), and non-allergic non-infectious perennial rhinitis (NANIPER). The first is sometimes associated with eosinophilic nasal poliposis and even asthma, and the latter is a form of persistent rhinitis with no clear etiology.

Otitis media has multifactorial causes. Contributing factors to the genesis of otitis media are eustachian tube dysfunction, bacterial or viral infections of the middle ear, nasal inflammation resulting from allergic rhinitis or upper airway infections.

Some risk factors for otitis media (acute or chronic) have been pointed: upper airways viral infections, allergic rhinitis, Eustachian tube dysfunction, cigarette smoking, bottle feed, male sex, immunologic deficiency, ciliary dysfunction, cleft palate disease, genetic predisposition.

The role of rhinitis in middle ear disease is very controversial. The reported incidence of patients with OME and allergy varies from 5 to 80%, usually around 23%. Although there is clinical evidence of nasal inflammatory disease affecting Eustachian tube function in the middle ear mucosa, there is no scientific evidence

to prove that fact. In 1931, Proetz noted a relationship between patients with allergic and chronic otitis media. Koch's study of 222 patients was the first to include observations of eosinophilia in otorrhea "supporting the contention that the middle ear takes part in allergic reactions similar to those seen in the nose and sinuses". Shambaugh suspected allergy as an etiology, reporting empiric data issuing the following caution: "Surgical mastoidectomy, simple or radical, is not indicated. With competent allergic diagnosis and management, preferably by an otologist trained in allergic methods, the otorrhea is finally brought under control".

Mion et al found about 50% of patients with chronic otitis media presenting nasal disease and nasal eosinophilia (33,33% of allergic and 15,69% of NARES), showing a slight increase in nasal pathology compared to other reports. The conclusion was that nasal disease has an impact on otologic middle ear disease, considering that the normal nasal mucosa do not have eosinophils.

In the genesis of chronic otitis media, rhinitis can be related by two ways: the Eustachian tube dysfunction caused by the allergic reaction in the nasal mucosa and the decrease of the ciliary beat frequency. According to Bernstein, there are three possibilities leading the inflammatory reaction to block the Eustachian tube. The dysfunction can represent retrograde extension of nasal mucosal edema and congestion; the mucociliary activity could cause the secretion to cover the ostium and lead to intraluminal inflammation. Many of the substances released are known to cause hypersecretion. They could also stimulate the seromucinous glands of the Eustachian tube to hypersecrete, obstructing the lumen.

Although protection of the middle ear from nasopharyngeal secretions and mucociliary clearance of the middle ear are known to be two important functions of the Eustachian tube, perhaps the most critical function of the Eustachian tube is the replacement of respiratory gases into the middle ear cleft to maintain atmospheric pressure in the middle ear. Disruption of the mechanism for middle ear pressure regulation is associated with pathophysiologic changes including the developement of significant low pressures and, if prolonged, otitis media with effusion.

The Eustachian tube offers a potential pathway for antigen access to the middle ear. Although most clinical studies did not show elevation of middle ear IgE levels, Bernstein's early studies suggested that about 23% of allergic patients with otitis media with effusion may have had a local allergic reaction within the middle ear. Most clinical and experimental studies using specific antigen or inflammatory mediators have failed to demonstrate the development of middle ear effusion despite the presence of Eustachian tube dysfunction. Therefore, it is likely that other factors, including bacterial and viral infections involving the nasopharyngeal area, contribute to the development of middle ear fluid and middle ear infection.

Forty to fifty percent of the children with otitis media with effusion have allergic rhinitis diagnosed with positive skin prick test and specific IgE.

High levels of histamine are present in middle ear secretions in allergic patients, even though the treatment with oral antihistamines has not shown significant difference when compared allergic and non-allergic patients with otitis media with effusion. This data confirms once more the complex nature of these diseases.

The level of cellular inflammatory components is also involved. Mast cells degranulation mainly triptase, is found in higher levels in the middle ear secretions of allergic children with otitis media with effusion. Eosinophils degranulation of eosinophilic cationic protein is present in 87% of patients with otitis media with effusion.

Eosinophils penetrate the nasal mucosa through adhesion and diapedesys. Respiratory viruses also penetrate the respiratory mucosa the same way. The expression of the endothelial adhesion molecules, specially ICAM-1, during allergic rhinitis and NARES, can facilitate viral infections of the upper airway, including the middle ear.

Tobacco smoking has an important role in chronic otitis media as well as in allergy. Among the risk factors for upper respiratory tract infections are passive smoking, low income and child care. Atopy was confirmed in 35% to 38% of the children with upper respiratory tract infections. Adenoid tissue removed from tobacco exposed children showed increased thickness in histopathology.

However, authors cited by several investigators found no clinical or pathologic basis for allergy in the etiology of otitis media with effusion. According to Bernstein, the relationship between allergy and otitis media with effusion will remain controversial until well controlled clinical studies are conducted documenting that in select populations therapy for allergic conditions is efficacious in preventing or limiting the duration of otitis media with effusion.

Nevertheless, we must always suspect of nasal inflammatory disease in patients with chronic otitis media who are refractory to treatment or do not respond accordingly after appropriate surgery.

Recommended readings

1. Fireman, P. Otitis media and Eustachian tube disfunction: conection to allergic rhinitis. J Allergy Clin Immunol. 1997; 99: S787-797.
2. Mion, O. et al. The Role of Rhinitis in Chronic Otitis Media. Otolaryngol Head Neck Surg, Volume 128(1): 27-31, January 2003.
3. Bousquet J et al. The Workshop Expert Panel. Allergic rhinitis and its impact on asthma.In collaboration with the World Health Organization.Executive Summary of the Workshop Report,7 –10 December 1999, Geneva, Switzerland. Allergy 2002; 57 :841 –855.