Eosinophile tissue infiltration as predictor in nasosinus mucous membrane disease

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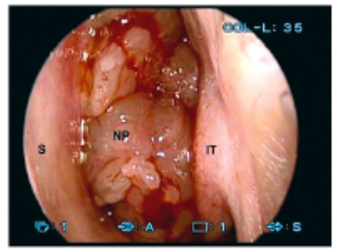
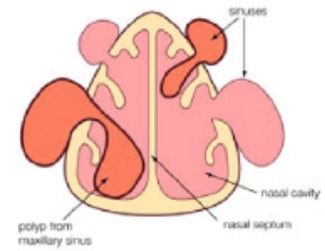
Allergic rhinitis affects one in 4 people worldwide. It is commonest atopic disease in human population and its prevalence is on the rise.

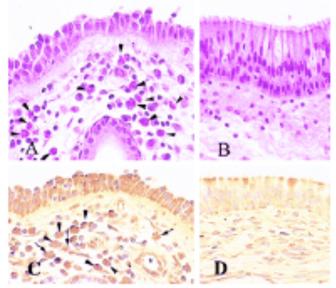




It is a type I allergic reaction mediated by mast cell bound IgE characterized by proliferation of pseudostratified respiratory epithelium, stromal oedema, thickening of basal membrane, fibrosis and inflammatory infiltration of lamina propria. Over time proliferation of stroma results in polyp formation.

Proliferation mainly originates from respiratory mucosa covering ostiomeatal complex of ethmoid bones and descend into nasal cavity through middle meatus causing symptoms of obstruction, anosmia, rhinorhoea, fullness of ears etc.

Aetiology is still unclear but stresses such bacterial, fungal and viral infection, environmental pollution are to blame. Those coincide with downregulation of TLR involved in immune response.



Unlike allergic rhinitis, nonallergic rhinitis does not involve the immune system. Often, what causes nonallergic rhinitis is unknown. And the condition is often confirmed only after other conditions such as allergic rhinitis or infection are ruled out.

Environmental irritants are common triggers of nonallergic rhinitis. Some are found in the home and others are more common in the workplace.

Common in any form of all allergic nasosinus mucous sinus membrane disease is tissue eosinophilia whose secretions (vasoactive substances, ECP, EPO, cytokines, chemotactic factors) are responsible for majority of symptoms.

Aim of this cross sectional study was (a) to investigate if number of tissue eosinophiles is in correlation with severity of disease and its propensity to earlier relapse and (b) to compare tissue eosinophilia between patents with allergic rhinitis and and non allergic disease. Study included 40 patients, 33 with allergic rhinitis (with and without nasal polyposis) and 7 non allergic rhinitis as control group. All patients gave written informed consent according to Helsinki Declaration.

Allergy was determined by measuring total IgE by ELISA kit and ELISA reader in collected venous blood.

Presence and number of tissue eosinophiles in nasal mucosa and or polyps was analyzed under 400x magnigication and counting was performed on 10 non covering fields. Staging was done according to number of eosinophiles into 3 stages (1. <10 eopf 2.10-20eopf 3.>20eopf*) Eosinophiles per field

Disease type	Non allergic disease	Allergic rhinitis	Atopic nasal polyposis
Eosinophile count in nasal mucosa	8,99 ± 7,44	15,34 ± 2,41	56,35 ± 3,94

Table 1. Eosinophile count in tissue samples

There is positive correlation between tissue eosinophilia and type of disease. Also, more progressed the disease (with formation of polyps) larger the tissue eosinophilia.

	Total	Group 1	Group 2	Group 3
Mean age/std. dev.	41,45± 16,32	38,24±19,35	43,34±18,74	40,98±17,33
Men/women	32/8	14/4	8/3	10/1
Atopy*	33	12	13	8
Total serum IgE/IU ml		298±33,71	311,93±39,41	369±42,98
Positive prick test	100% (33/33)	100% (12/12)	100% (13/13)	100% (8/8)
Relapse **	25	3	9	13

Table 2 Patient group profile allergic rhinitis

*allergy established by cutaneous prick test to one or more inhalatory allergens and presence of symptoms for at least a month
**1 year follow up

There is significant increase of disease relapse in patient whose tissue samples show marked eosinophilia (group 2. and 3.) Greater tissue eosinophila warrants higher relapse rate.

	Total
Mean age	7
Men/women	4/3
Positive skin prick test	0%
Total serum IgE/IU ml	43,65±22,37
Relapse*	100%

Table 3. Patient group non allergic disease

*Surgery is not an option for such patients and only biopsy was performed

Keith P, Dolovich J. Allery and nasal polyps (1997) In: Mygind N, Lildholdt T. Nasal polyposis. An inflammatory disease and its treatment. Eds Munksgaard, Copenhagen pp. 68-77 Pawliczak R, Kowalski ML, Danilewicz M, Danilewicz MW, Lewandowski A (1997) Distribution of mast cells and eosinophils in nasal polyps from atopic and nonatopic subjects: a morphometric study. Am J Rhinology 11: 257-262, Sorensen H, Mygind N, Tygstrup I, Felnsborg EW (1977) Histology of nasal polyps of different etiology. Rhinology 25: 121- 128. Stopp AE, Van Der Heijden H, Biewenga J, Van Der Baan S(1993) Eosinophils in nasal polyps and nasal mucosa : an immunohistochemical study. J Allergy Clin Immunol 91: 616-622.