



## ORIGINAL ARTICLE

## Study of Relationship of Serum and Tissue Eosinophilia in Patients with Allergy

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## ABSTRACT

**Introduction and Aim:** In ENT clinical practice, allergy is a common presentation. Allergic rhinitis is an Ig E- mediated allergic reaction and cause of inflammatory reaction often associated with adenotonsillar disease, sinusitis. **Materials and Methodology:** In the Department of ENT, patients between 5-60 years of age presenting with or without symptoms of allergy and diagnosed with adenoid/ adenotonsillar hypertrophy, turbinate hypertrophy, nasal polyp, undergoing surgery had clinical and laboratory assessment, primarily consisting of serum IgE, AEC, DNE, tissue eosinophil count. Then relationship of serum eosinophilia and tissue eosinophilia analyzed in patients with and without allergy. **Results:** In patients, aged between 5-60 years included during the study period, High serum IgE levels are seen in 27.5% of the people without allergy and is higher i.e. 51.0% among people with allergy, which is statistically important ( $p=0.02$ ). Tissue eosinophilia was high overall is 22.5% among patients with no allergy, 51% in patients with allergy, this is statistically significant ( $p=0.01$ ). Among individuals with normal serum IgE levels, 41.5% had high tissue eosinophil counts, while among those with high serum IgE levels, 69.4% had high tissue eosinophil counts so this difference in proportion is statistically significant. There is positive association of Serum IgE, AEC and tissue eosinophilia with individuals with history of allergy. Association of AEC with serum IgE, Serum IgE with tissue eosinophilia and Tissue eosinophilia with AEC are not statistically significant. **Conclusion:** In our study, proportion of people with high Serum IgE, high AEC and high tissue eosinophilia is higher among those who have history of allergy as compared to non-allergic people. No significant association was found between high tissue eosinophilia and blood parameters (Serum IgE and AEC). So, tissue eosinophilia has to be assessed for all patients irrespective of Serum IgE and AEC estimates.

**Keywords:** Adenoid; AEC; Allergy; Asthma; Eosinophilia; Tonsil

## INTRODUCTION

Allergic diseases represent a significant global health burden, affecting millions of individuals worldwide<sup>1</sup>. These conditions encompass a broad spectrum of disorders, including allergic rhinitis, asthma, chronic rhinosinusitis with nasal polyps (CRSwNP), and atopic dermatitis, among others. A common pathological feature shared by these disorders is eosinophilic inflammation, which plays a pivotal role in disease pathogenesis and symptomatology<sup>2</sup>.

The assessment of eosinophilia in allergic diseases can be performed through serum eosinophil count and tissue eosinophil quantification. While peripheral blood eosinophil count provides a readily accessible biomarker for systemic eosinophilic inflammation, tissue eosinophilia is

considered a more direct measure of local inflammatory burden<sup>3</sup>. However, the relationship between serum and tissue eosinophilia remains incompletely understood. Some studies suggest a strong correlation between peripheral eosinophilia and disease severity, whereas others indicate discordance between circulating and tissue-resident eosinophils. This variability underscores the need for further investigation into the association between serum and tissue eosinophilia in allergic conditions<sup>4,5</sup>.

*Pathophysiology of Eosinophilia in Allergy*

Eosinophils are key effector cells in allergic inflammation, playing a crucial role in both innate and adaptive immune responses. In allergic rhinitis and asthma, eosinophils infil-

trate the nasal mucosa and bronchial epithelium, leading to mucosal oedema, increased mucus production, and airway hyperresponsiveness. In CRSwNP, eosinophil accumulation within the sinonasal mucosa is associated with polyp formation and chronic inflammation<sup>6,7</sup>. The persistence of eosinophils within tissues is mediated by survival-promoting cytokines, resulting in prolonged inflammatory responses even in the absence of ongoing allergen exposure. Understanding the mechanisms underlying eosinophilic infiltration and persistence is critical for elucidating the relationship between serum and tissue eosinophilia<sup>8,9</sup>.

### ***Serum Eosinophilia as a Biomarker***

Peripheral blood eosinophil count is frequently utilized as a biomarker for eosinophilic diseases. Elevated serum eosinophil levels have been reported in patients with allergic rhinitis, asthma, and CRSwNP, often correlating with disease severity and treatment response. A higher eosinophil count in the blood may reflect systemic Th2 inflammation and increased eosinophil trafficking to target tissues. However, serum eosinophilia alone may not always accurately represent local tissue inflammation. Some patients with severe tissue eosinophilia may exhibit normal or only mildly elevated peripheral eosinophil counts<sup>10,11</sup>.

### ***Tissue Eosinophilia in Allergic Diseases***

Allergic rhinitis which is an Ig E- mediated allergic reaction and cause of inflammatory reaction after allergen exposure is the most common disease in patients.

Histopathological examination of affected tissues / barrier provides valuable insights into the extent of eosinophilic inflammation in allergic diseases. In allergic rhinitis, eosinophils infiltrate the nasal mucosa, contributing to epithelial damage and mucosal hyperplasia. In CRSwNP, eosinophilic inflammation is a defining histopathological feature, with studies demonstrating a correlation between tissue eosinophil density and disease recurrence<sup>7,9</sup>.

Despite the clear role of tissue eosinophilia in disease pathogenesis, its relationship with serum eosinophilia remains variable.

### ***Clinical Implications***

Understanding the relationship between serum and tissue eosinophilia has significant clinical implications for the diagnosis, prognosis, and management of allergic diseases<sup>12</sup>, if a strong correlation exists, serum eosinophil count could serve as a non-invasive biomarker for disease severity and treatment response, reducing the need for invasive biopsies. However, if discordance is frequently observed, reliance on serum eosinophilia alone may be insufficient for guiding clinical decision-making<sup>13</sup>.

Biologic therapies targeting eosinophils, such as anti-IL-5 monoclonal antibodies (mepolizumab, reslizumab, benral-

izumab), have revolutionized the treatment of eosinophilic diseases<sup>14,15</sup>. These therapies selectively reduce eosinophil levels in circulation and tissues, improving clinical outcomes in patients with severe eosinophilic asthma and CRSwNP. Investigating the relationship between serum and tissue eosinophilia could provide insights into treatment response variability and help identify patients who may benefit most from targeted eosinophil-depleting strategies<sup>16</sup>.

## **MATERIALS AND METHODS**

The study setting was Department of Otorhinolaryngology (ENT) of a tertiary hospital in Dakshina Kannada district of Karnataka. Total duration of the study was 16-18 months. It is a cross-sectional study. Considering 95% confidence interval, the sample size estimated for study is 89. Purposive sampling technique was adopted.

### ***Eligibility criteria***

#### ***Inclusion criteria:***

- All patients, aged between 5 years and 60 years visiting the ENT Department of AJIMS&RC during the study period; diagnosed to have adenoid / adeno-tonsillar hypertrophy, turbinate hypertrophy, nasal polyp who required surgical intervention were included in the study.

#### ***Exclusion criteria***

- Patients diagnosed with Asthma / immunodeficiency conditions / auto-immune diseases / genetic syndromes were excluded from the study.
- Patients who did not consent to take part in the study were also excluded from the study.

### ***Data collection technique***

Interview based techniques were used to ensure eligibility criteria and then to obtain a detailed history of study participants. Data obtained secondarily from medical records department and laboratory / department of pathology were also used in the present study. They are as follows:

- Blood investigations for measurement of eosinophils that is Absolute Eosinophil counts (AEC), Serum IgE levels.
- Diagnostic nasal endoscopy, other necessary radiological investigations.
- Histopathological examination of tissue for eosinophils.

### ***Statistical analysis***

Data was entered in Microsoft Excel and was analysed using trial version of SPSS-22. Median and inter-quartile range (IQR) were used to represent measures of central tendency



as the continuous variables were not normally distributed. Proportions were used to summarise and understand the distribution of data. Appropriate graphical representations are used for easy understanding.

To understand the association between blood parameters of eosinophils and tissue eosinophils, chi-square test was used. To compare the difference between the medians across various grades of tissue eosinophilia, Mann-Whitney U test and Kruskal-Wallis test were used. Additionally, Spearman's rho correlation co-efficient was computed to determine the relationship between serum IgE levels and AEC. All the above tests of significance were considered as statistically significant if p value was less than 0.5.

### Ethical considerations

Data collection was initiated only after the process of obtaining ethical clearance from Institutional ethics committee. All the four principles of medical ethics were abided to, as per the researcher's best knowledge. Individual data was not shared at any level throughout the process of data dissemination.

### RESULTS

In patients, aged between 5-60 years included during the study period. From Table 1, it is evident that participants in age group <10years, 24.5% were found to have allergy, in 10-49years age group, 30% had allergy, and age group of 50 years and above, 14.3% participants had history of allergy showing no significant association between age and allergy.

High serum IgE levels are seen in 27.5% of the people without allergy and is higher i.e. 51.0% among people with allergy, which is statistically important ( $p=0.02$ ) which is evident from Table 2. In Table 3, it is evident that out of 49 participants with history of allergy, 33 participants (67.3%) had high AEC levels, showing statistically significant association between allergy and AEC.

Tissue eosinophilia was high overall is 22.5% among patients with no allergy, 51% in patients with allergy, this is statistically significant ( $p=0.01$ ) seen in Table 4. There is positive association of Serum IgE, AEC and tissue eosinophilia with individuals with history of allergy. Association of Tissue eosinophilia with AEC, Serum IgE with tissue eosinophilia are not statistically significant as seen in Tables 5 and 6 respectively.

### DISCUSSION

The current study aimed to evaluate serum IgE and Absolute Eosinophil Count (AEC) among participants and compare these parameters with varying grades of tissue eosinophilia in individuals diagnosed with adenoid or adeno-tonsillar hypertrophy, turbinate hypertrophy, and nasal polyps requiring surgical intervention. A sample of 89 subjects aged 5years to 60years was studied and the results

**Table 1: Distribution of study participants as per age**

Age group	No allergy	H/O Allergy	Total
< 10 years	13 (32.5%)	12 (24.5%)	25 (28.1%)
10 – 19 years	10 (25.0%)	7 (14.3%)	17 (19.1%)
20 – 29 years	6 (15.0%)	10 (20.4%)	16 (18.0%)
30 – 39 years	6 (15.0%)	6 (12.2%)	12 (13.5%)
40 – 49 years	3 (7.5%)	7 (14.3%)	10 (11.2%)
≥ 50 years	2 (5.0%)	7 (14.3%)	9 (10.0%)
<b>Total</b>	<b>40 (100.0%)</b>	<b>49 (100.0%)</b>	<b>89 (100.0%)</b>
Chi-square test: $\chi^2$ : 5.089; df: 5; p value: 0.41 (Not significant)			

**Table 2: Distribution of study participants as per Serum IgE levels**

Serum IgE	No allergy	H/O Allergy	Total
Normal	29 (72.5%)	24 (49.0%)	53 (59.6%)
High	11 (27.5%)	25 (51.0%)	36 (40.4%)
<b>Total</b>	<b>40 (100.0%)</b>	<b>49 (100.0%)</b>	<b>89 (100.0%)</b>
Chi-square test: $\chi^2$ : 5.058; df: 1; p value: 0.02 (Significant)			

**Table 3: Distribution of study participants as per AEC levels**

AEC	No allergy	H/O Allergy	Total
Normal	26 (65.0%)	16 (32.7%)	42 (47.2%)
High	14 (35.0%)	33 (67.3%)	47 (52.8%)
<b>Total</b>	<b>40 (100.0%)</b>	<b>49 (100.0%)</b>	<b>89 (100.0%)</b>
Chi-square test: $\chi^2$ : 9.246; df: 1; p value: 0.002 (Significant)			

**Table 4: Distribution of study participants as per Tissue Eosinophilic counts**

Tissue EC	No allergy	H/O Allergy	Total
Low	31 (77.5%)	24 (49.0%)	55 (61.8%)
Moderate	4 (10.0%)	7 (14.3%)	11 (12.4%)
High	5 (12.5%)	18 (36.7%)	23 (25.8%)
<b>Total</b>	<b>40 (100.0%)</b>	<b>49 (100.0%)</b>	<b>89 (100.0%)</b>
Chi-square test: $\chi^2$ : 8.231; df: 2; p value: 0.01 (Significant)			

**Table 5: Association between Tissue EC and Absolute EC levels**

Tissue Eosinophils	Absolute Eosinophil counts		Chi-square test
	Normal	High	
Low	29 (69.0%)	26 (55.3%)	$\chi^2$ : 2.111; df: 2; p value: 0.34 (Not significant)
Moderate	5 (11.9%)	6 (12.8%)	
High	8 (19.0%)	15 (31.9%)	

**Table 6: Association between Tissue EC and Serum IgE levels**

Tissue Eosinophils	Serum IgE levels		Chi-square test
	Normal	High	
Low	37 (69.8%)	18 (50.0%)	$\chi^2$ : 3.943; df: 2; p value: 0.14 (Not significant)
Moderate	6 (11.3%)	5 (13.9%)	
High	10 (18.9%)	13 (36.1%)	



found are discussed as below:

### **Baseline comparisons**

In the present study, peak prevalence was seen in children <10 years (28.1%), next peak in adolescents (19.1% aged 10–19 years) and only 10% aged >50 years. This paediatric predominance is seen in many other studies where lymphoid tissue hypertrophy especially adenoid hypertrophy peaks at the age of 6 years and atrophies after the age of 12 years<sup>17–19</sup>. Another study by Rout *et al.*, reported 21% of adult nasal obstructions from adenoid hypertrophy, primarily in 16–25-year-olds (60%)<sup>20</sup>. This suggests adult-onset hypertrophy may involve distinct triggers (e.g., pollution, chronic infection). With respect to gender, male predominance seen at 67% in the present study. Study conducted by Rout *et al.* found 70% of male predominance<sup>21</sup>. Other studies also show similar findings suggesting environmental / gender-specific risk factors amplify male susceptibility<sup>17</sup>. Also, male predominance warrants research into androgen-driven lymphoid proliferation or exposure biases<sup>22</sup>. With respect to demographics, other studies<sup>17–20</sup> linked adult hypertrophy to pollution / occupation (e.g., roadside workers, construction site workers etc.), which may affect all demographics uniformly. These aspects could be explored in future studies. This demographic alignment with pediatric-focused studies validates the present study's cohort's representativeness, while adult disparities underscore the condition's evolving epidemiology. The uniform age / sex distribution across allergic / non-allergic subgroups suggests environmental or systemic drivers (e.g., IL-6/CRP elevation) may transcend traditional atopic pathways.

### **Laboratory findings across the groups with respect to allergy**

In the present study, high serum IgE levels were observed in 51.0% of allergic individuals compared to 27.5% of non-allergic individuals. A study by Agha *et al.* found significantly elevated serum IgE levels in patients with allergic disorders (e.g., asthma, allergic rhinitis, urticaria) compared to healthy controls ( $P < 0.001$ ). The mean IgE levels were higher in allergic individuals across all age groups, consistent with our findings<sup>23</sup>. Similarly, a longitudinal study reported that persistent serum IgE levels  $\geq 200$  kU/L were strongly associated with allergic rhinitis, asthma, and eczema during childhood<sup>24</sup>. This difference was statistically significant, reinforcing the role of serum IgE as a biomarker for allergic conditions. Overlap between allergic and non-allergic groups limits the standalone diagnostic utility of these markers.

In the present study, high AEC was observed in 67.3% of allergic individuals compared to 35.0% of non-allergic individuals. Elevated AEC is commonly linked to allergic conditions such as asthma and rhinitis. A study on

eosinophilia found that allergic patients frequently exhibit peripheral eosinophilia due to IL-5-mediated recruitment and activation of eosinophils<sup>24,25</sup>. This difference was statistically significant, highlighting the role of eosinophilia as a marker of Th2-mediated inflammation.

Overall, the combination of serum IgE and AEC improves diagnostic accuracy for allergic diseases compared to either marker alone.

### **Histopathological findings across the groups with respect to allergy**

In the present study, a significantly higher proportion of allergic individuals exhibited high (overall: 51% i.e. more than half; high: 36.7% and moderate: 14.3%) tissue eosinophilia compared to non-allergic individuals. This statistically significant difference underscores the role of eosinophils as a hallmark of Th2-mediated inflammation in allergic conditions. Another study aligns with the findings of the present study; it was a study on children undergoing adenotonsillectomy which found significantly higher tissue eosinophil counts in atopic patients compared to non-atopic patients<sup>26</sup>. Research activities on CRSwNP patients reported elevated tissue eosinophilia in allergic individuals, correlating with type 2 cytokines like IL-5 and eotaxins. These cytokines drive eosinophil recruitment and activation, consistent with the higher levels observed in your allergic group<sup>27</sup>.

### **Association between Laboratory findings and Histopathological findings**

In the present study, the observation that 31.9% of individuals with high AEC exhibited high tissue eosinophilia compared to 19.0% of those with normal AEC suggests a possible correlation between systemic eosinophilia and localized tissue inflammation. High tissue eosinophilia was seen in 36.1% of individuals with high serum IgE, compared to 18.9% in those with normal serum IgE levels. Studies have shown that elevated AEC often correlates with increased tissue eosinophilia in allergic conditions such as asthma and rhinitis. For instance, a study found that patients with asthma had significantly higher tissue eosinophil counts when their AEC was elevated, suggesting that systemic eosinophilia reflects ongoing local inflammation<sup>28</sup>. Conversely, some studies report cases where patients exhibit high tissue eosinophilia despite normal AEC levels, indicating that local factors (e.g., cytokine milieu) can drive eosinophilic infiltration independent of systemic counts<sup>28,29</sup>.

The findings in present study highlight the complexity of eosinophilic responses, where local tissue environments can modulate eosinophil activity regardless of systemic levels. This aligns with research indicating that local production of chemokines (e.g., eotaxins) can attract eosinophils to tissues even when peripheral counts are normal. Similarly,



the presence of high serum IgE does not always guarantee high tissue eosinophilia; some patients may have elevated IgE levels without significant eosinophilic infiltration due to factors such as desensitization or variations in individual immune responses.

The observed trends highlight the importance of considering both AEC and serum IgE levels when evaluating patients for allergic conditions or eosinophilic disorders. However, clinicians should be cautious about relying solely on these markers for diagnosis or treatment decisions due to their variability. Understanding the relationship between these markers can guide personalized treatment strategies for patients with allergic diseases or conditions characterized by eosinophilic inflammation. For example, patients exhibiting high tissue eosinophilia despite normal systemic markers might benefit from localized therapies targeting the inflammatory process.

Given the complexity observed in the relationships among AEC, serum IgE, and tissue eosinophilia, it is crucial for clinicians to conduct comprehensive evaluations that include clinical history, physical examination, and possibly additional testing (e.g., allergen-specific tests) to accurately assess the underlying pathologies.

## LIMITATIONS OF STUDY

Before concluding, it is vital to mention that the study was well structured within the limits of study setting and duration. Despite this, the study may have few constraints which are listed below:

- Cross-Sectional Design: Cannot establish causality between allergy and tissue remodelling.
- Sample Size: The study especially Subgroup analyses may lack power due to sample size.
- Unmeasured Confounders: Environmental factors (pollution, viral load) were not assessed.

Based on current understanding of the topic, few research opportunities arise such as the following. They provide better understanding of the topic and aid in planning patient care in more specific manner.

- Longitudinal Tracking where monitoring of IgE/AEC and tissue changes post-surgery can be done to identify relapse predictors.
- Cytokine Profiling i.e. measurement of Interleukins (IL-4, IL-5, and IL-13) in serum / tissue to clarify Th2 polarization patterns maybe studied.
- Allergen-Specific IgE may be studied i.e. Correlation of serum IgE (e.g., dust mites, molds) with eosinophil density may be explored.

## CONCLUSION

This study highlights the complementary roles of serum biomarkers and histopathology in managing hypertrophic

lymphoid disorders. While IgE/AEC aid in identifying allergic predisposition, tissue analysis remains indispensable for personalized therapeutic strategies. Future studies integrating molecular profiling with clinical outcomes could further refine management protocols.

## Conflict of Interest

None.

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None.

## REFERENCES

1. Shin YH, Hwang J, Kwon R, et al. Global, regional, and national burden of allergic disorders and their risk factors in 204 countries and territories, from 1990 to 2019: A systematic analysis for the Global Burden of Disease Study 2019. *Allergy*. 2023;78(8):2232–2254. Available from: <https://doi.org/10.1111/all.15807>.
2. Martin LB, Kita H, Leiferman KM, Gleich GJ. Eosinophils in allergy: role in disease, degranulation, and cytokines. *International Archives of Allergy and Immunology*. 1996;109(3):207–215. Available from: <https://doi.org/10.1159/000237239>.
3. Roufosse F, Weller PF. Practical approach to the patient with hypereosinophilia. *Journal of Allergy and Clinical Immunology*. 2010;126(1):39–44. Available from: <https://dx.doi.org/10.1016/j.jaci.2010.04.011>.
4. Ackerman SJ, Bochner BS. Mechanisms of Eosinophilia in the Pathogenesis of Hypereosinophilic Disorders. *Immunology and Allergy Clinics of North America*. 2007;27(3):357–375. Available from: <https://doi.org/10.1016/j.jiac.2007.07.004>.
5. Rothenberg ME, Hogan SP. The eosinophil. *Annual Review of Immunology*. 2006;24(1):147–174. Available from: <https://dx.doi.org/10.1146/annurev.immunol.24.021605.090720>.
6. Wardlaw J, Brightling C, Green R, Woltmann G, Pavord I. Eosinophils in asthma and other allergic diseases. *British Medical Bulletin*. 2000;56(4):985–1003. Available from: <https://doi.org/10.1258/0007142001903490>.
7. Adamko D, Lacy P, Moqbel R. Mechanisms of eosinophil recruitment and activation. *Current Allergy and Asthma Reports*. 2002;2(2):107–116. Available from: <https://doi.org/10.1007/s11882-002-0005-2>.
8. Ilmarinen P, Kankaanranta H. Eosinophil Apoptosis as a Therapeutic Target in Allergic Asthma. *Basic & Clinical Pharmacology & Toxicology*. 2013;114(1):109–117. Available from: <https://doi.org/10.1111/bcpt.12163>.
9. Akuthota P, Wang H, Weller PF. Eosinophils as antigen-presenting cells in allergic upper airway disease. *Current Opinion in Allergy & Clinical Immunology*. 2010;10(1):14–19. Available from: <https://dx.doi.org/10.1097/aci.0b013e328334f693>.
10. Kanuru S, Sapra A. Eosinophilia. Treasure Island (FL). StatPearls Publishing. 2023. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK560929/>.
11. Palacionyte J, Januskevicius A, Vasyle E, et al. Novel Serum Biomarkers for Patients with Allergic Asthma Phenotype. *Biomedicine*. 2024;12(1):1–18. Available from: <https://doi.org/10.3390/biomedicine12010232>.
12. Ekici NY, Kulahci Ö. Relationship between tissue and serum eosinophilia in children undergoing adenotonsillectomy with allergic rhinitis. *Turkish Journal of Medical Sciences*. 2019;49(6):1754–1759. Available from: <https://doi.org/10.3906/sag-1904-105>.
13. Day KS, Rempel L, Rossi FMV, Theret M. Origins and functions of eosinophils in two non-mucosal tissues. *Frontiers in Immunology*. 2024;15:1–14. Available from: <https://doi.org/10.3389/fimmu.2024.1368142>.



14. Farne HA, Wilson A, Powell C, Bax L, Milan SJ. Anti-IL-5 therapies for severe asthma. *Cochrane Database of Systematic Reviews*. 2017;2017(9):1–127. Available from: <https://doi.org/10.1002/14651858.CD010834.pub3>.
15. Norman G, Faria R, Paton F, et al. Omalizumab for the treatment of severe persistent allergic asthma: a systematic review and economic evaluation. *Health Technology Assessment*. 2013;17(52):1–342. Available from: <https://doi.org/10.3310/hta17520>.
16. Bachert C, Desrosiers MY, Hellings PW, Laidlaw TM. The Role of Biologics in Chronic Rhinosinusitis with Nasal Polyps. *The Journal of Allergy and Clinical Immunology: In Practice*. 2021;9(3):1099–1106. Available from: <https://doi.org/10.1016/j.jaip.2020.11.017>.
17. Ekici NY, Görgülü O, Yücel G, Külahcı Ö, Arıkan OK, Durmaz C. Can the number of eosinophils in adenoid and tonsil tissue determine the allergy in children? *International Journal of Pediatric Otorhinolaryngology*. 2018;108:35–39. Available from: <https://dx.doi.org/10.1016/j.ijporl.2018.02.008>.
18. Huang X, Gong X, Gao X. Age-related hypertrophy of adenoid and tonsil with its relationship with craniofacial morphology. *BMC Pediatrics*. 2023;23(1):163. Available from: <https://dx.doi.org/10.1186/s12887-023-03979-2>.
19. Niedzielski A, Chmielik LP, zyna Mielnik-Niedzielska G, Kasprzyk A, Bogusławska J. Adenoid hypertrophy in children: a narrative review of pathogenesis and clinical relevance. *BMJ Paediatrics Open*. 2023;7(1):e001710. Available from: <https://dx.doi.org/10.1136/bmjpo-2022-001710>.
20. Evcimik MF, Dogru M, Cirik AA, Nepesov MI. Adenoid hypertrophy in children with allergic disease and influential factors. *International Journal of Pediatric Otorhinolaryngology*. 2015;79(5):694–697. Available from: <https://dx.doi.org/10.1016/j.ijporl.2015.02.017>.
21. Rout MR, Mohanty D, Vijaylaxmi Y, Bobba K, Metta C. Adenoid Hypertrophy in Adults: A case Series. *Indian Journal of Otolaryngology and Head & Neck Surgery*. 2013;65(3):269–274. Available from: <https://dx.doi.org/10.1007/s12070-012-0549-y>.
22. Bidaye R, Desarda K, Thakkar J, William CS. Adenoid Hypertrophy in Adults. *JSM Head and Face Medicine*. 2018;3(1):1–3. Available from: <https://doi.org/10.47739/2578-3793/1007>.
23. Agha F, Sadaruddin A, Abbas S, Ali SM. Serum IgE levels in patients with allergic problems and healthy subjects. *Journal of the Pakistan Medical Association*. 1997;47(6):166–169. Available from: <https://pubmed.ncbi.nlm.nih.gov/9301170/>.
24. Wong CY, Yeh KW, Huang JL, Su KW, Tsai MH, Hua MC, et al. Longitudinal analysis of total serum IgE levels with allergen sensitization and atopic diseases in early childhood. *Scientific Reports*. 2020;10(1):1–6. Available from: <https://dx.doi.org/10.1038/s41598-020-78272-8>.
25. Aung T. Immunoglobulin E tests. DermNet NZ. 2019. Available from: <https://dermnetnz.org/topics/immunoglobulin-e-tests>.
26. Gitomer SA, Fountain CR, Kingdom TT, Getz AE, Sillau SH, Katial RK, et al. Clinical Examination of Tissue Eosinophilia in Patients with Chronic Rhinosinusitis and Nasal Polyposis. *Otolaryngology-Head and Neck Surgery*. 2016;155:173–178. Available from: <https://dx.doi.org/10.1177/0194599816637856>.
27. Pavord ID, Pizzichini MM, Pizzichini E, Hargreave FE. The use of induced sputum to investigate airway inflammation. *Thorax*. 1997;52(6):498–501. Available from: <https://doi.org/10.1136/thx.52.6.498>.
28. Ramirez GA, Yacoub MR, Ripa M, et al. Eosinophils from Physiology to Disease: A Comprehensive Review. *BioMed Research International*. 2018;2018:1–28. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5829361/>.
29. Valent P, Degenfeld-Schonburg L, Sadovnik I, et al. Eosinophils and eosinophil-associated disorders: immunological, clinical, and molecular complexity. *Seminars in Immunopathology*. 2021;43(3):423–438. Available from: <https://doi.org/10.1007/s00281-021-00863-y>.

