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Research Article

Association of Allergic Rhinitis with Sinonasal Polyposis: A Clinical and Laboratory-Based Observational Study

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ABSTRACT

Background: Allergic rhinitis and sinonasal polyposis share several inflammatory mechanisms, including eosinophilic infiltration, type 2 inflammation and immunoglobulin E-mediated immune activity. However, the contribution of allergy to sinonasal-polyp formation remains uncertain. Combined clinical, endoscopic and laboratory evaluation may help identify allergic rhinitis patients who are more likely to have sinonasal polyposis. **Aim:** To determine the association of allergic rhinitis with sinonasal polyposis using clinical, endoscopic and laboratory parameters. **Materials and Methods:** This hospital-based cross-sectional observational study included 200 patients with allergic rhinitis attending the Department of ENT, ANIIMS, Port Blair between January 2026 to June 2026. Demographic information, nasal and non-nasal symptoms and family history of allergy were recorded using a structured proforma. Participants underwent otorhinolaryngological examination and diagnostic nasal endoscopy to identify sinonasal polyposis. Absolute eosinophil count and total serum IgE were measured. Clinical and laboratory parameters were compared between allergic rhinitis patients with and without polyposis. Categorical variables were compared using the chi-square or Fisher's exact test, while continuous variables were compared using Welch's independent-samples t-test. Associations were expressed as odds ratios with 95% confidence intervals, and $P<0.05$ was considered statistically significant. **Results:** Of the 200 patients with allergic rhinitis, 31 had sinonasal polyposis, giving a proportion of 15.5% (95% CI: 11.1%-21.2%). Patients with polyposis were significantly older than those without polyposis (38.06±15.39 versus 31.86±9.79 years; mean difference=6.20 years, 95% CI: 0.38-12.02; P=0.037). Hyposmia was present in 77.4% of patients with polyposis compared with 27.2% of those without polyposis and was significantly associated with polyposis (OR=9.17, 95% CI: 3.70-22.72; P<0.001). Headache was reported by 96.8% and 29.0% of the respective groups (OR=73.47, 95% CI: 9.75-553.79; P<0.001). An absolute eosinophil count above 440 cells/mm³ was observed in

64.5% of patients with polyposis and 26.0% of those without polyposis (OR=5.17, 95% CI: 2.29-11.64; P<0.001). Total serum IgE above 320 IU/mL was found in 54.8% and 25.4%, respectively (OR=3.56, 95% CI: 1.63-7.78; P=0.001). Sex, family history, nasal discharge, nasal obstruction, nasal pruritus and sneezing were not significantly associated with polyposis. **Conclusion:** Sinonasal polyposis was present in approximately one-sixth of patients with allergic rhinitis. Older age, hyposmia, headache, elevated absolute eosinophil count and elevated total serum IgE were significantly associated with polyposis. Nasal endoscopy should be considered in allergic rhinitis patients with suggestive clinical features or raised inflammatory markers. AEC and serum IgE may be useful supportive markers but should not replace clinical and endoscopic assessment.

KEYWORDS: Allergic rhinitis; sinonasal polyposis; absolute eosinophil count.

INTRODUCTION

Allergic rhinitis is a common, chronic, immunoglobulin E (IgE)-mediated inflammatory disorder of the nasal mucosa that develops following exposure to sensitizing allergens. It is characterized by sneezing, watery rhinorrhoea, nasal itching and obstruction, and may be accompanied by ocular itching, watering, postnasal discharge, headache and reduced sense of smell. Allergic rhinitis substantially affects sleep, daily activities, academic or occupational performance and quality of life. Its pathogenesis involves allergen-specific IgE, mast-cell activation, T-helper type 2 inflammation and recruitment of eosinophils into the nasal mucosa [1].

Sinonasal polyposis is a chronic inflammatory condition characterized by benign, oedematous protrusions of sinonasal mucosa, usually arising from the middle meatus and ethmoidal region. Patients commonly present with progressive nasal obstruction, hyposmia or anosmia, nasal discharge, postnasal drip and facial pressure. Nasal polyps are frequently associated with chronic rhinosinusitis, asthma, aspirin-exacerbated respiratory disease and other inflammatory disorders. Chronic rhinosinusitis with nasal polyps represents a distinct clinical phenotype with marked mucosal oedema, tissue remodelling and, particularly in type 2 disease, eosinophilic inflammation and increased local IgE production [2,3].

Allergic rhinitis and sinonasal polyposis share several inflammatory characteristics, including eosinophilic infiltration, increased production of type 2 cytokines and activation of IgE-related immune pathways. Persistent exposure to allergens may promote chronic sinonasal inflammation, increase mucosal permeability and contribute to oedema and tissue remodelling. Nevertheless, allergy is not present in every patient with nasal polyposis, and the precise contribution of systemic atopy to the development and severity of nasal polyps remains uncertain. The detection of allergic sensitization may therefore require the combined assessment of clinical history, examination and laboratory markers rather than symptoms alone [3,4].

Absolute eosinophil count, nasal-smear eosinophilia and total serum IgE are readily available laboratory indicators of allergic and type 2 inflammation. Elevated blood eosinophil and serum IgE levels may identify patients with a greater inflammatory burden, although these markers are neither completely specific nor direct substitutes for allergen-specific testing. Nasal endoscopy is essential for detecting polyps and assessing their extent, while computed tomography of the paranasal sinuses may be used to evaluate sinonasal involvement in selected patients [2,5].

A combined clinical and laboratory evaluation may clarify whether patients with

allergic rhinitis and increased eosinophilic or IgE-mediated activity have a greater frequency of sinonasal polyposis. The present study was therefore undertaken to determine the association between allergic rhinitis and sinonasal polyposis and to examine the relationship of nasal polyps with clinical manifestations, family history of allergy, absolute eosinophil count, nasal eosinophilia and total serum IgE.

AIM

To determine the association of allergic rhinitis with sinonasal polyposis using clinical, endoscopic and laboratory parameters.

OBJECTIVES

1. To determine the proportion of sinonasal polyposis among patients with allergic rhinitis.
2. To compare clinical characteristics and family history of allergy between allergic rhinitis patients with and without sinonasal polyposis.
3. To assess the association of absolute eosinophil count, nasal-smear eosinophilia and total serum IgE with sinonasal polyposis.

MATERIALS AND METHODOLOGY

Source of Data

The study data were obtained from patients who attended the outpatient and inpatient services of the Department of Otorhinolaryngology with symptoms suggestive of allergic rhinitis. Information was collected through patient interviews, clinical examinations, diagnostic nasal endoscopy, laboratory investigations and relevant hospital records. All eligible patients who fulfilled the selection criteria during the study period were considered for inclusion.

Study Design

This was a hospital-based, cross-sectional observational study. The clinical and laboratory characteristics of allergic rhinitis patients with sinonasal polyposis were compared with those of allergic rhinitis patients without sinonasal polyposis.

Study Location

The study was conducted in the Department of ENT, ANIIMS, Port Blair. Laboratory investigations were performed in the institution's central clinical laboratory and pathology laboratory.

Study Duration

Study conducted between January 2026 to June 2026.

Sample Size

A total of 200 patients with allergic rhinitis were included in the study. Participants were recruited consecutively from eligible patients attending the Department of Otorhinolaryngology during the study period.

Inclusion Criteria

1. Patients presenting with clinical features suggestive of allergic rhinitis, including one or more of the following:
    - Recurrent sneezing
    - Watery nasal discharge
    - Nasal obstruction
    - Nasal itching
    - Ocular itching or watering
    - Pharyngeal itching
    - Hyposmia or anosmia
    - Headache or postnasal discharge
2. Patients whose clinical history and examination were consistent with allergic rhinitis.
3. Patients with an absolute blood eosinophil count of ≥350 cells/mm³, as applied in the study protocol.
4. Patients who provided written informed consent or whose legally authorized representative provided consent, where applicable.

Exclusion Criteria

1. Patients with vasomotor or other non-allergic rhinitis.
2. Patients with atrophic rhinitis.
3. Patients with suspected or confirmed benign or malignant tumours of the nose, paranasal sinuses or nasopharynx.
4. Patients with drug-induced rhinitis.
5. Patients with an acute upper respiratory tract infection at the time of assessment.
6. Patients with bleeding disorders or other conditions that prevented nasal examination or specimen collection.
7. Patients with incomplete clinical or laboratory information.
8. Patients who declined to participate in the study.

Procedure and Methodology

After obtaining approval from the Institutional Ethics Committee, patients presenting with symptoms of allergic rhinitis were screened for eligibility. Written informed consent was obtained before enrolment. Each participant was evaluated using a predesigned and pretested case-record form.

A detailed history was recorded regarding age, sex, residence, duration of symptoms, seasonal or perennial pattern, precipitating factors, exposure to dust, pollen, smoke or animals, previous treatment and associated asthma, dermatitis or drug hypersensitivity. A family history of allergic rhinitis, bronchial asthma, eczema or other atopic disorders was documented.

The severity of nasal and non-nasal symptoms was graded using a four-point symptom-evaluation scale:

- 0: Absent no symptom
- 1: Mild occasionally present but not troublesome
- 2: Moderate frequently present and annoying
- 3: Severe continuously present and interfering with work or sleep

The nasal symptoms evaluated included sneezing, rhinorrhoea, nasal itching, nasal obstruction and hyposmia. Non-nasal symptoms included ocular itching, watering, pharyngeal itching and headache.

A complete general physical examination and systemic examination were performed. Otorhinolaryngological examination included external nasal examination, anterior rhinoscopy and examination of the oral cavity, oropharynx and ears. Diagnostic nasal endoscopy was carried out whenever clinically indicated to identify sinonasal polyps and evaluate their site, extent and laterality. Sinonasal polyposis was diagnosed when pale, glistening, insensitive and non-bleeding polypoidal masses were visualized within the nasal cavity or middle meatus. Computed tomography of the paranasal sinuses was obtained in selected patients when required to define the extent of disease or support treatment planning.

The participants were subsequently classified into two groups:

- Allergic rhinitis without sinonasal polyposis.
- Allergic rhinitis with sinonasal polyposis.

Clinical manifestations, family history and laboratory parameters were compared between these groups.

Sample Processing

Under aseptic precautions, peripheral venous blood was collected from each participant. A blood sample collected in an ethylenediaminetetraacetic acid tube was used for complete blood count and calculation of the absolute eosinophil count. Absolute eosinophil count was expressed as cells/mm^3 and was interpreted according to the predefined study cut-off values.

A separate blood sample was collected in a plain tube for measurement of total serum IgE. The sample was allowed to clot and was centrifuged to separate the serum. Serum was tested promptly or stored under

recommended laboratory conditions until analysis. Total serum IgE was measured using the immunoassay method routinely employed by the institutional laboratory and was expressed in IU/mL.

A nasal smear was obtained by gently collecting secretions or scraping the mucosal surface from the inferior turbinate or nasal cavity. The material was transferred onto a clean glass slide, air-dried, stained using the laboratory's standard staining procedure and examined microscopically for eosinophils. All samples were appropriately labelled and processed according to institutional quality-control procedures.

Data Collection

Data were collected using a structured case-record form containing the following:

- Sociodemographic characteristics.
- Duration and pattern of allergic symptoms.
- Nasal and non-nasal symptom scores.
- Personal and family history of allergy.
- General and otorhinolaryngological examination findings.
- Anterior rhinoscopy and nasal endoscopy findings.
- Presence, site and extent of sinonasal polyposis.
- Absolute eosinophil count.
- Nasal-smear eosinophilia.
- Total serum IgE level.
- Relevant radiological findings, where available.

The collected forms were reviewed for completeness. Data were coded and entered into an electronic database. Personal identifiers were removed before statistical analysis to maintain confidentiality.

Statistical Methods

Data were analysed using appropriate statistical software. Continuous variables such as age, absolute eosinophil count and total serum IgE were examined for normality and summarized as mean with standard deviation or median with interquartile range, as appropriate. Categorical variables were presented as frequencies and percentages.

The prevalence of sinonasal polyposis among allergic rhinitis patients was calculated with a 95% confidence interval. The independent-samples Student's *t* test was used to compare normally distributed continuous variables between patients with and without polyposis. The Mann-Whitney *U* test was used for non-normally distributed variables.

The chi-square test was employed to assess associations between categorical variables, including sex, symptom categories, family history, raised eosinophil count, elevated serum IgE and sinonasal polyposis. Fisher's exact test was applied when the expected frequency in any cell was less than five. Effect sizes were expressed as odds ratios with 95% confidence intervals.

Pearson's or Spearman's correlation coefficient was calculated to assess relationships among symptom severity, eosinophil count and serum IgE, depending on the distribution of the data. Binary logistic regression analysis was used, where appropriate, to identify factors independently associated with sinonasal polyposis after controlling for potential confounders such as age, sex, symptom duration and family history. All tests were two-tailed, and a p value <0.05 was considered statistically significant.

OBSERVATION AND RESULTS

Table 1: Overall association of clinical, endoscopic and laboratory parameters with sinonasal polyposis among patients with allergic rhinitis (N = 200)

Study parameter	Allergic rhinitis with	Allergic rhinitis without	Effect estimate (95% CI)	Test of significance	P value

	polyposis (n=31)	**polyposis (n=169)**			
Age, years, Mean (SD)	38.06 (15.39)	31.86 (9.79)	Mean difference: 6.20 (0.38-12.02)	Welch's t=2.16	0.037*
Male sex, n (%)	18 (58.1%)	71 (42.0%)	OR: 1.91 (0.88-4.15)	χ^2=2.73	0.098
Hyposmia, n (%)	24 (77.4%)	46 (27.2%)	OR: 9.17 (3.70-22.72)	χ^2=29.02	<0.001*
Headache, n (%)	30 (96.8%)	49 (29.0%)	OR: 73.47 (9.75-553.79)	χ^2=50.36	<0.001*
Family history of allergy, n (%)	7 (22.6%)	57 (33.7%)	OR: 0.57 (0.23-1.41)	χ^2=1.50	0.221
AEC >440 cells/mm³, n (%)	20 (64.5%)	44 (26.0%)	OR: 5.17 (2.29-11.64)	χ^2=17.80	<0.001*
Total serum IgE >320 IU/mL, n (%)	17 (54.8%)	43 (25.4%)	OR: 3.56 (1.63-7.78)	χ^2=10.80	0.001*

Statistically significant at P<0.05. AEC: absolute eosinophil count; IgE: immunoglobulin E; OR: odds ratio; CI: confidence interval.

Among the 200 patients with allergic rhinitis, 31 had sinonasal polyposis and 169 did not. Patients with polyposis were significantly older than those without polyposis, with mean ages of 38.06±15.39 years and 31.86±9.79 years, respectively. The mean difference was 6.20 years (95% CI: 0.38-12.02; Welch's t=2.16, P=0.037). Males constituted 58.1% of the polyposis group compared with 42.0% of the non-polyposis group; however, this difference was not statistically significant (OR=1.91, 95% CI: 0.88-4.15; P=0.098).

Hyposmia was reported by 77.4% of patients with polyposis compared with 27.2% of those without polyposis. Patients with hyposmia had approximately nine times greater odds of having sinonasal polyposis (OR=9.17, 95% CI: 3.70-22.72; P<0.001). Headache was also considerably more frequent in the polyposis group than in the non-polyposis group (96.8% versus 29.0%), demonstrating a strong association with polyposis (OR=73.47, 95% CI: 9.75-553.79; P<0.001).

A family history of allergy was present in 22.6% and 33.7% of the respective groups and was not significantly associated with polyposis (OR=0.57, 95% CI: 0.23-1.41; P=0.221).

An absolute eosinophil count above 440 cells/mm³ was observed in 64.5% of patients with polyposis compared with 26.0% of those without polyposis. An elevated AEC was associated with approximately five times higher odds of polyposis (OR=5.17, 95% CI: 2.29-11.64; P<0.001). Similarly, total serum IgE above 320 IU/mL was found in 54.8% of patients with polyposis and 25.4% of those without polyposis. Elevated serum IgE was significantly associated with polyposis (OR=3.56, 95% CI: 1.63-7.78; P=0.001).

Table 2: Proportion of sinonasal polyposis among patients with allergic rhinitis (N = 200)

Endoscopic finding	Frequency, n (%)	95% CI for proportion	Test of significance	P value
Sinonasal polyposis present	31 (15.5%)	11.1%-21.2%	One-sample proportion z=2.59†	0.010*
Sinonasal polyposis absent	169 (84.5%)	78.8%-88.9%		
Total	200 (100.0%)			

†The observed prevalence of 15.5% was compared with a predefined reference proportion of 10%. Wilson's method was used to calculate the 95% confidence interval.
Statistically significant at P<0.05.

Sinonasal polyposis was detected endoscopically in 31 of the 200 patients with allergic rhinitis, giving an observed proportion of 15.5% (95% CI: 11.1%-21.2%). The remaining 169 patients, constituting 84.5% of the study population (95% CI: 78.8%-88.9%), did not have sinonasal polyposis. The observed proportion of 15.5% was significantly higher than the prespecified reference proportion of 10% (one-sample proportion z=2.59, P=0.010).

Table 3: Comparison of clinical characteristics and family history between allergic rhinitis patients with and without sinonasal polyposis

Clinical characteristic	With polyposis (n=31)	Without polyposis (n=169)	Effect estimate (95% CI)	Test of significance	P value
Age, years, Mean (SD)	38.06 (15.39)	31.86 (9.79)	MD: 6.20 (0.38-12.02)	Welch's t=2.16	0.037*
Male sex, n (%)	18 (58.1%)	71 (42.0%)	OR: 1.91 (0.88-4.15)	χ^2=2.73	0.098
Nasal discharge, n (%)	31 (100.0%)	157 (92.9%)	OR: 5.00 (0.29-86.66)†	Fisher's exact test	0.220
Nasal obstruction, n (%)	31 (100.0%)	164 (97.0%)	OR: 2.11 (0.11-39.06)†	Fisher's exact test	1.000
Nasal pruritus, n (%)	22 (71.0%)	123 (72.8%)	OR: 0.91 (0.39-2.13)	χ^2=0.04	0.835
Sneezing, n (%)	30 (96.8%)	157 (92.9%)	OR: 2.29 (0.29-18.30)	Fisher's exact test	0.696
Hyposmia, n (%)	24 (77.4%)	46 (27.2%)	OR: 9.17 (3.70-22.72)	χ^2=29.02	<0.001*

Headache, n (%)	30 (96.8%)	49 (29.0%)	OR: 73.47 (9.75-553.79)	χ^2=50.36	<0.001*
Family history of allergy, n (%)	7 (22.6%)	57 (33.7%)	OR: 0.57 (0.23-1.41)	χ^2=1.50	0.221

†Haldane-Anscombe correction was applied because of a zero cell.
Statistically significant at P<0.05; MD: mean difference.

Patients with sinonasal polyposis had a significantly higher mean age than patients without polyposis (38.06±15.39 versus 31.86±9.79 years), with a mean difference of 6.20 years (95% CI: 0.38-12.02; P=0.037). Males accounted for 58.1% of patients with polyposis and 42.0% of patients without polyposis. Although males had higher odds of polyposis, the association did not reach statistical significance (OR=1.91, 95% CI: 0.88-4.15; P=0.098).

Nasal discharge was present in all patients with polyposis and in 92.9% of those without polyposis. Nasal obstruction was similarly present in 100.0% and 97.0% of the two groups, respectively. Neither nasal discharge (P=0.220) nor nasal obstruction (P=1.000) differed significantly between the groups. Nasal pruritus was reported by 71.0% of patients with polyposis and 72.8% of those without polyposis (OR=0.91, 95% CI: 0.39-2.13; P=0.835). Sneezing was also common in both groups, occurring in 96.8% and 92.9%, respectively, without a statistically significant difference (OR=2.29, 95% CI: 0.29-18.30; P=0.696).

In contrast, hyposmia was substantially more frequent among patients with polyposis than among those without polyposis (77.4% versus 27.2%). Hyposmia was associated with more than ninefold higher odds of polyposis (OR=9.17, 95% CI: 3.70-22.72; P<0.001). Headache was reported by 96.8% of the polyposis group compared with 29.0% of the non-polyposis group and showed a particularly strong association with polyposis (OR=73.47, 95% CI: 9.75-553.79; P<0.001). A family history of allergy was less frequent in the polyposis group than in the non-polyposis group (22.6% versus 33.7%), but the difference was not statistically significant (OR=0.57, 95% CI: 0.23-1.41; P=0.221).

Table 4: Association of laboratory inflammatory parameters with sinonasal polyposis among patients with allergic rhinitis

Laboratory parameter	With polyposis (n=31)	Without polyposis (n=169)	Effect estimate (95% CI)	Test of significance	P value
Absolute eosinophil count, cells/mm³, Mean	478.96	432.76	Mean difference: 46.20	†	†
AEC >440 cells/mm³, n (%)	20 (64.5%)	44 (26.0%)	OR: 5.17 (2.29-11.64)	χ^2=17.80	<0.001*
AEC ≤440 cells/mm³, n (%)	11 (35.5%)	125 (74.0%)	Reference		
Total serum IgE, IU/mL, Mean	465.56	296.70	Mean difference: 168.86	†	†
Total serum IgE >320 IU/mL, n (%)	17 (54.8%)	43 (25.4%)	OR: 3.56 (1.63-7.78)	χ^2=10.80	0.001*

Total serum IgE ≤320 IU/mL, n (%)	14 (45.2%)	126 (74.6%)	Reference		
Nasal-smear eosinophilia	Not reported	Not reported	Not estimable	Not estimable	Not estimable

†

The dissertation reported group means but did not report the corresponding standard deviations; therefore, a valid confidence interval and independent-samples test could not be calculated from the summarized values.

Statistically significant at P<0.05.

The mean absolute eosinophil count was higher among patients with sinonasal polyposis than among those without polyposis (478.96 versus 432.76 cells/mm³), producing a mean difference of 46.20 cells/mm³. However, as the corresponding standard deviations were unavailable, a confidence interval and significance test for this mean difference could not be calculated. When AEC was categorized using a cut-off of 440 cells/mm³, an elevated count was present in 20 patients with polyposis (64.5%) compared with 44 patients without polyposis (26.0%). Conversely, an AEC of 440 cells/mm³ or below was found in 35.5% and 74.0% of the respective groups. Patients with an AEC above 440 cells/mm³ had approximately fivefold higher odds of sinonasal polyposis (OR=5.17, 95% CI: 2.29-11.64; χ^2=17.80, P<0.001).

The mean total serum IgE level was also higher in patients with polyposis than in those without polyposis (465.56 versus 296.70 IU/mL), corresponding to a mean difference of 168.86 IU/mL. A valid statistical comparison of these means could not be performed because group-specific standard deviations were not reported. Total serum IgE above 320 IU/mL was observed in 54.8% of patients with polyposis and 25.4% of those without polyposis. Elevated serum IgE was associated with approximately 3.6 times greater odds of polyposis (OR=3.56, 95% CI: 1.63-7.78; χ^2=10.80, P=0.001). Nasal-smear eosinophilia could not be analysed because its results were not reported.

DISCUSSION

Overall association of clinical, endoscopic and laboratory parameters

The present study included 200 patients with allergic rhinitis, of whom 31 had endoscopically confirmed sinonasal polyposis. Patients with polyposis were significantly older than those without polyposis (38.06±15.39 versus 31.86±9.79 years; P=0.037). This finding is biologically plausible because nasal polyposis usually reflects prolonged mucosal inflammation and cumulative tissue remodelling rather than an immediate manifestation of allergic rhinitis. Stevens et al. (2016)[1] similarly described chronic rhinosinusitis with nasal polyps (CRSwNP) as predominantly an adult disease, with prevalence increasing in middle age. Fokkens et al. (2020)[2] also reported that nasal polyps are uncommon in young children and generally become clinically evident in adulthood. Nevertheless, the wide standard deviation in the polyposis group indicates considerable age variation.

Males constituted 58.1% of the polyposis group compared with 42.0% of the group without polyposis. Although the odds of polyposis were approximately 1.9 times higher among males, the difference was not statistically significant (P=0.098). The direction of this association is consistent with the modest male predominance described by Stevens et al. (2016)[1] and in the EPOS 2020 evidence synthesis [2]. The failure to attain statistical significance in the present study may be explained by the relatively small number of patients with polyposis and the resulting wide confidence interval.

Hyposmia was observed in 77.4% of patients with polyposis compared with 27.2% of those without polyposis, corresponding to a ninefold increase in the odds of polyposis. Soler et al. (2016)[3] demonstrated that olfactory dysfunction is one of the most prominent manifestations of chronic rhinosinusitis, particularly in patients with polyps. Olfactory impairment in polyposis develops through mechanical obstruction of airflow to the olfactory cleft and inflammatory injury to the olfactory neuroepithelium. Fokkens et al. (2020)[2] and Orlandi et al. (2021)[4] consequently recognize reduction or loss of smell as one of the cardinal symptoms of CRS and as a clinically important indicator of CRSwNP.

Headache was reported by 96.8% of patients with polyposis and 29.0% of those without polyposis. Although the estimated odds ratio was very high, its wide confidence interval indicates limited precision caused by the near-zero cell in the polyposis group. Facial pain or pressure can accompany CRS, but headache alone is nonspecific and may arise from migraine, tension-type headache or other neurological disorders. Therefore, Orlandi et al. (2021)[4] emphasized that headache should be interpreted together with nasal symptoms and objective endoscopic or radiological findings rather than being used independently to diagnose sinonasal disease.

A family history of allergy was not significantly associated with polyposis. This finding supports the concept that systemic allergy is only one of several contributors to nasal-polyp formation. Wang et al. (2016)[5] demonstrated marked inflammatory heterogeneity in CRSwNP across geographic populations, with type 2/eosinophilic inflammation predominating in many Western patients but mixed type 1, type 2 and type 3 patterns occurring elsewhere. Thus, nasal polyposis can develop in patients without a demonstrable family history of allergy, and the absence of such a history does not exclude local type 2 inflammation.

Proportion of sinonasal polyposis in allergic rhinitis

Sinonasal polyposis was detected in 31 of the 200 patients with allergic rhinitis, giving a proportion of 15.5% (95% CI: 11.1%-21.2%). This proportion was significantly higher than the prespecified reference value of 10% (P=0.010). However, it should be interpreted as the proportion among hospital-attending patients with allergic rhinitis rather than the prevalence in the general population. Patients attending a tertiary ENT facility are more likely to have persistent nasal obstruction, olfactory disturbance or symptoms unresponsive to routine treatment, potentially increasing the observed proportion of polyposis.

Stevens et al. (2016)[1] reported that CRSwNP affects approximately 1%-4% of the general population, but its frequency is considerably higher in selected patients with chronic sinonasal disease, asthma or atopic disorders. Dietz de Loos et al. (2019)[6], using a population-based approach combining symptoms with nasal endoscopy, showed that objective examination substantially improves the accuracy of CRS prevalence estimates. The 15.5% proportion in the present study therefore cannot be directly compared with population prevalence estimates because all participants already had allergic rhinitis and were recruited from an ENT department.

The observed association is nevertheless compatible with the unified-airway concept, according to which persistent allergic inflammation can contribute to mucosal oedema, impaired sinus ventilation and chronic sinonasal inflammation. Fokkens et al. (2020)[2] noted that allergy is frequently present in CRSwNP but is neither necessary nor sufficient for polyp development. Accordingly, the present results demonstrate coexistence and association but do not

establish that allergic rhinitis directly caused sinonasal polyposis.

Clinical characteristics and family history

Nasal discharge, nasal obstruction, pruritus and sneezing were common in both study groups. Nasal discharge occurred in 100% of patients with polyposis and 92.9% of those without polyposis, while nasal obstruction was present in 100% and 97.0%, respectively. These differences were not statistically significant because both symptoms were already highly prevalent among patients selected for allergic rhinitis. This ceiling effect reduced their ability to discriminate between patients with and without polyps.

Nasal pruritus and sneezing also showed no significant group differences. These symptoms are more characteristic of allergen-induced nasal mucosal activation than of the structural presence of polyps. Wise et al. (2023)[7] identified sneezing, nasal itching and watery rhinorrhoea as characteristic features of allergic rhinitis, whereas persistent obstruction and olfactory loss are more suggestive of concomitant CRS or polyposis. Thus, the similar frequency of pruritus and sneezing in both groups was expected because every participant had allergic rhinitis.

In contrast, hyposmia had a strong and statistically significant association with polyposis. This finding agrees with the international consensus statements of Fokkens et al. (2020)[2] and Orlandi et al. (2021)[4], which identify smell loss as a major feature of CRSwNP. Kohli et al. (2016)[8] further demonstrated that nasal polyposis is an important determinant of olfactory dysfunction and that olfactory outcomes can improve following appropriate surgical management, although recovery varies with age, disease duration and inflammatory phenotype.

The absence of a significant relationship between family history of allergy and polyposis suggests that self-reported familial atopy may not accurately represent the inflammatory endotype of sinonasal tissue. Family history is vulnerable to recall error and may underestimate undiagnosed allergy among relatives. Moreover, local IgE production can occur within nasal-polyps independently of circulating IgE or conventional systemic allergy markers. Therefore, objective testing with allergen-specific IgE or skin-prick testing would have provided a more precise assessment than family history alone.

Absolute eosinophil count, nasal eosinophilia and serum IgE

The mean absolute eosinophil count was higher in patients with polyposis than in those without polyposis (478.96 versus 432.76 cells/mm³). More importantly, 64.5% of patients with polyposis had an AEC above 440 cells/mm³ compared with 26.0% of those without polyposis. Elevated AEC was associated with more than fivefold higher odds of sinonasal polyposis ($P<0.001$). This finding supports eosinophilic or type 2 inflammation as an important mechanism linking allergic rhinitis with polyp formation. Ammu Sreeparvathi et al. (2017)[9] found that peripheral eosinophil counts were significantly higher in patients with CRSwNP and proposed blood eosinophilia as an accessible marker of eosinophilic inflammation. Tokunaga et al. (2015)[10] incorporated peripheral blood eosinophilia into the JESREC clinical scoring system and showed that increasing eosinophilic disease severity was associated with refractory disease and recurrence. Fujieda et al. (2019)[11] similarly described eosinophilic CRS as a severe CRSwNP endotype characterized by blood and tissue eosinophilia, olfactory dysfunction and a tendency toward recurrence.

Zhong et al. (2021)[12] reported a mean blood eosinophil count of $0.79\pm0.27\times10^9$/L in eosinophilic CRSwNP compared with $0.30\pm0.22\times10^9$/L in non-eosinophilic

CRSwNP (P<0.001). A cut-off of 0.39×10^9/L distinguished the phenotypes, while a higher cut-off predicted recurrence. These findings are consistent with the present AEC threshold of 440 cells/mm^3, although differences in population, disease severity and laboratory definitions prevent direct comparison of cut-offs. Blood eosinophils should therefore be interpreted as a supportive rather than definitive diagnostic marker.

Total serum IgE was also appreciably higher in the polyposis group than in the non-polyposis group (465.56 versus 296.70 IU/mL). Serum IgE above 320 IU/mL was observed in 54.8% and 25.4% of the respective groups, giving an odds ratio of 3.56 (P=0.001). This agrees with the frequent presence of type 2 inflammation, B-cell activation and local IgE production in CRSwNP. The clinical efficacy of anti-IgE treatment reported by Gevaert et al. (2020)[13] further supports a functional role for IgE in at least a subgroup of patients with severe CRSwNP.

Nevertheless, total serum IgE is influenced by allergic rhinitis, asthma, parasitic infestation, smoking and other atopic conditions. It does not identify the responsible allergen and may correlate imperfectly with local tissue IgE. The association observed in this study should therefore not be interpreted as evidence that systemic IgE alone caused polyposis. Combined evaluation of serum IgE, allergen-specific IgE, blood eosinophils, nasal cytology and tissue eosinophilia would provide more accurate inflammatory endotyping.

Nasal-smear eosinophilia could not be compared because the corresponding results were unavailable. This represents an important limitation because blood and local nasal eosinophilia are not interchangeable. Some patients may have tissue or nasal eosinophilia despite normal peripheral counts, while systemic eosinophilia may occur without extensive local disease. Future investigations should report standardized nasal-cytology findings and examine their agreement with blood eosinophil count, serum IgE and histopathological eosinophilia.

CONCLUSION

Sinonasal polyposis was identified in 15.5% of patients with allergic rhinitis, indicating a clinically relevant coexistence of the two conditions. Patients with sinonasal polyposis were significantly older and had substantially higher frequencies of hyposmia and headache than patients without polyposis. Elevated absolute eosinophil count and total serum IgE were also significantly associated with sinonasal polyposis, supporting the involvement of eosinophilic and IgE-mediated inflammation in its development. In contrast, sex, family history of allergy, nasal discharge, nasal obstruction, nasal pruritus and sneezing were not significantly associated with polyposis.

These findings emphasize the importance of nasal endoscopic evaluation in allergic rhinitis patients, particularly those presenting with hyposmia, persistent headache or elevated inflammatory markers. Absolute eosinophil count and serum IgE may serve as accessible supportive markers for identifying allergic rhinitis patients who are more likely to have sinonasal polyposis. However, these laboratory parameters should be interpreted along with clinical and endoscopic findings rather than used as independent diagnostic tests.

LIMITATIONS

1. The study was conducted at a single tertiary-care institution; therefore, the findings may not be generalizable to the wider community or to other geographical populations.
2. The hospital-based sampling method may have introduced referral bias because patients attending an ENT department may have had

more persistent or severe symptoms than patients managed in primary care.
3. The cross-sectional observational design established associations but could not demonstrate a temporal or causal relationship between allergic rhinitis and sinonasal polyposis.
4. Only 31 patients had sinonasal polyposis, resulting in limited statistical power and wide confidence intervals for some associations.
5. Participants were recruited consecutively rather than through population-based random sampling, which may have introduced selection bias.
6. Allergic rhinitis was primarily evaluated using clinical features and routine laboratory parameters. Allergen-specific serum IgE and standardized skin-prick testing were not consistently available.
7. Total serum IgE is a nonspecific marker and may be affected by asthma, parasitic infection, smoking and other allergic or inflammatory disorders.
8. Absolute eosinophil count may not accurately represent the extent of eosinophilic inflammation within sinonasal tissues.
9. Nasal-smear eosinophilia was mentioned in the methodology, but complete participant-level results were unavailable; consequently, its association with polyposis could not be analysed.
10. Group-specific standard deviations for mean AEC and serum IgE were not reported, preventing calculation of confidence intervals and formal statistical comparison of the mean values.
11. Histopathological examination and tissue eosinophil counts were not available for all patients, limiting the inflammatory endotyping of sinonasal polyps.
12. The extent and severity of polyposis were not uniformly quantified using standardized endoscopic or radiological scoring systems such as the Lund-Kennedy and Lund-Mackay scores.
13. Potential confounding factors such as bronchial asthma, aspirin-exacerbated respiratory disease, smoking, environmental exposure and previous medical treatment were not comprehensively adjusted through multivariable analysis.
14. Symptom information and family history were self-reported and were therefore susceptible to recall and reporting bias.
15. Long-term follow-up was not performed, so the study could not assess treatment response, progression, recurrence or the subsequent development of polyposis.

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