

Clinicopathological Correlation of Nasal Smear Eosinophilia in Allergic and Non-Allergic Rhinitis: An Observational Study

Rutul Panchal¹, Trupal Patel²

¹Assistant Professor, Department of ENT, BJ Medical College and Hospital, Ahmedabad, Gujarat, India

²Assistant Professor, Department of ENT, BJ Medical College and Hospital, Ahmedabad, Gujarat, India

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Corresponding Author: Dr. Trupal Patel

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Abstract:

Background: Allergic rhinitis (AR) is a prevalent persistent inflammatory disorder of the nasal passages characterized by histamine-mediated symptoms. Non-allergic rhinitis (NAR) presents similar symptoms but does not have systemic allergic characteristics. Nasal smear eosinophilia is a simple and inexpensive tool for differentiating between AR and NAR and for identifying eosinophilic non-allergic rhinitis (ENR).

Aim: To evaluate the clinicopathological relationship of nasal smear eosinophilia in allergic and non-allergic rhinitis.

Methods: A prospective observational study was conducted at a tertiary care hospital, Ahmedabad, including 45 patients who were clinically diagnosed with AR, and 45 age- and sex-matched healthy controls. Nasal smears were obtained and subsequently stained using May-Grünwald and Giemsa methods. Percentages of eosinophils were assessed morphologically under a microscope, with statistical analysis conducted using SPSS version 25. Statistical significance was considered for p-values less than 0.05.

Results: Moderate eosinophil counts (>11 -50%) were found in 62.2% of the AR cases. Pathological eosinophilia (eosinophils $>10\%$) was significantly higher in patients (71.1%) than in controls (2.2%) ($p < 0.05$). Intermittent rhinitis was associated with primarily moderate to high eosinophil counts; persistent rhinitis demonstrated a broader range of eosinophil counts.

Conclusion: Nasal smear eosinophilia correlates well with AR and NAR and can serve as a useful diagnostic adjunct in differentiating the various subtypes of rhinitis, assist in medical management, and in determining eosinophilic non-allergic rhinitis (ENR).

Keywords: Allergic rhinitis, Nasal smear, Eosinophilia, Clinicopathological correlation, Eosinophilic non-allergic rhinitis, non-allergic rhinitis.

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Introduction

Allergic rhinitis (AR) is a frequent chronic inflammatory disease of the nasal mucosa, found in a considerable percentage of the world population. It is due to an exaggerated immune response to airborne allergens like pollen, dust mites, animal dander, or molds, and results in continual inflammation of the nose [1]. This hypersensitivity reaction causes typical symptoms involving nasal congestion, sneezing, itching, rhinorrhea, and conjunctival symptoms in the form of watery, red, and itchy eyes. These symptoms tend to appear within minutes of coming in contact with an allergen and last from several hours to days, impacting sleep, occupation, and daily life, and causing social, psychiatric, and economic burdens. Certain patients present with seasonal symptoms due to pollen, while others have perennial symptoms due to uninterrupted exposure to indoor allergens, reflecting the recurrent and persistent nature of the disease.² Allergic rhinitis commonly co-exists with other atopic diseases like asthma, allergic

conjunctivitis, and atopic dermatitis, reflecting its systemic nature and the notion of common atopic predisposition [2].

Allergic rhinitis pathophysiology results from an interaction of genetic predisposition and environmental exposures. There is an increased risk of acquiring allergic rhinitis with a first-degree relative history of atopy [3]. Immunologically, antigen exposure results in the activation of antigen-presenting cells, which activate T-helper 2 (Th2) lymphocytes, stimulating B lymphocytes to synthesize allergen-specific immunoglobulin E (IgE) antibodies. Repeat antigen exposure results in IgE cross-linking on basophils and mast cells, with the consequent release of inflammatory mediators like histamine, leukotrienes, and prostaglandins, causing acute symptoms of sneezing, itching, rhinorrhea, and congestion [4]. Chronic inflammation, epithelial damage, and sub-epithelial remodeling of the nasal mucosa from

repeated exposure may cause disease persistence and refractoriness to treatment.

Allergic rhinitis is diagnosed by history, clinical examination, and verification tests like prick testing or measurement of serum allergen-specific IgE. While helpful, these tests have their drawbacks, including the potential to yield false positives and variable accessibility in clinical practice. Diagnostically, allergic rhinitis should also be differentiated from viral infections of the upper respiratory tract, which typically present with self-limiting illness accompanied by systemic symptoms like fever, while allergic rhinitis does not resolve over two weeks without fever [5]. Treatment targets symptomatic relief and quality-of-life enhancement. Intranasal steroids, oral or intranasal antihistamines, and leukotriene receptor antagonists reduce inflammation effectively and also relieve symptoms. In patients with moderately to severely affected diseases who do not respond to drug therapy, allergen immunotherapy (AIT) has the capability of altering the basic immune response and achieving long-term remission while possibly averting the development of asthma [6].

Non-allergic rhinitis (NAR) is also an important cause of chronic nasal symptoms and presents nasal congestion, rhinorrhea, and sneezing without demonstrable sensitization to an allergen or systemic atopy [7]. The subtypes include vasomotor rhinitis and perennial non-allergic rhinitis and account for up to 40% of patients who present to otolaryngologic clinics. Symptoms of NAR are generally similar to allergic rhinitis but with persistent, non-variable symptoms elicited by irritants like smoke, odors, or changes in temperatures are more typical of NAR, but paroxysmal sneezing and nose itch are more characteristic of allergic rhinitis [8].

Nasal smear cytology has been identified as a quick, easy, and affordable diagnostic modality in chronic rhinitis. The presence of eosinophils in the nasal secretions, known as nasal smear eosinophilia, provides evidence of allergic inflammation and can differentiate allergic from non-allergic rhinitis. Nasal cytology is cheaper compared to skin tests or radioallergosorbent tests (RAST), results are given immediately, and it does not require referral to specialist centers because it can easily be done in general practice. Curiously, a subgroup of patients with non-allergic rhinitis has considerable eosinophilic invasion of the nasal secretions with negative tests to allergens, a clinical condition known as Eosinophilic Non-Allergic Rhinitis (ENR) or Non-Allergic Rhinitis with Eosinophilia Syndrome (NARES) [9]. Such patients respond to topical steroids, pointing out the clinical implications of diagnosing eosinophilic inflammation in therapeutic decision-making.

Owing to the shared symptoms of allergic and non-allergic rhinitis, there is still a need for accurate, convenient, and inexpensive diagnostic methods.

Nasal smear eosinophilia provides immediate information regarding the presence of inflammatory processes in the nose, allowing clinicians to distinguish allergic rhinitis, non-allergic rhinitis, and ENR, and select effective therapies accordingly. This clinical observation investigation tries to find out the correlation of the clinicopathological characteristics of nasal smear eosinophilia in allergic and non-allergic rhinitis patients and estimate the incidence of ENR among patients with chronic or recurrent nasal symptoms presenting to clinical observation. Through these correlations, the investigation tries to elevate diagnostic specificity, help in targeted therapy, and define management of chronic rhinitis in clinical practice with precision.

Methodology

Study Design: This was an observational study aimed at evaluating the clinicopathological correlation of nasal smear eosinophilia in allergic and non-allergic rhinitis patients.

Study Area: The study was conducted in the Department of ENT at a tertiary care hospital, Ahmedabad, Gujarat, India.

Study Duration: The study was carried out over a period of one year.

Study Population: The study included a total of 90 subjects, with 45 clinically diagnosed allergic rhinitis patients as cases and 45 age- and sex-matched healthy individuals as controls.

Inclusion Criteria

- Patients present with two or more nasal symptoms: nasal obstruction, rhinorrhea, itching, and sneezing.
- Intermittent allergic rhinitis: symptoms <4 days/week or <4 consecutive weeks.
- Persistent allergic rhinitis: symptoms >4 days/week and >4 consecutive weeks.
- Patients willing to provide informed consent.

Exclusion Criteria

- Patients with vasomotor rhinitis.
- Patients on antiallergic treatment.
- Patients with chronic nasal or systemic illnesses.
- Non-consenting individuals.

Sampling Technique: Subjects were selected using simple random sampling after applying the inclusion and exclusion criteria.

Data Collection: After implementing the inclusion and exclusion criteria, an evaluation of the eligible individuals was conducted based on a detailed history and ENT examination to confirm a diagnosis of allergic rhinitis or establish healthy controls. Demographic information, along with the duration of symptoms and severity, were documented, and the

nasal mucosal specimens were placed on cytology slides to count the eosinophils for both cases and controls.

Procedure: Nasal mucosal specimens were obtained by taking a scraping from the middle third of the inferior turbinate using a sterile cotton swab. In addition, a second sample was collected from each individual as a nasal swab, and nasal secretion was collected in a sterile container. The prepared smears were fixed with methanol for 10 minutes and then stained with either May-Grunwald or Giemsa stains. May-Grunwald stain was diluted to working strength using phosphate buffer and applied for 5 minutes, followed by Giemsa diluted with phosphate buffer for 10–15 minutes, all procedures performed at room temperature. The glass slides were then washed with phosphate buffer (pH 6.8), air-dried, and mounted with DPX. Slides were evaluated microscopically for the percentage of eosinophils per 100 leukocytes, and the eosinophil counts were compared between allergic rhinitis cases and controls.

Statistical Analysis: The data collected were analyzed using SPSS version 25.0. Descriptive statistics of mean, standard deviation, and percentage were performed to analyze the demographic and clinical variables. For the cross-sectional study comparing allergic rhinitis to controls, comparisons between cases and controls were conducted using Chi-square statistics for categorical variables and independent t-tests for continuous variables. A p-value of <0.05 was used to determine statistical significance."

Result

Table 1 shows the frequency distribution of nasal smear eosinophilia in 45 patients. Grade I ($\leq 5\%$ eosinophils) and Grade IV ($>50\%$ eosinophils) were each observed in 4 patients (8.9%), Grade II (6–10%) in 9 patients (20%), and Grade III (11–50%) in 28 patients (62.2%). This indicates that the majority of patients (62.2%) had moderate eosinophilia (Grade III) in their nasal smears.

Table 1: Frequency distribution of nasal smears for eosinophilia			
Eosinophil % in nasal smear	Grades	N	%
≤ 5	I	4	8.9
6–10	II	9	20
11–50	III	28	62.2
>50	IV	4	8.9
Total	–	45	100

Table 2 compares eosinophil counts between the study group ($n = 45$) and the control group ($n = 45$). In the study group, 4 patients (8.9%) had normal eosinophil counts ($\leq 5\%$), 9 patients (20%) were classified as doubtful (6–10%), and 32 patients (28 with 11–50% and 4 with $>50\%$) had pathological counts. In contrast, the control group had predominantly

normal counts, with 40 individuals (88.9%) showing $\leq 5\%$, 4 (8.9%) classified as doubtful, and only 1 individual (2.2%) with pathological counts (11–50%). This demonstrates a markedly higher prevalence of nasal eosinophilia in the study group compared to controls.

Table 2: Comparison of eosinophil count in study and control group			
No. of eosinophils (%)	Interpretation	Cases ($n=45$)	Control ($n=45$)
≤ 5	Normal	4	40
6–10	Doubtful	9	4
11–50	Pathological	28	1
>50	Pathological	4	0
Total	–	45	45

Table 3 presents the distribution of nasal smear eosinophilia across different disease types in 45 patients. Among patients with intermittent disease ($n = 22$), none had $\leq 5\%$ eosinophils, 2 (9.1%) had 6–10%, 18 (81.8%) had 11–50%, and 2 (9.1%) had $>50\%$. In the persistent disease group ($n = 23$), 4

patients (17.4%) had $\leq 5\%$, 7 (30.4%) had 6–10%, 10 (43.5%) had 11–50%, and 2 (8.7%) had $>50\%$. Overall, moderate eosinophilia (11–50%) was most common, especially in intermittent disease, indicating a higher eosinophilic burden in this group.

Table 3: Nasal smear eosinophilia vs. disease type					
Disease type	$\leq 5\%$	6–10%	11–50%	$>50\%$	Total
Intermittent	0	2	18	2	22
Persistent	4	7	10	2	23
Total	4	9	28	4	45

Discussion

The current study confirms a significant correlation between allergic rhinitis and nasal smear eosinophilia and provides further support to the accumulating evidence pointing towards eosinophilic infiltration as a characteristic of allergic inflammation of the nose. In our study population, moderate eosinophilia (11–50%) was the predominant result, occurring in 62.2% of subjects. The reduced representation of high eosinophil counts (>50%) and mild eosinophilia ($\leq 5\%$), with both occurring in 8.9% of cases, provides supportive evidence that moderate eosinophilia is the principal consideration in the examination of allergic rhinitis while the extremes for eosinophilia are rare. In contrast, the control group consistently exhibited eosinophil counts of $\leq 5\%$ in nearly all instances, highlighting a striking difference in the degree of inflammatory activity when compared to the affected individuals. This clear disparity underscores the role of eosinophils as a marker of allergic inflammation. The observed trend aligns closely with the findings of previous studies, which have suggested that the presence of nasal eosinophilia serves as a reliable and valuable indicator of allergic inflammation (Sood, 2005; Bhadari & Baldwa, 1976) [10,11]. These earlier investigations have provided strong evidence supporting the diagnostic utility of eosinophil measurement in differentiating allergic from non-allergic nasal conditions."

When we observe disease patterns, it appears as if intermittent allergic rhinitis is more strongly and consistently associated with moderate-to-high eosinophilia. Persistent allergic rhinitis had a wider range of eosinophil levels with lower overall eosinophilia. The noticeable difference in distribution patterns supports Miller et al.'s findings showing that intermittent allergic rhinitis showed a stronger association with nasal smear positivity. These findings could indicate that the episodic nature of intermittent diseases related to exposure to specific allergens leads to a greater eosinophil reaction which is likely both episodic and reactive upon exposure (Elkhalil et al., 1983) [12]. In addition, Bradding et al. (1995) [13] found in their study that nasal eosinophilia had distinct peaks based upon the season, with spring and summer consistently referred to as peak seasons. The association of eosinophilia during the spring and summer with peak environmental allergens, such as pollen, supports the notion that allergic rhinitis occurs cyclically, with periods of increased eosinophil activity concurrent with peak seasons of allergens.

In the present study, we found a slight rise in eosinophil numbers in the majority of participants, which closely follows the reports of Sood (2005) [10]. Sood found that 80% of patients with allergic rhinitis had eosinophils in nasal secretions, with only approximately 5% of control participants

demonstrating nasal eosinophilia. The significant and consistent difference we detected between the study and control groups further emphasizes nasal eosinophilia as a strong adjunct to diagnosis. This strengthens the argument that identifying eosinophils in a nasal smear offers high specificity for allergic rhinitis. As Salib et al. (2005) [14] noted, while it is a moderately sensitive test and must be interpreted in the context of a good clinical assessment, it has high specificity that is particularly helpful for confirming the diagnosis of allergic rhinitis.

An interesting observation from our data was that, even for those who are chronic allergic rhinitis sufferers, a substantial percentage had low eosinophil rates ($\leq 10\%$). This finding suggests that while the underlying condition is chronic, the eosinophil infiltration (or at least one sample) was not persistently elevated. This variability is consistent with the work of Bradding et al. (1995) [13], who showed eosinophil recruitment and accumulation is driven entirely by local cytokine signaling in the nasal mucosa. Thus, despite being a chronic disease, eosinophil levels may shift depending on local inflammatory condition, or erratic inflammatory conditions and physician baseline treatment may alter eosinophil levels. Additionally, this fluctuation of recruitment is consistent with the allergen rhinitis disease process which has a number of immunological pathways and local tissue influences.

Our study also supports the possible clinical application of eosinophilia on nasal smear as an assessment and diagnostic tool. Being inexpensive, repeatable, and non-invasive makes it a suitable choice for further evaluation and subsequent assessment of the diagnosis, at least in resource-poor environments. In contrast, Elkhalil et al. (1983) [15] raised the issue of use of nasal cytology as a monitoring tool for the assessment of treatment effect of children with perennial allergic rhinitis. Assessment of the trend in eosinophils may assist with treatment decisions on for example, the use of corticosteroids or with respect to predicting flare-ups of allergic rhinitis.

There are clearly limitations of the sensitivity of nasal smear results that have been pointed out in the literature. Sood et al. (2005) found sensitivity was as low as 18% with a specificity of 96% [10], thus the presence of eosinophilia is a valuable indicator of allergic rhinitis, whereas the absence of eosinophilia does not allow consensus exclusion of the diagnosis. This again highlights the value of correlating smear results with clinical history, prick testing, or serum IgE level as a general indication of the patients' diagnosis for allergic rhinitis [11,16] (Bhadari & Baldwa, 1976; Jirapongsananuruk & Vichyanond, 1998)

Additionally, we have found that there might be a differential association of eosinophilia with the

nature of nasal discharge. The cases with watery rhinorrhea mostly demonstrated greater to severe eosinophilia, which corresponds with the pathophysiologic understanding that mast cell activation due to IgE causes an increase in vascular permeability and eosinophil recruitment (Bradding et al., 1995) [13]. This is clinically useful in supporting prior reports that clinical phenotyping + cytologic assessment enhances diagnostic sensitivity and guides individualized treatment approaches.

Overall, the current study demonstrates a substantial correlation of nasal smear eosinophilia with allergic rhinitis clinically and pathologically. The study confirms earlier studies with high specificity and moderate sensitivity of nose smears in detecting allergic inflammation, particularly in those with intermittent disease (Sood, 2005) [10]. While persistent rhinitis has a broad eosinophilic spectrum, the presence of moderate eosinophilia in intermittent rhinitis can potentially serve as a reliable diagnostic marker. Nasal smear eosinophilia is a quick and non-invasive procedure making it an ideal screening, diagnostic and follow-up test, especially in an outpatient basis. Standardization studies to set thresholds of eosinophils and correlation of cytologic data with molecular biomarkers in future studies will certainly enhance specificity across the spectrum of allergic and non-allergic rhinitis.

Conclusions

The present study provides additional evidence of a strong relationship between eosinophilia in nasal smears and the differentiation of allergic rhinitis (AR) and non-allergic rhinitis (NAR). The analysis of nasal smears indicated that a considerable proportion of patients exhibited eosinophilia, reflecting an underlying pathological inflammatory response, while the vast majority of controls consistently displayed normal eosinophil counts. When eosinophil count was compared by rhinitis subtype, eosinophilia was present in both intermittent and persistent rhinitis, but exhibited different distributions: in intermittent rhinitis, the eosinophilia was larger in amplitude and episodic, whereas in persistent rhinitis, eosinophilia could be detected and was more evenly spread, but lower in concentration. The findings indicate that eosinophil counts are not only proportional to the presence of allergic disease but also consistent temporally with the symptomatic pattern of rhinitis presentation, as well as with potential variations in exposure/allergen or immune response. Taken together, the data support nasal smear cytology as an objective and clinically relevant diagnostic modality for the differentiation of AR and NAR. In addition, nasal eosinophilia is an important marker for pathophysiology of rhinitis and serves as an informative aspect of the comprehensive clinical evaluation in the diagnosis and management of patients with nasal symptoms.

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