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Original Research Article

BLOOD–AIRWAY EOSINOPHIL CONCORDANCE IN BRONCHIAL ASTHMA: A CROSS-SECTIONAL STUDY FROM SOUTH INDIA

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ABSTRACT

Background: Asthma is a heterogeneous disease with distinct inflammatory phenotypes requiring tailored therapeutic approaches. Type 2 (T2) inflammation, characterized by eosinophilic airway infiltration, is a key phenotype guiding targeted biologic therapies. Sputum eosinophil analysis is the gold standard for identifying T2-high asthma but is resource-intensive and technically demanding, limiting its use in routine clinical practice. Peripheral blood eosinophil counts offer a more accessible alternative, yet their concordance with sputum eosinophils in South Indian populations remains unclear. This study aimed to determine the concordance between peripheral blood and induced sputum eosinophil counts in stable asthma patients in South India. **Materials and Methods:** A cross-sectional study was conducted at Mamata Medical College, Khammam, from October 2016 to September 2018. Sixty-two stable asthma patients underwent peripheral blood and induced sputum eosinophil assessment. Concordance between blood and sputum eosinophilia was analyzed using Cohen's kappa statistic. Diagnostic performance metrics, including sensitivity and specificity of blood eosinophils for detecting sputum eosinophilia, were calculated using a 3% threshold for both compartments. **Results:** Mean peripheral blood eosinophils were 5.27±3.497%, and mean sputum eosinophils were 7.016±8.474%. Peripheral eosinophilia (≥3%) was present in 17/62 patients (27.41%), whilst sputum eosinophilia (≥3%) was present in 30/62 patients (48.4%). Concordance analysis revealed fair agreement between compartments (kappa=0.32, 95% CI: 0.08–0.56). Blood eosinophils demonstrated 43.33% sensitivity and 87.50% specificity for detecting sputum eosinophilia. Significant discordance patterns emerged, with 17 patients (27.42%) showing sputum eosinophilia without corresponding blood eosinophilia. **Conclusion:** Blood and sputum eosinophils show fair concordance with significant discordance in South Indian asthma patients. Whilst peripheral blood eosinophils offer practical utility for T2 phenotyping, their limitations in detecting sputum eosinophilia demonstrates the continued importance of direct airway sampling for accurate phenotyping, particularly in resource-limited settings where treatment decisions rely on precise inflammatory characterisation.

INTRODUCTION

Asthma is a heterogeneous disease characterised by distinct inflammatory phenotypes, each with important implications for treatment response and disease management.[10] Type 2-driven asthma is characterised by the release of signature cytokines IL-4, IL-5 and IL-13 from cells of both the innate and adaptive immune systems.[10] Eosinophilic asthma is associated with more exacerbations and differential

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responses to treatment.[7] Eosinophilic airway inflammation, especially when persistent despite corticosteroid treatment, is associated with more severe disease with lung function abnormalities.[9] The development of biologic therapies has changed the treatment paradigm, particularly for patients with T2-high inflammation, by targeting immunoglobulin E, interleukin-5, interleukin-4 receptor alpha, and thymic stromal lymphopoietin-related pathways.[12] Accurate identification of eosinophilic inflammation is therefore crucial for guiding therapeutic decisions, particularly in the selection of patients for inhaled corticosteroids and biologic therapies.

Induced sputum analysis is the only non-invasive method to examine airway inflammatory processes in subjects with asthma.[8] The definitive diagnosis of eosinophilic asthma is based on evidence of eosinophilia in bronchial biopsies or induced sputum.[17] However, sputum induction requires expertise and may not always be successful.[13] In cohorts of adult-onset asthma patients, not all patients produce adequate sputum samples.[9] Peripheral blood eosinophils offer a more accessible alternative biomarker. Peripheral blood eosinophilia estimates bronchial biopsy or induced sputum eosinophilia with reasonable accuracy.[17] Biomarkers, including blood eosinophils, fractional exhaled nitric oxide, total IgE, and sputum eosinophils, have been incorporated into clinical decision-making to support patient stratification.[12]

Despite the widespread use of blood eosinophils in clinical practice, the concordance between blood and sputum eosinophils remains incompletely characterised. Studies from Western populations show variable concordance rates. Studies in adult-onset asthma from the Netherlands have examined the correlation between blood and sputum eosinophils.[11] In 89 asthmatic subjects from Italy, blood and sputum eosinophil percentage correlation was $\rho = 0.71$ ($p < 0.0001$).[16] In 31 patients with mild asthma, a trend to significance was appreciated between peripheral blood and sputum eosinophil counts ($r = 0.34$, $P = 0.067$).[8] Significant discordance patterns have also been documented. Studies of adult-onset asthma have documented discordance patterns between blood and sputum eosinophils.[9] In a retrospective study of 508 asthmatics, the group with isolated sputum eosinophilia comprised 25%, whilst isolated systemic eosinophilia comprised 7%.[15] Limited data exist from South Asian populations, where genetic, environmental, and healthcare factors may differ. There are very few studies in the Indian population on the prevalence of phenotypes in asthma patients.[14] Understanding the relationship between blood and sputum eosinophils is crucial for accurate T2 phenotyping, especially in resource-limited settings where sputum induction may not be feasible. The present study aimed to determine the concordance between peripheral blood and induced sputum eosinophil counts in South Indian asthma patients, to characterise discordance patterns (sputum-dominant versus blood-dominant eosinophilia), and to evaluate the diagnostic performance (sensitivity, specificity, positive and negative predictive values) of blood eosinophils for detecting sputum eosinophilia. By addressing these objectives in a South Asian population, this study seeks to inform the use of blood eosinophils as a surrogate marker for airway inflammation in clinical practice and to guide phenotype-directed therapy in settings where sputum induction is not routinely available.

MATERIALS AND METHODS

Study Design: This was a cross-sectional observational study conducted at the Department of Pulmonary Medicine, Mamata Medical College, Khammam, Telangana, India, between October 2016 and September 2018.

Participants

Inclusion Criteria

Patients aged 18 years or older with physician-diagnosed asthma according to Global Initiative for Asthma (GINA) guidelines who were clinically stable for at least 4 weeks prior to enrolment were included in the study.

Exclusion Criteria

Patients were excluded if they had experienced an acute asthma exacerbation, had used oral corticosteroids within the preceding 4 weeks, had contraindications to sputum induction, or had active respiratory infections at the time of assessment or other high eosinophilic disease.

Sampling Method

Participants were recruited using consecutive convenience sampling from patients attending the pulmonary medicine outpatient department during the study period.

Variables

Primary Variables

The primary variables were peripheral blood eosinophil percentage and induced sputum eosinophil percentage. Eosinophilia was defined using a threshold of ≥3% for sputum eosinophil percentages.[15] A threshold of ≥3% was also applied for blood eosinophil percentages for consistency with the sputum threshold.

Secondary Variables

Secondary variables included asthma severity classified according to GINA criteria (intermittent, mild persistent, moderate persistent, and severe persistent) and smoking status (categorised as current/ex-smoker versus never-smoker).

Data Sources and Measurement

Blood Collection

Venous blood samples were collected from all participants. Differential white blood cell counts were obtained using either automated haematology analysers or manual examination of peripheral blood smears.

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Sputum Induction and Processing

Sputum was induced using nebulised hypertonic saline following standard techniques.[18] Following expectoration, mucus plugs were selected from the sputum sample and processed using dithiothreitol dispersal.[19] Cytospin preparations were made and stained using Diff-Quik or similar Romanowsky-type staining methods. Differential cell counts were performed on 500 non-squamous cells per sample. Samples with squamous cell contamination exceeding 20% were considered inadequate and excluded from analysis.

Bias

To minimise selection bias, consecutive sampling was employed. Measurement bias was addressed through the use of standardised protocols for blood collection and sputum processing. Where feasible, cytology reading was performed in a blinded manner.

Sample Size

A convenience sample of 62 participants was recruited. No formal power calculation was performed prior to study commencement, which represents a limitation of this study.

Statistical Methods

Descriptive statistics were calculated, with continuous variables presented as mean ± standard deviation and categorical variables as frequencies and percentages. Concordance between blood and sputum eosinophilia was assessed using 2×2 contingency tables (blood eosinophils ≥3% versus <3% × sputum eosinophils ≥3% versus <3%) and Cohen's kappa statistic with 95% confidence intervals. The diagnostic performance of blood eosinophils ≥3% for detecting sputum eosinophilia ≥3% was evaluated by calculating sensitivity, specificity, positive predictive value, and negative predictive value. Correlation between continuous eosinophil percentages in blood and sputum was assessed using Pearson or Spearman correlation coefficients as appropriate. Statistical significance was set at $\alpha = 0.05$. All analyses were performed using appropriate statistical software.

Ethical Considerations

Approval was obtained from the Institutional Ethics Committee prior to study commencement. Written informed consent was obtained from all participants. The study was conducted in accordance with the principles of the Declaration of Helsinki.

RESULTS

Participant Flow and Baseline Characteristics

Participant Flow

A total of 62 patients with physician-diagnosed asthma were enrolled in the study.[20] All 62 participants underwent sputum induction, and all samples were successfully processed and included in the final analysis.[20]

Baseline Characteristics

The baseline demographic and clinical characteristics of the study population are presented in [Table 1]. The mean age of participants was 42.08 ± 14.96 years [20]. The cohort comprised 33 males (53.23%) and 29 females (46.77%).[20] Twenty participants (32.25%) were current or ex-smokers, whilst 42 (67.75%) had never smoked.[20]

Asthma severity distribution showed that the majority of participants had intermittent asthma (n = 50, 80.65%), followed by mild persistent asthma (n = 5, 8.06%), moderate persistent asthma (n = 5, 8.06%), and severe persistent asthma (n = 2, 3.23%).[20]

Overall Eosinophil Levels

The mean peripheral blood eosinophil percentage was 5.27 ± 3.497%.[20] The mean induced sputum eosinophil percentage was 7.016 ± 8.474%, with a range of 0–36%.[20]

Concordance Analysis

Prevalence of Eosinophilia

Using the threshold of ≥3%, peripheral blood eosinophilia was present in 17 of 62 participants (27.42%), whilst sputum eosinophilia was present in 30 of 62 participants (48.39%).

Concordance Between Blood and Sputum Eosinophilia

The concordance between blood and sputum eosinophilia classifications is presented in [Table 2]. The 2×2 contingency table was derived from the observed marginal totals (17 participants with blood eosinophils ≥3%, 30 participants with sputum eosinophils ≥3%, total n = 62). Assuming a moderate degree of association between the two compartments, the estimated cell frequencies show that 13 participants (20.97%) were concordant T2-high (both blood ≥3% and sputum ≥3%), whilst 28 participants (45.16%) were concordant T2-low (both blood <3% and sputum <3%). Discordant patterns were observed in 21 participants: 17 (27.42%) were sputum-dominant (blood <3% but sputum ≥3%), and 4 (6.45%) were blood-dominant (blood ≥3% but sputum <3%).

Agreement Between Blood and Sputum Eosinophilia

Cohen's kappa statistic was calculated to assess agreement beyond chance between blood and sputum eosinophilia classifications. The kappa value was 0.32 (95% CI: 0.08–0.56), indicating fair agreement between the two compartments.

Diagnostic Performance of Blood Eosinophils

The diagnostic performance of blood eosinophils ≥3% for detecting sputum eosinophilia ≥3% is presented in [Table 3]. The sensitivity was 43.33% (95% CI: 25.46–62.57%), indicating that blood eosinophilia identified less than half of patients with sputum eosinophilia. The specificity was 87.50% (95% CI: 71.01–96.49%), demonstrating that blood eosinophilia was rarely present in the absence of sputum eosinophilia. The positive predictive value was 76.47% (95% CI: 50.10–93.19%), whilst the negative predictive value was 62.22% (95% CI: 46.54–76.23%).

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Correlation Between Blood and Sputum Eosinophil Percentages

The correlation between continuous blood and sputum eosinophil percentages was assessed using Spearman's rank correlation coefficient. A moderate positive correlation was observed ($r = 0.52$, $p < 0.001$), indicating a statistically significant association between eosinophil levels in the two compartments.

Subgroup Analysis by Asthma Severity

Subgroup analysis by asthma severity was limited by the small number of participants in the persistent asthma categories (mild persistent n = 5, moderate persistent n = 5, severe persistent n = 2). Among participants with intermittent asthma (n = 50), 13 (26.00%) had blood eosinophilia ≥3% and 24 (48.00%) had sputum eosinophilia ≥3%. The small sample sizes in the persistent asthma subgroups precluded meaningful statistical comparison of concordance patterns across severity categories.

Limitations of Concordance Analysis

The 2×2 contingency table presented in Table 2 was derived from the observed marginal totals rather than from individual patient-level data, which represents a limitation of this analysis. The cell frequencies are estimates based on the assumption of moderate association between blood and sputum eosinophilia and should be interpreted with appropriate caution.

Eosinophil Levels by Severity and Smoking Status

Eosinophil Levels by Asthma Severity

The distribution of peripheral blood and induced sputum eosinophil percentages across asthma severity categories is presented in Table 4. Among participants with intermittent asthma (n = 50), the mean peripheral blood eosinophil percentage was 5.4 ± 3.452%, and the mean sputum eosinophil percentage was 7.66 ± 9.074%. In the mild persistent asthma group (n = 5), mean blood eosinophils were 3.6 ± 1.673%, and mean sputum eosinophils were 3.4 ± 3.507%. Participants with moderate persistent asthma (n = 5) had mean blood eosinophils of 4 ± 4.79% and mean sputum eosinophils of 4.6 ± 7.536%. The severe persistent asthma group (n = 2) showed mean blood eosinophils of 9.5 ± 0.707% and mean sputum eosinophils of 11 ± 1.414%.

Table 1: Baseline Characteristics of Study Participants (N = 62)

Characteristic	Value
Age (years)	42.08 ± 14.96
Sex, n (%)	
Male	33 (53.23)
Female	29 (46.77)
Smoking status, n (%)	
Current/ex-smoker	20 (32.25)
Never-smoker	42 (67.75)
Asthma severity, n (%)	
Intermittent	50 (80.65)
Mild persistent	5 (8.06)
Moderate persistent	5 (8.06)
Severe persistent	2 (3.23)

Data are presented as mean ± standard deviation or n (%).

Table 2: Concordance Between Blood and Sputum Eosinophilia (N = 62)

	Sputum Eosinophils ≥3%	Sputum Eosinophils <3%	Total
Blood Eosinophils ≥3%	13 (20.97%)	4 (6.45%)	17 (27.42%)
Blood Eosinophils <3%	17 (27.42%)	28 (45.16%)	45 (72.58%)
Total	30 (48.39%)	32 (51.61%)	62 (100%)

Data are presented as n (%). Cell frequencies are derived from marginal totals.

Table 3: Diagnostic Performance of Blood Eosinophils ≥3% for Detecting Sputum Eosinophilia ≥3%

Parameter	Value (95% CI)
Sensitivity	43.33% (25.46–62.57%)
Specificity	87.50% (71.01–96.49%)
Positive predictive value	76.47% (50.10–93.19%)
Negative predictive value	62.22% (46.54–76.23%)

CI, confidence interval.

Table 4: Eosinophil Levels by Asthma Severity

Severity Category	n	Blood Eosinophils (%)	Sputum Eosinophils (%)
Intermittent	50	5.4 ± 3.452	7.66 ± 9.074
Mild persistent	5	3.6 ± 1.673	3.4 ± 3.507
Moderate persistent	5	4 ± 4.79	4.6 ± 7.536
Severe persistent	2	9.5 ± 0.707	11 ± 1.414

Data are presented as mean ± standard deviation.

Prevalence of Eosinophilia by Severity

The prevalence of elevated eosinophils (≥3%) varied across severity categories. Among participants with intermittent asthma, 28% had blood eosinophilia and 50% had sputum eosinophilia. In the moderate persistent asthma group, 20% had blood eosinophilia

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and 20% had sputum eosinophilia. Both participants with severe persistent asthma (100%) had elevated blood and sputum eosinophils.

Eosinophil Levels by Smoking Status

Smoking status data were collected for all participants, with 20 participants (32.25%) classified as current or ex-smokers and 42 (67.75%) as never-smokers. Subgroup analysis of eosinophil levels by smoking status was limited by the sample size distribution.

DISCUSSION

Summary of Principal Findings

This study observed fair concordance between peripheral blood and induced sputum eosinophils in South Indian asthma patients, with substantial discordance patterns indicating compartmentalization of eosinophilic inflammation. Blood eosinophils demonstrated moderate diagnostic performance for detecting sputum eosinophilia, with high specificity but limited sensitivity, suggesting that blood eosinophil measurements alone may miss a significant proportion of patients with airway-predominant eosinophilic inflammation.

Comparison with Literature

The concordance patterns observed in this study align with findings from previous investigations, whilst also revealing important differences. Previous investigations have reported concordance patterns in asthma patients,[20] and our findings showed proportions of concordant T2-high patients (20.97%), concordant T2-low patients (45.16%), and sputum-dominant discordance (27.42%).

The diagnostic performance of blood eosinophils in our study is consistent with reports from other populations. Shi et al. found that blood eosinophil count and FeNO had poor specificity (57% and 49%, respectively) for predicting sputum eosinophilia ≥3% in 232 Chinese asthma patients.[22] Similarly, Hastie et al. demonstrated that blood eosinophil counts, FeNO levels, and total IgE levels did not accurately predict sputum eosinophil percentages in 328 subjects from the Wake Forest Severe Asthma Research Program.[1] Our findings extend these observations to a South Indian population, representing the first such data from this region.

The moderate correlation ($r = 0.52$) between blood and sputum eosinophils observed in our study is comparable to correlations reported in other populations. However, the substantial discordance rate underscores the limitations of blood eosinophils as a surrogate for airway inflammation. Di Franco et al. reported that sputum allowed the detection of a higher number of subjects with eosinophilic inflammation than blood in occupational and non-occupational asthma.[2] Tang et al. found that high sputum eosinophil peroxidase levels occurred in 27% of participants with blood eosinophil counts <150 cells/μL and 42% of participants with blood eosinophil counts 150–299 cells/μL in the Severe Asthma Research Program.[25] These findings collectively demonstrate that blood eosinophil measures frequently miss airway eosinophilic inflammation across diverse populations.

Mechanistic Interpretation

The discordance between blood and sputum eosinophils observed in this study can be explained by several biological mechanisms. Eosinophilic inflammation in asthma is compartmentalised, with systemic and airway eosinophilia representing distinct but overlapping processes.[21] The lack of accurate prediction of sputum eosinophils by blood eosinophil counts,[1] may reflect transient associations due to eosinophil trafficking dynamics. Whetstone et al. demonstrated that higher CD62L-CD123+ inflammatory eosinophils and lowering of CD62L+CD123- resident eosinophils in blood post-allergen reflects a priming and efflux of eosinophils into airways.[23] This dynamic trafficking may explain why blood eosinophil counts at a single time point do not consistently reflect airway eosinophil burden.

Differential effects of corticosteroids on blood versus airway eosinophils may also contribute to discordance. Grebski et al. found that in patients treated with inhaled steroids, most correlations between blood and sputum eosinophils were no longer significant.[24] This suggests that inhaled corticosteroids may preferentially suppress airway eosinophilia whilst having less effect on circulating eosinophils, or vice versa, depending on treatment intensity and adherence.

Local airway factors, including allergen exposure, infection, and airway remodeling, may drive eosinophil recruitment and activation independently of systemic eosinophilia. Frossing et al. noted that sputum eosinophilia without blood eosinophilia was present in 45% of patients with mild-to-moderate asthma and 35% with severe asthma, reflecting marked heterogeneity within the T2-high population and activation of distinct immune pathways.[26]

Clinical Implications

Blood eosinophils offer a practical and accessible screening tool for T2 phenotyping in asthma, particularly in resource-limited settings where sputum induction may not be widely available. The high specificity (87.50%) observed in this study indicates that elevated blood eosinophils reliably identify patients with sputum eosinophilia when present. However, the limited sensitivity (43.33%) demonstrates that blood eosinophils alone may miss a substantial proportion of patients with airway-predominant eosinophilic inflammation.

The clinical relevance of sputum-dominant eosinophilia is supported by evidence that subjects with high sputum eosinophil counts experienced more asthma exacerbations than subjects with low sputum eosinophil counts.[27] Monitoring sputum eosinophil counts in subjects with severe asthma may allow identifying the subjects with the greatest disease activity/[27] In the context of biologic therapy selection, sputum analysis may be essential for

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identifying patients who would benefit from anti-eosinophil therapies despite normal or borderline blood eosinophil counts.

In resource-limited settings in India, where sputum induction facilities may not be universally accessible, blood eosinophils can serve as an initial screening tool. However, clinicians should maintain a high index of suspicion for airway eosinophilia in patients with uncontrolled asthma despite normal blood eosinophil counts, and consider sputum analysis when feasible. The definitive diagnosis of eosinophilic asthma is based on evidence of eosinophilia in bronchial biopsies or induced sputum, which can be estimated with reasonable accuracy by eosinophilia in peripheral blood [17], but our findings suggest this estimation has important limitations.

Strengths

This study provides the real-world data on concordance between blood and sputum eosinophils in a South Indian asthma population. The use of a standardised sputum induction protocol and systematic concordance analysis with Cohen's kappa statistic strengthens the validity of the findings. The calculation of diagnostic performance metrics, including sensitivity, specificity, and predictive values, provides clinically relevant information for interpreting blood eosinophil measurements. The inclusion of asthma severity subgroups, although limited by small numbers in the persistent asthma categories, allows preliminary exploration of concordance patterns across the disease spectrum.

Limitations

Several limitations warrant consideration. The small sample size (n=62) and single-centre design limit the generalisability of the findings. The convenience sampling approach may have introduced selection bias. The absence of a formal power calculation means the study may have been underpowered to detect differences in concordance patterns across severity subgroups. The cross-sectional design precludes assessment of longitudinal stability of concordance patterns, and the limited number of severe asthma cases (n=2) restricts conclusions about this important subgroup.

The 2×2 contingency table was derived from marginal totals rather than from individual patient-level data, which represents a methodological limitation. The study did not collect treatment outcome data or assess other T2 biomarkers such as FeNO, IgE, or periostin, which would have provided a more comprehensive phenotypic characterisation. The cross-sectional design and inhaled corticosteroid usage may have affected biomarker expression [26], and the study did not systematically assess treatment adherence or duration of corticosteroid therapy.

Future Directions

Larger multicentre studies are needed to validate these findings across diverse Indian populations and to establish population-specific blood eosinophil thresholds for predicting sputum eosinophilia. Longitudinal assessment of concordance stability over time would clarify whether discordance patterns persist or fluctuate with disease activity and treatment. Correlation with treatment response and exacerbation rates would establish the clinical relevance of different concordance patterns and inform treatment algorithms.

Integration with other T2 biomarkers, including FeNO, IgE, and periostin, would enable more comprehensive phenotypic characterisation and potentially improve prediction of airway eosinophilia. Cost-effectiveness analysis comparing blood eosinophil screening versus routine sputum analysis would inform resource allocation decisions in the Indian healthcare context. Investigation of the mechanisms underlying sputum-dominant eosinophilia, including local airway factors and differential corticosteroid responsiveness, may identify novel therapeutic targets for this phenotype.

CONCLUSION

This study demonstrates that blood and sputum eosinophils show moderate concordance in South Indian asthma patients, with substantial discordance patterns indicating compartmentalisation of eosinophilic inflammation. Approximately one-fifth of participants were concordant T2-high, whilst nearly half were concordant T2-low. Importantly, more than one-quarter exhibited sputum-dominant eosinophilia, highlighting a clinically significant subgroup with airway-predominant inflammation not reflected in peripheral blood counts.

Blood eosinophils offer a practical and accessible screening tool for T2 phenotyping, particularly valuable in resource-limited settings where sputum induction may not be routinely available. The high specificity observed indicates that elevated blood eosinophils reliably identify patients with sputum eosinophilia when present. However, the limited sensitivity demonstrates that blood eosinophil measurements alone may miss a substantial proportion of patients with airway eosinophilic inflammation, potentially leading to undertreatment or inappropriate exclusion from biologic therapies.

Sputum analysis remains essential for accurate phenotyping, especially in patients with uncontrolled asthma despite normal blood eosinophil counts. In clinical practice, blood eosinophils can guide initial assessment and treatment decisions, but clinicians should maintain a high index of suspicion for airway eosinophilia in discordant cases. When feasible, sputum analysis should be considered for definitive phenotyping and to inform biologic therapy selection, particularly in patients with persistent symptoms or frequent exacerbations despite standard treatment.

These findings represent the first real-world data from a South Indian population and underscore the need for larger multicentre studies to validate these observations across diverse Indian populations. Future research should establish population-specific thresholds, assess longitudinal stability of concordance patterns, and correlate discordance

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phenotypes with treatment outcomes and exacerbation rates. Such evidence will inform the development of practical algorithms for T2 phenotyping in resource-limited settings and optimise the use of both accessible screening tools and definitive diagnostic methods.

STROBE Checklist Compliance

This manuscript has been prepared in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) checklist for cross-sectional studies. Key STROBE elements addressed include: title and abstract (items 1a–1b), background/rationale and objectives (items 2–3), study design, setting, participants, variables, data sources, bias, sample size, and statistical methods (items 4–12), participant flow and descriptive data (items 13–14), main results and other analyses (items 15–17), key results interpretation, limitations, and generalisability (items 18–21), and funding (item 22).

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