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Analysis of Peripheral Blood T Lymphocyte Subsets in Children with Adenoid Hypertrophy and Allergic Rhinitis

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Abstract

This study sought to investigate the impact of adenoid hypertrophy (AH), allergic rhinitis (AR) and their coexistence on peripheral blood T lymphocyte subsets in pediatric patients. A cohort of 916 children aged 3 to 14 years, recruited from hospital visits between January and December 2022, was divided into four groups: AH only [n=316], AR only [n=176], AH+AR [n=379] and healthy controls [n=145]. T lymphocyte subsets were assessed via flow cytometry. Compared with the control group, all three disease groups exhibited significantly elevated absolute counts of $CD3^+$ and $CD8^+$ T cells ($P < 0.0083$), although no notable differences were observed in the proportional distribution of these subsets. The AR group demonstrated a significantly higher absolute $CD4^+$ T cell counts relative to both the control and AH groups ($P < 0.0083$), while the AH group had a greater percentage of $CD8^+$ T cells than the AR group. These findings indicate that both AH and AR contribute to an increase in total peripheral T lymphocyte counts. AR more substantially affects $CD4^+$ T cells, yet across all conditions, the predominant effect is an expansion of the overall lymphocyte population rather than an alteration in the proportional balance among T cell subsets.

Keywords: Adenoid Hypertrophy, Allergic Rhinitis, Children, Non-parametric Test, T Lymphocyte Subsets.

Introduction

Adenoid hypertrophy (AH) and allergic rhinitis (AR) are frequently encountered conditions in childhood and often coexist as comorbid disorders that may significantly affect respiration, sleep quality and overall immune function in affected children (1, 2). Adenoid hypertrophy, characterized by abnormal enlargement of lymphoid tissue in the nasopharynx, can lead to upper airway obstruction, mouth breathing, recurrent infections and sleep-disordered breathing. Allergic rhinitis, on the other hand, is a chronic inflammatory disease of the nasal mucosa triggered by immunoglobulin E (IgE)-mediated hypersensitivity reactions to environmental allergens. These two conditions frequently overlap in pediatric populations and their coexistence may exacerbate airway inflammation and immune dysregulation, thereby increase disease burden and negatively influence children's quality of life and development (3).

As central components of the adaptive immune system, T lymphocytes play critical roles in immune surveillance, pathogen clearance and the regulation of inflammatory responses. In the context of allergic diseases and chronic inflammatory conditions, alterations in the composition and functional status of T lymphocyte subsets are frequently observed (4). Among these subsets, $CD8^+$ T lymphocytes—also referred to as cytotoxic T cells—serve as key effector cells responsible for eliminating virus-infected or damaged cells. Their functional repertoire is multifaceted. First, $CD8^+$ T cells secrete pro-inflammatory cytokines such as interferon-γ (IFN-γ) and tumor necrosis factor-α (TNF-α), which enhance antimicrobial activity and activate macrophages, thereby amplifying immune responses. Second, these cells participate in immune regulation by influencing the balance between Th1 and Th2 responses and potentially limiting excessive immune activation. Third, a proportion of activated $CD8^+$ T cells differentiate into long-lived memory cells that provide rapid and robust immune responses upon re-exposure to previously encountered antigens (5). The adenoids are part of the nasopharynx-associated lymphoid tissue and represent an important component of the mucosal immune system in the upper airway.

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They serve as an initial immunological represents pathological lymphoid hyperplasia driven by persistent inflammatory stimulation. Within this pathological process, $CD8^+$ T cells appear to play a complex and potentially dual role. On the one hand, they act as defenders by combating chronic or latent viral infections—such as Epstein–Barr virus (EBV) or adenoviruses—through recruitment and activation to eliminate virus-infected epithelial cells. In addition, $CD8^+$ T cells may contribute to the control of bacterial biofilms, including those formed by Streptococcus pneumoniae, through cytotoxic activity or cytokine secretion (6). On the other hand, excessive or prolonged activation of $CD8^+$ T cells may contribute to local inflammation and tissue damage. Persistent cytotoxic activity may trigger repeated cycles of tissue injury and repair under chronic antigenic stimulation, promoting lymphoid hyperplasia and fibrosis. Furthermore, the pro-inflammatory cytokines IFN-γ and TNF-α released by these cells can upregulate adhesion molecules, recruit additional lymphocytes, stimulate fibroblast proliferation and interact with other immune cells—such as Th17 cells—to amplify local inflammatory responses. In certain circumstances, dysregulated $CD8^+$ T-cell activity may also interfere with regulatory T-cell function, thereby perpetuating chronic immune activation and tissue remodeling.

$CD4^+$ T lymphocytes, commonly referred to as helper T cells, function as the "command center" of the adaptive immune system. Unlike $CD8^+$ T cells, $CD4^+$ T cells do not directly kill infected cells but orchestrate immune responses by regulating the activity of nearly all other immune cell types. Naïve $CD4^+$ T cells can differentiate into several distinct effector subsets depending on the surrounding cytokine environment. Th1 cells, characterized by the secretion of IFN-γ and interleukin-2 (IL-2), primarily mediate cellular immunity against intracellular pathogens. Th2 cells produce cytokines such as IL-4, IL-5 and IL-13 and play a central role in humoral immunity and allergic inflammation by promoting IgE production and activating eosinophils and mast cells. Th17 cells, which secrete IL-17 and IL-22, contribute to host defense against extracellular pathogens but are also involved in pro-inflammatory processes associated with autoimmune and chronic inflammatory diseases. In contrast, regulatory T cells (Tregs), characterized by the expression of Foxp3 and the secretion of IL-10 and transforming growth factor-β (TGF-β), function as critical immune suppressors that limit excessive immune activation and maintain immune tolerance (7, 8).

Allergic rhinitis is fundamentally driven by Th2-type $CD4^+$ T-cell responses that lead to IgE production, eosinophilic inflammation and hypersensitivity to environmental allergens. In both AR and AH, $CD4^+$ T cells are believed to play central roles in disease pathogenesis through distinct but overlapping immunological pathways. In AR, Th2-dominated immune responses drive allergic inflammation, whereas in AH, inflammatory pathways involving Th1 and Th17 cells may contribute to persistent immune activation and lymphoid tissue proliferation (9). The interaction between these immune pathways may partly explain the high rate of comorbidity between AH and AR observed in pediatric populations.

Despite increasing recognition of the immunological mechanisms underlying these conditions, there remains a lack of comprehensive investigations systematically comparing peripheral blood T lymphocyte subsets among children with isolated adenoid hypertrophy, isolated allergic rhinitis, their coexistence and healthy controls. Most existing studies have focused primarily on local tissue immune responses within adenoid tissues or nasal mucosa, while relatively few have examined systemic immune characteristics reflected in peripheral blood lymphocyte populations (10). Understanding these systemic immune changes may provide valuable insights into the broader immunological landscape associated with these diseases and may help identify potential immunological markers relevant to diagnosis, disease monitoring, or therapeutic targeting.

Therefore, the present study aimed to characterize the absolute counts and percentages of peripheral blood T lymphocyte subsets in four pediatric populations: children with isolated adenoid hypertrophy, children with allergic rhinitis, children with both conditions and healthy controls. By comparing these groups, this study seeks to explore how different disease states influence systemic cellular immune parameters and to provide further insight into the immunological mechanisms underlying these common pediatric airway diseases.

Methodology

Patient Selection and Recruitment

To minimize selection bias, a consecutive recruitment strategy was employed. All children aged 3 to 14 years who presented to the Department of Otolaryngology at the Affiliated Hospital of North Sichuan Medical College (approximate GPS coordinates 106.10°E, 30.83°N) with symptoms suggestive of upper respiratory disorders (e.g., nasal obstruction, rhinorrhea, snoring, or mouth breathing) between January 1 and December 31, 2022, were initially screened for potential eligibility. This approach aimed to capture a representative sample of the clinical population rather than a selected subgroup. The screening was based on chief complaints and preliminary history. Subsequently, each child underwent standardized diagnostic evaluations to confirm the presence of AH, AR, both, or neither. Only those meeting the strict inclusion and exclusion criteria (detailed below) were enrolled into the respective study groups. The healthy control group was recruited from children attending the hospital for routine health check-ups during the same period, ensuring they had no history of allergic or chronic inflammatory diseases. This consecutive enrollment process helps reduce referral bias and increases the generalizability of the findings to the typical pediatric outpatient population.

General Information

Peripheral blood samples were collected from 916 children who visited the hospital between January 1 and December 31, 2022, for flow cytometric analysis of T lymphocyte subsets. Based on clinical diagnoses, the participants were stratified into four groups: Group 1 (isolated adenoid hypertrophy, AH): 316 cases (203 males, 113 females; mean age 6.74 ± 0.131 years); Group 2 (isolated allergic rhinitis, AR): 176 cases (97 males, 79 females; mean age 6.35 ± 0.332 years); Group 3 (AH with AR): 379 cases (228 males, 151 females; mean age 6.34 ± 0.121 years); and Group 4 (healthy controls): 145 cases (79 males, 66 females; mean age 6.60 ± 0.203 years). No statistically significant differences were observed in gender distribution or age across the four groups ($P > 0.05$), confirming group comparability. The study protocol was approved by the Ethics Committee of the Affiliated Hospital of North Sichuan Medical College (Approval No. 2024ER263-1).

Inclusion Criteria

Participants in the disease groups (AH, AR, AH+AR) had to meet all the following criteria:

(A) Diagnosis of Allergic Rhinitis (AR): Diagnosis was established in accordance with the "Chinese Guidelines for the Diagnosis and Treatment of Allergic Rhinitis (2022, Revised Edition)" (11). This required:

a) A typical clinical history of two or more of the following symptoms: watery rhinorrhea, nasal obstruction, sneezing and nasal itching, persisting for more than 4 days per week and for more than 4 consecutive weeks.

b) Objective confirmation via a skin prick test (SPT). A positive SPT was defined as a wheal diameter ≥3 mm larger than the negative control in response to at least one common inhalant allergen (including but not limited to house dust mites, mold, pollen, animal dander).

(B) Diagnosis of Adenoid Hypertrophy (AH): Diagnosis was confirmed by either nasal endoscopy or lateral nasopharyngeal radiography. The adenoid-to-nasopharynx ratio (A/N ratio) was calculated and an A/N ratio ≥0.6 was used as the objective diagnostic criterion for significant hypertrophy obstructing the nasopharynx (12).

(C) Age between 3 and 14 years.

(D) No history of autoimmune diseases, hematological disorders, or other systemic diseases and no acute upper respiratory tract infection within the preceding 4 weeks.

(E) No concurrent acute or chronic otitis media or sinusitis at the time of enrollment.

(F) Written informed consent obtained from a parent or legal guardian.

Exclusion Criteria

Children were excluded from the study if they met any of the following criteria:

(A) Presence of autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus), primary immunodeficiency disorders, hematological malignancies, or any other chronic systemic disease that could affect immune cell counts.

(B) Any acute infection (e.g., upper respiratory tract infection, gastroenteritis, urinary tract infection) or fever (axillary temperature ≥37.3°C) within the preceding 4 weeks.

(C) Age > 14 years or < 3 years.

(D) Recent acute upper respiratory tract infection, otitis media, or sinusitis.

(E) A history of otitis media or sinusitis within the past 3 months (to exclude recent/local inflammatory confounders).
(F) Failure to obtain informed consent.

Inclusion Criteria for the Healthy Control Group:

(A) No history of allergic diseases.
(B) Age between 3 and 14 years.
(C) Informed consent obtained.
(D) No systemic diseases and no recent acute infection history.
(E) No significant abnormalities in liver or kidney function.

Methods

Examination Method for AH

AH was diagnosed by nasal endoscopy or lateral nasopharyngeal X-ray; an adenoid-to-nasopharynx ratio (A/N) ≥0.6 was used as the diagnostic criterion.

Measured parameters included: absolute counts (cells/μL) and percentages (%) of $CD3^+$, $CD4^+$ and $CD8^+$ T lymphocytes, the CD4/CD8 ratio and the percentage of T suppressor/cytotoxic cells (13, 14).

Diagnostic Method for Allergic Rhinitis

Diagnosis was based on medical history combined with a skin prick test.

Detection Method for T Lymphocyte Subset Levels

For both patients during examination and healthy subjects during check-ups, 5 mL of morning fasting peripheral venous blood was drawn from the antecubital vein. From this, 2 mL of peripheral blood was used for detection. The absolute counts of $CD3^+$, $CD4^+$ and $CD8^+$ T lymphocytes and their percentages within the total lymphocyte population were measured using a Mindray BriCyte E6 flow cytometer.

Statistical Methods

Data were analyzed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). Normality of measurement data was assessed with the Shapiro-Wilk test; percentage data underwent arcsine square root transformation to approximate a normal distribution. Homogeneity of variances was examined via Levene's test. For percentage or ratio indicators exhibiting heterogeneous variances, overall group comparisons were performed using Welch's ANOVA, followed by post-hoc pairwise comparisons with the Games-Howell method. For absolute count indicators that were non-normally distributed, the Kruskal-Wallis H test was employed for overall comparisons, with data presented as median (interquartile range) [M(IQR)]. When overall differences reached statistical significance, pairwise comparisons were conducted using the Mann-Whitney U test and the significance threshold was adjusted via the Bonferroni correction (adjusted $\alpha = 0.0083$). Effect sizes were reported as follows: epsilon-squared (ε^2) for the Kruskal-Wallis test and the r value ($|r| = Z/\sqrt{N}$) for the Mann-Whitney U test. The significance level was set at $\alpha = 0.05$.

Sample Size Consideration and Power Analysis:

The present study employed a real-world, observational design, leading to uneven group sizes that reflected the natural prevalence and clinical presentation patterns of the conditions (e.g., higher frequency of AH+AR comorbidity). While unequal group sizes can influence statistical power, the non-parametric tests used (Kruskal-Wallis H test, Mann-Whitney U test) are robust to such imbalances. To address potential concerns regarding statistical power in pairwise comparisons, particularly involving smaller groups (e.g., AR-only group, n=176), we conducted a post-hoc power analysis for the key significant findings (e.g., CD4+ count: AR vs. Control). The observed effect sizes and sample sizes yielded a statistical power exceeding 80% for detecting these differences at $\alpha=0.05$, indicating that the study was sufficiently powered to identify the reported significant effects despite the uneven distribution.

Results

The study cohort comprised 316 children with adenoid hypertrophy (AH), 176 with allergic rhinitis (AR), 379 with comorbid AR and AH and 145 healthy controls. For multi-group comparisons, the non-parametric Kruskal-Wallis H test was employed, followed by post-hoc pairwise analyses using the Mann-Whitney U test. To account for multiple comparisons and control Type I error, the Bonferroni correction was applied, adjusting the significance level to $\alpha = 0.05/6 \approx 0.0083$ (15). Data are expressed as median with interquartile range [M(IQR)]. The results of the overall Kruskal-Wallis H tests for all measured parameters are presented in Table1.

Table 1: Kruskal-Wallis H Tests for Overall Differences Across Study Groups

Variable	H Statistic	df	P value
Absolute CD3+T cell count (cells/μL)	21.23	3	< 0.001
Absolute CD4+T cell count (cells/μL)	19.644	3	< 0.001
Absolute CD8+T cell count (cells/μL)	17.833	3	< 0.001
CD3+T lymphocytes (%)	4.968	3	0.174
CD4+T lymphocytes (%)	3.768	3	0.288
CD4/CD8 ratio	8.671	3	0.034

H: Kruskal-Wallis H statistic; df: degrees of freedom.

A P value < 0.05 indicates a statistically significant overall difference among the four groups for that variable.

A) Regarding $CD3^+$ T Lymphocytes (Total T Lymphocytes):

Significant differences in absolute $CD3^+$ T lymphocyte counts were observed across the four groups (H = 21.23, P < 0.001).

Median counts were highest in the allergic rhinitis (AR) group (2137 [1709–2971.5] cells/μL), followed by the comorbid AR + adenoid hypertrophy (AH) group (1960 [1573–2451] cells/μL), the AH-only group (1900.5 [1592.25–2345.25] cells/μL) and healthy controls [1373.5–2241] cells/μL). Pairwise comparisons revealed that the AR group had significantly higher counts than both the control group (P < 0.001) and the AH group (P = 0.004). Similarly, the AH group showed elevated counts relative to controls (P = 0.003) and the comorbid group also exceeded control levels (P = 0.001). No significant differences were found between the comorbid group and either the AR group (P = 0.021) or the AH group (P = 0.458).

The median percentages of $CD3^+$ T lymphocytes / total lymphocytes were similar across groups, ranging from 65.34% to 68.33%. The Kruskal-Wallis test showed no statistically significant overall difference in the percentage of total T lymphocytes among the four groups (H=4.968, P=0.174).

B) Results for $CD4^+$ T Lymphocytes:

The absolute count of $CD4^+$ T lymphocytes was highest in the allergic rhinitis group (1061 (797 - 1511) cells/μL), followed by the combined group (944 (727 - 1242) cells/μL), the adenoid hypertrophy group (925.5 (729.25 - 1164) cells/μL) and the healthy control group (823 (632.5 - 1103) cells/μL).

The Kruskal-Wallis H test revealed a statistically significant overall difference in absolute $CD4^+$ T lymphocyte counts among the four groups (H = 19.644, P < 0.001). The allergic rhinitis (AR) group exhibited significantly higher $CD4^+$ counts compared to both the adenoid hypertrophy (AH) group (P = 0.002) and healthy controls (P < 0.001). Although the AR group showed elevated counts relative to the comorbid (AH + AR) group, this difference did not reach statistical significance after Bonferroni correction (P = 0.025 > 0.0083). No significant differences were observed between the AH group and either the control group (P = 0.025) or the comorbid group (P = 0.181). However, the comorbid group demonstrated significantly higher $CD4^+$ counts than the healthy control group (P = 0.002).

The median percentages were similar across groups, ranging from 31.74 % to 32.48%. There was no statistically significant overall difference in the percentage of $CD4^+$ T cells among the four groups [H=3.768, P=0.288].

C) Results for $CD8^+$ T Lymphocytes:

The absolute count of $CD8^+$ T lymphocytes was 781.5 [622.75 - 985.5] cells/μL in the adenoid hypertrophy group, 740 [577 – 1246] cells/μL in the allergic rhinitis group, 788 [579 – 996] cells/μL in the allergic rhinitis combined with adenoid hypertrophy group and 661 [477 – 905] cells/μL in the healthy control group. The Kruskal-Wallis test showed a highly statistically significant overall difference in the absolute count of $CD8^+$ T lymphocytes among the four groups (H=17.833, P<0.001). The absolute counts of $CD8^+$ T cells in the adenoid hypertrophy group, allergic rhinitis group and allergic rhinitis combined with adenoid hypertrophy group were all significantly higher than that in the healthy control group (P < 0.001, P=0.006, P < 0.001, respectively). However, all pairwise comparisons among the three disease groups showed no statistically significant differences (all corrected P-values > 0.0083). The percentages were 27.3 [23.52 - 31.31] % for the adenoid hypertrophy group, 24.23 [20.93 - 30.28] % for the allergic rhinitis group, 26.3 [22.72 - 31.26] % for the combined group and 26.1 [21.46 - 31.01] % for the healthy control group. Only the difference in percentage between the allergic rhinitis group and the adenoid hypertrophy group was statistically significant (corrected P=0.002),

with the adenoid hypertrophy group having a higher $CD8^+$ percentage.

D) CD4/CD8 Ratio

The CD4/CD8 ratio was 1.29 (0.98 - 1.75) in the allergic rhinitis group, 1.23 (0.99 - 1.58) in the combined group, 1.16 (0.94 - 1.45) in the adenoid hypertrophy group and 1.23 (1 - 1.63) in the healthy control group. The overall difference in the CD4/CD8 ratio among the four groups was statistically significant (H=8.671, P=0.034). Although the overall test P-value was <0.05, after strict multiple comparison correction, all pairwise comparison P-values between groups were greater than the corrected significance level (0.0083) and therefore none were statistically significant.

Summary and Interpretation of Key Findings

Analysis of CD3+ T lymphocytes: Compared to healthy children, children with allergic rhinitis (AR), adenoid hypertrophy (AH) and the combination of both showed significantly elevated absolute counts of peripheral blood $CD3^+$ T lymphocytes. This suggests that AH itself is associated with systemic T-cell immune activation, regardless of allergic factors, while AR exhibits the strongest effect on increasing T-cell counts.

Disease Co-existence Did Not Produce an Additive Effect: The T-cell counts in children with AR combined with AH showed no statistically significant difference compared to those with AH but were significantly lower than those with AR (although this difference became non-significant after correction, the trend was consistent with the original P=0.021 value). This suggests that when the two diseases coexist, AH may exert a moderating effect on the stronger T-cell response triggered by AR, rather than a simple additive effect.

T Cell Relative Proportions Remained Stable: Despite significant increases in absolute T-cell numbers across disease groups, no statistically significant differences were found in the percentage of total T lymphocytes among lymphocytes ($CD3^+$%) between any groups, including compared to the healthy control group. This indicates that the observed T-cell increase is more likely due to an overall expansion of the lymphocyte pool rather than an imbalance in the proportions of specific T-cell subsets.

This study confirms that both pediatric AH and AR can independently cause a significant increase in the absolute count of peripheral blood $CD3^+$ T cells, with AR having a more prominent effect. However, these pathological states did not alter the fundamental proportion of T cells within the lymphocyte population. When the two diseases coexist, the immunological profile more closely resembles that of AH. These findings provide a quantitative basis for understanding the systemic immune status in children with such upper airway diseases.

Statistically significant inter-group differences exist in the absolute counts of peripheral blood T lymphocyte subsets in children with AH and/or AR, but the actual effect size of these differences is small.

The Elevation in Absolute $Cd4^+$ T Cell Counts Appear to Be Disease-Specific: Children with AR, whether alone or in combination with AH, exhibited significantly higher $CD4^+$ counts compared to healthy controls. In contrast, no statistically significant difference was observed between the AH-only group and the control group. These findings suggest that the increase in $CD4^+$ T cell numbers is predominantly driven by the presence of AR, whereas AH alone may not directly contribute to notable changes in this lymphocyte subset.

The Impact of AR On $CD4^+$ T Cells is More Prominent: Among all disease groups, the AR group had the highest $CD4^+$ T cell count, which was significantly higher than that in the AH group. This further confirms that AR, as a disease dominated by a Th2-type immune response, is more closely associated with the systemic activation of helper T ($CD4^+$) cells.

The Relative Proportion of $CD4^+$ T Cells Remained Constant Across All Groups: Despite the increase in absolute $CD4^+$ T cell numbers in the AR and combined groups, their percentage within the total lymphocyte population showed no statistically significant difference across groups (including between disease and healthy groups). This result, consistent with the patterns observed for $CD3^+$ and $CD8^+$ T cell percentages. Collectively supports the main observation of this study: upper airway diseases primarily cause an overall expansion of the lymphocyte pool, rather than a fundamental change in the proportions of specific T-cell subsets.

Summary Conclusion: This study clarifies the distinct impact patterns of pediatric AH and AR on

$CD4^+$ T cells: a significant increase in $CD4^+$ T cell numbers is a characteristic immune alteration in AR (whether combined with AH or not), while this effect is not seen in AH. Combined with the previous analysis of $CD3^+$ and $CD8^+$ cells, this study systematically reveals the peripheral immune characteristics in children with these diseases, which are dominated by an expansion of the total T lymphocyte count while maintaining a stable subset proportional architecture.

Based on the statistical analysis results strictly interpreted against the corrected significance level (α=0.0083), the following conclusions are drawn regarding $CD8^+$ T lymphocytes:

$CD8^+$ T Cell Absolute Counts are Generally Elevated in Disease Groups: Compared to the healthy control group, children with AH, AR and the combination of both showed significantly elevated absolute counts of peripheral blood $CD8^+$ T lymphocytes. This indicates that the disease states are all associated with an enhancement of systemic $CD8^+$ T cell (cytotoxic T cell) immune responses.

No Specific Differences Between Disease Types: No statistically significant differences were found in the absolute counts of $CD8^+$ T cells among the three disease states (AH, AR, AH+AR). This suggests that the impact of AH and AR on inducing an increase in $CD8^+$ T cell numbers is similar in magnitude and no additive or antagonistic effects are observed when the two diseases coexist.

The Percentage of $CD8^+$ T Cells Remained Relatively Stable Across All Groups: Despite notable increases in absolute $CD8^+$ counts in disease groups, no statistically significant differences were observed in their relative proportions among total lymphocytes, including when compared to healthy controls. When considered alongside the findings for $CD3^+$ and $CD4^+$ subsets, this consistency reinforces the central observation of the study: in pediatric patients, AH and AR predominantly drive an overall increase in lymphocyte pool size rather than a redistribution or selective expansion of specific T-cell subsets.

Summary Conclusion: This study confirms that an increase in $CD8^+$ T cell numbers is a shared peripheral immune feature of pediatric AH and AR, while the relative proportional architecture remains stable. This pattern is consistent with the changes in $CD3^+$ and $CD4^+$ T cells, collectively depicting a systemic immune response picture characterized by total lymphocyte expansion against the background of these upper airway diseases.

Based on the statistical analysis results strictly interpreted against the corrected significance level (α=0.0083), the following conclusions are drawn regarding $CD8^+$ T lymphocytes:

$CD8^+$ T Cell Absolute Counts are Universally Elevated but Show No Differences Between Diseases: Compared to healthy children, children with AH, AR and the combination of both showed significantly elevated absolute counts of peripheral blood $CD8^+$ T lymphocytes. However, no statistically significant differences were found in the absolute $CD8^+$ T cell counts among these three disease states. This indicates that these diseases can all lead to a systemic increase in cytotoxic T cell numbers, but the magnitude of increase is similar.

Differences in $CD8^+$ T Cell Relative Proportion Exist Between Specific Diseases: Unlike the generally stable patterns of $CD3^+$ and $CD4^+$ T cell percentages, a statistically significant difference was found in the relative proportion of $CD8^+$ T cells ($CD8^+$%) between the AR group and the AH group, with the AR group having a lower proportion. This finding suggests that although both conditions lead to an increase in $CD8^+$ T cell numbers, AR might be accompanied by a more pronounced expansion of other subsets within the lymphocyte pool, thereby relatively reducing the proportion accounted for by $CD8^+$ cells.

The Proportion in the Combined Disease State Shows No Difference from the Healthy Group: Despite the elevated absolute $CD8^+$ T cell counts in the AR+AH group, its $CD8^+$% showed no significant difference compared to the healthy control group. Combined with conclusion 2, this further indicates that when the two diseases coexist, the immune state does not exhibit the extreme characteristics of either disease but rather may reach a new balance point.

Summary Conclusion: This study reveals the specificity of the $CD8^+$ T cell response pattern in pediatric upper airway diseases: its absolute count is universally elevated across disease groups to a similar degree, but its relative proportion differs between patients with AR and AH. This indicates that although the overall T-cell response manifests as numerical expansion, different diseases may exert differential effects on the peripheral immune

microenvironment by modulating the proliferation degrees of different T-cell subsets. The difference in $CD8^+$ T cell proportion could be a potential indicator for distinguishing the immune characteristics of these two diseases.

Based on the statistical results after strict multiple comparison correction, this study draws the following conclusions regarding the CD4/CD8 ratio:

No Clear Inter-group Differences were Found: Although the overall test for the CD4/CD8 ratio suggested potential differences between groups, no statistically significant differences were identified between any two groups (including between each disease group and the healthy control group and between different disease groups) after controlling for multiple comparison errors. Therefore, the data from this study do not support the notion that pediatric adenoid hypertrophy, allergic rhinitis, or their combined state leads to a significant alteration in the peripheral blood CD4/CD8 ratio.

Numerical Distribution Trends were Observed: Based on the data distribution, the median CD4/CD8 ratio in the adenoid hypertrophy group (1.16) was slightly lower than that in the other three groups (1.23-1.29). Although this trend is not statistically significant, it may correspond with the previously observed pattern of a more pronounced increase in the absolute $CD4^+$ T cell counts in the allergic rhinitis group, suggesting that the effects of the two diseases on T cell subsets might have subtle differences, which are insufficient to disrupt the overall ratio balance.

Summary Conclusion: Integrating the analysis of all T cell subsets in this study, pediatric adenoid hypertrophy and allergic rhinitis primarily induce a generalized increase in the absolute numbers of $CD3^+$, $CD4^+$ and $CD8^+$ T lymphocytes. Although the degree of increase in each subset varies slightly between different diseases (e.g., the effect of allergic rhinitis on $CD4^+$ cells is more prominent), this increase is relatively balanced and does not lead to a statistically significant shift in the core immune balance index, the CD4/CD8 ratio. This further confirms that the peripheral immune characteristic of such diseases is primarily an expansion of the total lymphocyte pool while maintaining the fundamental stability of the cellular immune architecture.

Absolute $CD8^+$ T lymphocyte counts in the healthy control group were consistently lower than those in all disease groups, suggesting that pathological conditions may be associated with a modest elevation in $CD8^+$ cell numbers. In the isolated allergic rhinitis (AR) group, absolute counts of $CD4^+$ and $CD3^+$ T cells were significantly elevated compared to healthy controls, with moderate effect sizes, indicating that AR alone may exert a more substantial influence on these subsets. In contrast, no significant intergroup differences were detected in the percentages or ratios of T lymphocyte subsets, implying that disease states do not markedly alter the proportional distribution of these cells.

Significant heterogeneity in the absolute count of $CD3^+$ T lymphocytes existed among different groups. Specifically, Group 2 exhibited the highest levels, while Group 4 had the lowest, indicating that this parameter can effectively differentiate the four defined groups in this study. This likely reflects fundamental differences in key pathophysiological or immunological states among them.

The percentage of total T lymphocytes remained relatively stable across groups. Although pairwise comparisons revealed statistical differences between some specific groups, the overall test did not show significant differences. This suggests that the proportional composition of total T lymphocytes within the lymphocyte pool may not be a sensitive distinguishing indicator. Its stability implies a degree of consistency in immune cell proportions at this macroscopic level across the different groups.

Therefore, the data from this study do not support the notion that pediatric adenoid hypertrophy, allergic rhinitis, or their combined state leads to a significant alteration in the peripheral blood CD4/CD8 ratio. The detailed profiles of all T lymphocyte subset parameters for the four groups are summarized in Table 2. The comparative immunological profiles and the shared feature of lymphocyte pool expansion among the four study groups are schematically summarized in Figure 1.

Table 2: Profiles of Peripheral Blood T Lymphocyte Subsets in the Study Groups

Group	n	Absolute CD8+Count (cells/μL)	Absolute CD4+ Count (cells/μL)	Absolute CD3+ Count (cells/μL)	CD3+%	CD4+ %	CD4/C8Ratio	CD8+%
1 (AH)	316	781.5 (622.8-985.5) ^a	925.5 (729.3-1164.0) ^a	1900.5 (1592.3 - 2345.3) ^a	67.63 (62.90-71.70)	31.95 (28.07-35.92)	1.16 (0.94-1.45)	27.30 (23.52-31.31) ^*
2 (AR)	176	740.0 (577.0-1246.0) ^a	1061.0 (797.0-1511.0) ^{a,b}	2137.0 (1709.0 - 2971.5) ^{a,b}	65.34 (59.30-72.09)	31.74 (28.26-36.18)	1.29 (0.98-1.75)	24.23 (20.93-30.28) ^*
3(AH+AR)	379	788.0 (579.0-996.0) ^a	944.0 (727.0-1242.0) ^{a,c}	1960.0 (1573.0 - 2451.0) ^a	67.58 (62.90-71.74)	32.48 (28.76-36.84)	1.23 (0.99-1.58)	26.30 (22.72-31.26)
4(Healthy Control)	145	661.0 (477.0-905.0)	823.0 (632.5 - 1103.0)	1731.0 (1373.5 - 2241.0)	68.33 (62.11-73.54)	32.33 (27.85-38.36)	1.23 (1.00-1.63)	26.10 (21.46-31.01)

Note: Datas are presented as median (interquartile range). Group 1: Isolated adenoid hypertrophy (AH); Group 2: Isolated allergic rhinitis (AR); Group 3: Comorbid AH and AR; Group 4: Healthy controls. Superscript letters indicate statistically significant differences ($P < 0.0083$, Bonferroni-corrected) in absolute counts compared to: ^a vs. Group 4 (Control); ^b vs. Group 1 (AH); ^c vs. Group 2 (AR). *: A statistically significant difference ($P = 0.002$) was found in the percentage of CD8+ T cells between Group 1 (AH) and Group 2 (AR).

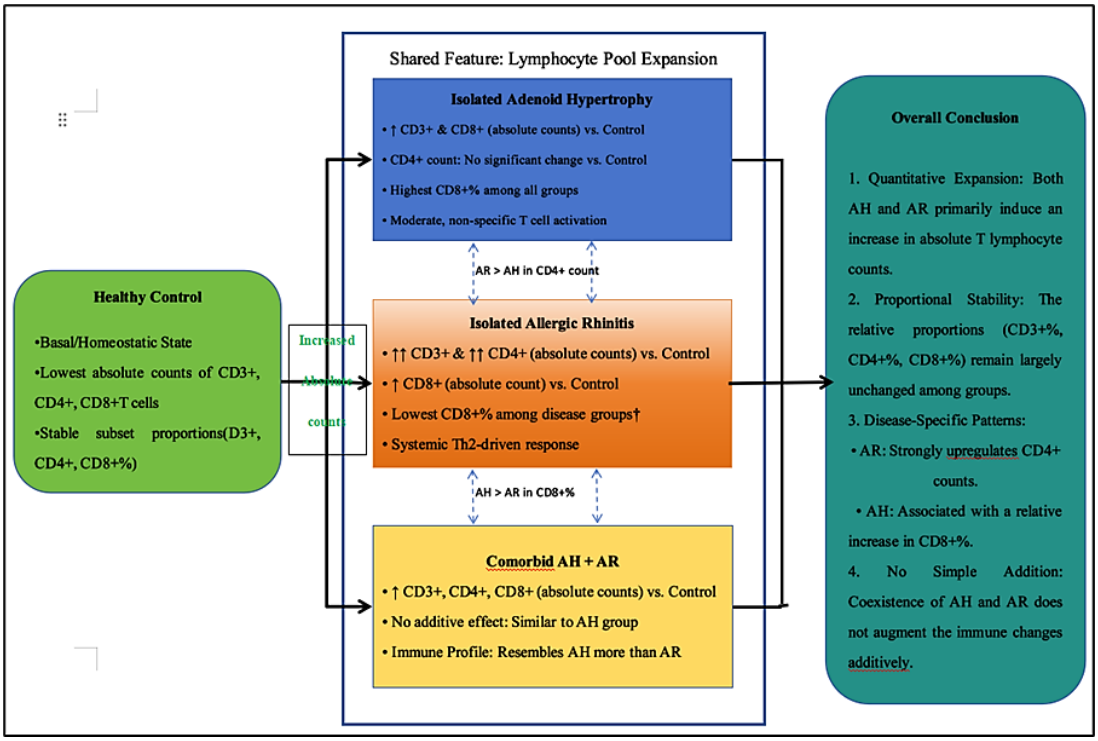

Figure 1: T Lymphocyte Subset Analysis Across Healthy, AH, AR and Comorbid Groups: A schematic Summary of Peripheral Blood T Lymphocyte Subset Profiles in the Studied Pediatric Cohorts.

In Figure 1, upward arrows (↑, ↑↑) indicate significant increases in absolute cell counts compared to healthy controls. The thickness of the arrows suggests the magnitude of the effect. Key comparative differences between disease groups are highlighted. The diagram integrates the main findings that both diseases drive a quantitative expansion of the lymphocyte pool while largely preserving subset proportions, with allergic rhinitis exhibiting a more pronounced effect on CD4+ T cells.

Discussion

Baseline Immune Differences Between Healthy and Diseased Children Were Identified: Contrary to common perception, the absolute counts of all T lymphocyte types (total $CD3^+$, helper $CD4^+$, cytotoxic $CD8^+$) in the healthy control children (Group 4) in this study were at their lowest levels. This finding suggests that under steady-state conditions without clear allergic or chronic inflammatory stimuli, effector T cells in children's peripheral circulation may be maintained at a relatively "basal" or "quiescent"

level. The elevated T-cell counts in the disease groups likely reflect a state of sustained immune system activation.

The elevated T-cell counts in the disease groups likely reflect a state of sustained immune system activation. This finding of the lowest absolute T lymphocyte counts in healthy controls aligns with the concept of a 'resting' or 'steady state' peripheral immune profile in the absence of overt antigenic challenge. This baseline level may reflect a homeostatic balance, as suggested by studies in healthy pediatric populations (16, 17). The elevated counts across all disease groups further underscore a state of systemic immune activation, consistent with the chronic inflammatory nature of both AH and AR (18, 19).

Different Diseases Exhibit Distinct Immune Activation Patterns:

a. Allergic Rhinitis (Group 2): Its characteristic feature is a significant increase in the absolute count of CD4+ T lymphocytes. This finding strongly supports its core pathological mechanism of Th2 cell-mediated type I hypersensitivity, indicating a dominantly activated state of systemic Th2 cell immune responses, as widely documented in the immunopathology of AR (20). Our quantitative data corroborate earlier observations of Th2 polarization in peripheral blood of AR patients (21, 22).

b. Adenoid Hypertrophy (Group 1) and the Comorbidity Group (Group 3): These two groups showed no significant differences in the absolute counts of most T cells, with their levels intermediate between the healthy group and the allergic group. This suggests that the immune activation accompanying adenoid hypertrophy (whether combined with allergy or not) differs in intensity and pattern from that of allergic rhinitis. It may be more associated with local recurrent infections or different inflammatory pathways (e.g., involving Th1 or Th17 responses) rather than systemic Th2 cell-dominant expansion, a notion supported by studies examining cytokine profiles in adenoid tissues (23).

c. No Additive Effect in the Comorbidity Group (Group 3): Children with both adenoid hypertrophy and allergic rhinitis did not show a further increase in T-cell subset counts beyond those seen in the disease groups. This suggests that shared or mutually restrictive immunoregulatory mechanisms may exist between these two diseases regarding their impact on the systemic T-cell pool. This absence of a simple additive effect aligns with some clinical observations that the immunological interplay in comorbid cases might represent a distinct phenotype, rather than a mere summation of two separate conditions (24).

d. The Core Clinical Value of T Cell Absolute Counts Was Clarified: Within the disease spectrum covered by this study, the absolute counts of T lymphocyte subsets are a more sensitive indicator for disease differentiation and immune status assessment than percentages. They more clearly reveal quantitative immune differences between health and disease states, as well as among different disease types.

Comprehensive Conclusion: Characteristic differences exist in the systemic T lymphocyte immune status among healthy children and children with allergic rhinitis, adenoid hypertrophy and the comorbidity of both. Children with allergic rhinitis exhibit a typical enhancement of systemic $CD4^+$ T cell responses; adenoid hypertrophy (regardless of comorbidity with allergy) is accompanied by a moderate, non-specific T cell activation, whereas healthy children present relatively basal T cell levels (25). Its persistent activation may, through causing tissue damage and secreting pro-inflammatory factors, inadvertently contribute to the pathological hyperplasia and remodeling of lymphoid tissue, a phenomenon observed in other chronic inflammatory settings (26). Future research should combine pathological and immunohistochemical studies of the adenoid tissue itself to more precisely elucidate the phenotype, function and causal role of CD8+ T cells within the local lesion in the development of hypertrophy, as recently explored in comparative studies of lymphoid tissue subpopulations (27). These findings provide new laboratory evidence for understanding the similarities and differences among these common childhood diseases from a systemic immunological perspective and suggest that the absolute counts of peripheral blood T lymphocyte subsets can serve as effective parameters for assisting in the assessment of immune status in children with related conditions.

Limitations

Several limitations of this study should be acknowledged. First, the retrospective and observational design limits the ability to infer

causal relationships between disease status and the observed changes in T-cell subsets. In addition, potential confounding factors such as medication use, disease duration and age were not fully controlled (28). Although strict inclusion and exclusion criteria were applied, residual confounding may still exist. Variables that may influence peripheral lymphocyte counts—such as subclinical infections, environmental allergen exposure at the time of sampling, nutritional status, psychological stress and circadian rhythm variations—were not directly assessed (29).

Second, the uneven sizes of the patient groups, although reflecting clinical prevalence, may have introduced some imbalance in statistical power for certain inter-group comparisons. While post-hoc analyses suggested that the primary findings retained adequate statistical power, future studies with prospectively balanced group sizes would be beneficial to confirm and extend these observations. In addition, the single-center nature of this study may limit the generalizability of the findings to other populations.

Third, the present analysis was limited to the quantitative assessment of peripheral blood T-cell subsets. Functional assays, such as cytokine profiling or lymphocyte proliferation tests, were not performed, nor were local immune responses in adenoid or nasal mucosal tissues examined (30). Consequently, the functional and tissue-specific significance of the observed numerical differences remains unclear.

Finally, although several comparisons reached statistical significance, the observed effect sizes were generally small, indicating that disease status explains only a modest proportion of the variance in lymphocyte counts. Therefore, the clinical and biological significance of these differences should be interpreted with caution. Future prospective, multi-center studies incorporating longitudinal sampling, balanced group designs, comprehensive measurement of potential covariates and functional immune assays will be important to validate and further elucidate these findings.

Conclusion

By comparing peripheral blood T lymphocyte subsets among four pediatric groups, this study found that disease states primarily influence the absolute numbers of lymphocytes rather than their relative proportions. The mild elevation of $CD8^+$ T cells in disease groups may be associated with immune activation under chronic inflammation or allergic conditions. Group 2 (AR) exhibited the most pronounced differences from the healthy control group in $CD4^+$ and $CD3^+$ counts, which may reflect the expansion effect of allergic inflammation on the systemic T-cell pool. This finding aligns with previous reports suggesting that children with AR exhibit Th2 polarization and T-cell activation.

However, the effect sizes for all significant differences were small ($\varepsilon^2 \approx 0.02$, mostly $r < 0.3$), indicating that the factor of "disease group" explains only a small proportion of the variance in lymphocyte counts. This suggests that although the differences are statistically significant, their clinical or biological significance may be limited and should be interpreted with caution. The findings therefore support the combined use of both absolute counts and percentage indicators for a more comprehensive assessment of immune status in children.

In this study, the absolute count of $CD8^+$ T lymphocytes were significantly higher in all disease groups (Groups 1, 2 and 3) compared with the healthy control group (Group 4). This observation suggests a state of systemic immune activation across these disease conditions. Whether in adenoid hypertrophy (AH), allergic rhinitis (AR), or their coexistence, the expansion of the $CD8^+$ T-cell pool may represent a shared immunological feature reflecting the body's adaptive response to chronic inflammation or persistent antigenic stimulation.

At the same time, the small effect size associated with these differences indicates that the increase in $CD8^+$ T-cell numbers, although statistically detectable, is unlikely to represent a dominant driver of adenoid hypertrophy. Instead, the development and progression of adenoid hypertrophy are more likely the result of complex interactions among multiple immune components, including different T-cell subsets (e.g., Th2, Th17, Treg), B cells, cytokine networks, the local microbiome and anatomical or physiological factors. In this context, the elevation of $CD8^+$ T cells may represent one component within a broader and more complex immune regulatory network.

Within the pathological context of adenoid hypertrophy, the role of $CD8^+$ T cells may extend beyond that of a traditional cytotoxic "defender" to

that of an active participant in the chronic inflammatory microenvironment. While these cells may function as effector mechanisms attempting to control local infection, persistent activation could also contribute to tissue damage and the secretion of pro-inflammatory mediators, potentially promoting pathological lymphoid hyperplasia and tissue remodeling. The present findings provide peripheral blood-level evidence that supports this theoretical framework.

Future Research Perspectives

Future studies should aim to further clarify the functional role of $CD8^+$ T cells in the pathogenesis of adenoid hypertrophy and allergic rhinitis. Combining peripheral immune profiling with pathological and immunohistochemical analyses of adenoid tissues would help identify the phenotype, functional state and spatial distribution of $CD8^+$ T cells within the local lesion. Functional assays examining cytokine production, cytotoxic activity and regulatory properties may also provide deeper insights into their immunological roles.

In addition, longitudinal and multi-center studies with larger and more balanced sample sizes would improve the generalizability of these findings and allow researchers to explore dynamic changes in T-cell subsets throughout disease progression and treatment. Integrating immunological data with clinical parameters, microbiome composition and environmental exposure factors may further contribute to a more comprehensive understanding of the immune mechanisms underlying adenoid hypertrophy and allergic rhinitis in children.

Such integrative approaches may ultimately help identify more precise immunological biomarkers and potential therapeutic targets for the management of chronic pediatric airway inflammatory diseases.

Abbreviations

None.

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Author Contributions

Fengbo Yang: Conceptualization, experimental design, data collection, data analysis, manuscript writing, Mohammed Abdelfatah Alhoot: supervision.

Conflicts of Interest

The authors declare no conflicts of interest, including but not limited to financial funding, collaborative relationships, employment, or any other potential conflicts that may affect the objectivity and independence of this study.

Data Availability

The datasets used in this study are fully described in the article. The raw and processed data are available from the first author Fengbo Yang upon reasonable request.

Declaration of Artificial Intelligence (AI) Assistance

Artificial intelligence tools were used to assist with language polishing during the writing and revision of this manuscript. These tools were only used as auxiliary means. All research ideas, experimental design, content writing, academic viewpoints and the final version of the manuscript were completed independently by the authors, who bear full academic responsibility.

Ethics Approval

This study was approved by the Ethics Committee of the Affiliated Hospital of North Sichuan Medical College (No. 2026ER53-1). Informed consent was obtained from all participants' guardians.

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