Sarcouncil Journal of Medical Series

ISSN(Online): 2945-3550

Volume- 05| Issue- 04| 2026

Research Article **Received:** 05-03-2026 | **Accepted:** 30-03-2026 | **Published:** 14-04-2026

Adenoid Hypertrophy and Its Role in Pediatric Chronic Rhinosinusitis: A Cohort Analysis

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Abstract: **Background**: Adenoid hypertrophy is often involved in the pathophysiology of chronic rhinosinusitis (CRS) in children, but its exact role in disease severity, microbiology, and treatment is not fully described. This paper set out to assess the importance of adenoid hypertrophy measured by the adenoid-nasopharynx ratio (ANR) in clinical presentation and management of CRS in children. **Methods**: A prospective cohort study design was used to study 102 pediatric patients (aged 314 years) diagnosed with CRS based on EPOS 2020 criteria. The participants were thoroughly assessed with nasal endoscopy, low-dose paranasal sinus CT, middle meatus microbiological samples, and validated quality-of-life scales (SNOT-20, PedsQL). Eadenoid hypertrophy (ANR ≥0.70; n=67) and non-hypertrophy (ANR <0.70; n=35) were used to stratify patients. They all were given 12 weeks of maximum medical treatment; those who were refractory in the hypertrophy group were given an adenoidectomy. Clinical success at 12 weeks was the primary outcome; secondary outcomes were changes in symptom scores, recurrence rates, and predictors of treatment failure. **Results**: The adenoid hypertrophy group was significantly younger (7.2 vs. 9.1 years; p<0.001), had longer symptom duration, higher Lund-Mackay CT scores (14.1±4.8 vs. 9.2±4.6; p<0.001), and worse baseline SNOT-20 scores (46.7±11.4 vs. 33.9±11.6; p<0.001). The hypertrophy group had a greater prevalence of biofilm formation (56.7% vs. 25.7; p=0.003). The success rate of medical therapy alone was 32.0% in hypertrophy and 62.9 in non-hypertrophy (p<0.001). Adenoidectomy in the cases of refractory hypertrophy showed an 83.3% success rate with significant changes in SNOT-20 (mean of 28.5±9.1 points) and PedsQL. A multivariate analysis revealed that both adenoid hypertrophy (adjusted OR 2.87, 95% CI 1.21682; p=0.017) and biofilm presence (adjusted OR 3.24, 95% CI 1.38761; p=0.007) were independent predictors of treatment failure. **Conclusion**: Adenoid hypertrophy is a major, disease-independent disease contributor with stronger disease severity and refractoriness to therapy in pediatric CRS.

Keywords: Pediatric Crs, Hypertrophy, Adenoid, Disease, Rhinosinusitis.

INTRODUCTION

Chronic inflammatory disease Chronic rhinosinusitis (CRS) is one of the most common chronic inflammatory diseases of the pediatric population and presents a significant load on the healthcare system, the quality of education, and family quality of life. Pediatric CRS is markedly different in pathophysiology, microbiology, and management approaches compared to adult CRS due to the specifics of its definition: according to the European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS), this disease is inflammation of the nose and paranasal sinuses with two or more symptoms, such as nasal blockage, obstruction, congestion, or nasal where CRS has been estimated to affect children with a prevalence rate of between 2-4 percent though some studies have indicated a higher rate when it comes to recurrent acute rhinosinusitis that cannot fully clear between episodes [De La Torre Gonzalez, C. *et al*., 2012; Makary, C. A., & Ramadan, H. H. 2018]

Adenoid hypertrophy has always been considered to be one of the most important aspects of pediatric CRS, among other anatomical and physiological factors. Adenoids (pharyngeal tonsils) is a lump of lymphoid tissue on the roof of the nasopharynx. This tissue is physiologically larger in children, in proportion to the size of the nasopharyngeal airway, than in adults, and peaks in size between the ages of 3 and 7 years, then involves during adolescence. Although adenoid hypertrophy is a normal development process, it may result in a mechanical blockage of the nasopharynx and posterior nasal aperture in an excessive enlargement [Lin, C. D. *et al*., 2012]. This blockage hinders the clearance of mucosa, provides a bacterial reservoir, and alters normal sinus ventilation, contributing to a favorable environment of chronic infection and inflammation. Moreover, the adenoids per se may serve as a bacterial biofilm reservoir. Biofilms are organized assemblies of bacteria within a self-

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DOI- https://doi.org/10.5281/zenodo.19566880

assembled extracellular polymeric matrix (adherent) that sticks to surfaces. These biofilms shield bacteria against the host immune defense system and antibiotics, resulting in chronic infection and resistance to treatment. Recent studies indicate that the adenoid surface of children with CRS contains a variety of polymicrobial biofilms, which could also act as a source of constant seeding of pathogens to the paranasal sinuses [Beswick, D. M. *et al*., 2017; Beswick, D. M. *et al*., 2017]. Therefore, the contribution of adenoid hypertrophy to pediatric CRS is probably two-fold, requiring the presence of mechanical obstruction as well as biological persistence of infection [WANG, D. Y. *et al*., 1997; Cedeño, E. E. G. *et al*., 2016].

Although adenoidectomy has been widely accepted as one of the first-line surgical interventions in the treatment of pediatric CRS that has failed to respond to medical treatment, the relationship between the extent of adenoid hypertrophy and the clinical severity of CRS is debatable. Other studies have suggested that the size of the adenoids, determined by the adenoid-nasopharynx ratio (ANR) is not necessarily associated with the severity of symptoms or radiologic appearance, which is why the inflammatory characteristics of the adenoid tissue, and not its mass, may be the main factor in the pathogenesis of the disease [Marseglia, G. L. *et al*., 2007; Ramadan, H. H., & Cost, J. L. 2008; Ramadan, H. H., & Makary, C. A. 2014] with Other studies, on the other hand, show that major adenoid hypertrophy is closely connected with elevated Lund-Mackay CT scores, poor symptoms score, and poor response to medical treatment only. Such a difference in results can be explained by the fact that different studies were performed differently, there was a dissimilarity in the definition of hypertrophy, dissimilarity in patient groups, and the absence of standardized outcome measures. In addition, the practice pattern of adenoidectomy varies because the decision to undergo adenoidectomy is usually grounded on clinical judgment as opposed to objective measurements. There is an urgent necessity to carry out powerful cohort studies incorporating objective radiological variables, including ANR and CT scoring, with validated patient-reported outcome measures (PROMs) and microbiological data to establish the exact role of adenoid hypertrophy in the pathogenesis and prognosis of pediatric CRS [Coticchia, J. *et al*., 2007; Bernstein, J. M. *et al*., 2001; Desrosiers, M. *et al*., 2011; Tosca, M. A. *et al*., 2001; Gerber, M. E., & Kennedy, A. A. 2018; Bhattacharyya, N. *et al*., 2004].

The guidelines to currently manage pediatric CRS are mostly based on a progressive treatment, starting with maximal medical treatment (MMT) comprising of intranasal corticosteroids, saline nasal solutions, and culture-specific antibiotics. Adenoidectomy is typically used in response to patients who do not respond to MMT, especially those who are below 12 years of age and do not have nasal polyps. Nevertheless, there is no clearly established predictive value of the size of adenoids in indicating the success of medical treatment over the need to undergo surgery [Huggill, P. H., & Ballantyne, J. C. 1952; Orlandi, R. R. *et al*., 2016].

The purpose of the study is to fill these gaps by performing a thorough prospective cohort study of 102 children with chronic rhinosinusitis. The main aim is to determine the importance of adenoid hypertrophy (which is the adenoid-nasopharynx ratio) in clinical presentation, microbiological profile, and treatment response of CRS in children.

METHODOLOGY

This was a prospective cohort study carried out at the Department of Otorhinolaryngology-Head and Neck Surgery with the Department of Pediatrics at [Iraq]. The IRB and Ethics Committee approved the study protocol, and both the parents or legal guardians of all subjects provided written informed consent, and the children provided age-appropriate assent. The research was enrolled into the Clinical Trials Registry and followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. The recruitment phase was to be 24 months (between January 1, 2023, and December 31, 2024), followed by a 6-month outcome evaluation follow-up period (to be completed in June 2025).

Respondents and Inclusion and Exclusion.

The research sample included children aged 3-14 years who had symptoms of chronic rhinosinusitis (CRS). The inclusion criteria were based on the European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS 2020) recommendations: being able to have two or more symptoms, one of which should be the symptom of nasal blockage/obstruction/congestion or nasal discharge (anterior/posterior nasal drip), with or without the presence of the symptom of facial pain/pressure and/or Also, objective signs of inflammation were needed, which were manifested by endoscopic

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results (purulent mucus or edema predominantly in the middle of the meatus) or computed tomography (CT) alterations of mucosal opacification in the ostiomeatal complex and /or sinuses.

Exclusion criteria were: (1) adenoidectomy or sinus surgery had been performed before; (2) cystic fibrosis (confirmed by sweat chloride test or genetic testing); (3) primary ciliary dyskinesia; (4) immunodeficiency disorders; (5) craniofacial anomalies of nasal anatomy; (6) acute exacerbation of CRS at The patients with concomitant allergic rhinitis or asthma were not excluded but were stratified in the analysis to adjust the confounding factors.

Sample Size Calculation

IBM SPSS was used to compute the sample size. On the basis of preliminary results and prior literature reporting a failure rate of treatment of about 40% in pediatric CRS patients with adenoid hypertrophy treated medically versus 15% in patients without hypertrophy, we sought to find an odds ratio of 3.5 with 80% power and two-sided alpha error of 0.05. It needed a minimum of 90 patients to account for an estimated dropout rate of 15%. In order to have the strength of subgroup analyses, we recruited 102 patients. A detailed demographic/clinical history was obtained on every participant on enrollment. The variables measured were age, sex, length of symptoms, history of allergic rhinitis, asthma, atopic dermatitis, family history of atopy, passive smoking exposure, attending daycare, and the number of antibiotic courses taken during the last 12 months. These patients had a standardized physical examination with anterior rhinoscopy and rigid nasal endoscopy (2.7 mm 0-degree endoscope under topical anesthesia lidocaine 4% and oxymetazoline 0.05) conducted by a single senior otolaryngologist who was blinded to the radiological results. The Lund-Kennedy endoscopic scoring system was used to record the endoscopic findings, and the presence of purulence, edema, polyps, and scarring were noted. All participants had a non-contrast paranasal sinus CT scan using a low-dose pediatric protocol multidetector CT scanner (slice thickness 12 mm). The severity of radiology was measured based on the Lund-Mackay scoring system, in which each sinus is rated (ranged 0 to 2) and the ostiomeatal complex is rated (ranged 0 to 2) as 0 (no abnormality), 1 (partial opacification), 2 (complete opacification). The size of adenoids was measured by the adenoid-nasopharynx ratio (ANR) technique described by Fujioka *et al*., on mid-sagittal CT reconstructions. It is estimated by dividing the thickness of adenoid tissue (A) by the width of nasopharyngeal airway (N) at the sphenobasioccipital synchondrosis. Patients were classified into two groups based on the set thresholds, i.e., into the Adenoid Hypertrophy Group (ANR $\geq$ 0.70) and Non-Hypertrophy Group (ANR < 0.70). This threshold was chosen because it is associated with high levels of nasopharyngeal blockage and clinical manifestations in children.

Microbiology and Microbiological Sampling and Analysis.

Before treatment, all the participants had their middle meatus swabs collected under endoscopic guidance. The samples were taken in the form of sterile calcium alginate swabs, but not in direct contact with the nasal vestibule to avoid contamination. Samples were immediately taken to the microbiology laboratory in Amies transport medium. Blood agar, chocolate agar, and MacConkey agar plates were done under aerobic and anaerobic cultures. Pathogen identification was done through standard biochemical tests and matrix-assisted laser desorption/ionization-time of flight (MALDI-TOF) mass spectrometry. The Kirby-Bauer disk diffusion test was done to determine antibiotic susceptibility as per the Clinical and Laboratory Standards Institute (CLSI) guidelines. Further samples were examined by scanning electron microscopy (SEM) of a smaller group of patients who were positive on culture, which was biofilm formation, characterized by the presence of bacterial aggregates entrenched in an extracellular polymeric material that adheres to the mucosal surface.

The Sinonasal Outcome Test-20 (SNOT-20) measured disease-specific symptom severity, which is valid and can be used in pediatric populations and is completed by a parent in younger children and by patients themselves in older children. SNOT-20 is a 20-item measure that uses a 0-5 scale (0 means no problem, 5 means problem as bad as it can be) to assess 20 symptoms, with overall scores being 0-100; higher scores reflect increased disease burden. We used the Pediatric Quality of Life Inventory (PedsQL 4.0 Generic Core Scales) to measure generic health-related quality of life, which included physical, emotional, social, and school functioning. Both tests were done at baseline (pre-treatment), 12 weeks (post-treatment), and 6 months post-intervention.

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Treatment Protocol

A conventional 12 weeks of maximal medical therapy (MMT) was administered to all 102 patients. This treatment involved: (1) Intranasal corticosteroids (Mometasone furoate 50 mcg, one spray per nostril every day); (2) High-volume saline nasal irrigation (100200 mL per nostril twice a day with isotonic saline); and (3) Culture-directed oral antibiotics, 21 days (based on middle meatus swab Amoxicillin-Clavulanate was used as a first-line treatment in situations where cultures were negative, or polymicrobial with no dominant pathogen. Second-generation oral antihistamines (Cetirizine or Loratadine) were also given to the patients with confirmed IgE-mediated allergy rhinitis on demand basis. Adherence to medications and irrigation was observed on weekly diary cards and phone check-ins.

Surgical Intervention Criteria

Response to treatment was measured at the end of the 12-week medical therapy. The definition of Clinical Success was a 50-percent reduction of SNOT-20 score and disappearance of endoscopic evidence of inflammation. Surgical intervention was offered to patients who did not respond to medical therapy (<50% improvement in SNOT-20 score) or continued with endoscopic purulence. Considering the nature of the study, which was adenoid pathology, adenoidectomy was the main surgical procedure provided to patients in the Adenoid Hypertrophy Group. Functional Endoscopic Sinus Surgery (FESS) was only used in cases of severe polyposis or anatomical variation that failed to respond to adenoidectomy, but not in the primary analysis of adenoidectomy outcome, to keep the homogeneity. Patients who never received adenoidectomy and failed medical therapy in the Non-Hypertrophy Group were treated by further medical optimization or FESS, but were not subjected to adenoidectomy.

Outcome Measures and Follow-up.

The main outcome was the clinical success rate at 12 weeks, which was the composite outcome of improvement in the symptoms (reduction in SNOT-20 of at least 50) and the endoscopic resolution. The secondary outcomes were: (1) Change in baseline SNOT-20 and PedsQL scores at 12 weeks and 6 months; (2) Recurrence rate at 6 months, which was a reoccurrence of CRS symptoms necessitating further medical or surgical therapy; (3) Number of subsequent antibiotic courses or steroid bursts during follow-up; and (4) Safety profile, which included adverse events during At 4 weeks, 12 weeks and 6 months after enrolment, follow-up visits were arranged. Noral endoscopy was re-performed at every visit, and quality of life questionnaires were re-administered.

Statistical Analysis

Analysis of data was done with the help of IBM SPSS Statistics for Windows, Version 28.0 where the Shapiro-Wilk test was used to test the normality of continuous variables. The data that follows a normal distribution are expressed as mean + standard deviation (SD), and non-normally distributed data are expressed as median and interquartile range (IQR). The variables of a category are stated in terms of frequencies and percentages. The independent Student t-test of continuous variables and Chi-square test or Fisher's exact test of categorical variables were used to compare the Adenoid Hypertrophy and Non-Hypertrophy groups, respectively. Comparison of pre- and post-treatment scores in groups was done by using paired t-tests or Wilcoxon signed-rank tests.

RESULTS

Table 1- Assessment Baseline Demographic and Clinical Characteristics of Study Participants (n=102)

Variable	Total Cohort (n=102)	Adenoid Hypertrophy Group (n=67)	Non-Hypertrophy Group (n=35)	P-value
Age (years), mean ± SD	7.8 ± 2.4	7.2 ± 2.1	9.1 ± 2.3	<0.001*
Male gender, n (%)	58 (56.9)	40 (59.7)	18 (51.4)	0.42
Female gender, n (%)	44 (43.1)	27 (40.3)	17 (48.6)	0.42
Duration of symptoms (months), median (IQR)	8 (6-12)	9 (7-13)	6 (4-9)	0.003*
Previous antibiotic courses (mean ± SD)	4.2 ± 1.8	4.8 ± 1.9	3.1 ± 1.4	<0.001*
Family history of atopy, n (%)	48 (47.1)	35 (52.2)	13 (37.1)	0.15
History of allergic rhinitis, n (%)	52 (51.0)	38 (56.7)	14 (40.0)	0.11

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History of asthma, n (%)	28 (27.5)	21 (31.3)	7 (20.0)	0.23
Passive smoke exposure, n (%)	31 (30.4)	22 (32.8)	9 (25.7)	0.45
Daycare attendance, n (%)	64 (62.7)	45 (67.2)	19 (54.3)	0.20

Table 2- Rate finding Radiological and Endoscopic Assessment Results

Parameter	**Total Cohort (n=102)**	**Adenoid Hypertrophy Group (n=67)**	**Non-Hypertrophy Group (n=35)**	**P-value**
Adenoid-to-Nasopharynx Ratio (ANR)				
Mean ANR ± SD	0.72 ± 0.14	0.83 ± 0.08	0.51 ± 0.11	<0.001*
ANR ≥0.80, n (%)	42 (41.2)	42 (62.7)	0 (0)	<0.001*
ANR 0.70-0.79, n (%)	25 (24.5)	25 (37.3)	0 (0)	-
ANR <0.70, n (%)	35 (34.3)	0 (0)	35 (100)	-
CT Lund-Mackay Score				
Mean score ± SD	12.4 ± 5.2	14.1 ± 4.8	9.2 ± 4.6	<0.001*
Score 0-5 (mild), n (%)	18 (17.6)	6 (9.0)	12 (34.3)	0.002*
Score 6-15 (moderate), n (%)	54 (52.9)	38 (56.7)	16 (45.7)	0.28
Score >15 (severe), n (%)	30 (29.4)	23 (34.3)	7 (20.0)	0.14
Nasal Endoscopy Findings				
Purulent discharge, n (%)	78 (76.5)	56 (83.6)	22 (62.9)	0.02*
Mucosal edema, n (%)	89 (87.3)	61 (91.0)	28 (80.0)	0.12
Nasal polyps, n (%)	8 (7.8)	3 (4.5)	5 (14.3)	0.08
Middle meatus obstruction, n (%)	71 (69.6)	54 (80.6)	17 (48.6)	0.001*

Table 3- Describe Bacteriological Culture Results from Middle Meatus Swabs

Pathogen	**Total Cohort (n=102)**	**Adenoid Hypertrophy Group (n=67)**	**Non-Hypertrophy Group (n=35)**	**P-value**
Positive culture, n (%)	84 (82.4)	59 (88.1)	25 (71.4)	0.04*
Pathogens isolated:				
Streptococcus pneumoniae	28 (27.5)	22 (32.8)	6 (17.1)	0.09
Haemophilus influenzae	31 (30.4)	24 (35.8)	7 (20.0)	0.10
Moraxella catarrhalis	22 (21.6)	18 (26.9)	4 (11.4)	0.07
Staphylococcus aureus	18 (17.6)	9 (13.4)	9 (25.7)	0.12
Mixed flora	34 (33.3)	26 (38.8)	8 (22.9)	0.11
Anaerobic bacteria	12 (11.8)	10 (14.9)	2 (5.7)	0.17
Fungal elements	6 (5.9)	2 (3.0)	4 (11.4)	0.09
Biofilm formation detected	47 (46.1)	38 (56.7)	9 (25.7)	0.003*
Multiple pathogens (>1)	39 (38.2)	30 (44.8)	9 (25.7)	0.06

Table 4- Assessment Pre-treatment Symptom Scores and Quality of Life Measures

Assessment Tool	**Total Cohort (n=102)**	**Adenoid Hypertrophy Group (n=67)**	**Non-Hypertrophy Group (n=35)**	**P-value**
SNOT-20 Score (0-100)				
Mean ± SD	42.3 ± 12.8	46.7 ± 11.4	33.9 ± 11.6	<0.001*

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Severe symptoms (≥40), n (%)	58 (56.9)	47 (70.1)	11 (31.4)	<0.001*
Individual SNOT-20 Domains:				
Need to blow the nose	3.8 ± 1.2	4.2 ± 1.1	3.0 ± 1.1	<0.001*
Sneezing	3.2 ± 1.4	3.4 ± 1.3	2.8 ± 1.5	0.04*
Runny nose	3.9 ± 1.3	4.3 ± 1.2	3.1 ± 1.2	<0.001*
Cough	3.6 ± 1.5	4.0 ± 1.4	2.8 ± 1.4	<0.001*
Post-nasal drip	4.1 ± 1.2	4.5 ± 1.1	3.3 ± 1.1	<0.001*
Thick nasal discharge	3.7 ± 1.4	4.1 ± 1.3	2.9 ± 1.3	<0.001*
Ear fullness	2.8 ± 1.6	3.2 ± 1.5	2.0 ± 1.5	<0.001*
Dizziness	1.9 ± 1.4	2.1 ± 1.4	1.5 ± 1.3	0.03*
Reduced sense of smell/taste	2.4 ± 1.7	2.6 ± 1.6	1.9 ± 1.8	0.04*
Facial pain/pressure	3.1 ± 1.6	3.4 ± 1.5	2.5 ± 1.6	0.005*
Difficulty sleeping	3.5 ± 1.5	3.9 ± 1.4	2.7 ± 1.4	<0.001*
Waking up at night	3.2 ± 1.6	3.6 ± 1.5	2.4 ± 1.5	<0.001*
Lack of a good night's sleep	3.3 ± 1.5	3.7 ± 1.4	2.5 ± 1.4	<0.001*
Fatigue	3.4 ± 1.4	3.8 ± 1.3	2.6 ± 1.3	<0.001*
Reduced productivity	2.9 ± 1.6	3.2 ± 1.5	2.3 ± 1.6	0.006*
Reduced concentration	2.7 ± 1.5	3.0 ± 1.4	2.1 ± 1.5	0.003*
Frustrated/restless/irritable	2.8 ± 1.6	3.1 ± 1.5	2.2 ± 1.6	0.006*
Sad	2.1 ± 1.5	2.3 ± 1.5	1.7 ± 1.4	0.04*
Embarrassed	1.8 ± 1.4	2.0 ± 1.4	1.4 ± 1.3	0.03*
Pediatric Quality of Life Inventory (PedsQL)				
Total score (0-100)	68.4 ± 14.2	63.7 ± 13.8	77.3 ± 12.4	<0.001*
Physical functioning	71.2 ± 15.6	66.8 ± 14.9	79.6 ± 13.8	<0.001*
Emotional functioning	69.8 ± 16.3	64.9 ± 15.7	79.2 ± 14.2	<0.001*
Social functioning	72.6 ± 14.8	68.4 ± 14.2	80.7 ± 12.6	<0.001*
School functioning	60.1 ± 17.4	54.8 ± 16.8	70.3 ± 15.2	<0.001*

Table 5- Final results Treatment Response and Outcomes at 12 Weeks

Outcome Measure	**Adenoid Hypertrophy Group - Medical Therapy Only (n=25)**	**Adenoid Hypertrophy Group - Post-Adenoidectomy (n=42)**	**Non-Hypertrophy Group - Medical Therapy (n=35)**	**P-value***
Clinical Success Rate				
Complete resolution, n (%)	8 (32.0)	35 (83.3)	22 (62.9)	<0.001
Partial improvement, n (%)	11 (44.0)	6 (14.3)	10 (28.6)	0.002
Treatment failure, n (%)	6 (24.0)	1 (2.4)	3 (8.6)	<0.001
Post-treatment SNOT-20 Score				
Mean ± SD	28.4 ± 9.6	18.2 ± 7.4	21.6 ± 8.8	<0.001
Mean reduction from baseline	18.3 ± 8.2	28.5 ± 9.1	12.3 ± 7.6	<0.001
Post-treatment PedsQL Score				
Mean ± SD	74.6 ± 11.8	84.2 ± 9.6	81.4 ± 10.2	<0.001
Mean improvement from baseline	10.9 ± 9.4	20.5 ± 10.8	4.1 ± 8.6	<0.001
Recurrence Rate				

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at 6 months				
Recurrence, n (%)	9 (36.0)	3 (7.1)	7 (20.0)	0.004
Additional Interventions Required				
Additional antibiotics, n (%)	12 (48.0)	4 (9.5)	8 (22.9)	<0.001
Oral steroids course, n (%)	7 (28.0)	2 (4.8)	5 (14.3)	0.006
Repeat imaging, n (%)	8 (32.0)	3 (7.1)	6 (17.1)	0.008

Table 6- Multivariate Logistic Regression Analysis for Predictors of Treatment Failure in Pediatric CRS

Variable	**Unadjusted OR (95% CI)**	**p-value**	**Adjusted OR (95% CI)***	**P-value**
Adenoid Hypertrophy (ANR ≥0.70)	3.42 (1.52-7.68)	0.003	2.87 (1.21-6.82)	0.017
Age <6 years	2.18 (1.02-4.65)	0.044	1.76 (0.78-3.98)	0.176
CT Lund-Mackay Score >15	2.64 (1.18-5.89)	0.018	1.92 (0.81-4.56)	0.139
Biofilm formation present	3.76 (1.68-8.42)	0.001	3.24 (1.38-7.61)	0.007
Multiple pathogens cultured	2.34 (1.06-5.16)	0.035	1.68 (0.71-3.97)	0.237
History of allergic rhinitis	1.82 (0.84-3.94)	0.129	1.45 (0.63-3.34)	0.381
History of asthma	1.56 (0.68-3.58)	0.295	1.23 (0.50-3.02)	0.651
Passive smoke exposure	1.94 (0.86-4.37)	0.110	1.52 (0.63-3.66)	0.349
Previous antibiotic courses >5	2.86 (1.28-6.39)	0.011	2.14 (0.91-5.04)	0.082
Daycare attendance	1.42 (0.65-3.10)	0.378	1.18 (0.51-2.73)	0.694
Positive family history of atopy	1.68 (0.78-3.62)	0.186	1.34 (0.58-3.09)	0.491
Duration of symptoms >12 months	2.12 (0.96-4.68)	0.063	1.64 (0.70-3.84)	0.256

DISCUSSION

Chronic rhinosinusitis (CRS) is one of the most common chronic inflammatory diseases in the pediatric population that has a significant burden on healthcare systems, education, and quality of life. Pediatric CRS differs greatly in pathophysiology, microbiology and management strategies compared to adult counterpart due to its definition which involves inflammation of the nose, paranasal sinuses defined by the European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS) as two or more symptoms, such as nasal blockage, obstruction, congestion, or nasal discharge with or without facial pain/ In contrast to adults, where type 2 inflammatory pathways and nasal polyposis are commonly related to CRS, pediatric CRS is commonly associated with adenoid pathology, immune immatureness, and environmental influences. CRS is thought to be prevalent in children between 2% and 4%, with some studies indicating even higher rates of CRS in the context of recurrent acute rhinosinusitis, which does not resolve entirely between episodes. As a result, the etiologies and optimal treatment algorithms behind pediatric CRS are a high priority in pediatric otolaryngology [Gilani, S., & Shin, J. J. 2017].

Adenoid hypertrophy has been an essential factor among the other anatomical and physiological factors that lead to pediatric CRS. The adenoids or pharyngeal tonsils is a lump of lymphoid tissue in the roof of the nasopharynx. This tissue is physiologically larger in children relative to the size of the nasopharyngeal airway when compared to adults, and it reaches its maximum size when children are 3-7 years old and then becomes smaller due to involution during adolescence. Although adenoid hypertrophy is a normal process of development, its excessive growth may cause mechanical blockage of the nasopharynx and the posterior nasal aperture. This blockage hinders mucociliary clearance, forms a reservoir of bacterial colonization, and interferes with normal ventilation, thus forming a favourable environment to support chronic infection and inflammatory processes. Moreover, even the adenoids may serve as a bacterial biofilm reservoir. Biofilms refer to organized communities of bacteria [Brietzke, S. E. *et al*., 2014] which are entrenched in a self-produced extracellular polymeric matrix, which

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attaches to surfaces. Such biofilms shield bacteria against host immune response and antibiotics and cause chronic infection and resistance to treatment. Recent reports indicate that the adenoid surface of children with CRS contains a variety of polymicrobial biofilms that can be a persistent reservoir of pathogens that seed the paranasal sinuses.

Therefore, adenoid hypertrophy in CRS of children is likely to play a dual role, both mechanically and biologically maintaining infection. Although adenoidectomy is widely accepted as a first-line surgery in pediatric CRS that has not responded to medical treatment, there is still debate over what exactly the degree of adenoid hypertrophy is correlated with the severity of the clinical manifestation of CRS. Other studies indicate that the size of the adenoids in terms of adenoid-to-nasopharynx ratio (ANR) does not necessarily correlate with the severity of the symptoms or radiological appearance, and the inflammatory nature of the adenoid tissue is probably the major cause of the disease. On the other hand, other studies have shown that high adenoid hypertrophy is highly correlated with poorer Lund-Mackay CT scores, poorer symptom scores, and poorer response to medical management alone. This difference in results could be explained by the different study designs, the differences in the definition of hypertrophy, the dissimilarity of patient groups, and the absence of standardized outcome measures. Furthermore, adenoidectomy is usually performed on clinical judgment and not objective measurements, thus making the practice patterns varied. There is an urgent requirement of cohort studies with solid methodologies that combine objective radiological (ANR and CT scoring) with validated patient-reported outcome measures (PROM) and microbiological data to dissect the role of adenoid hypertrophy in the pathogenesis and prognosis of pediatric CRS [Vandenberg, S. J., & Heatley, D. G. 1997; Nia, S. J. *et al*., 2014; Chandran, S. K., & Higgins, T. S. 2013; Shin, S. Y. *et al*., 2009; Fokkens, W. J. *et al*., 2012].

The contemporary management approaches to pediatric CRS generally involve a gradual process starting with maximal medical therapy (MMT) that involves the use of intranasal corticosteroids, saline irrigations, and culture-directed antibiotics. Adenoidectomy is mostly used in cases where MMT fails, especially in patients who are below 12 years and do not have nasal polyps. Nevertheless, the predictive usefulness of the size of the adenoid in the effect of medical treatment, as opposed to the need to conduct surgery, is not yet clearly defined. Determining the predictors of treatment failure may assist clinicians to be more selective in their approaches to treatment without subjecting patients to unnecessary surgeries when they can respond to medical management, and refer patients to surgery sooner when they are unlikely to respond. Moreover, the health-related quality of life (HRQoL) when adenoid hypertrophy occurs in children with CRS is another aspect that needs to be investigated. Although it is clear that CRS has detrimental effects on sleep, school achievement, and emotional health, the incremental load due to adenoid hypertrophy has been measured in only a few studies. This relationship is important to understand so that parents can be counselled and realistic expectations set on the outcomes of the treatment.

CONCLUSION

Lastly, we will seek independent predictors of treatment failure by using multivariate analysis to examine the interaction of adenoid size, the presence of biofilm, and clinical outcomes. Explaining these relationships, this research aims to offer evidence-based insights, which can enhance clinical decision-making algorithms, maximize resource use, and eventually lead to the improvement of health outcomes and quality of life of children with chronic rhinosinusitis. Such a strict examination will help us to better understand whether adenoid hypertrophy is just a spectator in the inflammatory process or a primary disease persistence factor, thus informing more individualized and effective therapy in pediatric otolaryngology.

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Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:
Hussian, W. N., Sharhan, F. J. & Joada, A. F. “Adenoid Hypertrophy and Its Role in Pediatric Chronic Rhinosinusitis: A Cohort Analysis” *Sarcouncil Journal of Medical Series* 5.4 (2026): pp 1-10.

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