Systematic Review
Iranian Journal of Otorhinolaryngology, Vol.38(5), Serial No.148, Sep-2026

Mechanistic Insights into the Role of Adenoid Hypertrophy and Chronic Adenoiditis in the Pathogenesis of Pediatric Chronic Rhinosinusitis: A Systematic Review

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Abstract

Objective:
To synthesize available evidence on the mechanistic role of adenoid hypertrophy and chronic adenoiditis in the pathogenesis of pediatric chronic rhinosinusitis (CRS), given that the mechanistic relationship between adenoid disease and pediatric CRS remains incompletely synthesized.

Data Sources:
PubMed/MEDLINE, Embase, Scopus, Web of Science Core Collection, and Cochrane Library were systematically searched in accordance with PRISMA 2020 guidelines.

Methods of Review:
Eligible studies included children with CRS and evaluated adenoid-related mechanisms, including mechanical obstruction, impaired drainage, microbial reservoir function, biofilm formation, immune dysregulation, epithelial dysfunction, mucociliary impairment, or clinical outcomes after adenoidectomy. Study quality was appraised using the JBI Critical Appraisal Tools. Due to heterogeneity in study design, diagnostic criteria, and reported outcomes, a narrative synthesis was performed.

Results:
Nine studies involving 927 pediatric participants were included. Evidence for mechanical obstruction was mixed, as adenoid size did not consistently correlate with CRS severity. Stronger evidence supported the role of the adenoid as a microbial reservoir and biofilm-bearing tissue, with studies reporting pathogenic bacterial colonization and extensive adenoidal biofilm in children with CRS. Limited molecular evidence suggested reduced surfactant protein-D expression, while epithelial barrier dysfunction and mucociliary impairment were supported mainly by indirect findings. Adenoidectomy improved symptoms and quality of life in selected children with medically refractory CRS.

Conclusion:
Adenoid hypertrophy and chronic adenoiditis appear to be associated with pediatric CRS through combined obstructive, microbial, biofilm-related, inflammatory, and immunological pathways, although causal relationships cannot be established from the available evidence. Adenoidectomy appears beneficial in selected refractory cases, although further prospective mechanistic studies are needed.

Keywords: Adenoid Hypertrophy, Chronic Adenoiditis, Pediatric Chronic Rhinosinusitis, Biofilm, Microbial Reservoir, Adenoidectomy

Received date: 14 Jul 2026
Accepted date: 15 Aug 2026

__Please cite this article;__ Pratomo GRA, Trihandoko F. Mechanistic Insights into the Role of Adenoid Hypertrophy and Chronic Adenoiditis in the Pathogenesis of Pediatric Chronic Rhinosinusitis: A Systematic Review. Iran J Otorhinolaryngol. 2026:38(5):289-300. Doi: 10.22038/ijorl.2026.97252.4200

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Introduction

Chronic rhinosinusitis (CRS) in children is a persistent inflammatory disorder of the nasal and paranasal sinus mucosa lasting more than 12 weeks. It commonly presents with nasal obstruction, anterior or posterior rhinorrhea, chronic cough, facial pressure, and sleep disturbance, with potential effects on quality of life, growth, and school performance. Diagnosis remains challenging because symptoms often overlap with other pediatric upper airway disorders, particularly adenoid hypertrophy, chronic adenoiditis, and allergic rhinitis (1).

The pathogenesis of pediatric CRS is multifactorial, involving recurrent upper respiratory tract infection, allergy, gastroesophageal reflux, anatomical variation, impaired mucociliary clearance, microbial colonization, and local immune dysfunction (1–4). Among these factors, adenoid disease is particularly relevant in children because the adenoid is located in the nasopharynx and functions as part of Waldeyer's lymphoid ring. Adenoid hypertrophy may impair posterior nasal airflow, sinus ventilation, and mucus drainage, whereas chronic adenoiditis may contribute to persistent sinonasal inflammation through non-obstructive mechanisms (5–7).

Increasing evidence suggests that adenoid disease contributes to pediatric CRS beyond mechanical obstruction. Chronically inflamed adenoid tissue may serve as a reservoir for pathogenic microorganisms and bacterial biofilms, allowing persistent infection despite host immune responses and antimicrobial therapy (8–10).

In addition, adenoid-related immune and epithelial alterations, including impaired innate defense, altered Toll-like receptor signaling, epithelial barrier dysfunction, and impaired mucociliary clearance, may facilitate microbial persistence and chronic mucosal inflammation (11,12).

Despite these findings, it remains unclear whether adenoid hypertrophy and chronic adenoiditis directly cause pediatric CRS, exacerbate pre-existing sinonasal inflammation, or share overlapping pathogenic pathways with CRS. Previous literature has often emphasized clinical diagnosis and adenoidectomy after failed medical therapy, while the mechanistic pathways linking adenoid disease and pediatric CRS have not been consistently synthesized across clinical, microbiological, histopathological, and molecular studies (1,13).

Several prior reviews have addressed the relationship between adenoid disease and pediatric CRS, but each has limitations that remain to be addressed. These include a systematic review restricted to studies through 2018 without mechanism-based stratification or formal quality appraisal (5), a narrative overview centered on clinical presentation and management rather than pathogenic mechanisms (6), and a broader narrative review of adenoid hypertrophy spanning multiple conditions beyond CRS (7); none applied a PRISMA-based methodology with formal quality appraisal or organized evidence within a mechanistic framework, and several relevant studies have since been published.

Therefore, this systematic review aims to synthesize available evidence on the mechanistic role of adenoid hypertrophy and chronic adenoiditis in the pathogenesis of pediatric CRS, focusing on obstruction, impaired drainage, microbial reservoir function, biofilm formation, immune dysregulation, epithelial dysfunction, mucociliary impairment, and the therapeutic implications of adenoidectomy.

Materials and Methods

This systematic review synthesized evidence on the mechanistic role of adenoid hypertrophy and chronic adenoiditis in the pathogenesis of pediatric CRS. The review was reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses 2020 statement (PRISMA 2020) (14).

Eligibility Criteria

Eligibility criteria were structured using the PECO framework. Eligible studies included children and adolescents aged 0–18 years with CRS and evaluated adenoid-related factors, including adenoid hypertrophy, chronic adenoiditis, adenoid biofilm, microbial colonization, microbial reservoir function, inflammatory changes, epithelial barrier dysfunction, or impaired mucociliary clearance. Comparators included healthy children, children without CRS, children with different grades of adenoid hypertrophy, or preoperative versus postoperative comparisons.

Studies without a formal comparator were also included if they provided relevant mechanistic data.

Information Sources and Search Strategy

A systematic literature search was conducted in PubMed/MEDLINE, Embase, Scopus, Web of Science Core Collection, and Cochrane Library using controlled vocabulary and free-text terms related to pediatric CRS and adenoid-related mechanisms. The search strategy in PubMed/ MEDLINE was: (("Rhinosinusitis "[Mesh] OR "Sinusitis"[Mesh] OR rhinosinusitis [tiab] OR "chronic rhinosinusitis" [tiab] OR "chronic sinusitis" [tiab] OR CRS [tiab]) AND ("Child" [Mesh] OR " Adolescent" [Mesh] OR "Infant" [Mesh] OR child*[tiab] OR pediatric* [tiab] OR paediatric* [tiab] OR adolescent*[tiab] OR infant* [tiab]) AND ("Adenoids"[Mesh] OR adenoid* [tiab] OR "adenoid hypertrophy" [tiab] OR "adenoidal hypertrophy"[tiab] OR adenoiditis s[tiab] OR "chronic adenoiditis" [tiab] OR "nasopharyngeal tonsil" [tiab]) AND (pathogenes*[tiab] OR mechanism*[tiab] OR obstruct* [tiab] OR ventilation [tiab] OR drainage [tiab] OR biofilm* [tiab] OR microbiome [tiab] OR microbiota tiab] OR bacteria* [tiab] OR colonization [tiab] OR colonization [tiab] OR reservoir[tiab] OR inflammation [tiab] OR cytokine* [tiab] OR chemokine*[tiab] OR "Toll-like receptor"[tiab] OR TLR[tiab] OR "NF-kB" [tiab] OR "NF-κB"[tiab] OR "epithelial barrier"[tiab] OR epitheli* [tiab] OR "mucociliary clearance" [tiab] OR cilia* [tiab])). The complete search strategy for other databases is provided in (Table 1).

Table 1. Search strategy used for each electronic database.

Database	String
PubMed/ MEDLINE	(("Rhinosinusitis"[Mesh] OR "Sinusitis"[Mesh] OR rhinosinusitis[tiab] OR "chronic rhinosinusitis"[tiab] OR "chronic sinusitis"[tiab] OR CRS[tiab]) AND ("Child"[Mesh] OR "Adolescent"[Mesh] OR "Infant"[Mesh] OR child*[tiab] OR pediatric*[tiab] OR paediatric*[tiab] OR adolescent*[tiab] OR infant*[tiab]) AND ("Adenoids"[Mesh] OR adenoid*[tiab] OR "adenoid hypertrophy"[tiab] OR "adenoidal hypertrophy"[tiab] OR adenoiditis[tiab] OR "chronic adenoiditis"[tiab] OR "nasopharyngeal tonsil"[tiab]) AND (pathogenes*[tiab] OR mechanism*[tiab] OR obstruct*[tiab] OR ventilation[tiab] OR drainage[tiab] OR biofilm*[tiab] OR microbiome[tiab] OR microbiota[tiab] OR bacteria*[tiab] OR colonization[tiab] OR colonisation[tiab] OR reservoir[tiab] OR inflammation[tiab] OR cytokine*[tiab] OR chemokine*[tiab] OR "Toll-like receptor"[tiab] OR TLR[tiab] OR "NF-kB"[tiab] OR "NF-κB"[tiab] OR "epithelial barrier"[tiab] OR epitheli*[tiab] OR "mucociliary clearance"[tiab] OR cilia*[tiab]))
Embase	('rhinosinusitis'/exp OR 'sinusitis'/exp OR rhinosinusitis:ti,ab OR 'chronic rhinosinusitis':ti,ab OR 'chronic sinusitis':ti,ab OR CRS:ti,ab) AND ('child'/exp OR 'adolescent'/exp OR 'infant'/exp OR child*:ti,ab OR pediatric*:ti,ab OR paediatric*:ti,ab OR adolescent*:ti,ab OR infant*:ti,ab) AND ('adenoid'/exp OR adenoid*:ti,ab OR 'adenoid hypertrophy':ti,ab OR 'adenoidal hypertrophy':ti,ab OR adenoiditis:ti,ab OR 'chronic adenoiditis':ti,ab OR 'nasopharyngeal tonsil':ti,ab) AND (pathogenes*:ti,ab OR mechanism*:ti,ab OR obstruct*:ti,ab OR ventilation:ti,ab OR drainage:ti,ab OR biofilm*:ti,ab OR microbiome:ti,ab OR microbiota:ti,ab OR bacteria*:ti,ab OR colonization:ti,ab OR colonisation:ti,ab OR reservoir:ti,ab OR inflammation:ti,ab OR cytokine*:ti,ab OR chemokine*:ti,ab OR 'toll like receptor':ti,ab OR TLR:ti,ab OR 'NF-kB':ti,ab OR 'NF-κB':ti,ab OR 'epithelial barrier':ti,ab OR epitheli*:ti,ab OR 'mucociliary clearance':ti,ab OR cilia*:ti,ab)
Scopus	TITLE-ABS-KEY (rhinosinusitis OR "chronic rhinosinusitis" OR "chronic sinusitis" OR sinusitis OR CRS) AND TITLE-ABS-KEY (child* OR pediatric* OR paediatric* OR adolescent* OR infant*) AND TITLE-ABS-KEY (adenoid* OR "adenoid hypertrophy" OR "adenoidal hypertrophy" OR adenoiditis OR "chronic adenoiditis" OR "nasopharyngeal tonsil") AND TITLE-ABS-KEY (pathogenes* OR mechanism* OR obstruct* OR ventilation OR drainage OR biofilm* OR microbiome OR microbiota OR bacteria* OR colonization OR colonisation OR reservoir OR inflammation OR cytokine* OR chemokine* OR "Toll-like receptor" OR TLR OR "NF-kB" OR "NF-κB" OR "epithelial barrier" OR epitheli* OR "mucociliary clearance" OR cilia*)
Web of Science Core Collection	TS=(rhinosinusitis OR "chronic rhinosinusitis" OR "chronic sinusitis" OR sinusitis OR CRS) AND TS=(child* OR pediatric* OR paediatric* OR adolescent* OR infant*) AND TS=(adenoid* OR "adenoid hypertrophy" OR "adenoidal hypertrophy" OR adenoiditis OR "chronic adenoiditis" OR "nasopharyngeal tonsil") AND TS=(pathogenes* OR mechanism* OR obstruct* OR ventilation OR drainage OR biofilm* OR microbiome OR microbiota OR bacteria* OR colonization OR colonisation OR reservoir OR inflammation OR cytokine* OR chemokine* OR "Toll-like receptor" OR TLR OR "NF-kB" OR "NF-κB" OR "epithelial barrier" OR epitheli* OR "mucociliary clearance" OR cilia*)
Cochrane Library	([mh Rhinosinusitis] OR [mh Sinusitis] OR rhinosinusitis:ti,ab,kw OR "chronic rhinosinusitis":ti,ab,kw OR "chronic sinusitis":ti,ab,kw OR CRS:ti,ab,kw) AND ([mh Child] OR [mh Adolescent] OR [mh Infant] OR child*:ti,ab,kw OR pediatric*:ti,ab,kw OR paediatric*:ti,ab,kw OR adolescent*:ti,ab,kw OR infant*:ti,ab,kw) AND ([mh Adenoids] OR adenoid*:ti,ab,kw OR "adenoid hypertrophy":ti,ab,kw OR "adenoidal hypertrophy":ti,ab,kw OR adenoiditis:ti,ab,kw OR "chronic adenoiditis":ti,ab,kw OR "nasopharyngeal tonsil":ti,ab,kw) AND (pathogenes*:ti,ab,kw OR mechanism*:ti,ab,kw OR obstruct*:ti,ab,kw OR ventilation:ti,ab,kw OR drainage:ti,ab,kw OR biofilm*:ti,ab,kw OR microbiome:ti,ab,kw OR microbiota:ti,ab,kw OR bacteria*:ti,ab,kw OR colonization:ti,ab,kw OR colonisation:ti,ab,kw OR reservoir:ti,ab,kw OR inflammation:ti,ab,kw OR cytokine*:ti,ab,kw OR chemokine*:ti,ab,kw OR "Toll-like receptor":ti,ab,kw OR TLR:ti,ab,kw OR "NF-kB":ti,ab,kw OR "NF-κB":ti,ab,kw OR "epithelial barrier":ti,ab,kw OR epitheli*:ti,ab,kw OR "mucociliary clearance":ti,ab,kw OR cilia*:ti,ab,kw)
Google Scholar	"pediatric chronic rhinosinusitis" AND adenoid AND (hypertrophy OR adenoiditis OR biofilm OR reservoir OR obstruction OR inflammation OR immune OR mucociliary)
Clinical Trials.gov	pediatric chronic rhinosinusitis AND adenoid; chronic rhinosinusitis AND adenoidectomy; adenoid hypertrophy AND rhinosinusitis

Selection Process

Prior to this process, all records retrieved from the databases were imported into Mendeley reference manager to undergo record deduplication, if any duplication was identified. Subsequently, the study selection process was performed in two main stages. First, initial screening was done to evaluate the relevance of the records to the present review; irrelevant records were excluded at this stage. Second, a full-text review was carried out to assess the eligibility of the articles for inclusion based on the predetermined criteria. This process was performed by two investigators (G.R.A.P. and F.T.), and any discrepancies that arose during the process were addressed through in-depth discussion until a consensus was achieved.

Data Collection Process

Data were extracted independently by two reviewers using a standardized extraction form. Extracted data included study characteristics, population characteristics, CRS diagnostic methods, adenoid assessment methods, mechanistic findings, microbiological findings, immunological and epithelial findings, and clinical outcomes. The primary outcomes were mechanistic findings linking adenoid hypertrophy or chronic adenoiditis with pediatric CRS, while secondary outcomes included clinical implications such as response to medical therapy, symptom improvement after adenoidectomy, recurrence, and need for further surgical intervention.

Quality Assessment

Two reviewers (G.R.A.P. and F.T.) independently assessed the quality of the included studies using the JBI Critical Appraisal Tools (https://jbi.global/critical-appraisal-tools), tailored to the specific design of each study. Any disagreements were resolved through discussions between the reviewers.

Data Synthesis

A narrative synthesis was performed because of substantial clinical and methodological heterogeneity across study designs, diagnostic criteria, adenoid assessment methods, sampling techniques, laboratory methods, and reported outcomes. Studies were grouped according to predefined mechanistic domains, including mechanical obstruction, impaired ventilation and drainage, chronic inflammation, microbial reservoir function, biofilm formation, immune dysregulation, epithelial barrier dysfunction, impaired mucociliary clearance, and therapeutic implications of adenoidectomy. Meta-analysis was not performed because the included studies did not report sufficiently comparable quantitative outcomes.

Results

Study Selection

The initial search across five databases yielded 76 records. After 41 duplicate records were removed, 35 records remained for title and abstract screening, of which 21 were excluded for clear irrelevance to the review question. The remaining 14 reports were retrieved in full-text, and all were successfully obtained. Following full-text assessment against the predefined eligibility criteria, five articles were excluded for the following reasons: ineligible population or clinical condition; adenoid-related exposure not evaluated; and no relevant outcome, leaving nine studies that met the inclusion criteria and were carried forward into the final narrative synthesis. The included studies investigated the relationship between adenoid hypertrophy, chronic adenoiditis, and pediatric chronic rhinosinusitis through clinical, radiological, microbiological, biofilm, histopathological, molecular, and surgical approaches. The study selection process is summarized in the PRISMA flow diagram (Figure 1).

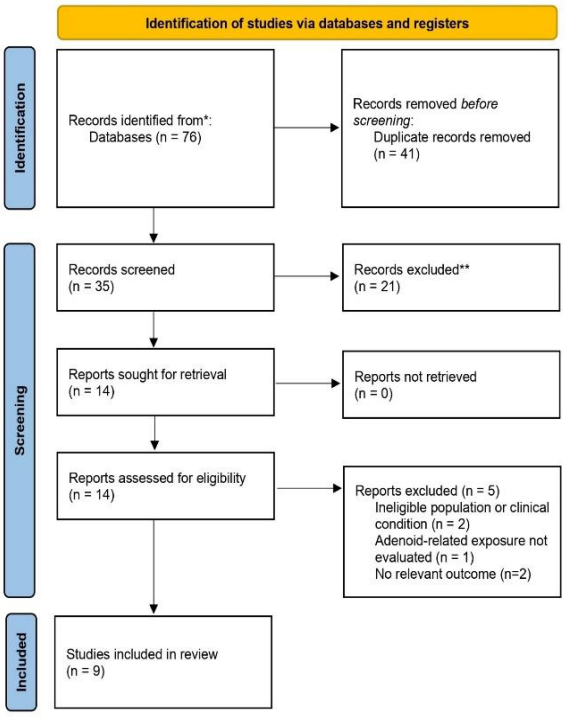

Fig 1. PRISMA framework

Characteristics of Included Studies

The nine included studies were published between 2007 and 2024 and involved a total of 927 pediatric participants. The sample size ranged from 16 to 410 children. The age range across studies varied from 3 months to 16 years, although most studies focused on children younger than 12–15 years. The included studies were conducted in the United States, Turkey, South Korea, China, India, Egypt, and Romania, reflecting heterogeneous clinical settings and populations.The study designs included retrospective cohort studies, retrospective chart reviews of prospectively collected data, prospective interventional studies, clinical observational studies, comparative microanatomical studies, preliminary molecular case-control studies, and prospective comparative morphological studies. No randomized controlled trial was identified among the included studies.Three studies primarily addressed clinical or therapeutic outcomes after adenoidectomy or differentiation of CRS from chronic adenoiditis (15–17).

Three studies primarily evaluated microbiological or biofilm-related mechanisms using adenoid tissue culture or scanning electron microscopy (8,9,18). One study evaluated innate immune gene expression in adenoidal epithelial cells (11). Two studies evaluated the relationship between adenoid size, radiological sinus disease, and adenoid-related obstruction (19,20).

The diagnostic criteria for pediatric CRS varied across studies. Some studies used symptom duration and clinical criteria, while others incorporated CT findings, endoscopy, Lund–Mackay scoring, or radiographic assessment. Several studies excluded children with systemic or structural conditions that could confound CRS pathogenesis, such as cystic fibrosis, primary ciliary dyskinesia, immunodeficiency, craniofacial abnormality, asthma, allergy, nasal polyps, or prior sinonasal surgery. However, exclusion criteria were not uniformly reported across all studies. The characteristics of all included studies along with their clinical outcomes were summarized in (Table 2).

Table 2. Study characteristics and clinical outcomes.

Study	Country	Sample Size (% Female) and Age	Inclusion and Exclusion Criteria	Clinical Outcomes		
				Response to Medical Treatment	Response to Adenoidectomy or Surgical Treatment	Key Clinical Outcome
Coticchia et al., 2007	United States	16 children (25%); age range 3 months–10 years	Inclusion criteria: Children undergoing adenoidectomy for chronic rhinosinusitis (CRS) or obstructive sleep apnea (OSA); CRS defined as paranasal sinus infection lasting >3 months and failing ≥4 weeks of oral antibiotics. Exclusion criteria: N/R.	CRS cases had failed prolonged oral antibiotics for at least 4 weeks before adenoidectomy	No symptom outcome was directly measured; the study proposes that removing biofilm-bearing adenoid tissue may explain clinical benefit of adenoidectomy	The primary outcome was biofilm coverage, not clinical response
Berçin et al., 2007	Turkey	42 children (38.09%); age range 6–16 years; mean age 10.36 ± 2.89 years	Inclusion criteria: Children with adenoid vegetation and/or nasal discharge who underwent paranasal sinus CT; children with adenoid enlargement and chronic sinusitis underwent adenoidectomy. Exclusion criteria: N/R.	N/R	Children with adenoid enlargement and chronic sinusitis underwent adenoidectomy, but postoperative response was not the primary outcome	No significant relationship between adenoid volume and CT sinusitis extent
Shin et al., 2008	South Korea	410 children (40.2%); <14 years, mean age 8.5 years	Inclusion criteria: Children undergoing adenoidectomy from 2005 to 2007 for symptoms of adenoid hypertrophy, including mouth breathing, snoring, and nasal airway obstruction. Exclusion criteria: Unilateral sinusitis on radiograph; antibiotic treatment within 2 weeks before surgery.	Antibiotic use within 2 weeks before surgery was an exclusion criterion; response to previous medical therapy was not analyzed as an outcome	Adenoidectomy was performed in all included children; clinical response after surgery was not measured	Bacterial isolation from adenoid tissue, rather than adenoid size, correlated with sinusitis grade
Qu et al., 2015	China	18 children: 9 with adenoid hypertrophy and pediatric CRS (44.4%) and 9 with simple adenoid hypertrophy without	Inclusion criteria: Children undergoing adenoidectomy; pediatric CRS group met EPOS 2012 criteria with sinonasal symptoms >12 weeks and mucopurulent drainage from the middle meatus; AH group had adenoid hypertrophy without clinical or endoscopic sinus disease. Exclusion criteria: Nasal allergy, asthma, positive skin-prick test, nasal polyps,	N/R	All patients underwent adenoidectomy for AH with or without pediatric CRS; postoperative clinical response was not evaluated	Pediatric CRS group had higher SNOT-22 score than AH-only group: 30.9 ± 18.5 vs. 11.8 ± 7.0, p = 0.011

		CRS (44.4%); age range 4–14 years	immunodeficiency, immotile ciliary syndrome, recent common cold.			
Bettadahalli and Chakravarti, 2017	India	60 children (30%); age range 4–12 years; mean age 7.32 ± 2.39 years	Inclusion criteria: Children aged 4–12 years undergoing adenoidectomy or adenotonsillectomy for CRS refractory to 4 weeks of amoxicillin–clavulanate or second-generation cephalosporin; adenoid hypertrophy grades II–IV. Exclusion criteria: Craniofacial abnormalities, cleft palate, submucous cleft palate, marked nasal anatomical abnormality, cystic fibrosis, immunodeficiency, primary ciliary dyskinesia, chronic respiratory or cardiac disease, known allergy, nasal polyps, asthma, Down syndrome, other neurological syndromes.	Only children with refractory CRS after 4 weeks of amoxicillin–clavulanate or second-generation cephalosporin were included	Significant postoperative improvement in global symptom severity: total score decreased from 36.73 ± 7.70 to 16.17 ± 8.61, p = 0.000; overall SN-5 improved from 4.59 ± 0.98 to 1.55 ± 1.06; clinically meaningful quality-of-life improvement in 53/60 children (88.3%)	Adenoidectomy or adenotonsillectomy improved symptoms and quality of life in refractory pediatric CRS
Ramadan and Ashry, 2021	Egypt	60 children (50%); age range 3–12 years; mean age 6.00 ± 2.05 years	Inclusion criteria: Children presenting with symptoms suggestive of adenoid hypertrophy, including mouth breathing, snoring, and nasal airway obstruction; all underwent endoscopic adenoid grading, CT sinus assessment, and adenoidectomy. Exclusion criteria: Septal deviation, previous sinus surgery, previous adenoidectomy, antibiotic treatment, age <3 or >12 years.	Antibiotic-treated patients were excluded; medical response was not evaluated	Adenoidectomy was performed in all patients; postoperative symptom response was not quantified	Positive CT findings were present in 55% of children with AH; CT rhinosinusitis findings were significantly associated with adenoid grade
Bugari et al., 2021	Romania	107 children (43.93%); age range 4–15 years; mean age 7.24 ± 2.61 years	Inclusion criteria: Children with adenoid hypertrophy and either chronic rhinosinusitis or obstructive sleep apnea; diagnosis supported by clinical screening forms, endoscopy, nasal bacteriology, and postoperative tissue evaluation. Exclusion criteria: Asthma was clinically excluded; other exclusion criteria were not clearly specified.	CRS was defined as disease lasting >3 months with failure of oral antibiotic treatment even after 5 weeks	All patients underwent endoscopically guided adenoidectomy; the authors stated that adenoidectomy relieved symptoms, but standardized symptom response was not quantitatively reported in the extracted data	Children with CR had markedly higher adenoid biofilm coverage than children with OSA; recurrence was linked to allergic status
Orabi et al., 2022	United States	107 children (N/R); 27 chronic adenoiditis, 42 CRS, 38 controls; age range 2–12 years	Inclusion criteria: Children aged 2–12 years with chronic sinonasal symptoms for ≥12 weeks who underwent CT imaging; chronic adenoiditis defined as CT Lund–Mackay score <5; CRS defined as CT Lund–Mackay score ≥5; controls had non-sinonasal conditions. Exclusion criteria: Cystic fibrosis, primary ciliary dyskinesia, complicated acute sinusitis, immune deficiency disorders, prior sinonasal surgery including adenoidectomy or FESS.	Medical treatment response was not directly evaluated	No postoperative adenoidectomy outcome was measured in this study	SN-5 differentiated CRS from chronic adenoiditis: CRS 4.53 ± 0.77, chronic adenoiditis 3.49 ± 1.00, controls 2.03 ± 0.71, p < 0.0001
Kais et al., 2024	United States	107 children (41.1%); mean age 4.88 ± 2.54 years	Inclusion criteria: Children meeting pediatric CRS criteria who underwent adenoidectomy after failed maximal medical therapy; pediatric CRS defined by ≥2 symptoms for ≥12 weeks plus endoscopic or CT evidence of sinonasal inflammation. Exclusion criteria: ESS as initial surgical intervention before adenoidectomy; cystic fibrosis, primary ciliary dyskinesia, complicated acute sinusitis, immune deficiency disorders.	Included only children with pediatric CRS after failed maximal medical therapy	Adenoidectomy success was defined as resolution of ≥50% of cardinal pediatric CRS symptoms; the success rate was 67.9% in children aged ≥6 years and 39.2% in children <6 years; successful patients were older and had lower Lund-Mackay CT scores in univariate analysis	Adenoidectomy was supported as a first-line surgical treatment for children ≤12 years, especially those aged 6–12 years, but higher CT burden may predict lower response

CRS, chronic rhinosinusitis; N/R, not reported; OSA, obstructive sleep apnea.

Study Quality

Based on the evaluation using the JBI Critical Appraisal Tools (Table 3), all included studies were judged to have moderate concern, with one study with an overall judgement of low-to-moderate concern (17) and one study with high concern (8).

Table 3. JBI critical appraisal

Study	Domain											Overall judgment
	D1	D2	D3	D4	D5	D6	D7	D8	D9	D10	D11	
JBI critical appraisal for analytical cross-sectional and comparative observational study (A)												
Coticchia 2007	Y	Y	Y	Y	N	N	Y	Y				High concern
Berçin 2007	Y	Y	Y	Y	N	N	Y	Y				Moderate concern
Shin 2008	Y	Y	Y	Y	U	N	Y	Y				Moderate concern
Ramadan & Ashry 2021	Y	Y	Y	Y	U	N	Y	Y				Moderate concern
Bugari 2021	Y	Y	Y	Y	U	U	Y	Y				Moderate concern
JBI critical appraisal for case-control study (B)												
Qu et al. 2015	Y	Y	Y	Y	Y	Y	Y	Y	U	Y		Moderate concern
JBI critical appraisal for quasi-experimental study (C)												
Bettadahalli & Chakra varti 2017	Y	NA	Y	N	Y	Y	Y	Y	Y			Moderate concern
JBI critical appraisal for cohort study (D)												
Orabi et al., 2022	Y	Y	Y	Y	U	NA	Y	NA	NA	NA	Y	Moderate concern
Kais et al., 2024	Y	Y	Y	Y	Y	Y	Y	Y	U	U	Y	Low-to-moderate concern

A = D1: Inclusion criteria clearly defined; D2: Study subject and setting described; D3: Exposure measured validly and reliably; D4: Objective criteria used for outcome/condition measurement; D5: Confounding factors identified; D6: Strategies used to deal with confounding; D7: Outcomes measured validly and reliably; D8: Appropriate statistical analysis used.
B = D1: Groups comparable other than presence of disease; D2: Cases and controls appropriately matched or selected; D3: Same criteria used for identification of cases and controls; D4: Exposure measured in a standard, valid, and reliable way; D5: Exposure measured in the same way for cases and controls; D6: Confounding factors identified; D7: Strategies used to deal with confounding; D8: Outcomes assessed in a standard, valid, and reliable way; D9: Exposure period sufficient to be meaningful; D10: Appropriate statistical analysis used.
C = D1: Clear cause-and-effect relationship stated; D2: Participants included in comparisons were similar; D3: Participants received similar care apart from intervention; D4: Control group included; D5: Multiple measurements before and after intervention; D6: Follow-up complete or adequately described; D7: Outcomes measured in the same way before and after intervention; D8: Outcomes measured reliably; D9: Appropriate statistical analysis used.
D = D1: Groups similar and recruited from the same population; D2: Exposures measured similarly across groups; D3: Exposure measured validly and reliably; D4: Confounding factors identified; D5: Strategies used to deal with confounding; D6: Participants free of outcome at start of study; D7: Outcomes measured validly and reliably; D8: Follow-up time sufficient; D9: Follow-up complete or losses described; D10: Strategies used to address incomplete follow-up; D11: Appropriate statistical analysis used.
Y, yes; N, no; U, unclear; NA, not applicable.

Discussion

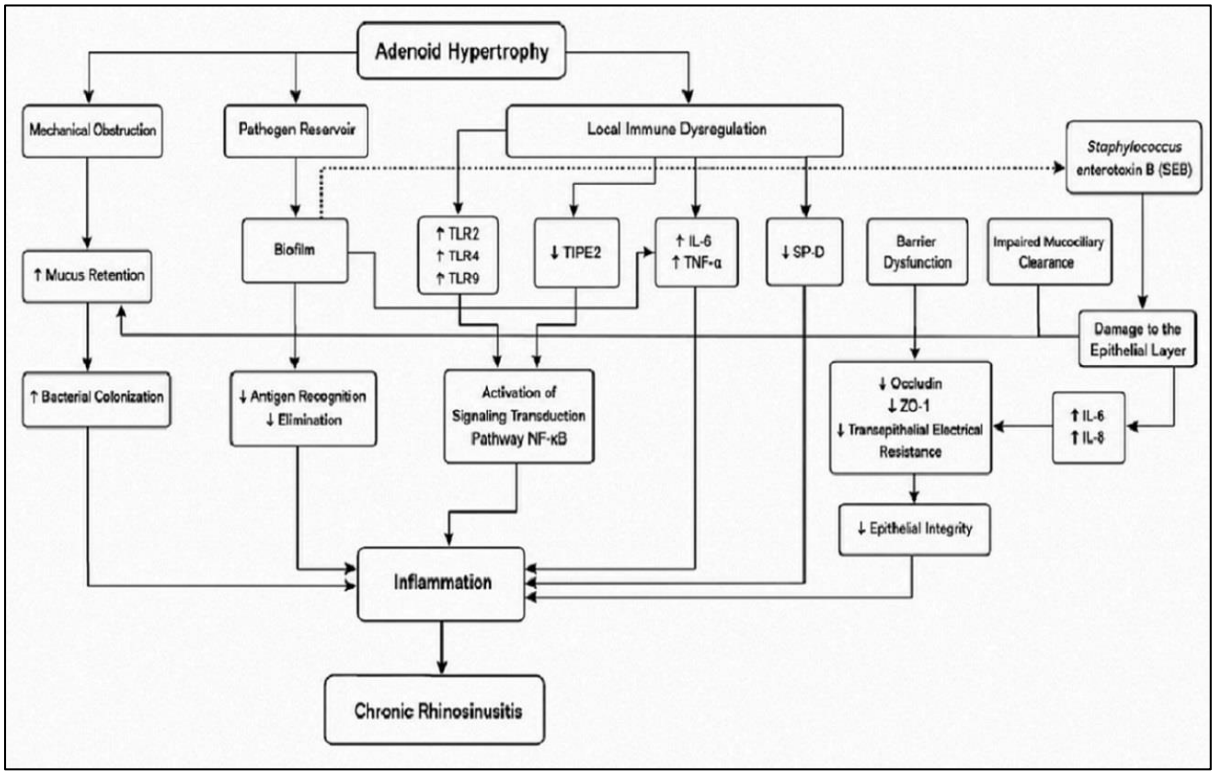

Fig 2. Mechanistic framework linking adenoid hypertrophy/chronic adenoiditis with pediatric chronic rhinosinusitis.

Mechanical Obstruction and Impaired Drainage

Several included studies suggest that adenoid hypertrophy may contribute mechanically to pediatric CRS by narrowing the posterior nasal airway and obstructing the nasopharynx, thereby impairing nasal airflow, reducing sinus ventilation, and disturbing sinonasal drainage (Figure 2). This mechanism is particularly relevant in younger children, in whom adenoid tissue is physiologically more prominent and may become clinically obstructive when hypertrophied. Ramadan and Ashry reported that positive CT findings of maxillary rhinosinusitis were present in 55% of children with adenoid hypertrophy and were significantly associated with endoscopic adenoid grade, supporting the hypothesis that higher-grade adenoid obstruction may promote secretion stasis and radiological sinonasal inflammation in selected patients (19). However, this association was not consistent across all studies. Berçin et al. found no significant relationship between adenoid volume and Lund–Mackay CT score, while Shin et al. reported no statistically significant association between sinusitis grade and adenoid size, although bacterial isolation from adenoid tissue increased significantly with sinusitis grade (18,20).

Overall, these findings indicate that mechanical obstruction may contribute to pediatric CRS in some children, particularly those with more severe adenoid hypertrophy, but adenoid size alone does not consistently predict the presence or severity of sinus disease. This suggests that inflammatory and microbiological properties of the adenoid may be at least as important as mechanical obstruction alone.

Adenoid as a Microbial Reservoir

Microbiological studies support the role of the adenoid as a reservoir for pathogenic bacteria in pediatric CRS (Figure 2). Shin et al. evaluated adenoid bacteriology in 410 children undergoing adenoidectomy and reported an overall bacterial isolation rate of 79.3%, with the most common isolates being Haemophilus influenzae (28.5%), Streptococcus pneumoniae (21.7%), Streptococcus pyogenes (21.0%), and Staphylococcus aureus (15.6%). Bacterial isolation increased significantly with sinusitis grade, particularly for H. influenzae and S. pneumoniae, whereas adenoid size was not significantly associated with sinusitis grade or bacterial isolation rate (18). Similarly, Ramadan and Ashry reported bacterial growth in all adenoid tissue specimens, including H. influenzae, S. pneumoniae, S. pyogenes, S. aureus, and MRSA, with 80.0% showing single-organism growth and 20.0% showing multiple-organism growth (19).

Taken together, these findings suggest that adenoid tissue harbors pathogenic bacteria, whose bacterial burden, unlike adenoid size, was consistently correlated with sinusitis severity, suggesting that colonization status may be mechanistically more relevant than mechanical obstruction alone. However, it should be noted that this conclusion rests on only two studies, both of which measured bacterial colonization through tissue culture rather than molecular methods; the evidence should therefore be regarded as suggestive rather than conclusive, and is further limited by variability in sampling techniques, culture-based versus molecular methods, prior antibiotic exposure, and differences in definitions of pathogenic colonization versus commensal carriage.

Adenoid Biofilm Formation

Biofilm formation was one of the most consistently supported mechanisms among the included studies and provides a biologically plausible explanation for persistent infection and treatment resistance in pediatric CRS (Figure 2). Bacterial biofilms consist of structured microbial communities embedded within an extracellular matrix, enabling microorganisms to adhere to mucosal surfaces and evade antimicrobial therapy and host immune responses. Coticchia et al. used scanning electron microscopy to compare adenoid tissue from children with CRS and children with obstructive sleep apnea (OSA), reporting dense mature biofilms covering a mean of 94.9% of the mucosal surface in the CRS group compared with only 1.9% in the OSA group ($p < 0.001$) (8).

Similarly, Bugari et al. found substantially greater biofilm coverage on adenoid tissue from children with CRS than from children with OSA, with mean coverage of 81.27% versus 3.871%, respectively ($p<0.001$). Histopathological and immunohistochemical findings also demonstrated epithelial erosion,

squamous metaplasia, lymphoid follicle hypertrophy, inflammatory infiltration, and immune-cell involvement in adenoid tissue (9).

These findings suggest that adenoidal biofilm is strongly associated with pediatric CRS and may contribute to recurrent symptoms, reduced response to conservative therapy, and persistent sinonasal inflammation.

The biofilm mechanism also provides a rationale for symptom improvement after adenoidectomy in selected medically refractory cases, as removal of biofilm-bearing adenoid tissue may reduce microbial burden and decrease repeated exposure of the sinonasal mucosa to pathogens and inflammatory stimuli. However, while consistent, these findings should still be considered preliminary pending confirmation in larger and more diverse cohorts.

Local Immune Dysregulation

Studies evaluating immune-related markers suggest that adenoid hypertrophy and chronic adenoiditis may contribute to pediatric CRS through altered local immune responses rather than obstruction alone (Figure 2).

The adenoid epithelium participates in innate mucosal defense through mucociliary clearance, antimicrobial molecule secretion, antigen recognition, and interaction with adaptive immune cells; however, these functions may become dysregulated in diseased adenoid tissue. Qu et al. evaluated innate immune gene expression in isolated adenoidal epithelial cells from children with adenoid hypertrophy with and without pediatric CRS and found significantly lower surfactant protein-D (SP-D) expression in the pCRS group compared with the adenoid hypertrophy-only group (0.73 ± 0.10 vs. 1.21 ± 0.15; $p = 0.0173$). In contrast, no significant differences were observed for HBD-2, HBD-3, SP-A, TLR1–10, NOD1, NOD2, NALP3, RIG-1, MDA-5, or NF-κB p65 (11). These findings suggest that pediatric CRS associated with adenoid hypertrophy may involve selective impairment of adenoidal epithelial innate defenses, particularly through reduced SP-D expression, rather than broad dysregulation of pattern-recognition receptor pathways. However, because only one included study directly evaluated these molecular markers and immune-marker findings remain methodologically heterogeneous, the evidence for immune dysregulation should be considered preliminary.

Epithelial Barrier Dysfunction and Impaired Mucociliary Clearance

A smaller group of studies addressed epithelial barrier dysfunction and impaired mucociliary clearance as potential mechanisms in pediatric CRS (Figure 2). Disruption of epithelial integrity may increase mucosal permeability to pathogens, allergens, and environmental irritants, thereby amplifying inflammatory responses, while impaired mucociliary clearance may reduce the ability of the nasal and sinus mucosa to remove mucus, debris, and microorganisms. These mechanisms are biologically plausible because pediatric CRS is characterized by persistent inflammation and secretion stasis, which may be aggravated by adenoid hypertrophy and chronic adenoiditis through obstruction of airflow and drainage, altered epithelial function, and sustained inflammatory exposure within the nasopharyngeal region. Bugari et al. provided histopathological evidence of epithelial erosion, squamous metaplasia, lymphoid follicle hypertrophy, and inflammatory infiltration in adenoid tissue from children with CRS, supporting the presence of chronic epithelial and inflammatory remodeling (9). However, direct evidence for adenoid-specific epithelial barrier dysfunction and mucociliary impairment remains limited, as only a single study addressed these mechanisms, and none of the included primary studies directly measured tight-junction proteins such as ZO-1 or occludin, transepithelial electrical resistance, ciliary beat frequency, or mucociliary clearance time.

Therapeutic Implications of Adenoidectomy

Clinical outcome studies suggest that adenoidectomy may improve symptoms in selected children with medically refractory pediatric CRS. Its therapeutic effect may be explained by several mechanisms, including relief of nasopharyngeal obstruction, restoration of airflow and sinonasal drainage, reduction of pathogenic bacterial reservoirs, removal of biofilm-bearing tissue, and decreased chronic inflammatory stimulation. Bettadahalli and Chakravarti reported significant postoperative improvement after adenoidectomy or adenotonsillectomy, with the total symptom severity score decreasing from

36.73 ± 7.70 preoperatively to 16.17 ± 8.61 postoperatively (p = 0.000). The overall SN-5 score also improved from 4.59 ± 0.98 to 1.55 ± 1.06, and clinically meaningful quality-of-life improvement was observed in 53 of 60 children (88.3%) (15). Kais et al. evaluated 107 children with pediatric CRS following failure of maximal medical therapy and found that adenoidectomy success was associated with older age and lower Lund–Mackay CT scores. Successful outcomes were reported in 67.9% of children aged 6 years and older compared with 39.2% of children younger than 6 years, and children with successful outcomes had lower CT scores than those with failed surgery (6.93 ± 3.81 vs. 8.63 ± 3.48; p = 0.045). Among children with failure of adenoidectomy, 12 required subsequent endoscopic sinus surgery (17). Although Orabi et al. did not evaluate postoperative outcomes, their study showed that SN-5 scores could differentiate CRS from chronic adenoiditis, with higher mean scores in CRS than in chronic adenoiditis and controls (4.53 ± 0.77 vs. 3.49 ± 1.00 vs. 2.03 ± 0.71; $p <$ 0.0001), supporting the clinical utility of validated symptom tools in distinguishing overlapping pediatric upper airway conditions (16). Overall, adenoidectomy should be understood as an effective surgical option for selected children with refractory pediatric CRS, but not as a universal definitive treatment. Children with allergic rhinitis, asthma, immunodeficiency, primary ciliary dyskinesia, severe sinus disease, or other comorbidities may have persistent symptoms and require additional medical therapy or functional endoscopic sinus surgery.

Certainty of Evidence *and Directions for* Future Research

Not all mechanistic domains discussed above rest on equally solid ground. The biofilm hypothesis is the most consistently supported mechanism in this review, converging across independent studies using different sampling and imaging approaches and offering a coherent rationale for symptom improvement after adenoidectomy, which warrants moderate certainty. The microbial reservoir hypothesis is similarly compelling—bacterial burden correlated with sinusitis severity in studies where adenoid size did not; however, this evidence is tempered by variability in sampling technique, culture-based versus molecular detection methods, prior antibiotic exposure, and inconsistent definitions of pathogenic colonization versus commensal carriage, which together justify only low-to-moderate certainty. The obstruction hypothesis is weak, as adenoid size did not consistently predict CRS severity, although the relationship may still hold in children with more severe hypertrophy, suggesting that the adenoid's inflammatory and microbiological properties may matter as much as, or more than, its mechanical bulk; certainty for obstruction as an independent predictor is accordingly low. Immune dysregulation rests on a single molecular study and should be treated as a plausible lead rather than established evidence, warranting very low certainty. The same caution applies to epithelial barrier dysfunction and impaired mucociliary clearance, neither of which was measured directly in any included study; what is presented as evidence for these pathways is an inference from histopathology, not functional data; therefore, it again warrants very low certainty. The clinical benefit of adenoidectomy is comparatively well replicated across three observational studies, but outcomes varied by patient selection, and because none of these studies were randomized, how much of the observed benefit reflects the mechanisms proposed here rather than natural history or a nonspecific surgical effect remains uncertain, supporting only low-to-moderate certainty.

This pattern of evidence, therefore, points to several priorities for future research. Reservoir studies need standardized sampling protocols, molecular rather than culture-dependent detection, and a consistent operational definition separating pathogenic colonization from commensal carriage, since these inconsistencies likely account for much of the heterogeneity seen across studies. Obstruction studies need standardized, objective measurement of adenoid size or grade alongside CRS severity, ideally with stratified analysis by hypertrophy severity, to evaluate whether the relationship is genuinely absent or simply obscured by measurement variability. Finally, the immune dysregulation findings require replication in larger, independent cohorts before they can be considered anything more than hypothesis-generating, and epithelial and mucociliary function should be assessed

directly rather than inferred from tissue morphology.

Limitations

This review has several limitations that should temper the strength of the conclusions drawn. First, the review protocol was not prospectively registered (e.g., in PROSPERO), which precludes independent verification that the eligibility criteria, search strategy, and planned synthesis were specified in advance and raises the possibility of post hoc refinement of the review methodology. Second, only nine studies met the eligibility criteria with all were observational studies, which limits causal inference regarding the mechanistic role of adenoid disease in pediatric CRS.

Third, substantial heterogeneity in diagnostic criteria for CRS, methods of adenoid assessment, sampling techniques, and reported outcomes precluded meta-analysis and necessitated a narrative synthesis, which is inherently more susceptible to bias in interpreting the findings.

Fourth, quality appraisal using the JBI Critical Appraisal Tools indicated that most included studies raised moderate methodological concern, with one study rated as high concern and only one as low-to-moderate concern, further limiting confidence in the reported associations. Finally, the included studies were conducted across heterogeneous populations and clinical settings, and small sample sizes in several studies further constrain the generalizability of findings. Given these limitations, the mechanistic pathways discussed in this review should be regarded as hypothesis-generating rather than definitive, and the conclusions warrant cautious interpretation pending confirmation from larger, prospective, and mechanistically focused studies.

Conclusion

The available evidence is consistent with an association between adenoid hypertrophy, chronic adenoiditis, and pediatric chronic rhinosinusitis, and points to several biologically plausible mechanisms that may underlie this association. Mechanical obstruction may be associated with impaired nasopharyngeal airflow and sinonasal drainage, while chronic adenoiditis may be linked to sustained local inflammation. Adenoid tissue also appears to be associated with a microbial reservoir and biofilm-bearing structure, allowing persistent infection despite medical therapy. In parallel, immune dysregulation, epithelial barrier dysfunction, and impaired mucociliary clearance may perpetuate chronic sinonasal inflammation. Taken together, the findings indicate that adenoid disease should not be viewed solely as an anatomical obstruction. Instead, the available evidence points toward multiple interacting obstructive, infectious, inflammatory, and immunological factors that may jointly characterize the relationship between adenoid disease and pediatric CRS. Because all included studies were observational, these findings should be interpreted as hypothesis-generating associations rather than established causal pathways, and confirmation through prospective mechanistic studies is warranted.

Acknowledgements

The authors would like to acknowledge that no specific funding or external assistance was received for this study.

Conflicts of interest

None declared.

Funding

None.

Authors' contributions

Conceptualization: GRAP; Data curation: GRAP; Investigation: GRAP, FT; Methodology: GRAP, FT; Project administration: GRAP; Supervision: FT; Validation: FT; Visualization: GRAP; Writing – original draft: GRAP, FT; Writing – review & editing: GRAP, FT.

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