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Original Article

Nasal polyps in childhood: insights from a pediatric pulmonology cohort

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ABSTRACT

Introduction. Nasal polyps (NP) are benign inflammatory growths originating from the nasal/paranasal sinus mucosa. NPs can occur independently or signify underlying respiratory conditions. This study aimed to evaluate the demographic and clinical characteristics of NPs in children at a pediatric pulmonology department.

Method. This cross-sectional study included children aged 0-18 years diagnosed with NP at the pediatric pulmonology department from 2007 to 2025. Patients were categorized into four groups: cystic fibrosis (CF), primary ciliary dyskinesia (PCD), asthma/allergic rhinitis (AR), and undefined etiology. Demographic and clinical data were compared across these groups.

Results. A total of 47 (40.4% female) patients were included in the study. The median age of NP diagnosis was 11 years (4.6-17.8). The most common presenting symptoms were nasal obstruction (47, 100%) and mouth breathing during sleep (37, 78.7%). The etiological causes of NPs were PCD (13, 27.7%), CF (9, 19.1%), asthma/AR (11, 23.4%), and an undefined etiology (14, 29.8%). Bilateral NPs were present in 32 (68.1%) cases. In 27 (57.4%) patients, NP was the first presentation, with no other symptoms. Among all PCD patients, NP was the first presentation in 7 (53.8%) of cases. NP recurrence was documented in 14 patients (29.8%). No significant differences were observed among the groups in sex, growth z-scores, or age at NP diagnosis ($p>0.05$). Bilateral NPs were more common in the PCD group (92.3%) than in the CF (66.7%), asthma/AR (36.3%), and undefined groups (71.4%) ($p=0.034$). NP recurrence was more frequent in the CF group (66.7%) compared with the PCD (38.5%) and undefined groups (13.6%), while no recurrence was observed in the asthma/AR group ($p=0.021$).

Conclusion. This study highlights the heterogeneity of pediatric NP in causes, presentation, and course. Patients with PCD often present bilaterally, while CF patients have the highest recurrence rate. In PCD, NP was the initial sign in over half of cases, suggesting isolated NP should prompt suspicion of PCD.

Key words: nasal polyp, cystic fibrosis, primary ciliary dyskinesia, asthma, allergic rhinitis.

Nasal polyps (NP) originate from any part of the mucosa of the nose or paranasal sinuses. NPs are benign inflammatory growths that range in color from grayish-pink to yellow. They may appear as solitary or multiple masses and are usually easily movable during examination. NPs can occur as an isolated condition, but they may also be a sign of various underlying respiratory disorders. These include cystic fibrosis (CF), primary ciliary dyskinesia (PCD), asthma, allergic rhinitis (AR), and immunodeficiency, among others. Moreover, in

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some cases, NP may be the first manifestation of a severe underlying disease. When identified, clinicians should raise suspicion for potential underlying conditions.[1,2]

NPs are relatively rare in the general pediatric population, with an estimated incidence of approximately 0.1%.[1] However, their prevalence is higher with certain chronic respiratory diseases. The literature reports that the incidence of NPs in children with CF varies from 6% to 48%.[2,3] Similarly, studies report a prevalence ranging between 15%-30% in patients with PCD, likely due to impaired mucociliary clearance leading to persistent sinonasal congestion and infection.[4] In pediatric asthma populations, NPs are less frequent but still noteworthy, with reported rates around 7%, particularly in those with coexisting AR. In addition to causing persistent nasal obstruction, NPs can impair children's quality of life by disrupting breathing, sleep, and daily activities. They are linked to chronic upper airway colonization, serving as a reservoir for pathogens. This colonization may increase pulmonary exacerbations in children with chronic respiratory conditions like CF and PCD.[2-7]

This study aimed to evaluate the demographic and clinical features of NPs in children who were followed up at a pediatric pulmonology department, and to examine their distribution across primary underlying conditions, thereby improving the understanding of disease patterns.

Methods

This was a cross-sectional study. All procedures involving human participants in the study adhered to the ethical standards established by the institutional research committee (Gazi University Ethics Committee, date: September 24, 2024, meeting: 15, reference number: 2024-1502), as well as the Declaration of Helsinki and its subsequent amendments or similar ethical guidelines. All children aged 0 to 18 years who were followed up with a diagnosis of NP at the pediatric pulmonology department between 2007 and 2025 were included in the study. The study population consisted of patients referred to the pediatric pulmonology department because NP was identified at their initial visit, as well as those who developed NP during follow-up for underlying conditions such as CF or PCD. Only patients with a confirmed NP diagnosis and complete medical records were included, while those with an uncertain diagnosis or missing data were excluded.

The medical data of the patients were retrospectively collected from their medical records. Demographic and clinical information, including sex, symptoms, growth parameter z-scores, etiological causes of NP, history of polypectomy, and age at polypectomy, were recorded, along with whether patients had pulmonary function test (PFT) and home sleep study (HSS) results documented.

Pulmonary function tests were performed according to the American Thoracic Society/ European Respiratory Society (ATS/ERS) guidelines, using acceptable criteria for children. Measurements included forced expiratory volume in the first second (FEV1), forced vital capacity (FVC), peak expiratory flow (PEF), and forced expiratory flow from 25% to 75% (FEF25–75%), all recorded as percentages. Additionally, the FEV1/FVC ratio was documented and assessed based on age, sex, and height. PFTs are generally conducted for individuals aged six and older.[8] PFT values obtained at the time of NP diagnosis were recorded.

A home sleep study (Type 3 polygraphy, ApneaLink(') was used to diagnose sleep-disordered breathing (SDB) when in-laboratory polysomnography is not available. It was performed when obstructive sleep apnea (OSA) was suspected in patients who reported nighttime symptoms such as snoring or witnessed apneas. During the study period, access to HSS testing was sometimes restricted or temporarily unavailable.[9] The apnea-hypopnea index (AHI) was defined as follows: AHI < 1 is normal, 1 ≤ AHI < 5 is classified as

mild OSA, 5 ≤ AHI < 10 is classified as moderate OSA, and AHI ≥ 10 is classified as severe OSA. The term "apnea" refers to parent-reported or witnessed breathing pauses during sleep.

Medical records of patients who had NP were evaluated, and their clinical and diagnostic findings were recorded. The assessment included a detailed medical history, physical examination, PFT, allergy testing (including serum IgE levels and skin prick tests), sweat chloride testing when clinically indicated, and, when clinically indicated, high-speed video microscopy (HSVM) and/or genetic testing. CF diagnosis was established according to the European Cystic Fibrosis Society guidelines. Sweat testing was performed using the chloride titration method, with sweat chloride values >59 mmol/L considered diagnostic for CF, values <30 mmol/L considered normal, and values between 30 and 59 mmol/L requiring repeat testing. Accordingly, CF was diagnosed based on either two sweat chloride measurements >59 mmol/L, or one sweat chloride measurement >59 mmol/L together with the identification of two CF-causing mutations by DNA analysis, or the identification of two CF-causing mutations in the presence of typical clinical features of CF.[10]

Diagnosis of PCD was based on a compatible clinical presentation, characteristic radiological findings, HSVM results showing abnormal ciliary beat patterns consistent with PCD (including stiff-beating and immotile cilia patterns), and/or genetic testing in accordance with ERS/ATS diagnostic guidelines.[11] Among genotyped patients, for patients carrying apparently heterozygous variants, parental segregation analysis was performed, demonstrating compound heterozygosity, with each parent carrying one variant.

Asthma diagnosis was determined through pediatric allergy consultation, clinical history, spirometry, and adherence to the Global Initiative for Asthma guidelines.[12] The diagnosis of AR was established based on clinical symptoms, physical examination, and a positive skin prick test and/or serum-specific IgE results.[13]

Patients were divided into four diagnostic subgroups based on etiology: CF, PCD, asthma/AR, and undefined etiology. Demographic and clinical data were compared across groups.

Diagnosis of NP

The diagnosis of NP was made by otolaryngologists using one or more of these methods: direct inspection, anterior rhinoscopic examination, nasal endoscopy, or sinonasal imaging, including paranasal sinus computed tomography or magnetic resonance imaging. NP was confirmed when polypoid lesions were found in the nasal or paranasal cavity with any of these techniques.

Definition of NP regression

Regression of NPs was defined as a ≥25% reduction in polyp size or complete resolution, as documented by nasal endoscopic and clinical examination findings following medical and/or surgical treatment. Regression was determined by the attending physician's assessment, based on a visible reduction in obstruction or the disappearance of polyps.[14,15]

Definition of recurrent NP

Recurrent NP were defined as the reappearance of polypoid lesions after prior medical and/or surgical treatment, confirmed by clinical examination, endoscopic evaluation, and/or sinonasal imaging.

Treatment of NP

At our institution, children with NP are initially treated with intranasal corticosteroids and saline irrigation. Systemic corticosteroids are rarely used and are reserved for selected cases with severe obstruction or inadequate response to topical therapy. Surgical intervention is considered for patients who do not respond to medical treatment or who experience

recurrence, taking into account the underlying primary disease. The final decision to proceed with surgery is made after evaluation by an otolaryngologist, with the indication mainly based on the severity of nasal obstruction and symptom burden.

Medical treatment consisted mainly of intranasal corticosteroid sprays (such as mometasone furoate, triamcinolone acetonide, and fluticasone furoate) in combination with regular nasal saline irrigation. Intranasal corticosteroids were administered once or twice daily for at least one month (≥4 weeks) or longer depending on clinical response. Antihistamines and short courses of oral corticosteroids were used only in selected cases, taking into account the underlying disease.

Statistical analysis

IBM SPSS Statistics version 22.0 (IBM, Armonk, NY, USA) was used for the statistical analyses. In the descriptive statistics section, categorical variables are presented as counts and percentages, while continuous variables are shown with means ± standard deviations, and medians (minimum–maximum values). The Pearson chi-square test and Fisher's exact test were used to evaluate categorical variables. The Mann–Whitney U-test was employed for comparing two independent variables in data that did not meet normal distribution assumptions, and the independent sample t-test was used for data that followed a normal distribution. When comparing three or more variables, one-way analysis of variance (ANOVA) was performed under conditions suitable for parametric tests, and the Kruskal–Wallis H test was used when parametric conditions were not met. The relationship between non-normally distributed data was assessed using Spearman's correlation test, while Pearson's correlation test was used for data that conformed to a normal distribution. P-values less than 0.05 were considered statistically significant.

The required sample size was calculated using G*Power 3.1.9.7. Assuming a large effect size (Cohen's w = 0.50) for the chi-square test comparing NP rates across four etiological groups, with a power of 80% and a two-sided alpha of 0.05, a minimum of 44 patients was estimated. Since this retrospective study included all eligible cases (n= 47), the required sample size was achieved.

Results

The demographic and clinical data for all patients, along with comparisons between groups, are shown in Table I. A total of 47 patients were included in the study. Nineteen (40.4%) were female and 28 (59.6%) were male. The median age at NP diagnosis was 11 (4.6-17.8) years. The median weight z-score was -0.41 (-2.33- 4.87), height z-score was -0.32 (-2.69-9.62), and BMI z-score was 0.0 (-3.93-2.49) at the time of diagnosis of NP. The most common presenting symptoms were nasal obstruction 47 (100%), mouth breathing during sleep 37 (78.7%), and snoring 25 (53.2%). NPs were unilateral in 15 (31.9%) cases and bilateral in 32 (68.1%) cases.

In 27 (57.4%) patients, NP were the first presentation, with no other symptoms. Among these patients, one had a diagnosis of CF, seven had a diagnosis of PCD, one had a diagnosis of asthma, and four had a diagnosis of AR. Among the 13 confirmed PCD patients included in the study with NP, NP were the first presenting feature in 7 (53.8%) of them.

Intranasal corticosteroids combined with nasal saline irrigation were the most commonly used medical treatments (40 patients, 85.1%). Antihistamines were prescribed in 4 (8.5%) and short courses of oral corticosteroids were prescribed in 2 (4.2%) patients, respectively.

Thirty (63.8%) patients had a history of polypectomy, with a mean age at surgery of 12.7 years (range: 5–25 years). Regression of NP was

Table I. Demographic and clinical data of the CF, PCD, and Asthma-AR and Unknown etiology groups .

	CF n (%) (Total: 9 patients)	PCD n (%) (Total: 13 patients)	Asthma/AR n (%) (Total: 11 patients)	Unknown etiology n (%) (Total: 14 patients)	p
The age of diagnosis of NP (year) ‡	10.8 (5.8-17.8)	11.9 (5.1-15.6)	10 (5.1-16)	12.6 (4.6-17.4)	0.659
Female	4 (44.4)	9 (69.3)	2 (18.2)	4 (28.6)	0.055
Male	5 (55.5)	4 (30.7)	9 (81.8)	10 (71.4)	
Weight z-score ‡	-0.66 (-1.80-4.74)	-0.49 (-2.33-2.18)	0.82 (-1.81-4.87)	-0.28 (-1.64-2.42)	0.367
Height z-score ‡	0.13 (-1.47-7.0)	-0.40 (-1.37-2.31)	-0.49 (-1.84-9.62)	-0.30 (-2.69-1.96)	0.915
BMI z-score ‡	-0.4 (-3.93-1.91)	-0.39 (-2.25-1.44)	0.39 (-1.58-2.01)	-0.11(-2.31-2.49)	0.117
Symptoms					
Nasal obstruction	9 (100)	13 (100)	11 (100)	14 (100)	-
Mouth breathing during sleep	7 (77.8)	12 (92.3)	9 (81.8)	9 (64.2)	-
Snoring	3 (33.3)	6 (46.2)	6 (54.5)	10 (71.4)	-
**Apnea*	2 (22.2)	-	1 (9.1)	1 (7.1)	-
Morning headache	1 (11.1)	1 (7.6)	1 (9.1)	1 (7.1)	-
Daytime sleepiness	-	1 (7.6)	1 (9.1)	2 (14.2)	-
Spirometry	n=8	n=12	n= 4	n=6	
FEV1% ‡	91 (58-128)	94 (48-114)	96 (78-135)	103 (71-127)	0.361
FVC% ‡	90 (58-124)	90 (52-116)	97 (79-122)	105 (65-112)	0.667
FEV1/FCV ‡	103 (62-110)	99 (75-108)	106 (84-116)	103 (86-116)	0.574
FEF_{25-75}% ‡	87 (24-133)	90 (37-115)	110 (47-160)	84 (60-143)	0.591
HSS	n=3	n=1	n=2	n=1	
Mild OSA	2 (22.2)	1 (7.7)	2 (18.2)	-	
Moderate OSA	-	-	-	1 (7.1)	-
Severe OSA	1 (11.1)	-	-	-	
NP presentation					
Unilateral	3 (33.3)	1 (7.7)	7 (63.7)	4 (28.6)	0.034
Bilateral	6 (66.7)	12 (92.3)	4 (36.3)	10 (71.4)	
Presenting with NP as the first manifestation	1 (11.1)	7 (53.8)	5 (45.5)	14 (100)	-
Medical treatments					
Intranasal steroids& nasal lavage	6 (66.6)	12 (92.3)	9 (81.8)	13 (92.8)	-
Antihistamines	1 (11.1)	1 (7.7)	1 (9.1)	1 (7.1)	
Oral steroid	-	2 (15.4)	-	-	
History of polypectomy	6 (66.7)	10 (76.9)	5 (45.5)	9 (64.3)	0.442
The age of polypectomy (year) ‡	11.5 (9.6-25)	13.2 (7.2-20.6)	11 (6-18.5)	13 (5-21.3)	0.988
Regression of NP	3 (33.3)	8 (61.5)	11 (100)	11 (78.5)	0.021
Recurrence of NP	6 (66.7)	5 (38.5)	-	3 (13.6)	0.021
Follow-up duration (months)	148 (21–201)	31 (1–146)	4.5 (1–76)	1 (1–15)	<0.001

AR: Allergic Rhinitis, BMI: Body mass index, CF: Cystic Fibrosis, HSS: Home Sleep Study, NP: Nasal Polyp, OSA: Obstructive Sleep Apnea, PCD: Primary Ciliary Dyskinesia

‡:[median (min-max)]

* The term "apnea" indicates parent-reported or witnessed breathing pauses during sleep.

observed in 33 (70.2%) patients. NP recurrence was documented in 14 patients (29.8%).

Spirometry data were available for 30 patients. The median FEV1 was 95% (range: 48–135%), FVC was 91% (52–124%), the FEV1/FVC ratio was 101% (62–116%), and the mean FEF_{25-75} was 91% (24–160%).

Aspirin provocation testing was performed in two patients suspected of NSAID hypersensitivity, and neither met the criteria for aspirin-exacerbated respiratory disease. The other children with NP had no history of suspected NSAID hypersensitivity. HSS was performed in seven patients, all of whom were diagnosed with OSA; five had mild OSA, one had moderate OSA, and one had severe OSA.

Among the 47 patients, 33 (70.2%) had an identifiable underlying disease, including PCD (13, 27.7%), CF (9, 19.1%), asthma (5, 10.6%), and AR (6, 12.8%). Fourteen (29.8%) patients had no defined etiology. In our pediatric pulmonology clinic, 57 patients were followed up with a diagnosis of CF, and 24 with PCD. Among these, NPs were detected in 9 (15.8%) patients with CF and in 13 (54.2%) patients with PCD.

Demographic and clinical characteristics of patients with NP were compared across four etiological groups: CF (n = 9), PCD (n = 13), asthma/AR (n = 11), and unknown etiology (n = 14). No statistically significant differences were observed regarding sex (p = 0.055), current age (p = 0.345), weight z-score (p = 0.367), height z-score (p = 0.915), BMI z-score (p = 0.117), or age at NP diagnosis (p = 0.659) among the groups.

Regarding NP laterality, bilateral presentation was significantly more common in the PCD group (92.3%) than in the other groups (p = 0.034).

The history of polypectomy was reported in 66.7% of CF patients, 76.9% of PCD patients, 45.5% of asthma/AR patients, and 64.3% of patients with unknown etiology (p = 0.442), with no significant difference in the age at polypectomy across the groups (p = 0.988). Notably, NP recurrence was significantly more frequent in the CF group (66.7%) (p = 0.021).

Spirometry was available for subsets of each group (n=8 for CF, n=12 for PCD, n=4 for asthma/AR, and n=6 for the unknown etiology group). No significant differences were found in median values of FEV1, FVC, FEV1/FVC ratio, or FEF_{25-75} among the groups (respectively, p= 0.361, p= 0.667, p= 0.574, p= 0.591).

Among patients, the median follow-up durations were 148 (21–201) months for the CF group, 31 (1–146) months for the PCD group, 4.5 (1–76) months for the asthma/AR group, and 1 (1–15) months for the unknown etiology group (p < 0.001).

In the CF group, five CF patients received CFTR modulator therapy during the study. Four patients were treated with elexacaftor/tezacaftor/ivacaftor for a median of 11 months (range: 5–18 months), and one patient received on ivacaftor for 18 months. When considering all CFTR modulator–treated CF patients together, the median treatment duration was 17 months (range: 5–18 months). Three of them had a history of polypectomy and recurrence of NP.

Because of the retrospective design and the historical period of patient evaluation, genetic analysis was available for only 8 of the 13 patients diagnosed with PCD. Five patients with PCD were lost to follow-up. In the remaining five patients, the genetic analysis results were unavailable. The results of the genetic analysis for the CF and PCD patients are presented in Table II.

Discussion

This study is among the few pediatric pulmonology cohorts examining NPs across various etiologic groups rather than focusing on a single disease. Including children with CF, PCD, Asthma–AR, and unknown causes provides a broader clinical perspective. It emphasizes disease-specific patterns, such as

Table II. The genetic analysis reports of the CF and PCD patients.

CF	Patient 1. *CFTR*: 3199del6 /3199del6
	Patient 2. *CFTR*: R1070Q / G178R / S466X (TAA)
	Patient 3. *CFTR*: E831X / E831X
	Patient 4. *CFTR*: G85E / G85E
	Patient 5. *CFTR*: 1677delTA/ F508del
	Patient 6. *CFTR*: R1162X/ 2789+5G>A
	Patient 7. *CFTR*: F508del / F508del
	Patient 8. *CFTR*: R334W / N1303K
	Patient 9. *CFTR*: F508del / N1303K
PCD	Patient 1. *TTC12*: c.1480G>A, *DNAAF4*: c.791A>G, *DNAAF3*: c.1445C>A, *DNAH1*: c.7809C>T
	Patient 2. *KIF9*: c.1515-1G>A, c.1960G>A, *DNAH7:* c.16-6A>G / c.8945+652G>A, *DNAH1*: c.7759G>T, *DRC1*: c.26C>T
	Patient 3. *DNAJB13*: c.721-37C>G, c.607-314dup, *CFAP74*: c.2241+34G>A, c.3012-56G>A, *CCDC40*: c.2445A>G, *DNAL1*: c.306C>T
	Patient 4. *DNAAF1*: c.455G>A, *DNAI1*: c.1286G>A, c.7975A>G, c.11788-29G>A, *DNAH9:* c.2427C>T
	Patient 5. *DNAH5*: c.6037C>T / c.6037C>T
	Patient 6. *CCDC151*: c.1291C>T / c.1291C>T
	Patient 7. *DNAH5:* c.12596G>A / c.12596G>A
	Patient 8. *DNAH5:* c.5563dupA / c.2511dupC

CF: Cystic Fibrosis, PCD: Primary Ciliary Dyskinesia .

increased recurrence in CF and predominantly bilateral presentation in PCD. In PCD, NP was the initial presentation in over half of the cases, highlighting that isolated NP should prompt suspicion of an underlying PCD diagnosis. Underlying genetic mucociliary disorders predispose patients to more pronounced and recurrent NP.

Nasal polyps in children often are related to chronic sinonasal inflammation and may signal underlying genetic, allergic, or inflammatory conditions. In our group, PCD and CF were primary causes, seen in 27.7% and 19.1% of patients, respectively, with fewer cases of AR and asthma. These findings are consistent with evidence that impaired mucociliary clearance and ongoing inflammation contribute to NP.[16,17] Notably, 70.2% of patients had underlying diseases, emphasizing that NP often indicates broader systemic or respiratory issues. In some cases, NP may be the first sign of systemic diseases like CF or PCD, especially in children without initial pulmonary symptoms. In our cohort, 57.4% presented with NP first, and 13 were diagnosed with an underlying disease. However, 29.8% had no identifiable cause, highlighting diagnostic challenges. Therefore, children presenting with NPs should undergo a structured initial evaluation, including assessments for CF, PCD, and allergic airway diseases. Additional genetic, immunologic, or radiologic tests should be reserved for cases in which initial findings suggest an underlying systemic or chronic condition. This highlights the need for a multidisciplinary approach to NP in children for proper management and early detection of serious conditions. Long-term follow-up is recommended. Assessing children with isolated NP may help detect systemic diseases early. The high initial NP rate in our study may be due to increased awareness and routine evaluations by pulmonologists.

Nasal polyps are considered the final stage of chronic mucosal inflammation caused by

ongoing inflammatory exposure in the upper airways. The prevalence of NP has been shown to increase with age, with studies reporting higher occurrence in older children and adolescents.[18-20] Additionally, previous research has indicated an age range from early childhood to adolescence. Supporting this, our study also included a notably broad age spectrum. In our cohort, the age at diagnosis varied widely from 4.6 to 17.8 years, suggesting that, besides cumulative inflammatory burden, other factors such as genetic susceptibility, host response, and underlying comorbidities may also significantly influence the timing of presentation.

In CF, the reported prevalence of NP in pediatric groups varies widely across studies, ranging from 6% to 86%.[21,22] In our cohort, NP was present in 15.8% of children with CF, a rate lower than that reported in other studies. This variability between studies may be influenced by multiple factors, including genetic background, differences in study design, patient age distributions, diagnostic methods for NP, and clinical follow-up practices across centers. In PCD, the prevalence of NP has been reported to range from 15% to 30% across most studies.[4] In our cohort, NP was detected in 54.2% of patients with PCD, a rate notably higher than in most previous reports. This difference may be due to our tertiary referral setting, where patients generally have more advanced or symptomatic disease, as well as our systematic ENT assessments, which enable more comprehensive detection of NP throughout the disease course.

Previous studies have shown that highly effective CFTR modulator therapies are associated with decreased sinonasal symptoms, improved endoscopic and radiologic findings, and regression of NPs in some patients with CF. In our study, nine CF patients were included in the NP subgroup, of whom five received highly effective *CFTR* modulator therapy during follow-up. Among those patients, three had a history of nasal polypectomy and experienced NP recurrence. However, due to the small number of CF patients, the limited number receiving therapy, and differences in follow-up duration and therapy length, we could not perform a statistical analysis of the impact of CFTR modulators on NP regression or recurrence. As a result, our data do not establish a direct link between *CFTR* modulator therapy and the clinical progression of NP. Nonetheless, based on existing research, *CFTR* modulator therapies represent a significant advance in treating CF-related sinonasal disease, and larger, prospective studies are needed to better understand their effects on NP.[23]

Nasal obstruction is an independent contributor to SDB.[24] In our cohort, it was the most common symptom, present in all patients, followed by mouth breathing during sleep and snoring. These findings support the known effect of NP in increasing upper airway resistance, especially in children with smaller anatomies, making them more vulnerable to airflow restriction. Mouth breathing and snoring, often seen as harmless, are linked to SDB and can lead to poor sleep, neurocognitive issues, and fatigue. Less common symptoms include apnea, headache, and daytime sleepiness. Apnea in children with NP may indicate undiagnosed OSA. All seven patients assessed with HSS had OSA, emphasizing the role of NP in pediatric SDB. Chronic sinonasal inflammation may also contribute to headaches and fatigue through cytokines and systemic responses. This diversity of symptoms highlights the importance of a thorough clinical assessment in children with nasal obstruction or mouth breathing, as these may signal more serious upper airway conditions beyond simple rhinitis.

Differences in NP characteristics across disease groups reflect different mechanisms. Bilateral NP was more common in PCD, likely due to mucociliary impairment causing extensive sinonasal inflammation and bilateral NP formation. An international study of 345 PCD patients found NP in 14%, with 47% bilateral.[4] In our study, NP recurrence was most common in CF, likely due to persistent inflammation from thick secretions and infections, leading to ongoing sinonasal issues and regrowth after

surgery. The asthma/AR group showed the highest rate of unilateral NP and no recurrence, indicating a milder pattern. Although pediatric studies on AR-associated NP are limited, evidence from atopy-related NP suggests a less aggressive course. Steehler et al.[25] identified central atopic disease as an NP phenotype associated with inhalant allergy, with lower recurrence and revision rates than those of other CRS subtypes. These findings support the idea that NP in atopic conditions may be more localized and milder than in CF or PCD, which involve more severe airway pathology.[4,17,24-29] These insights highlight that clinical features such as NP can inform different management strategies depending on the disorder, underscoring the importance of disease-specific surveillance in pediatric NP.

Spirometric parameters were similar across disease groups, including CF, PCD, and asthma/AR. In children, nasal disease and NP mainly reflect upper-airway issues and are only weakly related to spirometric indices.[30-32] CF and PCD pulmonary impairment mainly result from lower-airway disease, while in asthma, airflow limitation depends on control. Our data showed similar spirometric profiles across groups. A recent meta-analysis found no significant postoperative improvements in FEV1, FVC, or FEV1/FVC after sinus surgery in CF, suggesting that sinonasal disease affects morbidity but not pulmonary decline.[25] Adult studies link NP and chronic rhinosinusitis with more severe asthma, but limited pediatric data show no spirometric decline related to NP.[33] Our findings support the idea that NP primarily causes sinonasal issues, not directly affecting lung function.

This study has some limitations. The retrospective design introduces potential risks of missing data and selection bias. Heterogeneity within subgroups and relatively small sample sizes may have limited the ability to detect subtle differences, which is a common challenge in rare disease cohorts. Not all patients had an HSS, which is another limitation. Additionally, systematic screening for NSAID hypersensitivity was not available for all patients. Since this was a cross-sectional study, follow-up duration and visit frequency varied among patients, which could have impacted the accuracy of recurrence rate assessment and limited the evaluation of long-term disease progression and causal relationships.

In conclusion, our findings highlight the variability of pediatric NP in terms of underlying causes, presentation, and clinical progression. Patients with PCD were more likely to present with bilateral NP, whereas those with CF exhibited the highest recurrence rates. Identifying underlying chronic diseases in many children with NP emphasizes the importance of comprehensive evaluation in this group. Additionally, when NP is diagnosed, it should be evaluated for underlying conditions. Specifically, disorders like CF and PCD, which involve genetically impaired mucociliary clearance, are associated with a higher burden and recurrence of NPs and should be considered during follow-up. Variations in NP features across different underlying causes indicate that detailed characterization of these features could help evaluate disease progression and guide follow-up care. These findings underscore the importance of routine upper airway monitoring in high-risk populations.

Ethical approval

The study was approved by Gazi University Ethics Committee (date: September 24, 2024, number: 2024-1502).

Author contribution

The authors confirm contribution to the paper as follows: Study conception and design: HK, TSE, ATA; data collection: ABÇ, NK; analysis and interpretation of results: FB; draft manuscript preparation: HK, TSE, ATA. All authors reviewed the results and approved the final version of the manuscript.

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Conflict of interest

The authors declare that there is no conflict of interest.

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