

Observational Study of Effect of Intranasal Corticosteroids in Improving Nasal Obstruction in Patients with Symptomatic Adenoidal Hypertrophy

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ABSTRACT

Background: Adenoidal hypertrophy commonly causes nasal obstruction, snoring, and mouth breathing. This study evaluated the effectiveness of intranasal corticosteroids in improving symptoms and reducing adenoid size.

Methods: This prospective observational study included 60 patients aged 8-25 years with symptomatic adenoidal hypertrophy. Clinical symptoms and adenoid grading were assessed at baseline, four weeks, and eight weeks following intranasal corticosteroid therapy. Forty patients completed six-month follow-up.

Results: Snoring (86.6%) and mouth breathing (83.3%) were the commonest presenting symptoms. At eight weeks, complete resolution of snoring increased from 13.3% to 75.0%, mouth breathing from 16.7% to 56.7%, nasal obstruction from 46.7% to 78.3%, and nasal discharge from 60.0% to 98.3% (all $p=0.001$). Grade III-IV adenoidal hypertrophy decreased from 51.6% to 21.7%, while 15.0% achieved complete radiological/endoscopic resolution (Grade 0). Clinical improvement was largely maintained at six months.

Conclusion: Intranasal corticosteroids were associated with significant symptomatic improvement and reduction in adenoid hypertrophy, supporting their role as a conservative treatment option.

Keywords: Adenoidal Hypertrophy, Intranasal Corticosteroids, Nasal Obstruction, Snoring, Mouth Breathing, Diagnostic Nasal Endoscopy.

INTRODUCTION

Adenoidal hypertrophy (AH) is one of the most common causes of upper airway obstruction in children, particularly between 3 and 10 years of age [1]. The adenoids are lymphoid tissues located in the roof and posterior wall of the nasopharynx and form an important component of Waldeyer's ring, contributing to mucosal immunity during early childhood [2,3]. Physiologically, adenoidal tissue enlarges during childhood, reaching its maximum size around 6–7 years, followed by gradual involution during adolescence. However, recurrent infections, chronic inflammation, allergic conditions and environmental factors may cause persistent enlargement, resulting in

nasopharyngeal airway obstruction [4,5]. Although predominantly affecting younger children, adenoidal hypertrophy may also occur in older children and adolescents, particularly in association with chronic inflammatory and allergic conditions [6]. The clinical manifestations depend on the degree of airway obstruction and commonly include nasal blockage, persistent mouth breathing, snoring, rhinorrhoea and hyponasal speech [7]. Prolonged obstruction may contribute to abnormal craniofacial development, including a long face, narrow maxilla, high-arched palate and dental malocclusion, collectively termed adenoid facies [8]. Significant adenoidal enlargement may also cause sleep-disordered breathing, ranging from habitual snoring to obstructive sleep apnoea, resulting in disturbed sleep, daytime somnolence,

behavioural changes and impaired academic performance [9,10]. Additionally, Eustachian tube dysfunction associated with enlarged adenoids may predispose children to otitis media with effusion, conductive hearing loss and delayed speech development. Severe untreated cases may occasionally result in growth impairment and cardiopulmonary complications

[11,12].Diagnosis is primarily based on clinical history and physical examination, supported by radiological and endoscopic investigations. Lateral nasopharyngeal radiography provides an accessible and inexpensive method of assessing adenoidal enlargement, whereas flexible fiberoptic nasopharyngoscopy allows direct visualisation of adenoidal tissue and the extent of airway obstruction

[13,14].Management depends on symptom severity, degree of obstruction and associated complications. While mild cases may respond to conservative treatment, persistent nasal obstruction, recurrent infections or significant sleep-disordered breathing may necessitate surgical intervention [15]. Adenoidectomy remains an established treatment for symptomatic adenoidal hypertrophy; however, potential complications, including bleeding, postoperative pain, infection and anaesthesia-related risks, highlight the need for effective non-surgical alternatives [16].Intranasal corticosteroids have emerged as a promising conservative treatment owing to their potent local anti-inflammatory effects. They suppress inflammatory mediators, reduce mucosal oedema and potentially improve nasopharyngeal airway patency with relatively low systemic exposure [17]. Commonly used agents include mometasone furoate, fluticasone propionate, budesonide and beclomethasone dipropionate. Previous clinical studies have demonstrated improvements in nasal obstruction and reductions in adenoidal size following intranasal corticosteroid therapy, suggesting their potential to avoid or defer surgical intervention in selected patients [18,19]. Nevertheless, uncertainties remain regarding optimal treatment duration, sustainability of symptomatic improvement and recurrence following discontinuation. Further observational evidence from routine clinical settings is therefore required to establish their therapeutic utility. Therefore, the present observational study was undertaken to evaluate the effect of intranasal corticosteroids in improving nasal obstruction among patients with symptomatic adenoidal hypertrophy and to assess

their potential role as a non-surgical treatment option.

MATERIALS AND METHODS

This prospective, single-arm observational follow-up study was conducted in the Department of Otorhinolaryngology (ENT) at a tertiary care hospital over 18 months, from October 2024 to March 2026, to evaluate the effectiveness of intranasal corticosteroids in patients with symptomatic adenoidal hypertrophy. A total of 60 patients aged 8–25 years with clinically and radiologically diagnosed adenoidal hypertrophy were enrolled using purposive sampling.

Inclusion Criteria: Patients aged 8–25 years presenting with nasal obstruction, mouth breathing or snoring, with adenoidal hypertrophy confirmed by lateral nasopharyngeal X-ray and diagnostic nasal endoscopy (DNE), wherever feasible, and providing the required informed consent.

Exclusion Criteria: Patients with previous adenoidectomy, hypersensitivity or contraindications to corticosteroids, craniofacial anomalies, other nasal pathologies mimicking adenoidal hypertrophy (e.g., nasal polyps), age below eight years or absence of informed consent.

Sample Size Calculation

The sample size was estimated using an anticipated clinical improvement rate of 62.2%, a 95% confidence level and 13% absolute precision, based on Sadeghi-Shabestari et al. (2022) [20].

$$n = \frac{Z^2 pq}{d^2} = \frac{(1.96)^2 (0.622)(0.378)}{(0.13)^2} \approx 54$$

After adjusting for an anticipated 10% loss to follow-up, the final sample size was 60 patients.

Study Procedure

Baseline Assessment: All participants underwent detailed history-taking, clinical examination, symptom severity assessment, lateral nasopharyngeal radiography and DNE using a 0-degree endoscope in cooperative patients. Adenoidal grade and the adenoid/choana ratio were documented. **Treatment Protocol:** All participants received intranasal mometasone furoate or fluticasone furoate spray, prescribed as two puffs twice daily for eight weeks. Subsequently, maintenance

therapy was prescribed as one puff twice daily for one month, followed by one puff once daily for the next three months.

Follow-up Assessment: Clinical reassessment was performed at four and eight weeks using symptom severity scores. Lateral nasopharyngeal radiography was repeated at four and eight weeks, while endoscopic reassessment was performed in cooperative patients at eight weeks. Final follow-up was conducted at six months to document symptomatic and objective improvement. Patients with inadequate improvement after eight weeks were advised regarding surgical management.

Study Parameters and Outcome Measures

Primary Outcome: Improvement in nasal obstruction following intranasal corticosteroid therapy, assessed using a visual analogue scale (VAS) at baseline, four weeks and eight weeks. **Secondary Outcomes:**

- Changes in snoring, mouth breathing and nasal discharge severity.
- Changes in adenoidal grade on lateral nasopharyngeal radiography.
- Changes in the adenoid/choana ratio assessed through DNE.
- Sustained symptomatic improvement at sixmonth follow-up.

Data Collection

Demographic characteristics, clinical findings, symptom scores, radiological grades, endoscopic measurements and follow-up outcomes were documented using structured case record forms. Patient/caregiver-reported symptoms and relevant radiological and endoscopic findings were recorded for comparative assessment.

Statistical Analysis

Data were analysed using IBM SPSS Statistics version 25.0. Continuous variables were expressed as mean $\pm$ standard deviation or median (interquartile range), while categorical variables were presented as frequencies and percentages. Paired t-tests or Wilcoxon signedrank tests were applied for pre- and posttreatment comparisons, as appropriate. Chisquare or Fisher's exact tests were used for categorical comparisons. A two-sided p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 60 patients with symptomatic adenoidal hypertrophy were enrolled in the study. The majority of patients belonged to the 8–10-year age group (51.6%), followed by 11–13 years (16.6%) and 14–16 years (11.6%). Male predominance was observed, with 42

(70.0%) males and 18 (30.0%) females (Table 1). Snoring was the most common presenting symptom, reported in 52 (86.6%) patients, followed by mouth breathing in 50 (83.3%), nasal obstruction in 32 (53.3%) and nasal discharge in 24 (40.0%). Among the features of adenoid facies, open-mouth posture was most prevalent (80.0%), followed by elongated and dull facial appearance (65.0%), dental misalignment (60.0%), pinched nose (56.6%) and high-arched palate (41.6%) (Table 2). Baseline lateral nasopharyngeal radiography demonstrated Grade II adenoidal hypertrophy in 36.7% of patients, followed by Grade III (35.0%), Grade IV (13.3%) and Grade I (10.0%), while 5.0% showed no radiological enlargement. Diagnostic nasal endoscopy (DNE) revealed Grade II and Grade III hypertrophy in 33.3% of patients each, followed by Grade IV (18.3%) and Grade I (15.0%). The difference in the overall grading distributions between radiographic and endoscopic assessments was not statistically significant ($p=0.27$). Based on DNE findings, 51.6% of patients had Grade III or IV adenoidal hypertrophy at baseline (Table 3, Figure 1). Significant improvement was observed in all four assessed clinical symptoms following intranasal corticosteroid therapy over eight weeks. The proportion of patients without snoring increased from 13.3% at baseline to 60.0% at four weeks and 75.0% at eight weeks ($p=0.001$). Similarly, the absence of mouth breathing increased progressively from 16.7% to 41.7% and 56.7%, respectively ($p=0.001$). Moderate-to-severe snoring and mouth breathing were completely absent at eight weeks.

Nasal obstruction also showed significant improvement, with the proportion of asymptomatic patients increasing from 46.7% at baseline to 75.0% at four weeks and 78.3% at eight weeks ($p=0.001$). Moderate-to-severe nasal obstruction was no longer reported after four weeks. Likewise, complete resolution of nasal discharge increased from 60.0% at baseline to 98.3% at both four and eight weeks ($p=0.001$). All reported baseline-to-eight-week comparisons were statistically significant (Table 4). A progressive reduction

in adenoidal hypertrophy was observed following intranasal corticosteroid therapy. At baseline, Grade III and IV hypertrophy accounted for 51.6% of patients, which decreased to 31.7% at four weeks and 21.7% at eight weeks. Conversely, the proportion of patients with Grade I hypertrophy increased from 15.0% at baseline to 26.7% at four weeks and 33.3% at eight weeks, while Grade 0 increased from 0% to

15.0%. Although the change in adenoidal grading at four weeks was not statistically significant compared with baseline ($p=0.09$), a significant improvement was observed at eight weeks ($p=0.001$), indicating a progressive reduction in adenoidal size following treatment (Table 5, Figure 2). Of the 60 enrolled patients, 40 (66.7%) completed the six-month follow-up. Clinical improvement achieved at eight weeks was largely maintained at six months. Complete resolution of

snoring increased from 70.0% at eight weeks to 75.0% at six months ($p=0.42$), while the absence of mouth breathing increased from 65.0% to 70.0% ($p=0.38$). Similarly, the proportion of patients without nasal obstruction increased from 75.0% to 80.0% ($p=0.51$), whereas nasal discharge remained absent in 97.5% of patients at both assessments ($p=1.00$). None of these changes was statistically significant (Table 6A). Regarding adenoidal grading, Grade I hypertrophy increased from 32.5% at eight weeks to 37.5% at six months, while Grade II increased from 30.0% to 32.5%. The proportions of Grade III (17.5%) and Grade IV (5.0%) remained unchanged, whereas Grade 0 decreased from 15.0% to 7.5%. These findings demonstrated that the reduction in adenoidal hypertrophy achieved during the initial treatment period was largely sustained among patients completing six-month follow-up (Table 6B, Figure 3).

Table 1. Baseline Demographic Characteristics of the Study Population (N=60)

Parameter	Frequency (n)	Percentage (%)
Age group (years)		
8–10	31	51.6
11–13	10	16.6
14–16	7	11.6
17–19	5	8.3
20–22	3	5.0
23–25	4	6.6
Sex		
Male	42	70.0
Female	18	30.0

Table 2. Baseline Distribution of Clinical Symptoms and Features of Adenoid Facies (N=60)

Clinical characteristics	Frequency (n)	Percentage (%)
Presenting symptoms		
Snoring	52	86.6
Mouth breathing	50	83.3
Nasal obstruction	32	53.3
Nasal discharge	24	40.0
Features of adenoid facies		
Open-mouth posture	48	80.0
Elongated and dull face	39	65.0
Dental misalignment	36	60.0
Pinched nose	34	56.6
High-arched palate	25	41.6

Table 3. Comparison of Baseline Adenoidal Hypertrophy Grading by X-ray and Diagnostic Nasal Endoscopy (N=60)

Adenoidal grade	X-ray findings n (%)	DNE findings n (%)
Grade 0 (Absent)	3 (5.0)	0 (0.0)

Grade I	6 (10.0)	9 (15.0)
Grade II	22 (36.7)	20 (33.3)
Grade III	21 (35.0)	20 (33.3)
Grade IV	8 (13.3)	11 (18.3)
Total	60 (100.0)	60 (100.0)

Reported p-value = 0.27.

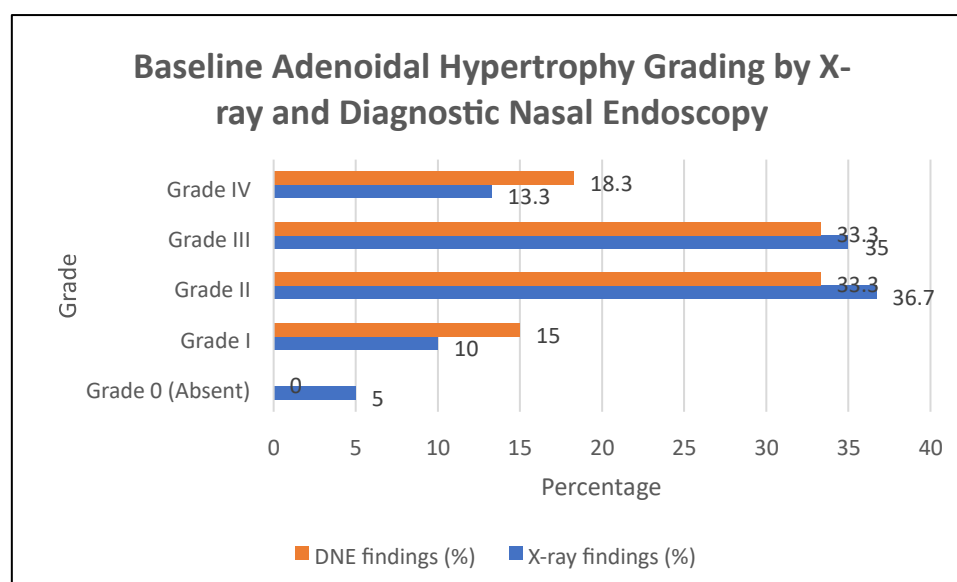


Figure 1. Comparison of Baseline Adenoidal Hypertrophy Grading by X-ray and Diagnostic Nasal Endoscopy (N=60)

Table 4. Changes in Clinical Symptom Severity Following Intranasal Corticosteroid Therapy at Baseline, 4 Weeks and 8 Weeks (N=60)

Clinical symptom and severity	Baseline n (%)	4 weeks n (%)	8 weeks n (%)	χ^2 value*	pvalue*
Snoring				43.89	0.001
Absent (0)	8 (13.3)	36 (60.0)	45 (75.0)		
Mild (1)	30 (50.0)	20 (33.3)	15 (25.0)		
Moderate (2)	17 (28.3)	4 (6.7)	0 (0.0)		
Severe (3)	5 (8.3)	0 (0.0)	0 (0.0)		
Mouth breathing				56.67	0.001
Absent (0)	10 (16.7)	25 (41.7)	34 (56.7)		
Mild (1)	20 (33.3)	23 (38.3)	26 (43.3)		
Moderate (2)	24 (40.0)	12 (20.0)	0 (0.0)		
Severe (3)	6 (10.0)	0 (0.0)	0 (0.0)		
Nasal obstruction				76.52	0.001
Absent (0)	28 (46.7)	45 (75.0)	47 (78.3)		
Mild (1)	18 (30.0)	15 (25.0)	13 (21.7)		
Moderate (2)	13 (21.7)	0 (0.0)	0 (0.0)		
Severe (3)	1 (1.7)	0 (0.0)	0 (0.0)		
Nasal discharge				89.54	0.001
Absent (0)	36 (60.0)	59 (98.3)	59 (98.3)		
Mild (1)	19 (31.7)	1 (1.7)	1 (1.7)		
Moderate (2)	5 (8.3)	0 (0.0)	0 (0.0)		
Severe (3)	0 (0.0)	0 (0.0)	0 (0.0)		

Table 5. Changes in Adenoidal Hypertrophy Grading Following Intranasal Corticosteroid Therapy (N=60)

Adenoidal grade	Baseline n (%)	4 weeks n (%)	8 weeks n (%)
Grade 0	0 (0.0)	3 (5.0)	9 (15.0)
Grade I	9 (15.0)	16 (26.7)	20 (33.3)
Grade II	20 (33.3)	22 (36.7)	18 (30.0)
Grade III	20 (33.3)	13 (21.7)	10 (16.7)
Grade IV	11 (18.3)	6 (10.0)	3 (5.0)
Total	60 (100.0)	60 (100.0)	60 (100.0)
p-value (vs. baseline)	—	0.09	0.001

Statistical test reported in the original results: Chi-square test.

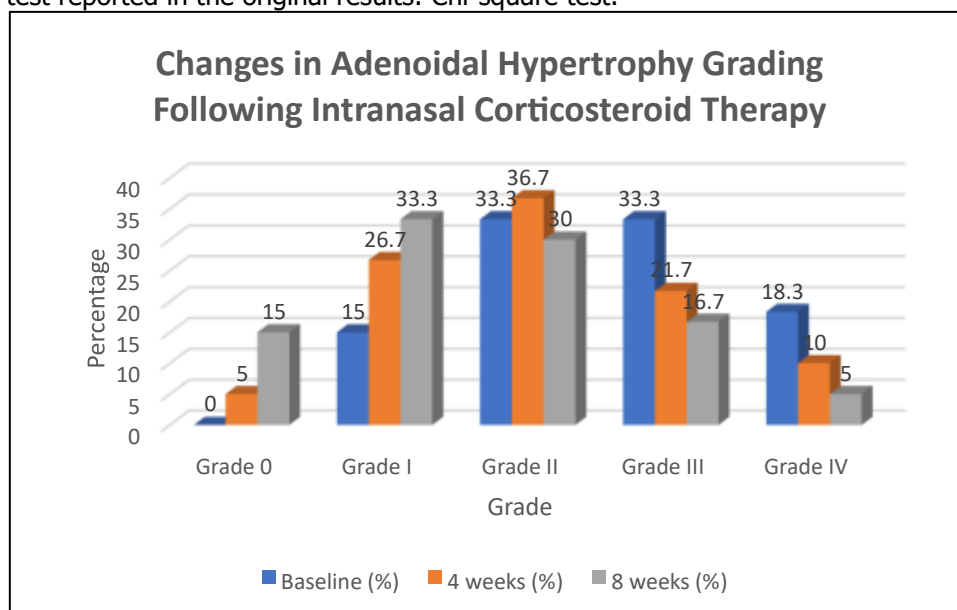


Figure 2. Changes in Adenoidal Hypertrophy Grading Following Intranasal Corticosteroid Therapy

Table 6. Comparison of Clinical Outcomes and Adenoidal Grading at Eight Weeks and Six Months (n=40)

A. Clinical symptom resolution

Clinical outcome	8 weeks n (%)	6 months n (%)	p-value
Absence of snoring	28 (70.0)	30 (75.0)	0.42
Absence of mouth breathing	26 (65.0)	28 (70.0)	0.38
Absence of nasal obstruction	30 (75.0)	32 (80.0)	0.51
Absence of nasal discharge	39 (97.5)	39 (97.5)	1.00

B. Adenoidal hypertrophy grading

Adenoidal grade	8 weeks n (%)	6 months n (%)
Grade 0	6 (15.0)	3 (7.5)
Grade I	13 (32.5)	15 (37.5)
Grade II	12 (30.0)	13 (32.5)
Grade III	7 (17.5)	7 (17.5)
Grade IV	2 (5.0)	2 (5.0)
Total	40 (100.0)	40 (100.0)

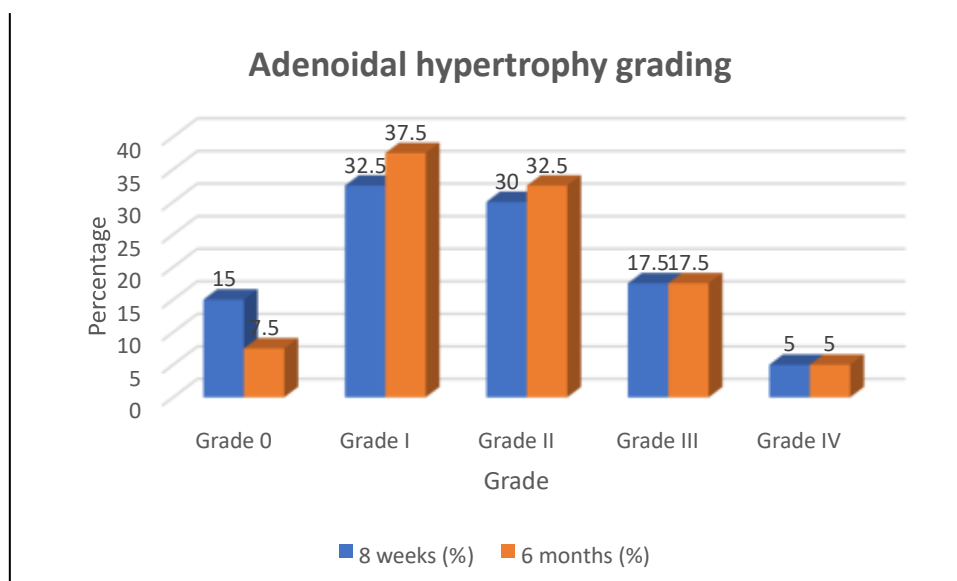


Figure 3. Adenoidal hypertrophy grading

DISCUSSION

The present prospective observational study evaluated intranasal corticosteroid therapy in 60 patients with symptomatic adenoidal hypertrophy. Significant improvement in clinical symptoms and adenoid grading was observed over eight weeks, with benefits largely maintained among the 40 patients completing six-month follow-up. Most patients were aged 8–10 years (51.6%), consistent with the younger age predominance reported by Khadgi et al. [21] (mean age 7.67 years), Khan et al. [22] (predominantly 3–7 years), Solmaz et al. [15] (7.92±1.81 years), and Hashim et al. [23] (6.85±2.62 years). Males constituted 70.0% of our cohort, comparable to the male predominance reported in these studies [21,22]. Snoring (86.6%) and mouth breathing (83.3%) were the predominant symptoms, followed by nasal obstruction (53.3%) and nasal discharge (40.0%). Similar presentations were documented by Khadgi et al. [21], Hashim et al. [23], and Sadeghi-Shabestari et al. [20], whereas Singh et al. [24] reported greater prevalence of nasal obstruction and discharge. Open-mouth posture was the commonest feature of adenoid facies (80.0%), consistent with the chronic obstructive manifestations discussed by Colavita et al. [25] and Elbeltagy et al. [26]. On radiography, Grade II (36.7%) and III (35.0%) hypertrophy predominated, while DNE demonstrated Grade II and III disease in 33.3% each and Grade IV in 18.3%. Similar predominance of moderate-to-advanced hypertrophy was reported by Khadgi

et al. [21], Solmaz et al. [15], Hashim et al. [23], and Singh et al. [24].

Snoring improved significantly, with complete symptom resolution increasing from 13.3% at baseline to 75.0% at eight weeks ($p=0.001$). Comparable improvement was reported by Arslan et al. [28] ($p<0.001$), Hashim et al. [23] (mean score reduction from 7.63 ± 0.85 to 5.53 ± 2.06), and Sadeghi-Shabestari et al. [20]. Zhang et al. [27], reviewing six randomized trials involving 394 children, also documented symptomatic improvement in five studies. Mouth breathing showed significant improvement, with asymptomatic patients increasing from 16.7% to 56.7% ($p=0.001$). Similar findings were reported by Hashim et al. [23] and Sadeghi-Shabestari et al. [20]. Khan et al. [22] demonstrated greater treatment success with intranasal steroids than saline (88% vs. 74%; $p<0.001$), while Solmaz et al. [15] reported marked clinical and endoscopic improvement following mometasone therapy. Nasal obstruction resolved in 78.3% of patients at eight weeks compared with 46.7% at baseline ($p=0.001$). These findings agree with Arslan et al. [28], Hashim et al. [23], and Sadeghi-Shabestari et al. [20]. Zhang et al. [27] similarly demonstrated significant symptomatic improvement and reduced surgical requirements in responsive patients. Nasal discharge also improved markedly, with 98.3% becoming symptom-free by four weeks and remaining asymptomatic at eight weeks ($p=0.001$). Hashim et al. [23] similarly demonstrated significant improvement in rhinorrhoea following six weeks of

corticosteroid therapy. A significant reduction in adenoidal hypertrophy was observed, with Grade III–IV disease decreasing from 51.6% at baseline to 21.7% at eight weeks, while Grade I increased from 15.0% to 33.3%, and 15.0% achieved Grade 0 ($p=0.001$). Similar regression was demonstrated by Arslan et al. [28], Hashim et al. [23], Solmaz et al. [15], and Singh et al. [24]. Notably, Solmaz et al. [15] reported Grade I disease in 81% of patients following treatment, with complete disappearance of Grade III–IV hypertrophy. These observations are supported by Elbeltagy et al. [26], who reported significant reductions in adenoid size; Alqutub et al. [29], who demonstrated reduced adenoid size ($SMD=-1.33$) and adenoidectomy rates ($RR=0.30$); and Ripp et al. [30], who observed a greater reduction in severe hypertrophy with corticosteroids than controls (43.5% vs. 13.3%). Khadgi et al. [21] additionally associated larger adenoids with increased conductive hearing loss risk, highlighting the clinical relevance of reducing disease severity. Sadeghi-Shabestari et al. [20] and Khan et al. [22] likewise demonstrated favourable clinical responses to intranasal corticosteroids. Among the 40 patients completing six-month follow-up, improvement was largely maintained. Complete resolution of snoring increased from 70.0% at eight weeks to 75.0% at six months, mouth breathing from 65.0% to 70.0%, and nasal obstruction from 75.0% to 80.0%, while 97.5% remained free of nasal discharge. These interval changes were not statistically significant. Grade III and IV hypertrophy remained unchanged at 17.5% and 5.0%, respectively, although Grade 0 decreased from 15.0% to 7.5%, suggesting possible limited regrowth. These findings are broadly consistent with the therapeutic benefits reported by Arslan et al. [28], SadeghiShabestari et al. [20], Hashim et al. [23], Solmaz et al. [15], and Singh et al. [24], although their follow-up durations differed. Colavita et al. [25] highlighted persistent or recurrent symptoms following adenoidectomy in children with allergic rhinitis, emphasizing the importance of controlling underlying inflammation. Systematic evidence from Elbeltagy et al. [26], Alqutub et al. [29], and Ripp et al. [30] further supports the role of intranasal corticosteroids in reducing disease severity and surgical requirements.

CONCLUSION

Intranasal corticosteroid therapy was associated with significant improvement in nasal obstruction, snoring, mouth breathing, and nasal discharge among patients with symptomatic adenoidal hypertrophy. A significant reduction in adenoid grading was observed after eight weeks of treatment ($p=0.001$). Clinical improvement was largely maintained at six months, supporting intranasal corticosteroids as a conservative treatment option for symptomatic adenoidal hypertrophy.

Limitations

The single-centre observational design, relatively small sample size ($N=60$), and absence of a control group limit the generalisability and causal interpretation of the findings. Only 40 patients completed six-month follow-up, and the limited follow-up duration restricts assessment of long-term recurrence and sustained treatment efficacy.

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