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Effectiveness of Evaluation of Adenoid Hypertrophy in Children by Flexible Nasopharyngoscopy Examination (FNE), Proposed Schema of Frequency of Examination: Cohort Study

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Abstract: Objectives: To demonstrate the effectiveness of endoscopic assessment of the pharyngeal tonsil in defining the size of the adenoid hypertrophy in endoscopic examination that would be equivalent to intraoperative assessment as a large adenoid, and to determine the lowest necessary frequency of tests to assess the variability of its size. Methods: The study is based on an analysis of two groups of children diagnosed and treated in a children's ENT outpatient clinic and ENT department. In the first group, adenoid size was assessed based on flexible endoscopy, and then with a consequent adenoid surgery with assessment of the intraoperative size of the adenoid, we compared the size of the adenoid. The second part of the study included a group of 81 children. We analyzed performed flexible nasopharyngoscopy examinations (FNE) of each child, and compared the change of adenoid size in a minimum of two subsequent examinations over a period of 1 year or more. Results: The sensitivity of flexible endoscopic examination in the assessment of the pharyngeal tonsil was determined at 97.3%, and specificity at 72.7%. The ROC curve shows the value of adenoid-to-choana (A/C) ratio as 75% or more in the preoperative FNE, indicating that the tonsil during surgery is assessed as large. Among the children, 26.3% had a change in adenoid size of more than 15% in the A/C ratio in 1 year of observation, and 45% of the children had A/C ratio changed above 15% in a period of 3 years of observation. Conclusions: FNE examination is highly effective in assessing the size of the pharyngeal tonsil. We proposed a schema for frequencies of FNE examinations and treatment dependent on A/C ratio and worsening of ailments.

Keywords: adenoid size; adenoid hypertrophy; adenoidectomy indications; flexible nasofiberoscopy

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1. Introduction

The adenoid (pharyngeal tonsil) has been a mysterious cause of numerous ailments in children for years. It is perceived as the cause of the recurrence of upper respiratory infection, snoring, hypoacusis and otitis media with effusion, obstructive sleep apnea, facial growth disorders, malocclusion, and, finally, may have an influence on behavioral symptoms [1–3]. Problems of examination and visualization of the adenoid make it difficult to assess its true role in these disorders. Hans Wilhelm Meyer was the first to diagnose adenoid hypertrophy with his finger in 1868, and then removed the adenoid [4]. Since then, numerous diagnostic techniques have been introduced, seeking the most effective, but also the most comfortable and least burdensome for the patient. These techniques may be divided into two groups: invasive and imaging. The first group consists of manual finger or mirror examination through the mouth, rigid or flexible nasopharyngoscopy

examination (FNE), videofluoroscopy, and acoustic rhinomanometry. The second group comprises ultrasonography, lateral nasopharyngeal X-ray (lateral cephalogram), multirow detector CT with virtual laryngoscopy, or MRI of the nasopharynx [5–9]. However, the effectiveness of these tests is related to unobjective results, such as the severity of adenoid hypertrophy symptoms, otitis media with effusion (OME), tympanometry, or air flow through the nose. However, not all adenoid hypertrophy symptoms must be associated with adenoid hypertrophy. In some cases, it may be simulated by other causes of nose and nasopharyngeal obstruction, such as septal deviation, nasal polyps, nasal concha hypertrophy, and allergic rhinitis [6,10]. Some of the adenoid examination methods refer to intraoperative evaluation using a transoral mirror examination or to nasal endoscopy [6,9–11]. Patel et al. showed that in patients who had less obstructive adenoid hypertrophy with less than 75% in adenoid-to-choana scale (A/C ratio), preoperative FNE and intraoperative mirror exam may not correlate, suggesting that intraoperative mirror examination performed in a horizontal position in anesthesia with relaxation may also be fraught with observation errors [6]. We should ask the following question: How accurate is the FNE examination as a defined gold standard? Therefore, in the following work, an attempt was made to relate the results of the endoscopic examination to the size of the tonsil being removed during the surgery. Another issue is the question of how often in the clinical observation should such an examination be performed, and more precisely, the question should be answered for how the size of the tonsil changes with age. There is prevalent opinion, often based on ENT doctors' experience or based on the almost 100-year-old Scammon's theory in accordance with proposed Scammon curves, that the adenoid undergoes hypertrophy during childhood and involution in adulthood [12]. To the best of our knowledge, only three longitudinal observational studies, one performed in 1976 by Handelman and two performed in Japan, published in 2018 and 2021, have assessed the sizes of the adenoid, all based on lateral cephalometric radiography [13–15]. Therefore, there is the need for longitudinal adenoid observations to investigate the process of adenoid involution, and to assess its influence on the reduction of adenoid symptoms in children. We used FNE to analyze the term change of the adenoid size.

The aim of this study was to determine the effectiveness of endoscopic assessment of the pharyngeal tonsil and its correlation with intraoperative assessment of adenoid size. The second aim of this study was to analyze longitudinal changes in adenoid size to obtain knowledge of its yearly variations. This may be useful in determining the lowest necessary frequency of tests to monitor the variability in its size.

2. Materials and Methods

2.1. Research Participants

The study is based on a retrospective analysis of two groups of children diagnosed and treated in a children's ENT outpatient clinic. The first group consisted of 108 children aged 3 to 9 who were qualified and underwent subsequent adenoidectomy because of adenoid hypertrophy and recurrent adenoid symptoms, obstructive sleep apnea (OSA), and as an adjuvant treatment of otitis media with effusion with grommet insertion between 2019 and 2021. The second analyzed group consisted of children who were the patients of the ENT outpatient clinic for a longer period of time, at least 1 year, and had undergone subsequent endoscopic choana examination between 2016 and 2021.

2.2. Inclusion and Exclusion Criteria

In the first part of the study, we included children admitted to the ENT outpatient clinic who subsequently underwent adenoidectomy or adenoidectomy with grommet insertion. In the second sample, we enrolled patients who did not undergo adenoid surgery during the observation period. We excluded patients with genetic diseases (Down, Treacher–Collins Syndrome, 9th chromosome trisomy) and craniofacial anomalies, cleft palate, or submucosal cleft from the studies. Children who had an active upper

respiratory infection during the performed endoscopy or those who had previously undergone adenoidectomy or maxillofacial surgery or trauma were eliminated from the study.

2.3. Study Methods

In the first analyzed group, adenoid size was assessed based on flexible endoscopy and consequent adenoid surgery with use of an age-appropriate adenotome. We analyzed 108 subsequent adenoidectomies. Before each surgery, ENT doctors (PB, KM) analyzed the endoscopy previously performed in the ENT outpatient clinic and assessed the adenoid size in adenoid-to-choana ratio (A/C) as a percentage. All adenoidectomies were performed by a third ENT doctor (AZ), who was blind to the results of the endoscopies. Intraoperatively, the surgeon assessed the adenoid size as "small," "medium," or "large." The assessment of the size of the tonsil during surgery was expressed as "large" if the removed tonsil tissue was larger than the hole in the Beckmann adenotome. "Medium" was smaller than the size of the adenotome opening, but occupying more than three-fourths of its lumen. "Small" tonsil tissue occupied less than three-fourths of the adenotome opening (Figure 1). The intraoperative size of the tonsil was compared with an endoscopic assessment. We also performed control visits at least 2 weeks, 6 months, and 12 months after surgery, asking parents if the children had any problems with breathing through the nose, presence of snoring, mouth-breathing, and hyponasal voice or recurrent middle-ear disease; and in any suspicion of adenoid regrowth, we performed flexible endoscopy. If we observed that regrowth of the adenoid tissue to the A/C ratio reached 30% without middle-ear disturbance symptoms, we did not perform revision surgery.

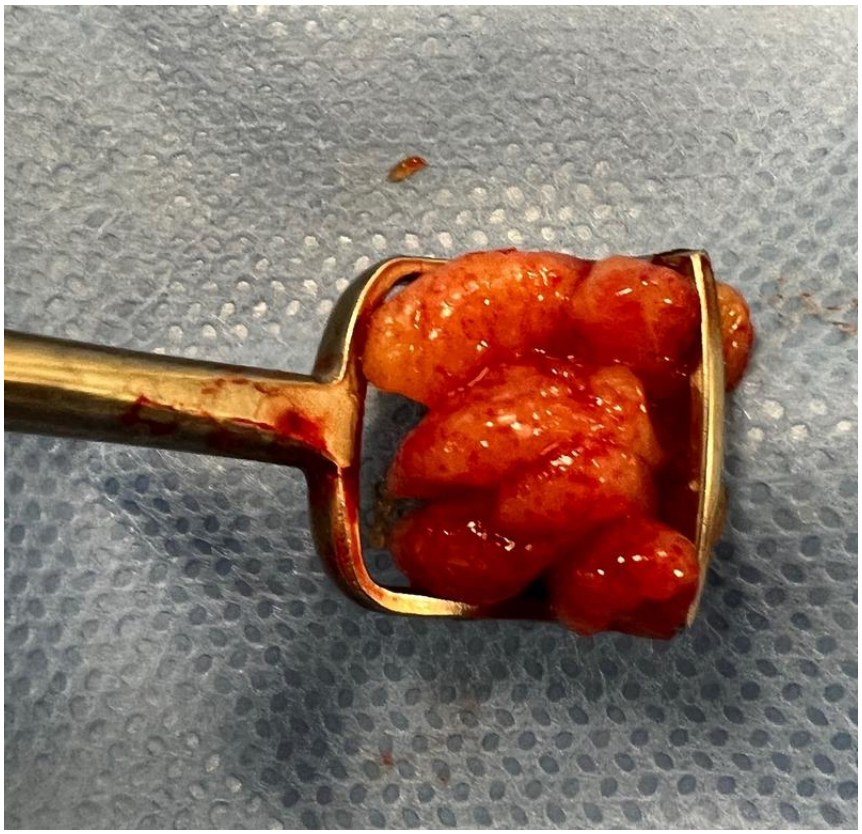

Figure 1. Intraoperative adenoid size measurement.

The second part of the study included a group of 81 children. We analyzed endoscopic examinations (FNE) performed by one children's ENT doctor (AZ) on each child

and compared the change in adenoid size in a minimum of two subsequent examinations over a period of 1 year or more.

2.4. Endoscopy

Flexible nasopharynx endoscopic (FNE) examinations were performed in the ENT outpatient clinic by an ENT specialist using the Karl Storz Germany Tele Pack compact endoscopy system (18 kilo pixels, 2.8 mm outer diameter, flexible nasopharyngoscope; Medit Inc.). Based on the recorded video files, we used DaVinci Resolve 17 software (Blackmagic Design) to evaluate and calculate the percentages of obturation of the choanae (A/C ratio, adenoid-to-choana ratio as a percentage). The A/C ratio was assessed with an accuracy of up to 5%. Additionally, we classified endoscopic adenoid size in accordance with the section of the Bolesławska scale: grade I, adenoid tissue filling less than one-third of the vertical portion of the choanae; grade II, adenoid tissue filling between one-third and two-thirds of the choanae; and grade III, adenoid tissue filling more than two-thirds of the choanae [16].

2.5. Surgery

Surgery was performed under general anesthesia. The child's mouth was opened widely with a McIvor retractor. Next, the palate was palpated for evidence of submucous cleft. Then, the catheter was inserted into the nose, retrieved through the mouth, and pulled anteriorly to retract the soft palate forward. The adenoid was removed through the mouth with the use of a Beckmann adenotome. The size of the adenotome used was selected according to the age of the child, in accordance with Shaalan's recommendations (3–4 years: 18 mm; 5–6 years: 19–20 mm; 7–9 years: 20–21 mm) [17]. A cotton pledget was used to stop any bleeding. Indirect mirror visualization of the choana was performed at the end of the surgery to confirm complete adenoid removal.

2.6. Statistical Analysis

Quantitative data are presented as mean ± standard deviation (*SD*) and median with interquartile range (Q25–Q75). For the categorical variables, we used numbers (*n*) and percentages (%). Differences in the distribution of categorical variables were evaluated using Pearson's $\chi 2$ or Fisher's exact test, as appropriate. Student's *t* test was used for comparison of quantitative variables between two independent groups. For comparing more independent groups, a one-way analysis of variance (ANOVA) was used. The difference between the maximum and minimum values (range) of the A/C ratio in each patient was used to evaluate changes in the adenoid size over time. In order to determine the diagnostic value of the A/C ratio to detect the operative adenoid size, the receiver operating characteristic (ROC) method was used, analyzing the area under the curve (AUC), as well as the sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) for the selected cutoff.

For all these tests, two-tailed *p* values were used, and a p value < 0.05 was considered statistically significant. The Bonferroni correction for multiple testing was used. All statistical analyses were performed with SPSS software (Statistical Package for the Social Sciences version 26, Armonk, NY, USA).

2.7. Ethics

Ethical approval for this study was obtained from the ethics committee of Nicolaus Copernicus University (KB 136/2022).

3. Results

3.1. Association of A/C Ratio with Operative Adenoid Size

3.1.1. Patients' Characteristics

We performed subsequent adenoidectomies in 108 children (43 girls and 65 boys, mean age 5.2 ± 1.8 years) who were previously endoscopically evaluated for A/C ratio and Bolesławska scale [16] (Table 1). The range of A/C ratio was 40% to 95%; the majority of children had 80%, 85%, and 90% A/C ratio (21.3%, 20.4%, and 14.8%, respectively). In our study, 23 (21.3%) children were classified as grade II adenoid hypertrophy by Bolesławska scale, and 85 (78.7%) were classified as grade III. During the surgery, 75 (69.4%) of the removed adenoids were assessed as large, 12 (11.1%) as medium, and 21 (19.4%) as small (operative adenoid size). There were no statistically significant differences between gender and age. The mean age of the girls was 5.4 years, and that of the boys was 5 years. In our sample, we did not report gender-dependent differences in the prevalence of adenoid hypertrophy in Bolesławska scale and adenoid size reported during the surgery. In our study, eight (18.6%) girls and 15 (23.1%) boys were classified as grade II by Bolesławska, and 35 (81.4%) girls and 50 (76.9%) boys were classified as grade III. During the surgery, the adenoids of eight (18.6%) girls and 13 (20.0%) boys were classified as small, six (14.0%) in each gender were classified as medium, and 29 (67.4%) in girls and 46 (70.8%) in boys as a large operative adenoid size. Detailed data for the whole group and according to gender are presented in Table 1.

Table 1. Patients' characteristics in the whole first group who underwent adenoidectomy and according to gender.

Characteristics		All Patients	Female	Male	*p* Value
n		108	43	65	
Gender	female	43 (39.8%)	43 (39.8%)		-
	male	65 (60.2%)		65 (60.2%)	
Age (years)	mean ± *SD*	5.2 ± 1.8	5.4 ± 1.9	5.0 ± 1.7	0.267
	median (Q25–Q75)	5.0 (4.0–6.0)	5.0 (4.0–6.0)	4.5 (4.0–5.5)	
Adenoid size (A/C ratio *, %)	mean ± *SD*	77.5 ± 13.2	78.8 ± 13.4	76.6 ± 13.2	0.396
	median (Q25–Q75)	80.0 (75.0–85.0)	80.0 (75.0–90.0)	80.0 (70.0–85.0)	
	40	1 (0.9%)	1 (2.3%)	0 (0.0%)	0.505
	50	10 (9.3%)	2 (4.7%)	8 (12.3%)	
	60	9 (8.3%)	4 (9.3%)	5 (7.7%)	
	65	3 (2.8%)	1 (2.3%)	2 (3.1%)	
	70	3 (2.8%)	1 (2.3%)	2 (3.1%)	
	75	13 (12.0%)	7 (16.3%)	6 (9.2%)	
	80	23 (21.3%)	7 (16.3%)	16 (24.6%)	
	85	22 (20.4%)	7 (16.3%)	15 (23.1%)	
	90	16 (14.8%)	8 (18.6%)	8 (12.3%)	
	95	8 (7.4%)	5 (11.6%)	3 (4.6%)	
Adenoid size (A/C ratio on Bolesławska scale, %)	35–65 (B II)	23 (21.3%)	8 (18.6%)	15 (23.1%)	0.587
	>65 (B III)	85 (78.7%)	35 (81.4%)	50 (76.9%)	
Operative adenoid size, 3 categories	small	21 (19.4%)	8 (18.6%)	13 (20.0%)	0.857
	medium	12 (11.1%)	6 (14.0%)	6 (9.2%)	
	large	75 (69.4%)	29 (67.4%)	46 (70.8%)	
Operative adenoid size, 2 categories	not large	33 (30.6%)	14 (32.6%)	19 (29.2%)	0.713
	large	75 (69.4%)	29 (67.4%)	46 (70.8%)	
	true	96 (88.9%)	37 (86.0%)	59 (90.8%)	0.536

Consistency between A/C ratio in Bolesławska scale and operative adenoid size **	false	12 (11.1%)	6 (14.0%)	6 (9.2%)

* A/C ratio—adenoid-to-choana ratio. ** Consistency when second degree in the Bolesławska scale (B II) = not large operative adenoid size (small and medium) or B III = large operative adenoid size.

3.1.2. Diagnostic Value of A/C Ratio

Comparing the size of the tonsil assessed on the A/C ratio and Bolesławska scales with the macroscopic assessment of the removed adenoid depending on whether it was assessed as large or medium and small, a high agreement was shown ($p < 0.001$ for all) (Table 2).

Table 2. Association between macroscopic adenoid size and demographic variables and A/C ratio.

Characteristics	Operative Adenoid Size, 3 Categories			*p* Value [1]	*p* Value [2]
	Small	Medium	Large		
n	**21**	**12**	**75**		
Gender					
female	8 (38.1%)	6 (50.0%)	29 (38.7%)	0.857	0.713
male	13 (61.9%)	6 (50.0%)	46 (61.3%)		
Age (years)					
mean ± *SD*	5.0 ± 1.5	4.7 ± 1.3	5.3 ± 1.9	0.559	0.336
median (Q25–Q75)	4.5 (4.0–6.5)	4.3 (4.0–5.8)	5.0 (4.0–6.0)		
Adenoid size (A/C ratio, %)					
mean ± *SD*	59.5 ± 13.6	69.6 ± 10.1	83.8 ± 6.8	<0.001 [3]	<0.001
median (Q25–Q75)	50.0 (50.0–65.0)	67.5 (60.0–77.5)	85.0 (80.0–90.0)		
Adenoid size (A/C ratio on Bolesławska scale, %)					
35–65 (B II)	16 (76.2%)	6 (50.0%)	1 (1.3%)	<0.001	<0.001
>65 (B III)	5 (23.8%)	6 (50.0%)	74 (98.7%)		
Consistency between A/C ratio on Bolesławska scale and operative adenoid size					
true	16 (76.2%)	6 (50.0%)	74 (98.7%)	<0.001	<0.001
false	5 (23.8%)	6 (50.0%)	1 (1.3%)		

[1] p value for comparison of small vs. medium vs. large adenoid size; [2] p value for comparison of not large (small + medium) vs. large adenoid size. [3] Bonferroni post hoc tests: small vs. medium, $p = 0.007$; medium vs. large, $p < 0.001$; small vs. large, $p < 0.001$.

Based on the ROC curve, the diagnostic value of A/C ratio in large operative adenoid size diagnosis was evaluated (Figure 2). In our study group, high diagnostic accuracy was found for A/C ratio (AUC = 0.894; 95% CI = 0.818–0.969; $p < 0.001$). The course of the ROC curve indicates the existence of two potential cutoff points for the A/C ratio with significant discrimination values: 75% (<75% vs. ≥75%) and 80% (<80% vs. ≥80%). According to the determined cutoff, a higher A/C ratio in the endoscopic examination indicates that the adenoid size during surgery is assessed as large. For the cutoff value of 75%, the sensitivity was 97.3%, and the specificity was 72.7%. For A/C ratio at the cutoff value of 80%, the sensitivity was 84.0% and the specificity was 81.8% (Table 3). Among these two threshold values, the A/C ratio of 75% has greater prognostic accuracy in detecting a large operative adenoid size.

Table 3. Diagnostic value of A/C ratio for diagnosis of large operative adenoid size.

	A/C Ratio	
AUC	0.894	
95% CI	0.818–0.969	
***p* value**	<0.001	
Cutoff value (%)	75.0	80.0
Sensitivity	97.3%	84.0%
Specificity	72.7%	81.8%
PPV	89.0%	91.3%
NPV	92.3%	69.2%

AUC—area under the curve. 95% CI—95% confidence interval. PPV—positive predictive value. NPV—negative predictive value.

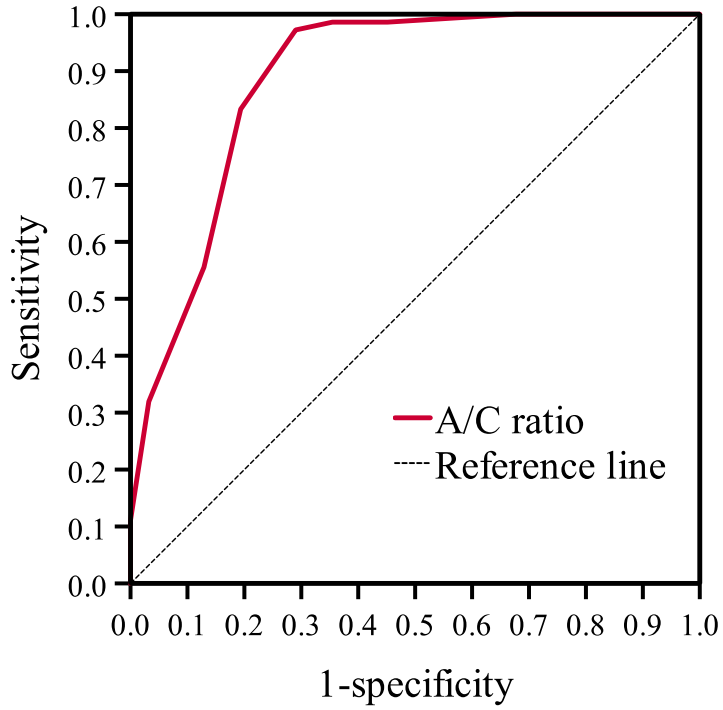

Figure 2. Diagnostic accuracy of A/C ratio for diagnosis of large operative adenoid size.

3.2. Volatility of the A/C Ratio over Time

The second retrospective study group of patients consisted of 81 children; 43 (53.1%) were girls and 38 (46.9%) were boys. All patients' characteristics data are presented in Table 4. The mean age of the children was 3.9 years (*SD* ± 1.2). Forty-seven (58.0%) children were examined three times, and others more often. The mean period of observation was 2.4 years. In this analyzed group, 38 (46.9%) children were examined three times over a period of 1 year. Fifty-seven (70.4%) children proceeded with flexible nasopharyngoscopy at least one time per year. Thirty-seven (45.7%) children were subsequently examined in a 2-year period, and 20 (24.7%) in a 3-year period.

Table 4. Characteristics of the group of patients undergoing long-term observation.

Characteristics		**All Patients**
n		81
Gender	female	43 (53.1%)
	male	38 (46.9%)
Age at the first visit (years)	mean ± *SD*	3.9 ± 1.2
	median (Q25–Q75)	3.5 (3.0–4.5)
Number of measurements	3	47 (58.0%)
	4	16 (19.8%)
	5	15 (18.5%)
	6	2 (2.5%)
	7	1 (1.2%)
Period of observation (years)	mean ± *SD*	2.4 ± 1.2
	median (Q25–Q75)	2.0 (1.5–3.0)
	1–3	63 (77.8%)
	3.5–5.5	18 (22.2%)
First visit	mean ± *SD*	65.4 ± 13.2
	median (Q25–Q75)	65.0 (55.0–75.0)
Bolesławska scale	<35	1 (1.2%)
	35–65	46 (56.8%)
	>65	34 (42.0%)
Last visit	mean ± *SD*	61.2 ± 16.1
	median (Q25–Q75)	60.0 (50.0–75.0)
Bolesławska scale	<35	4 (4.9%)
	35–65	46 (56.8%)
	>65	31 (38.3%)
Change; first vs. last visit	decrease	21 (25.9%)
	no change	17 (21.0%)
	increase	43 (53.1%)
	≤10	54 (66.7%)
	>10	27 (33.3%)
	≤15	61 (75.3%)
	>15	20 (24.7%)
Max	mean ± *SD*	71.0 ± 12.3
	median (Q25–Q75)	70.0 (60.0–80.0)
Min	mean ± *SD*	56.3 ± 14.6
	median (Q25–Q75)	55.0 (50.0–70.0)
Range	mean ± *SD*	14.7 ± 11.2
	median (Q25–Q75)	10.0 (5.0–20.0)
	≤10	42 (51.9%)
	>10	39 (48.1%)
	≤15	56 (69.1%)
	>15	25 (30.9%)
Semi-annual continuous measurements, 1 year	*n*	38 (46.9%)
Range	≤10	28 (73.7%)
	>10	10 (26.3%)
	≤15	35 (92.1%)
	>15	3 (7.9%)
Annual continuous measurements (at least one measurement per year)	*n*	57 (70.4%)
Range	≤10	34 (59.6%)

	>10	23 (40.4%)
	≤15	42 (73.7%)
	>15	15 (26.3%)
Two-year period	*n*	37 (45.7%)
Range	≤10	21 (56.8%)
	>10	16 (43.2%)
	≤15	29 (78.4%)
	>15	8 (21.6%)
Three-year period	*n*	20 (24.7%)
Range	≤10	7 (35.0%)
	>10	13 (65.0%)
	≤15	11 (55.0%)
	>15	9 (45.0%)

Only 7.9% of the children had a change in tonsil size of more than 15% on the A/C scale in 1 year of observation (Table 4). In the group of 37 children examined at least three times over a period of 2 years, eight (21.6%) of them had a change in the size of the tonsil above a 15% A/C ratio. In both groups of patients, less than half of the adenoids were changed more than 10% in A/C ratio scale (26.3% and 43.2%, respectively). From the group of 20 children with at least three endoscopic examinations over a period of 3 years, in nine (45.0%) of them, the A/C ratio changed above 15%.

4. Discussion

Our study showed that flexible endoscopic adenoid assessment is related to real adenoid size evaluated during its surgical removal; 97.3% sensitivity and 72.7% specificity of flexible endoscopic examination were obtained in the assessment of the size of the pharyngeal tonsils. Inaccuracy of the flexible adenoid assessment during the endoscopic examination may be caused by incomplete relaxation of the soft palate. In our experience, we recommend to the child to blow their nose ("blow the camera") when we reach the adenoid view. Discomfort with the examination caused the children to want to help us and remove the endoscope by trying to blow their nose. It causes soft palate relaxation and gives us the opportunity to accurately measure the adenoid size when the soft palate is relaxed. This makes the examination more reproducible. A comparison with the lateral x-rays should also be performed at the end of the inspiration, but in that case, it is really difficult to take a single picture at the right moment when we examine a frightened and noncooperating child [18]. Lateral cephalograms reached 61% to 75% sensitivity and 41% to 96% specificity [7,19]. A systematic review performed by Major suggested that lateral x-rays overestimated the size of the adenoid, and should be used for the measurement of the size of the airway rather than the adenoid size [19]. This technique is static and produces a two-dimensional summation picture [14,18,19]. On the other hand, the lateral cephalometric radiograph is a simple, inexpensive, and sufficiently informative diagnostic technique with a low radiation dose [14,18]. A comparative study performed by Mlynarek showed that, in contrast to FNE, lateral X-ray measurements, such as adenoid thickness or A/C ratio, did not correlate with obstructive symptom score [20]. From the other side, Caylakli, in a performed study, found a correlation between the achieved results of lateral x-rays and FNE [21]. Handelman sequentially analyzed 12 children, from 9 months of age to 18 years, using lateral cephalometric radiographs [15]. His study allowed describing the adenoid development in a single patient, and found the greatest obstruction of nasopharyngeal space during pre- and early school children. However, the effectiveness of surgical treatment presented in this work undermines the reliability of the radiological assessment of the tonsil itself. In 5 out of 10 operated children, the authors reported no complete removal of the tonsil in the radiological examination. This is much more than that described by other authors assessing the surgical effects of adenoidectomy

(e.g., with endoscopic examination) [22]. Moreover, it has also been proven that evaluation of adenoid size by cephalometric radiography is much less effective than CT, and what was mentioned above overestimated the adenoid size [19,23]. Unfortunately, CT and cone-beam CT have disadvantages, such as high radiation exposure and high cost, which may disqualify them from being performed repeatedly [24]. Analyses of CT examinations in 200 children of different ages divided into five subgroups were carried out by Cohen. He reported involution of adenoid tissue in a group of children between 5.1 and 8 years old. However, this is quite a long period when it comes to observing and deciding on the treatment of the patient [25]. In addition, the reduction of adenoid symptoms and increase of nasal air flow, which are commonly observed at about 7 years of age, may not be related to adenoid involution, but to the expansion of bony nasopharynx confirmed in lateral cephalometry [25–27]. Bergland reported an increase of 38% of nasopharyngeal space from 6 years of age to maturity [28]. A study performed by Papaioannou involving the analysis of the size of the pharyngeal tonsil in an MRI study of children of different ages showed that in children who do not snore, the size of the tonsil increases up to 7 to 8 years of age, and then it slowly decreases. In the group of children who snore (more than 1 night per week), the reduction of the tonsil occurred very slowly until 18 years of age [29]. On one hand, this study may indicate that such a slow process of tonsil involution occurs in children presenting to the ENT clinic; on the other hand, the discrepancy in results and curves in both groups—snorers and non-snorers—may derive from the analysis of a too-small group of snoring children: 33 compared with the bigger sample of 149 non-snoring children. It is particularly important in the performed study to analyze large groups of patients, because each patient's adenoid size was examined only once, and then it was compared with other age patients' adenoids to analyze the adenoid development from the perspective of years. It is known that in different children, the size of the tonsil is different, which has already been confirmed by Handelman and Pruzansky's report and numerous subsequent works [30]. Research on ultrasonography of the adenoids is promising, but is still not very common [31]. In contrast to the imaging techniques, invasive diagnostic tools not only provide information about anatomical structures of the nose and nasopharynx, but also visualize the functional state of the nasopharynx. Some of them show the color, mucous coverage, or inflammation characteristics. Invasive methods can cause discomfort and pain for the patient and require his or her collaboration. Otherwise, there is a need for the use of general anesthesia. Video fluoroscopy has good sensitivity—100%—and specificity of 90%, but it produces a 260 microsievert irradiation dose [7,19]. Flexible endoscopy seems to be the least traumatic of the invasive techniques and, as we have shown, in experienced hands, may be performed without anesthesia and gives plenty of information about adenoid state [32,33]. In the era of commonly performed COVID-19 tests, this examination is less painful than a nasal swab, in the opinion of patients.

Sensitivity and specificity of the adenoid size assessment during the surgery may also be burdened with an incomplete resection of the adenoid. Lesinskas reported 31.3% adenoid regrowth in children younger than 5 years old [34]. The reasons for the regrowth may be incomplete resection or persistent infections of the upper respiratory tract in postsurgical children's lives, asthma, gastroesophageal reflux (GERD), and allergic rhinitis. [34,35]. It is also dependent on the surgeon's experience and surgical technique [22,34]. Yildirim shows that blind curettage adenoidectomy may have left in 18% large residual adenoid. For precise resection of the adenoid, we controlled the nasopharynx with a mirror [22]. In our study, one patient (fewer than 1%) needed revision surgery because of adenoid regrowth (A/C ratio 70%) and concomitance hypoacusis caused by middle-ear effusion. This might be consistent with Dearking's observation that children with ear-related indications and obstructive adenoid symptoms were significantly likelier to require revision adenoidectomy [36]. The overall rate of revision adenoidectomy is estimated from 1.6% to 2.5% [35,37]. In the performed study, we obtained similar effectiveness of

treatments compared to other authors, which validates the proper surgical technique and precise adenoid removal.

This study verified the gold standard in adenoid examination by comparing the endoscopic adenoid assessment with the real size of the removed adenoid. Moreover, it shows that an adenoid measured in endoscopic examination as a 75% A/C ratio is assessed intraoperatively as a large adenoid.

Our research showed that in performing the first endoscopic examination and reporting an A/C ratio below 60%, we have about a 26% probability that within 1 year and 21% in a 2-year period, the tonsil will reach 75% A/C ratio and it will be assessed during the surgical procedure as large. In such children, an examination to assess the size of the tonsil should not be performed more frequently than 3 years if adenoid symptoms do not increase. In these children, conservative treatment of adenoid hypertrophy symptoms should be applied. However, in the group of children with a baseline tonsil of 60–70% in A/C ratio, this examination should be performed more often, every 2 years, if the adenoid hypertrophy symptoms do not intensify. In case of an increase in those symptoms, FNE should be performed every year. Children whose tonsil size exceeds 70% in A/C ratio should also be controlled every year. As mentioned above, there are many statements about the time of involution of the adenoid in children in the specialized literature, but there are few studies that confirm these theses [13–15,31]. Ishida performed sequential lateral cephalometric radiography in 90 children from 6 to 19 years old over a 10-year period of time. This performed study shows slow multiannual tendency of adenoid involution, but there was no decrease in adenoid size among groups of children in neighboring groups (for example: lower primary school, 8 years old, vs. upper primary school children, 10 years old) [14]. As mentioned earlier, lateral x-rays overestimated the size of the adenoid and should be used for the measurement of the size of the airway rather than the adenoid [19]. However, this study proves the tendency toward very slow adenoid involution. Yamada performed a similar retrospective study in Japan. He analyzed, in a 5-year period of time, a sample involving 99 individuals of the ages of 8–12 years. He stated that the adenoid-to-nasopharynx index decreased significantly through elementary school (in Japan, ages 6 to 12 years) [13]. However, the most interesting are the studies concerning children aged 3 to 8 years, because at these ages, adenoid symptoms seem to be the most burdensome for children. Wang analyzed the adenoid change in an ultrasound measurement of adenoid thickness in children aged 3 to 12 years and showed that the mean value of ultrasound measurements of adenoids in children aged 6 years was significantly greater than that of children of other ages [31]. We performed the first analysis of adenoid size change in periods of time in children 2.5 to 8 years old with the use of FNE. Based on our study, we propose a schema for the frequency of FNE for adenoid size assessment and adenoid hypertrophy treatment (Figure 3).

A limitation of this study was the selection of a group of patients undergoing long-term observation. After the diagnosis, parents were informed about the possibility of conservative or surgical treatment of adenoid hypertrophy in their children. Conservatively-treated children received a 12-week course treatment with mometasone furoate nasal spray and saline irrigation, which is a standard pharmacological treatment for adenoid hypertrophy symptoms, hoping for improvement [38]. We should add that our newest study did not reveal any change in adenoid size 3 to 6 months after finishing a 12-week course of intranasal steroid treatment, but in other studies, we showed seasonal variability of symptoms [33,39]. The need to wait for the adenoidectomy procedure in our country and, in some cases, the temporary (seasonal) relief of symptoms made such an observation possible until the procedure of adenoidectomy. In some cases, parents did not decide to undergo surgery because of symptom relief or concerns about surgical complications. This meant that, with the follow-up period, the number of patients decreased. The influence on the size of the sample is the fact that those groups were examined and also operated on by one children's ENT specialist (A.Z.) in the same ENT outpatient clinic and hospital.

On the other hand, it affects the repeatability of the tests performed by the same doctor using the same flexible endoscopic system and surgical technique.

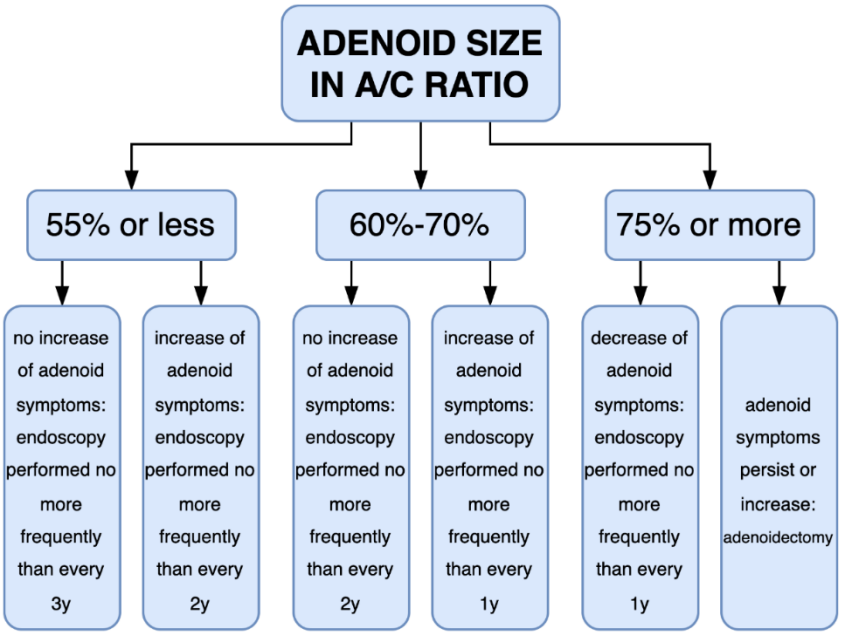

Figure 3. Schema of frequency of adenoid examinations and treatment.

5. Conclusions

FNE examination is highly effective in assessing the size of the pharyngeal tonsil—a high compliance of endoscopic examination with intraoperative assessment of the size of the tonsil has been demonstrated. In contrast to imaging tests, such as X-ray or ultrasound, this examination determines not only the size of the tonsil, but also its mucous coverage, edema, and inflammation status. Sensitivity and specificity of the flexible nasopharyngoscopy were calculated as 97.3% and 72.7%, respectively. We confirmed that a 75% A/C ratio or more is equivalent with an intraoperative large adenoid. Failure to remove a large adenoid during surgery in children who had a high A/C ratio before surgery may be a signal to the surgeon that he or she did not cut the whole tonsil, especially if he or she performed a blind curettage adenoidectomy. We proposed a schema for frequencies of FNE examinations and treatment depending on A/C ratio and worsening of ailments.

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The Impact of the Thermal Seasons on Adenoid Size, Its Mucus Coverage and Otitis Media with Effusion: A Cohort Study

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Abstract: Background: The purpose of this study is to analyze seasonal differences in adenoid size and related mucus levels via endoscopy, as well as to estimate changes in middle ear effusion via tympanometry. Methods: In 205 children with adenoid hypertrophy, endoscopic choanal assessment, adenoid hypertrophy assessment using the Bolesławska scale, and mucus coverage assessment using the MASNA scale were performed in two different thermal seasons, summer and winter. The study was conducted in two sequences of examination, summer to winter and winter to summer, constituting two separate groups. Additionally, in order to measure changes in middle ear effusion, tympanometry was performed. Results: Overall, 99 (48.29%) girls and 106 (51.71%) boys, age 2–12 (4.46 ± 1.56) were included in the study. The first group, examined in summer (S/W group), included 100 (48.78%) children, while the group first examined in winter (W/S group) contained 105 (51.22%) children. No significant relationship was observed between the respective degrees of adenoid hypertrophy as measures by the Bolesławska scale between the S/W and W/S groups in winter ($p = 0.817$) and in summer ($p = 0.432$). The degrees of mucus coverage of the adenoids using the MASNA scale and tympanograms were also comparable in summer ($p = 0.382$ and $p = 0.757$, respectively) and in winter ($p = 0.315$ and $p = 0.252$, respectively) between the S/W and W/S groups. In the total sample, analyses of the degrees of adenoid hypertrophy using the Bolesławska three-step scale for seasonality showed that patients analysed in the summer do not differ significantly when compared to patients analysed in the winter (4.39%/57.56%/38.05% vs. 4.88%/54.63%/40.49%, respectively; $p = 0.565$). In contrast, the amount of mucus on the adenoids increased in winter on the MASNA scale ($p = 0.000759$). In addition, the results of tympanometry showed deterioration of middle ear function in the winter ($p = 0.0000149$). Conclusions: The obtained results indicate that the thermal seasons did not influence the size of the pharyngeal tonsils. The increase and change in mucus coverage of the adenoids and deterioration of middle ear tympanometry in winter may be the cause of seasonal clinical deterioration in children, rather than tonsillar hypertrophy. The MASNA scale was found to be useful for comparing endoscopy results.

Keywords: adenoid hypertrophy; seasons; mucus on adenoid; OME; tympanometry

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1. Introduction

Adenoid hypertrophy and otitis media with effusion are one of the most common childhood disorders, and can cause various ailments of the upper respiratory tract. The prevalence of adenoid hypertrophy in preschool and primary school children is estimated to be 49.7% [1]. The adenoid tissue may cause otitis media with effusion due to supranormal size, disruption of nasopharyngeal ventilation, Eustachian tube obstruction, mucus accumulation, oedema, and upper respiratory tract infection [2]. Other ethological factors are craniofacial malformations, mechanical obstruction of the nasopharynx, allergies, and immunodeficiency [3]. OME is reportedly experienced by 30–40% of all children [3]. The

consequent obstruction of the upper respiratory tract causes mouth breathing, snoring, recurrent sinusitis, asthma, and sleep apnea, potentially leading to serious health damage including impaired development in children [1,4]. Quick and accurate diagnosis of the etiology can help in the proper treatment of obstructions [5]. Four or more episodes of recurrent purulent sinusitis, nasal obstruction, or otitis media with effusion in children aged four or older may indicate adenoid hypertrophy. There is a prevailing opinion that in cases of adenoid hypertrophy conservative treatment using topical steroids should be continued for at least two to three months; however, if there is no significant improvement, especially regarding sleep disturbances, adenoidectomy should be considered [6,7]. Various diagnostic methods can be used to assess the size of the pharyngeal tonsils, including nasopharyngeal lateral radiographs, computed tomography, videofluoroscopy, ultrasound, and mirror examination; the gold standard is flexible endoscopic examination [8–10]. Furthermore, a relationship between the size of the pharyngeal tonsils and the thermal seasons has been suggested, which may be based on the frequency of infections or allergy [11]. To the best of our knowledge and based on review of the literature in the PubMed database, there are no works analyzing adenoid size using flexible endoscopy in different thermal seasons, which prompted us to conduct this research.

In this study, we aimed to analyze changes in the size of the pharyngeal tonsils and their mucus depending on the thermal seasons by using endoscopic assessment. Furthermore, we evaluated changes in middle ear effusion by using tympanometry measurement. To better compare the population in this study with studies performed in other countries (often in different geographic zones with only two dominant seasons, winter and summer, such as the Mediterranean zone), we divided the year into two main seasons, winter and summer, considering the cutoff point for temperature to be 10 °C [12,13].

2. Materials and Methods

2.1. Study Population

205 children who visited a medical outpatient clinic with symptoms suggestive of chronic adenoid hypertrophy between 2016 and 2021 were included in the study. This was the first ENT consultation for those children; thus they were not given the standard conservative treatment for adenoid hypertrophy of a long course of antibiotics and steroids. Children with obstructive sleep apnea and upper airway obstruction, recurrent adenoid infection where two courses of antibiotics had failed, and four recurrent purulent rhinorrheas occurring in the preceding 12 months were included. Children with craniofacial anomalies, such as cleft lip/cleft palate, genetic diseases (Down Syndrome), neurological diseases, cardiovascular diseases, nasal septal deviation, nasal polyp or inferior turbinate hypertrophy, tympanic membrane perforation, and active upper respiratory infection within two weeks of enrolling in the study, or those who had previously undergone adenoidectomy or tympanostomy tube placement, were excluded from the study. We also excluded children with confirmed allergic rhinitis.

Each child was examined at least twice in two different seasons. The interval between the sequential seasonal examinations was 6 to 8 months. The summer examination occurred in the early summer, summer, and late summer (from 22 April to 12 October), when the average temperature in our region was above 10 °C, and the winter examination occurred in the early spring, spring, autumn, early winter, and winter (from 13 October to 21 April), when the average temperature was below 10 °C [11]. This study was a crossover group study, where approximately half of the children were first examined in winter and then in summer, and the remaining half were first examined in summer and then in winter. Initial assessment of each patient after enrollment in the study included a history and physical examination, parental questionnaire, flexible fiberoptic rhinoscopy, and tympanogram. After the first visit, patients received 12 weeks of conservative treatment with Mometasone Furoate nasal drops as a standard pharmacological treatment for adenoid hypertrophy [14,15]. Ethical approval for this study was obtained by the ethics committee of Nicolaus Copernicus University (KB 559/2021).

2.2. Endoscopy

Endoscopic examinations were performed by a pediatric otorhinolaryngologist (A.Z.) with 15 years of experience using the Karl Storz Tele Pack endoscopic system. This endoscopic system is equipped with a flexible nasopharyngoscope with a 2.8-mm outer diameter and 300-mm length. The percentage of obturation (adenoid-to-choanae ratio in percentage) of the choanae and mucus coverage of the adenoids were compared based on videoendoscopy with the freeze-frame option. Obstructions were assessed with an accuracy of up to 5%. Otoscopic examination was performed; if needed, the external auditory canal was cleaned, and tympanometry was performed using a GSI 39 Auto Tymp^{TM} by Grason-Stadler (Eden Prairie, MN, USA).

The percentage of choanal obstruction was measured and compared with the pretreatment value from the patient's medical history score. The adenoid size and mucus coverage recorded on the endoscopic system were compared with those in the previously recorded video by a second doctor (K.M). If there was a discrepancy in the assessment, the score was reassessed by a third ENT doctor (P.B).

Change in adenoid size was considered as the percentage difference in the amount of nasopharyngeal obstruction by the tonsil. For statistical analysis to assign the grade of adenoid hypertrophy we applied the classification described by Boleslavska: grade I, adenoid tissue filling less than one-third of the vertical portion of the choanae; grade II, adenoid tissue filling between one-third and two-thirds of the choanae; and grade III, adenoid tissue filling more than two-thirds of the choanae [16].

We devised a scale for assessing the mucus coverage of the adenoid, called the Mucus of Adenoid Scale by Nasopharyngoscopy Assessment (MASNA), which describes the amount of mucus covering the tonsil on a 4-point scale (0—no mucus, 1—residue of clear watery mucus, 2—some amount of dense mucus, 3—copious thick dense mucus) (Figure 1). The degree of seasonal change in adenoid mucus on the MASNA scale was compared for the total number of patients and between the designated groups. An increase in mucus coverage was represented by an increase in the degree of scale, while a decrease in the degree of scale implied reduced mucus coverage.

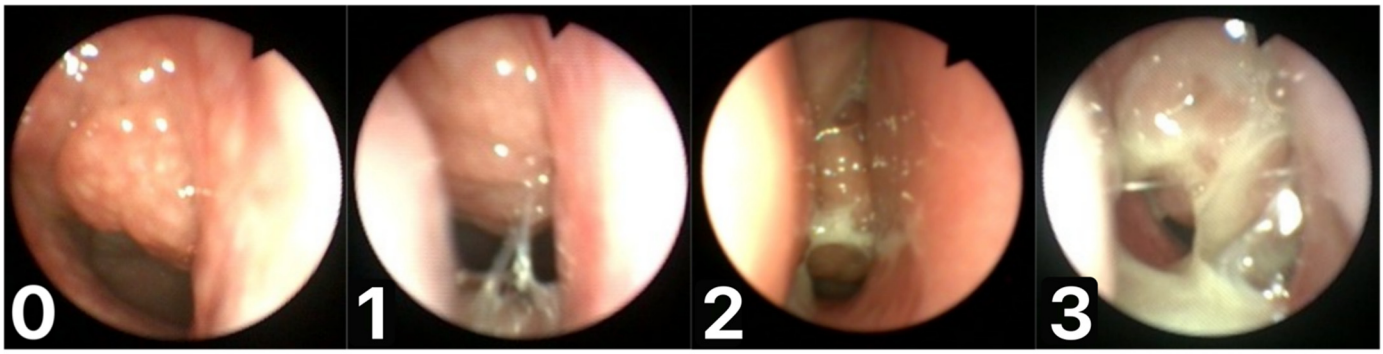

Figure 1. Mucus of Adenoid Scale by Nasopharyngoscopy Assessment (MASNA): (**0**)—no mucus; (**1**)—residue of clear watery mucus; (**2**)—some amount of dense mucus; (**3**)—copious thick dense mucus.

2.3. Tympanometry

The middle ear effusion in each ear was analyzed by tympanometry measurement and graphic tympanogram. The results were classified using the classification system for tympanograms developed by Liden and Jerger [17,18]. The sequence of saved tympanograms for each patient's ear was first right, then left. We posited a type B tympanogram as the worst result, type C as bad, and A as good (normal). For further statistical analysis, we divided children into three groups, taking into consideration worse tympanogram in each child. Group A consisted of children with tympanogram A in both ears (AA), group C of children with tympanogram C (CC, AC, CA), and group B of children with tympanogram B (BB, BC, CB, AB, BA).

2.4. Ethics

All recorded videos of the nasopharynx were coded and blindly analyzed.

2.5. Statistical Analysis

We used descriptive statistics to present the characteristics of the study groups. We summarized continuous variables such as age and adenoid size through their means ± standard deviation (SD) and medians with 25th and 75th percentiles (Q25–Q75), and categorical variables such as gender, mucus coverage in the MASNA scale, adenoid size in the Boleslavska scale, and tympanograms using frequency counts and percentages. This study analyzed whether the seasonal order of the study, that is, winter to summer examination or summer to winter examination, had an impact on the results. In order to determine the effect of the sequence of examination on the clinical results, we compared qualitative data using the chi-squared method or Fisher's exact test where appropriate, and quantitative data using Student's *t*-test for independents variables. In order to assess the impact of the thermal seasons on adenoid size, adenoid mucilage coverage, and tympanograms, we evaluated the statistical significance by analysis for dependent variables. Continuous variables were compared with Student's *t*-test test for paired samples. McNemar–Bowker tests were used for analysis of categorical variables.

For all these tests, two-tailed *p*-values were used and *p*-values < 0.05 were considered to be statistically significant. All statistical analyses were conducted with SPSS (Statistical Package for the Social Sciences version 26, Armonk, NY, USA) software.

3. Results

A total of 205 children with sequential seasonal ENT examinations were studied, 99 girls (48.29%) and 106 boys (51.71%), age 2–12 with a mean of4.46 ± 1.56 (Table 1). The study was conducted in two sequences of examination, summer to winter and vice versa, constituting two separate groups with comparable sample sizes. The group which was first examined in the summer (S/W group) included 100 children (48.78%), and group first examined in the winter (W/S group) included 105 children (51.22%).

For the next step of our analysis, we estimated associations between the sequence of examinations and demographical and clinical characteristics. These analyses were aimed at determining whether or not the obtained results depend on the season in which the patient's clinical analysis was started. As shown in Table 2, there were no statistically significant differences in the demographical and clinical characteristics between the S/W and W/S groups. Both groups were similar in terms of age and gender. In both groups the mean adenoid size was similar, 61.70% and 64.05%, respectively, in winter was and 60.70% and 63.57%, respectively, in summer, and did not differ statistically significantly ($p = 0.303$ and $p = 0.432$, respectively, for winter and summer). The groups were also similar in terms of mucus coverage of adenoids and tympanograms. The degree of mucus coverage of adenoids using the MASNA scale was comparable. Comparing winter examinations between the group first examined in summer and the group first examined in winter, we did not find statistically significant differences ($p = 0.315$). Comparing summer examinations, the group first examined in summer and the group first examined in winter were again similar, and we found no statistically significant differences between the groups ($p = 0.382$). Moreover, the type and frequency of occurrence of different types of tympanograms were similar. There were no statistical significance between the S/W and W/S groups in tympanometry performed in winter ($p = 0.254$) and in summer ($p = 0.757$) (Table 2).

Table 1. Patient characteristics.

Characteristic		All Patients
n		205
Age at first visit (years)	mean ± SD	4.46 ± 1.56
	median (Q25–Q75)	4.00 (3.00–5.00)
Gender	female	99 (48.29%)
	male	106 (51.71%)
Sequence of examination	summer –> winter (S/W)	100 (48.78%)
	winter –> summer (W/S)	105 (51.22%)
Summer adenoid size	mean ± SD	62.17 ± 15.20
	median (Q25–Q75)	60.00 (55.00–75.00)
	grade I	9 (4.39%)
	grade II	118 (57.56%)
	grade III	78 (38.05%)
Winter adenoid size	mean ± SD	62.90 ± 16.27
	median (Q25–Q75)	60.00 (50.00–75.00)
	grade I	10 (4.88%)
	grade II	112 (54.63%)
	grade III	83 (40.49%)
Seasonal change in adenoid size	mean ± SD	0.73 ± 8.74
	median (Q25–Q75)	0.00 (−5.00–5.00)
	decrease	56 (27.32%)
	no change	76 (37.07%)
	increase	73 (35.61%)
Summer adenoid mucus coverage (MASNA scale)	0	47 (22.93%)
	1	82 (40.00%)
	2	53 (25.85%)
	3	23 (11.22%)
Winter adenoid mucus coverage (MASNA scale)	0	25 (12.20%)
	1	67 (32.68%)
	2	65 (31.71%)
	3	48 (23.41%)
Seasonal change in adenoid mucus coverage	decrease	51 (24.88%)
	no change	57 (27.80%)
	increase	97 (47.32%)
Summer tympanogram	AA	129 (62.93%)
	AB/BA	11 (5.37%)
	AC/CA	15 (7.32%)
	BB	23 (11.22%)
	BC/CB	11 (5.37%)
	CC	16 (7.80%)
	A	129 (62.93%)
	B	45 (21.95%)
	C	31 (15.12%)
Winter tympanogram	AA	86 (41.95%)
	AB/BA	6 (2.93%)
	AC/CA	17 (8.29%)
	BB	55 (26.83%)
	BC/CB	15 (7.32%)
	CC	26 (12.68%)
	A	86 (41.95%)
	B	76 (37.07%)
	C	43 (20.98%)

Data are presented as frequency (%) unless otherwise indicated.

Table 2. Relationship between sequence of examination and clinical and demographic variables.

Characteristic		The Sequence of Examination		*p* Value
		Summer –> Winter (S/W)	Winter –> Summer (W/S)	
n		100 (48.78%)	105 (51.22%)	
Age at first visit (years)	mean ± SD median (Q25–Q75)	4.43 ± 1.54 4.00 (3.00–5.00)	4.49 ± 1.58 4.00 (3.00–5.00)	0.782
Gender	female male	45 (45.00%) 55 (55.00%)	54 (51.43%) 51 (48.57%)	0.357
Summer adenoid size	mean ± SD median (Q25–Q75)	60.70 ± 15.75 60.00 (50.00–72.50)	63.57 ± 14.59 60.00 (60.00–75.00)	0.177
	grade I grade II grade III	6 (6.00%) 59 (59.00%) 35 (35.00%)	3 (2.86%) 59 (56.19%) 43 (40.95%)	0.432
Winter adenoid size	mean ± SD median (Q25–Q75)	61.70 ± 16.18 60.00 (50.00–75.00)	64.05 ± 16.35 65.00 (55.00–80.00)	0.303
	grade I grade II grade III	6 (6.00%) 54 (54.00%) 40 (40.00%)	4 (3.81%) 58 (55.24%) 43 (40.95%)	0.817
Seasonal change in adenoid size	mean ± SD median (Q25–Q75)	1.00 ± 9.24 0.00 (−5.00–5.00)	0.48 ± 8.28 0.00 (−5.00–5.00)	0.669
	decrease no change increase	29 (29.00%) 35 (35.00%) 36 (36.00%)	27 (25.71%) 41 (39.05%) 37 (35.24%)	0.804
Summer adenoid mucus coverage (MASNA scale)	0 1 2 3	26 (26.00%) 42 (42.00%) 24 (24.00%) 8 (8.00%)	21 (20.00%) 40 (38.10%) 29 (27.62%) 15 (14.29%)	0.382
Winter adenoid mucus coverage (MASNA scale)	0 1 2 3	11 (11.00%) 38 (38.00%) 32 (32.00%) 19 (19.00%)	14 (13.33%) 29 (27.62%) 33 (31.43%) 29 (27.62%)	0.315
Seasonal change in adenoid mucus coverage	decrease no change increase	22 (22.00%) 30 (30.00%) 48 (48.00%)	29 (27.62%) 27 (25.71%) 49 (46.67%)	0.604
Summer tympanogram	AA AB/BA AC/CA BB BC/CB CC	61 (61.00%) 7 (7.00%) 8 (8.00%) 13 (13.00%) 5 (5.00%) 6 (6.00%)	68 (64.76%) 4 (3.81%) 7 (6.67%) 10 (9.52%) 6 (5.71%) 10 (9.52%)	0.757
	A B C	61 (61.00%) 25 (25.00%) 14 (14.00%)	68 (64.76%) 20 (19.05%) 17 (16.19%)	0.576
Winter tympanogram	AA AB/BA AC/CA BB BC/CB CC	49 (49.00%) 3 (3.00%) 8 (8.00%) 21 (21.00%) 5 (5.00%) 14 (14.00%)	37 (35.24%) 3 (2.86%) 9 (8.57%) 34 (32.38%) 10 (9.52%) 12 (11.43%)	0.254
	A B C	49 (49.00%) 29 (29.00%) 22 (22.00%)	37 (35.24%) 47 (44.76%) 21 (20.00%)	0.154

The primary analysis in this study focused on the association between thermal season and adenoid size, adenoid mucus coverage and tympanograms. We observed that in the summer first-degree of adenoid hypertrophy by the Bolesławska scale was present in 9 (4.39%) children, second-degree in 118 (57.56%) children, and third-degree in 78 (38.05%) children, whereas in winter we found first-degree adenoid hypertrophy in 10 (4.88%) children, second-degree in 112 (54.63%) children and third-degree in 83 (40.49%) children. In our series, there were no significant differences in the means of adenoid size or the Boleslavska scale between thermal seasons ($p = 0.232$, $p = 0.565$, respectively; Table 3). In contrast, when assessing the amount of mucus on the adenoids between thermal seasons, we observed deterioration in winter and an increase in the degree of mucus using our proposed MASNA scale. We found high statistical significance in seasonal dependence of mucus coverage of the adenoids ($p = 0.000759$). Middle ear effusion, confirmed by the results of tympanometry, also seems to be strongly associated with seasonality, as the number of incorrect tympanograms (type B or C) was significant higher in winter than in summer ($p = 0.0000149$) (Table 3).

Table 3. Impact of the thermal season on adenoid size, adenoid mucus coverage and tympanograms.

Characteristic		Thermal Season		*p* Value
		Summer	Winter	
Adenoid size	mean ± SD	62.17 ± 15.20	62.90 ± 16.27	0.232
	median (Q25–Q75)	60.00 (55.00–75.00)	60.00 (50.00–75.00)	
	grade I	9 (4.39%)	10 (4.88%)	0.565
	grade II	118 (57.56%)	112 (54.63%)	
	grade III	78 (38.05%)	83 (40.49%)	
Adenoid mucus coverage (MASNA scale)	0	47 (22.93%)	25 (12.20%)	0.000759
	1	82 (40.00%)	67 (32.68%)	
	2	53 (25.85%)	65 (31.71%)	
	3	23 (11.22%)	48 (23.41%)	
Tympanogram	AA	129 (62.93%)	86 (41.95%)	0.0000149
	AB/BA	11 (5.37%)	6 (2.93%)	
	AC/CA	15 (7.32%)	17 (8.29%)	
	BB	23 (11.22%)	55 (26.83%)	
	BC/CB	11 (5.37%)	15 (7.32%)	
	CC	16 (7.80%)	26 (12.68%)	
	A	129 (62.93%)	86 (41.95%)	0.00000323
	B	45 (21.95%)	76 (37.07%)	
	C	31 (15.12%)	43 (20.98%)	

4. Discussion

According to the medical experience of many ENT doctors, the number of upper respiratory infections in children decreases in summer. This has led to speculation that the size of the adenoids is also reduced in the summer.

Flexible endoscopy is the best method for assessing the adenoids in children, with a low rate of complications [8–10,19]. Unfortunately, it is not a routinely performed procedure in outpatients ENT clinics or hospitals because of its time-consuming nature, high cost, and the degree of experience required. However, it is possible to perform an assessment using a thin and flexible endoscope without anesthesia or premedication in more than 95% of children in age 2 to 12 years. It is essential to maintain contact with child patients, to allow play and to reward them. Parental collaboration and determination to obtain an objective diagnosis is also important, and pays off in correct diagnosis for oft-misdiagnosed patients and in a better possibility of using the most appropriate treatment.

Our study based on nasopharyngoscopy did not reveal any changes in adenoid size between the thermal seasons. There is no other study with which to compare results

on seasonal change of adenoids in children based on flexible endoscopy; however, there are some works analyzing seasonal polysomnography (PSG) for the diagnosis of sleep-disordered breathing (SDB) and indirect estimation of changes of adenoid hypertrophy in children. Greenfeld et al. conducted a study in Israel based on polysomnography which showed that the seasonal difference in adenoid size was particularly significant in children younger than five years of age [12]. They stated that enlarged tonsils play an important role in sleep-disordered breathing (SDB) in children. They also suggested that their findings may indirectly support the role of the seasonal viral burden as a major determinant in season-dependent changes in SDB. Nakayama et al. undertook a similar study in Japan, which showed changes in the severity of SDB and snoring in different seasons [20]. Frimer et al. conducted a study in Israel in which they performed PSG and analyzed the prevalence of obstructive sleep apnea (OSA) in children in different seasons. However, they did not find any significant differences [2]. As the study of sleep, polysomnography deals with the air flow through the nasopharynx, but not its anatomical and functional structure.

The present study based on flexible nasopharyngoscopy revealed high volatility of adenoid mucus by season. Mucus provides a barrier against viruses and bacteria, while chronic inflammation may destroy this barrier and induce contamination [21]. Inflammatory nasal secretion has an impact on the severity of upper respiratory tract diseases because of the ability of microorganisms present in the secretion to multiply rapidly and produce a biofilm, leading to bacterial resistance against the host's immune system. Biofilms most often form in humid and non-sterile environments. The multicellular bacterial structure surrounded by organic and nonorganic substances produced by the bacteria makes the biofilm cover porous surfaces, such as the tonsil [22,23]. The adenoid is a reservoir of microorganisms, including those with the ability to produce extracellular mucus [24,25]. Microorganisms from one or more species combine to form small colonies, which together constitute approximately 10% of the total volume of the biofilm [25]. The remainder includes extracellular polymeric substances (EPS), which keep the biofilm intact despite the adverse environmental conditions [23]. The glycocalyx is a particularly important substance in the EPS; it absorbs the necessary nutrients and promotes bacterial multiplication [24,25]. It is produced by bacteria such as *Pseudomonas* spp., *Moraxella* spp., or *Klebsiella* spp., which exist on the surface of the pharyngeal tonsil and can lead to the formation of non-sensitive bacterial colonies surrounded by thick mucus. Mucus-containing bacteria and viruses may contribute to the intensification of symptoms associated with adenoiditis [26]. Bellinghausen et al. state that pre-exposure of airway epithelial cells to pathological bacteria aggravates the production of proinflammatory cytokines in response to subsequent infections [27]. Clinical observations and clinical literature data confirm that the mechanical eradication of thick mucus by rinsing the nose with saline solution reduces the subjective and objective symptoms of adenoiditis [28].

In our opinion, the assumption of temperature dependence also undermines the theory of the influence of allergens and infections on the size of the pharyngeal tonsil, which corresponds with Greenfeld's results [12]. Mucus on the pharyngeal tonsils seems to play a main role in the context of the exacerbation of the ailments related to the presence of the adenoids. This was also suggested by Nakayama et al. concerning the seasonal exacerbation of OSA in children [20]. Individuals allergic to certain environmental factors may present with seasonal allergies, which may impact adenoid size. However, most clinicians disagree on whether or not allergic rhinitis impacts adenoid hypertrophy [22,23,29–33]. Modrzyński et al. and Sadeghi-Shabesteri et al. described the relationship between adenoid hypertrophy and allergy related to dust mite hypersensitivity [22,29]. Dogru et al. suggested that only children hypersensitive to fungi may have seasonal adenoid hypertrophy, while those allergic to *Alternaria alternata* and dust mites do not [23]. In contrast, Karaca et al. did not find any correlation between adenoid size and skin prick test results in children aged 5 to 14 years old, but concluded that allergic hypersensitivity may play an important role in children with tonsillar hypertrophy [30]. Furthermore, Eren et al. and Ameli et al. found a negative correlation between allergies and adenoid size in their studies [31,32]. The study

performed by Colavita et al. failed to demonstrate benefits of adenoidectomy in 80% of children with allergic rhinitis and hypertrophy, which seems to confirm the absence of or a negative correlation between allergies and adenoid size [33]. They concluded that only an endoscopic analysis of nasal secretions has significant predictive value for allergic rhinitis.

Our work based on performed tympanometry revealed seasonal tympanogram variability, and indicated seasonal change of otitis media with effusion (OME.) This is defined as an accumulation of fluid behind the intact tympanic membrane without signs and symptoms of acute ear infection in children, and seems to be strictly related to nasopharyngeal conditions such as chronic sinusitis and adenoid hypertrophy [34]. There are many postulated factors that may influenced OME, including age, allergies, breastfeeding, bottle feeding, presence of atopy or allergy, snoring, cough, more than five instances of tonsilitis in twelve months, presence of pets, attending a daycare center, passive smoking, number of siblings, family income, and seasonality [34–36]. For many years both otoscopy and tympanometry have been the gold standard for diagnosis of otitis media with effusion (OME). In 1981, Holm-Jensen showed great variations in repetitive tympanometry in four-year-old children when performed from winter to spring, which was connected with climate conditions [37]. In a continuation of this work based on tympanometry performed in 288 children, Mirko Tos found a significant increase in the frequency of otitis secretory and a significant deterioration in tympanometric condition in the winter [38]. For many years, OME was related with seasons [35]. In contrast to previously performed studies, Knopke in 2015 showed no significant seasonal difference between the winter and the summer period in intraoperative middle ear examination in connection with preoperative tympanograms [39].

This study has strongly demonstrated association between seasonality and OME as well as the co-occurrence of OME and increased amounts of nasopharyngeal mucus. The amount of mucus significantly changes between seasons. For describing mucus on the adenoid surface, we devised a scale called MASNA, which is useful for comparing the results of endoscopic examinations [40]. It may be speculated that it is difficult to compare the mucus coverage of adenoid in children, as some may blow their nose directly before the endoscopy and others may do not. Our experience shows that it is difficult for a child to eliminate deep-seated mucus from the nose during nasopharyngoscopy. This observation corresponds with parental opinion that it is impossible for their children to blow their nose despite the impression that the nose is blocked. One of the most useful methods for removing mucus is rinsing the nose with saline solution [41,42], which should thus be avoided prior to endoscopy.

5. Conclusions

The obtained results indicate that the thermal seasons do not influence the size of the pharyngeal tonsils. The increase and change in mucus coverage of the adenoids and deterioration of middle ear tympanometry in winter may be the cause of seasonal clinical deterioration in children instead of tonsillar hypertrophy. The proposed MASNA scale is useful for describing mucous coverage on the adenoids and for comparing endoscopy results. Flexible endoscopy is recommended and could be used more often for assessing the state of the adenoids in children. Further studies on the impact of the thermal seasons on adenoid size and mucus coverage, performed in different countries, are advisable.

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Impact of Isolation on Adenoid Size and Symptoms in Preschool Children Who Previously Qualified for Adenoidectomy: A Case–Control Study

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Abstract

Objectives: To analyze the role of factors that influence adenoid-related symptoms, and the influence of 3 months of isolation in preschool children who were qualified for adenoidectomy. **Methods:** This was a cohort study of the impact of 3 months of isolation on children aged 3 to 6 years with adenoid-related symptoms and endoscopically confirmed grade II and III adenoid hypertrophy. The children had previously qualified for adenoidectomy. After 3 months, 141 children were asked about their symptoms, 71 of whom were randomly chosen for medical examination, including endoscopic adenoid examination. Additionally, pre- and post-isolation tympanometry results were analyzed. **Results:** In our study, significant or mild improvements in health were observed in approximately 73% of the children; 92% of the surveyed parents reported that their children exhibited improved nasal patency, 63% of children discontinued snoring, and 30% of children showed a decrease in snoring. Data collected through surveys correlated with endoscopic findings wherein the size of the adenoid decreased by an average of 5.4%, but the amount of mucus covering the adenoid decreased more significantly in 76% of patients, which may be the main problem determining symptoms reported before isolation. In addition, the amount of residual mucus in the nasopharynx significantly affected the middle ear effusion in adenoid hypertrophy. **Conclusions and Relevance:** This study found that isolation in preschool children stabilizes the bacterial microbiome of the nose and nasopharynx, thereby having a significant effect not only on the number of recurrent infections of the upper respiratory tract but also on the patency of the nose. The only symptom that depended on the size of the pharyngeal tonsil hypertrophy was snoring. There was a weak correlation between prevalence of infections and adenoid size. During the isolation period, the adenoid size reduced by approximately 5.4% only, statistically more significant in the group with grade III adenoid hypertrophy, but the prevalence of infections rapidly decreased. Residual mucous in the nasopharynx found to be in correlation with middle ear effusion.

Keywords

adenoid hypertrophy, adenoiditis, otitis media with effusion, OME

Introduction

In 1661, Conrad Victor Schneider first discovered and described a prominent nasopharyngeal structure as follows: "a whitish color, the adjoining membranes being bloody or dusky. It is fuller than they and like fat. It is always moist and exudes a glutinous substance."[1] Hans Wilhelm Meyer coined the name "adenoid" in 1868. Many physicians began to study the role of this nasopharynx structure in the pathogenesis of upper respiratory tract diseases in children. This commenced an era of the diagnosis of overgrown tonsils and development of surgical removal techniques as a treatment for snoring and sleep disturbances, mouth breathing, gasping, open mouth

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posture, dry mouth, halitosis, hyponasal speech, and nasal obstruction with or without rhinorrhea in young children. Additionally, in 1842, James Yearsley found a relationship between pharyngeal tonsil hypertrophy and otitis.[2]

Since then, the adenoid has also been considered a source of mucus and runny nose. This secretion also contains commensal and pathological microbes. The concept of commensalism as a biological association between 2 species was theorized in the 1860s by Pierre-Joseph Van Beneden.[3] A few decades later, nasal swab bacteriology cultivation and analysis was introduced. The important role of biofilms, organized structures of microorganisms in human pathogenesis, has been known and investigated since the 1980s. In 2003, Matthew Parsec proposed criteria for biofilm infection in the respiratory tract and indicated its importance in recurrent upper respiratory tract diseases in children.[4,5] James Coticchia indicated the role of biofilms covering the adenoid in pediatric chronic rhinosinusitis (CRS), which led to adenoidectomy as a treatment for pediatric CRS.[6]

Currently, 4 or more incidences of recurrent purulent rhinorrhea, nasal obstruction, or otitis media with effusion in children $\geq$4 years lead to the suspicion of adenoiditis.[7] Indications for treatment may be established based on medical history and physical examination, which confirm adenoid hypertrophy. The appropriate diagnosis should be based on a flexible endoscopic examination, and not on radiographic assessment.[8] Adenoidectomy is recommended in cases of persistent symptoms despite pharmacological therapy and sleep disturbances with nasal airway obstruction persisting for at least 3 months. However, there remains a question: Is adenoid hypertrophy an indication for adenoidectomy?

Many doctors observe a decrease in the rate of upper respiratory infections if parents attempt to isolate their preschool children, keeping them at home and not sending them to kindergarten. However, until now, there has been no relevant study or opportunity to allow all children to remain at home. Is it not a problem of civilization and its related excessive social contacts that do not allow for maturation of the immune system and excessive stimulation of Waldeyer's ring? Alternatively, is there no stability of the adenoid microbiome?

The coronavirus disease (COVID-19) pandemic forced the governments of many countries to order citizens to remain at home. In Poland, the period of isolation was 3 months. We analyzed the symptoms and ailments in isolated preschool children who were qualified for adenoidectomy before the pandemic.

Patients and Methods

We analyzed 141 preschool children aged 3 to 6 years who would qualify for adenoidectomy based on the reported ailments. All children had recurrent purulent rhinorrhea: 4 or more incidents of nasal blockage, snoring, and mouth breathing in the previous 12 months. There were no improvements after 3 months of treatment. Flexible endoscopic examinations were performed by one pediatric otorhinolaryngologist doctor with 15 years of experience using the Karl Storz Tele Pack endoscopic system before isolation. All recorded videos of the nasopharynx, acquired while maintaining the endoscope in a stable position until the child relaxed the soft palate, were coded and blindly analyzed by a second doctor to assign the grade of adenoid hypertrophy based on the classification described by Boleslavská et al[9]: grade I, adenoid tissue filling less than one-third of the vertical portion of the choanae; grade II, adenoid tissue filling between one-third and two-thirds of the choanae; and grade III, adenoid tissue filling more than two-thirds of the choanae.

Table 1. Endoscopically Controlled Random Group of Patients.

		Number of patients (n = 71)		%	
Adenoid size change before and after isolation					
Increase		11		15.4	
The same		24		33.8	
Decrease		36		50.7	
Amount of mucous before and after isolation					
Increase		2		2.8	
The same		15		21.1	
Decrease		54		76	
Tympanometry before and after isolation					
Improved		47		66.2	
The same	Normal	19	23	26.8	32.4
	Pathological	4		5.6	
Deterioration		1		1.4	

We excluded children with a grade I adenoid size. The children were divided into 2 groups depending on the degree of adenoid hypertrophy: II and III. After 3 months of isolation, the parents were asked to complete a survey concerning previously reported and unresolved ailments, and comparisons of the symptoms were made.

Additionally, a total of 71 randomly selected children from both groups were medically examined, and resolution of adenoiditis was endoscopically confirmed after 3 months of isolation. The size of the adenoid recorded on the Tele Pack endoscopic system was randomly compared with that in a previously recorded video by a second experienced doctor. The percentages of obturation of the choanae were compared based on video endoscopy with the freeze frame option. Mucous coverage of the adenoid was also analyzed and categorized among 4 stages: (1) no mucus, (2) remnants of mucus, (3) some mucus, and (4) abundant thick mucus.

During each visit, we performed tympanometry using the Grason-Stadler GSI 39 screening device and related the results with those of tympanometry performed before isolation (Table 1).

Statistical Analysis

The purpose of this study was to assess the effect of adenoid size on the incidence of ailments: infections, nasal patency, and

Table 2. Statistical Analysis of the Surveys.

Improving the health of the child	Grade II adenoid hypertrophy	Grade III adenoid hypertrophy	χ^2	*P*
Significant	31 (41.3%)	26 (39.4%)	5.888	.117
Mild	23 (30.7%)	22 (33.3%)		
Small	13 (17.3%)	17 (25.8%)		
Lack	8 (10.7%)	1 (1.5%)		
Number of infections			χ^2	*P*
Less	62 (82.7%)	59 (89.4%)	1.305	.253
Unchanged	13 (17.3%)	7 (10.6%)		
Improving nasal patency			χ^2	*P*
Yes	70 (93.3%)	60 (90.9%)	0.287	.592
No	5 (6.7%)	6 (9.1%)		
Snoring			χ^2	*P*
Yes, the same level	4 (5.3%)	16 (18.2%)	8.115	**.017**
Yes, but less	22 (29.3%)	20 (36.4%)		
No (snoring has resolved)	49 (65.4%)	30 (45.4%)		

snoring. The H0 was no difference, and we attempted to verify this hypothesis using Pearson χ^2 test at the significance level $\alpha = .05$. Based on the responses to the survey, the empirical values of the χ^2 test and the corresponding *P* values were calculated. The obtained values allowed for the inference to accept or reject H0.

Results

In both groups of children with different levels of adenoid hypertrophy maintained at 3 months of isolation, significant or mild improvements in health were observed in approximately 73% of the children. No statistical significance ($P = .117$) was observed in either group. The number of infections decreased by 83% and 90% in the grade II and III hypertrophy groups, respectively ($P = .253$). More than 90% of children in both groups showed improvements in nasal patency during this period of isolation, and 65% of the children in group I and 45% of the children in the second group stopped snoring, while it decreased in approximately 30% of children in both groups. Snoring remained at the same level in 18% of children in the grade III hypertrophy group, which was statistically significant ($P = .017$; Table 2).

Almost all calculated *P* values for the test characteristics (questions asked) exceeded the critical value of the assumed level of significance α; thus, we could not justify rejecting H0 "that the size of the adenoid does not determine symptoms," with one exception wherein the size of the adenoid determined snoring.

In our study, 92% of the surveyed parents reported that their children exhibited improved nasal patency, 63% of children discontinued snoring, and 30% of children showed a decrease in snoring. The size of the adenoid in the endoscopic controlled group decreased by an average of 5.4%, and almost did not change in the group with grade II adenoid hypertrophy (average decrease of 0.4%) and decreased in the group with grade III adenoid hypertrophy by an average of 10.2%, which was statistically significant ($P = .001$; Table 3). The distribution of the number of children in the appropriate groups of adenoid hypertrophy depending on the change in adenoid size is shown in Figure 1. The amount of mucus covering the adenoid decreased more significantly in 76% of patients (Table 1, Figure 2). During the control examination, 66 (93%) patients had a tympanogram on the same normal level or improved. In one case, deterioration was observed, and a negative result persisted in 4 cases.

Table 3. Comparison of Group of Children Qualified to Selected Category of Adenoid Size Change Depending of Previously Stated Grade Adenoid Hypertrophy.

Change of the adenoid size before and after isolation	Grade II adenoid hypertrophy, n = 36	Grade III adenoid hypertrophy, n = 35	χ^2	*P*
$\leq -20\%$	3	7	20.57	**.001**
-15%	1	5		
-10%	5	9		
-5%	1	5		
0	15	9		
>5%	11	0		

Discussion

COVID-19 isolation in Poland was a special situation not previously experienced, in which one of the factors of infection was deactivated: contact with other children and contaminations. Other parameters such as allergies and adenoid hypertrophy persisted. Although the use of FFP2 or FFP3 masks might have helped prevent allergic reactions at the beginning of lockdown, at that time masks were scarce. Additionally, in Poland, young children rarely wear masks. The authors acknowledge potential seasonal limitations to this research. Although the study started in winter, the children were assessed during spring although we intended to examine patients immediately after isolation. Vila et al analyzed patients' medical examinations for Eustachian tube dysfunction or otitis media with effusion and compared the results between adults and children. Among children (2.6 billion ENT visits) 24% of visits were in winter and 27% were in spring,[10] suggesting there were no significant seasonal changes. Frimer et al also noted that, in children, the frequency of moderate and severe obstructive sleep apnea (OSA) did not vary significantly by season.[11] Similarly, we found that adenoid size exhibited no seasonal variations. Lastly, the time between the first and second examinations was too short (3 months) to consider the potential influence of age-related adenoid atrophy.

Isolation remarkably influenced children's health in both the patient groups—confirmed grades II and III adenoid hypertrophy. It may have influenced the prevalence of infection, which declined by 86%. The percentage of children with a good nasal

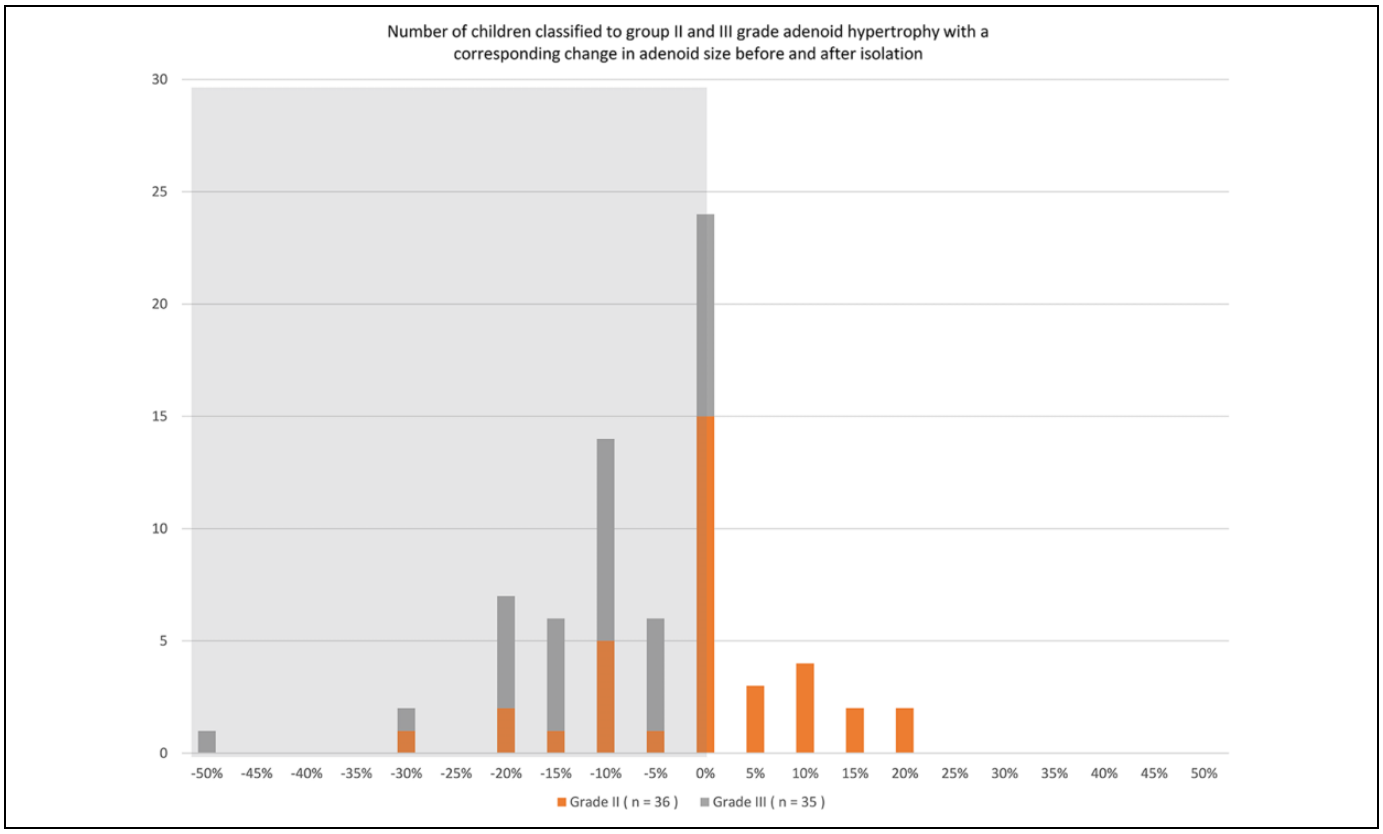

Figure 1. The distribution of the number of children in the appropriate groups of adenoid hypertrophies.

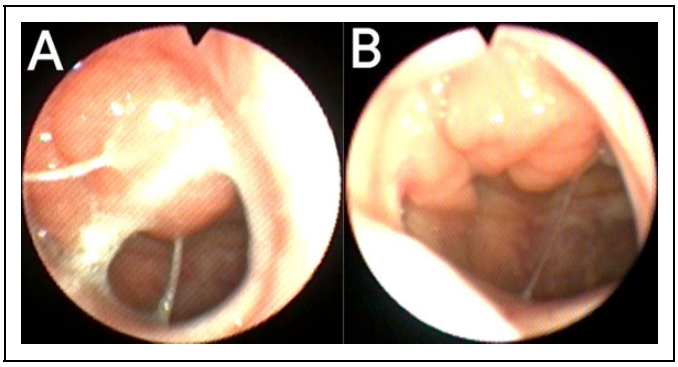

Figure 2. Endoscopy of the same patient—amount of mucous in the nasopharynx. (A) before, (B) after isolation.

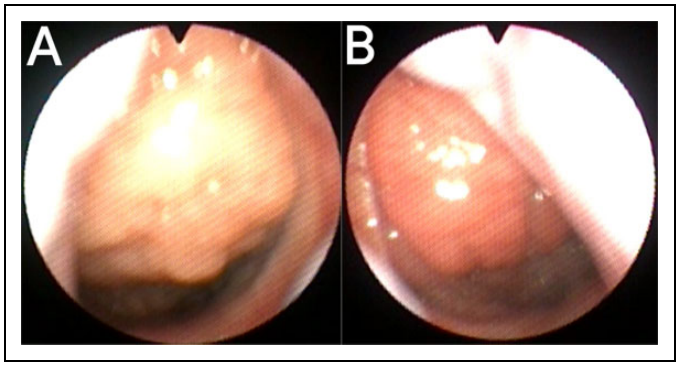

Figure 3. Endoscopy of the same patient with adenoid hypertrophy: (A) left side before isolation, (B) right side after isolation.

patency and no snoring improved to 93.3% and 90.9%, respectively. Our study showed a slight effect of infection on the size of the adenoid. A lower rate of infection caused a decrease in the adenoid size by an average of 5.4% (Figure 3). In addition, the tissue of the adenoid was less edematous and slimy.

The mucosa of the nose, nasopharynx, and adenoid is colonized by commensal microbiota after birth[12] and affects the innate mucosal immune response. Moreover, many potentially pathogenic microbes are often cultivated from swabs obtained from healthy patients' adenoids.[13] The potentially pathogenic microbes that are found on the adenoid include Neisseria, *Staphylococcus aureus*, *Haemophilus influenza*, *Streptococcus sp*, Actinomyces, Bacteroides, Prevotella, Peptostreptococcus, or Fusobacterium. The role of early *Helicobacter pylori* infection in chronic adenoiditis has also been reported.

Analyzing the microbiome based on swabs obtained from the tonsils is difficult because different bacteria occupy different localizations on the surface of the tonsils. *Haemophilus influenzae* infiltrates tissues, whereas Bacteroides and Streptococcus are located deep in its folds.[14] Furthermore, Stępińska et al stated that *H influenzae* and *S aureus* are intracellular.[15] Analysis of the microbiome on the removed tonsil is not fully objective, because at the time of treatment, the children are healthy and pathogenic microbes disappear.[16] This leads to the

conclusion that the tonsil microbiome is difficult to assess and is continuously changing. Stabilization of the microbiome is the most important aspect of children's health. The isolation of patients provides supportive evidence for this. During the 3 months of isolation, not only did the frequency of infection among children decrease but the patency of the nose also improved. This may have been due to the abatement of chronic inflammation of the adenoid, caused by a reduction in the number of bacteria. Slow changes allow young patients to obtain an optimal acquired immunity.

The biofilm on the adenoid leads to recurrent inflammation of the nasal cavity and throat in children. Numerous studies have suggested the need to remove the biofilm, as it is a bacterial reservoir contributing to recurrent nasal mucosa inflammation.[5,6,17] The structure of the microbiome is an important factor in the recurrence of infections, and its modification with isolated bacterial strain supplementation results in an improvement in patients.[18] Three months of isolation of children not only led to a decrease in upper respiratory tract infections but also improved nasal patency, regardless of the grade of the tonsil hypertrophy. In our study, snoring remained the only symptom dependent on the size of the pharyngeal tonsil. Moreover, the important role of biofilm is also postulated in the pathogenesis of otitis media with effusion, wherein apposition of the overgrown tonsil seems to significantly impair the patency of the Eustachian tube. This study showed that the severity of effusion in the middle ear depends on the amount of mucus on the adenoid and in the nasopharynx and not on the adenoid size. Despite a significant improvement in the child's health in the assessment by parents, 7% of children maintained an abnormal tympanogram. Aldè et al found that 933% of tympanograms normalized during the 2-month lockdown in Italy.[19] This indicates that this group should be periodically followed up.

In the last few decades, overgrowth of the adenoid has been relatively easy to assess macroscopically in terms of both palpation and radiological diagnosis, but it did not always provide a complete picture of the pathological condition. Only endoscopic examinations have allowed us to assess both the adenoid size and the surface covered with mucus, its density, and its color, which seem to be more important.

This study revealed that isolation of preschool children, resulting in stabilization of the bacterial microbiome of the nose and nasopharynx, has a significant effect not only on the number of recurrent infections of the upper respiratory tract but also on the patency of the nose. The only symptom that depended on the size of the pharyngeal tonsil hypertrophy was snoring. There was a weak correlation between the prevalence of infections and adenoid size. In the isolation time, the adenoid size reduced by approximately 5.4% only, statistically more significant in the group with grade III adenoid hypertrophy, and the prevalence of infections rapidly decreased. Residual mucous in the nasopharynx found to be in correlation with middle ear effusion.

We analyzed the effects of lockdown (isolation) on adenoid hypertrophy, OSA, and other middle ear diseases in children. Such issues are very important in the COVID-19 era, when governments often order deferments of elective surgeries and the conversion of pediatric ENT departments to COVID-19 wards. Knowledge about influence of isolation on adenoid and related diseases is important and should be based objective assessments of the risk of worsening illness. This is the first study to examine these issues based on endoscopic examinations of preschool-aged children conducted pre- and post-lockdown for COVID-19.

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Supplemental Material

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Impact of Breastfeeding Duration on Adenoid Hypertrophy, Snoring and Acute Otitis Media: A Case-Control Study in Preschool Children

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Abstract: Background: The aim of this study was to analyze the relationship between breastfeeding duration and adenoid size, snoring and acute otitis media (AOM). Methods: We analyzed the medical history of children admitted to the ENT outpatient clinic in 2022 and 2023, reported symptoms, ear, nose and throat (ENT) examination, and flexible nasopharyngoscopy examination of 145 children aged 3–5 years. Results: Breastfeeding duration of 3 and 6 months or more had a significant effect on the reduction of snoring ($p = 0.021$; $p = 0.039$). However, it had no effect on the adenoid size, mucus coverage and sleeping with an open mouth. Snoring was correlated with open mouth sleeping ($p < 0.001$), adenoid size with a 75% A/C ratio or more ($p < 0.001$), and adenoid mucus coverage in the Mucus of Adenoid Scale by Nasopharyngoscopy Assessment—MASNA scale ($p = 0.009$). Children who were breastfed for less than 3 months had more than a four-fold greater risk of snoring. There was a statistically significant correlation between AOM and gender ($p = 0.033$), breastfeeding duration in groups fed 1, 3 or 6 months or more ($p = 0.018$; $p = 0.004$; $p = 0.004$) and those fed with mother's breast milk 3 or 6 months or more ($p = 0.009$; $p = 0.010$). Moreover, a correlation was found between adenoid size and mucus coverage, tympanogram, and open-mouth sleeping ($p < 0.001$). Independent factors of snoring in 3- to 5-year-old children were breastfeeding duration of less than 3 months ($p = 0.032$), adenoid size with an A/C ratio of 75% or more ($p = 0.023$) and open mouth sleeping ($p = 0.001$). Conclusions: Children breastfed for 3 and 6 months or more exhibited reduced rates of snoring. There was no effect of breastfeeding duration on adenoid size in children aged 3 to 5 years, suggesting that the link between breastfeeding duration and snoring is primarily associated with craniofacial development and muscle tone stimulation. A breastfeeding duration of 1 month or more plays a key role in reducing the rate of AOM. The mother's milk plays a protective role against AOM. The presence of mucus might be responsible for snoring in preschool children. A medical history of breastfeeding should be taken into consideration when snoring children are suspected of adenoid hypertrophy.

Keywords: adenoid hypertrophy; breastfeeding; snoring; open mouth breathing; acute otitis media

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1. Introduction

The World Health Organization (WHO) strongly advocates breastfeeding for infants, recommending it from birth until at least six months due to its remarkable impact on bolstering the infant's immune system and promoting optimal craniofacial development [1,2]. Furthermore, the United Nations International Children's Emergency Fund (UNICEF)

recommends the introduction of complementary foods alongside breastfeeding from 6 to 24 months [1].

The quality and composition of breast milk exhibit variations depending on the postpartum period. Colostrum, produced from birth to the 7th day, transitions into transitional milk from the 7th to the 15th day, ultimately giving way to mature milk four weeks postpartum [3,4]. Initially abundant in immune components, colostrum undergoes a decrease, stabilizing as breast milk matures [5]. Simultaneously, the milk's composition, inclusive of minerals, vitamins, hormones, enzymes, nucleic acids, polysaccharides, lipids, breast-derived cells, blood-derived cells, and extracellular vesicles, undergoes alterations [4–6]. Consequently, the duration of breastfeeding can impact a child's susceptibility to infectious diseases, allergies, or immune disorders in the future [7].

Breast milk encompasses various bioactive components, with a pivotal role attributed to sIgA immunoglobulins. These immunoglobulins safeguard the infant's intestinal barrier, reduce the risk of upper and lower respiratory tract infections, and mitigate inflammation [8]. This immunoprotective effect is believed to be mediated through the passive transfer of maternal immune components during breastfeeding. Furthermore, breast milk is instrumental in shaping the upper respiratory tract microbiome and fostering greater alpha diversity [9]. Breast-derived microbiota (BDM) contribute to the development of offspring microbiota through mucosal colonization, influencing the maturation of the immune system [10]. Notably, significant variations in the microbiome exist between infants fed mother's milk and those fed formula [11,12].

Breastfeeding contributes significantly to the proper development of the oropharyngeal apparatus, including the peristaltic movement of the tongue during suckling, which in turn aids in the development and coordination of the oropharyngeal muscles engaged in swallowing [13]. Additionally, it diminishes the risk of malocclusion disorders in children and supports the healthy formation of the palate [14–16]. Importantly, scientific evidence has underscored a connection between the duration of breastfeeding and the subsequent occurrences of habitual snoring and apnea in preschool and early school-aged children. Habitual snoring is observed in 9% of preschool children, with adenoid hypertrophy being the predominant causative factor [17,18]. The aim of this study is to pursue and elucidate the possible association between the duration of breastfeeding, snoring, adenoid size and acute otitis media (AOM). Specifically, it explores whether sustained exposure to immune components in breast milk has an influence on adenoid growth and mucus coverage, or if the link between breastfeeding duration and snoring is primarily attributable to craniofacial development and muscle tone stimulation.

2. Materials and Methods

Study Population: The study encompassed 145 children (60 girls and 85 boys) aged 3–5 years, presenting with symptoms suggestive of adenoid hypertrophy, who were evaluated at an ear, nose, and throat (ENT) outpatient clinic in 2022 and 2023. Detailed assessments included allergy history, symptoms linked to adenoid hypertrophy (e.g., snoring, mouth breathing), stated by parents' hypoacusis, frequency of upper respiratory tract infections, and the presence and severity of a runny nose. Snoring was defined in accordance with criteria based on the International Classification of Sleep Disorders (ICSD-3) [19]. The children were categorized into six groups based on breastfeeding duration: up to 3 month, 3 to 6 months, 6 to 12 months, 12 to 18 months, 18 to 24 months, and over 24 months. To determine the critical duration of breastfeeding, various analyses were conducted on the entire cohort of children, involving the subdivision into two subgroups with variable cut-off points: below and above 1 month, up to 3 months and over 3 months, up to 6 months and over 6 months, up to 12 months and over 12 months, up to 24 months and over 24 months.

Additionally, we analyzed children according to the same categorized periods of mother's milk feeding, both from the breast and the bottle. The groups were analyzed according to adenoid size, mucus coverage and related symptoms, such as snoring and sleeping with an open mouth. Additionally, a history of AOM and the results of tym-

panometry examinations were recorded and compared with the breastfeeding period. Exclusion criteria included recent and prolonged treatment for adenoid hypertrophy with nasal steroid spray, craniofacial anomalies, genetic disorders (e.g., Down syndrome), nasal septal deviation, nasal polyps, inferior turbinate hypertrophy, and active upper respiratory infections. The patient characteristics and reported symptoms are presented in Table 1.

Table 1. Characteristics of the study group divided into a 6-month breastfeeding period.

Characteristic		All Patients (n = 145)	Breastfeeding Groups		p Value
			≥6 Months (n = 100)	<6 Months (n = 45)	
Age (years)	mean ± SD	3.9 ± 0.8	3.9 ± 0.8	3.9 ± 0.8	0.981
Gender	female	60 (41.4%)	41 (41.0%)	19 (42.2%)	0.890
	male	85 (58.6%)	59 (59.0%)	26 (57.8%)	
Breastfeeding (months)	mean ± SD	13.0 ± 11.2			
	0–3	26 (17.9%)	-	-	-
	3–6	14 (9.7%)			
	6–12	31 (21.4%)			
	12–18	21 (14.5%)			
	18–24	16 (11.0%)			
	>24	37 (25.5%)			
Adenoid size (A/C ratio, %)	mean ± SD	65.3 ± 18.8	64.4 ± 19.0	67.3 ± 18.5	
	<75	90 (62.1%)	66 (66.0%)	24 (53.3%)	0.146
	≥75	55 (37.9%)	34 (34.0%)	21 (46.7%)	
Adenoid mucus coverage (MASNA scale)	0	40 (27.6%)	27 (27.0%)	13 (28.9%)	0.615
	1	36 (24.8%)	28 (28.0%)	8 (17.8%)	
	2	40 (27.6%)	26 (26.0%)	14 (31.1%)	
	3	29 (20.0%)	19 (19.0%)	10 (22.2%)	
	0	40 (27.6%)	27 (27.0%)	13 (28.9%)	0.814
	1–3	105 (72.4%)	73 (73.0%)	32 (71.1%)	
Tympanogram	AA	65 (44.8%)	48 (48.0%)	17 (37.8%)	0.602
	AB/BA	6 (4.1%)	5 (5.0%)	1 (2.2%)	
	AC/CA	15 (10.3%)	10 (10.0%)	5 (11.1%)	
	BB	34 (23.4%)	23 (23.0%)	11 (24.4%)	
	BC/CB	6 (4.1%)	4 (4.0%)	2 (4.4%)	
	CC	19 (13.1%)	10 (10.0%)	9 (20.0%)	
	A	65 (44.8%)	48 (48.0%)	17 (37.8%)	0.306
	B	46 (31.7%)	32 (32.0%)	14 (31.1%)	
	C	34 (23.4%)	20 (20.0%)	14 (31.1%)	
Allergy	yes	24 (16.6%)	14 (14.0%)	10 (22.2%)	0.137
	no	25 (17.2%)	21 (21.0%)	4 (8.9%)	
	not tested	96 (66.2%)	65 (65.0%)	31 (68.9%)	
Snoring	yes	106 (73.1%)	68 (68.0%)	38 (84.4%)	**0.039**
	no	39 (26.9%)	32 (32.0%)	7 (15.6%)	
Sleeping with an open mouth	yes	72 (49.7%)	53 (53.0%)	19 (42.2%)	0.394
	periodic	50 (34.5%)	31 (31.0%)	19 (42.2%)	
	no	23 (15.9%)	16 (16.0%)	7 (15.6%)	
Hypoacusis (stated by parents)	yes	35 (24.3%)	25 (25.3%)	10 (22.2%)	0.579
	periodic	25 (17.4%)	15 (15.2%)	10 (22.2%)	
	no	84 (58.3%)	59 (59.6%)	25 (55.6%)	
History of acute otitis media	yes	84 (57.9)	50 (50.0%)	11 (24.4%)	**0.004**
	no	61 (42.1%)	50 (50.0%)	34 (75.6%)	

Table 1. *Cont.*

Characteristic		All Patients (n = 145)	Breastfeeding Groups		p Value
			≥6 Months (n = 100)	<6 Months (n = 45)	
rURTIs	yes no	120 (82.8%) 25 (17.2%)	83 (83.0%) 17 (17.0%)	37 (82.2%) 8 (17.8%)	0.909
Rhinitis (weeks in a month)	mean ± SD	1.8 ± 1.0	1.9 ± 1.0	1.6 ± 0.9	0.072
Cough	yes no	48 (33.1%) 97 (66.9%)	37 (37.0%) 63 (63.0%)	11 (24.4%) 34 (75.6%)	0.237
Blocked nose	yes no	11 (7.6%) 134 (92.4%)	7 (7.0%) 93 (93.0%)	4 (8.9%) 41 (91.1%)	0.739

A/C ratio: adenoid-to-choana ratio, rURTIs: recurrent upper respiratory tract infections.

Endoscopy: Flexible endoscopic examinations of the nasopharynx were conducted on each child using a common nasal meatus. These examinations were carried out by a pediatric otorhinolaryngologist (AZ) using the Karl Storz Tele Pack endoscopic system, which was equipped with a flexible nasopharyngoscopy tool (2.8 mm outer diameter, 300 mm length). The assessment included choanal obstruction percentages (adenoid-to-choanae ratio—A/C ratio) and mucus coverage of the adenoids based on flexible nasopharyngoscopy. Choanal obstructions were assessed with up to 5% accuracy. To assess adenoid size in this study, we divided children into two groups: those with an adenoid size less than 75% in the A/C ratio, and those with 75% or more in the A/C ratio in the flexible nasopharyngoscopy assessment. This categorization was derived from our intraoperative comparisons of adenoid size with preoperative endoscopic adenoid assessments, indicating that a 75% A/C ratio or more was equivalent to a surgically removed large adenoid [20], which was also confirmed in other clinicians' opinions and studies [21,22]. Videos of the nasopharynx were coded and analyzed blindly by the second ENT doctor (KM). Furthermore, the Mucus of Adenoid Scale by Nasopharyngoscopy Assessment (MASNA) was utilized to quantify adenoid mucus coverage, employing a four-point scale (0, no mucus; 1, residue of clear watery mucus; 2, some amount of dense mucus; 3, copious thick dense mucus) [23]. In cases of assessment discrepancies, a third ENT doctor (PB) reassessed the score.

Tympanometry: The study involved otoscopic examinations and external auditory canal cleaning when required. Tympanometry was carried out using the GSI 39 AutoTymp TM by Grason-Stadler. Effusion in the middle ear was assessed through tympanometry measurements and tympanogram graphs. The results were classified using the Liden and Jerger classification system for tympanograms [24,25]. Tympanograms were saved for each patient's right and left ears, but to simplify the score, we divided them into three groups in accordance with the worst tympanogram in both ears. We adopted type-B tympanograms to consider the worst, type-C worst, and type-A indicative of normal function.

Statistical Analysis: Descriptive statistics were used to summarize variables within the study group. Quantitative variables were presented as means ± standard deviation (SD), while categorical variables were summarized using frequency counts and percentages. Statistical significance was determined using the Chi-square method or Fisher's exact test for categorical variables and Student's t-test or one-way ANOVA for quantitative variables to assess differences between independent variables.

Variables significantly related to snoring in the univariate analysis were included in the logistic regression analysis to identify independent prognostic factors useful in assessing snoring. Prediction of snoring was assessed via two separate analyses: using (1) breastfeeding < 3 months and (2) breastfeeding < 6 months for the logistic regression models. Odds ratios (OR) and 95% confidence intervals (95% CIs) were also calculated for considered clinical variables in regression models. For all these tests, two-tailed p-values were used, and differences at the level of $p < 0.05$ were considered significant. All statistical

analyses were performed with SPSS (Statistical Package for the Social Sciences, version 28, Armonk, NY, USA) software.

Ethics: Ethical approval for this study was obtained from the ethics committee of Nicolaus Copernicus University (KB 141/2022).

3. Results

The study comprised 145 children with an average age of 3.9 years, a mean breastfeeding duration of 13 months, and an average adenoid size corresponding to a 65.3% A/C ratio (Table 1). The duration of breastfeeding 6 months or more had a significant effect on the reduction of snoring and incidence of AOM ($p = 0.039$ and $p = 0.004$, respectively). However, it had no effect on the adenoid size, mucus coverage, nasal blockage, sleeping with an open mouth, frequency of recurrent upper respiratory tract infection, rhinitis, cough, hypoacusis and tympanogram (Table 1).

To assess the factors that impact snoring within the study sample, a comparison was made between children who snored and those who did not (Table 2). There was no significant difference between age, gender, allergy history, hypoacusis, recurrent upper respiratory tract infections, rhinitis, cough, or nasal blockage. However, a statistically significant difference in snoring rates was observed based on breastfeeding duration. Children breastfed for 3 months, 6 months or more exhibited reduced rates of snoring ($p = 0.021$ and $p = 0.039$, respectively). Also, children fed with mother's milk for 3 months or more snored less ($p = 0.014$). Snoring was also correlated with open mouth sleeping ($p < 0.001$), adenoid size with an A/C ratio of 75% or more ($p < 0.001$), and adenoid mucus coverage with the presence of any mucus (MASNA 1 to 3 degrees) ($p = 0.009$) (Table 2). Children with an adenoid size with an A/C ratio of 75% or more and mucus coverage on the adenoid snore more often. No relationship was observed between snoring and allergy, AOM, recurrent respiratory tract infections, rhinitis, nose blockage, cough, hypoacusis and tympanogram.

Table 2. The relationship of snoring and the occurrence of acute otitis media with clinical and demographic data in children aged 3–5 years with suspected adenoid hypertrophy.

Characteristic		History of Acute Otitis Media		*p* Value	Snoring		*p* Value
		Yes (*n* = 84)	No (*n* = 61)		Yes (*n* = 106)	No (*n* = 39)	
Age (years)	mean ± SD	4.0 ± 0.8	3.8 ± 0.9	0.345	4.0 ± 0.8	3.8 ± 0.8	0.211
Gender	female male	41 (48.8%) 43 (51.2%)	19 (31.1%) 42 (68.9%)	**0.033**	46 (43.4%) 60 (56.6%)	14 (35.9%) 25 (64.1%)	0.416
Breastfeeding (months)	mean ± SD	11.8 ± 11.5	14.8 ± 10.5	0.107	12.5 ± 11.3	14.6 ± 10.7	0.318
	≥1 <1	66 (78.6%) 18 (21.4%)	57 (93.4%) 4 (6.6%)	**0.018**	87 (82.1%) 19 (17.9%)	36 (92.3%) 3 (7.7%)	0.128
	≥3 <3	59 (70.2%) 25 (29.8%)	55 (90.2%) 6 (9.8%)	**0.004**	78 (73.6%) 28 (26.4%)	36 (92.3%) 3 (7.7%)	**0.021**
	≥6 <6	50 (59.5%) 34 (40.5%)	50 (82.0%) 11 (18.0%)	**0.004**	68 (64.2%) 38 (35.8%)	32 (82.1%) 7 (17.9%)	**0.039**
	≥12 <12	38 (45.2%) 46 (54.8%)	33 (54.1%) 28 (45.9%)	0.292	49 (46.2%) 57 (53.8%)	22 (56.4%) 17 (43.6%)	0.277
	≥24 <24	18 (21.4%) 66 (78.6%)	19 (31.1%) 42 (68.9%)	0.185	26 (24.5%) 80 (75.5%)	11 (28.2%) 28 (71.8%)	0.652
Mother's milk feeding	mean ± SD	12.3 ± 11.2	15.0 ± 10.3	0.139	12.9 ± 11.0	14.9 ± 10.5	0.329
	≥1 <1	71 (84.5%) 13 (15.5%)	58 (95.1%) 3 (4.9%)	0.060	87 (82.1%) 19 (17.9%)	36 (92.3%) 3 (7.7%)	0.236
	≥3 <3	63 (75.0%) 21 (25.0%)	56 (91.8%) 5 (8.2%)	**0.009**	78 (73.6%) 28 (26.4%)	36 (92.3%) 3 (7.7%)	**0.014**

Table 2. *Cont.*

Characteristic		History of Acute Otitis Media		*p* Value	Snoring		*p* Value
		Yes (*n* = 84)	No (*n* = 61)		Yes (*n* = 106)	No (*n* = 39)	
Mother's milk feeding	mean ± SD	12.3 ± 11.2	15.0 ± 10.3	0.139	12.9 ± 11.0	14.9 ± 10.5	0.329
	≥6 <6	54 (64.3%) 30 (35.7%)	51 (83.6%) 10 (16.4%)	**0.010**	68 (64.2%) 38 (35.8%)	32 (82.1%) 7 (17.9%)	0.059
	≥12 <12	40 (47.6%) 44 (52.4%)	34 (55.7%) 27 (44.3%)	0.334	49 (46.2%) 57 (53.8%)	22 (56.4%) 17 (43.6%)	0.246
	≥24 <24	18 (21.4%) 66 (78.6%)	19 (31.1%) 42 (68.9%)	0.185	26 (24.5%) 80 (75.5%)	11 (28.2%) 28 (71.8%)	0.652
Adenoid size (A/C ratio, %)	mean ± SD	66.6 ± 19.0	63.4 ± 18.6	0.320	68.2 ± 18.7	57.4 ± 17.2	**0.002**
	<75 ≥75	47 (56.0%) 37 (44.0%)	43 (70.5%) 18 (29.5%)	0.075	56 (52.8%) 50 (47.2%)	34 (87.2%) 5 (12.8%)	**<0.001**
Adenoid mucus coverage (MASNA scale)	0 1 2 3	22 (26.2%) 22 (26.2%) 22 (26.2%) 18 (21.4%)	18 (29.5%) 14 (23.0%) 18 (29.5%) 11 (18.0%)	0.888	23 (21.7%) 29 (27.4%) 30 (28.3%) 24 (22.6%)	17 (43.6%) 7 (17.9%) 10 (25.6%) 5 (12.8%)	0.060
	0 1–3	22 (26.2%) 62 (73.8%)	18 (29.5%) 43 (70.5%)	0.659	23 (21.7%) 83 (78.3%)	17 (43.6%) 22 (56.4%)	**0.009**
Tympanogram	AA AB/BA AC/CA BB BC/CB CC	29 (34.5%) 3 (3.6%) 9 (10.7%) 25 (29.8%) 5 (6.0%) 13 (15.5%)	36 (59.0%) 3 (4.9%) 6 (9.8%) 9 (14.8%) 1 (1.6%) 6 (9.8%)	0.054	50 (47.2%) 3 (2.8%) 11 (10.4%) 26 (24.5%) 3 (2.8%) 13 (12.3%)	15 (38.5%) 3 (7.7%) 4 (10.3%) 8 (20.5%) 3 (7.7%) 6 (15.4%)	0.486
	A B C	29 (34.5%) 33 (39.3%) 22 (26.2%)	36 (59.0%) 13 (21.3%) 12 (19.7%)	**0.011**	50 (47.2%) 32 (30.2%) 24 (22.6%)	15 (38.5%) 14 (35.9%) 10 (25.6%)	0.643
Allergy	yes no not tested	15 (17.9%) 11 (13.1%) 58 (69.0%)	9 (14.8%) 14 (23.0%) 38 (62.3%)	0.295	17 (16.0%) 21 (19.8%) 68 (64.2%)	7 (17.9%) 4 (10.3%) 28 (71.8%)	0.402
Sleeping with an open mouth	yes periodic no	44 (52.4%) 28 (33.3%) 12 (14.3%)	28 (45.9%) 22 (36.1%) 11 (18.0%)	0.709	61 (57.5%) 37 (34.9%) 8 (7.5%)	11 (28.2%) 13 (33.3%) 15 (38.5%)	**<0.001**
Hypoacusis (stated by parents)	yes periodic no	24 (28.9%) 14 (16.9%) 45 (54.2%)	11 (18.0%) 11 (18.0%) 39 (63.9%)	0.315	26 (24.8%) 17 (16.2%) 62 (59.0%)	9 (23.1%) 8 (20.5%) 22 (56.4%)	0.830
Snoring	yes no	65 (77.4%) 19 (22.6%)	41 (67.2%) 20 (32.8%)	0.173	- -	- -	-
History of acute otitis media	yes no	- -	- -	-	41 (38.7%) 65 (61.3%)	20 (51.3%) 19 (48.7%)	0.173
rURTIs	yes no	72 (85.7%) 12 (14.3%)	48 (78.7%) 13 (21.3%)	0.269	91 (85.8%) 15 (14.2%)	29 (74.4%) 10 (25.6%)	0.104
Rhinitis (weeks in a month)	mean ± SD	1.8 ± 0.9	1.8 ± 1.1	0.711	1.9 ± 0.9	1.6 ± 1.1	0.176
Cough	yes no	30 (35.7%) 54 (64.3%)	18 (29.5%) 43 (70.5%)	0.433	35 (33.0%) 71 (67.0%)	13 (33.3%) 26 (66.7%)	0.972
Blocked nose	yes no	7 (8.3%) 77 (91.7%)	4 (6.6%) 57 (93.4%)	0.761	9 (8.5%) 97 (91.5%)	2 (5.1%) 37 (94.9%)	0.498

Further relationships between the history of AOM and other factors were analyzed (Table 2). We found a statistically significant correlation between AOM and gender ($p = 0.033$). The girls became sick more often. Breastfeeding duration, especially in groups fed 1, 3 or 6 months and more, impacted the recurrence of AOM ($p = 0.018$, $p = 0.004$ and $p = 0.004$, respectively). One month of breastfeeding was enough to reduce the amount of AOM. Also, in the groups of children fed with breast milk for 3 months, 6 months or more, a statistically significant difference in the incidence of AOM was shown ($p = 0.009$ and $p = 0.010$, respectively). A high correlation was also found depending on the depending on the type A tympanogram ($p = 0.003$). Tympanometry, indicating any presence of fluid in the middle ear, fostered the occurrence of AOM. There was no stated correlation between the history of AOM and adenoid size, adenoid mucus coverage, rhinitis, or upper respiratory tract infection, as observed by parents' hypoacusis, cough, or snoring.

In the next step, correlations between the adenoid size and previously analyzed clinical data were analyzed. In addition to the previously presented correlations between adenoid size and snoring and the lack of correlation with breastfeeding duration, a statistically significant correlation was found between adenoid size and mucus coverage, tympanogram or open mouth sleeping (Table 3). In all of these correlation cases, the calculated p-value was <0.001. Adenoid hypertrophy of 75% and more was conducive to greater adenoid mucus coverage, C or B tympanogram, and sleeping with an open mouth. Additionally, a relationship was detected between adenoid size and mucus coverage on the MASNA scale, whereas larger adenoids more frequently displayed pathological mucus coverage. The presence of effusion in the middle ear was also linked to bigger adenoid size (Table 3). Moreover, it was determined that adenoid mucus coverage influenced snoring and tympanometry results.

Table 3. Relationship of adenoid size and demographic or clinical factors in children aged 3–5 years with suspected adenoid hypertrophy.

Characteristic		Adenoid Size (A/C Ratio)		*p* Value
		n	Mean ± SD	
Gender	female	60	67.5 ± 18.0	0.234
	male	85	63.7 ± 19.4	
Breastfeeding (months)	0–3	31	66.0 ± 21.1	0.753
	3–6	14	70.4 ± 10.6	
	6–12	29	61.6 ± 20.0	
	12–18	18	63.1 ± 20.4	
	18–24	16	67.5 ± 17.3	
	>24	37	65.8 ± 18.8	
Mother's milk feeding	0–3	26	66.5 ± 19.4	0.742
	3–6	14	70.4 ± 10.6	
	6–12	31	62.1 ± 19.6	
	12–18	21	62.4 ± 23.1	
	18–24	16	67.5 ± 17.3	
	>24	37	65.8 ± 18.8	
Adenoid mucus coverage (MASNA scale)	0	40	53.5 ± 19.9	**<0.001**
	1	36	65.3 ± 19.6	
	2	40	70.1 ± 14.5	
	3	29	74.8 ± 13.3	
	0	40	53.5 ± 19.9	**<0.001**
	1–3	105	69.8 ± 16.4	
Tympanogram	AA	65	58.8 ± 20.6	**0.010**
	AB/BA	6	75.8 ± 12.8	
	AC/CA	15	67.7 ± 13.7	
	BB	34	70.1 ± 18.7	

Table 3. *Cont.*

Characteristic		Adenoid Size (A/C Ratio)		*p* Value
		n	Mean ± SD	
Tympanogram	BC/CB	6	72.5 ± 12.1	**0.010**
	CC	19	71.1 ± 13.0	
	A	65	58.8 ± 20.6	**<0.001**
	B	46	71.2 ± 17.2	
	C	34	69.6 ± 13.2	
Allergy	yes	24	64.4 ± 19.3	0.904
	no	25	64.2 ± 17.7	
	not tested	96	65.8 ± 19.2	
Sleeping with an open mouth	yes	72	71.9 ± 15.7	**<0.001**
	periodic	50	60.2 ± 19.8	
	no	23	55.4 ± 18.8	
Hypoacusis (stated by parents)	yes	35	69.1 ± 18.0	0.283
	periodic	25	61.8 ± 22.8	
	no	84	64.3 ± 17.7	
Snoring	yes	106	68.2 ± 18.7	**0.002**
	no	39	57.4 ± 17.2	
History of acute otitis media	yes	84	66.6 ± 19.0	0.320
	no	61	63.4 ± 18.6	
rURTIs	yes	120	66.3 ± 18.1	0.156
	no	25	60.4 ± 21.7	
Cough	yes	48	64.8 ± 17.1	0.829
	no	97	65.5 ± 19.7	
Blocked nose	yes	11	72.7 ± 13.3	0.173
	no	134	64.7 ± 19.1	

Finally, we assessed the importance of breastfeeding in the prediction of snoring using logistic regression analysis. Regression analysis model using a 3-month breastfeeding cut-off point showed that a breastfeeding duration of less than 3 months ($p = 0.032$), adenoid size with an A/C ratio of 75% or more ($p = 0.023$) and open mouth sleeping ($p = 0.001$) were independent factors of snoring in children 3 to 5 years old (Table 4). The obtained estimates indicate that children who were breastfed for less than 3 months had more than a 4-fold greater risk of snoring than those who were breastfed for more than 3 months (OR = 4.33, 95% CI = 1.14–16.50). Analyzing the model with a 6-month breastfeeding cut-off point, sleeping with an open mouth ($p < 0.001$) and adenoid size of 75% or more ($p = 0.030$) were shown as significant factors of snoring.

Table 4. Logistic regression analysis for the prediction of snoring in children aged 3–5 years.

Characteristic	*p* Value	OR	95% CI
Model including breastfeeding with a 3-month cut-off point			
Sleeping with an open mouth, yes	**0.001**	**2.57**	**1.46–4.51**
Adenoid mucus coverage (MASNA scale), 1–3	0.197	1.83	0.73–4.60
Adenoid size, A/C ratio ≥ 75%	**0.023**	**3.59**	**1.19–10.83**
Breastfeeding, <3 months	**0.032**	**4.33**	**1.14–16.50**
Model including breastfeeding with a 6-month cut-off point			
Sleeping with an open mouth, yes	**<0.001**	**2.71**	**1.52–4.84**
Adenoid mucus coverage (MASNA scale), 1–3	0.240	1.73	0.69–4.29
Adenoid size, A/C ratio ≥ 75%	**0.030**	**3.37**	**1.13–10.09**
Breastfeeding, <6 months	0.055	2.66	0.98–7.24

4. Discussion

The study did not show an effect of breastfeeding duration on the adenoid size in children aged 3–5 years. No other studies in the available literature database have analyzed this relationship.

However, the study affirmed the beneficial effect of breastfeeding for 3 and 6 months or more in reducing snoring rates among children aged 3 to 5 years. This finding aligns with previous studies by Brew et al. indicating that breastfeeding for over one month is associated with a reduced risk of parent-reported habitual snoring [26]. Moreover, breastfeeding for longer than 3 months was linked to a significant decrease in the risk of witnessed sleep apnea. Beebe et al. and Montgomery-Downs et al. also support this observation, suggesting that breastfeeding may confer protection against sleep-disordered breathing [17,27]. One proposed hypothesis centers on the immunological protection of maternal milk, which reduces early childhood infections and inhibits lymphoid tissue proliferation. At ages 3 to 5, adenoids represent the most developed lymphoid tissue within Waldeyer's ring [20]. However, this study did not validate a correlation between breastfeeding duration and adenoid size as measured via flexible nasofiberscopy. Moreover, no such relationship emerged when considering the duration of breastfeeding. Multivariate analysis indicated that breastfeeding duration and adenoid size independently contributed to snoring. This raises questions regarding the second hypothesis postulated by Montgomery-Downs, suggesting that the link between breastfeeding duration and snoring might be primarily associated with craniofacial development and muscle tone stimulation [27,28].

Our study has shown the beneficial effects of breastfeeding for more than one month on reducing the risk of AOM in preschool children. This was already reported in the literature and meta-analysis performed by other authors [29–33]. The population studies in Canada by Karunanayake et al. indicated that breastfeeding for more than 3 months protects against ear infection. We have also shown a beneficial effect of feeding the baby with breast milk for more than 3 months on reducing the incidence of AOM. This confirms the suggested effect of immune modulation, epigenetic changes, and colonization of beneficial microbiota supplemented with mother's milk [32,34]. Moreover, mothers' milk has a bacteriostatic effect and contains lactoperoxidase, which destroys pathogenic AOM bacteria [32]. The positive effect of a short breastfeeding period (one month in our study) on the reduction of the incidence of AOM supports the protective role of mother's milk against AOM rather than the breastfeeding process itself, influencing the development of muscle tone of the oropharyngeal apparatus and improving Eustachian tube function. Perhaps this period is sufficient to shape baby's immunological system by mother's milk, which is then exceptionally rich in immune components [5].

The incidence of AOM correlated with tympanometry results, indicating effusion in the middle ear confirmed by a type C or B tympanogram. This clearly confirmed that effusion in the middle ear promotes the growth of pathogens and increases the risk of otitis media [35].

One noteworthy revelation in this study was the identification of a potential link between adenoid mucus coverage and snoring in preschool children. The presence of mucus is responsible for constriction of the nose and nasopharynx, increasing upper airway resistance and influencing snoring. Consequently, snoring in preschool children may be misinterpreted as a symptom of adenoid hypertrophy, even though it can be substantially caused by mucus covering the adenoid. Nonetheless, appropriate treatments such as anti-allergic medications, nasal steroids, and nasal lavage can potentially mitigate this effect. Research by Masna et al. emphasized that other factors, such as the thermal season, can also impact adenoid mucus [23]. This observation was also stated by Wang et al., who showed seasonal dependence of snoring problems related to nasal blockage in the general population [36]. A reduction in nasal resistance plays an important role in reducing snoring [37]. This was corroborated by Värendh et al., who identified nasal symptoms as independent risk factors for snoring [38].

Our study did not show a relationship between the length of breastfeeding and open mouth sleeping. This is opposite to the results of a meta-analysis conducted by Savian et al., indicating a possible protective effect against the occurrence of mouth breathing [39]. A study by Lopes et al. indicated that an increased duration of breastfeeding increases the likelihood of developing a normal breathing pattern in 2.5- to 4-year-old children [40]. Similar positive results of breastfeeding for more than 3 months in similar groups of preschool children were shown by Travitzki [41]. Similar to our findings, Ieto did not find that feeding correlated with predominant breathing patterns [42].

In addition, we confirmed the reports that other authors have already shown regarding the relationship between adenoid hypertrophy and the presence of effusion in the middle ear confirmed by tympanometry examination or adenoid size and sleeping with the mouth open [43–47]. The high convergence of the obtained results and the resulting correlations with previous studies by other authors indicate that the studied group is representative. This increases the credibility of our results, particularly the lack of correlation between breastfeeding periods and adenoid hypertrophy.

Finally, it has been shown that the breastfeeding period and adenoid hypertrophy are independent factors influencing the snoring of preschool children. Therefore, a snoring child breastfed under 3 months may be misdiagnosed due to adenoid hypertrophy. A medical history of breastfeeding should be taken into consideration when snoring children are suspected of adenoid hypertrophy.

The study's robustness lies in the considerable size of the studied children's group, substantial congruence with previous studies by other researchers, and a pioneering attempt to explore the correlation between breastfeeding duration and adenoid size assessed with optimal objectivity, utilizing flexible endoscopy. It is plausible that further analyses on larger patient cohorts would afford a clearer determination of the significance level for the observed relationships, particularly in cases where p-values hover near 0.05.

5. Conclusions

This study underscored the positive impact of breastfeeding for a minimum of 3 months in reducing snoring rates and preventing open month breathing among children aged 3 to 5 years. However, it did not establish any effect of breastfeeding duration on adenoid size. This reinforces the hypothesis that the favorable effect of breastfeeding on snoring is related to its influence on proper facial skeletal development, occlusion, and muscle tension in the mouth and throat. Moreover, it was found that breastfeeding duration of 3 months or more and adenoid hypertrophy with a 75% A/C ratio or more are independent factors of snoring in preschool children. Breastfeeding duration should be taken into account when, based on symptoms such as snoring, the doctor makes a diagnosis of suspected adenoid hypertrophy. The presence of mucus might be responsible for snoring in preschool children. Breastfeeding duration of 1 month or more plays a key role in reducing the rate of AOM. The nutrients of a mother's milk play a protective role against AOM.

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Abbreviations

FNE	flexible nasopharyngoscopy examination
A/C ratio	adenoid-to-choanae ratio
MASNA	mucus of adenoid scale by nasopharyngoscopy assessment
rURTI	recurrent upper respiratory tract infections
ICSD-3	International Classification of Sleep Disorders
AOM	acute otitis media
BDM	breast-derived microbiota

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Siblings' Risk of Adenoid Hypertrophy: A Cohort Study in Children

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Abstract: Background: The aim of this study was to compare adenoid size in preschool-age siblings using flexible nasopharyngoscopy examination (FNE) when they reach the same age. The occurrence of adenoid symptoms in these patients was also analyzed. This study was conducted to analyze the adenoid size in siblings when they reach the same age and substantiate a correlation between adenoid hypertrophy (AH) and adenoid symptoms. Methods: We analyzed and reported on the symptoms, ENT examination results, and FNE of 49 pairs of siblings who were examined at the same age. Results: There was a strong association in adenoid size between siblings when they are at a similar age (r = 0.673, $p < 0.001$). Second-born children whose older sibling had III° AH (A/C ratio > 65%) had a risk of III° AH 26 times greater than patients whose older sibling did not have III° AH (OR = 26.30, 95% CI = 2.82–245.54). Over 90% of snoring children whose siblings had confirmed III° AH would develop III° AH by the time they reach the same age. Second-born children in whom snoring occurs and whose older siblings have a III° AH have about a 46 times higher risk of III° AH compared to patients who did not meet these two conditions ($p < 0.001$, OR = 46.67, 95% CI = 8.37–260.30). Conclusions: A significant familial correlation between adenoid size in siblings when they reach the same age was shown. If the older sibling has a confirmed overgrown adenoid (III° AH) and their younger sibling presents adenoid symptoms, particularly snoring, it is highly probable that they will also have an overgrown adenoid.

Keywords: adenoid hypertrophy; AH; siblings; flexible nasopharyngoscopy; adenoid symptoms

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1. Introduction

Adenoid hypertrophy (AH) is one of the most common diseases among preschool children, usually associated with adenoid symptoms, such as mouth breathing, persistent rhinitis, snoring, and a nasal voice [1]. If the disease presents in an older child of a family, parents will often suspect that the same symptoms described above will resurface later in a younger sibling in the family. A common question is whether this problem is familial, especially if the parents also underwent or were considered as a child for adenoidectomy. In 1980, Katznelson and Gross first confirmed a significantly higher incidence of prior tonsillectomy and adenoidectomy performed on an analyzed group of siblings and parents than controls [2]. However, all later performed analyses and surgical procedures were based on presented adenoid symptoms, not on a true measurement of adenoid size. Thus, it is difficult to compare parents and their children, because diagnostic techniques have changed and improved over the years. Nowadays, flexible nasopharyngoscopy examination (FNE) seems the gold standard, not only for assessing adenoid size but also for checking the mucus coverage of the adenoid [3].

It is commonly believed that the adenoid undergoes hypertrophy during childhood, and eventually, involution in adulthood [4]. Over the years, several longitudinal studies,

Handelman and Osborne (1976), Ishida et al. (2018), and Yamada et al. (2021), have assessed the size of the adenoid using lateral cephalometric radiography [5–7]. Yamada showed that an overgrowth of adenoids occurred in preschool children, but there were no significant changes in the adenoid size from 8–12 years of age [7]. A previous study based on flexible endoscopic examinations revealed that adenoid involution proceeds rather slowly; only 7.9% of preschool children (aged 3–7 years) underwent a change in the adenoid size by >15% on the adenoid to choana (A/C) ratio over one year of observation, 21.6% over a period of 2 years, and 45% over a period of 3 years [8]. The growth and development patterns of the nasopharyngeal lymphoid tissues vary for each individual; accordingly, we believe that in studies comparing the adenoid size in a pair of siblings, the permissible age difference should not exceed 12 months.

This study aimed to compare the adenoid size of siblings who were raised in the same household. In most cases, the pairs of children studied were raised with exposure to the same environmental factors, such as cigarette smoke, pollution, and mold allergens, which are considered risk factors for the development of AH [9,10]. Tobacco smoke exposure has been particularly reported as a risk factor for AH [10]; however, nowadays, numerous preventive campaigns have been conducted in our country, and parents are abundantly aware of the risks associated with cigarette smoke exposure in children. Therefore, there is a common practice of avoiding smoking in rooms where children reside. Nevertheless, parents who violate this rule will usually not admit it. Furthermore, the city and rural living conditions may vary in terms of air pollution and allergen exposure, so we analyzed the influence of area of residence on AH. Notably, the duration of breastfeeding is a potential factor distinguishing between babies living in one environment, and breastfeeding has been indicated as a risk factor for snoring and obstructive sleep apnea syndrome (OSAS) [11–13]. Xu et al. stated this correlation and highlighted the need for further investigations to confirm the relationship between breastfeeding and OSAS and the mechanisms underlying it [11]. Chng et al. suggest that breastfeeding independently increases the risk of snoring and possible obstructive sleep apnea syndrome [12]. In another study, Montomery-Downs et al. indicated that OSAS severity is reduced in association with breastfeeding, but it should not be interpreted to suggest that breastfeeding entirely prevents the development of sleep disorder breathing [13]. Presumably, AH may be operational in this mechanism; however, to the best of our knowledge, no study has confirmed a relationship between breastfeeding and AH. Moreover, no genetic factors associated with AH have been discovered so far to demonstrate a similarity in the adenoid size of siblings and justify further research.

FNE is a common procedure to evaluate adenoid size, and this study used FNE to compare adenoid sizes in siblings when they reach the same age. This study was conducted to analyze the adenoid size in siblings when they reach the same age and substantiate a correlation between AH and adenoid symptoms.

2. Materials and Methods

2.1. Study Population

We retrospectively analyzed a group of 1247 preschool children (3–7 year of age) who visited a medical outpatient ear, nose, and throat (ENT) clinic with symptoms suggestive of chronic AH between 2016 and 2021. We searched the medical history of all preschool children admitted to the ENT outpatient clinic. Then, 82 pairs of siblings were selected. We included in the study each pair of children if they were examined in the ENT outpatient clinic at around the same age, where the permissible age difference should not exceed 12 months. We then called their caretakers to confirm if the siblings had the same parents. Exclusion criteria from the study were: children brought up in a common household who have the same last name but different parents, craniofacial anomalies, such as cleft lip/cleft palate; genetic diseases (Down Syndrome); septal nasal deviation; nasal polyp or inferior turbinate hypertrophy; active upper respiratory infection within 2

weeks of enrolling in the study; or previously performed adenoidectomy. In the end, 49 pairs of siblings qualified for participation in the study.

The initial assessment of each patient after study enrollment included a parental questionnaire concerning recurrent upper respiratory infections, defined as a frequent runny nose, pharyngitis, or a cough [14]. We also analyzed the symptoms of rhinitis—at least two nasal symptoms: rhinorrhea, blockage, sneezing, or itching and snoring—defined as persistent, occasional, or non-existent [15]. All children performed an ENT physical examination, flexible fiberoptic rhinoscopy, and tympanometry.

Additionally, we analyzed whether residing in the city or rural regions affects the adenoid size. We divided the children into two groups: those living in the city (population: 170,000–340,000 citizens), and those living in the countryside.

Seasons may influence adenoid mucus coverage and tympanometry type [3]. To avoid any seasonal influence on the obtained results and compare better the sibling population from this study, we divided the year into two main seasons, winter and summer, and we considered the cut-off temperature to be 10°C and also analyzed seasons of performed examination.

2.2. Endoscopy

Each child underwent flexible endoscopic examinations using common nasal meatus, performed by one pediatric otorhinolaryngologist (A.Z.) using the Karl Storz Tele Pack endoscopic system, which was equipped with a flexible nasopharyngoscope (2.8-mm outer diameter and 300-mm length). The percentages of obturation (adenoid-to-choanae ratio in percentage-A/C ratio) of the choanae and mucus coverage of the adenoids were analyzed based on videoendoscopy with the freeze-frame option. Choanal obstructions were assessed with an accuracy of up to 5%. For a better statistical assessment, patients were divided into groups for which we used the 3-degree Bolesławska scale, particularly the part concerning adenoid size in relation to the nasopharynx [16]. All recorded videos of the nasopharynx were coded and blindly analyzed. The percentage of choanal obstruction by the adenoid was measured and compared between siblings. Adenoid size and mucus coverage recorded on the endoscopic system were compared by a second doctor (K.M.). If there was a discrepancy in the assessment, the score was reassessed by a third ENT doctor (P.B.).

The difference in adenoid size between each sibling pair was considered a percentage difference in the amount of nasopharyngeal obstruction by the adenoid. In addition, we used the previous proposed scale to assess the mucus coverage of the adenoid, called the Mucus of Adenoid Scale by Nasopharyngoscopy Assessment (MASNA), which describes the amount of mucus covering the adenoid on a four-point scale (0, no mucus; 1, residue of clear watery mucus; 2, some amount of dense mucus; 3, copious thick dense mucus) [3].

2.3. Tympanometry

An otoscopic examination was performed, and if needed, the external auditory canal was cleaned. In addition, tympanometry was performed using the GSI 39 Auto Tymp™ by Grason-Stadler. The middle ear effusion in each ear was analyzed by tympanometry measurement, and tympanogram graphs were generated. The results were classified using the classification system for tympanograms developed by Liden and Jerger [17,18]. The sequence of saved tympanograms for each patient ear was right, left. We posit that type-B tympanograms produced the worst result, type-C, significant negative pressure in the middle ear, was worse, and type-A, normal middle ear status, was good. For a further statistical analysis, we divided the children into three groups, considering the worse tympanogram result for each child: Group A children with type-A tympanogram in both ears (AA), group C children with tympanogram C (CC, AC, and CA), and group B children with tympanogram B (BB, BC, CB, AB, and BA).

2.4. Ethics

Ethical approval for this study was obtained by the ethics committee of Nicolaus Copernicus University (KB 559/2021).

2.5. Statistical Analysis

We used descriptive statistics to summarize and describe the variables for the study group. We summarized quantitative variables, such as age and adenoid size, based on their means ± standard deviations (SDs). For the categorical variables, we used frequency counts and percentages. To determine differences between variables, statistical significance was estimated using the Chi-square method or Fisher's exact test for categorical variables and the Student's t-test or ANOVA for quantitative variables. Associations between variables were analyzed using Pearson's correlation.

Variables significantly related to adenoid size in a univariate analysis were included in the linear and logistic regression analyses. The linear regression analysis assessed variables of significance for the prediction of adenoid size (A/C ratio, %) volatility in the whole study group. In our linear regression analysis, variables such as recurrent upper respiratory tract infections, rhinitis, snoring, adenoid mucus coverage, and type of tympanogram were assigned appropriate values (for recurrent upper respiratory tract infections (rURTI), rhinitis, snoring: 0, symptom not present; 1, symptom present; for adenoid mucus coverage: from 0 to 4 according MASNA scale; for tympanogram type: 0–A, 1–C, 2–B).

To check for any differences between the pairs of analyzed siblings, the children were divided into two groups: the first including the first examined child from the pair, usually the older of the siblings, and the second including the second examined child, usually the younger. If large families were analyzed, only one pair of siblings from a given family was preferred. We selected the sibling pair with the smallest age difference at the time of examinations.

To analyze the associations between a significant increase in adenoid size (AH) in the second-born child and clinical factors, such as recurrent upper respiratory tract infections, rhinitis, snoring, adenoid mucus coverage, and tympanogram type as categorical variables, a logistic regression analysis was performed. III^{o} AH was defined as an A/C ratio of >65%, based on the Bolesławska scale, where 65% is the cut-off point between II^{o} and III^{o} AH. To predict an A/C ratio of >65% in second-born children, we conducted two separate assessments of: (1) second-born factors and (2) second-born factors and first-born adenoid size. Odds ratios (ORs) and 95% confidence intervals (95% CIs) were also calculated for the considered clinical variables in the regression models.

For all these tests, two-tailed p-values were used, and differences at the level of $p < 0.05$ were considered significant. All statistical analyses were performed using the SPSS (Statistical Package for the Social Sciences version 26, Armonk, NY, USA) software.

3. Results

The mean age of the first examined child group was 5.0 years (SD = 2.2), and that of the second examined child group was 5.1 (SD = 2.2). The mean adenoid size as an A/C ratio for the first sample was 63%, and it was 59% for the second. In total, 71.4% of parents reported rURTI in the first group of siblings and 51% in the second. Rhinitis was present in 77.6% of children from the first group and in 65.3% from the second group. Persistent and occasional snoring were present in, respectively, 36.7% and 28.6% of children from the first group and 34.7% and 26.5% from the second group. Mucous coverage of the adenoid according to MASNA scale grades 0 to 3 was, respectively, 30.6%, 44.9%, 18.4%, and 6.1% in the first group and 28.6%, 38.8%, 24.5%, and 8.2% in the second group. Analyzing the tympanometry results, we found 53.1% type-A tympanograms, 20.4% type-C tympanograms, and 26.5% type-B tympanograms in the first group and 67.3% type-A tympanograms, 12.2% type-C tympanograms, and 20.4% type-B tympanograms in the

second group. Comparing examinations in thermal seasons, 42.9% of children were examined in the summer and 57.1% in the winter in the first group and 55.1% in the summer and 44.9% in the winter in the second group. No differences between groups were found in terms of the analyzed data, except rURTI. All presented data are included in Table 1.

Table 1. Demographic and clinical characteristics of study population according to birth order of siblings.

Characteristic		First-Born Children	Second-Born Children	*p* Value
n		49	49	
Age (years)	Mean ± SD	5.0 ± 2.2	5.1 ± 2.2	0.110
	Median (Q25–Q75)	4.3 (3.7–5.6)	4.6 (3.6–5.7)	
Sex	Female	23 (46.9%)	15 (30.6%)	0.134
	Male	26 (53.1%)	34 (69.4%)	
rURTI *	No	14 (28.6%)	24 (49.0%)	0.021
	Yes	35 (71.4%)	25 (51.0%)	
Rhinitis	No	11 (22.4%)	17 (34.7%)	0.210
	Yes	38 (77.6%)	32 (65.3%)	
Snoring	No	17 (34.7%)	13 (26.5%)	0.630
	Occasionally	14 (28.6%)	19 (38.8%)	
	Persistent	18 (36.7%)	17 (34.7%)	
Adenoid size (A/C ratio and (Bolesławska scale, %)	Mean ± SD	63.0 ± 17	59.0 ± 20	0.163
	Median (Q25–Q75)	60.0 (50.0–75.0)	60.0 (50.0–75.0)	
	<35 (B I)	2 (4.1%)	6 (12.2%)	0.396
	35–65 (B II)	27 (55.1%)	25 (51.0%)	
	>65 (B III)	20 (40.8%)	18 (36.7%)	
Adenoid mucus coverage (MASNA scale)	0	15 (30.6%)	14 (28.6%)	0.387
	1	22 (44.9%)	19 (38.8%)	
	2	9 (18.4%)	12 (24.5%)	
	3	3 (6.1%)	4 (8.2%)	
Tympanogram	AA	26 (53.1%)	33 (67.3%)	-
	AB/BA	2 (4.1%)	0 (0.0%)	
	AC/CA	5 (10.2%)	4 (8.2%)	
	BB	10 (20.4%)	7 (14.3%)	
	CB/BC	1 (2.0%)	3 (6.2%)	
	CC	5 (10.2%)	2 (4.1%)	
	A	26 (53.1%)	33 (67.3%)	0.340
	B	13 (26.5%)	10 (20.4%)	
	C	10 (20.4%)	6 (12.2%)	
Thermal season	Summer	21 (42.9%)	27 (55.1%)	0.263
	Winter	28 (57.1%)	22 (44.9%)	

* rURTI—recurrent upper respiratory tract infections.

An association between adenoid size in siblings was determined by Pearson's correlation analysis. The correlation coefficient between adenoid size on the A/C ratio between the first- and the second-born siblings showed a strong positive association ($r = 0.673$, $p < 0.001$ (Figure 1).

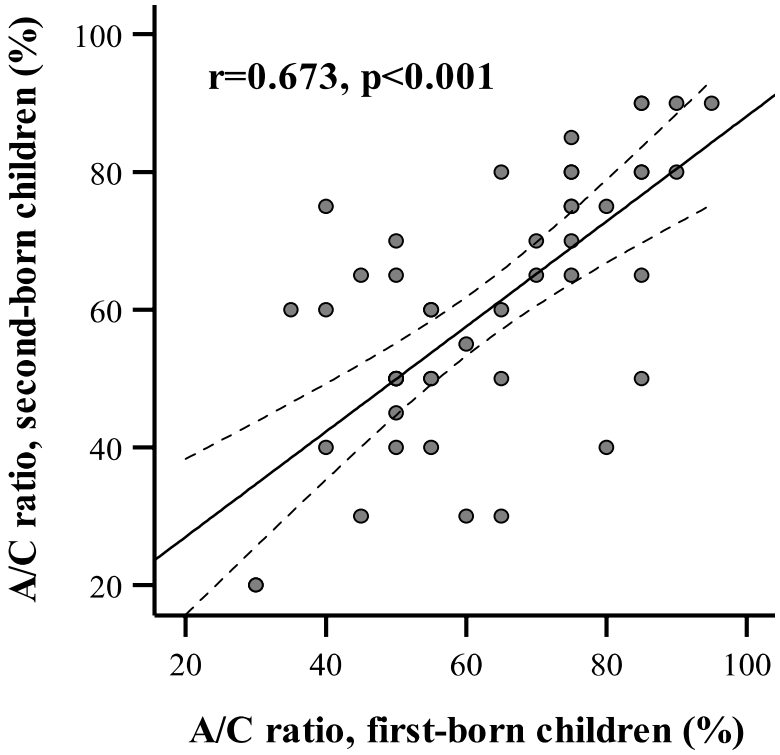

Figure 1. A/C ratio correlation between the first- and second-born children in the family.

In the next analysis step, the relationships between demographic or clinical factors and adenoid size were analyzed for the entire group of children (Table 2). Statistically significant differences in A/C ratios were obtained for rURTI ($p < 0.001$), rhinitis ($p < 0.001$), snoring ($p < 0.001$), tympanometry type ($p < 0.001$), and adenoid mucus coverage ($p = 0.002$). Patients with rURTI and rhinitis, snoring, impaired tympanogram, or high adenoid mucus coverage according to the MASNA scale had an increased A/C ratio. There were no associations between adenoid size and sex and seasonality. Moreover, we analyzed adenoid size change according to age for the whole patient sample (Figure 2). A linear correlation analysis showed no significant correlation between age and adenoid size ($r = -0.125$, $p = 0.219$). We also analyzed the living conditions (in the city or countryside) as an environmental factor influencing the adenoid size. No significant correlation between adenoid size in children living in city and countryside was found.

Table 2. Relationships between demographic or clinical factors and adenoid size in children with symptoms suggestive of chronic AH.

Characteristic ($n = 98$)		Adenoid Size (A/C Ratio), % Mean ± SD	*p* Value
Sex	Female	64.1 ± 20.5	0.195
	Male	59.1 ± 17.1	
rURTI	No	50.0 ± 15.9	<0.001
	Yes	68.0 ± 16.7	
Rhinitis	No	51.6 ± 15.6	0.001
	Yes	64.8 ± 18.3	
Snoring	No	50.5 ± 19.3	<0.001
	Occasionally	60.0 ± 15.4	
	Persistent	71.0 ± 15.4	
	No	50.5 ± 19.3	<0.001
	Yes	65.7 ± 16.3	

Adenoid mucus coverage (MASNA scale)	0	52.8 ± 18.2	0.002
	1	60.0 ± 18.1	
	2	70.2 ± 15.8	
	3	73.6 ± 13.5	
Tympanogram	A	54.5 ± 17.8	<0.001
	B	74.6 ± 15.2	
	C	65.6 ± 13.5	
	A	54.5 ± 17.8	<0.001
	Non-A	70.9 ± 15.0	
Thermal season	Summer	58.2 ± 19.8	0.145
	Winter	63.7 ± 17.0	
Place of residence	countryside	59.8 ± 19.9	0.901
	city	60.3 ± 19.4	

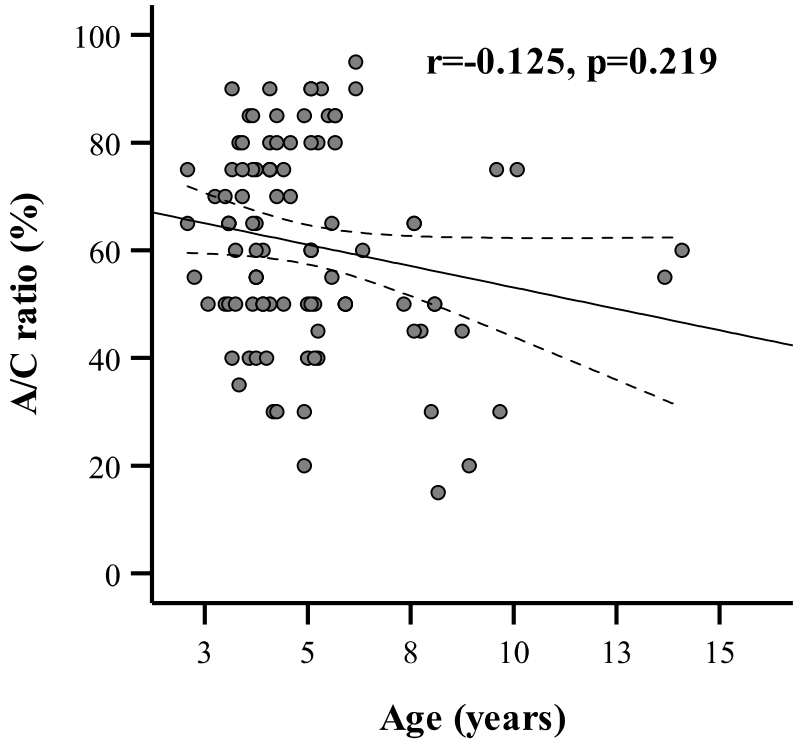

Figure 2. Correlation between A/C ratio and age of study population (*n* = 98).

Variables significantly related to adenoid size in the univariate analysis were included in the linear and logistic regression analyses to identify independent prognostic factors useful in assessing adenoid size. Linear regression analysis for predicting the adenoid size variance in the whole study group revealed that rURTI, snoring, adenoid mucus coverage, and tympanogram type impact the assessment of adenoid size, but not rhinitis (Table 3). Interestingly, the most important aspect is whether the patient is snoring (β = 0.329), followed by the tympanogram type (β = 0.269).

Table 3. Linear regression analysis for the prediction of adenoid size in children (*n* = 98).

Characteristic	*p* Value	B with 95% CI	Beta (β)
rURTI	0.015	9.04 (1.81–16.27)	0.24
Rhinitis	0.470	2.73 (−4.74–10.20)	0.07
Snoring	<0.001	7.47 (3.94–10.99)	0.33
Adenoid mucus coverage	0.019	3.98 (.67–7.30)	0.19
Tympanogram	0.001	6.25 (2.58–9.92)	0.27

The main analysis in this study focused on assessing the significance of clinical factors in the prognosis of III° AH in siblings. For this, logistic regression analyses for the detection of III° AH (A/C ratio > 65%) in second-born children were performed. Using second-born child factors, logistic regression analyses showed importance only for snoring (p = 0.006) and tympanogram type (p = 0.029; Table 4). Patients diagnosed with snoring have more than a 6-fold greater risk of III° AH (OR = 6.23, 95% CI = 1.7–22.70) compared to patients who do not snore. Likewise, the presence of an ear impairment demonstrated by type-B or -C tympanogram relates to an over 6-fold increase in the risk of III° AH (OR = 6.30, 95% CI = 1.20–32.99).

Table 4. Logistic regression analysis for the prediction of III° AH (A/C ratio > 65%) in second-born children (*n* = 49).

Characteristic	*p* Value	OR	95% CI
Second-born child factors			
rURTI, yes	0.736	1.37	0.22–8.43
Rhinitis, yes	0.897	1.14	0.15–8.57
Snoring, yes	0.006	6.23	1.7–22.70
Adenoid mucus coverage (MASNA scale), per category	0.14	1.96	0.80–4.79
Tympanogram, non-A	0.029	6.30	1.20–32.99
Second-born child factors and adenoid size of the first-born child			
rURTI, yes	0.543	0.48	0.04–5.15
Rhinitis, yes	0.575	2.05	0.17–25.08
Snoring, yes	0.015	8.43	1.51–46.95
Adenoid mucus coverage (MASNA scale), per category	0.335	1.68	0.59–4.81
Tympanogram, non-A	0.178	4.34	0.51–36.77
Adenoid size of the first-born child, A/C ratio > 65%	0.004	26.30	2.82–245.54

As shown, there was a strong association in adenoid size between siblings at a similar age. Therefore, we subsequently analyzed the importance of adenoid size in older siblings in relation to assessing adenoid size in younger siblings using logistic regression analysis. For this analysis, the adenoid size of the first-born child was categorized as the binary clinical variable (A/C ratio ≤ 65% vs. >65%). According to the assumed criterion, an A/C ratio of >65% was detected in 20 first-born children (40.8%). The logistic regression analysis showed that the assumed variable had the strongest significant effect on the prediction of III° AH (p = 0.004; Table 4). The obtained estimates indicate that second-born children whose older siblings had III° AH had more than a 26-fold greater risk of III° AH compared to patients whose older sibling did not have III° AH (OR = 26.30, 95% CI = 2.82–245.54). In addition to the adenoid size of the first-born child, from all the analyzed factors that affected the second-born child, only snoring was shown to be a predictive factor of III° AH in the younger sibling (p = 0.015, OR = 8.43, 95% CI = 1.51–46.95). The resulting data showed that knowledge of adenoid size in older siblings eliminates the importance of type of tympanogram in the younger child to estimate their risk of III° AH.

Finally, we assess the potential predictive value of snoring in the second-born child by relating it with an A/C ratio of >65% in the first-born child. Over 90% of snoring

children whose sibling had confirmed III° AH would later develop III° AH (Table 5). In our series, second-born children in whom snoring occurs and whose older siblings have a known A/C ratio of >65% have about a 46-fold higher risk of III° AH (A/C > 65%) compared to patients who did not meet these two conditions ($p < 0.001$, OR = 46,67, 95% CI = 8.37–260.30) (Table 5).

Table 5. The relationship between snoring in the second-born child and an A/C ratio of >65% in the first-born child and an A/C ratio > 65% in the second-born children group with symptoms suggestive of chronic AH.

		Adenoid Size (A/C Ratio, %) of the Second-Born Child		*p* Value	OR (95% CI)
		<=65	>65		
Snoring in the second-born child and A/C ratio > 65% in the first-born child	yes	28 (90.3%)	3 (16.7%)	<0.001	46.67 (8.37–260.30)
	no	3 (9.7%)	15 (83.3%)		

These results indicate that combining snoring in second-born children with adenoid size in first-born children in the same family clearly improves the prediction of III° AH in second-born patients.

4. Discussion

Our work shows a significant correlation between adenoid size in siblings if an FNE is performed at the same age. Katznelson and Gross observed a difference in the number of adenoidectomies between operated parents and siblings and the control group, which might suggest a familial susceptibility to AH [2]. On the other hand, the authors suggest that parents who were previously operated on or who have a child who was previously operated on might be more willing to allow the surgery to be performed on their second child. Still, our results confirmed the hypothesis of familial susceptibility to hypertrophy based on endoscopically assessed adenoid size. However, Bani-Ata et al. indicated a low significance of tonsillectomy in parental and sibling histories [19], but the size of palatine tonsils is not the main indication for tonsillectomy; therefore, it is difficult to compare a family predisposition to adenoidectomy with tonsillectomy. However, recurrent or chronic inflammation susceptibility of the adenoids or palatine tonsil tissue may lead to chronic activation of the cell-mediated and humoral immune response, which may play a role in hypertrophy [20]. This susceptibility to infection may be caused by genetic dispositions. The role of different variations in inflammatory genetic factors, such as polymorphisms of mannose binding lectin (MBL), toll-like receptors (TLRs), secretoglobulins (SCGBs), or IL-10, were analyzed [20–23]. Grasso et al. found that the MBL2 00 genotype is a prognostic marker of AH in children [21]. Meanwhile, Babademez et al. stated that *TLR4* polymorphisms were associated with an increased risk of AH, but they did not find the same association when they analyzed *TLR2* polymorphisms [20]. In addition, in the work of Özdaş et al., the presence of single nucleotide polymorphisms (SNPs) of secretoglobulins were associated with an increased risk of AH [22]. Another study demonstrated the role of the *IL-10* genotype GG in resistance to hypertrophy [23]. All these data support the hypothesis that the inheritance of AH is likely polygenic, involving aspects of physiology determined by multiple genes. Moreover, other non-genetic (environmental) factors, such as cytomegalovirus, human herpesvirus type 6, and infections, may play a role in AH [23]. These factors may co-occur in siblings from the same family who are in constant contact with each other. A study performed by Trask et al. shows that both allergic and non-allergic sibling groups showed a larger mean adenoid size on radiographs than controls [24].

Our study offers practical knowledge for pediatricians. Snoring children have a 6-fold greater risk of AH (III° in Bolesławska scale, A/C ratio > 65%) compared to patients who do not snore. In addition, children with an abnormal (not type-A) tympanogram and indirect effusion in the middle ear indicated a six-times greater chance of III° AH compared to children with a type-A tympanogram. The obtained results indicate that second-born children whose older sibling had III° AH have more than a 26-fold greater risk of III° AH compared to patients whose older siblings do not have AH. Second-born children will have a 46-fold increased chance of developing III° AH if they snore and if their older sibling has previously confirmed III° AH.

AH is the one of the main etiological factors for pediatric sleep disordered breathing (SDB). Lundkvist et al. analyzed parents affected by obstructive sleep apnea (OSAS) and their children, and they concluded that children whose parents were affected by OSAS had a substantially higher risk of hospitalization for SDB [25]. These symptoms were associated with pediatric OSAS or either adenoid or palatine tonsillar hypertrophy. Carmelli et al. analyzed genetic factors in self-reported snoring and excessive daytime sleepiness in twins, arguing that the inheritance of sleep apnea symptoms may be polygenic, but it can also be modulated by the environmental factors in which the twins grow up [26]. The issue of OSAS and hypertrophy of the adenoid and palatine tonsils in siblings was also analyzed by Friberg [27], who showed a significant risk of OSAS in children whose sibling has an OSAS diagnosis, significantly higher than in children with adenoid and palatine tonsils hypertrophy. This study, database research that analyzed AH, was based on a medical diagnosis described in the patient medical history by the ICD-10 code.

In addition, we showed in the whole analyzed group of children a correlation between adenoid size and such adenoid symptoms and related illnesses as rURTI, rhinitis, snoring, poor mucus coverage, and poor tympanometry type. This confirms the role of III° AH in the mentioned factors also described by other authors [1,28–31].

In our study, we showed a close relationship between AH in children and snoring. We stated that involution of the adenoid in children who snore and who have AH decreases slowly and linearly (Figure 2). The shape of the curve on the graph is similar to that presented by Papaioannou et al. [32]. They analyzed adenoid size in an MRI study in children of different ages and concluded that in children who do not snore, adenoid size increases to 7–8 years of age and then it slowly decreases (parabolic curve), and in the group of children who snore (more than 1 night per week), the reduction in adenoid size occurred slowly until 18 years (linear curve).

In summary, this study shows a great familial correlation between adenoid size in siblings based on real adenoid sizes measured by FNE. Other similar studies were based on a history of performed surgery and reported symptoms or ICD-10 code. Due to a lack of a historical possibility to analyze adenoid size via an endoscopic examination in parents because this technology was unavailable when parents were at their children's age, only endoscopic images of siblings' adenoids were comparable.

A limitation in this study was the difficulty of selection of siblings who were examined at the same age, because some parents whose older child was diagnosed with AH or who had presented adenoid symptoms and related illness decided to diagnose their younger child earlier and, based on their own experience earlier, opted for surgery. The influence on sample size is related to the fact that this group was examined by one children's ENT specialist (A.Z.) in the same ENT outpatient clinic. Further, the repeatability of the tests performed is affected by the use of the same doctor using the same flexible endoscopic system.

5. Conclusions

We showed a significant familial correlation between adenoid size in siblings when they reach the same age. The obtained result indicates the environmental and genetic mechanisms of AH, but due to the polygenicity of the issue, more research is necessary.

Our results also suggest that experiences and observations related to the medical history and examination of the older child can be helpful in making a timely diagnosis of the younger child. It is especially important for pediatricians to consider that when an older sibling has a confirmed overgrown adenoid (III° AH) and their younger sibling presents adenoid symptoms, particularly snoring, it is highly probable that they will also have an overgrown adenoid (46 times greater risk).

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Data Availability Statement: Additional data supporting reported results may be available for request.

Conflicts of Interest: The authors declare no conflict of interest.

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The Long-Term Effects of 12-Week Intranasal Steroid Therapy on Adenoid Size, Its Mucus Coverage and Otitis Media with Effusion: A Cohort Study in Preschool Children

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Abstract: Background: The purpose of this study is to analyse the long-term effects of a 12-week course of topical steroids on adenoid size and its mucus using endoscopy and on middle ear effusion measured by tympanometry. Methods: The study presents an endoscopic choanal assessment of the change in adenoid size (adenoid to choanae ratio, A/C ratio) and its mucus coverage in 165 children with Grade II and III adenoid hypertrophy three to six months after finishing a 12-week course of intranasal steroid treatment with mometasone furoate. Additionally, tympanometry was performed to measure middle ear effusion. Changes in the tympanograms were analysed. Results: The mean A/C ratio before treatment was 65.73%. Three to six months after finishing a 12-week course of intranasal steroid treatment, the mean A/C ratio decreased to 65.52%, although the change was not statistically significant ($p = 0.743$). There was no change in adenoid mucus according to the MASNA scale before and three to six months after the end of the steroid treatment ($p = 0.894$). Long-term observations of tympanograms before and three to six months after the end of the treatment did not show improvement ($p = 0.428$). Conclusions: The results indicate that there was no effect of topical steroids on adenoid size, its mucus and otitis media with effusion (OME) three to six months after finishing a 12-week course of treatment. In the light of performed study, decision of adenoidectomy and tympanostomy should not be procrastinated.

Keywords: adenoid hypertrophy; mucus; endoscopic evaluation; topical steroids; intranasal steroids

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1. Introduction

Nasal obstructions, recurrent upper respiratory tracts infections, mouth breathing, persistent rhinorrhoea, snoring, nasal voice and recurrent otitis media in preschool children suggest enlarged adenoids (pharyngeal tonsil) and incline pediatricians to refer the patient to an ear, nose and throat (ENT) specialist [1]. The percentage of adenoid hypertrophy in young children admitted to ENT outpatient clinics because of nasal obstructions is estimated to be 57.7% [2]. Upon confirmation of an enlarged adenoid and its symptoms, conservative treatment with the use an intranasal steroid and saline irrigation should be applied [3–5]. The study concerning on number of eosinophils of allergic rhinitis children demonstrate that combined steroid and saline treatment improves the efficacy of treatment [5]. Unfortunately, almost 90% of children with adenoid hypertrophy and adenoid symptoms undergo surgery in the two-year period after the initial diagnosis [6]. Adenoidectomy is also recommended for children suffered from bilateral OME lasting for over 3 months of evolution, or unilateral OME lasting for over 6 months of evolution as a surgical restoration of physiological tubal function and combined surgery of ventilation tubes installation [7]. From the other side, effectiveness of adenoid surgery in children with

recurrent or chronic nasal symptoms remains unclear and may not be effective treatment in children with sinusitis [8]. In fact, adenoidectomy is one of most frequently performed surgeries in children [9]. This is particularly puzzling since numerous publications have shown the beneficial effect of conservative treatment of intranasal steroids on reducing the size of the pharyngeal tonsil and related symptoms [1,3,10–14]. The impact of corticosteroids on reducing adenoid tissue proliferation was also confirmed by in vitro clinical trials [15]. Two human isoforms of glucocorticoid receptors (GCR-α and GCR-β) have been identified in adenoid tissue that play a role in glucocorticoid ligand efficacy [16]. However, there are limited studies on how long the effects of intranasal steroids on adenoids persist.

In this study, we aimed to analyse changes in the size of Grade II and III pharyngeal tonsil hypertrophy its mucus coverage and middle ear effusion controlled by tympanometry three to six months after finishing a 12-week course of intranasally administered mometasone furoate.

2. Materials and Methods

2.1. Study Population

We performed retrospective analysis of data from two sequential visits of 165 preschool children aged 3–6 years (mean 4.14; SD = 0.97) before and three to six months after finishing a course of 12-week intranasal steroid therapy. We enrolled patients in the study who were classified at the first visit by fibroscopic examination as Grade 2 and 3 adenoid hypertrophy according to Bolesławska [17]. The study group consisted of 83 girls and 82 boys admitted to an ENT outpatient clinic between 2016 and 2021. We eliminated children from the study who had a history of epistaxis, immunodeficiency and hypersensitivity or allergy or atopy to mometasone furoate. Additionally, we excluded patients with genetic diseases (Down, Treacher–Collins Syndrome), craniofacial anomalies, nasal septal deviation, nasal polyp, or inferior turbinate hypertrophy. Children who had an active upper respiratory infection within two weeks of enrolling in the study or those who had previously undergone adenoidectomy or tympanostomy tube insertion were excluded from the study. We further excluded six children from the analysis because of irritation of the nose and throat, crusting, transient dryness and epistaxis, which made it necessary to discontinue the course of intranasal steroids.

2.2. Methods

Each child was examined twice before and after the 12-week course of steroid administration, with a period of at least three to six months without steroid intake before the visits. Because seasonality may influence adenoid mucus coverage and tympanometry, for detailed analyses patients was divided to two subgroups of thermal sequence examination, in which 85 children were first examined in winter and then in summer, and the remaining 80 children were first examined in summer and then in winter [18]. Each patient enrolled in the study underwent a parental questionnaire, history and physical examination, nasopharyngoscopy and tympanometric evaluation.

2.3. Endoscopy

Flexible fibreoptic rhinoscopy examinations were performed by an ENT children specialist (A.Z.) doctor with over 15 years of experience using the Karl Storz Germany, Tele Pack compact endoscopy system (18 kilopixels, 2.8-mm outer diameter, flexible nasopharyngoscope; Medit Inc., Winnipeg, MB, Canada). Based on the recorded video files, we used DaVinci Resolve 17 software (Blackmagic Design) to evaluate and calculate the percentages of obturation of the choanae (A/C ratio, adenoid-to-choana ratio in percentage) and analysed mucus coverage of the adenoids. The A/C ratio was assessed with an accuracy of up to 5% and then classified according to the Bolesławska scale. Mucous coverage of the adenoids was classified according to a previously devised and described scale (Figure 1) called the Mucus of Adenoid Scale by Nasopharyngoscopy Assessment (MASNA) [18]. This is a four-point scale describing the amount of mucus covering the

adenoid: 0 = no mucus, 1 = residue of clear watery mucus, 2 = some amount of dense mucus, 3 = copious thick dense mucus. The adenoid size and mucus coverage recorded on the endoscopic system before and after treatment were blindly assessed by a second doctor (K.M.), and then the results were compared with those scored by first doctor (A.Z.). If there was a discrepancy in the assessment, the score was reassessed by a third ENT doctor (P.B.). We statistically considered the total number of patients depending on the amount of adenoid reduction, ingrowth and lack of change.

The change in adenoid size was considered as the percentage difference in the A/C ratio before and after steroid treatment. The degree of change in adenoid mucus on the MASNA scale was also assessed. A higher score of change in adenoid mucus scale represented an increase in mucus coverage, while a lower score of change indicated decreased mucus coverage.

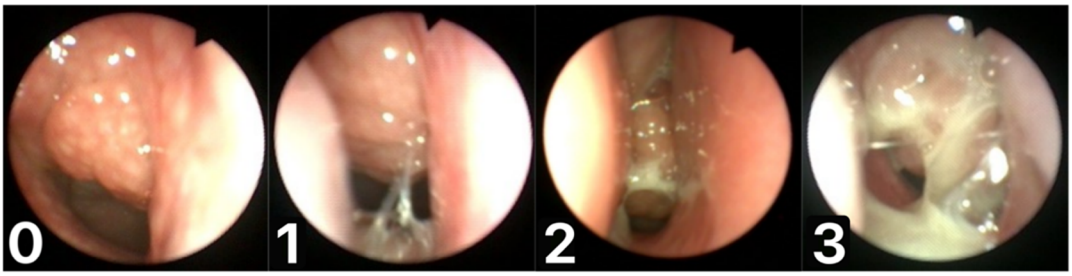

Figure 1. Mucus of Adenoid Scale by Nasopharyngoscopy Assessment (MASNA): ((**0**)—no mucus; (**1**)—residue of clear watery mucus; (**2**)—some amount of dense mucus; (**3**)—copious thick dense mucus).

2.4. Tympanometry

To evaluate effusion in the middle ear, we analysed the type of tympanograms based on the Jerger classification before and three to six months after treatment [19]. A shift from a Type B to a Type C/A tympanogram or from a Type C to a Type A tympanogram was considered an improvement. The persistence of the same type of tympanogram was considered as no change. A shift from a Type C to a Type B or from a Type A to a Type C/B was considered a deterioration.

2.5. Medical Treatment

After the first visit, patients received a 12-week course of conservative treatment with mometasone furoate nasal spray and saline irrigation, which are a standard pharmacological treatment for adenoid hypertrophy symptoms [11,14,20]. One hundred micrograms of the steroid were puffed in each nostril once daily before sleep. We recommended use of hypertonic saline misting sprays twice a day to each nostril, the second dose was administrated at least 15 min before steroid puffs.

We analysed the late changes in the adenoids and tympanometry after topical steroid treatment with a leeway period of three to six months without drug intake.

2.6. Statistical Analysis

We used descriptive statistics to summarize and describe variables for the study group. We summarized quantitative variables, such as age and adenoid size, based on their means ± standard deviation (SD) and medians using the 25th and 75th percentiles (Q25–Q75). For the categorical variables, including gender, mucus coverage according to the MASNA scale, adenoid size according to the Boleslavska scale and tympanograms, we used frequency counts and percentages. In order to determine the differences between the independent variables, statistical significance was estimated using the Chi-square method or Fisher's exact test for categorical variables and a Student's t-test for quantitative variables. To determine the impact of the steroid treatment and thermal seasons on adenoid

size, adenoid mucilage coverage and tympanogram we used analysis for the dependent variables. Quantitative variables were compared with a Student's *t*-test tests for paired samples. The McNemar–Bowker test was used for analysis of categorical variables.

To multivariate analysis of the associations between other factors determined during the first medical visit and intranasal corticosteroid response such as gender, sequence of examination, and age, adenoid size (C/A ratio), adenoid mucus coverage and tympanogram as category variables, logistic regression analysis was performed. Odds ratios (OR) and 95% confidence intervals (95% CIs) were also calculated for considered clinical variables in regression models.

Intranasal corticosteroids response we assessed in three separate analyses: by improvement in (1) adenoid size (C/A ratio), (2) adenoid mucus coverage and (3) tympanogram, as the binary clinical variables. Adenoid size (C/A ratio) improvement was defined as a decrease in value of the C/A ratio by at least 15% from baseline (assessed at first visit). Adenoid mucus coverage improvement was defined as achievement of the lower category of mucilage in MASNA scale from baseline. Tympanogram improvement was based on a three-categorical tympanogram variable: A, B or C and was defined as achievement of better category from baseline (detection of changes: from B to A, B to C and C to A). As these binary variables were created by comparing the results before and after therapy, in the regression models the relevant baseline variables were not included in the creation of individual models.

For all these tests, two-tailed *p*-values were used, and differences at the level of $p < 0.05$ were considered significant. All statistical analyses were performed with SPSS (Statistical Package for the Social Sciences version 26, Armonk, NY, USA) software.

2.7. Ethics

Ethical approval for this study was obtained from the ethics committee of Nicolaus Copernicus University (KB 581/2021).

3. Results

We analysed data from the sequential pediatric ENT examinations of 165 children in the age group of 3–6 years, (mean age 4.14 ± 0.97) before and three to six months after treatment with mometasone furoate, an intranasal topical steroid (Table 1). Of the children, 83 (50.30%) were girls and 82 (49.70%) were boys. The demographic and clinical findings of the patients are summarized in Table 1.

We analysed the impact of steroids on the change in the A/C ratio, adenoid mucus and tympanometry for each patient. The A/C ratio decreased in 53 (32.12%) children, remain unchanged in 62 (37.58%) and increased in 50 (30.30%) (Table 1). The mean A/C ratio before treatment was 65.73%, and three to six months after finishing the 12-week course of intranasal steroid treatment the ratio decreased to 65.52% ($p = 0.743$) (Table 2). There were no statistically significant differences found when comparing selected groups of the same grade of adenoid hypertrophy using the Bolesławska scale ($p = 0.541$). Only in one patient did adenoid size decrease three to six months after treatment to the first degree in the Bolesławska scale (Table 2). We did not observe any change in adenoid mucus based on the MASNA scale before and three to six months after the end of the steroid treatment ($p = 0.894$) (Table 2). Moreover, long-term observations of tympanograms before and three to six months after the end of the treatment did not show improvement in tympanometry ($p = 0.428$) (Table 2). There was no change on tympanometry results. From a total of 80 (48.48%) children, there was improvement in 32 (19.39%) children and deterioration in 53 (32.12%) (Table 1).

Comparing the efficacy of the steroid on adenoid mucous coverage and tympanometry depending on the season in which the intranasal steroid was administrated, we observed statistically significant differences in the changes in adenoid mucus coverage ($p = 0.002$) and in tympanograms ($p = 0.000000505$) (Table 3). A statistically significant impact of thermal season on adenoid mucus coverage was confirmed by the MASNA scale ($p = 0.003$)

and tympanograms (p = 0.0000548), but there was no impact of season on adenoid size (p = 0.280) (Table 4).

As shown, sequence of examination associated with different thermal seasons affects the final therapeutic outcome, evaluated by analysing adenoid size (C/A ratio), adenoid mucus coverage and tympanogram before and at last 12 weeks after completing the treatment. Therefore, we subsequently analysed which factors besides thermal sequence of examination affect the assumed response rates using logistic regression analysis. For this analyses adenoid size (C/A ratio), adenoid mucus coverage and tympanogram was categorised as the binary clinical variables (improvement vs. no improvement). A detailed description of the adopted criteria for indicating improvement for these variables can be found in the in statistical analysis section.

According with assumed criteria, improvement for adenoid size (decrease C/A ratio by at least 15%) was detected in 53 (32.12%), for adenoid mucus coverage in 55 (33.33%) and for tympanogram in 53 children (32.12%).

Logistic regression analyses have shown that the assumed variables have no significant effect on reducing the adenoid size. We showed that, the process of achieving of the adenoid mucus coverage improvement is significantly influenced only by the sequence of examination. The obtained estimates indicate that patients from the winter→summer (W/S) group have more than 2.5 times greater chance of obtaining a favorable result compared to patients from the summer→winter (S/W) group (OR [odds ratio] = 2.86, 95% CI [95% confidence internal] = 1.38–5.91).

Beside sequence of examination, the gender, baseline adenoid size (C/A ratio) in Bolesławska scale and baseline adenoid mucus coverage (MASNA scale) were showed as significant covariates in the model assessing the achievement of tympanogram improvement, with the greatest importance for the sequence of examination. Resulting data showed that conducting therapy and analysing the results of treatment in the winter–summer regimen is about seven times more effective in improving hearing compared to patients treated in the reverse thermal regimen (OR = 7.08, 95% CI = 2.89–17.31) (Table 5).

Table 1. Patients characteristics.

Characteristic		All Patients
n		165
Age at the first visit (years)	mean ± SD median (Q25–Q75)	4.14 ± 0.97 4.00 (3.00–5.00)
Gender	female male	83 (50.30%) 82 (49.70%)
Sequence of examination	Summer→winter (S/W) winter→summer (W/S)	80 (48.48%) 85 (51.52%)
Adenoid size (A/C ratio and (Bolesławska scale)—first visit	mean ± SD [%] median (Q25–Q75) [%]	65.73 ± 12.57 65.00 (55.00–75.00)
	II III	93 (56.36%) 72 (43.64%)
Adenoid size (A/C ratio and (Bolesławska scale)—second visit	mean ± SD [%] median (Q25–Q75) [%]	65.52 ± 12.68 65.00 (60.00–75.00)
	I II III	1 (0.61%) 88 (53.33%) 76 (46.06%)
Impact of steroid on A/C ratio change	mean ± SD [%] median (Q25–Q75) [%]	−0.21 ± 8.29 0.00 (−5.00–5.00)
	decrease no change increase	53 (32.12%) 62 (37.58%) 50 (30.30%)

Table 1. *Cont.*

Characteristic		All Patients
Adenoid mucus coverage (MASNA scale)—first visit	0	29 (17.58%)
	1	59 (35.76%)
	2	46 (27.88%)
	3	31 (18.79%)
Adenoid mucus coverage (MASNA scale)—second visit	0	26 (15.76%)
	1	58 (35.15%)
	2	51 (30.91%)
	3	30 (18.18%)
Impact of steroid on adenoid mucus coverage change	decrease	55 (33.33%)
	no change	48 (29.09%)
	increase	62 (37.58%)
Tympanogram—first visit	AA	74 (44.85%)
	AB/BA	9 (5.45%)
	AC/CA	16 (9.70%)
	BB	36 (21.82%)
	BC/CB	15 (9.09%)
	CC	15 (9.09%)
	A	74 (44.85%)
	B	60 (36.36%)
	C	31 (18.79%)
Tympanogram—second visit	AA	87 (52.73%)
	AB/BA	3 (1.82%)
	AC/CA	14 (8.48%)
	BB	30 (18.18%)
	BC/CB	11 (6.67%)
	CC	20 (12.12%)
	A	87 (52.73%)
	B	44 (26.67%)
	C	34 (20.61%)
Impact of steroid on tympanogram change	improvement	32 (19.39%)
	no change	80 (48.48%)
	deterioration	53 (32.12%)

First visit—visit before start of treatment; second visit—visit over 3 months after end of treatment, A/C ratio—adenoid to choana ratio.

Table 2. Impact of the intranasal steroid on the adenoid size, adenoid mucus coverage and tympanograms.

Characteristic		Intranasal Steroid Treatment		*p* Value
		First Visit (Before Beginning of Treatment)	Second Visit (From >3 to <6 Months after End of Treatment)	
Adenoid size (A/C ratio and (Bolesławska scale)	mean ± SD (%)	65.73 ± 12.57	65.52 ± 12.68	0.743
	median (Q25–Q75) (%)	65.00 (55.00–75.00)	65.00 (60.00–75.00)	
	I	0 (0.00%)	1 (0.61%)	0.541
	II	93 (56.36%)	88 (53.33%)	
	III	72 (43.64%)	76 (46.06%)	
Adenoid mucus coverage (MASNA scale)	0	29 (17.58%)	26 (15.76%)	0.894
	1	59 (35.76%)	58 (35.15%)	
	2	46 (27.88%)	51 (30.91%)	
	3	31 (18.79%)	30 (18.18%)	

Table 2. *Cont.*

Characteristic		Intranasal Steroid Treatment		*p* Value
		First Visit (Before Beginning of Treatment)	Second Visit (From >3 to <6 Months after End of Treatment)	
Tympanogram	AA	74 (44.85%)	87 (52.73%)	0.428
	AB/BA	9 (5.45%)	3 (1.82%)	
	AC/CA	16 (9.70%)	14 (8.48%)	
	BB	36 (21.82%)	30 (18.18%)	
	BC/CB	15 (9.09%)	11 (6.67%)	
	CC	15 (9.09%)	20 (12.12%)	
	A	74 (44.85%)	87 (52.73%)	0.126
	B	60 (36.36%)	44 (26.67%)	
	C	31 (18.79%)	34 (20.61%)	

Table 3. Impact sequence of examination on clinical and demographic variables.

Characteristic		The Sequence of Examination		*p* Value
		Summer→Winter (S/W)	Winter→Summer (W/S)	
n		80 (48.48%)	85 (51.52%)	
Age at the first visit (years)	mean ± SD	3.99 ± 0.92	4.28 ± 1.00	0.050
	median (Q25–Q75)	4.00 (3.00–5.00)	4.00 (4.00–5.00)	
Gender	female	36 (45.00%)	47 (55.29%)	0.186
	male	44 (55.00%)	38 (44.71%)	
Adenoid size (A/C ratio and (Bolesławska scale)—first visit	mean ± SD (%)	65.00 ± 11.72	66.41 ± 13.35	0.473
	median (Q25–Q75) (%)	65.00 (55.00–75.00)	65.00 (55.00–80.00)	
	II	47 (58.75%)	46 (54.12%)	0.549
	III	33 (41.25%)	39 (45.88%)	
Adenoid size (A/C ratio and (Bolesławska scale)—second visit	mean ± SD (%)	65.50 ± 13.54	65.53 ± 11.90	0.988
	median (Q25–Q75) (%)	65.00 (60.00–75.00)	65.00 (60.00–75.00)	
	I	1 (1.25%)	0 (0.00%)	0.695
	II	41 (51.25%)	47 (55.29%)	
	III	38 (47.50%)	38 (44.71%)	
Impact of steroid on A/C ratio change	mean ± SD (%)	0.50 ± 8.37	−0.88 ± 8.21	0.286
	median (Q25–Q75) (%)	0.00 (−5.00–5.00)	0.00 (−5.00–0.00)	
	decrease	23 (28.75%)	30 (35.29%)	0.268
	no change	28 (35.00%)	34 (40.00%)	
	increase	29 (36.25%)	21 (24.71%)	
Adenoid mucus coverage (MASNA scale)—first visit	0	18 (22.50%)	11 (12.94%)	**0.003**
	1	35 (43.75%)	24 (28.24%)	
	2	20 (25.00%)	26 (30.59%)	
	3	7 (8.75%)	24 (28.24%)	
Adenoid mucus coverage (MASNA scale)—second visit	0	9 (11.25%)	17 (20.00%)	0.308
	1	28 (35.00%)	30 (35.29%)	
	2	25 (31.25%)	26 (30.59%)	
	3	18 (22.50%)	12 (14.12%)	
Impact of steroid on adenoid mucus coverage change	decrease	16 (20.00%)	39 (45.88%)	**0.002**
	no change	26 (32.50%)	22 (25.88%)	
	increase	38 (47.50%)	24 (28.24%)	

Table 3. *Cont.*

Characteristic		The Sequence of Examination		*p* Value
		Summer→Winter (S/W)	Winter→Summer (W/S)	
Tympanogram—first visit	AA	47 (58.75%)	27 (31.76%)	**0.004**
	AB/BA	6 (7.50%)	3 (3.53%)	
	AC/CA	7 (8.75%)	9 (10.59%)	
	BB	10 (12.50%)	26 (30.59%)	
	BC/CB	5 (6.25%)	10 (11.76%)	
	CC	5 (6.25%)	10 (11.76%)	
	A	47 (58.75%)	27 (31.76%)	**0.002**
	B	21 (26.25%)	39 (45.88%)	
	C	12 (15.00%)	19 (22.35%)	
Tympanogram—second visit	AA	35 (43.75%)	52 (61.18%)	0.106
	AB/BA	1 (1.25%)	2 (2.35%)	
	AC/CA	7 (8.75%)	7 (8.24%)	
	BB	21 (26.25%)	9 (10.59%)	
	BC/CB	5 (6.25%)	6 (7.06%)	
	CC	11 (13.75%)	9 (10.59%)	
	A	35 (43.75%)	52 (61.18%)	0.062
	B	27 (33.75%)	17 (20.00%)	
	C	18 (22.50%)	16 (18.82%)	
Impact of steroid on tympanogram change	improvement	23 (28.75%)	9 (10.59%)	**0.000000505**
	no change	10 (12.50%)	43 (50.59%)	
	deterioration	47 (58.75%)	33 (38.82%)	

First visit—visit before start of treatment; second visit—visit over 3 months after end of treatment.

Table 4. Impact of the thermal season on the adenoid size, adenoid mucus coverage and tympanograms.

Characteristic		Thermal Season		*p* Value
		Winter	Summer	
Adenoid size (A/C ratio and (Bolesławska scale)	mean ± SD (%)	65.97 ± 13.41	65.27 ± 11.78	0.280
	median (Q25–Q75) (%)	65.00 (60.00–80.00)	65.00 (60.00–75.00)	
	I	1 (0.61%)	0 (0.00%)	0.307
	II	87 (52.73%)	94 (56.97%)	
	III	77 (46.67%)	71 (43.03%)	
Adenoid mucus coverage (MASNA scale)	0	20 (12.12%)	35 (21.21%)	**0.003**
	1	52 (31.52%)	65 (39.39%)	
	2	51 (30.91%)	46 (27.88%)	
	3	42 (25.45%)	19 (11.52%)	
Tympanogram	AA	62 (37.58%)	99 (60.00%)	**0.0000548**
	AB/BA	4 (2.42%)	8 (4.85%)	
	AC/CA	16 (9.70%)	14 (8.48%)	
	BB	47 (28.48%)	19 (11.52%)	
	BC/CB	15 (9.09%)	11 (6.67%)	
	CC	21 (12.73%)	14 (8.48%)	
	A	62 (37.58%)	99 (60.00%)	**0.00000819**
	B	66 (40.00%)	38 (23.03%)	
	C	37 (22.42%)	28 (16.97%)	

Table 5. Factors associated with the adenoid size (C/A ratio), adenoid mucus coverage and tympanogram in patients treated with intranasal corticosteroids (logistic regression models).

Characteristic	*p* Value	OR	95% CI
C/A ratio improvement (≥15% C/A ratio decrease from baseline)			
Gender, male	0.602	0.83	0.42–1.65
Age, per year	0.297	1.20	0.85–1.70
The sequence of examination, winter→summer (W/S)	0.808	0.92	0.45–1.87
Baseline adenoid mucus coverage (MASNA scale), per category	0.067	1.43	0.98–2.10
Baseline tympanogram, per category	0.127	1.38	0.91–2.07
Adenoid mucus coverage improvement (achievement of better category in MASNA scale from baseline)			
Gender, male	0.850	1.07	0.53–2.14
Age, per year	0.661	1.08	0.76–1.54
The sequence of examination, winter→summer (W/S)	**0.005**	**2.86**	**1.38–5.91**
Baseline adenoid size (Bolesławska scale), per category	0.323	1.43	0.70–2.94
Baseline tympanogram, per category	0.051	1.50	0.99–2.25
Tympanogram improvement (achievement of better category from baseline *)			
Gender, male	**0.009**	**0.33**	**0.14–0.76**
Age, per year	0.325	0.82	0.55–1.22
The sequence of examination, winter→summer (W/S)	**0.000018**	**7.08**	**2.89–17.31**
Baseline adenoid size (Bolesławska scale), per category	**0.011**	**2.87**	**1.27–6.48**
Baseline adenoid mucus coverage (MASNA scale), per category	**0.002**	**2.04**	**1.31–3.16**

Baseline values were collected from data obtained at the first pre-treatment visit. * Analysis based on three-categorical tympanogram variables: A, B and C; tympanogram improvement if detection of changes: from B to A, B to C and C to A.

4. Discussion

Our study based on 165 children with adenoid hypertrophy treated with mometasone furoate did not reveal any change in adenoid size and its mucus three to six months after finishing a 12-week course of intranasal steroid treatment ($p = 0.541$, $p = 0.894$ respectively). Additionally, there were no differences in middle ear effusion on tympanometry examination ($p = 0.428$).

These results are in contrast to those of the vast majority of studies, which have shown reductions in adenoid hypertrophy and related symptoms after the use of intranasal steroids [10,11,14,21–28]. One exception is the work of Anjali Lepcha, but her results showing no effect of beclomethasone on tonsils was based on an analysis of only 13 patients, and the results were not statistically significant ($p < 0.060$) [29]. None of these trials established the optimal duration of the treatment in children. Almost all of them evaluated adenoid size immediately following conservative treatment and not after a leeway period without topical steroid treatment. The clinical findings of the previously published papers are summarized in Table 6.

Table 6. Review of similar studies. MF—mometasone fluroate, F—flunisolide, B—beclomethasone.

Author Year Country	Age	Number of Patients Treated with Steroids	Medication	Time of Treatment	Time of Final Results Counting	Main Results
Cengel 2005 Turkey [21]	3–15	122	MF	6 weeks	at the end of therapy	42.2% improvement of OME
Ciprandi 2007 Italy [22]	3–6	139	F	8 weeks	at the end of therapy	reduction of A/H index in 72% children

Table 6. *Cont.*

Author Year Country	Age	Number of Patients Treated with Steroids	Medication	Time of Treatment	Time of Final Results Counting	Main Results
Demirhan 2010 Turkey [14]	4–16	25	MF	8 weeks	at the end of therapy	symptoms improvement, 76% of children do not need adenoidectomy
Mohebbi 2014 Iran [23]	2–11 (2–4 and 5–11)	51	MF	3 months	at the end of therapy	improvement
Gupta 2015 India [24]	4–12	55	MF	4 weeks	at the end of therapy	improvement
Monga 2020 India [25]	3–11	30	MF	8 weeks	at the end of therapy	improvement
Rezende 2015 Brazil [26]	4–8	55	MF	6 weeks	at the end of therapy	reduction of adenoid size
Hassanzadeh 2016 Iran [27]	4–12	20	MF	4 weeks	at the end of therapy	reduction of adenoid size
Lepcha 2002 India [29]	3–12	13	B	8 weeks	at the end of therapy	**no improvement**
Berlucchi 2008 Italy [20]	3–7	21	MF	1–3 months before surgery or 15–31 months (mean 23) (2 weeks every month, suspended during the summer)	before surgery (9 children) or at the end of the maintenance therapy (12 children)	regular continuity MF therapy may obtain successful results
Criscuoli 2003 Italy [11]	Mean 3, 8	53	B	26 weeks	24,52,100 weeks after treatment	relevant clinical improvement in 45% children but 70% children performed surgery
Jazi 2011 Iran [28]	2–10	20	MF	6 weeks	1 and 8 weeks after treatment	clinical improvement more significant than adenoid regression in nasofiberoscopy
Bhargava 2014 India [10]	2–12	100	MF	24 weeks	24 weeks after treatment	clinical improvement

Only a few papers mentioned the long-term effects of adenoid hypertrophy treatment with intranasal steroids and only those study could be used to assess if steroid therapy could prevent the need for surgery. The longest follow-up period of 31 months was presented by Berlucchi [20]. In his study, he advised an increase in the duration of steroid treatment to 31 months and proposed a different schema of steroid therapy: two weeks of intranasal steroids taken every month, suspended during the summer. He claimed this could free children from adenoid symptoms and reduce adenoid size. The study group was relatively small, containing only 12 patients, and this treatment may not be acceptable because of the potential side effects. A larger study, with a group of 53 patients and a long follow-up time of up to two years, was presented by Criscuoli. However, despite a clear improvement in the health of 45% of the children as a result of steroid treatment, 70% of them needed to undergo adenoid surgery at the end of the study [11]. Jazi also reported clinical improvement in patients eight weeks after the end of treatment, but the study group was limited to 20 patients [28]. Arguably the most reliable study was presented by

Bhargava [10], who analysed a large cohort (100 children) over a long follow-up period (six months) after the end of steroid therapy. The endoscopic evaluation results indicated a long-term reduction in the size of the adenoid as a result of treatment. Unfortunately, the group of children aged 2–5 years contained only 13 patients. Generally, studies analysing changes in the size of the pharyngeal tonsil in children treated with intranasal steroids have employed small samples, with only three including samples exceeding 100 children (Table 6). This makes proper inference challenging.

It seems that in order to achieve the beneficial effect of treatment and avoid surgery, steroids should be used for a longer time over a period of up to several years with possible periods of suspension when symptoms are reduced. This period may be summer, where we observed an improvement in adenoid mucus coverage and tympanometry results [18]. The scheme proposed by Berlucchi may warrant consideration as well [20]. However, research is required on a larger group of preschool children to determine the lowest effective dose as well as the side effects of long-term steroid use. Local adverse effects in children, such as nasal irritation, sneezing, epistaxis, a burning and dry sensation in the nose and septal perforation, are uncommon [30,31]. The use of mometasone fluorinate for one year in children aged six to nine years showed only minimally higher rates of epistaxis in the sample group compared to a placebo, and there has been no study focused on preschool children [32]. Moreover, parents should be informed not to administer intranasal topical steroids with topical decongestants to their children for longer than a few days because of tachyphylaxis and rebound congestion [30]. Intranasal steroids may influence children's growth or bone metabolism. Such an effect was shown in a study analysing beclomethasone [33]. However, a study on a newer intranasal steroid (mometasone furoate) at a dose of 100 micrograms per day in 98 children three to nine years of age contradicted these findings [32]. One year of continuous steroid treatment did not retard the growth of the children [32]. However, McDonnell concluded that children using intranasal steroids on a regular basis should be checked for growth, following clinical growth charts (CDC) growth curves. This may be performed by the child's pediatrician [30].

This study confirmed our previous performed study concerning on seasonality change of adenoid size and mucus coverage and change in tympanogram [18]. Moreover, it shows that sequence of steroid treatment and observation may influence on positive results. We obtained a 2.5 times greater chance of favorable results for mucus coverage in the MASNA scale if we began treatment in winter and assessed the results in summer. It shows that seasonality impact on obtained results should be always taken into account if impacts on other factors are assessed on the adenoid change in a long-time period.

Age group selection is particularly important. Combining a group of children 2–6 years of age with a group 7–12 years of age may affect the results. In the younger age group, ailments associated with the pharyngeal tonsil are usually more burdensome and significant. In schoolchildren, these ailments are less bothersome [34].

In our long-term study, we found no improvement in tympanometry results after 12-week treatment with topical steroids. This is consistent with the international consensus based on 12 studies of 945 patients, which showed no improvement in long-term OME clinical symptoms after intranasal steroid treatment. Hence, steroid treatments are not recommended due to their cost, possible side effects and lack of long-term efficacy [35]. Tympanostomy ventilation tube insertion with or without adenoidectomy is the only treatment to have been validated by the international scientific community for persistent OME with functional impartment of hearing between 25 and 40 dB or with damage of tympanic membrane [36,37].

Recently, new publications on adenoid hypertrophy and OSA and the impact of adenoidectomy on the improvement of behavioral symptoms in children indicate the adverse effect of long-term impaired nasal ventilation on the child's cognitivist development [38]. Confirmed in our study was the absence of long-term effectiveness of steroid pharmacological treatment of the adenoid hypertrophy and OSA symptoms indicate the need to look

for another effective method of pharmacological treatment or to decide more quickly on adequate surgery.

5. Conclusions

The results indicate that there was no effect of intranasal mometasone furoate on adenoid size, its mucus and OME three to six months after finishing a 12-week course of treatment. Topical steroids seem to have a temporary effect on adenoid size and its mucus, which decreases when they are not used. The durability of the long-term effects of newer intranasal steroids used for a moderate period on adenoid size should be analysed and compared with those of steroids used for a longer duration to establish an effective treatment for adenoid hypertrophy and to avoid adenoidectomy. Further studies are needed on the impact of multiple-year courses of topical steroids on adenoid size and its mucus coverage as well as side effects on preschool children. We are inclined to use the Berrlucchi scheme, which reduces the risk of complications with long-term topical steroid use. We were not able to confirm the beneficial effect of intranasal steroids on tympanometry results reported in other studies. In light of the performed study decision, adenoidectomy and tympanostomy should not be procrastinated. The seasonality impact on the adenoid mucus coverage and tympanometry should be always taken into account if the impact of other factors is assessed on the adenoid change in a long-time period.

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RHINOLOGY

Maximal medical treatment of adenoid hypertrophy: a prospective study of preschool children

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Abstract

Purpose This study aimed to examine the effectiveness of the combined maximal medical treatment for adenoid hypertrophy in preschool children.

Methods Sixty-four children underwent one-year combined therapy with intranasal mometasone furoate, oral desloratadine, nasal saline irrigation, and bacteriotherapy. Additionally, decongestion drops were applied during scheduled breaks.

Results Of the 64 treated children, 72% showed clinical improvement in adenoid symptoms while 28% did not improve and underwent surgery. These groups differed significantly in terms of the overall reduction in ailments after treatment ($p < 0.001$), infection rate ($p < 0.001$), catarrh severity ($p < 0.001$) and nasal patency ($p < 0.001$). Endoscopic examination confirmed that responders experienced, on average, a decrease of 8.4% in the adenoid/choana ratio and an improvement in mucosal coverage of the adenoid. These effects were not observed in the group of children whose parents opted for surgery after nine months of conservative treatment.

Conclusions The proposed new schema of long-term maximal medical treatment with the use of combined intermittent treatment of intranasal mometasone furoate and decongestion drops, oral desloratadine, nasal saline irrigation, and bacteriotherapy can be attempted in patients with adenoid hypertrophy symptoms, and responders may avoid the need for surgery. The applied treatment breaks resulted in a low number of therapeutic side effects.

Keywords Adenoid hypertrophy treatment · Intranasal steroids · Antihistamine drugs · Flexible nasopharyngoscopy · Adenoidectomy

Introduction

Adenoid hypertrophy (AH) is one of the most common causes of nasal blockage, rhinitis, snoring, mouth breathing, and upper respiratory tract infection in preschool children [1]. These effects of AH are commonly called adenoid symptoms. Various diagnostic methods are used to confirm AH, but the most effective is flexible nasopharyngoscopy [2].

Surgical treatment of AH has been shown to be highly effective, but like other surgical treatments, it involves the risk of serious and potentially fatal complications [3]. In addition, an adenoidectomy can cause stress and anxiety in children, as it is often the first surgery they undergo in life [4, 5]. Therefore, many parents seek to avoid surgery in favor of conservative treatment methods. Over 150 years ago, various pharmacological substances, including silver nitrate, sodium bicarbonate, and salt solutions, were used to reduce adenoid size or to identify adenoid symptoms more precisely before surgery [6]. Doctors in that era generally believed that such methods were effective, although they did not exclude surgical intervention. For example, Mayer, the father of adenoid surgery, in summarizing his observations on adenoid treatment, discussed medico-surgical treatment [6]. Today, doctors still attempt to treat adenoid hypertrophy conservatively, utilizing various therapies, including systemic antibiotics, nasal steroid sprays, hypertonic saline solutions, proton pump inhibitors, bacteriotherapy, traditional Chinese

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herbal medicine and, more recently, anti-leukotriene drugs [7]. Several weeks of treatment with systemic antibiotics is not currently used because its effectiveness is unproven and it alters children's microbiome [8, 9]. In preliminary studies, Chung et al. showed that local antibiotic nasal irrigation does not improve nose and sinus inflammation in AH [10]. An alternative trend involves stabilizing the microbiome using lyophilized bacteria supplementation [11]. A study by La Mantia et al. indicated that nasal application of *Streptococcus salivarius* 24SMB and *Streptococcus oralis* 89a may help to reduce AH [12]. While some studies have linked laryngopharyngeal reflux and AH, Iqbal et al. found that proton pump inhibitors (PPI) do not reduce AH [13, 14]. Moreover, although some research has suggested that anti-leukotriene therapy could be useful for AH treatment, montelukast is not approved for such use in Europe due to its serious side effects [15–17]. Studies on traditional Chinese medicines are difficult to interpret due to variable herbal mixtures and a lack of objective measurements [18].

Many studies have confirmed that intranasal steroids effectively reduce adenoid tissue and symptoms both in vitro and clinically, leading to their broad usage [7, 19–22]. However, most studies have focused on symptom reduction rather than adenoid size [7]. Steroids also affect the nasal mucosa, altering mucus quantitatively and qualitatively, further reducing adenoid symptoms [7]. In addition, it has been shown that the season can affect the mucus coverage of the adenoid more than steroids [23]. Therefore, it is important to evaluate treatment effects in the same season. Research has shown worse long-term outcomes after ending treatment compared to immediate post-treatment outcomes [24, 25]. Consequently, there is a tendency to prolong steroid therapy in AH treatment [22, 26].

Allergy rates in children range from 20 to 40%, and Eren et al. found they could reach 65% in children with AH [27–29]. However, most children suffering from adenoid symptoms that begin at the age of 3–4 years are not diagnosed due to their allergies, which are usually diagnosed at the age of 6–7 years. Therefore, some pediatricians empirically introduce antihistamine drug therapy for rhinitis symptoms and continue it for responders.

Nasal irrigation with hypertonic saline solution may help to flush the mucosa and eliminate dense mucous, inflammatory mediators, allergens and pathogens, reducing rhinitis and some adenoid symptoms [30]. It can also improve local drug efficiency [30, 31].

This study examined the effectiveness of combined maximal medical treatment (MMT) for AH in preschool children, analyzing parent-reported symptoms and pre- and post-treatment nasopharyngoscopy findings. We applied a combination of intranasal steroid therapy and mannitol (Narivent®) decongestion drops with antihistamine oral drugs and local bacteria supplementation [7].

Materials and methods

Study group

Seventy-nine 3–7-year-old children with suspected AH were diagnosed in an ENT outpatient clinic in the thermal winter of 2022, from January to April, when the average temperature was below 10 °C, using nasopharyngoscopy and tympanometry. During the study, five patients were lost from the controls, and another five children discontinued therapy. Two stopped due to their parents' fear of long-term steroid use, another two stopped due to heavy bleeding during steroid therapy and the parent of one child decided to treat the child with inhalations and a salt cave, reporting a reduction in symptoms. Finally, 69 children (32 girls and 37 boys) started the one-year therapy. Of these children, 10 (14.5%) had allergies, 12 did not (17.4%), and 47 (68.1%) were not tested for allergies.

As is widely recognized, chronic otitis media with effusion (OME) often coexists with adenoid hypertrophy. In many surgical centers, both issues are addressed in a single surgical procedure. In our approach, we adhered to the following strategy:

1. If adenoid symptoms persisted during the proposed schema of pharmacological treatment, and follow-up examinations revealed no reduction in adenoid size despite pharmacological treatment, and no OME was detected, we opted for adenoidectomy alone.
2. In cases where there was no improvement in adenoid symptoms during follow-up, no reduction in adenoid size was observed in flexible nasopharyngoscopy, and OME persisted for more than 3 months, we performed both adenoidectomy and tympanotomy.
3. When there was regression in adenoid size and improvement in symptoms, but OME persisted for more than 3 months, we conducted only tympanostomy while continuing pharmacological treatment
4. When there was regression in adenoid size and improvement in symptoms, and there was OME regression or OME was not stated the pharmacological treatment was continued

During the study, 23 children underwent surgical treatment for AH due to a lack of expected improvement in adenoid symptoms or persistent middle ear effusion during treatment. Eleven children underwent adenoidectomy, and 12 underwent adenoidectomy and ventilation tube insertion into the tympanic membrane. Additionally, two children underwent only tympanostomy because of middle ear effusion confirmed by tympanometry and hearing loss, with the resolution of adenoid symptoms. The remaining

46 patients continued their therapy. The patients' characteristics are presented in Table 1.

Exclusion criteria

We excluded children whose parents refused pharmacological treatment and immediately decided on surgery, children who were treated for asthma with anti-leukotriene therapy, children who were treated with nasal steroids for more than two weeks in the two months preceding the ENT medical visit, children with a history of active upper respiratory infection within two weeks of enrolling in the study and children who had previously undergone an adenoidectomy or tympanostomy tube placement. Additionally, we excluded children with craniofacial malformations and Down syndrome.

Diagnostics

All the examinations were performed by one pediatric ENT doctor (A.Z.), who has approximately 20 years of experience in the use of the Karl Storz Germany Tele Pack compact endoscopy system (18 kilo pixels, 2.8 mm outer diameter, flexible nasopharyngoscope; Medit Inc.). Based on the recorded video files, DaVinci Resolve 17 software (Blackmagic Design) was used by a second ENT doctor (K.M.), who evaluated and calculated the percentages of obturation of the choanae (adenoid-to-choana ratio (A/C ratio) as a percentage). The A/C ratio was assessed with an accuracy of up to 5%. To facilitate a better statistical assessment, patients were divided into groups, and the three-degree Bolesławska scale and the four-degree MASNA scale (mucous on adenoid scale by nasopharyngoscopy assessment) were used to categorize adenoid size and mucus coverage [23, 32]. Tympanometry was performed using a GSI 39 Auto Tymp™ (Grason-Stadler). Tympanograms were grouped as A (normal), C or B (worst). For further statistical analysis, we divided the children into three groups, considering the worst tympanogram result for each child: Group A children with type A tympanograms in both ears (AA), group C children with type C tympanograms (CC, AC and CA) and group B children with type B tympanograms (BB, BC, CB, AB, and BA).

Treatment

All parents were informed about their children's diagnosis and the available treatment methods as well as their complications and side effects. Some of them decided to undertake annual therapy following the proposed scheme, which involved one-year combined therapy of intranasal corticosteroid spray intermittent with decongestion drops, oral desloratadine, nasal saline irrigation and bacteriotherapy. The therapy included the use of intranasal mometasone furoate spray. Fifty micrograms of the steroid were puffed in each nostril once daily for three weeks in each month with a one-week break. During the break, the children were treated with mannitol decongestion drops (Narivent ®), with one puff to each nostril administered twice daily. Additionally, the children were treated with 2.5 ml–5 ml of desloratadine **0.5 mg/ml syrup** in accordance with their age once per day. Based on the reduction of complaints and adenoid mucous improvement in the summer, as shown in our previous works, the children were not administered medication from the second half of June to the beginning of September [23]. During this pharmacological break, intranasal Streptococcus salivarius 24SMBc and Streptococcus oralis 89a bacteriotherapy was used twice a day for one week in each of the three subsequent months. We also recommended nasal irrigation with hypertonic nasal saline spray twice a day or more if the child was able to perform high-volume flushing with a hypertonic saline solution (50 ml) once a day for the entire treatment period. Parents were informed about the possible side effects of the treatment and the possibility of ending treatment at any time. Standard rescue medications were permitted in the event of distress for all patients.

Final validation

After 9–12 months, the effect of the treatment on adenoid symptoms was analyzed, and parents elected to forego surgery. Additionally, during the final visit, a control endoscopy was performed to assess the adenoid size and mucus in the same season as the therapy started. Post-therapy, control-flexible nasopharyngoscopy was also performed in 18 children whose parents opted for surgery. In five children who underwent operations, flexible endoscopy was not performed because the adenoid symptoms persisted, and their parents decided for surgery. In these patients, the minimum period of conservative treatment and follow-up, which was at least nine months of conservative treatment, was not achieved. Finally, to compare the endoscopic results of pharmacological AH treatment, we analyzed the sample of 64 children, consisting of the group of 46 children who were not operated on and the group of 18 who underwent surgery but had a period of conservative treatment longer than nine months.

Side effects

The parents did not report sedation, nervousness, hyperactivity, dry mouth, rash, seizures, blood pressure variability, increased urine output, or gastrointestinal upset as antihistamine drug side effects. In two cases, children were not treated with intranasal bacteriotherapy due to non-acceptance by the children themselves. Additionally, one case involved bleeding from the nose, and in another case, there was a worsening

Table 1 Characteristics of the study group

Characteristic		All Patients
N		64
Age at the first visit (years)	Mean ± SD	3.9 ± 1.1
Gender	Female	31 (48.4%)
	Male	33 (51.6%)
Therapy	Medical–surgical	18 (28.1%)
	Medical	46 (71.9%)
Adenoid size (A/C ratio and Bolesławska scale)—first visit	Mean ± SD [%]	73.8 ± 13.7
	I°	2 (3.1%)
	II°	12 (18.8%)
	III°	50 (78.1%)
Adenoid size (A/C ratio and Bolesławska scale)—final visit	Mean ± SD [%]	68.1 ± 14.8
	I°	2 (3.1%)
	II°	31 (48.4%)
	III°	31 (48.4%)
Impact of MMT on A/C ratio change	Mean ± SD [%]	–5.7 ± 11.1
	Decrease	37 (57.8%)
	No change	14 (21.9%)
	Increase	13 (20.3%)
	Decrease ≥ 15%	20 (31.3%)
	Increase, no change or decrease < 15%	44 (68.8%)
Adenoid mucus coverage (MASNA scale)—first visit	0	9 (14.1%)
	1	21 (32.8%)
	2	18 (28.1%)
	3	16 (25.0%)
Adenoid mucus coverage (MASNA scale)—final visit	0	22 (34.4%)
	1	14 (21.9%)
	2	17 (26.6%)
	3	11 (17.2%)
Impact of MMT on adenoid mucus coverage change	Decrease	34 (53.1%)
	No change	14 (21.9%)
	Increase	16 (25.0%)
Tympanogram—first visit, n = 63	AA	18 (28.6%)
	AB/BA	1 (1.6%)
	AC/CA	8 (12.7%)
	BB	17 (27.0%)
	BC/CB	10 (15.9%)
	CC	9 (14.3%)
	A	18 (28.6%)
	B	28 (44.4%)
	C	17 (27.0%)
Tympanogram—final visit, n = 61	AA	28 (45.9%)
	AB/BA	1 (1.6%)
	AC/CA	6 (9.8%)
	BB	13 (21.3%)
	BC/CB	6 (9.8%)
	CC	7 (11.5%)
	A	28 (45.9%)
	B	20 (32.8%)
	C	13 (21.3%)

Table 1 (continued)

Characteristic		All Patients
Impact of MMT on tympanogram change	Improvement	18 (29.5%)
	No change	37 (60.7%)
	Deterioration	6 (9.8%)
Allergies	Not tested	43 (67.2%)
	No	12 (18.8%)
	Yes	9 (14.1%)
Rhinitis (weeks in a month)	Mean ± SD [%]	1.3 ± 0.8

First visit: visit before start of treatment; Final visit: visit over nine months after start of treatment or visit before adenoidectomy; A/C ratio: adenoid-to-choana ratio

of nasal swelling, leading to the discontinuation of intranasal bacteriotherapy for both of these children. Of the entire group of 69 children, two needed to discontinue nasal steroids due to heavy bleeding. Moreover, in one case, decongestion drops led to increased nasal swelling and had to be stopped; while in another case, they were not well tolerated by the child. We controlled the growth curves for each treated child and did not observe any growth retardation in the study group.

Statistical analysis

We used descriptive statistics to summarize and describe variables for the study group. We summarized quantitative variables, such as age and adenoid size, based on their means ± standard deviation (SD). For the categorical variables, including gender, mucus coverage (based on the MASNA scale), adenoid size (based on the Bolesławska scale) and tympanograms, we used frequency counts and percentages. To determine the differences between the independent variables, statistical significance was estimated using the Chi-square method or Fisher's exact test for categorical variables and a Student's t-test for quantitative variables. To determine the impact of the AH-MMT on adenoid size, adenoid mucilage coverage and tympanograms, we analyzed the dependent variables. Quantitative variables were compared using a Student's t-test tests for paired samples, and the McNemar–Bowker test was used for analysis of the categorical variables.

Ethics

Ethical approval for this study was obtained from the ethics committee of Nicolaus Copernicus University (KB 141/2022).

Results

Of the 69 children qualified to receive conservative treatment, 33% were operated on during the year due to a lack of improvement and persistent adenoid symptom complaints. Overall, the parents of 64 children (who were analyzed because they met the criterion of at least nine months of conservative treatment) reported a reduction in ailments (78%): 76.6% had a lower infection rate, and 73.4% experienced decreased snoring. However, during sleep, the mouths of 64.1% of the children were permanently or periodically open. Additionally, the severity of catarrh decreased in 76.6% of the children, and 79.7% had better nasal patency (Table 2).

In the whole group of patients, we observed a statistically significant reduction in adenoid size, mean adenoid size as well as in the Bolesławska scale comparing adenoid size in flexible endoscopy before and after treatment ($p < 0.001$). Additionally, we found a statistically significant reduction in the MASNA scale. However, we did not observe any improvement in tympanometry. Detailed results are presented in Table 3.

Comparing both endoscopically controlled groups, that is, the group of children who underwent surgery and the

Table 2 Parental assessment of the children's health status at the final visit compared to their status at the first visit

Symptom		All Patients
n		64
Ailments	Improvement	50 (78.1%)
	No improvement	14 (21.9%)
Recurrent upper respiratory tract infection	Improvement	49 (76.6%)
	No improvement	15 (23.4%)
Snoring	Improvement	47 (73.4%)
	Periodical improvement	5 (7.8%)
	No improvement	12 (18.8%)
Mouth breathing	Improvement	35 (54.7%)
	Periodical improvement	6 (9.4%)
	No improvement	23 (35.9%)
Rhinitis	Improvement	49 (76.6%)
	No improvement	15 (23.4%)
Nasal obstruction	Improvement	51 (79.7%)
	No improvement	13 (20.3%)

group treated conservatively, they did not differ statistically significantly initially in terms of gender, age, allergy rate or adenoid size based on the Bolesławska scale and mucus coverage based on the MASNA scale (Table 4). A difference was found in adenoid size based on the mean A/C ratio ($p=0.029$). Further, there were differences in both groups after treatment in adenoid size (Bolesławska scale, $p=0.002$), mean adenoid size (A/C ratio, $p<0.001$) as well as mucus coverage (MASNA scale, $p=0.015$). In 66.6% of the children whose parents decided to have surgery, the adenoid size remained unchanged or increased. In the group that continued pharmacological treatment and in which the parents reported a reduction in discomfort and did not decide on the surgical procedure, the adenoid size decreased in 67.4% of the children. Statistically significant differences between the two groups were found in both adenoid size and mucus coverage ($p=0.01$ and $p=0.002$, respectively). In the group continuing conservative treatment, adenoid size decreased based on the A/C ratio by an average of 8.4%, and in the operated group, the A/C ratio increased by 1.1%. This difference was statistically significant ($p=0.002$).

Both groups also differed in terms of tympanograms during the first and final visits. After treatment, a statistically significant difference in tympanograms was found ($p<0.001$).

However, the groups did not differ in the frequency of allergies, although it should be noted that 67% of the children were not tested (Table 1). Regarding the frequency of catarrh, in the group continuing pharmacological treatment, catarrh occurred on average 1.2 weeks per month. Meanwhile, in the group that decided to have surgery, the average frequency of catarrh during treatment was 1.7 weeks ($p=0.031$). Detailed results are presented in Table 4.

The two groups differed in terms of the overall reduction of ailments after treatment ($p<0.001$), infection rate ($p<0.001$), severity of catarrh ($p<0.001$) and nasal patency ($p<0.001$). There was no difference in the severity of snoring, but there was a difference in mouth openness during sleep ($p=0.041$) (Table 5).

Discussion

During the 12-month treatment, 70% of the children improved so much that their parents did not decide to undergo surgical treatment. However, about 30% of the children underwent operations within 9–12 months of the initiation of AH-MMT therapy. The parents of these children decided to have the procedure performed based on persistent ailments, despite the MMT treatment. The persistence or reduction of adenoid ailments determined the parents' decision to continue pharmacological therapy or elect surgical treatment. The analysis showed that the severity of adenoid symptoms correlated with the endoscopic clinical picture of the adenoid in the medical-treated and medical–surgical-treated children. In the first group, the size of

Table 3 Impact of conservative maximal medical treatment for adenoid hypertrophy on adenoid size, adenoid mucus coverage, and tympanograms

Characteristic ($n=64$)		MMT		*P*-value
		First visit (before beginning of treatment)	Final visit (from > 9 to < 12 months after start of treatment)	
Adenoid size (A/C ratio and (Bolesławska scale)	Mean ± SD [%]	73.8 ± 13.7	68.1 ± 14.8	**< 0.001**
	I°	2 (3.1%)	2 (3.1%)	**< 0.001**
	II°	12 (18.8%)	31 (48.4%)	
	III°	50 (78.1%)	31 (48.4%)	
Adenoid mucus coverage (MASNA scale)	0	9 (14.1%)	22 (34.4%)	**0.038**
	1	21 (32.8%)	14 (21.9%)	
	2	18 (28.1%)	17 (26.6%)	
	3	16 (25.0%)	11 (17.2%)	
Tympanogram, n = 61	AA	18 (28.6%)	28 (45.9%)	0.358
	AB/BA	1 (1.6%)	1 (1.6%)	
	AC/CA	8 (12.7%)	6 (9.8%)	
	BB	17 (27.0%)	13 (21.3%)	
	BC/CB	10 (15.9%)	6 (9.8%)	
	CC	9 (14.3%)	7 (11.5%)	
	A	18 (28.6%)	28 (45.9%)	0.090
	B	28 (44.4%)	20 (32.8%)	
	C	17 (27.0%)	13 (21.3%)	

MMT: maximal medical treatment

Table 4 Impact of the applied maximal medical treatment on adenoid size, adenoid mucus coverage, and tympanograms in the medical and medical–surgical groups

Characteristic		Treatment		*P*-value
		Medical	Medical–surgical	
N		46 (71.9%)	18 (28.1%)	
Age at the first visit (years)	Mean ± SD	3.9 ± 1.0	4.0 ± 1.2	0.581
Gender	Female	22 (47.8%)	9 (50.0%)	0.349
	Male	24 (52.2%)	9 (50.0%)	
Adenoid size (A/C ratio and (Bolesławska scale) – first visit	Mean ± SD [%]	71.6 ± 13.9	79.4 ± 11.7	**0.029**
	I°	2 (4.3%)	0 (0.0%)	0.663
	II°	7 (15.2%)	5 (27.8%)	
	III°	37 (80.4%)	13 (72.2%)	
Adenoid size (A/C ratio and (Bolesławska scale) – final visit	Mean ± SD [%]	63.3 ± 12.6	80.6 ± 12.6	**< 0.001**
	I°	2 (4.3%)	0 (0.0%)	**0.002**
	II°	28 (60.9%)	3 (16.7%)	
	III°	16 (34.8%)	15 (83.3%)	
Impact of MMT on A/C ratio change	Mean ± SD [%]	-8.4 ± 10.4	1.1 ± 9.9	**0.003**
	Decrease	31 (67.4%)	6 (33.3%)	**0.010**
	No change	10 (21.7%)	4 (22.2%)	
	Increase	5 (10.9%)	8 (44.4%)	
Adenoid mucus coverage (MASNA scale)—first visit	0	4 (8.7%)	5 (27.8%)	0.115
	1	15 (32.6%)	6 (33.3%)	
	2	16 (34.8%)	2 (11.1%)	
	3	11 (23.9%)	5 (27.8%)	
Adenoid mucus coverage (MASNA scale)—final visit	0	19 (41.3%)	3 (16.7%)	**0.015**
	1	12 (26.1%)	2 (11.1%)	
	2	11 (23.9%)	6 (33.3%)	
	3	4 (8.7%)	7 (38.9%)	
Impact of MMT on adenoid mucus coverage change	Decrease	29 (63.0%)	5 (27.8%)	**0.002**
	No change	11 (23.9%)	3 (16.7%)	
	Increase	6 (13.0%)	10 (55.6%)	
Tympanogram—first visit	AA	16 (34.8%)	2 (11.8%)	**0.007**
	AB/BA	0 (0.0%)	1 (5.9%)	
	AC/CA	5 (10.9%)	3 (17.6%)	
	BB	8 (17.4%)	9 (52.9%)	
	BC/CB	8 (17.4%)	2 (11.8%)	
	CC	9 (19.6%)	0 (0.0%)	
	A	16 (34.8%)	2 (11.8%)	**0.047**
	B	16 (34.8%)	12 (70.6%)	
	C	14 (30.4%)	3 (17.6%)	
Tympanogram—second visit	AA	27 (61.4%)	1 (5.9%)	**< 0.001**
	AB/BA	1 (2.3%)	0 (0.0%)	
	AC/CA	4 (9.1%)	2 (11.8%)	
	BB	3 (6.8%)	10 (58.8%)	
	BC/CB	3 (6.8%)	3 (17.6%)	
	CC	6 (13.6%)	1 (5.9%)	
	A	27 (61.4%)	1 (5.9%)	**< 0.001**
	B	7 (15.9%)	13 (76.5%)	
	C	10 (22.7%)	3 (17.6%)	

Table 4 (continued)

Characteristic		Treatment		*P*-value
		Medical	Medical–surgical	
Impact of MMT on tympanogram change	Improvement	16 (36.4%)	2 (11.8%)	**0.034**
	No change	26 (59.1%)	11 (64.7%)	
	Deterioration	2 (4.5%)	4 (23.5%)	
Allergies	Not tested	28 (60.9%)	15 (83.3%)	
	No	9 (19.6%)	3 (16.7%)	0.090
	Yes	9 (19.6%)	0 (0.0%)	
Rhinitis after MMT (week in a month)	Mean ± SD [%]	1.2 ± 0.7	1.7 ± 1.0	**0.031**

MMT: maximal medical treatment

Table 5 Improvement in symptoms in the medical and medical–surgical groups before making the decision about surgery

Symptom		Treatment		P-value
		Medical	Medical–surgical	
n		46 (71.9%)	18 (28.1%)	
Ailments	Improvement	42 (91.3%)	8 (44.4%)	**< 0.001**
	No improvement	4 (8.7%)	10 (55.6%)	
Recurrent upper respiratory tract infections	Improvement	41 (89.1%)	8 (44.4%)	**< 0.001**
	No improvement	5 (10.9%)	10 (55.6%)	
Snoring	Improvement	36 (78.3%)	11 (61.1%)	0.131
	Periodically improvement	4 (8.7%)	1 (5.6%)	
	No improvement	6 (13.0%)	6 (33.3%)	
Mouth breathing	Improvement	19 (41.3%)	4 (22.2%)	**0.041**
	Periodically improvement	6 (13.0%)	0 (0.0%)	
	No improvement	21 (45.7%)	14 (77.8%)	
Rhinitis	Improvement	41 (89.1%)	8 (44.4%)	**< 0.001**
	No improvement	5 (10.9%)	10 (55.6%)	
Nasal obstruction	Improvement	42 (91.3%)	9 (50.0%)	**< 0.001**
	No improvement	4 (8.7%)	9 (50.0%)	

the adenoid decreased by an average of 8.4%, and in the second, it increased by an average of 1.1%. In addition, endoscopic assessment of adenoid mucus coverage revealed a decrease in the degree of mucus based on the MASNA scale, which was not found in the group that underwent surgery. Thus, these groups responded differently to pharmacological treatment. It seems that the clinical response to MMT (or the lack of it) is crucial in making the decision to undergo surgery. This has been confirmed by numerous studies that have shown a positive effect of conservative treatment with nasal steroids on the reduction of adenoid symptoms, but few studies have analyzed changes in adenoid size [7]. In addition, the effect obtained following treatment only lasted about 3–6 months after the discontinuation of treatment [24]. Therefore, to maintain the positive effect, this should be a long-term therapy. However, to reduce the risk of side effects, breaks from treatment may be applied, including periods in which clinical improvement is naturally observed, holidays, and short weekly periods each month [23]. A similar treatment regimen with steroids with short-term breaks was proposed by Berlucchi, exhibiting positive effects, although his sample only included 21 patients [26]. Demirhan et al. reported that there was no need for surgery in 70% of children after the end of continuous cycles of treatment with nasal steroids [33]. This result is consistent with ours. However, Demirhan et al. did not employ breaks, and the described follow-up period was much shorter, with the results assessed after just eight weeks of therapy. In addition, only 25 children were analyzed [33]. Ciprandi also reported that after eight weeks of treatment, endoscopic assessment showed a reduction in the A/C ratio in 72% of patients [20].

There are discrepancies among studies in terms of the steroid dose puffed into each nostril. Bhat et al. recommended the application of 100 µg of mometasone furoate spray in each nostril once a day for 12 weeks. In contrast, we used fifty micrograms [34]. Similar to our findings,

Bhat et al. reported a reduction in snoring in 66% of patients, a reduction in recurrent colds in 85% of patients, an improvement in nasal patency in 65% of patients, and decreased adenoid size in 71% of patients based on endoscopic assessment [34]. Twice the topical steroid dose did not increase the effectiveness of treatment in our study. It should be noted that the studied group of children was older than in our study, as the children were 6 to 15 years of age. However, it is important to note that the effect of nasal steroids administered by a spray is local. No study has qualitatively assessed the mucus coverage of the adenoid or proposed scales for this purpose. In our study, we used the previously developed MASNA scale, which appears to be useful for assessing the effects of treatment, as the degree of mucus can affect the severity of adenoid symptoms [23].

In the era of united airways theory, links between allergies and upper and lower respiratory tract illness are assumed based on the idea of a single integrated anatomical and functional unit [35]. Hence, allergies can affect the degree of nasal and adenoid mucus and, consequently, adenoid symptoms. However, in patients with rhinitis allergies, the effect of treatment with topical steroids for adenoid hypertrophy was found to be worse than in those without allergies [36]. In the group we studied, 67% of the patients had not been previously tested for allergies. Considering the analysis of Eren et al. cited above, the percentage of allergies in children with AH symptoms may reach 65% [27]. Therefore, in our opinion, when using maximal conservative treatment, antihistamine drugs should be used even in the initial period of treatment, as AH is diagnosed between three and four years of age, whereas AR is usually diagnosed in 6–7-year-old children. Moreover, even parents of children without confirmed allergies insist to repeat the diagnostics in subsequent years. Certainly, this is due to the fact that they themselves are often allergic, and the symptoms observed in children are often similar to those of their allergic parents [7]. Selective histamine blockage caused by second-generation antihistamine drugs decreases the release of cell mediators from mast cells, reducing epithelial inflammation and decreasing mucosal thickness [37]. This was confirmed in our observations of responders (medical group), as the MASNA scale indicated improvement in 63% of the children in the medical group.

The MMT for AH we applied also included nasal cleaning with a hypertonic saline solution. Marcisio et al. demonstrated that it is helpful to rinse the nasal mucosa of residual secretions and allergens [31]. Such treatment does not pose an increased risk of side effects and can significantly support the effects of treatment. In the overall study group, after systematic nasal cleaning along with other elements of treatment, we observed a reduction in snoring (73.4%), catarrh severity (76.6%), and nasal patency (79.7%). This was also confirmed by endoscopic improvement in 53% of the patients based on the MASNA scale (Tables 1 and 2).

Local bacteria supplementation was used to modify the nasal and nasopharyngeal microbiomes in summer during the break from steroid and antihistamine treatment. We observed a reduction in recurrent upper respiratory tract infections in 76.6% of the children. A similar improvement was reported by Ciprandi et al. [11]. This indicates that probiotics can restore the native nasal ecology, which may be protective against recurrent infections [35].

The proposed long-term therapy with breaks did not reduce the effectiveness of treatment compared to the results obtained by other authors, and it made it possible to safely extend the period of therapy [38]. However, it is still unclear how long it should be continued [39]. Previous studies have shown that the process of adenoid involution with age is slow, but based on our observations, adenoid symptoms usually become much less intense after 6 years [2].

In addition, it should be noted that the group that responded to conservative treatment exhibited an improvement in tympanometry, which was not observed in the group undergoing surgical treatment. Importantly, this group also showed an improvement in the degree of adenoid mucus coverage based on the MASNA scale. This is a surprising result, as Griffin does not recommend the use of antihistamines drugs and/or decongestants in otitis media with effusion (OME) in children [40]. Rodit et al. indicated that the effect of antihistamines and steroids may even be disadvantageous in the case of isolated OME. However, the effect may be different in the case of AH coexistence, highlighting the need for further research to better understand the treatment effects of these medications in this subgroup of patients [41]. In our analyses, these medications were helpful in the responder group and constituted an important element of success in medical therapy. An explanation for this may found in the studies performed by Colins and Ulualp [42, 43]. In an experimental analysis, Colins et al. showed that histamine levels in adenoid tissue are higher in children with OME than in healthy children [42]. Moreover, Ulualp et al. confirmed that the concentration of mast cells in the subepithelial layer of adenoid tissue is higher in children with OME than in those without OME [43].

Despite promising results, this study has some limitations. For some children, we were unable to complete the full one-year treatment and assess its results due to parental decisions to either continue treatment or opt for surgery. As a result, the compared groups were not equal in quantity. We also calculated the overall percentage of children who received medical or medical–surgical treatment for the entire group of 69 patients who began the proposed treatment protocol. We believe that further studies with larger sample sizes are required to confirm the effectiveness of long-term MMT in children living in temperate climate zones. Additionally, it

was challenging to assess the patients' allergy rates, as most of the children had not yet been formally diagnosed with allergies, as shown in the patient characteristics table.

Conclusions

The applied combined therapy had a positive treatment effect and led to improvements in a large group of patients, using all available and, in our opinion, effective pharmacological agents. The applied treatment breaks resulted in a low number of therapeutic side effects. This strategy should be used in the initial period of treatment prior to surgical treatment and continued in responders. We recommended a stepwise medical therapy approach, where surgical treatment is only considered after a lack of improvement following six–nine months of therapy. This therapy should also be considered in children with contraindications for surgery.

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Declarations

Conflict of interest The authors declare that they have no competing interests.
Ethic approval and consent to participate.
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150th Anniversary of global adenoid investigations: unanswered questions and unsolved problems

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Although the problem of adenoid hypertrophy (AH) has been diagnosed and treated by doctors and scientists from around the world for the last 150 years, there is still no consensus regarding appropriate diagnosis, conservative treatment options, and qualification for surgery. This manuscript presents current knowledge on these issues and compares diagnostic methods and the effectiveness of treatment options. Factors that may influence the obtained treatment results are also described, and a questionnaire is proposed to compare the results of treatment. The objective of drawing attention to this problem is to obtain better results from conservative treatment in the future and better-qualified patients for surgical treatment.

KEYWORDS

adenoid hypertrophy, adenoid hypertrophy treatment, conservative treatment, AH, adenoid surgery

Introduction

Although the pharyngeal tonsil was first discovered and described by Conrad Victor Schneider in 1,661 as a prominent nasopharynx structure, it was only 150 years ago, in 1873, that the medical world started to concentrate its attention on this important field of children's disease (1, 2). Such interest was motivated by Hans Wilhelm Meyer's second publication, "Ueber adenoide Vegetationen in der Nasenrachenhohle," in Arch. f. Ohrenheilkunde 1873, T-11 Bd. S. 211. VIII B., S. 120 and 241. Meyer is the father of the term "adenoid" and published the first report on an adenoid surgical resection in 1868 (**Figure 1**). However, although this first scientific report was significant, it attracted little attention. What would be the breakthrough manuscript was published by Wilhelm Meyer five years later, when he described his observations and experience in the surgical treatment of adenoids (2). From that time on, the medical world started paying more attention to this important field of disease. Specifically, the role and function of this nasopharyngeal structure in the pathogenesis of recurrent upper respiratory tract infection and rhinorrhoea began to be investigated by doctors. Adenoid hypertrophy (AH) was also found to be related to otitis media with effusion (3). Moreover, the concept of a "united airway disease" suggested that AH and rhinitis may have impact on the lower respiratory tract, which was confirmed in children suffering from asthma (4).

AH is one of the most common disorders in children. All associated indications, such as mouth breathing, snoring, nasal blockage, chronic rhinitis, and nasal speech are called "adenoid symptoms". The consequent obstruction of the nose may also cause recurrent

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FIGURE 1
Hans Wilhelm Meyer.

sinusitis, asthma, sleep apnoea, and otitis media with effusion. In some cases, AH may cause serious health deterioration and impair child development (5). For instance, Bitar et al. showed that 57.7% of young children suffering from nasal blockage and admitted to ENT outpatient clinics had AH (6).

Diagnosis

What is the best method for diagnosing adenoid hypertrophy?

The father of adenoid diagnostics, Hans Wilhelm Meyer, used his finger to explore the patient's mouth to confirm hypertrophy. He described his own methods of diagnosis in 1868 and was the first to perform resection surgery with an adenotome (7). Since then, many diagnostic methods have been introduced in the search for the most accurate approach, as well as the one that is most comfortable and least burdensome for patients, most of whom are young, not well cooperative children. Invasive techniques and imaging technology were later implemented. For the first group of diagnostic techniques, physical examinations were performed through the mouth or nose. For many years, doctors would palpate the adenoids with a finger then use the less traumatic approach of transoral mirror examination. The development of rigid and later flexible nasopharyngoscopies allowed for a pharyngeal examination to be performed through the nose. Thinner rigid nasopharyngeal endoscopy (RNE) and flexible nasopharyngeal endoscopy (FNE) became common methods for nasopharyngeal examination. Additionally, video fluoroscopy and acoustic rynomanometry for nasal diagnosis were developed. The second diagnostic tool group consisted of lateral x-ray of the nasopharynx (lateral cephalogram), ultrasonography, computer tomography (CT), and magnetic resonance imaging (MRI) (8–12).

The results of these initial tests were often based on the doctors' own experiences of feeling what is often immeasurable. Other results are based on measurable parameters, which do not always have to be related only to AH. In some cases, the condition may be simulated by other reasons (i.e., thermal seasons). Other factors related to nose and nasopharyngeal obstruction include nasal concha hypertrophy, nasal septal deviation, polyps, and allergic rhinitis (9, 13). The presence of such conditions can make it difficult to objectify and compare the results. Newly introduced diagnostic methods should still be compared to the results of transoral mirror examination or nasal endoscopy to ensure effectiveness (9, 12–14). However, this only occurs in some cases. Furthermore, in selected cases, the results of the intraoperative mirror exam may not correlate with preoperative FNE. Such circumstances may arise in children with small- and medium-sized adenoid hypertrophy (A/C ratio beneath 75%) (9). Moreover, Patel suggests that intraoperative mirror examination performed in a horizontal position in anaesthesia with relaxation may also be fraught with observation errors (9).

The sensitivity and specificity of lateral x-rays of the nasopharynx (lateral cephalometry) have reached 61%–75% and 41%–96% respectively (10, 15). More objective results are achieved when the diagnostician takes an x-ray picture at the end of patient's inspiration phase. This is especially difficult in the case of young, non-cooperating, and often frightened children (16). According to Major et al., the size of the adenoid is often overestimated in lateral cephalometry. As a result, lateral x-rays are useful for measuring the free airway space between the adenoid and soft palate (15). This is due to the fact that the diagnostic results of lateral cephalometry are shown in a single two-dimensional summation picture (15–17). As an alternative, lateral cephalometric radiographs are a simple, non-expensive, sufficiently informative method. Moreover, new digital x-ray apparatuses decrease radiation expousure (15, 17). While Mlynarek at al. did not find a correlation between lateral cephalometry and obstructive symptoms scores (OSS), a relationship has been identified between FNE and OSS (18). However, in another study, Caylakli found a correlation between the results of lateral cephalometry and those of FNE (19).

The high radiation doses of other available imaging methods, such as computer tomography (CT), cone bean computer tomography (CBCT), and time consuming such as magnetic resonance imaging (MRI) exclude these methods from being used repeatedly (20). From that reasons promising seems to be ultrasonography of the adenoid tissue. Wang et al. tried to assess

AH with ultrasonography, and despite the encouraging results, further evaluation is needed (21).

A definite advantage of invasive diagnostics, in addition to the static assessment of anatomical structures, is their ability to obtain dynamic information on the functioning of the nose and nasopharynx. In these examinations, a colour image is obtained, which allows for differentiation between the physiological and inflammatory conditions of the mucous membrane, as characterised by the type of mucous in the nose and its coverage of the adenoids.

Invasive nasopharyngeal diagnostics may cause discomfort and pain. In the absence of the child's cooperation, general anaesthesia is required, but such circumstances are rare for an experienced paediatric laryngologist. According to Ysunza et al., video fluoroscopy of the nasopharynx shows high sensitivity (100%) and specificity (90%). Unfortunately, this diagnostic tool produces a 260 micro-sievert irradiation dose (10, 15). Flexible nasopharyngoscopy is less traumatic than rigid endoscopy, and mentioned above, it may be performed without anaesthesia and provide important information about the nose, nasopharynx, and adenoid state (22, 23). In the context of COVID-19 tests, in the patients' opinions, this examination is less painful than testing with a COVID-19 nasal-swab. The sensitivity and specificity of flexible nasopharyngoscopy in the assessment of AH have reached 97.3% and 72.7%, respectivly (24). Today, flexible endoscopic examination should be the gold standard in AH examination and serve as a reference test for newly introduced diagnostic methods.

What should be appreciated for adenoid hypertrophy classification?

In clinical adenoid evaluation in children, the percentage of nasopharyngeal space occupied by the adenoid is most often used for adenoid size assessment. This is referred to as the adenoid-to-choana scale or ratio (A/C scale, A/C ratio) and is usually measured with an accuracy of up to 5%. For better assessment of patient groups, different anatomical and clinical classifications are used. In fact, many authors have introduced their own AH classifications. Although they are often similar, each may contain its own modified concept of the anatomical assessment of nasopharyngeal structures in relation to a particular clinical condition. The most common classification has been proposed by Cassano and is based on a four-step pictorial scheme describing the occupation of the nasopharynx by the adenoid (I° - 0%–25%, II° - 26%–50%, III° - 51%–75%, or IV° - 76%–100%) (25). In another five-step scale introduced by Zalzal, 0° indicates 0% obstruction of the choanae,1° less than 40% obstruction, 2° 41%–70% obstruction, 3° 71%–90% obstruction, and 4° complete obstruction (91%–100%), with adenoid tissue touching the relaxed soft palate (26). Another three-degree classification of AH was proposed by Parikh and Bolesławska (27, 28). Some of these classifications also differentiate relations between the adenoid and eustachian tube (27, 29). Flexible endoscopic adenoid investigation also allows for the classification of mucous coverage of the adenoid. For these reasons, the Mucus of Adenoid Scale by Nasopharyngoscopy Assessment (MASNA) was proposed. This four-point classification scheme accounts for the amount of mucus covering the adenoid (0° corresponds to no mucus, 1° describes the residue of clear watery mucus, 2° indicates some amount of dense mucus, and 3° indicates copious, thick, dense mucus; **Figure 2**) (23). In light of many proposed and used scales, there is still an important question that remains: what degree of AH is indicative of a large adenoid which should be surgically removed? This would help unify the results and facilitate further analysis.

What does it mean for an adenoid to be large?

In our daily practice, we often encounter patients who have previously been examined by other doctors who, based on anterior rhinoscopy and symptoms reported by parents, declare that the adenoid is large and suggest its removal. Unfortunately, these statements often do not correlate with endoscopic examination of the nasopharynx. Our intraoperative comparison of adenoid size with preoperative endoscopic adenoid assessment indicated that a 75% A/C ratio or more is equivalent to an intraoperatively removed large adenoid (24). For this reason, the classification proposed by Cassano seems to be more adequate for AH assessment because the degree of AH in this scale is equal to a large adenoid, and, in this case, adenoidectomy should be considered (25).

Does the adenoid involute with age? Is it worth waiting for an adenoidectomy?

For the most part, knowledge about adenoid tissue involution is based on Scammons's theory, which is over 100 years old, and ENT doctors' own experiences in treating children (30). According to Scammon's curves, adenoid tissue grows during childhood, leading to involution in adulthood (30). Still, there is a general lack of longitudinal observational studies on adenoid development in children, and only three are based on lateral nasopharynx x-rays studies. As shown above, lateral cephalometry may overestimate adenoid size and should be used specifically for measuring the narrowest airway space between the nose and the nasopharynx (15, 17, 31, 32). Our study showed that the involution of the adenoid proceeds slowly (24). Endoscopic examinations in the analysed group of preschool children indicated that in only 7.9% of the children, adenoid size changed by more than 15% on the A/C scale after one year of observation. In 21.6% of children, this change occurred over a period of two years, and over a period of three years in 45%.

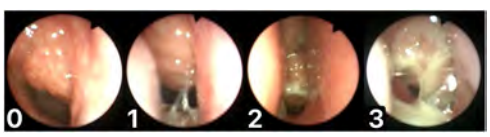

FIGURE 2
Mucus of adenoid scale by nasopharyngoscopy assessment (MASNA), 0—no mucus, 1—residue of clear watery mucus, 2—some amount of dense mucus, 3—copious thick dense mucus.

These findings are similar to the results of the longitudinal lateral cephalometric studies performed by Yamada, which showed that an overgrowth of adenoids appeared in preschool children, but there were no significant changes in the adenoid size at 8–12 years age (32). The growth and development patterns of nasopharynx lymphoid tissues are different for each patient. In our opinion, there is still a need for an accurate and broad analysis of adenoid involution in children with the use of objective adenoid size assessment.

Treatment

What is an adequate conservative medical treatment for adenoid hypertrophy?

Various methods of conservative treatment of AH have been used thus far. Their results are often difficult to evaluate because many studies have not undertaken a classic assessment of the size of the adenoid or its mucus coverage, instead only analysing the reduction of ailments and adenoid symptoms or, for example, performing a spirometry test. Therefore, the maximal conservative medical treatment of AH and how long it should last is still not known.

Intranasal topical steroids

Intranasal steroid treatment has been the most common treatment for AH and related symptoms for many years. Numerous studies have confirmed the beneficial effect of topical steroids on complaints related to AH or for decreases in adenoid symptoms (**Table 1**) (33–46). Significantly less publications refer to the objective postoperative assessment of adenoid size. According to Jazi at al., adenoid tissue regression after steroid treatment in FNE examination was less significant than what would be considered clinical improvement (44). However, *in vitro* clinical trials showed some impact of corticosteroids on reducing adenoid tissue proliferation (47). These effects were confirmed by the identification of the GCR-α and GCR-β human glucocorticoid receptors in adenoid tissue (48). These two receptors are ligands for glucocorticoid and regulate the tissue response for steroids (48). It should be emphasized that there is

TABLE 1 Research on the effect of topical steroids on adenoid size or symptoms.

Author/ year/ country	Age	Number of patients treated with steroids	Medication	Time of treatment	Time of final results counting	Main results
Cengel (33) 2005 Turkey	3–15	122	MF	6 weeks	at the end of therapy	improvement of OME in 42.2% of children
Ciprandi (34) 2007 Italy	3–6	139	F	8 weeks	at the end of therapy	reduction of A/C ratio in 72% patients
Demirhan (35) 2010 Turkey	4–16	25	MF	8 weeks	at the end of therapy	symptoms improvement, adenoidectomy was not necessary in 76% of children
Mohebi (36) 2014 Iran	2–11 (2–4 and 5–11)	51	MF	3 months	at the end of therapy	improvement
Gupta (37) 2015 India	4–12	55	MF	4 weeks	at the end of therapy	improvement
Monga (38) 2020 India	3–11	30	MF	8 weeks	at the end of therapy	improvement
Rezende (39) 2015 Brazil	4–8	55	MF	6 weeks	at the end of therapy	decrease of adenoid size
Hassanzadeh (40) 2016 Iran	4–12	20	MF	4 weeks	at the end of therapy	decrease of adenoid size
Lepcha (41) 2002 India	3–12	13	B	8 weeks	at the end of therapy	**no improvement**
Berlucchi (42) 2008 Italy	3–7	21	MF	1–3 months before surgery or 15–31 months (mean 23) (2 weeks every month, suspended during the summer)	Before surgery (9 children) or at the end of the maintenance therapy (12 children)	Long-time MF therapy may obtain successful results
Criscuoli (43) 2003 Italy	Mean 3,8	53	B	26 weeks	24,52,100 weeks after treatment	relevant clinical improvement in 45% children immediately after therapy, finally, after 2 years 70% children performed surgery
Jazi (44) 2011 Iran	2–10	20	MF	6 weeks	1 and 8 weeks after treatment	adenoid regression in FNE was less significant than clinical improvement
Bhargava (45) 2014 India	2–12	100	MF	24 weeks	24 weeks after treatment	clinical improvement
Zwierz (46) 2022 Poland	3–6	165	MF	3 months	3 to 6 months after end of the treatment	no long-time effect of intranasal mometasone furoate on adenoid size, its mucus

MF, mometasone fluroate; F, flunisolide; B, beclomethasone.

a lack of studies on the long-term outcomes of AH treatment with nasal steroids on adenoid size in children. Almost all the existing research has analysed changes of adenoid symptoms or measured adenoid size (A/C ratio) immediately after conservative treatment and not after a leeway period without the usage of topical steroids. The reports presented by Criscuoli are staggering, indicating that 70% children undergo adenoidectomy in the two-year follow-up period after treatment, despite the fact that immediately after treatment, 45% of children achieve significant improvement in their symptoms (43). Our long-term results 3–6 months after the discontinuation of medication did not indicate an adenoid size change and suggest that the therapy should be used continuously for a long-term period. A low rate of side effects allows for these steroids to be used topically for a long period of time (43, 46, 49, 50). This tendency is especially visible in recent works, where the period of use of nasal steroids has been extended (42, 43).

Antihistamine drug therapies

It is estimated that 20%–40% of children worldwide are affected by allergic rhinitis (AR) (51, 52). A study performed by Eren et al. showed that skin prick tests were positive in 65.2% of young patients with AH symptoms (53). The prevalence of AH has been increased in children with allergies, which means that this treatment could only be effective in this group of patients (54). However, there is a discrepancy in age predominance in children diagnosed with AH and AR. AH is diagnosed between 3 and 4 years of age, whereas AR is usually diagnosed in children 6–7 years old. Moreover, the remaining group of patients with nonallergic rhinitis was not homogenous. These cases included local allergic, drug-induced, gustatory, atrophic, occupational, hormonal, cold-air induced, and idiopathic rhinitis. It seems that in this nonallergic rhinitis group, the most common form is local allergic rhinitis (LAR). The incidence of LAR in children ranges from 3.7% to 66.6% (55). LAR seems to be an underdiagnosed entity and not considered for the doctors.

Both allergic and nonallergic rhinitis are cases of chronic rhinitis characterised by the presence of inflammatory cells that act on the nasal mucosa. Activation of the mast cells in nonallergic rhinitis cause histamine and a variety of other mediators (e.g., eosinophil chemotactic factor of anaphylaxis, PAF, leukotrienes, and prostaglandins) to release that exacerbate the inflammatory reaction (56). The release of histamine also acts chemotactically on neutrophils. Since a significant group of children with AH may be affected by both AR and LAR, local or systemic antihistamine treatment may be initiated in the case of strong symptoms, such as sneezing, rhinorrhoea, congestion, and nasal itching. The use of antihistamine therapy in patients with adenoid symptoms may be considered and should be further investigated. This treatment could be applied in all AH patients and continued for patients whose adenoid symptoms decrease after initial treatment.

Hypertonic saline solutions

This type of solution is used as an auxiliary and has been shown to be highly effective in cleansing the nasal mucosa of residual secretions and allergens. Hypertonic solutions are more effective in this respect; it is also important that the effectiveness in cleansing the mucosa increases with the volume of solution used (57). Such solutions should be used as a supportive treatment for adenoid symptoms.

Halotherapy

The release of micronized medical iodized sodium chloride in indoor climate-controlled conditions is another option for AH treatment. A study performed by Gelardi showed higher percentages of adenotonsillar tissue reduction in children after 10 daily sessions of micronized salt inhalation in a salt room when compared to placebo (58). This result was not statistically significant, however, and the authors concluded that a large sample of patients would be needed to show statistically significant rates of adenoid reduction.

Adrenomimetic agents

Although one study has shown the supporting effect of using oxymetazoline in AH treatment with nasal steroids, the lack of symptoms of rhinitis medicamentosa, and the rebound effect on the mucous membrane, most authors do not recommend their chronic use due to increasing rebound nasal congestion (50, 59).

Antibiotics

Even in the latest research by Zuo et al., it has been shown that the adenoid is a habitat for aerobic bacteria that can affect the development of AH, and the associated symptoms and appropriate antimicrobial therapy seem to be obvious (60). In the past, several weeks of antibiotic therapy have been used to treat AH, but this method was discontinued due to the negative impact of systemic antibiotic use on the entire microbiome of a child's body (44, 61). Currently, the local supplementation of bacteria to modify the nasal and nasopharyngeal microbiomes is more popular (62). Another approach is a 12-week treatment with OM85-BV, which may improve the Th1 immune response by weakening the local inflammatory response in the adenoids (63).

Proton pump inhibitors

Some studies have indicated a correlation between AH and gastroesophageal reflux, or AH and laryngo-pharyngeal reflux (64–67). Sagar demonstrated that adenotonsillectomy resulted in complete resolution of GER in 80% of children and improvement in 20% (66). However, in contrast, Iqbal's did not support the efficacy of PPIs for adenoid hypertrophy in children (68).

Anti-leukotriene therapies

Recently, some publications have pointed to the effectiveness of treating obstructive sleep apnea (OSA) with anti-leukotriene drugs for three months and adenoid hypertrophy (**Table 2**) (69–73). Ras showed better outcomes for oral montelukast with intranasal steroid in the treatment of AH than single-use mometasone (69). This is consistent with Tuhanoglu et al.'s findings, who described better symptom recovery in children treated with combined montelucast and mometasone furoate therapy; however, their objective assessment of adenoid size measured by lateral cephalometry showed no difference between this group and groups treated with mometasonel or montelucast

TABLE 2 Review of similar studies comparing effectiveness of anti-leukotriene therapy in children with adenoid hypertrophy. M: montelukast; MF: mometasone furoate.

Author year country	Age	Number of patients treated with montelucast/Control group	Medication	Time of treatment	Time of final result count	Main results
Ras (69) 2020 Egipt	3–10	50/50	M+MF Control MF	3 months	At the end of therapy, 3 months after treatment	Endoscopic A/C ratio examination Significantly better improvement over controls
Tuhanoglu (70) 2017 Turkey	4–10	30/30/30/30	M MF M+MF Placebo	3 months	At the end of therapy	Lateral cephalograms: Similar adenoid to air passage improvement in all groups, except placebo Best recovery in symptoms score in combined group
Shokouhi (71) 2015 Iran	4–12	30/30	M Placebo	3 months	At the end of therapy	Lateral cephalograms: Reduction of more than 25% in adenoid size in 76% treated patients vs. control (3.3%) Nasal endoscopy findings: Significant difference between groups after treatment
Liu (72) 2017 China	–	69/69	M+MF MF		At the end of therapy	M+MF more effectively reduced the adenoid nasopharynx ratio
Goldbart (73) 2012 Israel	2–10	23/23	M Placebo	3 months	At the end of the study	Lateral cephalogram radiography Decrease of the nasopharyngeal ratio in group treated with montelukast

alone (70). A study performed by Goldbart et al. showed that the adenotonsilar tissue of children with OSA contained higher leukotriene levels than that with infectious tonsilitis, and for this reason, this anti-leukotriene therapy should be applied to treat children with OSA symptoms rather than infectious adenoid symptoms (74). Montelukast is not approved for the treatment of AH and AR in Europe. Serious side effects, including hyperactive sleep disorders and depression, should be taken into consideration if anti-leukotriene therapy is to be applied (75, 76). Perhaps for this reason, all therapeutic regimens administered so far have lasted no longer than three months (**Table 2**).

Our single observations of patients treated with juvenile asthma with the use of leukotriene for a few years did not confirm its role in decreasing adenoid size. **Figure 3** shows an endoscopic view of an adenoid 8-year-old boy treated with 5 mg montelukast per day because of asthma for 3 years. Still, the role of anti-leukotriene therapies in decreasing adenoid size should be investigated.

In analysing the methods of conservative treatment described in the literature, there are still no spectacular effects or breakthroughs to be found. However, accurate diagnosis and clinical analysis should allow for the selection of patients who may be susceptible to medical treatment and able to avoid surgical treatment and related complications. Topical steroid treatment and saline irrigation should be applied before consideration of surgical treatment. Furthermore, antihistaminic drug and anti-leukotriene therapy studies should be analysed to evaluate possible benefits and side effects. Conservative treatment may be more effective with an A/C ratio beneath <75% (1–3 degrees of adenoid hypertrophy on the Cassano scale).

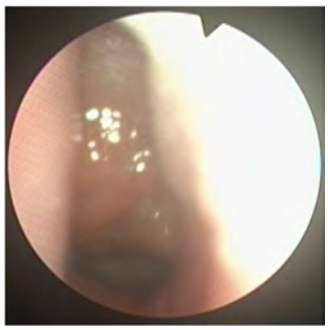

FIGURE 3
Endoscopic view of adenoid in 8-year-old boy treated with montelukast.

Is there an alternative treatment?

Some Chinese studies have indicated the efficacy of traditional Chinese herbal medicine for AH treatment in children, which showed better outcomes than Western medicine results (77–79). Zhao showed that oral Xiao-xian decoction combined with acupuncture (acupoint application) improved clinical symptoms of adenoid hypertrophy and may be suitable for long-term treatment (79).

However, there is still a variety of herbs mixtures used and no consensus on the treatment methods, as well as a lack of objectively evaluated measurements (80). Therefore, there is a need for long-term prospective clinical trials and a necessity to carry out evidence evaluation on the treatment of AH with Chinese or Western medicine to provide feasible and effective treatment options for clinics (78).

Acupuncture

Similar to Zaho's reports, the case presented by Deng showed the effectiveness of sphenopalatine ganglion electroacupuncture in widening the patency of the nasopharyngeal space in a 9-year-old boy (79, 81). While these are interesting reports, they do require further medical analysis.

What factors may influence the assessment of the effectiveness of conservative treatment?

To properly assess the effects of treatment, a questionnaire assessing the effects should be standardised. The proposed questionnaire is presented in **Table 3**. The child's parents should evaluate the change in symptoms and illness, such as snoring,

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TABLE 3 Proposed standardised questionnaire for children suspected of adenoid hypertrophy.

First visit Date: Comments:								Control Months after first visit: 1–3 / 3–6 / 7–9 / 9–12 The proposed conservative treatment was applied: Yes / No Overall improvement: Yes / No						
Season of visit	Winter/autumn			Summer/spring				Winter/autumn			Summer/spring			
Adenoid surgery	yes			no				yes			no			
Allergy	Yes		No		Not tested			Yes		No		Not tested		
Snoring	Yes		Occasionally			No		Improvement		No improvement				
Open mouth	Yes		Occasionally		No			Improvement		No improvement				
Hypoacusis	Yes		Occasionally		No			Yes		Occasionally		No		
Rhinitis	Persistent		Seasonal			No		Improvement		No improvement				
Rhinitis - weeks per month	<1		1		2	3	4	<1		1	2	3	4	
Recurrent upper respiratory tract infections	yes				no									
Courses of systematic antibiotics during the previous 6 months	0	1	2	3	4	5	6	0	1	2	3	4	5	6

sleeping with the mouth open, apnoea, periods of rhinorrhoea, allergies, recurrent infections, hearing loss, or otitis media. The season in which the assessments are performed should also be taken into consideration when evaluating the effects of the treatment. Our research has shown that seasonality itself significantly affects the condition of adenoid mucus and tympanometry, but not adenoid size (46). The results of treatment should be analysed with the most objective tool; currently, the gold standard is flexible nasopharynx examination.

Surgical adenoid treatment: adenoidectomy

Sclafani et al. reported that 90% of children with AH underwent surgery in the two-year period after the initial diagnosis (61). Slightly fewer children (70%) were operated on in Circuoli's studies regarding the effectiveness of conservative treatment of AH with intranasal beclomethasone (43). In fact, adenoidectomy is one of the most frequently performed surgeries in children (82). Bleeding is the most dangerous complications after surgery. The rate of haemorrhage following adenoidectomy is one in 200 (0.5%); taking into consideration the number of treatments performed, this affects many children. Attention should also be paid to the possibility of less frequent complications and to the child's stress associated with the first surgery. For this reason, children qualified for surgery should be well diagnosed to avoid ineffective and unjustified treatment (83). For example, adenoidectomy is recommended for the treatment of chronic rhinosinusitis in children. However, the effectiveness of adenoidectomy in chronic rhinosinusitis treatment in preschool and early-school children reaches only 47%–58% (84, 85). This could be attributed to the lack of normalised conservative treatment and appropriate diagnosis and qualification for surgery. In many cases, conservative therapy may allow time for the proper action of drugs on the adenoid and adenoid symptoms and maturation of the immunology system of the child.

Adenoid tissue regrowth after surgery may occur in 31.3% of operated children, especially those younger than five years of age (81). Such regrowth may cause a recurrence of symptoms. Some medical failures of adenoid surgery are caused by incomplete resection, whereas others can be attributed to persistent infections of the upper respiratory tract, asthma, gastroesophageal reflux (GERD), and AR (86, 87). Some of these illnesses can be diagnosed early and conservatively treated. Regrowth rate also depends on the surgeon's experience and applied surgical technique (86, 88). Yildirim showed that "blind curettage adenoidectomy" may leave up to 18% of a large residual adenoid. For total adenoid tissue resection, the nasopharynx should be controlled during the surgery by posterior rhinoscopy with the use of a mirror or trans-nasally with the use of an endoscope (88). Additionally, a study performed by Pagella et al. indicated that a greater length of the soft palate corresponds to a great risk of remnant adenoid tissue, with the authors suggesting a procedure with endoscopic control be performed, regardless of the surgical technique (89). Specifically, the authors recommended endoscopic control if the soft palate length is greater than 2.5 cm (89). The most important purpose of adenoid surgery is to precisely resect the adenoid tissue without leaving any macroscopic remnant. This increases the likelihood of resolving clinical problems related to AH. Compliance with these recommendations is expected to bring the overall rate of revision adenoidectomy down from 1.6% to 2.5% (87, 90).

Conclusion

The diagnosis of AH should be widely based on flexible endoscopy, and other newly introduced diagnostic methods should

be connected to this method. There is still no unified conservative treatment schema for AH or consensus on the length of treatment. In this respect, further research and a determination of the effects of different medical curations are indicated. Bearing in mind the fact of slow reduction of the hypertrophic adenoid under the influence of drugs, when undertaking conservative treatment, long-term therapy should be considered, with consideration of the side effects of the drugs used. The results of the treatment should be related to the most effective adenoid visualization method, which is flexible endoscopy and with the use of the Cassano and MASNA scales. Conservative therapy may be more effective when the A/C ratio remains <75%. In properly qualified patients, surgical treatment will be effective, provided that the adenoid tissue is radically resected, which is significantly more successful through intraoperative endoscopic control.

Author contributions

Conceptualization, AZ; methodology, AZ and KD; software, KD; validation, AZ and KD; formal analysis, AZ and KD investigation, AZ resources, AZ; data curation, AZ; writing—original draft preparation, AZ; writing—review and editing, KM; visualization, KM; supervision, PB; project administration, AZ. All authors contributed to the article and approved the submitted version.

Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

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Reply

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We wish to thank the author for raising the issues of how we performed the adenoid size classification and why we did not classify the condition of the nasopharyngeal orifice of the Eustachian tube [1].

The aim of our work, "The Long-Term Effects of 12-Week Intranasal Steroid Therapy on Adenoid Size, Its Mucus Coverage and Otitis Media with Effusion: A Cohort Study in Preschool Children", was to assess the change in adenoid size following intranasal steroid treatment [2]. Therefore, we assessed the long-term effects of intranasal steroid therapy on adenoid size, as measured using the percentage scale of nasopharyngeal obstruction (adenoid to choana (A/C) ratio). The size of the tonsil was analysed in relation to the height of the nasopharynx in a similar way to the assessment proposed by Wormald and Prescott [3]. To achieve a better statistical assessment, the patients were divided into groups, which we determined using the three-step Bolesławska scale, particularly the part regarding the size of the tonsil in relation to the nasopharynx [4]. This approach is described in detail in the Material and Methods section of the manuscript.

It is well-known that there are several similar classifications available for assessing the size of the tonsil in relation to the nasopharynx. For example, Cassano et al. proposed a four-step pictorial scheme for classifying nasopharyngeal obstruction by the tonsil (occupying 0–25%, 26–50%, 51–75% or 76–100% of the nasopharynx) [5]. Moreover, Zalzal et al. proposed a five-step scale for assessing adenoid size, wherein grade 0 indicates 0% obstruction of the choanae, grade 1 indicates less than 40% obstruction, grade 2 indicates 41–70% obstruction, grade 3 indicates 71–90% obstruction and grade 4 indicates complete obstruction (91–100%) of the choanae with lymphoid tissue touching the soft palate when at rest [6]. It should be noted that we primarily presented an accurate percentage analysis of the change in the adenoid and, further, that we used the secondary part of the Bolesławska scale as a means of simplification for the purposes of statistical analysis.

Our goal was to assess the change in the size of the tonsil as a result of treatment with intranasal steroids, which could indirectly cause anatomical changes in the structure of the nasopharynx. However, we did not analyse in detail the change in the relationship between the tonsil and the torus tubarius or salpingopharyngeal and salpingopalatine folds. Thus, we did not analyse the anatomical part of the Bolesławska scale. For this purpose, we preferred to use the newer scale proposed by Liu et al. [7]. Yet, we have not confirmed the long-term effects of intranasal steroid treatment on otitis media with effusion OME, which may suggest that the anatomical relations between adenoid and nasopharynx structures have not been altered. This issue requires further analysis and investigation in accordance with Liu classification because even a small change in adenoid size may be influenced by its relationship to the Eustachian tube. As described in studies conducted by Skoloudik and Hazem, anatomical relations of the adenoid with the torus tubarius may influence the results of OME treatment involving adenoidectomy, although we would like to point out

that there are many other factors that can also affect OME, for example, adenoid mucus coverage and seasonality [8–10]. Nevertheless, this issue was not a focus of our work, as we analysed the long-term effects of intranasal steroid treatment on OME.

Finally, we would like to add that we chose to use the Bolesławska scale in order to promote the researcher's work. It should be noted that the work and classification were published in Czech in the same year (2006) as the similar but much better-known Parikh classification [11]. In parts, both of these classifications concern the anatomical relations of the tonsil with the structure of the nasopharynx.

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