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Comparative Study between Different Medications in Treatment of Adenoid Hypertrophy

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Abstract

Adenoid tissue is the major constituent of Waldeyer's ring which is physiologically natural defense mechanism of the body against inhaled allergens and microorganisms. In all children, the volume of the adenoid increases with age with a maximal volume in the age group of 5-6 years, followed by a gradual decrease in volume by the age of 8-9 years. To study the effect of different medications on size and symptoms of adenoid.This study was conducted at Otorhinolaryngology Department of Al-Zahraa University Hospital and Damietta general hospital between October 2021 and October 2022. In total, 100 patients with adenoid hypertrophy with grade 3 or4 nasopharyngeal obstructions were included in this study 58 were female and 42 were males. The age ranged from 3 to 12 years. Medical treatment for adenoid hypertrophy using anti- inflammatory and anti-allergy, medications has gained importance in the last few years because of its low prevalence of complications and general good results.Medical treatment of adenoid especially combination of (antileukotriens as montelukast, anti histaminics, local corticosteroid spray as memotasone furonate) can decrease the symptoms of adenoid hypertrophy as snoring, breathing difficulty and snoring, rhinorrhea it also decrease the size of adenoid which evaluated by radiological assessment.

Keywords: Adenoid Hypertrophy - Allergic Rhinitis - Obstructive Sleep Ap*nea*

1. Introduction

Allergic rhinitis is a non-infectious chronic inflammatory disease of nasal mucosa mediated by immunoglobulin E and involved by a variety of immunocompetent cells (T, B lymphocytes, mast cells, etc.) after atopic individual contact with allergens. Adenoid hypertrophy and allergic rhinitis are common and frequently occurring diseases in children. Both of them may exist simultaneously and may cause similar clinical symptoms [1].

Majority of cases with adenoid hypertrophy are associated with infection and allergy i.e. descending infection in 33.3% of cases, ascending infection in 20% of cases and allergic rhinitis in 30% of

cases. Association of malignant sinonasal tumors, non-Hodgkin's lymphoma and HIV infections are rare i.e. 3.3% each. So, any cases of adult adenoid hypertrophy should be treated seriously to exclude the dangerous causes [2].

Adenotonsillar hypertrophy causes mouth breathing, nasal congestion, hyponasal speech, snoring, obstructive sleep apnea, chronic sinusitis and recurrent otitis media. Adenoidal hypertrophy results in the obstruction of nasal passages and Eustachian tubes, and blocks the clearance of nasal mucus[3].

Adenoid hypertrophy (AH) is an obstructive condition, with its symptomatology depending on the obstructed structure. Nasal obstruction by hypertrophic adenoid tissue can cause the patient to complain of rhinorrhea, difficulty breathing through the nose, chronic cough, post nasal drip, snoring, and/or sleep disordered breathing in children [4].

If nasal obstruction is significant, the patient can suffer from sinusitis as a result and may complain of facial pain or pressure Obstruction of the Eustachian tube can lead to symptoms consistent with Eustachian tube dysfunction such as muffled hearing, otalgia, crackling or popping sounds in the ear, and/or recurrent middle ear infections [4]. Adenotonsillectomy is a typical strategy for patients with AH but may lead to serious complications such as bleeding (4–5%) and postoperative respiratory compromise (27%), especially among young children, as well as recurrence of adenoid tissue (10–20%). Thus, non-surgical therapies have attracted considerable attention as an alternative strategy. The inflammatory mechanism proposed for AH has lead to the use of anti-inflammatory drugs to manage this condition. Leukotrienes are key inflammatory mediators in the respiratory system. These lipid mediators are involved in the pathogenesis of childhood diseases such as asthma. They are also systemically and locally involved in the process of inflammation in children with AH [5].

Human cysteinyl-leukotriene receptor-1 expression is elevated in the tonsillar tissues of children with obstructive sleep apnea (OSA) Accordingly, cysteinyl-leukotriene receptor-1, which interacts with leukotriene's and mediates the inflammatory pathway, was over expressed in adenotonsillar cells and tissues derived from children with AH thus, anti-inflammatory agents with a safe therapeutic profile may provide an interventional alternative to adenotonsillectomy [5].

Montelukast is an oral, bioavailable, cysteine-leukotriene receptor antagonist that is effective, safe, well tolerated and approved by the US Food and Drug administration (FDA) for preventive therapy of the inflammatory component in asthma and allergic rhinitis in children aged ≥1year [5].

Adenotonsillar hypertrophy and obstructive sleep apnea are associated with increased expression of various mediators of inflammatory responses in the tonsils, and respond to anti- inflammatory agents such as corticosteroids. Topical nasal steroids most likely affect the anatomical component by decreasing inspiratory upper airway resistance at the nasal, adenoidal or tonsillar levels. Corticosteroids, by their lymphocytic or anti- inflammatory effects, might reduce adenotonsillar hypertrophy Intranasal corticosteroids reduce cellular proliferation and the production of pro-inflammatory cytokines in a tonsil and adenoid mixed-cell culture system. Intranasal corticosteroids have been used in adenoidal hypertrophy and adenotonsillar hypertrophy patients, decreasing rates of surgery for adenotonsillar hypertrophy ***(3)*****.**

Intranasal corticosteroids may significantly improve nasal obstruction symptoms in children with moderate to severe adenoidal hypertrophy, and this improvement may be associated with a reduction of adenoid size [6].

Allergic rhinitis (AR) and adenoid hypertrophy (AH) are common in children and are often associated with each other. Recent studies have shown improvement of respiratory symptoms and reduction in the adenoid volume after anti-allergic

medical therapy (intranasal corticosteroids, antihistamines) [7].

A significant percentage (80%) of children suffering from AR did not present satisfactory benefits from adenoidectomy. They reported persistence or recurrence of rhinitic symptoms after surgery or only partial benefits, especially of recurrent respiratory tract infections and nasal obstruction. The local allergic persistent inflammation on nasal mucosa and adenoid tissue is probably the cause of the unsatisfactory results of adenoidectomy, therefore surgery cannot be the first therapeutic step for these children. It is important to extinguish the local inflammation by medical anti-allergic therapy to obtain improvements of nasal symptoms and to prevent adenoid regrowth [7].

2. Patients and Methods

This study was conducted at Otorhinolaryngology Department of Al-Zahraa University Hospital and Damietta general hospital between October 2021 and October 2022. In total, 100 patients with adenoid hypertrophy with grade 3 or4 nasopharyngeal obstructions were included in this study 58 were female and 42 were males. The age ranged from 3 to 12 years. Approval of this study obtained from Ethical Committee of the Faculty of medicine for girls, Al-Azhar University. Written consent was obtained from their parents prior to study.

2.1 Inclusion Criteria

Patients with adenoid hypertrophy symptoms: mouth breathing and snoring, patients aged between 3 and 18 years and both genders.

2.2 Exclusion Criteria

Patients out side the selected age, previous adenoidectomy or adenotonsillectomy, patients with other systemic diseases interfering the study (hypertension, diabetes, kidney diseases and liver diseases) and sever obstructive sleep apnea.

2.3 Methods of Research

Patients were divided into five groups each including 20 patients. There were 8 males and 12 females in group I, 12 males and 8 females in group II, 6 males and 14 females in group III, 6 males and 14 females in group IV and 10 males and 10 females in group V.

- Group I: patients received combined therapy using mometasone furate intranasal spray (2puff in each nostril 100 µg once daily) combined with oral montelukast (5mg-10mg) once daily for 12 weeks and anti histaminics (30mg twice daily for children aged 3 to 12 years and 120mg once daily for patients over 12 years).

- Group II patients received combined therapy using mometasone furate intranasal spray (2puff in each nostril 100 µg once daily) combined with oral montelukast (5-10mg) once daily for 12 weeks.

- Group III received oral montelukast either (5-10 mg)12 weeks.

- Group IV received mometasone furate intranasal spray (2puff in each nostril 100 µg once daily) either for 12 weeks.

- Group V received oral anti histaminics (30mg twice daily for children aged 3 to 12 years and 120mg once daily for patients over 12years).

All patients were subjected to clinical assessment before and after 12 weeks of treatment including:

- Taking history from the patient or his/her parents and answers the obstructive symptoms. These

symptoms are snoring, mouth-breathing, and obstructive sleep breathing. They were also asked about other symptoms: nocturnal enuresis and halitosis.

- Complete ENT examination including: Oral examination, nasal examination and ear examination.

- Radiological assessments (plain x-ray nasopharynx lateral view with open mouth were taken with a mild neck extension) for better visualization of AH. Calculation the thickness evaluated by the ratio of the adenoid thickness over the all nasopharyngeal thickness (A/N)based on Fujioka method [8].

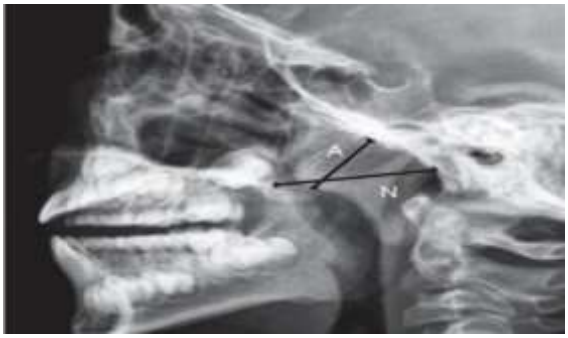


Figure 1: Method of assessing adenoid enlargement on lateral neck radiography. A) represents the distance from the point of maximal convexity of the adenoid shadow to a line along the anterior margin of the basiocciput. The nasopharyngeal measurement (N) is the distance between the posterior border of the hard palate and the antero-inferior edge of the sphenobasioccipital synchondrosis [8].

The soft tissue shadow seen in the X-ray was quantified, and the sizes of adenoid in relation to the size of the nasopharynx were graded: Mild 0-50% soft tissue shadow in nasopharynx, Moderate >50-75%soft tissue shadow in nasopharynx and Sever > 75-100% soft tissue shadow in nasopharynx.

All patients were examined by fibro optic endoscope. All patients had been evaluated and examined after 12 weeks of treatment. The evaluation included assessment of the symptoms, and all patients had been submitted for clinical examination using fibro-optic endoscopic examination and radiologically by X-ray.

Figure 2: X-ray showing sever adenoid hyper trophy pre treatment in group I.

Figure 3: X-ray showing moderate adenoid hypertrophy pretreatment in group II.

Figure 4: X ray showing sever adenoid hypertrophy pretreatment in group III.

Figure 5: Sever adenoid hypertrophy pretreatment in group IV.

Figure 6: Moderate adenoid hypertrophy pre treatment in group V.

2.4 Statistical analysis:

Data were analyzed using Statistical Program for Social Science (SPSS) version 24. Quantitative data were expressed as mean ±SD. Qualitative data were expressed as frequency and percentage. Mean (average): the central value of a discrete set of numbers, specifically the sum of values divided by the number of values. Standard deviation (SD): is the measure of dispersion of a set of values. A low SD indicates that the values tend to be close to the meaning of the set, while a high SD indicates that the values are spread out over a wider range. The following tests were done: Kruskal Willis test (KW): when comparing between more than two means (for abnormally distributed data). Chi-square test: was used when comparing between non-parametric data. Probability (P-value): P-value < 0.05 was considered significant, P-value < 0.001 was considered as highly significant and P-value > 0.05 was considered insignificant.

3. Results

In total 100 patients were included in this study, paients were divided into five groups each including 20 patients. There were 8 males and 12 females in group I, 12 males and 8 females in group II, 6 males and 14 females in group III, 6 males and 14 females in group IV and 10 males and 10 females in group V. Group I patients received combined therapy using mometasone furate intranasal spray (2puff in each nostril 100 µg once daily) combined with oral montelukast(5mg-10mg) once daily for 12 weeks and anti histaminics (30mg twice daily for children aged 3 to 12 years and 120mg once daily for patients over 12 years).Group II patients received combined therapy using mometasone furate intranasal spray (2puff in each nostril 100 µg once daily) combined with oral montelukast either patients(5mg-10mg) once daily for 12 weeks. Group III received oral montelukast either (5mg-10mg) in daily for 12 weeks. Group IV received mometasone furate intranasal spray (2puff in each nostril 100 µg once daily) either for 12 weeks. Group V received oral anti-style histaminic (30 mg twice daily for children aged 3 to 12 years and 120mg once daily for patients over 12years). As show in table 1 distribution of studied patients according to their demographic data including age and sex. There is no statistically significant difference (p-value = 0.233) between studied groups as regard sex. There were 8 males (40%) and 12 females (60%) in group I, 12 males (60%) and 8 females (40%) in group II, 6 males (30%) and 14 females (70%) in group III, 6 males (30%) and 14 females (70%) in group IV and 10 males (50%) and 10 females (50%) in group V. No statistical significant difference (p-value = 0.202) between studied groups as regard age. It was 5.9 ± 1.3 years in group I, 6.9 ± 2.02 years in group II, 6.3 ± 2.2 years in group III, 5.5 ± 2.3 years in group IV and 5.7 ± 1.8 years in group V. As shown in table 2 there is no statistically significant difference (p-value = 0.287) between studied groups as regards pre-treatment X-ray grading. There were 8 patients (40%) of grade III and 12 patients (60%) of grade IV in group I, 10 patients (50%) of grade III and 10 patients (50%) of grade IV in group II, 10 patients (50%) of grade III and 10 patients (50%) of grade IV in group III, 4 patients (20%) of grade III and 16 patients (80%) of grade IV in group IV and 8 patients (40%) of grade III and 12 patients (60%) of grade IV in group V. As shown in table 3 there is no statistically significant difference (p-value = 0.112)

between studied groups as regards pre-treatment rhinorrhea. It was present in 12 patients (60%) of group I, 12 patients (60%) of group II, 16 patients (80%) of group III, 16 patients (80%) of group IV and 18 patients (90%) of group V. No statistically significant difference (p-value = 0.107) between studied groups as regards pre-treatment mouth breathing. It was present in 20 patients (100%) of group I, 20 patients (100%) of group II, 18 patients (90%) of group III, 16 patients (80%) of group IV and 18 patients (90%) of group V. No statistically significant difference (p-value = 0.915) between studied groups as regards pre-treatment snoring. It was present in 12 patients (60%) of group I, 12 patients (60%) of group II, 10 patients (50%) of group III, 10 patients (50%) of group IV and 10 patients (50%) of group V. Statistically significant difference (p-value = 0.031) between studied groups as regard pre-treatment halitosis. It was present in 12 patients (60%) of group I, 14 patients (70%) of group II, 6 patients (30%) of group III, 8 patients (40%) of group IV and 6 patients (30%) of group V. No statistically significant difference (p-value = 0.341) between studied groups as regards pre-treatment nocturnal enuresis. It was present in 6 patients (30%) of group I, 10 patients (50%) of group II, 4 patients (20%) of group III, 8 patients (40%) of group IV and 8 patients (40%) of group V. As shown in table 4 as regards rhinorrhea, there were statistically significant differences (p-value = 0.028) between group I & group V, statistically significant differences (p-value = 0.028) between group II & group V and No statistically significant difference (p-value > 0.05) between each studied group. As regards mouth breathing, there were statistically significant differences (p-value = 0.035) between group I & group IV, statistically significant difference (p-value = 0.035) between group II & group IV and no statistically significant difference (p-value > 0.05) between each other studied groups. As regards snoring, there was no statistically significant difference (p-value > 0.05) between each other studied groups. As regards halitosis, there were statistically significant differences (p-value = 0.011) between group II & group III, statistically significant difference (p-value = 0.011) between group II & group V and no statistically significant difference (p-value > 0.05) between each other studied groups. As regards nocturnal enuresis, there were statistically significant differences (p-value = 0.047) between group II & group III and no statistically significant difference (p-value > 0.05) between each other studied groups. As shown in table 5 there is no statistically significant difference (p-value = 0.223) between studied groups as regards post-treatment rhinorrhea. It was present in 2 patients (10%) of group I, 4 patients (20%) of group II, 8 patients (40%) of group III, 6 patients (30%) of group IV and 4 patients (20%) of group V. No statistically significant difference (p-value = 0.308) between studied groups as regards post-treatment mouth breathing. It was present in 8 patients (40%) of group I, 10 patients (50%) of group II, 8 patients (40%) of group III, 10 patients (50%) of group IV and 14 patients (70%) of group V. Statistically significant difference (p-value = 0.002) between studied groups as regards post-treatment snoring. It was present in 4 patients (20%) of group I, 0 patients (0%) of group II, 2 patients (10%) of group III, 4 patients (20%) of group IV and 10 patients (50%) of group V. No statistically significant difference (p-value = 0.432) between studied groups as regards post-treatment halitosis. It was present in 8 patients (40%) of group I, 8 patients (40%) of group II, 4 patients (20%) of group III, 4 patients (20%) of group IV and 6 patients (30%) of group V. Sregardsically significant difference (p-value = 0.04) between studied groups as regard post-treatment nocturnal enuresis. It was present in 2 patients (10%) of group I, 2 patients (10%) of group II, 2 patients (10%) of group III, 6 patients (30%) of group IV and 8 patients (40%) of group V. As shown in table 6 as regard rhinorrhea, there were statistically significant differences (p-value = 0.028) between group I & group III and no statistically significant difference (p-

value > 0.05) between each other studied groups. As regard mouth breathing, there was no statistical significant difference (p-value > 0.05) between each other studied groups As regard snoring, there were statistically significant difference (p-value = 0.035) between group I & group II, statistically significant difference (p-value = 0.047) between group I & group V, statistically significant difference (p-value = 0.035) between group II & group V, highly statistical significant difference (p-value < 0.001) between group II & group V, statistically significant difference (p-value = 0.006) between group III & group V, statistically significant difference (p-value = 0.047) between group IV & group V and no statistical significant difference (p-value > 0.05) between each other studied groups.

As regards halitosis, there was no statistically significant difference (p-value > 0.05) between each other studied groups. As regards nocturnal enuresis, there were statistically significant differences (p-value = 0.028) between group I & group V, statistically significant difference (p-value = 0.028) between group II & group V, statistically significant difference (p-value = 0.028) between group III & group V and no statistically significant difference (p-value > 0.05) between each other studied groups. As show in table7 this table shows that there were statistically significant (p-value = 0.001) decreased percentage of post-treatment rhinorrhea (2 patients 10%) when compared with pre-treatment rhinorrhea (12 patients, 60%) in group I. Highly statistical significant (p-value < 0.001) decreased percentage of post-treatment mouth breathing (8 patients 40%) when compared with pre-treatment mouth breathing (20 patients, 100%) in group I. Statistically significant (p-value = 0.01) decreased percentage of post-treatment snoring (4 patients 20%) when compared with pre-treatment snoring (12 patients, 60%) in group I. No statistically significant difference (p-value = 0.206) between pre-treatment and post-treatment halitosis in group I. No statistically significant difference (p-value = 0.114) between pre-treatment and post-treatment nocturnal enuresis in group I.

As shown in table 8 this table shows statistically significant (p-value = 0.01) decreased percentage of post-treatment rhinorrhea (4 patients 20%) when compared with pre-treatment rhinorrhea (12 patients, 60%) in group II. Highly statistically significant (p-value < 0.001) decreased percentage of post-treatment mouth breathing (10 patients 50%) when compared with pre-treatment mouth breathing (20 patients, 100%) in group II. Highly statistically significant (p-value < 0.001) decreased percentage of post-treatment snoring (0 patients 0%) when compared with pre-treatment snoring (12 patients, 60%) in group II. No statistically significant difference (p-value = 0.057) between pre-treatment and post-treatment halitosis in group II. Statistically significant (p-value = 0.006) decreased percentage of post-treatment nocturnal enuresis (2 patients 10%) when compared with pre-treatment nocturnal enuresis (10 patients, 50%) in group II. As shown in table 9 this table shows statistically significant (p-value = 0.01) decreased percentage of post-treatment rhinorrhea (8 patients 40%) when compared with pre-treatment rhinorrhea (16 patients, 80%) in group III. Statistically significant (p-value = 0.001) decreased percentage of post-treatment mouth breathing (8 patients 40%) when compared with pre-treatment mouth breathing (18 patients, 90%) in group III. Statistically significant (p-value = 0.006) decreased percentage of post-treatment snoring (2 patients 10%) when compared with pretreatment snoring (10 patients, 50%) in group III. No statistically significant difference (p-value = 0.465) between pre-treatment and post-treatment halitosis in group III. No statistically significant difference (p-value = 0.376) between pre-treatment and post-treatment nocturnal enuresis in group III.

As As shown in table 10 this table shows Statistically significant (p-value = 0.001) decreased percentage of post-treatment rhinorrhea (6 patients 30%) when compared with pre-treatment rhinorrhea

(16 patients, 80%) in group IV. Statistically significant (p-value = 0.047) decreased percentage of post-treatment mouth breathing (10 patients 50%) when compared with pre-treatment mouth breathing (16 patients, 80%) in group IV. Statistically significant (p-value = 0.047) decreased percentage of post-treatment snoring (4 patients 20%) when compared with pre-treatment snoring (10 patients, 50%) in group IV. No statistically significant difference (p-value = 0.168) between pre-treatment and post-treatment halitosis in group IV. No statistically significant difference (p-value = 0.507) between pre-treatment and post-treatment nocturnal enuresis in group IV. As shown in table 11 this table shows Highly statistically significant (p-value < 0.001) decreased percentage of post-treatment rhinorrhea (4 patients 20%) when compared with pre-treatment rhinorrhea (18 patients, 90%) in group V. No statistically significant difference (p-value = 0.114) between pre-treatment and post-treatment mouth breathing in group V. No statistically significant difference (p-value = 1.0) between pre-treatment and post-treatment snoring in group V. No statistically significant difference (p-value = 1.0) between pre-treatment and post-treatment halitosis in group V. No statistically significant difference (p-value = 1.0) between pre-treatment and post-treatment nocturnal enuresis in group V. As show in table12 This table shows Statistically significant difference (p-value = 0.012) between pre-treatment and post-treatment X-ray grading in group I. Statistically significant difference (p-value = 0.014) between pre-treatment and post-treatment X-ray grading in group II. No statistically significant difference (p-value = 0.082) between pre-treatment and post-treatment grading in group III. No statistically significant difference (p-value = 0.344) between pre-treatment and post-treatment grading in group IV. No statistically significant difference (p-value = 1.0) between pre-treatment and post-treatment grading in group V.

Table 1: Comparisons between studied groups as regard age and sex:

		Groups										Stat. test	P-value
		Group I (n = 20)		Group II (n = 20)		Group III (n = 20)		Group IV (n = 20)		Group V (n = 20)			
Sex	Male	8	40%	12	60%	6	30%	6	30%	10	50%	$X^2 = 5.5$	0.233 NS
	Female	12	60%	8	40%	14	70%	14	70%	10	50%		
Age (years)	Mean	5.9		6.9		6.3		5.5		5.7		KW = 5.9	0.202 NS
	±SD	1.3		2.02		2.2		2.3		1.8			

NS: p-value > 0.05 is considered non-significant. KW: Kruskal Willis test. X^2: Chi-square test.

Table 2: Comparisons between studied groups as regard pre treatment X-ray grading:

X-ray grading		Groups										Stat. test	P-value
		Group I (n = 20)		Group II (n = 20)		Group III (n = 20)		Group IV (n = 20)		Group V (n = 20)			
pre	Grade III	8	40%	10	50%	10	50%	4	20%	8	40%	$X^2 = 5.0$	0.287 NS
	Grade IV	12	60%	10	50%	10	50%	16	80%	12	60%		

NS: p-value > 0.05 is considered non-significant. X^2: Chi-square test.
S: p-value < 0.05 is considered significant.

Table 3: Comparisons between studied groups as regards pre-treatment manifestations

Pre-treatment	Groups										X^2	P-value
	Group I (n = 20)		Group II (n = 20)		Group III (n = 20)		Group IV (n = 20)		Group V (n = 20)			
Rhinorrhea	12	60%	12	60%	16	80%	16	80%	18	90%	7.4	0.112 NS
Mouth breathing	20	100%	20	100%	18	90%	16	80%	18	90%	7.6	0.107 NS
Snoring	12	60%	12	60%	10	50%	10	50%	10	50%	0.96	0.915 NS
Halitosis	12	60%	14	70%	6	30%	8	40%	6	30%	10.6	0.031 S
Nocturnal enuresis	**6**	**30%**	**10**	**50%**	**4**	**20%**	**8**	**40%**	**8**	**40%**	**4.5**	**0.341 NS**

NS: p-value > 0.05 is considered non-significant. X^2: Chi-square test.
S: p-value < 0.05 is considered significant.

Table 4: Multiple comparisons between studied groups as regard pre-treatment manifestations

Pre-treatment		Rhinorrhea	Mouth breathing	Snoring	Halitosis	Nocturnal enuresis
I vs II	X^2	0.0	---	0.0	0.44	1.66
	p-value	1.0	---	1.0	0.507	0.197
I vs III	X^2	1.9	2.1	0.4	3.6	0.53
	p-value	0.168	0.147	0.525	0.057	0.465
I vs IV	X^2	1.9	4.4	0.4	1.6	0.44
	p-value	0.168	0.035	0.525	0.206	0.507
I vs V	X^2	4.8	2.1	0.4	3.6	0.44
	p-value	0.028	0.147	0.525	0.057	0.507
II vs III	X^2	1.9	2.1	0.4	6.4	3.9
	p-value	0.168	0.147	0.525	0.011	0.047
II vs IV	X^2	1.9	4.4	0.4	3.6	0.4
	p-value	1.68	0.035	0.525	0.057	0.525
II vs V	X^2	4.8	2.1	0.4	6.4	0.4
	p-value	0.028	0.147	0.525	0.011	0.525
III vs IV	X^2	0.0	0.78	0.0	0.44	1.9
	p-value	1.0	0.376	1.0	0.507	0.168
III vs V	X^2	0.78	0.0	0.0	0.0	1.9
	p-value	0.376	1.0	1.0	1.0	0.168
IV vs V	X^2	0.78	0.78	0.0	0.44	0.0
	p-value	0.376	0.376	1.0	0.507	1.0

S: p-value < 0.05 is considered significant. X^2: Chi-square test.
NS: p-value > 0.05 is considered non-significant.

Table 5: Comparisons between studied groups as regard post-treatment manifestations

Post-treatment	Groups										X^2	P-value
	Group I (n = 20)		Group II (n = 20)		Group III (n = 20)		Group IV (n = 20)		Group V (n = 20)			
Rhinorrhea	2	10%	4	20%	8	40%	6	30%	4	20%	5.7	0.223 NS
Mouth breathing	8	40%	10	50%	8	40%	10	50%	14	70%	4.8	0.308 NS
Snoring	4	20%	0	0%	2	10%	4	20%	10	50%	17.5	0.002 S
Halitosis	8	40%	8	40%	4	20%	4	20%	6	30%	3.8	0.432 NS
Nocturnal enuresis	2	10%	2	10%	2	10%	6	30%	8	40%	10	0.04 S

NS: p-value > 0.05 is considered non-significant. X^2: Chi-square test.
S: p-value < 0.05 is considered significant.

Table 6: Multiple comparisons between studied groups as regard post-treatment manifestations

Post-treatment		Rhinorrhea	Mouth breathing	Snoring	Halitosis	Nocturnal enuresis
I vs II	X^2	0.78	0.4	4.4	0.0	0.0
	p-value	0.376	0.525	0.035	1.0	1.0
I vs III	X^2	4.8	0.0	0.78	1.9	0.0
	p-value	0.028	1.0	0.376	0.168	1.0
I vs IV	X^2	2.5	0.4	0.0	1.9	2.5
	p-value	0.114	0.525	1.0	0.168	0.114
I vs V	X^2	0.78	3.6	3.9	0.44	4.8
	p-value	0.376	0.057	0.047	0.507	0.028
II vs III	X^2	1.9	0.4	2.1	1.9	0.0
	p-value	0.168 NS	0.525	0.147	0.168	1.0
II vs IV	X^2	0.53	0.0	4.4	1.9	2.5
	p-value	0.465	1.0	0.035	0.168	0.114
II vs V	X^2	0.0	1.66	13.3	0.44	4.8
	p-value	1.0	0.197	< 0.001	0.507	0.028
III vs IV	X^2	0.44	0.4	0.78	0.0	2.5
	p-value	0.507	0.525	0.376	1.0	0.114
III vs V	X^2	1.9	3.6	7.6	0.53	4.8
	p-value	0.168	0.057	0.006	0.465	0.028
IV vs V	X^2	0.53	1.66	3.9	0.53	0.44
	p-value	0.465	0.197	0.047	0.465	0.597

S: p-value < 0.05 is considered significant. X^2: Chi-square test.
HS: p-value < 0.001 is considered highly significant.
NS: p-value > 0.05 is considered non-significant.

Table 7: Comparisons between pre and post-treatment manifestations in group I

Group I	Pre (n = 20)		Post (n = 20)		X^2	P-value
Rhinorrhea	12	60%	2	10%	10.4	0.001 S
Mouth breathing	20	100%	8	40%	17.1	< 0.001 HS
Snoring	12	60%	4	20%	6.7	0.01 S
Halitosis	12	60%	8	40%	1.6	0.206 NS
Nocturnal enuresis	6	30%	2	10%	2.5	0.114 NS

S: p-value < 0.05 is considered significant. X^2: Chi-square test.
HS: p-value < 0.001 is considered highly significant.
NS: p-value > 0.05 is considered non-significant.

Table 8: Comparisons between pre and post-treatment manifestations in group II

Group II	Pre (n = 20)		Post (n = 20)		X^2	P-value
Rhinorrhea	12	60%	4	20%	6.7	0.01 S
Mouth breathing	20	100%	10	50%	13.3	< 0.001 HS
Snoring	12	60%	0	0%	17.1	< 0.001 HS
Halitosis	14	70%	8	40%	3.8	0.057 NS
Nocturnal enuresis	10	50%	2	10%	7.6	0.006 S

S: p-value < 0.05 is considered significant. X^2: Chi-square test.
HS: p-value < 0.001 is considered highly significant.
NS: p-value > 0.05 is considered non-significant.

Table 9: Comparisons between pre and post-treatment manifestations in group III

Group III	Pre (n = 20)		Post (n = 20)		X^2	P-value
Rhinorrhea	16	80%	8	40%	6.7	0.01 S
Mouth breathing	18	90%	8	40%	10.9	0.001 S
Snoring	10	50%	2	10%	7.6	0.006 S
Halitosis	6	30%	4	20%	0.53	0.465 NS
Nocturnal enuresis	4	20%	2	10%	0.78	0.376 NS

S: p-value < 0.05 is considered significant. X^2: Chi-square test.
NS: p-value > 0.05 is considered non-significant.

Table 10: Comparisons between pre and post-treatment manifestations in group IV

Group IV	Pre (n = 20)		Post (n = 20)		X^2	P-value
Rhinorrhea	16	80%	6	30%	10.1	0.001 S
Mouth breathing	16	80%	10	50%	3.9	0.047 S
Snoring	10	50%	4	20%	3.9	0.047 S
Halitosis	8	40%	4	20%	1.9	0.168 NS
Nocturnal enuresis	8	40%	6	30%	0.44	0.507 NS

S: p-value < 0.05 is considered significant. X^2: Chi-square test.
NS: p-value > 0.05 is considered non-significant.

Table 11: Comparisons between pre and post-treatment manifestations in group V

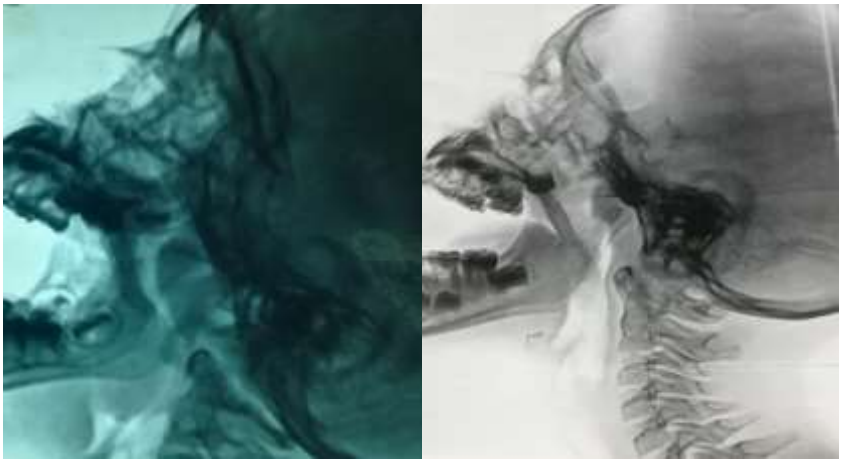
Group V	Pre (n = 20)		Post (n = 20)		X^2	P-value
Rhinorrhea	18	90%	4	20%	19.8	< 0.001 HS
Mouth breathing	18	90%	14	70%	2.5	0.114 NS
Snoring	10	50%	10	50%	0.0	1.0 NS
Halitosis	6	30%	6	30%	0.0	1.0 NS
Nocturnal enuresis	8	40%	8	40%	0.0	1.0 NS

S: p-value < 0.05 is considered significant. X^2: Chi-square test.
HS: p-value < 0.001 is considered highly significant.
NS: p-value > 0.05 is considered non-significant.

Table 12: Comparisons between pre and post-treatment X-ray grading

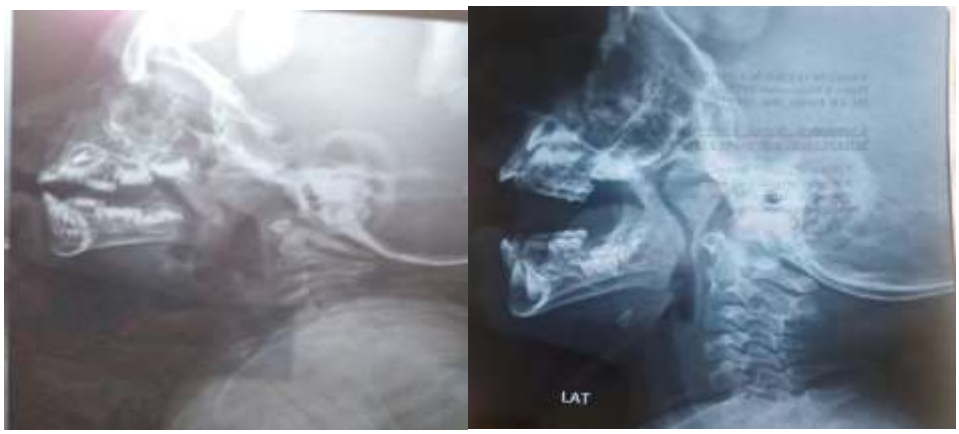
	X-ray grading	Pre (n = 20)		Post (n = 20)		X^2	P-value
Group I	Grade II	0	0%	4	20%	8.8	0.012 S
	Grade III	8	40%	12	60%		
	Grade IV	12	60%	4	20%		
Group II	Grade II	0	0%	6	30%	8.5	0.014 S
	Grade III	10	50%	10	50%		
	Grade IV	10	50%	4	20%		
Group III	Grade II	0	0%	4	20%	5.0	0.082 NS
	Grade III	10	50%	10	50%		
	Grade IV	10	50%	6	30%		
Group IV	Grade II	0	0%	2	10%	2.1	0.344 NS
	Grade III	4	20%	4	20%		
	Grade IV	16	80%	14	70%		
Group V	Grade II	0	0%	0	0%	0.0	1.0 NS
	Grade III	8	40%	8	40%		
	Grade IV	12	60%	12	60%		

S: p-value < 0.05 is considered significant. X^2: Chi-square test.
NS: p-value > 0.05 is considered non-significant.



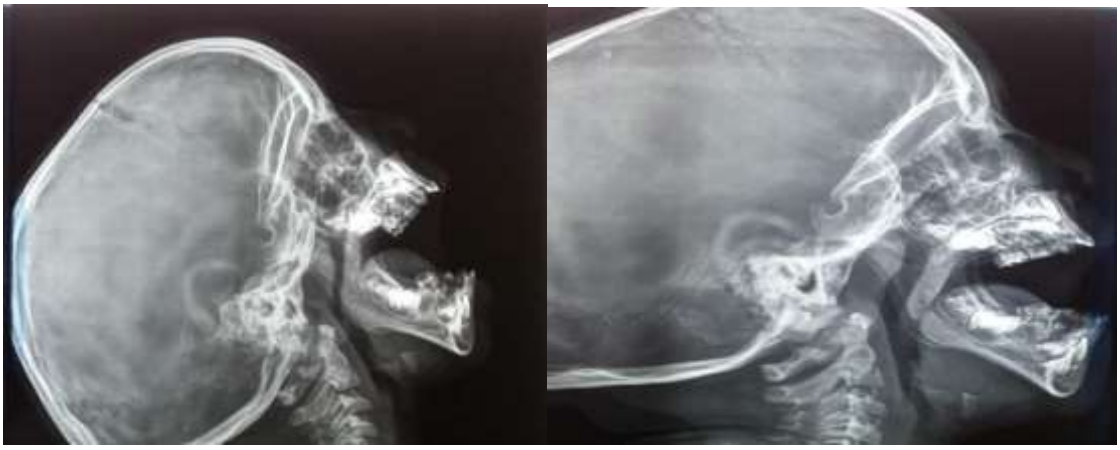
A **B**

Figure 7: Plain x-ray showing marked decrease of adenoid size pre(32 A) and post treatment(32 B) in group I.



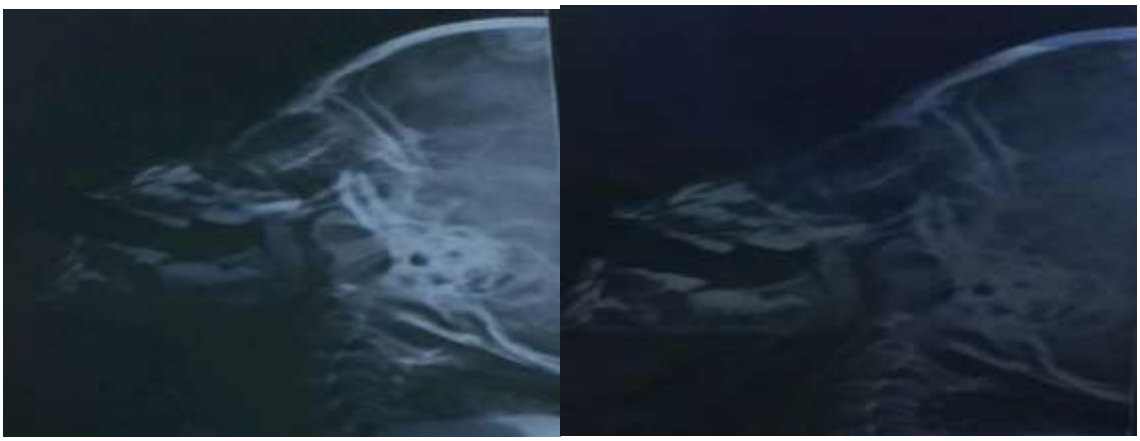
A: Pre treatment **B: Post treatment**

Figure 8: Plain x-ray nasopharynx showing moderate decrease of adenoid size pre (A) and post treatment(B) in group II.



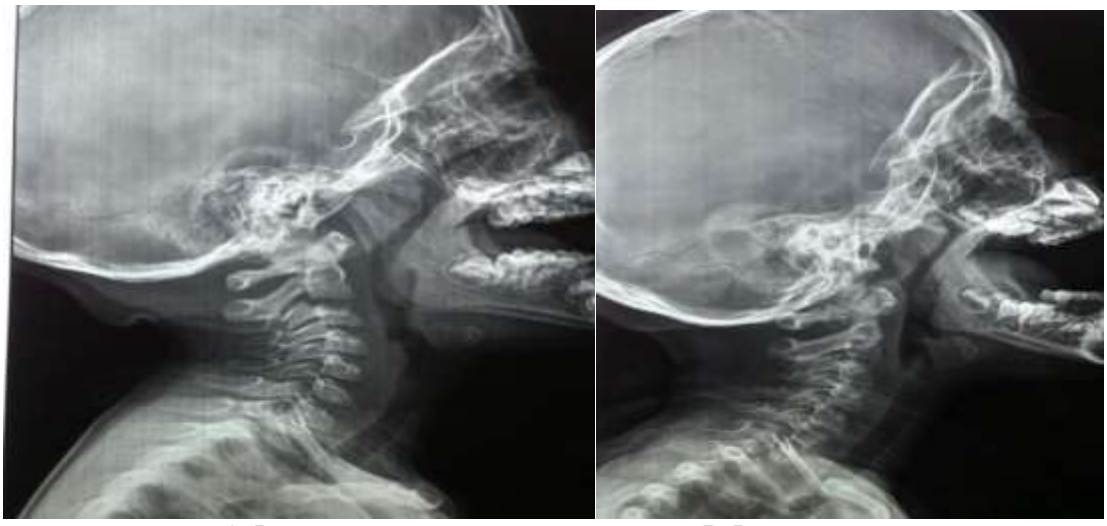
A: Pre treatment **B: Post treatment**

Figure 9: Plain x-ray nasopharynx showing mild decrease of adenoid size pre and post treatment in group III.



A: Pre treatment **B: Post treatment**

Figure 10*:* Plain x-ray nasopharynx showing no decrease of adenoid size pretreatment and post treatment in group IV.



A: Pre treatment **B: Post treatment**

Figure 11*:* Plain x-ray nasopharynx showing no decrease of adenoid size between pretreatment and post treatment in group V.

4. Discussion

Adenoids hypertrophy is a common problem in childhood, the incidence of adenoid hypertrophy is 2%–3% in children [5] .

Obstructive adenoids may cause nasal obstruction, mouth breathing, snoring, rhinorrhea, postnasal drip, cough, dry mouth, halitosis, swallowing difficulty, hyponasal voice, restlessness sleep, enuresis, and morning headache. In severe cases, they may induce obstructive sleep apnea, otitis media with effusion, and craniofacial growth abnormality [4].

X-ray has been picked like the basis standard found since it connect strongly with the size of the adenoid which removed among surgery. Moreover, radiographs are seems to be objective & noninvasive method for evaluating the degree of obstruction in the airway caused by adenoid [9].

The gold-standard for diagnosis of adenoid-related nasal obstruction is considered to be fiber- optic nasoendoscopy, which allows direct visualization of the nasal cavity with dynamic evaluation of any nasal airway obstruction [10].

Adenoidectomy is the definitive worldwide used method for treatment of adenoid hypertrophy, adenoid tissue may grow after infections or chronic allergic reactions and this surgery has some complications like; hemorrhage, infections and palate dysfunction besides the risks of general anesthesia [(11)]. Early or late bleeding (4%–5%), adenoid tissue recurrence (10%–20%), and postoperative respiratory problems (27%) [5].

In addition, anesthesia risks are also among the factors that should be taken into account [12].

Adenoid hypertrophy in children is a global health problem because of its negative impact on quality of life. In recent years; the medical treatment of obstructive adenoids has developed by using anti-inflammatory and anti-allergy medications. Many studies have demonstrated the usefulness of steroid sprays in patients with adenoidal hypertrophy; thus, adenoidectomy can be avoided [4].

The studied groups were matched in age and sex.. Patients assigned to groups randomly with no significant differences at the initial assessment as regards main symptoms and adenoid hypertrophy grades.

In our study, there is no statistically significant difference (p-value = 0.202) between studied groups as regards age. It was 5.9 ± 1.3 years in group I, 6.9 ± 2.02 years in group II, 6.3 ± 2.2 years in group III, 5.5 ± 2.3 years in group IV and 5.7 ± 1.8 years in group V.this matches with Tuhanıoğlu and Erkan (11), who included 30 patients in each studied groups, with age ranged from 4 to 10 years, the mean age in the group treated with momentasone was 6.83 ± 2.05 years, and in the group treated with combined therapy, it was 7.37 ± 1.85 years.

In our study in group, I 20 patients received combined therapy using mometasone furate intranasal spray (2puff in each nostril 100 µg once daily) combined with oral montelukast either (4 mg in patients <6 years) or (5mg in patients >6 years) once daily for 12 weeks and anti histaminics.
•Statistically significant difference (p-value = 0.012) between pre-treatment and post-treatment X-ray grading in group I.

Statistically significant (p-value = 0.001) decreased percentage of post-treatment rhinorrhea (2 patients 10%) when compared with pre-treatment rhinorrhea (12 patients, 60%) in group I.

Highly statistically significant (p-value < 0.001) decreased percentage of post-treatment mouth breathing (8 patients 40%) when compared with pre-treatment mouth breathing (20 patients, 100%) in group I.

Statistically significant (p-value = 0.01) decreased percentage of post-treatment snoring (4 patients 20%) when compared with pre-treatment snoring (12 patients, 60%) in group I. In literature there is no papers that studied combination of

montelukast, nasal corticosteroids, and anti-histaminic as treatment of adenoid hypertrophy. But Tuhanıoğlu and Erkan [11] studied combination of montelukast and mometasone furate intranasal spray. And Modrzyński et al. [13] studied use of antihistminics and topical nasal steroids.

Our results partially matches with results of Tuhanıoğlu and Erkan [11], they found that both montelukast and mometasone furoate therapies were similarly successful in the treatment of adenoid hypertrophy and the combination therapy is effective at reducing adenoid size. And matches also with results of Modrzyński et al. [13] examined changes in clinical symptoms score, and used acoustic rhinometry and endoscopy to evaluate the influence of three months anti-allergic treatment (topical nasal steroid and antihistaminic) on the adenoid size in children with adenoid hypertrophy. Results In children from the study group we observed the most significant decrease of clinical symptoms and endoscopic adenoid size and increase of nasopharyngeal cavity in acoustics rhinometry after the treatment.so, properly administered nasal glucocorticoid spray together with antihistaminic in standard doses can significantly reduce adenoidal hypertrophy and considerably eliminate airway obstructive symptoms in children with allergic rhinitis.

In group II 20 patients received combined therapy using mometasone furate intranasal spray (2puff in each nostril 100 µg once daily) combined with oral montelukast either (4 mg in patients <6 years) or (5mg in patients >6 years) once daily for 12 weeks.

Statistically significant difference (p-value = 0.014) between pre-treatment and post-treatment X-ray grading in group II.

Highly statistical significant (p-value < 0.001) decreased percentage of post-treatment mouth breathing (10 patients 50%) when compared with pre-treatment mouth breathing (20 patients, 100%) in group II.

Highly statistical significant (p-value < 0.001) decreased percentage of post-treatment snoring (0 patients 0%) when compared with pre-treatment snoring (12 patients, 60%) in group II.

Tuhanıoğlu and Erkan [11] made a randomized prospective clinical trial to evaluate the effects of montelukast, and mometasone furoate as combined therapy on adenoid size. Our results matche with results of Tuhanıoğlu and Erkan [11], they found that both montelukast and mometasone furoate therapies were similarly successful in the treatment of adenoid hypertrophy and the combination therapy is effective at reducing adenoid size. Our results agree with Yang et al. [14] in which The study was designed to investigate the clinical effect of montelukast sodium combined with inhaled corticosteroids in the treatment of children with obstructive sleep apnea syndrome (OSAS).

In group III 20 patients received oral montelukast either (4 mg in patients <6 years) or (5mg in patients >6 years) once daily for 12 weeks.

No statistical significant difference (p-value = 0.082) between pre-treatment and post-treatment grading in group III.

Statistically significant (p-value = 0.01) decreased percentage of post-treatment rhinorrhea (8 patients 40%) when compared with pre-treatment rhinorrhea (16 patients, 80%) in group III.

Statistically significant (p-value = 0.001) decreased percentage of post-treatment mouth breathing (8 patients 40%) when compared with pre-treatment mouth breathing (18 patients, 90%) in group III.

Statistically significant (p-value = 0.006) decreased percentage of post-treatment snoring (2 patients 10%) when compared with pre-treatment snoring (10 patients, 50%) in group III.

Shokouhi et al. [5] studied the effect of montelukast on size and symptoms of adenoid hypertrophy and said that montelukast as an anti-inflammatory agent gave promising results in reduction of

adenoid size and improving the related clinical symptoms.

Our results matched with Shokouhi et al. [5] in which Children were randomly assigned to the study or control groups (n=30). The study group received montelukast), 5 and 10 mg per day for children <6 and >6 years of age, respectively, whereas placebo tablets with the same shape, color and dosage were prescribed for the control group. They found no difference was observed between the two groups (P=0.111) as regards to Snoring before treatment. However, following treatment a significant difference was revealed between the two groups (P<0.007). Regarding sleep discomfort, no meaningful difference between the two groups was observed at study initiation (P=0.408). However, following treatment the difference was statistically significant (P<0.0001). Results were similar for mouth breathing; showing a strongly meaningful difference only after the therapeutic period (P=0.33 vs. P<0.0001).

About change of size of adenoid it doesn't match with Shokouhi et al. [5] as in our study No statistical significant difference (p-value = 0.082) between pre-treatment and post-treatment grading in group III, but in our study, our results matched with Karaer and Cimen [15]. There was no statistically significant difference identified between the adenoid sizes before and after treatment (p=0.304) in that study.

Group IV 20 patients received mometasone furate intranasal spray (2puff in each nostril 100 µg once daily) either for 12 weeks.

No statistical significant difference (p-value = 0.344) between pre-treatment and post-treatment grading in group IV.

Statistically significant (p-value = 0.047) decreased percentage of post-treatment mouth breathing (10 patients 50%) when compared with pre-treatment mouth breathing (16 patients, 80%) in group IV.

Statistically significant (p-value = 0.047) decreased percentage of post-treatment snoring (4 patients 20%) when compared with pre-treatment snoring (10 patients, 50%) in group IV.

In literature there are many papers studied the effect of nasal steroid, particularly intranasal mometasone for treatment of adenoid hypertrophy. Some authors [4,16,17] found some beneficial effect of mometasone nasal spray on some outcomes of nasal obstruction caused by adenoidal hypertrophy.

Our results matched with Bhargava and Chakravarti [4] with mometasone treatment in change in snoring and mouth breathing, there was an 89.8 per cent reduction in clinical symptom score, but about change of size of adenoid doesn't match with it as in our study No statistical significant difference (p-value = 0.344) between pre-treatment and post-treatment grading, in Bhargava and Chakravarti [4] the degree of obstruction dropped from 87 to 72 per cent (p < 0.0001). A statistically significant change in quality of life scores was seen in patients treated with the mometasone nasal spray.

Group V received oral anti histaminic as fexofenadine (30mg twice daily for children aged 3 to 12 years and 120mg once daily for patients over 12years) for 12 weeks.

Highly statistical significant (p-value < 0.001) decreased percentage of post-treatment rhinorrhea (4 patients 20%) when compared with pre-treatment rhinorrhea (18 patients, 90%) in group V.

No statistical significant difference (p-value = 1.0) between pre-treatment and post-treatment grading in group V

No statistical significant difference (p-value = 0.114) between pre-treatment and post-treatment mouth breathing in group V.

No statistical significant difference (p-value = 1.0) between pre-treatment and post-treatment snoring in group V.

This doesn't match with results of Modrzyński et al. [13] examined changes in clinical symptoms score, and used acoustic rhinometry and endoscopy to evaluate the influence of three months anti-allergic treatment (topical nasal steroid and

antihistaminic) on the adenoid size in children with adenoid hypertrophy. Results
In children from the study group we observed the most significant decrease of clinical symptoms and endoscopic adenoid size and increase of nasopharyngeal cavity in acoustics rhinometry after the treatment.so, properly administered nasal glucocorticoid spray together with antihistaminic in standard doses can significantly reduce adenoidal hypertrophy and considerably eliminate airway obstructive symptoms in children with allergic rhinitis. This doesn't match because we used anti histaminics alone for 12 weeks.
Also this doesn't match with Georgalas et al. [18] who used Intranasal corticosteroids in combination with antihistamine and antibiotic have also shown efficacy in reducing AH associated with rhinosinusitis in a small pediatric study.
In literature no papers studied the effect of anti histaminics alone on adenoid hypertrophy.
Anti-allergic drugs have been used from time to time despite the lack of adequate evidence to support their usage, as allergy is just one of the reasons for obstruction [19].
No papers studied association between medical treatment of adenoid and halitosis.
Abdollohi-Fakhim et al. [20] studied association between halitosis and adenoid. There was a statistically significant association between halitosis and adenoid hypertrophy, and a significant improvement in halitosis was obtained following adenoidectomy. The present study provides an association between halitosis and adenoid hypertrophy. If there is no other oral pathology causing halitosis, halitosis can be a sign of adenoid hypertrophy in children.
In our study statistically significant difference (p-value = 0.031) between studied groups as regard pre-treatment halitosis. It was present in 12 patients (60%) of group I, 14 patients (70%) of group II, 6 patients (30%) of group III, 8 patients (40%) of group IV and 6 patients (30%) of group V.
In group I: No statistical significant difference (p-value = 0.206) between pre-treatment and post-treatment halitosis.
In group II: No statistical significant difference (p-value = 0.057) between pre-treatment and post-treatment halitosis.
In group III: No statistical significant difference (p-value = 0.465) between pre-treatment and post-treatment halitosis.
In group IV: No statistical significant difference (p-value = 0.168) between pre-treatment and post-treatment halitosis.
In group V: No statistical significant difference (p-value = 1.0) between pre-treatment and post-treatment halitosis.
After 12 weeks of medical treatment no statistical significant difference (p-value = 0.432) between studied groups as regard post-treatment halitosis. It was present in 8 patients (40%) of group I, 8 patients (40%) of group II, 4 patients (20%) of group III, 4 patients (20%) of group IV and 6 patients (30%) of group V.
About nocturnal enuresis no papers studied association between nocturnal enuresis and medical treatment of adenoid hypertrophy but Abdollohi-Fakhim et al. [20] studied effects of adenotonsillar hypertrophy corrective surgery on nocturnal enuresis of children, In this longitudinal study, 184 children with adenotonsillar hypertrophy as case group and 200 healthy children as control group were randomly compared for nocturnal enuresis incidence and risk factors. Then they were followed after 6 months to estimate the cure rate after corrective operation.
Three months after surgery, 48% of children totally cured from enuresis (P = 0.001) and 71% cured both partially or totally (P = 0.03). The response rate after moderate obstruction relieving was 100% while that in severe cases was 60% (P = 0.2).
Cure rate of primary enuresis due to obstructive airway disease after 6 months of relieving was 48% in children over 5 years old [20].

In our study, No statistical significant difference (p-value = 0.341) between studied groups as regard pre-treatment nocturnal enuresis. It was present in 6 patients (30%) of group I, 10 patients (50%) of group II, 4 patients (20%) of group III, 8 patients (40%) of group IV and 8 patients (40%) of group V.

In group I: No statistical significant difference (p-value = 0.114) between pre-treatment and post-treatment nocturnal enuresis.

In group II: Statistically significant (p-value = 0.006) decreased percentage of post-treatment nocturnal enuresis (2 patients 10%) when compared with pre-treatment nocturnal enuresis (10 patients, 50%).

In group III: No statistical significant difference (p-value = 0.376) between pre-treatment and post-treatment nocturnal enuresis.

In group IV: No statistical significant difference (p-value = 0.507) between pre-treatment and post-treatment nocturnal enuresis.

In group V: No statistical significant difference (p-value = 1.0) between pre-treatment and post-treatment nocturnal enuresis.

In comparison between all groups, statistically significant difference (p-value = 0.04) between studied groups as regard post-treatment nocturnal enuresis. It was present in 2 patients (10%) of group I, 2 patients (10%) of group II, 2 patients (10%) of group III, 6 patients (30%) of group IV and 8 patients (40%) of group V.

5. Conclusion

In this study it found that medical treatment of adenoid especially combination of (antileukotriens as montelukast, anti histaminics, local corticosteroid spray as memotasone furonate) can decrease the symptoms of adenoid hypertrophy as snoring, breathing difficulty and snoring, rhinorrhea it also decrease the size of adenoid which evaluated by radiological assessment.

Also combination of (antileukotriens as montelukast, local corticosteroid spray as memotasone furonate) can be used but better results are for combination of (antileukotriens as montelukast, anti histaminics, local corticosteroid spray as memotasone furonate). Medical treatment for 3 month therapy can be used in adenoid hypertrophy alternative to surgery.

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