INDICATION AND THE EFFICACY OF ADENOIDECTOMY IN INFANCY

Wael Aly Alzamil, M.D. Essam Fathy Mohamed,** M.D. Ear nose and throat deparment, hearing and speech institute general organization for teaching hospitals and institutes, egypt, Sameer Attya,***M.D. Banha teaching hospital general organization for teaching hospitals and institutes, egypt, Said Abdelmonem, M.D. ****faculty of medicine zagazig university.*

ABSTRACT

Objective; this study is carried out to assess the efficacy of adenoidectomy in infants less than 1 year with OSA provided that no craniofacial anomalies or neuromuscular disease. **Background;** Pediatric obstructive sleep apnea is most commonly caused by adenoid hypertrophy. Although adenoidectomy is the only effective treatment for adenoid hypertrophy, it is rarely performed in infants less than 1 year old. **Materials and methods;** Twenty infants less than 1 year old with a triad of upper airway obstruction symptoms, findings of obstructing adenoids, and obstructive sleep apnea undergo adenoidectomy will be included in our study. This is a retrospective study reviewing each infant's clinical data, including presenting symptoms, physical examination findings, and results of the investigations such as polysomnography, endoscopy, and echocardiography. With careful preoperative and postoperative monitoring, the 20 infants underwent adenoidectomy. **Results**: The infants' ages ranged from 8 to 12 months, with a mean of 10 months. There were 13 boys and 7Girls. The length of follow-up ranged from 6 to 16 months, with a mean of 10 months (median, 10.5 months).The most common presenting symptoms were noisy breathing, usually snoring in19 infants (95 %), apnea in 18 infants (90%) After the adenoidectomy all infants showed dramatic improvement with disappearance of symptoms of upper airway obstruction, failure to thrive, and pulmonary hypertension. **Conclusions;** Adenoidectomy was found to be sufficient and curative for such infants.

Key Word: adenoid hypertrophy, adenoidectomy, obstructive sleep apnea, upper airway obstruction. .

Corresponding Author
Name: Essam Fathy Mohamed
E-mail: essament2014@gmail.com

Received: February 2016
Accepted: April 2016

INTRODUCTION

Nasal obstruction in children is a common clinical presentation to the pediatricians and pediatric Otorhinolaryngologists. An enlarged, obstructive adenoid has been implicated in some of the cases [1]. Adenoids are lymphoid tissues located at the postnasal space and their surgical removal is simply referred to as adenoidectomy. An enlarged adenoid is a common cause of obstructive sleep apnea syndrome, which is an absolute indication for adenoidectomy. Obstructive sleep apnea syndrome is clinically diagnosed when there is snoring, excessive daytime somnolence, restlessness and cessation of breath for at least five times, each episode lasting for at least 10 s in an hour of sleep. The situation is usually worse when accompanied by enlarged tonsils. Obstructive sleep apnea is a frequent, albeit underdiagnosed, condition in children and may lead to substantial morbidity if left untreated. In a neglected, long standing situation, this could result in hypoxia, pulmonary hypertension, right ventricular hypertrophy and cor-pulmonale [2].

Other indications for adenoidectomy include failure to thrive, recurrent or persistent middle ear effusion and recurrent or chronic sinusitis [3–5]. The contribution of adenoid hypertrophy to upper airway obstruction (UAO) is even more significant in the presence of other structural factors that compromise the upper airways, e.g. craniofacial anomalies [6]. The OSA incidence rate reported for infants and children is approximately 0.69 % to 2.9 %(9-11). In childhood, OSA is related to adenotonsillar hypertrophy, and in infancy, it is usually related to craniofacial anomalies [6]. The portion of these cases solely attributable to adenoid hypertrophy has not been reported in the literature. It is generally accepted that adenoid hypertrophy occurs mostly between 2 and 5 years of age [7, 8], and is rarely found in infants less than 1 year old. However, an analysis of incidence by age group has not yet been reported in the literature.

Although adenoidectomy is the only effective treatment for adenoid hyper-trophy, it is rarely performed in infants less than 1 year old. this study reports on the successful

use of adenoidectomy in infants less than 1 year old with the triad of UAO symptoms, findings of obstructing adenoids, and OSA (But no other anomalies).

MATERIAL AND METHODS

Ethical consideration: The study protocol was approved by the institutional ethical review committee. Informed consent was obtained from the parents or legal guardians. This is retrospective study , The study group consisted of 20 infants who met 3 criteria:1) UAO symptoms including snoring, respiratory distress, and/or apnea; 2) physical findings consistent with adenoid enlargement causing > 50% narrowing of the nasopharynx ; and 3) polysomnography demonstrating OSA.

Statistical analysis; polysomnography results are expressed as mean±SD. Paired *t* tests was used to compare mean values of polysomnography parameters before and after adenoidectomy.

All infants less than 1 year of age who underwent adenoidectomy at the Hearing and speech institute,and Banha teaching hospitals . These were otherwise healthy infants. In order to meet inclusion criteria, patients should have or present complaints of nasal obstruction or oral breathing, with suspected diagnosis of adenoid hypertrophy. Children with syndromes or head and neck malformations were excluded. Subjects with acute infection of the respiratory tract, with history of previous adenoidectomy or grade 3 - 4 tonsillar hypertrophy, were also dismissed. Each infant's clinical data were recorded. Failure to thrive was defined as weight and height under the third percentile. All infants underwent a standard evaluation consisting of history and physical examination, upper airway evaluation, radiographic evaluation, laboratory tests and polysomnography. These preoperative investigations are detailed below.

HISTORY AND PHYSICAL EVALUATION

Each infant's clinical data were recorded. Snoring and difficulty breathing during sleep are the most common complaints [9]. Parents often describe episodes of retractions with increased respiratory effort; infants appear to be very restless during the night, frequently changing sleep positions and hyperextending the neck, increased diaphoresis and enuresis, and mouth breathing**.**

Physical Examination; Mouth breathing, adenoidal faces, Hyponasal voice and a muffled voice were noted. The lateral facial profile inspected to exclude retrognathia, micrognathia, or midfacial hypoplasia. Failure to thrive was defined as weight and height under the third percentile.

UPPER AIRWAY EVALUATION

0ropharyngeal examination; cleft palate, a bifid uvula may be associated with submucosal cleft palate, a high-arched or elongated palate, or a low dependent palate were excluded. The size of the tonsils should be assessed. A scale from 0 to a maximum of +4 when the tonsils meet the midline is commonly used [10]. Some simply describe the tonsils' appearance as minimally visible (grade 1), visible to the pillars (grade2), visible beyond the pillars (grade3), and visible to the uvula (grade4).

Endoscopic examinations; were performed preoperatively with the patient in the supine position and 36-mm flexible fiberoptic endoscope introduced through the nasal cavity. The nose is assessed for septal deviation, mucosal thickening, polyps, and patency of either vestibule with the opposite naris occluded. grading according to endoscopic evaluation, According to this method [16], adenoid hypertrophy is classified according to its anatomical relationship with adjacent structures such as vomer, soft palate and torus tubaris: 1) grade 1, none of the above-cited structures contact with the adenoid tissue; 2) grade 2, the adenoid tissue contacts with the torus tubaris; 3) grade 3, the adenoid tissue contacts with torus tubaris and vomer; 4) grade 4, the adenoid tissue contacts with torus tubaris, vomer and soft palate in resting position.

Radiographic Evaluation

To evaluate the upper airway in children with OSAS, several radiologic techniques are available, including lateral neck radiographs, computerized tomography, and magnetic resonance imaging (MRI) The utility of these techniques varies, and as diagnostic tools they should be tailored to the child's condition. A

lateral neck radiograph is simple and may be obtained in clinic to evaluate the size of the adenoid and upper airway patency. Other radiographic techniques should be reserved for more complicated conditions, such as those involving alterations in craniofacial growth and neurologic conditions affecting upper airway collapsibility. The severity of adenoid hypertrophy was graded as follows: grade 1 was 100 % (complete) obstruction; grade 2 was 70 % to 99 % narrowing of the nasopharyngeal airways; and grade 3 was 50 % to 69 % narrowing.

Echocardiography: We performed echocardiography in 5 cases of severe OSA (AHI of > 10).

Polysomnography

Full night polysomnography is the only diagnostic method able to quantify sleep related breathing disorders and is considered the gold standard for the diagnosing of OSAS. It can be easily performed at any age, provided appropriate equipment and trained health care providers are available. The following parameters are recorded during polysomnography: respiratory movements, oronasal airflow, hemoglobin oxygen saturation, end-expiratory CO2, and electrocardiogram. Combined analysis of these parameters can lead to an accurate diagnosis.

All infants were treated using conventional adenoidectomy technique. All operations were performed under general anesthesia. The patients were positioned supine in Rose position and the neck was extended with a shoulder-roll placed under the shoulders. The adenoidectomies were performed using adenoidectomy curettes. A Boyle-Davis gag was inserted. The palate was palpated to exclude a possible submucosal cleft. Complete removal was confirmed by digital palpation or mirror examination. After adenoi-dectomy, pressure was applied to nasopharynx by tonsillar pack for three minutes. The use of cautery was rarely necessary for hemostasis. The patients were discharged the same day after the operation. They routinely received 7 days of postoperative ampicillin or amoxicillin. Three months after the operation the children were re-examined and their parents were interviewed about persistent symptoms.

Definitions in pediatrics; The term obstructive sleep related breathing disorders includes a variety of pathologic conditions ranging from primary snoring and upper airway resistance syndrome to obstructive sleep apnea- hypopnea syndrome ***Obstructive apnea*** ;was defined as a cessation of airflow through the nose and mouse for at least two respiratory cycles with paradoxical chest and abdominal movements. ***Hypopnea***; was defined as a reduction in airflow through the nose and mouse with paradoxical respiratory effort, resulting in either an arousal or an oxyhemoglobin desaturation of at least 4%. ***central apnea***; was defined as absence of airflow and respiratory effort for greater than or equal to 20 seconds or greater than or equal to 10 seconds but associated with a bradycardia or 4% oxygen desaturation. The following respiratory parameters were also measured: (table III) ***respiratory disturbance index***; defined as the average number of central and obstructive apneas and hypopneas per hour of sleep; ***obstructive apnea index***; defined as the number of obstructive apneas per hour of sleep. ***Obstructive hypopneas index***; defined as the number of obstructive hypopneas per hour of sleep. ***AHI***; defined as the number of obstructive apneas and hypopneas per hour of sleep. ***Central apnea index***; defined as the number of central apneas per hour of sleep. According to diagnostic guidelines proposed by the American Thoracic Society We defined OSA as an apnea/hypopnea index (AHI) of > 1/h [11].

RESULTS

The infants' ages ranged from 8 to 12 months, with a mean of 10 months. There were 13 boys and 7Girls. The length of follow-up ranged from 6 to 16 months, with a mean of 10 months (median, 10.5 months).The most common presenting symptoms were noisy breathing, usually snoring in19 infants (95 %), apnea in 18 infants (90%), and nocturnal cough (30%) table 1. On physical examination, the four most frequent findings were oral breathing, rhinitis, chest and suprasternal retractions, and failure to thrive (90%, 80%, 75 %, and 70%, respectively,)

table 2. Lateral neck radiographs revealed 2 cases of grade 1 adenoid hypertrophy, 13 cases grade 2, and 5 cases grade 3. The mean interval between pre- and postoperative polysomnograhy was 7.5(range, 2.2-14) months. Respiratory and non-respiratory parameters from polysomnography in infants before and after adenoidectomy for OSA are summarized in Table III. Changes were highly significant (*P*<.oo1) in the AHI, respiratory disturbance index, obstructive apnea index, obstructive hypopneas index, minimum oxygen saturation. For all infants, the preoperative AHI was higher than the postoperative value. The mean preoperative value of the AHI for the study population was 27.5, whereas the mean postoperative value was 3.5. Changes in the mean oxygen saturation and in the central apnea index were also significant (Table III). The preoperative echocardiography findings were normal for 18 infants, 2 infants showed pulmonary hypertension which improved post adenoidectomy. There were no intraoperative, immediate, or late postoperative complications, including bleeding or late nasopharyngeal stenosis. After adenoidectomy, all 20 infants showed marked clinical improvement. Chest retractions, as well as thoracoabdominal asynchrony, completely disappeared. The preoperative data indicated that 70% of the 20 infants demonstrated FTT. One month after the operation, this rate dropped to 35 %. Treatment of OSA by adenoidectomy lead to resolution of the somatic growth disturbance, and weight gain was observed in all infants. Echocardiography show improved pulmonary hypertension in the 2 infants with pulmonary hypertension after 2 weeks and discharged from ICU of Banha pediatric hospital.

Table 1; symptomatic presentation**.**

Symptoms	No.of patients	%
Apnea	18	90
Snoring	19	95
Dyspnea	4	20
Nocturnal cough	6	30
Cyanosis	2	10
Running nose	6	30

Table 2; data of physical finding.

Signs	No. of patients	%
Oral breathing	18	90
Rhinitis	16	80
FTT	14	70
Retraction	15	75
Pharyngomalacia	2	10
High/narrow palate	4	20
Laryngomalacia	2	10

Table 3: polysomnographic parameters before and after adenoidectomy for pediatric obstructive sleep apnea (n=20).

Parameter	Preoperative	postoperative	*P* value
Obstructive apnea/hypopnea index	27.5± 22.5	3.5± 4.9	<.001
Respiratory disturbance index	29.9± 28.2	4.6± 5.5	<.001
Obstructive apnea index	9.0 ± 14.8	0.4± 1.1	<.001
Obstructive hypopnea index	18.5± 16.9	3.1± 3.8	<.001
Central apnea index	2.4± 2.4	1.1± 1.2	.01
Mean oxygen saturation(Sao 2)	95.1± 3.0	96.1± 2.7	.02
minimum oxygen saturation(Sao 2)	82.0± 7.0	91.4± 3.7	<.001

DISCUSSION

Upper airway obstruction and obstructive sleep apnea (even without complete apnea) from adenotonsillar hypertrophy appear to be rising, either reflecting an increasing number of cases or more accurate, and thus more frequent, diagnosis. This is especially true for infants.

Anat Shatz, stated that he did not expect to find adenoid hypertrophy as a cause for UAO in infants less than 1 year of age. His view has now changed. Even moderate adenoid hypertrophy in infants can cause OSA. He therefore makes more extensive use of polysomnography and endoscopy in diagnosing UAO in such infants [12].

Adenoidectomy does not need to be accompanied by tonsillectomy when adenoid

hypertrophy is the only abnormal finding (no tonsillar enlargement), especially in infants less than 1 year of age who exhibit significant narrowing of the nasopharynx and OSA. In these circumstances, adenoidectomy alone was found to be curative treatment following which OSA either disappeared or improved significantly. Tonsillectomy is indicated only in the rare cases of infants with obstructing or "kissing" tonsils, for infants with OSA, we recommend individual consideration of each case, rather than automatic referral for tonsillectomy and adenoidectomy as advocated by others. [13-16].

We found correlation between the severity grading of adenoid hypertrophy (radiologically) and the severity of UAO and OSA, the 2 cases of grade 1(100% obstruction) develope pulmonary hypertension. Severe symptoms found in grade 2 more than grade 3.

Polysomnography can define the severity of OSA. It is the gold standard in the diagnosis of OSA in infants, given that the decision to perform or forgo a surgical procedure involves high levels of risk in the first 12 months of life. Polysomnography indicates the significance of the clinical problem and serves as a tool to follow up and measure the effectiveness of treatment.

Endoscopic evaluations are the key tools in investigating the causes of OSA. Endoscopy provides an accurate diagnosis of nasopharyngeal obstruction, because direct visualization can establish the nature and locations of the obstruction and determine whether there are multiple synchronous obstructions that require treatment [17].

One of the common complications of childhood OSA is the failure to thrive (FTT). Therefore, in infants and children with failure to thrive, pediatricians should be aware of OSA to be differentiated from other causes of FTT [18, 19]. The high incidence of FTT suggests a more pronounced effect of UAO on infants in comparison to older children. According to Marcus et al., FTT could be a by-product of the higher caloric expenditure caused by the increased effort required to breathe during sleep. This effect may explain the rapid catch-up in growth, and subsequent normal growth curves [20]. Our study complements prior reports that infants with enlarged adenoids and OSA, who are younger than 18 months, including infants with dysmorphism, demonstrate a significant increase in weight gain following adenotonsillectomy [18].

Children awaiting adenotonsillectomy for obstruction of the UA during sleep already exhibited altered cardiac morphology and function. Unfortunately we are unable to classify the severity of UA obstruction in these children in order to correlate the degree of echocardiographic findings with the number of obstructive respiratory events or of oxyhemoglobin desaturation episodes. Silke Anna et al, Saied that neither polysomnography nor cardiorespiratory monitoring are carried out as routine for adenotonsillectomy indicated due to UA obstruction in order to quantify sleeping obstructive respiratory events and confirm the diagnosis of OSA[21].

In infants with severe OSA, preoperative assessment by echocardiography is indicated to reveal the presence of pulmonary hypertension and right ventricular dysfunction, which contribute to the risk of arrhythmias during anesthesia. In our study the 2 cases with pulmonary hypertension improved within 2 weeks postoperatively.

Preoperative PSG also helps to identify which children are at greatest risk for persistent OSA following AT. Among 26 children reported by Suen and colleagues and 110 children reported by Tauman and colleagues, high preoperative AHI was associated with increased risk for residual SDB following surgery [22, 23]. Use of preoperative PSG to identify these high-risk patients is clinically important in several ways. Firstly, knowledge that a child may be at increased risk for persistent SDB after adenotonsillectomy should impact how the treating physicians communicate likelihood of surgical success or failure to the child's parents in advance of the procedure. Secondly, this knowledge also identifies the children who have the greatest need for follow-up clinical and PSG evaluation after their surgery.

Earlier studies concluded that AT resulted in polysomnographic resolution of OSA in

75% to 100% of otherwise healthy children. However, use of widely variable methodologies and definitions of OSA represented a significant limitation to these data [22-26]. More recently, several large pediatric case series have reported substantially lower success rates for AT. In a prospective study of 199 children with OSA reassessed following AT, 92 (46%) continued to demonstrate an elevated AHI upon follow-up PSG performed 3 to 5 months after surgery [27].

In a cohort of 110 children assessed before and after AT, complete normalization of the PSG was observed in only 25% of subjects. [23]. It is thought that there is a higher risk of surgical complications in children with very severe OSA or in children less than 1 year old. This was found to be true of infants with craniofacial disorders and severe refractory OSA. However, this was not found to be true in this study of infants less than year of age that had OSA but were otherwise healthy. After surgery, however, there is always a high risk of UAO in an obtunded patient for whom the anesthetic effects still compromise upper airway tone and reflexes. The development of idiopathic pulmonary edema following the relief of UAO also has been noted. These risks of postoperative complications seem to justify overnight cardiorespiratory monitoring in an intensive care unit after adenoidectomy in infants.

CONCLUSIONS

Obstructive sleep apnea appears more frequently than previous studies suggest, and adenoid hypertrophy seems to be more prevalent than is commonly reported in the literature. Obstructive sleep apnea due to adenoid hypertrophy with obstruction does occur in infants. Their young age should not prevent adenoidectomy whenever the procedure is indicated. Adenoidectomy (without tonsillectomy) may be sufficient and curative for these infants. Limitation is a lack of control group and small number of the cases.

REFERENCES

1) Greenfeld M, Tauman R, DeRowe A, Sivan Y (2003) Obstructive sleep apnea syndrome due to adenotonsillar hypertrophy in infants. Int J Pediatr Otorhinolaryngol 67:1055–1060
2) Subashini P, Ravikumar A, Ranjit MS, Sairam VK, Vatsanath RP, Jayasree S (2006) Adenoid hypertrophy presenting with systemic hypertension. Indian J Otolaryngol Head Neck Surg59:73–75
3) van den Aardweg MT, Rovers MM, Kraal A, Schilder AG (2010) Current indications for adenoidectomy in a sample of children in the Netherlands. B-ENT 6:15–18
4) van den Aardweg MT, Schilder AG, Herkert E, Boonacker CW,Rovers MM (2010) Adenoidectomy for otitis media in children.Cochrane Database Syst Rev. doi:10.1002/14651858.CD007810
5) American Academy of Family Physicians, American Academy of Otolaryngology-Head and Neck Surgery, American Academy of Pediatrics Subcommittee on Otitis Media With Effusion (2004) Otitis media with effusion. Pediatrics 113:1412–1429
6) Ward SL, Marcus CL. Obstructive sleep apnea in infants and young children. J. Clin Neurophysiol 1996,13 : 198 – 207.
7) Hui S, Wing YW. Obstructive sleep apnea syndrome in children. Hong Kong Med J 1997; 3:419-26.
8) Gaultier C. Sleep-related breathing disorders. 6 Obstructive sleep apnea syndrome in infants and children: established facts and unsettled issues. Thorax 1995; 50:1204-10.
9) Carroll JL, McColley SA, Marcus CL, Curtis S, Loughlin GM. Inability of clinical history to distinguish primary snoring from obstructive sleep apnea syndrome in children.Chest 1995;108:610–618.
10) Brodsky L. Modern assessment of tonsils and adenoids. Pediatr Clin North Am 1989;36:1551–1569.
11) Brouillette RT, Fernbach SK, Hunt CE. Obstructive sleep apnea in infants and children.J Pediatr 1982;100:31–40
12) Anat Shatz. Indication and outcome of adenoidectomy in infancy.Ann Oto Rhino Laryngol 2004;113:835-838.
13) Darrow DH, Siemens C. Indications for tonsillectomy and adenoidectomy Laryngoscope 2002; 112(suippl 100):6-10.
14) Deutsch ES. Tonsillectomy and adenoidectomy. Changing indications, Pediatr Clin North Am 1996; 43:1319-38.
15) Leiberman A, Tal A, Brama I, Sofer S. Obstructive sleep apnea in young infants. Int J Pediatr Otorhinolaryngol 1988:16:39-44.
16) Potsic WP, Shan UK, Nonsurgical and Surgical management of infants and children

with obstructive sleep apnea syndrome. Otolaryngol Clin North Am 1998:31:969-77

17) Croft CB, Thomson HG, Samuels MP, Southall DP. Endoscopic evaluation and treatment of sleep-associated upper airway obstruction in infants and young children. Clin Otolaryngol 1990;15:209-16
18) Freezer NJ, Bucens IK, Robertson CF. Obstructive sleep apnoea presenting as failure to thrive in infancy. J Paediatr Child Health 1995:31:172-5.
19) Schiffmann R, Faber, J. Eidelman Al. Obstructive hypertrophic adenoids and tonsils as a cause of infantile failure to thrive; reversed by tonsillectomy and adenoide-ctomyt Int J Pediatr Otorhinolarytngol 1985;0:183-7
20) Marcus CL, Carroll JL. Koemer CB, Hamer A, Lutz J, Loughlin GM. Determinants of growth in children with the obstructive sleep apnea syndrome . J Pediatr 1994:125:556-62.
21) Silke Anna Theresa Weber, Jair Cortez Montovani, Beatriz Matsubara,and José Roberto Fioretto. Echocardiographic abnormalities in children with obstructive breathing disorders during sleep, Obstructive sleep apnea, child, echocardiography. J Pediatr (Rio J). 2007;83(6):518-522
22) Suen JS, Arnold JE, Brooks LJ, Adenotonsillectomy for treatment of obstructive sleep apnea in children. Arch Otolaryngol Head Neck Surg 1995;121:525-30.
23) Tauman R, Gulliver TE, Krishna J, et al. Persistence of obstructive sleep apnea syndrome in children after adenotonsillectomy. J Pedi-atr 2006; 149:803-8.
24) Brietzke SE, Gallagher D, The effectiveness of tonsillectomy and adenoidectomy in the treatment of pediatric obstructive sleep apnea/hypopnea syndrome: a meta-analysis. Otolaryngol Head Neck Surg 2006;134:979-84
25) Section on Pediatric Pulmonology, Subcommittee on Obstructive Sleep Apnea Syndrome, American Academy of Pediatrics. Clinical practice guideline: diagnosis and management of child-hood obstructive sleep apnea syndrome. [See comment]. Pediatrics 2002; 109:704-12.
26) Nieminen P, Tolonen U, Lopponen H. Snoring and obstructive sleep apnea in children: a 6-month follow-up study. Arch Otolaryngol Head Neck Surg 2000; 126:481-6.
27) Guilleminault C, Huang YS, Glamann C, Chan A. Adenotonsillectomy and obstructive sleep apnea in children: a prospective survey. Otolaryngol Head Neck Surg 2007; 136:169-75.