Original Article

Correlation Between Upper Airways Obstructive Indexes in Adenotonsilar Hypertrophy with Mean Pulmonary Arterial Pressure

*Ehsan Khadivi[1], Mohsen Horri[2], Monavar Afzal Aghaee[3], Abolfazl Taheri[4]

Abstract

Introduction:
Hypertrophied tonsils and adenoids may cause upper airway obstruction and cardio-pulmonary complications due to pulmonary arterial hypertension. The aim of this study was to determine the correlation between mean pulmonary arterial pressure (mPAP) and selected adenotonsilar hypertrophy indexes.

Materials and Methods:
Thirty two patients with upper-airway obstruction resulting from hypertrophied tonsils and adenoids were included in our study. Mean pulmonary arterial pressure was measured by a non-invasive method using color doppler echocardiography. Upper airway obstruction was evaluated by clinical OSA (obstructive sleep apnea) scoring and also adenoidal-nasopharyngeal (A/N) ratio in the lateral neck radiography.

Results:
Fifty percent of the patients with a normal OSA score, 20% of those with a suspected OSA score and also 50% of cases with OSA had pulmonary hypertension (mPAP>20mmHg) which was not statistically significant (P=0.198). Mean Adenoidal-nasopharyngeal ratio in patients with a normal mPAP (mPAP≤20mmHg) was 0.61±0.048 and it was 0.75±0.09 in those with pulmonary hypertension; the difference was statistically significant (P=0.016).

Conclusion:
It seems that A/N ratio could be used as a predicting factor for increased mPAP in children with upper airway obstruction and a pediatric cardiologist consultation may be necessary before some surgical interventions.

Keywords:
Adenotonsilar, Echocardiography, Hypertrophy

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[1]*Ear, Nose, Throat Research Center, Department of otorhinolaryngolgy, Mashhad University of Medical Sciences, Mashhad, Iran*
[2]*Department of pediatrics, Mashhad University of Medical Sciences, Mashhad, Iran*
[3]*Department of social medicine, Mashhad University of Medical Sciences, Mashhad, Iran*
[4]*Department of otorhinolaryngolgy, Mashhad University of Medical Sciences, Mashhad, Iran*

[*]***Corresponding author:***
Imam Reza and Dr Sheikh Educational Hospitals, Mashhad University of Medical Sciences, Mashhad, Iran.
E-mail: khadivie@mums.ac.ir
Tel: +98 511 802 25 17
Fax: +98 511 859 40 82

Introduction

Increased upper airway resistance resulting from hypertrophied tonsils and adenoids can cause intermittent airway obstruction, chronic alveolar hypoventilation and can even lead to severe cardiopulmonary complications like cor-pulmonale (1-6). Although cardiopulmonary squeal of pulmonary hypertension including cor-pulmonale and ultimately death may develop in only a small number of children, these children appear to be part of a much larger group affected by partial upper airway obstruction, most commonly resulting from adenotonsilar hyperplasia (7). Presence of pulmonary hypertension in patients with adenotonsilar hypertrophy and its reversibility by surgical therapy has been assessed in many previous studies (8, 9). But the relation between severity of obstructive symptoms and pulmonary arterial pressure is not yet clear.

The aim of this study was to evaluate the relation between airway obstructive indexes and mean pulmonary arterial pressure in patients with adenotonsilar hypertrophy.

Materials and Methods

Children with obstructive or infectious symptoms due to adenotonsilar hypertrophy were randomly selected. Exclusion criteria was the presence of other causes of upper airway obstruction such as septal deviation, craniovertebral anomalies, subglottic stenosis and other secondary airway stenosis resulting from previous surgeries and cardiopulmonary diseases.

Thirty two children who were admitted in the otorhinolaryngology department for adenotonsilectomy due to upper airway obstructive or infectious symptoms underwent color doppler echocardiography and electrocardiography by a pediatric cardiologist using a General electric vivid 3 echocardiography machine, before surgery.

Mean pulmonary arterial pressure was evaluated by the noninvasive method of Mahan (mPAP=79-0.45× interval between onset to maximum flow) (8,9). OSA score was also assessed by clinical examination using the Brouillette questionnaire.

OSA score= 1.42 D+ 1.41 A + 0.71 S – 3.83

The D stands for sleep dyspnea (scored as: 0,1,2,3), A stands for apnea (scored as: 0,1) and S stands for snoring (scored as: 0,1,2,3). OSA score is borderline when -1<OSA<3.5 and in normal range when OSA<-1; it shows an obstructive condition in cases of an OSA>3.5 (10,11).

Lateral neck radiography was performed in all patients; adenoid to nasopharyngeal ratio was calculated for each case. This ratio has shown a direct correlation with the severity of obstructive symptoms according to previous studies (12).

The collected data were analyzed using Wilcoxon signed rank, Mann Whitney and Chi-Square tests.

Results

In total the male to female ratio was 1. Mean age was 7.06±1 years. In general, severe sleep dyspnea was observed in 28.1% of all cases, sleep apnea was seen in 50% and severe snoring was reported in 28.1%. Mean pulmonary arterial pressure (mPAP) was measured as 37.19±0.82 mmHg.

Mean adenoid to nasopharyngeal ratio (A/N ratio) was 0.65±0.05 (range, 0.4 to 0.94). Tonsilar size was very large (4+) in 34.4% and large (3+) in 37.5%, whereas the adenoid size was very large (4+) in 75% and large (3+) in 25%.

Mean OSA score was 1.13±0.38 (range, -3.83 to 3.97) and the mean OSA score for 1+ tonsilar size was 1.84, it was 0.636 in 2+ tonsilar size and 0.247 in 3+ tonsilar size but in 4+ tonsilar size the same score was 2.87; so the difference in the OSA score in these groups was statistically significant (P= 0.001). Mean OSA score in 3+ Adenoids was -0.81 and in 4+ large adenoids was 1.78 which also showed a significant difference (P=0.002).

OSA score was borderline (-1<OSA<3.5) in 63.5%, in normal range (<-1) in 12.5% and showed an obstructive condition (>3.5) in 25%.

Increased PAP (>20mmHg) was observed in 50% of patients with a normal OSA, 20% of those with borderline OSA and 50% of those with an obstructive condition based on the OSA score.
The difference between these groups was not statistically significant (*P*=0.198) (Table 1).

Table 1: Mean pulmonary arterial pressure in patients according to OSA score grouping

OSA score	Normal mPAP (>20 mmHg)	Increased mPAP (≤20mmHg)
Normal	2 (50%)	2 (50%)
Borderline	16 (80%)	4 (20%)
Obstructive	4 (50%)	4 (50%)

A/N ratio was in abnormal range (>0.53) in 82.1% of all children in our study.
Mean A/N ratio in those with a normal PAP was 0.61±0.048 and among those with an increased PAP was 0.75±0.09; so the difference in A/N ratio between the two groups was statistically significant (*P*= 0.016) (Fig 1).

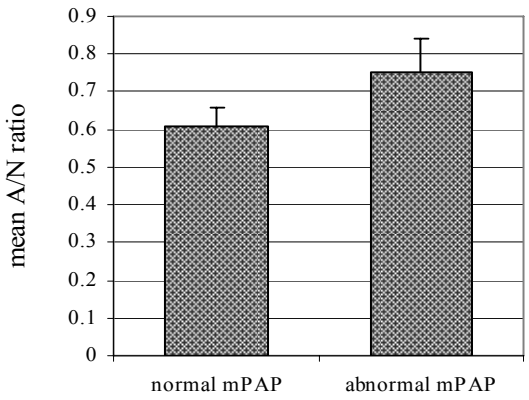

Fig 1: Mean A/N ratio in patients with normal and abnormal mPAP

Disscusion

This interventional study included 16 males and 16 females with age ranging from 3.5 to 13 years. Pulmonary arterial pressure could either be monitored by direct or indirect methods. Right heart catheterization was performed through the direct method which is an invasive approach. In the other method doppler echocardiography was used to estimate mPAP. Estimated mPAPs by Mahan's method have almost the same results as those of the invasive technique (8). Therefore, the non-invasive method was chosen in the current study in order to evaluate this index. In our patients mPAP was 19.37±82 mmHg preoperatively, with the minimum of 6 and a maximum of 25 mmHg. Resting mPAP in normal children is almost 20 mmHg and higher values are considered as pulmonary hypertension (8). Based on this definition, 10 of our studied children (31.3%) had pulmonary hypertension.
Obstructive symptoms were assessed by history taking using Brouillette et al questionnaire. They had concluded that a history of snoring, respiratory sleep disorders and obstructive apnea that can be reported by parents are the main criteria for OSA and suggested the following formula:
(OSA score= 1.42D+1.41A+0.71 S-3.83).
D stands for respiratory disorders, S stands for snoring and A stands for apnea (10). An OSA score more than 3.5 is considered as OSA (10). OSA scoring by Brouillette's questionnaire has a high predictive value, almost the same value as standard methods such as polysomnography (10). In our study, OSA score was normal (<-1) in 12.5%, borderline (-1<OSA<3.5) in 62.5% and abnormal (3.5<) in 25%. The difference between pulmonary hypertension in patients according to their OSA ranking was not statistically significant (*P*=0.198). mPAP was normal in 50% of those with OSA and also in those without OSA.
In Yilmaz et al study OSA was detected in 85% of patients with pulmonary hypertension; so they suggested OSA as a major causative factor for pulmonary hypertension (8).
Previous studies have recommended adenotonsillectomy for OSA scores ≥3.5 and have excluded surgical intervention for those with an OSA≤-1. Polysomnography would be helpful in the borderline group with 3.5>OSA>-1. Hence, this approach restricts the indications of polysomnography and facilitates the selection of surgical cases. We also performed lateral neck radiography and calculated the A/N ratio by Fujioka formula (12).

Mean A/N ratio was 0.65±0.05 in our series. An A/N ratio of less than 0.53 is considered normal according to previous studies (10). Regarding this fact 82.1% of our cases had an abnormal A/N ratio. Mean A/N ratio was 0.61±0.048 in children with a normal mPAP and 0.75±0.09 in children with pulmonary hypertension for which the difference was statistically significant (P= 0.016).

In another study the mean A/N ratio was 0.44 in children without obstructive airway symptoms, 0.49 in those with snoring, 0.62 in children with snoring and chronic mouth breathing and 0.75 in those with previous symptoms plus hyponasality. They concluded that A/N ratio should be higher than 0.73 in those with airway obstruction which is an indication for adenotonsillectomy (12).

So patients with A/N ratio higher than 0.73 are prone to severe airway obstructive complications and pulmonary hypertension and we may use lateral neck radiography to calculate A/N ratio as a new indicator for adenotonsillectomy.

Conclusion

According to the direct correlation between mPAP and A/N ratio in adenotonsilar hypertrophy, we suggest the A/N ratio as a predictor for pulmonary hypertension and a useful index in screening of those who need preoperative cardiologist consultation.

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