International Journal of Pediatric Otorhinolaryngology (2008) 72, 1425–1429

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International Journal of Pediatric Otorhinolaryngology

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Effect of upper airway obstruction on pulmonary arterial pressure in children

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Received 14 April 2008; received in revised form 31 May 2008; accepted 5 June 2008
Available online 17 July 2008

KEYWORDS
Adenotonsillar hypertrophy;
Pulmonary hypertension;
Upper airway obstruction

Summary

Objective: Our aim was to examine the elevation of pulmonary arterial pressure in children with upper airway obstruction caused by adenotonsillar hypertrophy according to their disease severity assessed with symptom scoring and to demonstrate the profit for echocardiographic monitorization of the children with adenotonsillar hypertrophy regardless of their clinical status.
Methods: Thirty-nine children with a diagnosis of upper airway obstruction caused by adenotonsillar hypertrophy were included for the study. There were 16 female and 23 male patients. Ages of the children were between 3 and 10 years with a mean age of 5.78 ± 1.98. Twenty children composed the control group with a similar age and sex distribution but without any sign and symptom of upper airway obstruction. Mean pulmonary arterial pressures were measured by Doppler echocardiography preoperatively and 6 months postoperatively. Symptom scores were calculated for each patient in the study group to assess their disease severity. The significances of changes and relations between pressure levels and symptom scores were calculated by statistical package for social sciences (SSPS) computer program in terms of Student's test, χ^2-test and Mc Nemar's test.
Results: Mean pulmonary arterial pressure were 26.26 ± 5.40 (14–36) preoperatively, $16{,}61 \pm 2.68$ (10.15–22.3) postoperatively and 16.54 ± 2.63 (10.5–21.7) in the control group. There were a statistically significant decrease at pressure levels postoperatively and a significant difference from the levels in the control group (Student's t-test, $p < 0.01$). We found no correlation between the pressure levels and disease severity assessed in terms of symptom scoring.
Conclusion: This study showed that upper airway obstruction caused by adenotonsillar hypertrophy causes significant elevation of pulmonary arterial pressures and adenotonsilectomy is an absolute therapeutic method in these children. Every child

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0165-5876/$ – see front matter
doi:10.1016/j.ijporl.2008.06.005

with adenotonsillar hypertrophy has some probability of having pulmonary hypertension regardless of his or her disease severity. Therefore, performing echocardiographic examination to all children with adenotonsillar hypertrophy is beneficial for assessing the cardiopulmonary status of the patient and may be useful at decision making for adenotonsilectomy.

1. Introduction

Upper airway obstruction (UAO) is known as the cause of various systemic abnormalities in children. It can have detrimental effects on the quality of life in children. There are some reports in the literature declaring these effects on somatic growth [1—3], poor school performance [4—7], hyperactivity [8] and cardiopulmonary system [9—15]. Cardiovascular deterioration is the most important one among these disorders which may lead to cor pulmonale and congestive heart failure. Although cardiopulmonary sequelae of UAO had been mentioned by anecdotal reports [16—18] in the literature, importance of this problem is best understood when considering the incidence of adenotonsillar hypertrophy (ATH) which is the most common cause of upper airway obstruction and obstructive sleep apnea in children.

Elevation of the pulmonary arterial pressure is the first step of the sequelae leading to cor pulmonale and congestive heart failure. Because of the absence of cardiovascular system-related symptoms in this period, changes at the pulmonary arterial pressure do not draw attention. At clinical practice, Doppler echocardiographic examination is not employed routinely unless severe obstructive sleep apnea (OSA) disease settles down and cardiovascular deterioration becomes established. Therefore, the following question arises. Should echocardiographic examination be done to all children with ATH and do the children with ATH but having milder symptoms are also under risk of pulmonary hypertension? Accurate diagnosis of OSA is made with polysomnography (PSG) but it is not easily employed, expensive and usually not possible at office practice. Because of these disadvantages of PSG, decision of referring the child to a cardiologist for echocardiographic examination and the decision of adenotonsilectomy is commonly made according to symptom questionnaire and physical examination findings by otolaryngologists.

Purpose of this study is to examine the relationship between UAO-related symptoms and pulmonary hypertension in order to have an idea about the answers of the questions asked above. The effect of adenotonsilectomy on pulmonary arterial pressure in children with ATH is also examined.

2. Materials and methods

The study group was selected among children who were admitted to ENToutpatient clinic of Haydarpasa Numune Education and Research Hospital with the complaint of recurrent adenotonsillar infection between March 2005 and November 2007. After getting approval from the ethic committee of our hospital, we obtained a written informed consent from the parents of the children. All children underwent a complete otolaryngologic examination including oropharyngal examination, otoscopy, anterior rhinoscopy and flexible nasopharyngoscopy if possible. Lateral radiographs are obtained. Hypertrophy of the tonsils was graded according to the system proposed previously by Brodsky [19]. Children having both grades 3 or 4 hypertrophied tonsils and hypertrophied adenoid large enough to obstruct the choana partially or completely are recruited for the study. Children having the pathologies which may cause UAO other than ATH such as allergic rhinitis, septum deviation, sinonasal infection and craniofacial anomalies were excluded from the study. Thirty-nine children meeting these criteria composed the study group.

In order to measure the symptom severity, parents of these children underwent a questionnaire including the questions about three major symptoms of UAO which are snoring, difficulty in breathing during sleep and apneas. A symptom score was calculated for each patient as proposed by Brouilette et al. [20] by applying the following the formula:

$$\text{OSA score} = 1.42D + 1.41A + 0.71S - 3.83$$

where D is difficulty breathing during sleep, A is apnea observed during sleep, and S is snoring. Values assigned to D and S were 0, never; 1, occasionally; 2, frequently; 3, always. Values assigned to A were 0, no and 1, yes.

A symptom score greater than 3.5 was interpreted as severe and diagnostic for OSA, symptom score between 3.5 and -1 was interpreted as moderate and suspicious for OSA; symptom score lower than -1 was interpreted as mild and indicate absence of OSA.

Control group constisted of 20 children within the same age range but without any sign and symptom of upper airway obstruction which were chosen among the children admitted to our clinic because of the reasons other than upper airway obstruction.

Table 1 mPAP levels and symptom scores in the study group

Preop mPAP ($n = 39$)	26.26 ± 5.40 (14 − 36)
Hypertensive, *n* (%)	33 (84.6%)
Normotensive, *n* (%)	6 (15.4%)
Postop mPAP	16.61 ± 2.68 (10.15 − 22.3)
Hypertensive, *n* (%)	2 (5.1%)
Normotensive, *n* (%)	37 (94.9%)
Preop symptom score ($n = 39$)	2.77 ± 1.30 (0.43 − 3.97)
Severe, *n* (%)	19 (48.7%)
Moderate, *n* (%)	20 (51.3%)
Postop symptom score	-2.11 ± 0.94 (−3.83 − 0.43)
Moderate, *n* (%)	3 (7.7%)
Mild, *n* (%)	36 (92.3%)

Complete two-dimensional, spectral Doppler, and color flow examinations by commercially available ultrasound instruments were performed to all subjects. Mean pulmonary arterial pressure (mPAP) were calculated by Mahan formula (mPAP (mmHg) = 79 − 0.45 × act; acceleration time of the pulmonary flow trace is the time interval between the beginning of the flow and its peak velocity) by the same cardiologist. The upper limit of pulmonary arterial pressure is 20 mmHg in children; therefore values exceeding this limit were evaluated as pulmonary hypertension. Mean pulmonary arterial pressure was estimated by Doppler echocardiography to all subjects in the control and study group. Adenotonsilectomy was performed to all subjects in the study group and Doppler echocardiografic examination was repeated 6 months postoperatively.

For statistical analysis, statistical package for social sciences (SPSS) program was used. Student's *t*-test was used for the assessment of quantitative data. Q^2-test, Fischer's exact q^2-test and Mc Near test were used for the assessment of qualitative data. Spearman's correlation analysis was used for the assessment of relation between pulmonary arterial pressure levels and symptom scores. Results were assessed between 95% confidence interval and $p = 0.05$ level.

3. Results

The mean age of the children in the study group was 5.78 ± 1.98 years (3—10 years). Sixteen (41%) of them were female whereas 23 (59%) of the were male. The mean pulmonary arterial pressure levels and symptom scores of the children in the study group preoperatively and postoperatively are shown in Table 1.

Preoperative mPAP levels were found to be very significantly higher than the mPAP levels of the children postoperatively and the children in the control group (Student's *t*-test, $p = 0.01$). But, postoperative mPAP levels do not statistically differ from the mPAP levels in the control group ($p > 0.05$) (Table 2). This shows us that pulmonary arterial pressure levels returned to normal at postoperative period.

Table 2 mPAP levels preoperatively and postoperatively

mPAP	Study, mean ± S.D.	Control, mean ± S.D.	*p*
Prop	26.26 ± 5.40	16.54 ± 2.63	0.001**
Postop	16.61 ± 2.68	16.54 ± 2.63	0.922

**statistically significant.

Proportion of pulmonary hypertensive children was found to be significantly higher than the children in the control group (χ^2-test, $p = 0.01$) but no statistically significant difference was observed between postoperative hypertensive children proportion and control group (χ^2-test, $p > 0.05$). Number of the children whom were hypertensive preoperatively was 33 out of 39. Only two children remained hypertensive after adenotonsilectomy. Change in the proportion of hypertensive children in the study group from preoperative period (84.6%) to postoperative period (5.1%) was found to be highly statistically significant (Mc Near test, $p = 0.01$) (Table 3).

Table 3 Distribution of the number of hypertensive and normotensive patients preoperatively and postoperatively

mPAP	Study, *n* (%)	Control, *n* (%)	*p*
Preop			
Hypertensive	33 (84.6%)	2 (10.5%)	0.001**
Normotensive	6 (15.4%)	17 (89.5%)	
Postop			
Hypertensive	2 (5.1%)	2 (10.5%)	0.591
Normotensive	37 (94.9%)	17 (89.5%)	

**statistically significant.

Table 4 Comparison of mPAP levels and symptom scores pre and postoperatively

	Symptom score preop		Symptom score postop	
	r	*p*	*r*	*p*
mPAP preop	0.080	0.630	–	–
mPAP postop	0.207	0.206	0.077	0.643

There is no statistically significant correlation between preoperative mPAP levels and preoperative symptom scores (r, 0.080; $p > 0.05$), and no significant correlation between postoperative mPAP levels and postoperative symptom scores (r, 0.077; $p > 0.05$). There seems to be a weak correlation between preoperative symptom scores and postoperative mPAP levels but it is not statistically significant (*Spearman correlation coefficient*, r, 0.207; $p > 0.05$) (Table 4).

There is no statistically significant difference at mPAP levels pre and postoperatively according to symptom score classification (Student's t-test, $p > 0.05$) (Table 5).

4. Discussion

It is well known that ATH is the most common cause of upper airway obstruction in children. This obstruction becomes more pronounced during sleep when the oropharyngeal musculature is relaxed. Sleep-related UAO in children may manifest as obstructive apnea or obstructive hypoventilation. Obstructive hypoventilation results from continuous partial upper airway obstruction, which leads to paradoxical respiratory efforts, hypercarbia and often hypoxemia. Hypoxemia and hypercarbia induced respiratory acidosis are potent mediators of pulmonary vasoconstriction leading to reversible or irreversible changes in the cardiopulmonary vasculature. Neurohumoral factors produced in response to hypoxemia may also promote the changes in pulmonary vasculature. Despite the absence of complete airway obstruction during sleep, children with obstructive hypoventilation are at risk for all of the reported complications of night time breathing obstruction [21]. There are various reported cases in the literature describing ATH as the cause of acute congestive heart failure and cor pulmonale [16–18]. Pulmonary hypertension is the first step of the sequelae leading to lethal cardiovascular complications. Cardiopulmonary system-related symptoms are absent or minimal during this period. This long lasting subclinical cardiac impairment may be attributed to a special unique physiological property of pulmonary vasculature. The normal vascular bed has high dispensability, and therefore increases in pulmonary blood flow may result in minimal change in pulmonary artery pressure. Hypoxemia and hypercarbia continues to exert their vasoconstrictive action. In order to maintain cardiac output, right ventricle compensate this situation for a long period by dilatation and hypertrophy. However, if this abnormal overload to RV is not altered, compensatory mechanisms become insufficient. This situation ends with rapid onset of cardiac decompensation [22].

Gold standard method for pulmonary arterial pressure measurement is by direct catheterization of right ventricle and pulmonary artery. This method is invasive, expansive and dangerous. Indirect measurement can be done by Doppler echocardiography using the Mahan formula. There is a good correlation between the mPAP calculated by Mahan formula in Doppler echocardiographic measurement and direct invasive catheterization method [23].

In our study, 33 children out of 39 were hypertensive in the study group and 2 children out of 20 were hypertensive at the control group. Among these 33 hypertensive children, only 2 of them remained hypertensive after adenotonsilectomy. Also, preoperative mPAP levels of the children in the study group were very significantly higher than the mPAP levels of the children in the control group. This agrees with the other studies in the literature [12–15] declaring ATH as the cause of pulmonary hypertension. Postoperative mPAP levels were statistically lower than the preoperative ones but were not statistically different from the mPAP levels of the children in the control group. This means that adenotonsilectomy is an curative method for reversing the changes in the pulmonary vasculature in terms of pulmonary arterial tension caused by ATH.

Sylmar et al. [12] used the same symptom scoring proposed by Brouilette et al. [20] in his study. He found 31 (59%) of the children having OSA out of 52 children with ATH. But, he did not make comparison between the mPAP levels of the children with OSA and without OSA. In our study, 19 (47%) children with ATH in the

Table 5 mPAP levels pre and postoperatively according to the symptom score calculated preoperatively

mPAP	Preop symptom score		*p*
	Severe, mean ± S.D.	Moderate, mean ± S.D.	
Preop	26.60 ± 5.14	25.92 ± 5.74	0.701
Postop	17.05 ± 3.07	16.20 ± 2.53	0.328

study group had severe symptoms and accepted as OSA while 20 (53%) children out of 39 had moderate symptom scores. We found no statistically significant difference between preoperative mPAP levels of the children with severe symptom scores and moderate symptom scores. Also, there were no significant correlation between postoperative mPAP levels and postoperative symptom scores. Regarding the fact that every child with ATH does not have OSA syndrome, we can conclude that every child with ATH is under risk of pulmonary hypertension and related cardiovascular complications even if he or she does not have severe symptom score reminding OSA syndrome. Also, we found that there is no statistically significant correlation between preoperative symptom scores and postoperative mPAP levels. This finding emphasizes the curative effect of adenotonsilectomy also at the children with severe disease.

In conclusion, we can say that UAO causes elevation of the pulmonary arterial pressure in children. If it is caused by ATH, adenotonsilectomy is an effective therapeutic method for reversing the arterial pressure change. Performing Doppler echocardiographic examination to the children with ATH is not mandatory but beneficial even if he or she does not have severe upper airway obstruction symptoms, because every child with ATH carries the risk of pulmonary hypertension independent from his or her symptom severity. The physician must take the mPAP level of the patient into consideration at the time of decision making for adenotonsilectomy when in doubt.

References

[1] C.L. Marcus, J.L. Carroll, C.B. Koerner, A. Hamer, J. Lutz, G.M. Laughlin, Determinants of growth in children with the obstructive apnea syndrome, J. Pediatr. 125 (1994) 556—562.

[2] R.M. Bland, S. Bulgarelli, J.C. Ventham, D. Jackson, J.J. Reilly, H. Paton, Total energy expenditure in children with obstructive apnea syndrome, Eur. Respire. J. 18 (2001) 164—169.

[3] A. Bar, A. Tarasiuk, Y. Segev, M. Philip, A. Tal, The effect of adenotonsilectomy on serum insulin-like growth factor-I and growth in children with obstructive sleep apnea, J. Pediatr. 135 (1999) 76—80.

[4] D. Gozal, Sleep-disordered breathing and school performance in children, Paediatrics 102 (1998) 616—620.

[5] G. Avior, G. Fishman, A. Leor, Y. Sivan, N. Keyser, A. Derowe, The effect of tonsillectomy and adenoidectomy on inattention and impulsivity as measured by the test of variables of attention in children with obstructive apnea syndrome, Otolaryngology. Head Neck Surge. 131 (2004) 367—371.

[6] B.C. Friedman, A. Hendeles-Amitai, E. Kozminsky, A. Leiberman, M. Friger, A. Torasik, et al., Adenotonsilectomy improves neurocognitive function in children with obstructive sleep apnea syndrome, Sleep 26 (2003) 999—1005.

[7] M. Weissbluth, A. Davis, J. Poncher, Signs of airway obstruction during sleep and behavioral, developmental and academic problems, Dev. Behave. Pediatr. 4 (1983) 119—121.

[8] R. Chervin, J. Dilton, C. Bassetti, D.A. Ganoczy, K.J. Pituch, Symptoms of sleep disorders, inattention, and hyperactivity in children, Sleep 20 (1997) 1185—1192.

[9] A. Tal, A. Leibermen, G. Margulis, S. Sofer, Ventricular dysfunction in children with obstructive sleep apnea: radio nuclide assessment, Pediatr. Palomino. 4 (1988) 139—143.

[10] G. Aljadeff, D. Gozal, V.L. Schectman, B. Burrell, R.W. Harper, S.L. Ward, Heart rate variability in children with obstructive sleep apnea, Sleep 20 (1997) 151—157.

[11] C.L. Marcus, M.G. Grene, J.L. Carrol, Blood pressure in children with obstructive sleep apnea, Am. J. Respir. Crypt. Care Med. 157 (1998) 1098—1103.

[12] M.D. Yılmaz, E. Ornat, A. Altuntaş, D. Kaya, O.K. Kahveci, O. Ozel, et al., The effects of adenoidectomy and tonsilectomy on pulmonary arterial pressure in children, Am. J. Otolaryngol. 26 (2005) 18—21.

[13] A. Pac, A. Karadag, H. Kurtaran, D. Aktas, Comparison of cardiac function and valvular damage in children with and without adenotonsillar hypertrophy, Int. J. Pediatr. Otorhinolaryngol. 69 (2005) 527—532.

[14] K. Görür, O. Döven, M. Unal, N. Akkus, C. Ozcan, Preoperative and postoperative cardiac and clinical findings of patients with adenotonsillar hypertrophy, Int. J. Pediatr. Otorhinolaryngol. 59 (2001) 41—46.

[15] M.C. Miman, T. Kirazli, R. Ozyurek, Doppler echocardiography in adenotonsillar hypertrophy, Int. J. Pediatr. Otorhinolaryngol. 54 (2000) 21—26.

[16] E.B. Kumar, N.S.V. Jaggaro, Adenotonsillar hypertrophy and cor pulmonale: clinical and echocardiographic operation, Postgrad. Med. J. 65 (1989) 473—475.

[17] S. Ramakrishna, V.S. Ingle, S. Patel, P. Bhatt, J.E. Dada, F.A. Shah, et al., Reversible cardiopulmonary changes due to adenotonsillar hypertrophy, Int. J. Pediatr. Otorhinolaryngol. 55 (2000) 203—206.

[18] C.Y. Kathleen, J. Sie, A. Perkins, A. Jonathan, S. Perkins, W.R. Clarke, Acute heart failure due to adenotonsillar hypertrophy, Int. J. Pediatr. Otorhinpharyngol. 41 (1997) 53—58.

[19] L. Brodsky, Modern assessment of tonsils and adenoids, Pediatr. Clin. North Am. 36 (1989) 1551—1569.

[20] R. Brouilette, D. Hanson, R. David, L. Kleimka, S. Anna, S. Fernbach, A diagnostic approach to suspected obstructive sleep apnea in children, J. Pediatr. 105 (1984) 10—14.

[21] L.M. Sternei, D.E. Tunkel, Obstructive sleep apnea, in: C.W. Cummings, J.M. Frederickson, L.A. Harker, C.J. Krause, M.A. Richardson, D.E. Schuller (Eds.), Otolaryngology Head and Neck Surgery, Mosey-Year Book Inc., St. Louis, 1998 , pp. 216—228.

[22] A. Widlitz, R.J. Barst, Pulmonary arterial hypertension in children, Eur. Respir. J. 21 (2003) 155—176.

[23] J.P. Laaban, B. Diebold, R. Zielinski, M. Lafey, H. Ralfaul, J. Rochemaure, Non-invasive estimation of systemic pulmonary arterial pressure using Doppler echocardiography in patients with chronic obstructive pulmonary disease, Chest 96 (1989) 1258—1262.