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## ORIGINAL ARTICLE

# Do pre-adenotonsillectomy echocardiographic findings change postoperatively in children with severe adenotonsillar hypertrophy

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## KEYWORDS

Childhood;  
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**Abstract** *Purpose:* Hypertrophy of adenotonsillar tissue is one of the most common problems in childhood age and causes upper airway obstruction and even obstructive sleep apnea symptoms in severe forms. The aim of this study is to evaluate the changes of pre-adenotonsillectomy echocardiographic findings after operation.

*Material and methods:* From August 2007 to November 2008, 55 children with adenotonsillar hypertrophy and obstructive-sleep symptoms (aged 3–11 years old of which 35 were males and 20 females) were randomly selected. Preoperatively echocardiography was performed for all patients by the pediatric cardiologist.

In control group 55 children who referred to otolaryngology clinic because of non adenotonsillar related disease evaluated. One month and six months after operation, patients with positive findings were followed up and again echocardiography was performed separately.

*Results:* No complaints of apnea were reported. Tonsillar grades of all cases were type III or IV. The preoperative mean pulmonary arterial pressure levels of the 4 (7.3%) children were higher than normal range (25 mm Hg) and it decreased significantly after operation ( $P < 0.000$ ) (Preoperative MPAP =  $32 \pm 3$  mm Hg, and six months postoperative follow up, MPAP =  $11 \pm 5$  mm Hg). All

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the patients' parents complained about severe open mouth snoring, agitated sleep and hyperpnoea of their children.

The preoperative tricuspid regurgitation pressure level of 7 children was higher than normal range and it decreased significantly after operation ( $P < 0.000$  preoperative TR =  $34 \pm 8$  mm Hg post-operative TR =  $19 \pm 6$  mm Hg). AC/ET in these seven patients were lower than 0.4. In control group echocardiography findings are normal.

**Conclusion:** Severe chronic hypertrophic adenotonsillar tissue caused higher tricuspid regurgitation pressure and mean pulmonary arterial pressure.

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## 1. Introduction

The lymphoid tissues hypertrophy of the pharynx is the most common causes of the obstructive symptoms and surgical tonsil intervention is observed in this age group (Anhtaseree et al., 2001; Gislason and Benediktadottir, 1995; Suen et al., 1995). Currently obstruction takes the first order among adenotonsillectomy indication (Miman et al., 1999). Increased upper airway resistance resulting from hypertrophied tonsils and adenoids can cause intermittent airway obstruction, chronic alveolar hypoventilation, and even lead to severe cardiopulmonary complications like cor pulmonale (Broadsky, 1993). The detail of cardiopulmonary echocardiographic changes in children with grade III and IV obstructive adenotonsillar tissue have not been investigated yet. The aim of our study is to compare the changes of preoperative echocardiographic findings of these patients with a sixth month follow up postoperatively.

## 2. Materials and methods

The study consisted of 55 children with grade III or IV adenotonsillar hypertrophy (35 males and 20 females, aged range 3–11 years). This study was approved by ethic board of Tabriz medical university. The most common complaints of the patients were snoring, mouth breathing and pausing of breath during sleep as well as apnea and hypopnea. Brodsky scale was used for the tonsillar hypertrophy grading as follows.

Grade I: tonsils in tonsillar fossa and barely visible behind the anterior pillars; Grade II: tonsils in tonsillar fossa and easily visible behind the anterior pillars; Grade III: tonsils extended three quarters of the way to the midline; and Grade IV: complete obstruction of the airway by enlarged tonsils. Children with any known or suspected cardiovascular disease, metabolic disease, neurological disease, obesity (BMI > 29), and upper airway obstruction secondary to other causes such as allergic rhinitis, recent upper respiratory tract infection, septal deviation and proven skull base anomaly by imaging were excluded from the study. All children underwent a complete otolaryngological examination, otoscopy and anterior rhinoscopy by the same otolaryngologist. They were examined using two dimensional coloured Doppler and M-Mode Doppler echocardiography (Vivid 3 Npro/Expert, made in Norway) with 2.5 MHZ duplex imaging transducer. Echocardiographic studies were performed by a single experienced pediatric cardiologist who was blinded to patients' names and diagnosis. We measured maximum interventricular septum thickness, acceleration time, ejection time, mean pulmonary

artery pressure (MPAP) (by tracing pulmonary flow and using Mahan formula  $MPAP \text{ mm Hg} = 90 - [0.62 \times ACT]$ ), systolic pulmonary pressure (by tricuspid regurgitation velocity using Bernoli equation:  $\text{systolic pulmonary pressure} = 4V^2 \times 1.23 \text{ mm Hg}$ ). We also measured peak velocity of flow and ejection time (the total duration of flow in systolic on the Doppler tracing). Moreover, we measured infra vena cava diameter and its change with respiratory cycle. Peak velocity was unchanged. However, the acceleration time was shortened in case of pulmonary hypertension ( $AT/ET = 0.45 \pm 0.05$  normal) (Allen et al., 2001; Snider et al., 1997; Arthur Garson et al., 1998; Marcus et al., 1994).

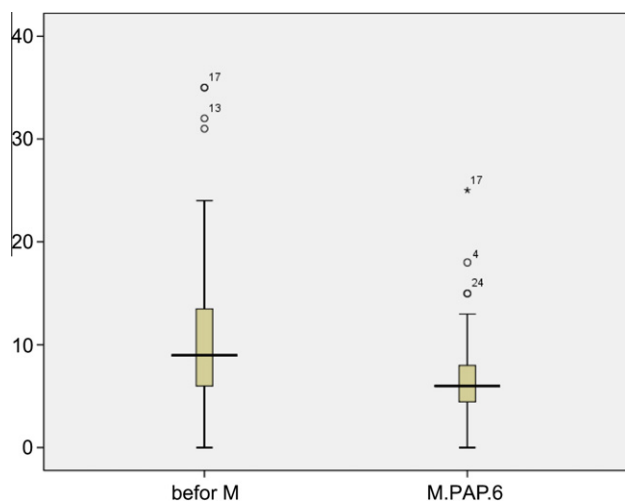
ACT = Acceleration time of the pulmonary flow trace: The time interval between the beginning of the flow and its peak velocity, MPAP = Mean pulmonary artery pressure, AT = Acceleration time, ET = Ejection time. The study was approved by Ethics committee of Tabriz medical university and parental informed consent was obtained. All subjects underwent adenotonsillectomy by cold knife technique under general anesthesia and Doppler echocardiographic measurements were repeated one and then six months postoperatively in whom had positive echocardiographic findings preoperatively. In control group 55 children who referred to otolaryngology clinic because of non adenotonsillar related disease evaluated. The results were analyzed findings preoperatively. The results were analyzed with the spss software. Continuous variables are presented as mean  $\pm$  SD. The qualitative values were compared by  $\chi^2$ -test, and the significance of difference quantitative values of pre and post-operation were estimated by means of independent paired-sample *t*-test. A *P*-value < 0.05 was considered significant.

## 3. Results

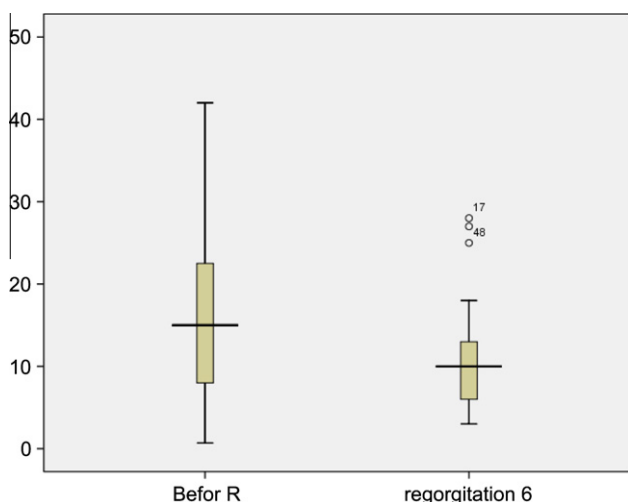
Forty seven (85/5%) children were identified to have grade IV enlarged tonsil. The minimum and maximum ages of the children were 3 and 11, respectively.

All children (100%) had night snoring with open mouth breathing and agitated sleep at least six months before their visits. Eleven (20%) had a history of respiratory pause similar to hypopnea, but none of them suffered from apnea. None of the patients had chest pain during physical activity, chronic fatigue, dizziness or fainting.

The preoperative MPAP of 4 (7.3%) cases were above 25 mm Hg ( $P < 0.000$  Npar test) (Preoperative MPAP =  $32 \pm 3$  mm Hg). The preoperative tricuspid regurgitations in 48 (87.3%) were lower than 25 mm Hg and in 7 (12.7%) were more than 25 mm Hg (TR =  $34 \pm 8$  mm Hg). Interventricular septal diameter in all children was lower than 4 mm.



The minimum, mean and maximum of preoperative MPAP were 5, 11, 35 mm Hg, respectively. Again the minimum, mean and maximum of preoperative tricuspid regurgitation pressure was 0.7, 15.56 and 42 mm Hg, respectively.



AT/ET ratio was lower than  $0.3 \pm 0.059$  in 7 patients who had increased tricuspid regurgitation. All of these 7 patients were male ( $P < 0.000$ ).

Caval index in all patients was higher than 50% in the first month's postoperative follow up. The mean pulmonary artery pressure and the tricuspid regurgitation pressure of those 7 patients decreased to normal levels after the first month follow up. All children of control group had normal echocardiographic findings.

#### 4. Discussion

Children with obstructive disorders during sleep were exposed to repeated hypopnea in addition to infective and obstructive symptoms.

During apnea, there is a progressive decrease in oxygen levels and increase in  $\text{CO}_2$  levels. Hypercapnia and hypoxemia

provoke respiratory acidosis and consequently vasoconstriction of the pulmonary artery.

In addition to increased venous return due to increased pulmonary resistance, there is also increased venous return to the right cardiac chambers, which is facilitated both by the decubitus horizontal position during sleep and by intra thoracic pressure which becomes more negative due to respiratory effort against the obstructive area. These types of changes can lead to an enlarged right atrium and ventricle and compromise ejection during systole (Phillips, 2005; Steiner and Straur, 2004; Verrier et al., 2000).

Pulmonary hypertension was defined as pulmonary artery systolic pressure at least 30 mm Hg corresponding to a peak tricuspid regurgitation jet velocity of 2.5 m/s. Mild pulmonary hypertension was defined as tricuspid regurgitation jet velocity 2.5–2.9; moderate pulmonary hypertension was defined as tricuspid regurgitation jet velocity 3 m/s (Lindberg et al., 2008).

Signs and symptoms of pulmonary hypertension may not be obvious at first, but they can be worsened over time and can begin to limit daily activities. Symptoms of Pulmonary hypertension include: breathlessness, chronic fatigue, dizziness, faintness, swollen ankles and legs, chest pain, especially during physical activity. Functional classification of pulmonary hypertension include as follows:

- (I) Without limited physical activity and comfortable at rest; ordinary physical activity does not cause dyspnea or fatigue, chest pain or near syncope.
- (II) Mild limited physical activity but comfortable at rest; ordinary physical activity cause undue dyspnea or fatigue, chest pain or near syncope.
- (III) Marked limited physical activity but comfortable at rest; less than ordinary activity cause undue dyspnea or fatigue, chest pain or near syncope.
- (IV) Inability to carry out any physical activity without symptoms; discomfort is increased by any physical activity (Farzana et al., 2009).

Maurizi et al. (1980) described that 65.7% of clinically normal children with adenoid hypertrophy showed pulmonary functional abnormalities. Also, higher MPAP values of children with diagnosis of upper airway obstruction resulting from hypertrophied tonsils and adenoids have been reported by Yilmaz et al. (2005).

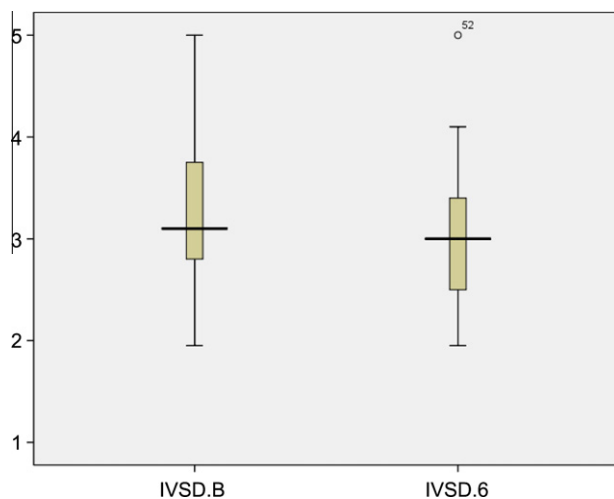
Guney in his study recorded a significant recovery in respiratory functional tests and blood gas levels after T&A in patients with adenotonsillar hypertrophy (Gueny, 1974). In our study, the preoperative MPAP values of four patients were more than 25 mm Hg and in 24 patients were more than 12 mm Hg. Additionally, significant decrease in MPAP values of those groups were detected in the first month and then at the sixth month follow up echocardiography postoperatively ( $P < 0.005$ ).

In our study preoperatively tricuspid regurgitation pressure in seven patients (12.7%) was higher than 25 mm Hg and all of the patients had normal tricuspid regurgitation pressure in the first month and sixth month post operative echocardiography.

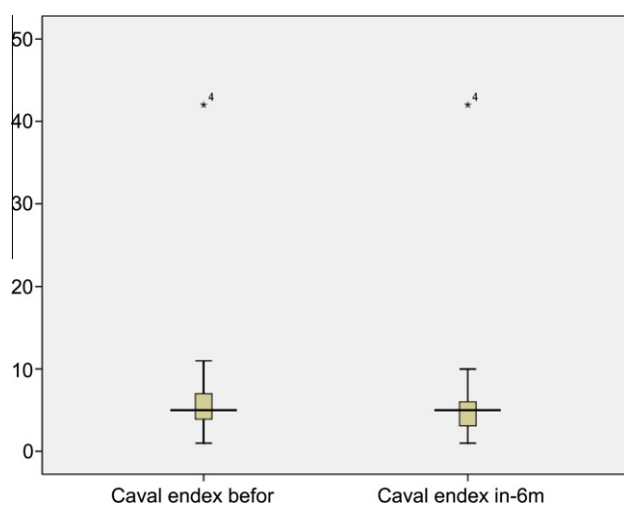
Similar to our results, Pac et al. compared cardiac functions and valvular damages in children with and without adenotonsillar hypertrophy and reported differences in tricuspid and diastolic time preoperatively ( $104.8 \pm 28.8$  ms) versus

postoperatively ( $86.4 \pm 17.32$  ms) ( $P < 0.005$ ) (Pac et al., 2005).

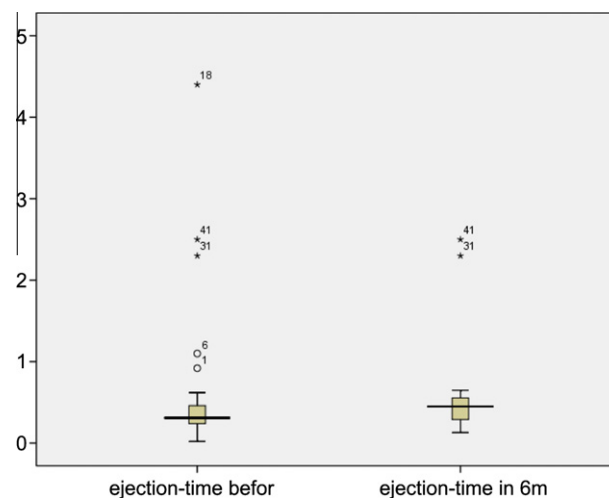
Marcus et al reported that increased  $O_2$  saturation and disappearance of apnea episodes after adenotonsillectomy (Marcus et al., 1994). In our study, IVSD in all patients were lower than 4 mm which Garur et al. reported significant improvement in right ventricular diameter (Gorun et al., 2001).



Also Miman et al. reported dramatic decrease in mean preoperative pulmonary arterial pressure after relief of upper airway obstruction ( $29.12 \pm 4$  mm Hg preoperatively versus  $12.06 \pm 3.09$  postoperatively) (Miman et al., 1999).



On the other hand, Kalra et al. demonstrated RV and IV cardiac abnormalities in children with respiratory complications after adenotonsillectomy due to OSAS suggesting that prior identification of these cardiac abnormalities may be of aid in preventing this complication (Kalra et al., 2005).



Duman et al. reported subclinical right ventricular dysfunction in advanced stage of adenotonsillar hypertrophy children (grad III–V), which may reverse these cardiac changes (Duman et al., 2008). In our study, subclinical right ventricular dysfunction was discovered as well. Naiboglu et al. in their study showed that pulmonary arterial pressure significantly raised in children with obstructive hypertrophic adenotonsillar tissues and concluded adenotonsillectomy was the absolute therapeutic method in these children (Naiboglu et al., 2008).

The AT/ET ratio was lower than  $13 \pm 0.059$  ( $AT/ET = 0.45 \pm 0.05$  normal) in seven patients who had higher tricuspid regurgitation pressure because some factors such as age, HR, RV preload, sample volume position and RV function (Snider et al., 1997) alter Doppler interval time in pulmonary hypertension. So only by lower AT/ET we could not come to an exact conclusion about patients and evaluate other echocardiographic findings.

We suggested that echocardiography is sometimes helpful in decision making for surgeon. In conclusion, this study shows that chronic obstructive adenotonsillar hypertrophy causes significant cardiovascular changes and early detection and treatment of these patients are necessary for the relief of cardiovascular threatening complications in future.

We didn't evaluate our patients by polysomnography; we have limited number of patients and may be it was a limitation for our study.

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