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Assessment Of Cardiac Functions In Patients With Adenotonsillar Hypertrophy

Adenotonsiller Hipertrofisi Olan Hastalarda Kardiyak Fonksiyonların Değerlendirilmesi

ABSTRACT

Background: The aim of this study is to compare cardiac function in children with and without adenotonsillar hypertrophy (ATH).

Materials and methods: Ninety-one children (26 female 65 male) who were diagnosed as ATH in the pediatric outpatient clinic and twenty-three completely healthy, age-sex matched children (10 female 13 male) were included in the study. All patients underwent a complete two-dimensional transthoracic echocardiographic and Doppler study.

Results: Mean mitral E, A and deceleration time were significantly longer in ATH group. Also chamber areas and volumes were bigger. Pulmonary and mitral regurgitation were statistically more frequent in ATH group. Adenotonsillar grade was positively related with mean pulmonary arterial pressure (r: 0.44 p: <0.001). Mitral valve thickness was strongly correlated with tonsillar hypertrophy grade (r: 0.73; p:<0.001).

Conclusions: ATH may lead to mild diastolic dysfunction and chamber dilatation. Mitral valve thickness was strongly correlated with adenotonsillar grade.

Keywords: Adenotonsillar hypertrophy, apnea, cardiac function, rheumatic heart disease, snoring, subclinical carditis

ÖZET

Amaç: Bu çalışmanın amacı, adenotonsiller hipertrofisi (ATH) olan ve olmayan çocuklarda kardiyak fonksiyonları değerlendirmektir.

Materyal ve Metod: Pediatri polikliniği tarafından adenotonsiller hipertrofi tanısı konmuş doksan bir çocuk (26 kız, 65 erkek) ve 23 sağlıklı olgu, yaş ve cinsiyet eşleştirilmeli sağlıklı çocuklar (10 kız, 13 erkek) çalışmaya dahil edilmiştir. Tüm hastalara 2D ekokardiyografi ve Doppler tetkiki yapılmıştır.

Bulgular: Ortalama mitral E, A dalgaları ve deselerasyon zamanı, ATH olan grupta daha uzun bulunmuştur. Aynı zamanda kalp boşlukları ve hacimleri de daha yüksek ölçülmüştür. Pulmoner ve mitral regürjitasyon oranı, ATH olan grupta daha sık saptanmıştır. Adenotonsiller grade, ortalama pulmoner arterial basınçla ilişkili bulunmuştur. (r: 0.44 p: <0.001). Mitral kapak kalınlığı, tonsiller hipertrofi derecesiyle güçlü bir şekilde korele saptanmıştır. (r: 0.73; p:<0.001).

Sonuç: ATH, hafif diastolik disfonksiyon ve kalp boşluklarında dilatasyona yol açabilmektedir. Mitral kapak kalınlığı, adenotonsiller grad ile güçlü bir şekilde korele bulunmuştur.

Anahtar Kelimeler: Adenotonsiller hipertrofi, apne, kardiyak fonksiyon, romatizmal kalp hastalığı, horlama, subklinik kardit

INTRODUCTION

Adenotonsillar Hypertrophy (ATH) is the most common obstructive manifestation in patients with recurrent upper respiratory tract infections. ATH is closely related with recurrent tonsillitis attacks resulting from streptococcus infections (1). Infection of the tonsillar crypt can lead to colonization of the core providing for further spread of the organism (2,3). ATH may be a risk factor for rheumatic heart disease (RHD)2,3 which is a major public health concern in developing nations and indigenous communities in industrialized countries (4). Cardiac involvement has been reported in different studies (2,3). A form of valvulitis can exist in a proportion of patients with RHD. Pathological valvular regurgitation is not evident clinically in these patients, however can be detected by echocardiography (5). One concern impeding the widespread acceptance of this subclinical carditis as part of the spectrum of

rheumatic valvular damage is uncertainty around the differentiation between normal and pathological valvular regurgitation, raising the possibility that normal children may be inappropriately labeled as having RHD (6). The issue of children who had ATH, are at increased risk for rheumatic cardiac complications or subclinical involvement is also unclear (3). The frequency and evolution of subclinical valvulitis in patients with ATH has not been established yet. The objectives of our study were to investigate cardiac functions in patients with ATH.

MATERIALS AND METHODS

Study population: The present study was conducted in the departments of otolaryngology and cardiology. Ninety-one children (26 female 65 male; with a mean age of 7) who were diagnosed as ATH in the pediatric outpatient clinic were included in the study. Twenty-three completely healthy, age-sex matched children (10 female 13 male; with a mean age of 7) were included as the control group. The parents of every subject signed an informed consent. Duzce University ethics committee has approved the study.

Otorhinolaryngologic evaluation: All patients underwent complete endoscopic otorhinolaryngologic examination and tonsillar hypertrophy was graded according to Brodsky scale as follows: Grade I: tonsils were in the tonsillar fossa, barely visible behind the anterior pillars; Grade II: tonsils were easily visible behind the anterior pillars; Grade III: tonsils extended three-quarters of the way to the midline; and Grade IV: tonsils were completely obstructing the airway. Adenoid hypertrophy was graded according to severity of the airway obstruction: Grade I: indicates <25%; Grade II: indicates 25—50%; Grade III: indicates 50—75%;

and Grade IV: indicates >75% obstruction. Children suffering from any cardio respiratory or renal diseases, or having upper airway obstruction due to other causes like nasal polyps were excluded from the study.

Echocardiographic measurements: All patients underwent a complete two-dimensional transthoracic echocardiographic and Doppler study in the left lateral decubitus position from multiple windows. Children younger than 4 years were anesthetized with oral chloral hydrate 50 mg/kg. Studies were performed using standard techniques, with subxiphoidal, precordial, apical, and suprasternal imaging measurements. All studies were performed with Vingmed Vivid-3 (General Electric, Haifa, Israel) echocardiograph and a 2.5 MHz transducer. Gain, depth, and sector angles were individualized for the best measurement. For each echocardiographic method, measurements of at least three cardiac cycles were averaged. A single echocardiographer who was blinded to the results of the study performed all echocardiographic studies.

Statistical analysis: All values are given as mean \pm standard deviation. Statistical Package for Social Sciences software (SPSS 10.0, Chicago, IL, USA) was used for comparisons of demographic and clinical variables. Unpaired Student's t test was used for group comparisons. Categorical data were compared with the chi-square test. Medcalc software (Medcalc, Mariakerke, Belgium) was used to calculate cut points for receiver operating characteristic curve (ROC) analyses, sensitivity, specificity, and positive and negative predictive values. A p value of < 0.05 was considered significant.

Characteristics	ATH (N:91)	Control (N:23)	P value
Sex (F)	26	10	0.169
Age(year)	7.58 \pm 3.1	7.04 \pm 2.8	0.428
Height(cm)	122.4 \pm 17.7	112.7 \pm 28.2	0.345
Weight(kg)	24.75 \pm 10.2	22.94 \pm 7.21	0.274
URT frequency (episode)	21.16\pm13.71	1.04\pm5.00	<0.001
Snoring	53	1	<0.001
Sickness period (month)	21\pm13	0	<0.001
Mouth Breathing	49	2	<0.001
Apnea	17	0	0.024

Table 1: Clinical and demographic characteristics

	ATH group	Control Group	P value
Left atrial Diameter (mm)	24.4±3.9	23.2±2.6	0.06
Septum (mm)	5.4±0.8	4.9±0.6	0.113
Posterior wall (mm)	5.6±0.8	5.3±0.6	0.07
LVDD (mm)	37.2±4.6	33.3±7.4	0.6
LVSS (mm)	23.6±4.0	21.6±2.5	0.1
Ejection Fraction %	66.6±3.8	69.1±1.9	0.003
Mitral E (m/sec)	1.1±0.15	1.07±0.09	0.014
Mitral A (m/sec)	0.70±0.15	0.61±0.08	0.025
Deceleration time (msec)	138.8±28.5	120.8±16.9	0.013
E/A	1.56±0.33	1.75±0.24	0.192
Isovolumetric contraction time(msec)	39.84±14.8	35.3±11.0	0.088
Isovolumetric relaxation time (msec)	62.3±17.8	63.7±14.9	0.490
Mitral excursion	1.15±0.18	1.18±0.12	0.062
Mitral valve thickness (mm)	3.68±0.82	2.57±0.55	0.106
Left ventricular systolic volume (ml)	16.4±7.4	14.5±4.0	0.02
Left ventricular systolic Area (cm²)	8.9±2.8	8.3±1.5	0.03
Left ventricular diastolic volume (ml)	57.2±14.3	44.5±17.2	0.01
Left ventricular diastolic area (cm²)	16.9±4.4	15.8±2.5	0.03
Left atrial systolic volume	6.7±3.7	5.5±2.2	0.08
Left atrial systolic area	5.0±1.7	4.5±1.1	0.057
Left atrial diastolic volume (ml)	20.1±8.8	17.0±3.7	0.006
Left atrial diastolic area (cm²)	10.3±2.9	9.3±1.3	0.002
Left atrial ejection Fraction	68.4±5.5	68.8±5.9	0.8
Pulmonary regurgitation (n)	54	13	<0.001
Mitral regurgitation (n)	42	4	<0.001
Mitral regurgitation (pathological) (n)	5	0	<0.001

[(LVDD: Left ventricular diastolic diameter (mm), LVSS: Left ventricular systolic diameter (mm)]

Table 2: Comparison of Doppler and 2-D echocardiographic measurements

RESULTS

Comparison of demographic and clinical variables of the patients and control subjects were shown in Table 1. Upper respiratory tract infection episodes were more frequent in the ATH group. Snoring, apnea and open-mouth breathing during sleeping were also more frequent in the ATH group. None of the patients had RHD according to Jones criteria.

Comparison of left heart chamber Doppler and 2-D echocardiographic measurements were shown in Table 2. Mean mitral E, A and deceleration time were significantly longer in ATH group. Also chamber

areas and volumes were bigger. Pulmonary and mitral regurgitation were statistically more frequent in ATH group.

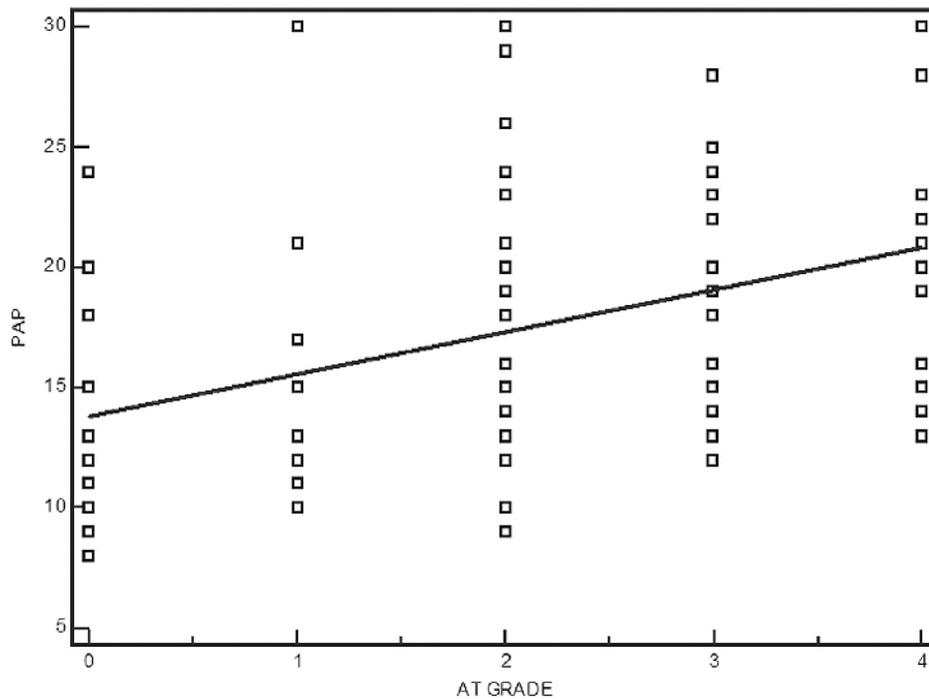
Comparison of right heart chamber Doppler and 2-D echocardiographic measurements were shown in Table 3. Right heart chamber diameters, areas and volumes were also higher in ATH group except right atrial systolic parameters.

Adenotonsillar grade was positively related with mean pulmonary arterial pressure ($r: 0.44$ $p: <0.001$, Figure 1). We also compared mitral valve thickness between the groups. Although statistically

	ATH group	Control Group	P value
Right ventricular systolic volume (ml)	6.8±4.1	5.87±1.9	0.027
Right ventricular systolic area (cm ²)	4.6±1.7	4.3±0.8	0.007
Right ventricular systolic diameter	2.8±0.6	2.7±0.3	0.009
Right ventricular diastolic volume (ml)	20.0±10.2	17.2±4.2	0.004
Right ventricular diastolic area (cm ²)	10.3±3.4	9.6±1.4	0.001

Table 3: Comparison of right heart Doppler and 2-D echocardiographic measurements

Figure 1: Correlation graph of adenotonsillar grade with pulmonary arterial pressure

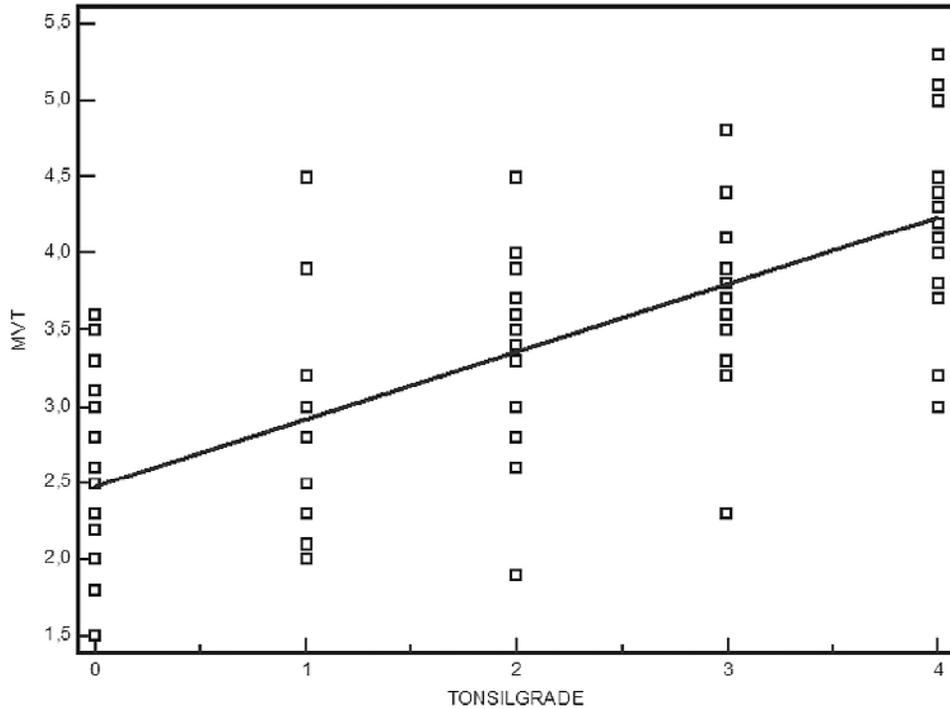


insignificant, mitral valves were thicker in ATH group (3.68 ± 0.82 vs. 2.57 ± 0.55 mm, Table 1). Furthermore, mitral valve thickness was strongly correlated with tonsillar hypertrophy grade ($r: 0.73$; $p < 0.001$, Fig. 2).

DISCUSSION

In the present study we found a significant positive correlation between adenotonsillar grade and pulmonary arterial pressure. Mitral valve thickness was strongly correlated with adenotonsillar grade. Right and left heart volumes were significantly higher in children with ATH than the age matched controls. Adenotonsillar hypertrophy is a common cause of upper airway obstruction in children. Children with ATH may develop cardiopulmonary complications associated with hypercarbia and hypoxemia. Pulmonary hypertension is a well-known complication of ATH. ATH can exacerbate pulmonary hypertension due to the vasoconstrictive effects of hypoxia and hypercarbia (7). In order to

maintain cardiac output, the RV compensates the progressive increases in pulmonary vascular resistance through a combination of dilation and hypertrophy. Gorur et al. investigated 33 children with ATH pre- and post-surgery, and compared findings to those in control subjects (8). At 6 months after adenotonsillectomy, they observed significantly improved right ventricular diameter, LVED and IVSS. The authors concluded that adenotonsillar disease led to RV and/or LV enlargement, and that these changes resolved over time postoperatively⁸. Pac et al also found same results and proposed prolonged tricuspid deceleration time suggests that early stage of right ventricular diastolic dysfunction³. We also compared the chamber volume, area and diameters and found that mean right ventricular, left ventricular, left atrial systolic and diastolic volumes were increased in patients with ATH.

Figure 2: Correlation graph of tonsillar grade with mitral valve thickness.

We also demonstrated that diastolic function variables (mitral E, Mitral E/A deceleration time) increased in ATH. These findings are concordant with the reports supporting that ATH leads an early stage of ventricular diastolic dysfunction.

Permanent valvular disease suggesting RHD was demonstrated in patients with ATH. However, there are conflicting manuscripts reporting no significance in valvular damage between ATH and control groups being that ATH was not a risk factor for RHD (2). In our study, we found that mean mitral valve thickness was strongly correlated with tonsillar grade supporting that there is an association between mitral valve degeneration and ATH. Valvular degeneration may be a manifestation of ATH and may not mean that it is a risk factor for RHD. Further prospective follow-up studies are needed for confirmation.

In conclusion, ATH may lead mild diastolic dysfunction and chamber dilatation. The chamber dilatation may lead further valvular regurgitation and increase in pulmonary artery pressure. ATH is also associated with valvular degeneration and RHD should be ruled in these patients. ATH patients should be considered to have RHD only if they meet Jones criteria to prevent overdiagnosis of RHD due to co-existing valvular degeneration.

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