



The effects of adenotonsillar hypertrophy corrective surgery on left ventricular functions and pulmonary artery pressure in children



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ARTICLE INFO

Article history:

Received 3 July 2017

Accepted 20 July 2017

Available online 22 July 2017

Keywords:

Adenotonsillar hypertrophy (ATH)

Left ventricle (LV)

Tissue doppler echocardiography

ABSTRACT

Objective: Comparison of left ventricular functions in preoperative and postoperative periods of children with adenotonsillar hypertrophy (ATH) who have findings of upper airway obstruction (UAO), using echocardiographic parameters.

Methods: Thirty children who were diagnosed with UAO due to ATH, and who have undergone adenoidectomy/adenotonsillectomy and 30 healthy children, between 2 and 11 years of age, were included in the study. Patient group was evaluated by the pulsed wave tissue Doppler echocardiography, as well as with conventional echocardiography, before and 6 months after the operation.

Results: Of 30 children in study group, 18 (60%) had adenotonsillectomy and 12 (40%) had adenoidectomy. The differences between groups regarding myocardial performance index (MPI) was not statistically significant ($p = 0.847$). There was not any statistically significant difference between groups in terms of mitral isovolemic acceleration (MIVA) (2.28 ± 0.67 , 2.24 ± 0.55 , 2.23 ± 0.49 ; $p = 0.943$, respectively). Interventricular septum diameter (IVSD) was significantly higher in preoperative group than postoperative and control groups (3.68 ± 0.52 , 3.50 ± 0.40 , 3.38 ± 0.60 ; $p = 0.028$, respectively). Pulmonary acceleration time (PACT) was found to be significantly lower in preoperative group compared to postoperative and control groups (107.64 ± 16.60 , 119.52 ± 15.95 , 120.47 ± 16.19 ; $p = 0.004$, respectively). Mean pulmonary arterial pressure (mPAP) was significantly higher in preoperative group than postoperative and control groups (30.58 ± 8.11 , 25.23 ± 9.07 , 25.00 ± 6.52 ; $p = 0.002$, respectively). In postoperative group mPAP was found to be similar to the control group.

Conclusions: Clinical or subclinical left ventricle (LV) dysfunction in children with ATH who have findings of UAO was not determined while mean pulmonary arterial pressure was significantly higher compared with the control cases. Besides early adenotonsillectomy is a beneficial treatment option for these patients.

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1. Introduction

Upper airway obstruction (UAO) during sleep comprises a large spectrum of symptoms varying from night time snoring to the obstructive sleep apnea-hypopnea syndrome. The most common symptoms defined are the snoring, respiratory pauses, difficulty in breathing, agitated sleep and nocturnal diaphoresis [1]. Adenotonsillar hypertrophy (ATH), the excessive lymphoid tissue proliferation on adenoids and tonsils, is the most common cause of upper air-way obstruction in pediatric population [2–4].

Severe UAO can result in chronic alveolar hypoventilation, which may cause hypoxemic pulmonary vasoconstriction and the resulting rise in pulmonary vascular resistance and pulmonary arterial pressure (PAP). Dilatation and hypertrophy of the right side of the heart and subsequent right ventricular dysfunction are the end results of persistent pulmonary vascular resistance [5,6]. Early recognition of ventricular dysfunction is important in prevention of further progression to heart failure and even death and also in decision of requirement of surgical treatment [7,8]. Recently, tissue Doppler echocardiography (TDI) has been defined as a reliable and accurate technique in evaluation of global and regional ventricular functions [9].

Although the data in literature is conflicting, majority of studies suggest that severe UAO contributes to the development of left

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ventricular systolic and diastolic dysfunctions and subsequently heart failure [10]. However, the exact pathophysiological mechanisms linking UAO to cardiovascular disease or the long-term impacts of UAO on cardiovascular system were not defined clearly, yet.

Even though there are many studies about the effects of ATH on left ventricle functions in adults [11,12], and there are many studies in children dealing with the effects of ATH on right ventricle; the data about the association of ATH with left ventricle functions in children is limited. The objective of this study was to evaluate the left ventricle (LV) functions in children with ATH, by means of TDI, and to determine the effects of adenotonsillectomy on LV functions by comparing pre- and post-operative data.

2. Methods

The study protocol was approved by the University of Yuzuncu Yil Ethics Committee. Informed consent was obtained from parents of the participants.

Thirty patients (17 male, 13 female, mean age 5.8 ± 3.0 years) with history and physical examination findings suggestive of UAO, who have undergone adenoidectomy/adenotonsillectomy due to stage III or stage IV adenoid and/or ATH were included in the study. In control group, there were 30 healthy children (15 male, 15 female, mean age 5.9 ± 2.1 years) who were admitted to pediatric cardiology outpatient clinic with murmur, syncope, or chest pain and whose echocardiographic evaluations revealed no pathologic findings. The participants were selected consecutively.

A questionnaire was applied to the study group with the purpose of determining whether there were symptoms related to upper respiratory tract obstruction (snoring, respiratory arrest during sleep, open mouth, sleeping, suddenly waking up at night, nocturnal diuresis, mouth open navigation and sleepiness during the day) (Table 3). Parents were asked to answer the questions in questionnaire. Answers such as always, frequently and yes were considered as positive and the ones like sometimes, never, and no were considered as negative responses. Exclusion criteria were as follows: presence of additional pathologies leading to UAO other than ATH, heart disease with congenital origin or secondary to another disease, primary pulmonary hypertension, any systemic diseases, obesity, craniofacial anomaly, and genetic syndrome or inability to obtain informed consent from family. Detailed histories of the patients were obtained and comprehensive physical examination including height, weight, and arterial blood pressure measurements were performed in all participants.

Degree of tonsillar hypertrophy of the study group was determined with paranasal sinus radiographs and clinical findings, according to the Brodsky scale, regarding the degree of oropharyngeal obstruction due to palatine tonsils [13]. Examination of adenoids was performed by fiberoptic endoscopy. According to this evaluation, percentage of obstruction of the passage by adenoids was determined and staging was performed [14]. Conventional Doppler echocardiography (CDE) and TDI values for all patients were recorded. Study group was evaluated by echocardiography in pre-operative period and on 6th month after operation.

2.1. Operative management

Preoperative routine anesthetic evaluations were including blood tests and chest X-ray. All children were operated as inpatient and under general anesthesia. Adenoidectomy in 12 patients and adenotonsillectomy in 18 patients were carried out by curettage and cold dissection methods. Postoperative period care was uneventful in all children. The average period of hospital stay was two days.

2.2. Echocardiography

Echocardiography was performed on the left lateral decubitus and supine positions with an ultrasound machine Vivid 6S (GE-Vingmed Ultrasound AS, Horten, Norway) and 3S-RS (3.5 Mhz) probe. Averages of three consecutive cycles were measured for all echocardiographic data. Images were obtained from parasternal and apical positions using 2D, M-mode and Doppler echocardiographic techniques.

M-mode echocardiographic measurements were performed according to standards outlined by the American Society of Echocardiography [15]. The following variables were measured: inter-ventricular septum diameter (IVSD), left ventricle fractional shorting (LVFS), left ventricle (LV) diameter and LV posterior wall thickness (LVpWD). IVSD and LVpWD were measured at the end of diastole in parasternal long axis. Left ventricular end-diastolic (LVedD) and end-systolic diameter (LVesD) were measured at the end of systole and diastole in parasternal long axis. LVFS was calculated as the percentage decrease of LV systolic diameter to the diastolic diameter.

The pulmonary acceleration time (PAcT), pulmonary peak velocity (PPV) and pulmonary ejection time (PET) were obtained using the pulse wave Doppler with the pulse wave sample volume placed within the RV outflow tract. PET was measured as the time from onset to completion of systolic pulmonary flow. PAcT is the time interval between the onset of the systolic velocity and the peak systolic velocity [16]. Using this measurement, the mean pulmonary artery pressure (mPAP) was calculated by the equation (Mahan formula): $mPAP = 79 - (0.45 \times PAcT)$ [17,18].

TDI was recorded from the apical 4C view with the pulse wave Doppler sample volume placed on the mitral lateral annulus. Peak systolic (Sm) velocity, peak early (Em), the deceleration time (DT) of Em wave and peak late (Am) diastolic myocardial annular velocity, isovolemic relaxation time (IVRT), and isovolemic contraction time (IVCT) were measured. Myocardial performance index (MPI) was calculated with the Tei index formula. Mitral isovolemic acceleration (MIVA) for the LV was calculated by dividing the isovolemic contraction peak velocity by the time interval between the onset of this wave and its peak velocity [19,20] (Fig. 1). Examinations were performed by single experienced pediatric cardiologists.

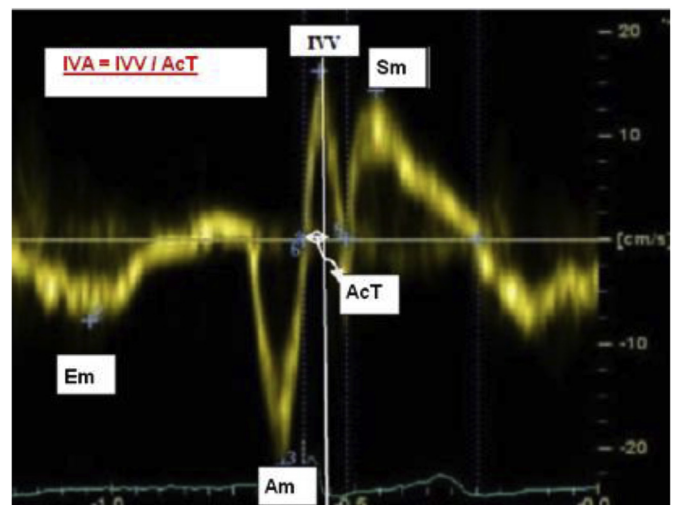


Fig. 1. Calculation of left ventricular MPI and MIVA with TDI. MIVA, mitral isovolemic acceleration; IVV, isovolemic contraction peak velocity; AcT, acceleration time; Sm, systolic myocardial velocity; Em, early diastolic myocardial velocity; Am, late diastolic myocardial velocity; IVRT, isovolemic relaxation time; IVCT, isovolemic contraction time; ET, ejection time; MPI, myocardial performance index.

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