Asymptomatic Cardiopulmonary Changes Caused by Adenoid Hypertrophy

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Abstract: Adenoid hypertrophy is the most common cause of pediatric upper airway obstruction, and it can lead to cardiopulmonary complications such as pulmonary hypertension, cor pulmonale, and even heart failure. The aim of this study was to detect the asymptomatic cardiopulmonary changes that could happen in children with adenoid hypertrophy.

Eighty children with adenoid hypertrophy were included in this study. Chest x-ray was used to assess the cardiothoracic ratio, whereas echocardiography was used for measuring the pulmonary arterial pressures, right ventricular diastolic filling parameters, and right ventricular end-diastolic diameters. All patients underwent adenoidectomy with or without tonsillectomy, and they were subjected again to echocardiographic assessment 6 months after the operation.

No patient showed an increase in the cardiothoracic ratio on x-ray. Preoperative echocardiography showed an increase in pulmonary artery pressure (22.7 [SD, 3.8] mm Hg), a decrease in right ventricular diastolic filling parameters (E/A = 1.03 [SD, 0.17]), and an increase in right ventricular end-diastolic diameters (1.89 [SD, 0.19] cm). Postoperatively, pulmonary artery pressure decreased to 17.2 [SD, 2.1] mm Hg, right ventricular diastolic filling (E/A) increased to 1.25 [SD, 0.11], and right ventricular end-diastolic diameters decreased to 1.68 [SD, 0.12] cm. The comparison between preoperative and postoperative results for each individual parameter was statistically significant.

Clinically asymptomatic cardiopulmonary changes due to adenoid hypertrophy are not rare. Early diagnosis and treatment of upper airway obstruction can prevent these serious complications. Echocardiographic examination should be recommended for these patients as a part of preoperative preparation to avoid anesthetic complications.

Key Words: Adenoid, cardiopulmonary changes, echocardiography, pediatric airway

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Adenoid hypertrophy is the most important cause of upper airway obstruction and obstructive sleep apnea syndrome (OSAS) in children.[1] This syndrome is relatively common in pediatric population because it may affect 1% to 3% of preschool children.[2]

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Obstructive sleep apnea syndrome, if left untreated, can result in serious morbidity from various adverse sequelae that occur because of chronic nocturnal hypoxemia. Cardiovascular complications such as pulmonary hypertension, cor pulmonale, and heart failure were used to be the common presentations of this syndrome in children.[3] However, children who have milder symptoms and signs of cardiovascular sequelae have not been investigated in detail.[4]

Echocardiography can be used to assess pulmonary pressures and cardiac functions in those patients. Adenoid hypertrophy causing upper airway obstruction and cardiac complications can be effectively treated with adenoidectomy operation.[5]

The aim of this study was to detect the asymptomatic cardiopulmonary changes that could be present in children with adenoid hypertrophy.

METHODS

This study was conducted on 80 snorer children evaluated to have adenoid hypertrophy with or without tonsillar hypertrophy. Their ages ranged from 2.5 to 7 years (with a mean of 4 years and 3 months), 46 boys and 34 girls. All cases were collected from the Otolaryngology Clinic of the Pediatric Hospital of Cairo University in the period from July 2006 to May 2009. Informed consents were obtained from the parents of the patients, and the principles outlined in the Declaration of Helsinki were followed.

The following inclusion criteria were adopted:

- Adenoid hypertrophy with or without tonsillar hypertrophy was the only cause of airway obstruction.
- All patients should be free of any craniofacial anomalies, laryngeal diseases, and congenital cardiopulmonary diseases.
- No age or sex was excluded.
- Patients who missed the follow-up were excluded.

All patients were subjected to the following:

1. Preoperative assessment
   - History taking and clinical examination
   - Radiologic examination: including lateral plain x-ray for nasopharynx to assess the size of the adenoid and chest posteroanterior radiographs to assess the cardiothoracic ratio. The cardiothoracic ratio was measured by relating the largest transverse diameter of the heart to the widest internal diameter of the chest. A cardiothoracic ratio of 0.5 or less is considered within normal limits.
   - Echocardiography: using transthoracic echocardiography with 5 MHz pediatric probe of Toshiba Sonolayer SSA-270A cardiac ultrasound (Toshiba, Tokyo, Japan). The children were sedated with chloral hydrate in a dose of 75 to 100 mg/kg when needed. Pulmonary arterial pressures, right ventricular diastolic filling parameters (peak E, peak A, and E/A ratio), and right ventricular end-diastolic diameters were measured.
2. Operative procedure
   - Adenoidectomy (with or without tonsillectomy) was carried out for all patients using conventional curettage technique. Sedative preoperative medications were not used to avoid collapse of the upper airway, and induction of anesthesia was

TABLE 1. Echocardiographic Findings of the Patients (Mean [SD])

Echocardiographic Parameter	Preoperative	Postoperative
Pulmonary artery pressure, mm Hg	22.7 (3.8)	17.2 (2.1)
Right ventricular diastolic filling (E/A)	1.03 (0.17)	1.25 (0.11)
Right ventricular end-diastolic diameters, cm	1.89 (0.19)	1.68 (0.12)

done using halothane in oxygen to prevent sudden carbon dioxide wash and apnea.

3. Postoperative assessment
   - All patients were subjected to echocardiographic examination at 6 months postoperatively.
   - Statistical method: the data were coded and entered using statistical package SPSS version 12 for Windows (SPSS Inc, Chicago, IL). Data were summarized using mean and SD for quantitative variables. Comparison between the preoperative and postoperative echocardiographic changes was done using paired sample *t*-test with a level of significance of $P < 0.05$.

RESULTS

Eighty children with snoring underwent adenoidectomy (with or without tonsillectomy). Regarding the results of chest x-ray, no cases showed enlarged cardiac size, and the cardiothoracic ratio was within normal limit. Regarding the results of echocardiography (Table 1), the following changes were detected:

- Pulmonary artery pressure: the preoperative value was 22.7 (SD, 3.8) mm Hg decreased to 17.2 (SD, 2.1) mm Hg postoperatively.
- Right ventricular diastolic filling parameters: the preoperative value for E/A was 1.03 (SD, 0.17) increased to 1.25 (SD, 0.11) postoperatively.
- Right ventricular end-diastolic diameters: the preoperative value was 1.89 (SD, 0.19) cm decreased to 1.68 (SD, 0.12) cm postoperatively.

The difference between the preoperative and postoperative results of each individual parameter of the echocardiographic changes was statistically significant.

DISCUSSION

It has been known that severe obstructive sleep apnea in children may lead to congestive heart failure and cor pulmonale. In the 1950s, childhood OSAS was diagnosed mainly by cardiologists and endocrinologists when children presented in heart failure or severe growth impairment. Currently, the more relevant question is whether milder forms of childhood sleep disorder breathing are associated with cardiovascular morbidity.[6]

In our study, we evaluated the cardiopulmonary changes in 80 children due to obstructive adenoids. Chest radiography showed no abnormalities, whereas echocardiography showed changes in the pulmonary artery pressure, right ventricular diastolic filling parameters, and right ventricular end-diastolic diameters; this may indicate that the changes are not gross to be detectable radiologically. However, the changes were improved and returned to their normal values after removal of the adenoids indicating that airway obstruction was the cause of these changes.

Khalifa et al[7] and Saito et al[8] stated that enlarged adenoids may be associated with ventilatory impairment that is usually reversible after adenoidectomy.

Luke et al[9] reported that hypoxia has 2 effects on the cardiopulmonary system. First, it leads to pulmonary vasoconstriction, an increase in pulmonary artery pressure, causing right ventricular hypertrophy and right ventricular heart failure; second, it causes pulmonary edema by increasing capillary permeability. Cronje et al[10] claimed that thickening of the muscular layer of the small arteries and pulmonary arterioles may cause pulmonary hypertension.

The symptoms of progressive pulmonary hypertension are minimal until the rapid onset of severe cardiac decompensation occurs.[11] Silent progression of pulmonary vascular disease is a function of the unique physiology of the pulmonary vascular bed and the response of the cardiovascular system, primarily the right ventricle, finally leading to increased pulmonary vascular resistance.[12]

The increase in pulmonary vascular resistance causes an increase in right ventricular work, right ventricular hypertrophy, and even heart failure. However, pulmonary hypertension in these patients may be reversible when the obstruction is relieved.[13]

In our study, the cardiopulmonary changes were not manifested clinically indicating that the patients were in an early stage of this problem. Brown et al[14] reported that the mild form of cor pulmonale due to adenotonsillar enlargement could not be diagnosed clinically, and also, Tal et al[15] concluded that the right ventricular function may be compromised in children with obstructive sleep apnea even before the clinical signs of cardiac involvement are being detectable.

El-Hoshy et al[16] detected 12 of 60 children having pulmonary hypertension and right ventricular hypertrophy with heart failure in 5 of them caused by severe upper airway obstruction and OSAS. After adenotonsillectomy, 11 children were cured completely with return of heart sizes to their normal parameters, but 1 child remained with right ventricular hypertrophy even after return of oxygen saturation to the normal level indicating irreversible cardiopulmonary changes. Gerald and Dungan[17] stated that pulmonary hypertension is usually reversible but the heart may not return to its normal size.

Based on the results of our study, we conclude that cardiopulmonary changes due to adenoid hypertrophy are not rare. Early diagnosis and treatment of upper airway obstruction can prevent these serious complications. In addition, we recommend echocardiographic examination of these patients as a part of preoperative preparation to avoid anesthetic complications.

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