MedCrave
Step into the World of Research

Journal of Pediatrics and Neonatal Care

Echocardiographic Evaluation of Cardiac Functions in Children with Chronic Adenotonsillar Hypertrophy

Abstract

Objective: Chronic adenotonsillar hypertrophy (ATH) causing upper airway obstruction may lead to pulmonary hypertension and other cardiac abnormalities. This study aimed at the clinical and echocardiographic evaluation of cardiac abnormalities in children with chronic ATH and evaluation of these abnormalities after adenotonsillectomy.

Subjects and method: We studied 40 children with ATH indicated for adenotonsillectomy (group I) compared with another 35 sex and age matched normal children (Group II). Both groups were examined by otorhinolaryngologist and adenotonsillar hypertrophy was diagnosed with lateral neck x-ray. Clinical and echocardiographic examinations were compared in both groups. Adenotonsillectomy were performed in group I and reevaluation of clinical and echocardiographic findings were performed 6 months later in this group and results were compared with the preoperative findings in the same group.

Results: Compared with group II, group I has a significantly higher pulmonary artery pressure, right ventricular size, Interventricular septal wall thickness, left ventricular end-diastolic dimension, and longer E-wave deceleration time. Postoperative echocardiographic findings showed statistically significant decrease only in pulmonary artery pressure and E-wave deceleration time. Significant improvement in clinical symptoms and growth parameters were also noted postoperatively in group II.

Conclusion: Upper airway obstruction in children with ATH is associated with cardiac structural and functional abnormalities as demonstrated by echocardiography. Some of these abnormalities i.e. pulmonary hypertension and delayed LV diastolic relaxation are reversible after adenotonsillectomy.

Keywords: Adenotonsillar hypertrophy; Adenotonsillectomy; Echocardiography; Cardiac dysfunction

Research Article

Volume 2 Issue 5 - 2015

Mostafa M El-Nashar[1], Mohamed A Oraby[2] and Osama A Abd El Hamed[3]*

[1]*Department of Oto-rhinolaryngeology, Ain Shams University, Egypt*
[2]*Department of Cardiology, Suez Canal University, Egypt*
[3]*Department of Pediatrics, Port Fouad General Hospital, Egypt*

***Corresponding author:** Osama A Abd El Hamed, Head of Pediatrics department, Port Fouad General Hospital, Egypt, Email: arafaclinic@yahoo.com

Received: July 21, 2014 | **Published:** August 29, 2015

Abbreviations: ATH: Chronic Adenotonsillar Hypertrophy; SDB: Sleep-Disordered Breathing; OSA: Obstructive Sleep Apnea; LVESD: Left Ventricular End Systolic Diameter; LVEDD: Left Ventricular End Diastolic Diameter; FS: Fractional Shortening; EF: Ejection Fraction; IVSD: Interventricular Septum Diastolic Thickness; LA: Left Atrium; RVD: RV Diastolic Diameter; DT: Deceleration Time; TR: Tricuspid Regurgitation

Introduction

Adenoids are located on the posterior nasopharyngeal wall posterior to the nasal cavity. They are a component of the Waldeyer ring of lymphoid tissue in the oropharynx and nasopharynx that consists mainly of the adenoids, the palatine tonsils, and the lingual tonsils [1]. Adenoids are present at birth and then begin to enlarge. They, along with the tonsils, continue to grow until individuals are aged 5-7 years [2].

Adenotonsillar enlargement is the most common cause of pharyngeal airway obstruction in infants and children [3]. Frequent and loud snoring is a very frequent condition in prepubertal children affecting approximately 10% of all 2-8 year old children. Often this is mild and has no sequalae. However when constant and severe obstruction is present, a condition of sleep-disordered breathing (SDB) develops which leads to disturbed sleep, snoring, behavioral abnormalities, and sometimes growth failure. Long standing SDB is known to cause hypoxia, polycythemia, elevation of pulmonary artery pressure and cor-pulmonale [4,5].

Adult patients with obstructive sleep apnea (OSA) have higher morbidity and mortality due to cardiovascular disease compared with the general population [6-12]. Cardiac dysfunction, nocturnal hypertension, and myocardial and cerebral ischemia during obstructive apnea have been documented in adults [13-16]. In pediatric patients, severe adenotonsillar hypertrophy can lead to pulmonary arterial hypertension, cor-pulmonale, and congestive heart failure [13]. However, neither the early stages of the pathophysiological changes that link adenotonsillar hypertrophy to cardiovascular disease nor the long-term impact of the disorder on the cardiovascular system are well understood. So, in the present study, we evaluated cardiac functions in children with chronic ATH mandating adenotonsillectomy, using echocardiographic and clinical evaluation before and after adenotonsillectomy.

Patients and Methods

Two groups were included in this study; group I included children who were admitted to ENT inpatient for adenotonsillectomy because of significant chronic ATH diagnosed by Otorhinolaryngeal examination and confirmed by lateral neck X-ray associated with marked sleep snoring for the last 6 months.

Group II included age and sex matched normal healthy children attending the pediatric outpatient clinic in the same hospital during the same period of time.

Phase (I): Pre-adenotonsillectomy phase

In this phase both groups underwent the following:

i. History taking (from parents) focusing on upper airway obstructive symptoms including snoring, mouth-breathing during sleep, restless sleeping, daytime somnolence, hyponasal voice and, nocturnal enuresis.

ii. Complete clinical examination including: pulse, blood pressure, weight, height and complete cardiac examination.

iii. Routine laboratory studies including complete blood count, Anti-streptolysine O titer, and erythrocyte sedimentation rate.

iv. 12 leads surface electrocardiography.

v. Trans-thoracic echocardiography.

Phase (II): Post-adenotonsillectomy phase

In this phase study group only was subjected to adenotonsillectomy in the same hospital within a week from the initial evaluation and re-evaluated by clinical and echocardiographic examination 6 months latter for comparison with the preoperative findings in the same group.

Echocardiography

Was performed in both groups pre-operatively and only in group I post-operatively. Two-dimensional, two-dimensionally directed M-mode, and color, pulsed and continuous-wave Doppler tracings were recorded using the Sonos 5,500 Ultrasound Imaging System (Agilent, Andover, MA) with 5 MHz phased-array transducer. We measured Left ventricular end systolic and end diastolic diameters (LVESD, LVEDD), fractional shortening (FS), ejection fraction (EF), Interventricular septum diastolic thickness (IVSD), Left atrium diastolic diameter (LA) and RV diastolic diameter (RVD). Doppler parameters included mitral E-wave (E), mitral A-wave (A) and mitral E-wave deceleration time (DT). Pulmonary artery systolic pressure was estimated using the tricuspid regurgitant velocity or the pulmonary acceleration time if no adequate tricuspid regurgitant Doppler signal could be obtained [8,10]. All measurements and estimations were made according to the recommendations of the American Society of Echocardiography [17]. All studies were recorded on VHS videotapes and analyzed by a blinded experienced cardiologist.

All measurements were made under standardized resting conditions with the child in the supine position after a minimum rest period of 5 minutes. Sedation was used only with non-cooperative children. Pre-operative and post-operative echocardiographic findings were compared within group I and pre-operative findings of group I were compared with group II.

Statistical analysis

Collected data was analyzed using the Statistical Package for Social Sciences 15 (SPSS Inc., Chicago, IL, USA).Chi square and the Fisher's exact test were used to test significance of the difference between qualitative data, Student's t test was used to test significance of the difference between quantitative data. Probability value (P) <0.05 was considered statistically significant.

Results

We enrolled 40 (23 males, and 17 females) children in group I with a mean age 6.5+1.76 years. Group II included 35 (19 males, and 16 females) normal healthy children with a mean age 6.1+1.68 years, Growth parameters including weight, height, their percentiles, and body mass index showed statistically significant difference between control group (II) and preoperative state of study group (I) (p-value < 0.05). In post operative state of group (I) these parameters improved significantly and become statistically not different from control group (II) (P-value > 0.05) (Table 1).

Table 1: Anthropometric measures in study group and in control group.

Anthropometric Measures	Study Group (Preoperative) (n=40)	Study Group (Postoperative) (n=40)	Control Group (n=35)
	Mean±SD	Mean±SD	Mean±SD
Weight (kg)	19.7±4.29*	23.7±5.31	25.8±6.23
Weight (Percentile)	23.1±15.74*	40.5±16.71	44.2±17.62
Height (m)	1.1±0.11*	1.2±0.12	1.25±0.13
Height (Percentile)	21.5±18.71*	38.1±16.45	40.3±19.32
Body Mass Index (BMI) (Kg/m2)	15.4±0.78*	15.9±0.56	16.1±0.64

*Statistically significant difference with control group and post operative state.

Table 2 demonstrates the improvement in obstructive symptoms postoperatively compared to the preoperative state (p-value <0.05). The highest improvement rates were noted in hyponasal voice and daytime somnolence (100%). All laboratory findings (red cell count, ESR, ASOT, and CRP) were significantly higher in the preoperative phase of group I compared with group II, and only ESR and CRP showed significant improvement postoperatively that reached normal levels of control group (Table 3).

Mild-moderate functional tricuspid regurgitation (TR) was reported by color Doppler in 32 (80%) children in group I, and in only 19 (54%) children in group II (P< 0.001). In cases with adequate continuous-wave TR Doppler signal, it was used for estimation of pulmonary artery pressure, while in the absence of

this clear signal, pulmonary artery acceleration time (obtained by pulsed-wave Doppler) was used [17].

Comparing pre-operative phase of group I with group II, pulmonary artery systolic pressure was significantly higher in group I (25.15±8.49 and 16.25±5.41 mmHg, P < 0.001). Similarly, RV size as measured by the diastolic diameter was significantly larger in group I (1.7±0.2 and 1.3±0.3 cm, P<0.02). LV size as measured by LVEDD was larger in the group I (3.7±0.6 and 3.2±0.5 cm, P<0.01) and similarly the ventricular septal thickness was higher in this group (7.1±1.2 and 6.2±1.4 cm, p< 0.02). Mitral E-wave deceleration time was longer in group I (175±46 and 128±26 ms, P< 0.001) indicating improper LV diastolic relaxation (Table 4).

Table 2: Clinical findings in study group in both pre and post operative states (n=40).

Clinical Findings		Study Group (Preoperative) (n=40)		Study Group (Postoperative) (n=40)		Success Rate	p-Value
		No.	%	No.	%		
Snoring	Yes	40	100%	3	7.5%	93%	0.001*
	No	0	0%	37	92.5%		
Mouth Breathing during Sleep	Yes	38	95%	8	20%	79%	0.004*
	No	2	5%	32	80%		
Mouth Breathing during Daytime	Yes	30	75%	7	17.5%	77%	0.001*
	No	10	25%	33	82.5%		
Hyponasal Voice	Yes	31	77.5%	0	0%	100%	0.000*
	No	9	22.5%	40	100%		
Restless Sleeping	Yes	27	67.5%	2	5%	93%	0.002*
	No	13	32.5%	38	95%		
Daytime Somnolence	Yes	21	52.5%	0	0%	100%	0.000*
	No	19	47.5%	40	100%		
Nocturnal Enuresis	Yes	17	42.5%	3	7.5%	82%	0.001*
	No	23	57.5%	37	92.5%		

*Statistically significant difference.

Table 3: Laboratory findings in both study group and control group.

Laboratory Findings	Study Group (Pre-operative) (n=40)	Study Group (Postoperative) (n=40)	Control Group (n=35)
	Mean±SD	Mean±SD	Mean±SD
Red cell count (X106/L)	6.2±0.51*	5.2±0.56*	4.6±0.45
A.S.O.T	753.4±64.32*	352.43±54.63*	155.4±43.52
E.S.R	66.4±21.35*	16.4±4.51	15.3±3.42
C.R.P	21.8±3.46*	3.9±2.43	3.1±1.54

*Statistically significant difference with control group.

Table 4: Echocardiographic findings in study group and control group.

Echocardiographic Findings	Study Group (Pre-operative) (n=40)	Study Group (Postoperative) (n=40)	Control Group (n=35)
	Mean±SD	Mean±SD	Mean±SD
Pulmonary Artery Pressure (PAP) (mmHg)	25.15±8.49*#	16.25±5.41	15.21±6.34
Right Ventricle (RV) (cm)	1.7±0.2*	1.6±0.5*	1.3±0.3
Interventricular Septum Thickness (IVS) (mm)	7.1±1.2*	6.5±1.7	6.2±1.4
Posterior Wall Thickness (PWT) (mm)	6.4±1.3	6.2±1.4	6.3±1.1
Left Atrium Diameter (LA) (cm)	2.7±0.3	2.6±0.7	2.6±0.1
Left Ventricular end Diastolic Diameter (LVEDD) (cm)	3.7±0.6*	3.5±0.4*	3.2±0.5
Left Ventricular end Systolic Diameter (LVESD) (cm)	2.4±0.5	2.4±0.1	2.3±0.3
Fractional Shortening (FS) (%)	38±5	37±6	36±4
Early Ventricular filling Velocity (E) (m/s)	1.12±0.17	1.11±0.23	1.1±1.8
Late Ventricular filling Velocity (A) (m/s)	0.64±0.1	0.63±0.32	0.68±0.21
E-wave Deceleration time (DT) (ms)	175±46*#	130±28	128±26
Ejection Fraction (EF) (%)	79.56±6.2	77.31±5.1	76.8±0.81

*Statistically significant difference with control group.

#Statistically significant difference with post-operative state of study group.

In the post-operative state of group I the only improved echocardiographic indices were the pulmonary artery systolic pressure (16.25±5.41j versus 25.15±8.49 mmHg in preoperative state, P< 0.001) (Figure 1) and the mitral E-wave deceleration time (130±28 versus 175±46 ms in the preoperative state, P< 0.0001) (Figure 2) indicating better LV diastolic relaxation. Most upper airway obstruction in an otherwise healthy child is caused by adenotonsillar hypertrophy. And obstructive sleep apnea caused by this abnormality is a definite indication for surgery [18-21]. Prolonged partial airway obstruction during sleep may result in significant hypercapnia and hypoxemia, as well as daytime somnolence, night sweats, irritability, hyperactivity, behavioral problems, personality changes, poor school performance, morning headache, failure to thrive, obesity, and enuresis [19-27]. Children with ATH may also develop cardiopulmonary complications associated with hypercarbia and hypoxemia [26,27].

In the present study, Pre-operative clinical findings showed high prevalence of symptoms of adenotonsillar hypertrophy including snoring (100%), mouth breathing during sleep (95%), mouth breathing during daytime (75%), hyponasal voice (77.5%), restless sleeping (67.5%), daytime somnolence (52.5%) and nocturnal enuresis (42.5%). There were dramatic improvement of these clinical findings postoperatively with complete disappearance of both daytime somnolence and hyponasal voice and overall improvement in symptoms ranging from 77% to 100%.

These findings are consistent with those reported by Miman et al. [28] who studied the improvement in symptom score of 17 children (mean age 41+37 months) with adenotonsillar hypertrophy after adenotonsillectomy. There was a significant improvement in symptoms after adenotonsillectomy as symptoms score dropped from 4.88±1.17 preoperatively to 0.53±0.72 postoperatively ($p < 0.05$). The significant improvement in growth parameters postoperatively reported in our study population is consistent with what has been reported by Selimoğlu et al. [29], who studied 29 prepubertal children with obstructive adenotonsillar hypertrophy and recorded significant improvement in weight and height scores post-adenotonsillectomy.

Results of echocardiographic examinations in our study revealed structural and functional abnormalities of both sides of the heart in patients with ATH. Mild-moderate functional TR was more prevalent in the case group than in the control group. PAP, RV size and Interventricular septal thickness were significantly higher in patients than in the control group and this observation has been reported in many other studies [30,31]. The etiological factors that lead to rise in pulmonary artery pressure from chronic upper airway obstruction are not completely understood.

Hypoxemia and hypercarbia associated with chronic upper airway obstruction are potent mediators of pulmonary vasoconstriction, resulting in rise in PAP, and right ventricular after load, finally causing right ventricular hypertrophy and right ventricular heart failure. (32 –35) Postoperative improvement was observed only in the level of pulmonary artery pressure but not in RV size. This could be explained in the light of the early assessment of cardiac function (6 months postoperatively) that may be enough for drop in pulmonary arterial pressure but not for regression of RV size which may occur later on [32-35].

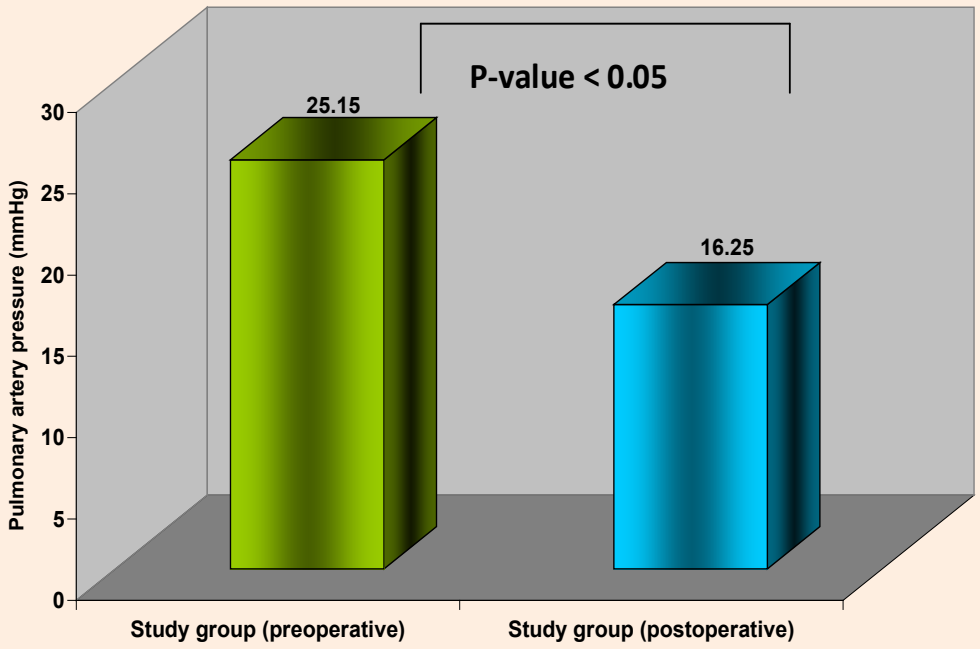

Figure 1: Mean pulmonary artery pressure in both pre and post operative states of study group.

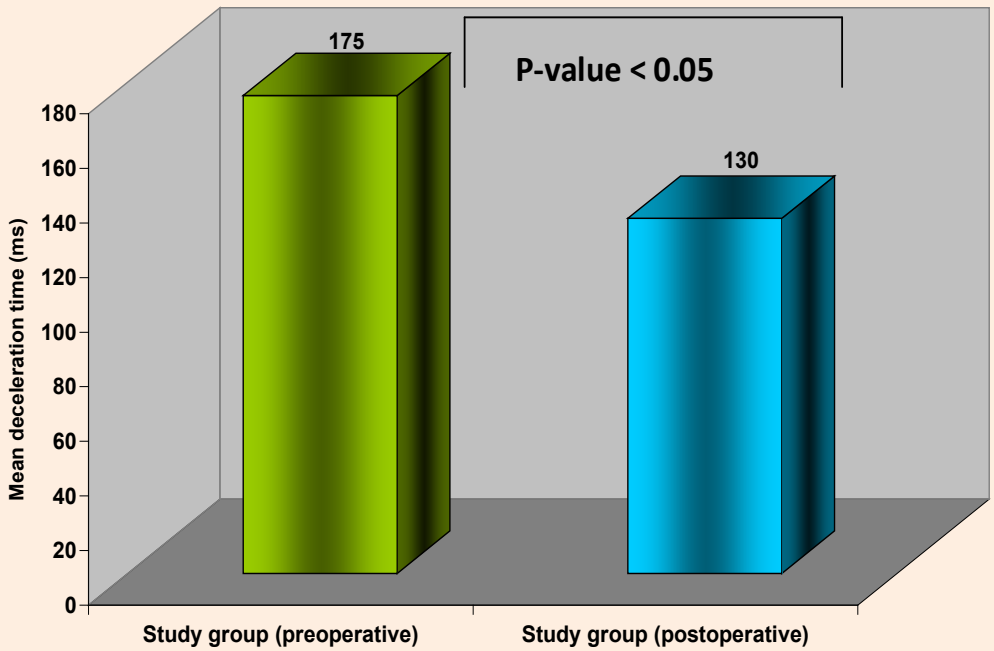

Figure 2: Mean deceleration time in both pre and post operative states of study group.

Left sided echocardiographic abnormalities were also noted in the case group in the form of significantly larger LVEDD and longer E-wave deceleration time. These changes reflect the inherent Interventricular interaction in both systole and diastole. The detected changes in the E-wave deceleration time may be the earliest finding among the measured left ventricular diastolic indices. There is slower drop in the rate of decrease of left ventricular pressure, and the duration of relaxation is prolonged into mid- or even late-diastole. So that, relaxation may continue into mid- or late-diastole, which means less filling of the left ventricle in mid-diastole and prolongation of the deceleration time on the transmitral flow velocity curve. Left ventricular diastolic relaxation as measured by E-wave deceleration time showed significant postoperative improvement. A similar observation has been reported by Görür et al. [36].

Study limitations

The small number of patients included in this study represents a methodological limitation and studying a larger patient population is recommended. Including patients with severe ATH indicated for adenotonsillectomy in this study may hinder the extrapolation of its results to milder forms of ATH. Indirect echocardiographic estimation of PAP using two different methods in this study is another limitation and a more objective and direct measurement of the pressure using invasive catheterization should be studied.

Conclusion

Upper airway obstruction in children with ATH is associated with cardiac structural and functional abnormalities as demonstrated by echocardiography. Some of these cardiac abnormalities i.e. pulmonary hypertension and delayed LV diastolic relaxation are reversible after adenotonsillectomy.

References

1. Richtsmeier WJ, Shikhani AH (1987) The physiology and immunology of the pharyngeal lymphoid tissue. Otolaryngol Clin North Am 20(2): 219-228.
2. Brodsky L, Moore L, Stanievich JF (1987) A comparison of tonsillar size and oropharyngeal dimensions in children with obstructive adenotonsillar hypertrophy. Int J Pediatr Otorhinolaryngol 13(2): 149-156.
3. Uruma Y, Suzuki K, Hattori H, Hattori C, Nishimura T (2003) Obstructive sleep apnea syndrome in children. Acta Otolaryngol Suppl (550): 6-10.
4. Hunt CE, Brouillette RT (2004) Disorders of breathing during sleep. In: Chernick V & Boat TF (Eds.), Kendig's Disorders of Respiratory tract in children. (7th edn), WB Saunders, Philadelphia, USA, pp. 225-250.
5. Leung RS, Bradley TD (2001) Sleep apnea and cardiovascular disease. Am J Respir Crit Care Med 164(12): 2147-2165.
6. McGuire M, Bradford A (1999) Chronic intermittent hypoxia increases hematocrit and causes right ventricular hypertrophy in the rat. Respir Physiol 117(1): 53-58.
7. Brouillette RT, Waters K (1996) Oxygen therapy for pediatric obstructive sleep apnea syndrome: How safe? How effective? Am J Respir Crit Care Med 153(1): 1-2.
8. Sahn DJ, DeMaria A, Kisslo J, Weyman A (1978) Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiographic measurements. Circulation 58(6): 1072-1083.
9. American Thoracic Society (1996) Standards and indications for cardiopulmonary sleep studies in children. Am J Respir Crit Care Med 153(2): 866-878.
10. Schechter MS, the American Academy of Pediatrics, Section on Pediatric Pulmonology, Subcommittee on Obstructive Sleep Apnea Syndrome (2002) Technical report: diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 109(4): e69.
11. Marcus CL, Curtis S, Koerner CB, Joffe A, Serwint JR, et al. (1996) Evaluation of pulmonary function and polysomnography in obese children and adolescents. Pediatr Pulmonol 21(3): 176-183.
12. Nelesen RA, Yu H, Ziegler MG, Mills PJ, Clausen JL, et al. (2001) Continuous positive airway pressure normalizes cardiac autonomic and hemodynamic responses to a laboratory stressor in apneic patients. Chest 119(4): 1092-1101.
13. Berger S (2004) Recent advances in treatment of Pediatric pulmonary arterial hypertension. In "Pediatric Cardiology". The Pediatric Clinics of North America 52(2): 331-44.
14. Leighton SE, Papsin B, Vellodi A, Dinwiddie R, Lane R (2001) Disordered breathing during sleep in patients with mucopolysaccharidoses. Int J Pediatr Otorhinolaryngol 58(2): 127-138.
15. Mitchell RB, Call E, Kelly J (2003) Diagnosis and therapy for airway obstruction in children with Down's syndrome. Arch Otolaryngol Head Neck Surg 129(6): 642-645.
16. Hung J, Whitford EG, Parsons RW, Hillman DR (2003) Association of sleep apnoea with myocardial infarction in men. Lancet 336(8710): 261-274.
17. Lai WW, Geva T, Shirali GS, Frommelt PC, Humes RA, et al. (2006) Guidelines and Standards for Performance of a Pediatric Echocardiogram: A Report from the Task Force of the Pediatric Council of the American Society of Echocardiography. J Am Soc Echocardiogr 19(12): 1413-1430.
18. Dana ST (1995) Clinical indicators compendium. Alexandria, Va, American Academy of Otolaryngology-Head and Neck Surgery, USA.
19. Noel PE, Guarisco JL (1994) Tonsillectomy and adenoidectomy in children: Current indications. J La State Med Soc 146(11): 473-478.
20. Paradise JL (1996) Tonsillectomy and adenoidectomy in children. In: Bluestone CD, Stool SE, Kenna MA, et al. (Eds.), Pediatric Otolaryngology. (3rd edn), WB Saunders, Volume 2, Philadelphia, USA.
21. Tonsils and Adenoids (1994): Guidelines for Parents. Elk Grove Village, Ill, American Academy of Pediatrics, USA.
22. Mauer KW, Staats BA, Olsen KD (1983) Upper airway obstruction and disordered nocturnal breathing in children. Mayo Clin Proc 58(6): 349-353.
23. Potsic WP (1989) Obstructive sleep apnea. Pediatr Clin North Am 36(6): 1435-1442.
24. Potsic WP, Pasquariello PS, Baranak CC, Marsh RR, Miller LM (1986) Relief of upper airway obstruction by adenotonsillectomy. Otolaryngol Head Neck Surg 94(4): 476-480.
25. Rosen CL, D'Andrea L, Haddad GG (1992) Adult criteria for obstructive sleep apnea do not identify children with serious obstruction. Am Rev Respir Dis 146(5 Pt 1): 1231-1234.
26. Guilleminault C, Eldridge FL, Simmons FB, Dement WC (1976) Sleep apnea in eight children. Pediatrics 58(1): 23-30.
27. Guilleminault C, Korobkin R, Winkle R (1981) A review of 50 children with obstructive sleep apnea syndrome. Lung 159(5): 275-287.

Citation: El-Nashar MM, Oraby MA, Hamed OAAE (2015) Echocardiographic Evaluation of Cardiac Functions in Children with Chronic Adenotonsillar Hypertrophy. J Pediatr Neonatal Care 2(5): 00090. DOI: 10.15406/jpnc.2015.02.00090

28. Miman MC, Kirazli T, Ozyurek R (2000) Doppler echocardiography in adenotonsillar hypertrophy. Int J Pediatr Otorhinolaryngol 54(1): 21-26.

29. Selimoğle E, Selimoğle MA, Orbak Z (2003) Does adenotonsillectomy improve growth in children with obstructive adenotonsillar hypertrophy? J Int Med Res 31(2): 84-87.

30. Levy AM, Tabakin BS, Hanson JS, Narkewicz RM (1967) Hypertrophied adenoids causing pulmonary hypertension and severe cardiac failure. N Engl J Med 277(1967) 506-511.

31. Widlitz A, Barst RJ (2003) Pulmonary arterial hypertension in children. Eur Respir J 21(1): 155-176.

32. Brown OE, Manning SC, Ridenour B (1988) Cor pulmonale secondary to tonsillar and adenoidal hypertrophy: management considerations. Int J Pediatr Otorhinolaryngol 16(2): 131-139.

33. Meyrick BO, Perkett EA (1989) The sequence of cellular and hemodynamic changes of chronic pulmonary hypertension induced by hypoxia and other stimuli. Am Rev Resp Dis 140(5): 1486-1489.

34. Menasch V, Farrehi C, Miller M (1965) Hypoventilation and cor pulmonale due to chronic upper airway obstruction. J Pediatr 67(2): 198-203.

35. Luke MJ, Mehrizi A, Fogler GM, Rowe RD (1966) Chronic nasopharyngeal obstruction as a cause of cardiomegaly, cor pulmonale, and pulmonale edema. Pediatrics 37(5): 762-768.

36. Görür K, Doven O, Unal M, Akkuş N, Ozcan C (2001) Preoperative and postoperative cardiac and clinical findings of patients with adenotonsillar hypertrophy. Int J Pediatr Otorhinolaryngol 59(1): 41-46.

Citation: El-Nashar MM, Oraby MA, Hamed OAAE (2015) Echocardiographic Evaluation of Cardiac Functions in Children with Chronic Adenotonsillar Hypertrophy. J Pediatr Neonatal Care 2(5): 00090. DOI: 10.15406/jpnc.2015.02.00090