Original Article

Pulmonary Arterial Hypertension and Body Mass Index in Children with Adeno-Tonsillar Hypertrophy

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Abstract

Background: Low body mass index (BMI) and pulmonary arterial hypertension (PAHT) are common findings among children with adenotonsillar hypertrophy (ATH). There are limited studies about the associations between these comorbidities. **Objective:** To evaluate the association between PAHT and low BMI in children with ATH. **Materials and Methods:** Seventy-nine children with ATH and 79 normal children (control) from both sexes were evaluated. ATH patients were evaluated by ear, nose, and throat surgeons and both ATH and control group were evaluated by pediatric cardiologists. The mean pulmonary artery pressure (MPA pr) was measured non-invasively by an echocardiography machine. **Results:** Children with low BMI (<5%) were significantly more among the ATH group (32.9%) with an odds ratio =3.38 and ($P = 0.01$) and their MPA pr was also significantly higher than the MPA pr of the control group (20.88 ± 7.36 and 16.27 ± 2.4 mm Hg) with $P = 0.04$. Among the ATH group, the MPA pr (24.8 ± 5.99 mm Hg) among the underweight patients was significantly higher than that (19.33 ± 5.92 mm Hg) of non-underweight children ($P = 0.00$) and low BMI cases were more frequent among PAHT group ($P = 0.03$). There is no significant correlation between low BMI and MPA pr. The grade of tonsils and adenoids was not significantly associated neither with the development of PAHT ($P = 0.22$ and 0.3) nor with the development of low BMI. **Conclusion:** Low BMI and PAHT are frequent among ATH patients. Although not correlated to each other, these associated comorbidities need special attention to prevent more serious future outcomes.

Keywords: Adenotonsillar hypertrophy, body mass index, children, pulmonary arterial hypertension

Introduction

The pulmonary arteries deliver the deoxygenated blood from the heart's right ventricle to the lungs for oxygenation.[1] The pulmonary vasculature constricts in response to hypoxia contrary to the systemic circulation, where it dilates to increase oxygen delivery to sustain normal tissue metabolism.[2] Prolonged upper airway obstruction can cause hypoxemia, hypercapnic respiratory acidosis, and pulmonary arterial vasoconstriction, which may cause pulmonary hypertension.[3] Adenotonsillar hypertrophy (ATH) is considered as the most common cause of pediatric upper airway obstruction and is described as an abnormal growth of the pharyngeal tonsil "adenoid" and palatine tonsils due to the hyperplastic process of the lymphoid cells of these tissues.[4-6] This process is enhanced between the age of 1 and 4 years due to the increasing activities of the immune system which can be triggered by different factors such as recurrent bacterial infections and passive cigarette smoking.[7] ATH is considered as one of the most important causes of failure to thrive due to low caloric intake resulting from difficulty eating a normal diet owing to obstruction caused by tonsils and adenoid, increased energy required to breathe against obstruction all night and, exercising to breathe, hypoxemia and its interference with the normal release of endocrine growth factors.[8,9] Decreased airflow due to ATH and the resultant hypoxemia will cause a

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reduction of growth hormone release.[9] This anabolic hormone stimulates growth either directly by binding to receptors within the cell membrane and stimulating the proliferation of the target cells or indirectly through synthesizing insulin-like growth factor 1 in the liver which stimulates protein synthesis, leading to bone and muscle growth.[10,11] Previous studies have evaluated ATH and PAHT associations with reported prevalence of PAHT in the studied samples of 21.9%-42.3%, or they studied low BMI among ATH patients and found a prevalence of around 24%, yet no one evaluated all of these comorbid disorders together.[8,9,11-17]

We are aiming in this study to investigate the associations between PAHT, low BMI, and ATH.

Materials and Methods

This cross-sectional analytical study was conducted in the researchers' hospitals in an outpatient setting over 1 year starting from March 12021 to February 30, 2022. The sample size (158) was calculated with a confidence level of 85% and a margin of error of 5.7% for a target population of 14,00,000 children according to the government's statistics. Written informed consents from the parents of children who were aged 2-15 years were collected for the participation in the study of non-invasive evaluation by an authorized pediatric cardiologist using a Phillips echocardiography machine Affinity 30 (Philips Ultrasound, Inc.) and age-compatible cardiac probes. Body mass index (BMI) was calculated as kg/m^2. We included children in the age range of 24-180 months in our study. Subjects were divided into two groups, the ATH group (n = 79) and the non-ATH as control group (n = 79). Every other child (to ensure randomization) with ATH who visited the ear, nose, and throat (ENT) outpatient unit was included and evaluated by the ENT surgeons who also graded their adenoid size endoscopically into four grades and their tonsillar size also into four grades depending on the Brodsky grading scale.[18,19] Involved children were then sent for evaluation for their growth parameters (weight, height, and BMI) and MPA pr by the pediatric cardiologist who was blinded for the above grades. Weight and height were measured using UNICEF-provided balance and stadiometer and their data were evaluated using the Center for Disease Control and Prevention (CDC) reference data, charts, and calculator. Patients were evaluated in the supine position using the standard echocardiographic views, tricuspid valve (TV) regurgitation and pulmonary valve regurgitation velocities were measured in the apical 4 chamber and short axis parasternal views, respectively, to get the highest readings.

(Estimated Pulmonary Artery Systolic Pressure (EPASP) was calculated using the formula:

EPASP = [(Peak TV regurgitation velocity)^2 × 4] + right atrial pressure*.

* Right atrial pressure is considered to be equal to 5-10 mmHg

MPAP was calculated using the following formulas:

MPAP = 0.61 × ESPAP + 2 mmHg or MPAP = peak pulmonary valve regurgitation pressure gradient). Whichever higher is recorded. Patients with MPA pr ≥ 25 mmHg were considered to have PAHT.[20,21]

Normal children (control) were every other one of those sent for echocardiographic evaluation because of cardiac murmur or chest discomfort and were found to be normal.

The exclusion was done for children with either tonsillar or adenoid hypertrophy alone. Children with any disease that may increase the PA pr and/or decrease the BMI are also excluded from the study such as craniofacial abnormalities, any cause of upper airway obstruction, BMI > 95th centile, congenital heart diseases, respiratory disease, neuromuscular disease, chronic renal disease, gastrointestinal disease, Down syndrome, etc.

Microsoft Excel 2020 and SPSS (Statistical Package of Social Sciences) 26 software packages were used for statistical analysis. Two samples *T*-test was used to compare means. Pearson's correlation coefficient was used to study correlations, and linear regression was used to study relationships. Statistical significance was set at a *P* value of <0.05.

Ethical approval

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. It was carried out with patients verbal and analytical approval before sample was taken. The study protocol and the subject information and consent form were reviewed and approved by a local ethics committee according to the document number 668 on January 18, 2021.

Results

The mean age of the ATH group ± SD was 81.9 ± 32.7 months (95% CI = 74.7-89.1 months) whereas the control (normal) group had a mean age ± SD of 90.5 ± 38.81 months (95% CI = 81.9-99.1 months). There was no significant difference between the two groups regarding their mean age as well as their male/female gender ratio (1.13 and 1.32) (P = 0.63, 0.29, respectively). Out of the 79 ATH patients, 22 (27.2%) had PAHT whereas the rest (72.8%) had normal MPA pr. None of the normal children were labeled with PAHT. From another aspect, the MPA pr was significantly higher (P = 0.04) among the ATH group compared to the normal group (20.88 ± 7.36 and 16.27 ± 2.4 mm Hg). There was a significant association between ATH and low BMI (defined as BMI < 5% on the BMI WHO centile growth chart). The number of low BMI cases was significantly

Table 1: Gender, age, BMI, and MPA pr characteristics of ATH patients compared to normal children

		ATH (n = 79)	Normal (n = 79)	P value
Gender	Male	42	45	0.63
	Female	37	34	
	Mean age ± SD (months)	81.9 ± 32.7	90.5 ± 38.81	0.29
BMI centile line	<5%	26 (32.9%)	10 (12.6%)	0.01 *
	5≤	53 (66.1%)	69 (87.4)	
	Mean PA pr ± SD	20.88 ± 7.36	16.27 ± 2.4	0.04 *

There was no significant age or gender difference between both groups, but low BMI cases were significantly higher among the ATH group, meanwhile, the MPA pr is significantly higher in this group compared to normal children

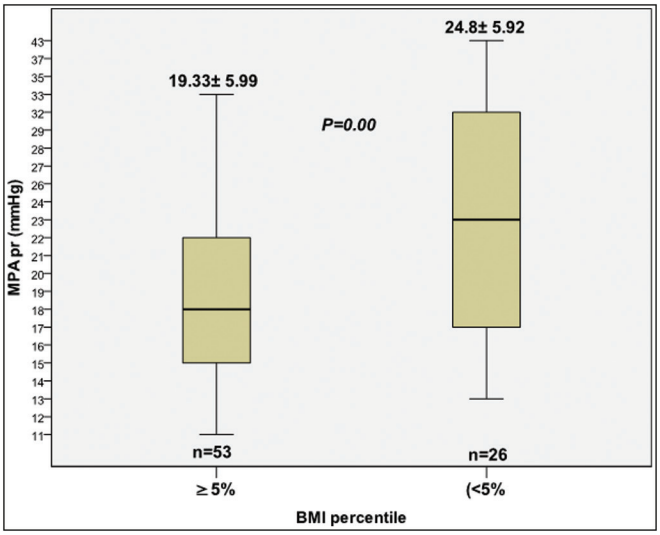

Figure 1: Mean pulmonary artery pressure (mmHg) among ATH patients. The MPA pr is significantly higher in the low body mass index (<5% or underweight) group compared to the non-underweight group (≥5%)

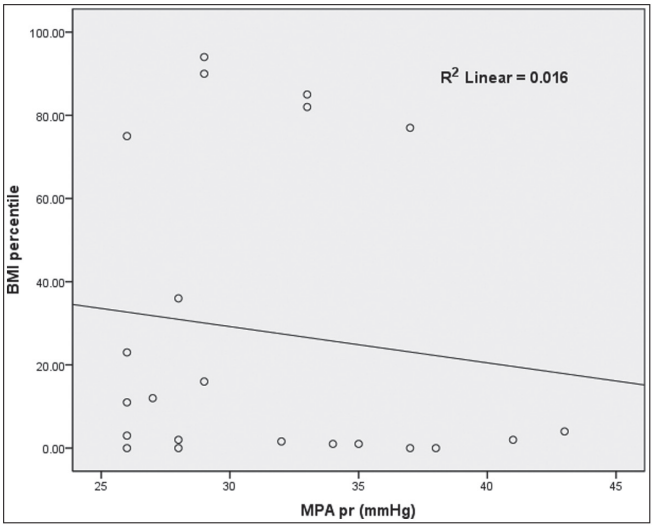

Figure 2: In person's correlation, there was no significant correlation between MPA pr and BMI percentiles (P = 0.46) among ATH who had PAHT, and linear regression for these variables showed a very weak inverse relationship

higher among the ATH group (P = 0.01) with an odds ratio of: 3.38, with a high significance level (P = 0.003) [Table 1].

In a subgroup analysis of the ATH group, as shown in Figure 1, the MPA pr (of 24.8 ± 5.99 mm Hg) among the underweight patients (BMI < 5%) was significantly higher than that (of 19.33 ± 5.92 mm Hg) of non-underweight children (BMI ≥ 5%) with P = 0.00. In Spearman correlation, we could not find a significant correlation between the MPA pr and BMI percentiles among this group. Linear regression graph revealed a very weak inverse relationship between these two variables [Figure 2].

In Table 2, the association between PAHT (defined as MPA pr ≥ 25 mm Hg), low BMI (<5%), clinical grading of tonsillar size, and endoscopic grading of adenoid size among the ATH group was analyzed. Low BMI cases were significantly higher among patients with PAHT (P = 0.03).

Neither the tonsillar size nor the adenoid size (from grade I to grade IV for both) showed significant association with the PAHT subgroups (P = 0.22 and 0.3). Further analysis of BMI percentiles with the different tonsillar and adenoid grades also could not find significant associations or correlations between these variables.

Discussion

Chronic airway obstruction in general and due to ATH, in particular, can result in serious consequences in terms of cellular hypoxia, increased MPA pr with subsequent reduction in the patient's appetite, disturbed body cells metabolism, and slowing of body growth and all of these changes can improve after adenotonsillectomy.[2,3,7] Low BMI is a frequent observation among ATH patients and at least to the best of our knowledge, there are no studies that evaluated the associations and correlations between MPA pr and low BMI among ATH patients.

It is known that PAHT may be asymptomatic in the beginning but it antecedes the serious outcome of *core pulmonale* making its early discovery a crucial step in the risky subjects. It is widely accepted and has been approved by many former studies that PAHT is highly prevalent

Table 2: Association of PAHT with Low BMI, clinical assessment of the tonsillar size and endoscopic assessment of adenoid size among ATH patients

		PAHT		*P*-value
		No (n = 57)	Yes (n = 22)	
BMI	<5% (n = 26)	15 (26%)	11 (50%)	0.03
	≤5% (n = 53)	42 (74%)	11 (50%)	
Clinical assessment of tonsillar size Brodsky grading scale (0, 1, 2, 3, 4)	I	2	1	0.22
	II	15	4	
	III	33	10	
	IV	7	7	
Endoscopic assessment of adenoid size Grade (1, 2, 3, 4)	I	4	0	0.3
	II	15	8	
	III	29	8	
	IV	9	6	

A significant relationship is found between PAHT and low MBI (<5%), whereas other variables did not show a significant association

among children with tonsillar, adenoid, or Adenotonsillar hypertrophy. In our study, 27.2% of ATH patients got PAHT, this figure is close to the results of the meta-analysis of Kong *et al*.[12] which was 35%. Our prevalence of PAHT was within the range of other researchers among their studied samples ranging from 21.9% to 42.3%.[12-17] In our study, the MPA pr ± SD among ATH patients in general (20.88 ± 7.36 mmHg) and among the hypertensive subgroup (24.8 ± 5.92 mmHg) were also comparable to the previous studies and similarly, both means were significantly higher than their control group.[13-17]

Being underweight is a common observation among ATH patients which has been confirmed by other studies, but the relationship between PAHT and being underweight (indicated by low BMI) was not studied. Fortunately, both PAH and low BMI in patients with ATH are reversible and can be reverted to normal after adenotonsillectomy.[3,8,9,11,14,17,22,23] In our study 32.9 % of ATH in general and 50% of the pulmonary hypertensive ATH patients were found to have low BMI, both were significantly higher than their control group. In Kang *et al*.[17] study, the prevalence of underweight among ATH patients was 26.4%, which was close to ours.

We could not find similar studies to compare our results about the association and correlation of low body BMI with PAHT among ATH patients. Our results confirm a significant association between these two variables among ATH patients; moreover, the MPA pr among the low BMI group was significantly higher than the normal BMI group among the ATH patients. Although there was a very weak inverse relationship between BMI percentiles and MPA pr, the correlation between these variables was not significant probably because of the size of the studied sample, and might be found to be positive in future studies. Like the results of some previous studies tonsillar or adenoid size did not show a significant association with the development of PAHT among ATH patients.[14]

Conclusion

Among ATH patients, the MPA pr is higher and PAHT is more frequent, and low BMI is more frequent too. There is an increased frequency of low BMI in patients with ATH and PAHT and their MPA pr is significantly higher compared to children with ATH and normal BMI. The MPA pr is not correlated with the BMI percentiles despite the very weak inverse relationship between them. Tonsillar and adenoid grades are not significantly associated with low BMI or the risk to develop PAHT. So finally, we can posit PAHT and Low BMI are frequent comorbidities associated with ATH, the presence of either one anticipates the other in such patients. Special attention should be given to those patients because they are at risk of the serious sequelae of PAHT.

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Author's contribution

All authors included in this article authors equally contributed to all steps of this work.

This manuscript has been read and approved by all the authors, the requirements for authorship have been met, and each author believes that this the manuscript represents honest work.

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Conflicts of interest

There are no conflicts of interest.

References

1. Kandathil A, Chamarthy M. Pulmonary vascular anatomy & anatomical variants. Cardiovasc Diagn Ther. 2018;8:201-7.

2. Shimoda LA. 55th Bowditch lecture: Effects of chronic hypoxia on the pulmonary circulation: Role of HIF-1. J Appl Physiol (1985) 2012;113:1343-52.
3. Kareem JM, Al-Waely NK, Al-Hashimi AF. Diaphragmatic thickness in chronic obstructive pulmonary disease. Med J Babylon 2022;19:427-33.
4. Jazi SM, Barati B, Kheradmand A. Treatment of adenotonsillar hypertrophy: A prospective randomized trial comparing azithromycin vs. fluticasone. J Res Med Sci 2011;16:1590-7.
5. Góis CRT, D'Ávila JS, Cipolotti R, Lira ADS, Silva ALL. Adenotonsillar hypertrophy in pre-school children with sickle cell disease and diagnostic accuracy of the sleep disturbance scale for children. Int Arch Otorhinolaryngol 2018;22:55-9.
6. Niedzielska G, Kotowski M, Niedzielski A. Assessment of pulmonary function and nasal flow in children with adenoid hypertrophy. Int J Pediatr Otorhinolaryngol 2008;72:333-5.
7. Samareh Fekri M, Arabi Mianroodi A, Shakeri H, Khanjani N. Effects of tonsil size on pulmonary function test results after tonsillectomy in children. Iran J Otorhinolaryngol 2016;28:61-6.
8. Cassano M, Russo G, Granieri C, Ciavarella D. Modification of growth, immunologic and feeding parameters in children with OSAS after adenotonsillectomy. Acta Otorhinolaryngol Ital 2018;38:124-30.
9. Farmarzi M, Shishegar M, Heydari ST, Haghighi A, Sharouny H. Effects of adenotonsillectomy on serum levels of IGF-1 and IGFBP-3 and growth indices in children with adenotonsillar hypertrophy or recurrent tonsillitis. Iran J Otorhinolaryngol 2016;28:329-35.
10. Chung JY, Sunwoo JS, Kim MW, Kim M. The neuroprotective effects of human growth hormone as a potential treatment for amyotrophic lateral sclerosis. Neural Regen Res 2015;10:1201-3.
11. Kiris M, Muderris T, Celebi S, Cankaya H, Bercin S. Changes in serum IGF-1 and IGFBP-3 levels and growth in children following adenoidectomy, tonsillectomy or adenotonsillectomy. Int J Pediatr Otorhinolaryngol 2010;74:528-31.
12. Kong F, Sun YL, Yuan B. Prevalence of pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy: A meta-analysis. Int J Pediatr Otorhinolaryngol 2022;153:111019.
13. Marangu D, Jowi C, Aswani J, Wambani S, Nduati R. Prevalence and associated factors of pulmonary hypertension in Kenyan children with adenoid or adenotonsillar hypertrophy. Int J Pediatr Otorhinolaryngol 2014;78:1381-6.
14. Martha VF, Moreira Jda S, Martha AS, Velho FJ, Eick RG, Goncalves SC. Reversal of pulmonary hypertension in children after adenoidectomy or adenotonsillectomy. Int J Pediatr Otorhinolaryngol 2013;77:237-40.
15. Naiboglu B, Deveci S, Duman D, Kaya KS, Toros S, Kinis V, *et al*. Effect of upper airway obstruction on pulmonary arterial pressure in children. Int J Pediatr Otorhinolaryngol 2008;72:1425-9.
16. Orji FT, Adiele DK, Umedum NG, Akpeh JO, Ofoegbu VC, Nwosu JN. The clinical and radiological predictors of pulmonary hypertension in children with adenotonsillar hypertrophy. Eur Arch Otorhinolaryngol 2017;274:1237-43.
17. Kang KT, Lee PL, Weng WC, Hsu WC. Body weight status and obstructive sleep apnea in children. Int J Obes (Lond) 2012;36:920-4.
18. Feres MF, Hermann JS, Sallum AC, Pignatari SS. Endoscopic evaluation of adenoids: Reproducibility analysis of current methods. Clin Exp Otorhinolaryngol 2013;6:36-40.
19. Ng SK, Lee DL, Li AM, Wing YK, Tong MC. Reproducibility of clinical grading of tonsillar size. Arch Otolaryngol Head Neck Surg 2010;136:159-62.
20. Budev MM, Arroliga AC, Jennings CA. Diagnosis and evaluation of pulmonary hypertension. Cleve Clin J Med 2003;70:9-17.
21. Roushdy AM, Ragab I, Abd El Raouf W. Noninvasive assessment of elevated pulmonary vascular resistance in children with pulmonary hypertension secondary to congenital heart disease: A comparative study between five different Doppler indices. J Saudi Heart Assoc 2012;24:233-41.
22. Mohammed AM, Al-Rawi RA, Abdulmajeed BY, Ayoub NI. The relationship between blood pressure and body mass index among primary-school children. Med J Babylon 2022;19:482-7.
23. Al-Murib AHA, Al-Jumaily HS, Falah Z, Al-Saad SF. Left Atrial Strain by 2D Speckle Tracking Echocardiography in Patients with Systemic Hypertension in Babylon Province. Med J Babylon 2024;21:485-90.