Adenotonsillar Hypertrophy and Cardiopulmonary Status: A Correlative Study

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Abstract

Introduction
Adenotonsillar hyperplasia in childhood is a common phenomenon. It has been reported in the literature that increased upper-airway resistance resulting from hypertrophied tonsils and adenoids can cause intermittent airway obstruction, chronic alveolar hypoventilation, and even lead to severe cardiopulmonary complications such as right ventricular (RV) failure and cor-pulmonale, a near-lethal culmination of pulmonary artery hypertension (PAH). This study was undertaken to explore and examine the association of adenotonsillar hypertrophy and cardiopulmonary status in patients with complaints of upper airway obstruction below the age of 20 years and to analyze the effect of adenotonsillar enlargement on pulmonary function tests and cardiac aberration reflected in the electrocardiography (ECG) changes.

Methods
This study included patients visiting or admitted to the Otolaryngology/Ear, Nose, and Throat (ENT) and Paediatrics department of Acharya Vinoba Bhave Hospital, Sawangi (Meghe), Wardha, Maharashtra, India. It is an Observational Prospective Study conducted on 75 Patients (50 cases and 25 controls) below the age of 20 years. Inclusion criteria for cases included all patients of adenoid hypertrophy (AH) and adenotonsillar hypertrophy (ATH). Inclusion criteria for controls comprised all patients with a history and clinical examination not suggestive of any upper airway and pulmonary disease. All the patients were subjected to pulmonary function tests (PFT) and electrocardiography (ECG), and the values were compared.

Results
It was found that there was a decrease in the value of the parameters of the pulmonary function tests (PFT), which includes forced expiratory volume in the first second (FEV1), forced vital capacity (FVC), the ratio of the forced expiratory volume in the first one second to the forced vital capacity of the lungs (FEV1/FVC) and peak expiratory flow rate (PEFR) with increasing severity of the endoscopic grades of adenoid hypertrophy. This was found to be statistically significant. All the PFT parameters were significantly lower in the subset of patients with ATH compared to those with AH only, emphasizing the compounding effect of tonsillar volume. Between case and control subjects also, these differences were statistically significant. Seventeen (34%) out of the 50 patients studied in the present work were found to have abnormalities in their ECG, while no subject in the control group had any departure from normal. Nine of these 17 patients had AH, and eight had the adenotonsillar disease. In the 9 patients (18%) with AH, sinus arrhythmia was seen in 5 (10%), sinus tachycardia in 3 (6%), and Mobitz type 1 block in 1 (2%) patients. In 8 patients (16%) with AH, sinus arrhythmia was seen in 4 (8%), sinus tachycardia in 3 (6%), and Mobitz type 1 block in 1 (2%) patients. Overall, sinus arrhythmia was the commonest finding seen in 9 patients (18%).

Conclusion
Chronic obstructive adenotonsillar hypertrophy causes significant cardiovascular and pulmonary changes, which is often overlooked in the clinical setting. Symptoms of progressive pulmonary hypertension are minimal until the rapid onset of severe cardiac decompensation occurs. These entire cascades of events are reversible in the early stages and thus mandate early detection and treatment. Performing PFT and ECG in children with adenotonsillar disease is not mandatory but profitable even in the absence of obvious symptoms of upper airway obstruction.
Hypertrophy, ventricular hypertrophy, and axis deviation. The ECG was read by a single experienced
physician using the BPL AR 2100 (Royal Health Care, Mumbai, India) machine. The parameters measured on the ECG
in this study were heart rate, rhythm, characteristics of P, QRS, and T waves, and the presence of evidence of atrial
hypertrophy, ventricular hypertrophy, and axis deviation.

Twelve-lead ECG was performed on all the patients diagnosed as having adenoid/adenotonsillar hypertrophy
until three closely matching forced expirations and inspirations were recorded.

Spirometric measurements were carried out on an RMS Helios 401 (Recorders & Medicare Systems Pvt. Ltd,
India), a spirometer, in a sitting position with the nose clipped. The patient was asked to make a series of
forced expirations from the position of full inspiration into the mouthpiece of the spirometric system and a series of
forced inspirations starting from the position of full expiration. These maneuvers were repeated
until three closely matching forced expirations and inspirations were recorded.

Twelve-lead ECG was performed on all the patients diagnosed as having adenoid/adenotonsillar hypertrophy
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included the heart rate, rhythm, characteristics of P, QRS, and T waves, and the presence of evidence of atrial
hypertrophy, ventricular hypertrophy, and axis deviation. The ECG was read by a single experienced
cardiologist. The findings were recorded under two headings - significant findings or normal ECG.

Materials And Methods
This study was conducted on patients visiting the outpatient department or admitted in the wards of the
department of Otolaryngology/Ear, Nose and Throat (ENT) and Paediatrics of Acharya Vinoba Bhave
Hospital, Sawangi (Meghe), Wardha, Maharashtra State, India. It is an observational prospective study
conducted on 75 patients (50 cases and 25 controls) below the age of 20 years. Inclusion criteria for cases
included all patients of adenoid hypertrophy (AH) and adenotonsillar hypertrophy (ATH) up to 20 years of age.
Inclusion criteria for controls comprised all patients with a history and clinical examination not
suggestive of any upper airway and pulmonary disease. Exclusion criteria for cases comprised of all patients
of AH and ATH above 20 years of age, adenotonsillar hypertrophy with acute exacerbation, isolated tonsillar
hypertrophy, patients with cardiopulmonary diseases, patients with nasal polyps, deviated nasal septum or
any structural abnormality of the thoracic cage affecting pulmonary function, primary pulmonary
hypertension, systemic diseases, obesity, craniofacial anomaly, and genetic syndrome and patient
not consenting. Exclusion criteria for controls included all patients with any cardiopulmonary diseases.

The study duration was from September 2015 to September 2017. Fifty patients less than or equal to 20 years
of age with signs and symptoms suggestive of adenoid or adenotonsillar hypertrophy were selected for the
study. After a thorough and careful ENT examination, patients were subjected to endoscopic assessment,
and grading of adenoid and adenotonsillar hypertrophy was done. Photographic documentation of various
modalities of assessment of adenoid size was also done. Once the diagnosis was established, patients were
further subjected to two major investigations: Pulmonary function test/spirometry and electrocardiography
(EGC). ECG was done per 25 patients less than or equal to 20 years of age fitting within the above-mentioned
selection criteria and was accrued as controls for the study. All the patients were subjected to pulmonary
function test/spirometry and ECG.

Ethical declaration
All procedures performed in this study involving human participants were in accordance with the ethical
standards of the internal Institutional Ethics Committee, Jawaharlal Nehru Medical College and Acharya
Vinoba Bhave Rural Hospital, Sawangi (Meghe), Wardha, Maharashtra, India (established under section three
of the UGC Act vide Notification Number F-9-48/2004 - U3 Govt of India), with reference number
DMIMS(DU)/IEC/2015-16/1636 approved on 26th of September, 2015. The approval has been granted on the
assumption that the proposed work will be carried out in accordance with the ethical guidelines prescribed
by Central Ethics Committee on Human Research (CECHR).

Pulmonary function test/Spirometry
Spirometric measurements were carried out on an RMS Helios 401 (Recorders & Medicare Systems Pvt. Ltd,
India), a spirometer, in a sitting position with the nose clipped. The patient was asked to make a series of
forced expirations from the position of full inspiration into the mouthpiece of the spirometric system and a series of
forced inspirations starting from the position of full expiration. These maneuvers were repeated
until three closely matching forced expirations and inspirations were recorded.

Electrocardiogram
Twelve-lead ECG was performed on all the patients diagnosed as having adenoid/adenotonsillar hypertrophy
using the BPL AR 2100 (Royal Health Care, Mumbai, India) machine. The parameters measured on the ECG
included the heart rate, rhythm, characteristics of P, QRS, and T waves, and the presence of evidence of atrial
hypertrophy, ventricular hypertrophy, and axis deviation. The ECG was read by a single experienced
cardiologist. The findings were recorded under two headings - significant findings or normal ECG.
Observations
Data gathered was entered in the proforma meant for the study and was statistically analyzed.

Informed Consent
The procedure was explained in detail to the parents, and written informed consent was obtained before enrollment.

Clinical Grading of Tonsils
All 50 patients were clinically examined for the presence of significant tonsillar hypertrophy, i.e., hypertrophy >2+, as per the assessment scale described by Brodsky (Table 1) [5].

| Grade | Features                                                                 |
|-------|--------------------------------------------------------------------------|
| +0    | Tonsils are situated in the tonsillar fossa                              |
| +1    | Tonsils sit just outside of the tonsillar fossa with obstruction of less than 25 percent of the airway |
| +2    | Tonsils are readily seen in the airway: 25 to 50 percent of the airway is obstructed |
| +3    | Tonsils causing 50 to 75 percent obstruction of the airway               |
| +4    | Tonsils involve a greater than 75 percent obstruction of the airway       |

TABLE 1: Clinical grading of tonsils

Endoscopic Grading of Adenoids
All the patients were subjected to post nasal space examination using nasopharyngeal examination by the rigid endoscope, Karl Storz zero-degree nasal endoscope (2.7 mm), made of stainless steel body was used. Endoscopy was done after treating the nose with 4% lidocaine mixed with adrenaline in both nostrils. The posterior end of the middle turbinate was considered a fixed anatomical landmark for viewing posterior choanae and adenoid hypertrophy. Adenoids were graded based on the classification given by Cassano as follows (Table 2) [6].

| Grade | Features                                                                 |
|-------|--------------------------------------------------------------------------|
| 1     | Choanal adenoid, occupying upper segment of nasopharynx (<25% of the choana) |
| 2     | Adenoid tissue occupying upper half of nasopharynx (25% to < 50%)         |
| 3     | Adenoids extending over the nasopharynx obstructing the choana and partially the tube (50% to <75%) |
| 4     | Total choanal obstruction (≥75%)                                         |

TABLE 2: Endoscopic grading of adenoids

Statistical analysis
Statistical analysis was done by using descriptive and inferential statistics using the chi-square test, student’s unpaired t-test, regression analysis, one-way analysis of variance (ANOVA), and Kappa Statistics and software used in the analysis were IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp. and GraphPad Prism 7.0 version and probability (p-value) were calculated.

Results
A total of 75 patients were enrolled in the study comprising 50 cases and 25 controls. Maximum patients with only adenoid hypertrophy (AH) as well as with adenotonsillar hypertrophy (ATH) were between the age group of 6 and 10 years (18, 36% (AH) and 13, 26% (ATH), respectively). The mean age of the patients was 10.33±3.63 years, while in the control group, it was 9.88±2.20 years. The mean age of patients with AH was 10.33±3.63 years, and with ATH was 9.50±1.98 years (Table 3).
### Table 3: Distribution of patients according to age

| Age Group(years) | Case Group | Control Group |
|------------------|------------|---------------|
|                  | AH         | ATH           |
| 0-5 years        | 2(4%)      | 0(0%)         | 0(0%)         |
| 6-10 years       | 18(36%)    | 13(26%)       | 15(80%)       |
| 11-15 years      | 8(16%)     | 7(14%)        | 10(40%)       |
| 16-20 years      | 2(4%)      | 0(0%)         | 0(0%)         |
| Total            | 30(60%)    | 20(40%)       | 25(100%)      |

#### Table

**AH**: Adenoid hypertrophy, **ATH**: Adenotonsillar hypertrophy

Males (28, 56%) marginally outnumbered females (22, 44%) with a male-to-female ratio (M:F ratio) of 1.27:1. In AH only patients, males and females were equal in number (15 each, 30%) with an M:F ratio of 1:1 while in patients of ATH males (13, 26%) were almost twice as females (7, 14%), the M:F ratio being 1.85:1. Table 4 shows the gender-wise distribution of patients.

### Table 4: Distribution of patients according to gender

| Gender  | Case Group | Control Group |
|---------|------------|---------------|
|         | AH         | ATH           |
| Male    | 15(30%)    | 13(26%)       | 18(72%)       |
| Female  | 15(30%)    | 7(14%)        | 7(28%)        |
| Total   | 30(60%)    | 20(40%)       | 25(100%)      |

**AH**: Adenoid hypertrophy, **ATH**: Adenotonsillar hypertrophy

The patients were also analyzed based on their geographical background and observed that the maximum number of patients (31, 62%) belonged to the urban area while 19 (38%) hailed from the rural area (Table 5) and out of a total of 50 patients studied, 30 (60%) had AH, and 20 (40%) had ATH (Table 6).

### Table 5: Geographic distribution of patients

| Area                  | Total (n=50) |
|-----------------------|--------------|
|                       | Rural | Urban |
| Adenoid Hypertrophy   | 13(26%) | 17(34%) | 30(60%) |
| Adenotonsillar Hypertrophy | 6(12%) | 14(28%) | 20(40%) |
| Total (n=50)          | 19(38%) | 31(62%) | 50(100%) |
The presenting symptoms of the patients with their frequency are displayed in Table 7. In patients with adenoid hypertrophy, the commonest presenting symptom was mouth breathing (23, 46%) followed by snoring (14, 28%) and earache (10, 20%). In patients with adenotonsillar hypertrophy, the commonest presenting symptoms were mouth breathing and snoring (17, 34% each), followed by obstructive symptoms causing disturbed sleep at night (6, 12%). Overall, mouth breathing (40, 80%), snoring (31, 62%), and earache (11, 22%) were the common manifestations.

Nasal endoscopy to grade adenoids was carried out, as per the guidelines given by Cassano [6]. Adenoid enlargement was graded depending on the percentage of choanal obstruction with the nasal endoscope (0 degrees, 2.7mm) placed at the posterior end of the middle turbinate. Overall, most patients had grade 3 AH (26, 52%). In patients with only AH, a maximum number of patients were observed in grade 4 (13, 26%), followed by grade 3 (11, 22%). While in patients with ATH, grade 3 enlargement (15, 30%) was most commonly observed. This is shown in Table 8.
The mean of the parameters of the pulmonary function test (PFT), which includes forced expiratory volume in the first second (FEV1), forced vital capacity (FVC), the ratio of the forced expiratory volume in the first one second to the forced vital capacity of the lungs (FEV1/FVC) and peak expiratory flow rate (PEFR) was studied with respect to the endoscopic grade of disease. Patients with AH: The values of FVC, FEV1 and PEFR were noted in grades 1, 2, and 4 AH, and the mean value of the aforementioned parameters was noted is given in Table 9. Patients with ATH: All four PFT parameters viz. FVC, FEV1, FEV1/FVC, and PEFR were noted along with the endoscopic severity of the disease, and the p-value was calculated as shown in Table 9. AH vs. ATH: The PFT values of the two subsets of patients were compared (Table 9).

### Table 9: PFT vs. ENDOSCOPIC GRADING

| Endoscopic Grade | AH | ATH |
|------------------|----|-----|
|                  | FVC (L) | FEV1 (L) | FEV1/FVC | PEFR (L/s) | FVC (L) | FEV1 (L) | FEV1/FVC | PEFR (L/s) |
| Grade 1          | 1.94±0 | 1.78±0 | 91±0 | 2.76±0 | - | - | - | - |
| Grade 2          | 1.23±0.58 | 1.08±0.56 | 86.68±4.92 | 2.15±0.55 | 0.93±0 | 0.77±0 | 82.80±0 | 1.55±0 |
| Grade 3          | 1.28±0.81 | 1.12±0.70 | 81.31±4.97 | 2.22±0.49 | 0.88±0.20 | 0.67±0.20 | 75.17±6.09 | 1.75±0.57 |
| Grade 4          | 0.92±0.29 | 0.71±0.25 | 75.88±7.73 | 1.73±0.57 | 0.78±0.19 | 0.58±0.10 | 73.11±1.85 | 1.56±0.31 |
| F-value          | 1.64 | 2.24 | 4.75 | 2.40 | 19.15 | 27.36 | 1.20 | 4.929 |
| p-value          | 0.20,NS | 0.10,NS | 0.009,S | 0.080,NS | <0.0001,S | <0.0001,S | 0.32,NS | 0.0017,S |

### TABLE 9: Comparison between endoscopic grade and PFT values

AH: Adenoid hypertrophy, ATH: Adenotonsillar hypertrophy, FVC(L): Forced vital capacity in liters, FEV1(L): Forced expiratory volume in the first second in liters, FEV1/FVC: Ratio of forced expiratory volume in first second to forced vital capacity, PEFR(L/s): Peak expiratory flow in liters per second, F-value: Ratio of two variances, or technically, two mean squares, p-value: Probability value, S: Significant, NS: Not significant

Seventeen (34%) out of the 50 patients studied in the present work were found to have abnormalities in their ECG, while no subject in the control group had any departure from normal (Table 10). Table 10 displays the frequency and type of abnormality found in the ECG of patients with the adenoid and adenotonsillar disease. Nine of the 17 patients had AH, and eight had the adenotonsillar disease. In the nine patients (18%) with AH, sinus arrhythmia was seen in five (10%), sinus tachycardia in three (6%), and Mobitz type 1 block in one (2%) patient. In eight patients (16%) with AH, sinus arrhythmia was seen in four (8%), sinus tachycardia in three (6%), and Mobitz type 1 block in one (2%) patient. Overall, sinus arrhythmia was the commonest finding seen in nine patients (18%).

### Table 10: Abnormal ECG findings in patients with adenoid and adenotonsillar hypertrophy

AH: Adenoid hypertrophy, ATH: Adenotonsillar hypertrophy

| ECG Findings       | AH     | ATH    | Total (n=50) |
|--------------------|--------|--------|--------------|
| Sinus Tachycardia  | 3 (6%) | 3 (6%) | 6 (12%)      |
| Sinus Arrhythmia   | 5 (10%)| 4 (8%) | 9 (18%)      |
| Mobitz Type 1 Block| 1 (2%) | 1 (2%) | 2 (4%)       |
| Total              | 9 (18%)| 8 (16%)| 17 (34%)     |

### Discussion

Sleep-disordered breathing (SDB) has an estimated prevalence of 11% in children [7]. Obstructive sleep apnea affects 2% of the pediatric population and is the most severe form of SDB [3]. Increased upper airway resistance during sleep is due to a combination of soft tissue hypertrophy, craniofacial dysmorphology, neuromuscular weakness, or obesity. Adenotonsillar hypertrophy is the most common cause of pediatric upper airway obstruction and can cause a multitude of cardiovascular and pulmonary complications like sleep apnea, sleeping disorders, pulmonary hypertension, cor-pulmonale, and even heart failure [8].

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2022 Deshmukh et al. Cureus 14(11): e31175. DOI 10.7759/cureus.31175
complications are silent in their progression, and timely intervention results in complete reversal [8].

Detailed history and symptoms were elicited, followed by clinical examination and clinical grading and endoscopic grading of adenoids, ECG, and spirometry carried out, and observations emanating from the study were recorded and statistically analyzed. The mean age of adenoid hypertrophy (AH) and adenotonsillar hypertrophy (ATH) in the present study was 10±3.09 years. The mean age reported by most of the investigators agrees well with the present study [9-14]. In a study by Tatlipinar, the mean age for AH was 6.96±2.11 years, while for ATH was 6.96±1.68 years [15]. In the present study, the mean age of patients with AH was 10.33±3.65 years, while that with ATH was 9.50±1.98 years. Exposure to the environment, to the pollutants, and most importantly, immunological stimulation coupled with allergic and inflammatory episodes makes this age group susceptible to AH and ATH.

Marginal male predominance was found in the present study. Variable preponderance has been reported by various investigators. Male predominance has been reported in some studies [10,12,13]. Female preponderance has been noted in another study [16]. Maximum patients (31, 62%) belonged to the urban area, while 19 (38%) hailed from the rural area. Rout et al. found an overwhelmingly high urban population (26/50, 87%) in their study, which aligns with the present study [17]. Niedzielska et al. observed that inhabitants of the towns reached poorer FVC results in comparison with those of rural areas [13]. It may result from air pollution or other factors and requires further research.

Out of a total of 50 patients with upper airway obstruction, 30 (60%) had AH, and 20 (40%) had ATH. Though not much literature is available on this dimension of the study, Tatlipinar et al. found that AH was seen in 40 patients (42%), and ATH was present in 41 patients (43%) [15]. The present study differs from this observation, but this being the only study available even after extensive search prevents us from drawing any firm inference regarding the predilection for the site of obstruction.

The range of symptoms in the present study was similar to that reported by other authors. Mouth breathing (80%) and snoring (62%) in general and disturbed sleep (16%) and nasal discharge (20%) in particular were observed less frequently in the present study. Racial factors, nasopharyngeal size, use of nasal decongestants, and variable significance attached to these otherwise important symptoms like disturbed sleep and nasal discharge may explain the less frequent reporting of these symptoms by the parents [18]. Nasal endoscopy to grade adenoids was carried out as per the guidelines given by Cassano [5]. In patients with only AH, a maximum number of patients observed had grade 4 (15, 26%) adenoid hypertrophy, followed by grade 3 (11, 22%). While in patients with ATH, grade 5 enlargement (15, 30%) of adenoids was most commonly observed. However, this difference was not found to be statistically significant (p<0.05). Patients with AH are mostly presenting with grade 4, while those with ATH are with grade 3. Seeking early medical help by patients with ATH could be due to the additive and compounding effect of enlarged tonsils.

In patients with AH a successive decrease in FVC, FEV1 and PEFR was noted over endoscopic grades 1, 2, and 4, but a higher mean value of the aforementioned parameters was seen in grade 3, as compared to grade 2 AH. However, a consistent and statistically significant decreasing trend with increasing endoscopic severity of the disease was noted in FEV1/FVC with a p-value of 0.009. In patients with ATH, a progressive decrease in all four PFT parameters viz. FVC, FEV1, FEV1/FVC, and PEFR were noted with an increase in the endoscopic severity of the disease. This decrease was found to be statistically significant (p<0.05) for all parameters except FEV1/FVC. AH vs. ATH-On comparing the PFT values of patients AH and ATH. The corresponding parameters were found to be lower in patients of ATH than those with only AH.

The paucity of studies studying this facet constrains us from a comparison. All 50 patients and 25 controls were subjected to 12-lead ECG. Seventeen (34%) out of the 50 patients studied in the present work were found to have abnormalities in their ECG, while no subject in the control group had any departure from normal. Overall, sinus arrhythmia was the commonest finding seen in nine patients (18%). Norte noted that the use of ECG to diagnose pulmonary hypertension in infants with AH associated with sleep apnea revealed low sensitivity [26]. Yilmaz evaluated the prevalence of arrhythmias, heart rate variability (HRV), and heart rate turbulence (HRT) using 24-hour Holter ECG monitoring pre and postoperatively in children with ATH [27]. They found that although some ECG and Holter findings, such as sinus tachycardia and Mobitz type 1 second-degree atrioventricular block, improved after the operation, the prevalence of arrhythmias and HRV and HRT values did not change significantly in the postoperative period. In a study by Fasunla, seven (9.46%) patients had abnormal ECG findings, of which three patients had isolated right
ventricular hypertrophy, one patient had right atrial hypertrophy, and another had right axis deviation, and the remaining two patients had bilateral ventricular hypertrophy [28]. Rosenzweig states that the ECG commonly shows right atrial enlargement, right-axis deviation, and right ventricular hypertrophy with secondary T-wave changes; however, these findings do not necessarily parallel the severity of the underlying pulmonary hypertension [29]. This area needs further research.

Chronic obstructive adenotonsillar hypertrophy causes significant cardiovascular and pulmonary changes. Recent studies have suggested that chronic pulmonary disorders in adolescents and adults have their origin in the first few years of life and may be related to chronic upper airway obstruction [30]. Unfortunately, this is often overlooked in the clinical setting. The symptoms of progressive pulmonary hypertension are minimal until the rapid onset of severe cardiac decompensation occurs. These entire cascades of events are reversible in the early stages and thus mandate early detection and treatment. Performing PFT, ECG, and Doppler echocardiographic examination in children with adenotonsillar disease is not mandatory but profitable even in the absence of obvious symptoms of upper airway obstruction [30]. Current literature supports performing PFT in children as young as 5–6 years as their development helps them to respond to given commands. As every child with ATH carries the risk of obstructive lung disease and/or pulmonary hypertension, the complementary, comprehensive, and correlative role of clinical, endoscopic, and radiological assessment cannot be over-emphasized.

The changes seen in PFT can be based on the fact that the respiratory system shares a unified airway with the same mucosal carpet hence showing common diseases. ECG was done in the patients purely out of the examiner’s interest to see incidental subclinical and subtle findings as respiratory comprise is directly linked with cardiac activity alterations and any changes in the early decade which is considered physiological can lead to pathological conditions if it persists for a long term.

The inadequate sample size is the limitation of this study. The period for which the patient had adenoid or adenotonsillar hypertrophy was not taken into account. Cardiopulmonary parameters other than pulmonary function tests and electrocardiograms like echocardiograms could be taken in further studies. Similar studies are needed to be conducted on larger groups with more parameters to establish the impact of adenoid hypertrophy and adenotonsillar hypertrophy on cardiopulmonary status.

**Conclusions**

Chronic obstructive adenotonsillar hypertrophy causes significant cardiovascular and pulmonary changes. Unfortunately, this is often overlooked in the clinical setting. The symptoms of progressive pulmonary hypertension are minimal until the rapid onset of severe cardiac decompensation occurs. These entire cascades of events are reversible in the early stages and thus mandate early detection and treatment. Performing PFT and ECG in children with adenotonsillar disease is not mandatory but profitable even in the absence of obvious symptoms of upper airway obstruction. Through this study, we attempt to throw light on the often-missed consequence of AH and ATH, which might help in counseling regarding surgical interventions required.

**Additional Information**

**Disclosures**

- **Human subjects:** Consent was obtained or waived by all participants in this study. Institutional Ethics Committee, Jawaharlal Nehru Medical College and Acharya Vinoba Bhave Rural Hospital, Sawangi (Meghe), Wardha, Maharashtra, India issued approval DMIMS(DU)/IEC/2015-16/1636.
- **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue.
- **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

**References**

1. Kaur R, Chandrashekhgar G, Mody D: An insight into relationship of hypertrophied adenoids & tonsils and dentofacial form. J Dental Med Sci. 2014, 13:48-54. 10.9790/0855-15974654
2. Winkelman A: Wilhelm von Waldeyer-Hartz (1836–1921): an anatomist who left his mark. Clin Anat. 2007, 20:231-4. 10.1002/ca.20400
3. Greenfeld M: Obstructive sleep apnea syndrome due to adenotonsillar hypertrophy in infants. Int J Pediatr Otorhinolaryngol. 2005, 67:1055-60. 10.1016/j.pediatr.05.00182-4
4. Tatlipinar A, Duman D, Uslu C, Egeii E: The effects of obstructive sleep apnea syndrome due to adenotonsillar hypertrophy on the cardiovascular system in children. Turk J Pediatr. 2011, 53:559–65.
5. Brodsky L: Modern assessment of tonsils and adenoids. Pediatr Clin North Am. 1989, 36:1551-69. 10.1016/s0031-3955(16)36806-7
6. Cassano P, Gelardi M, Cassano M, Fiorella MI, Fiorella R: Adenoid tissue rhinopharyngeal obstruction
grading based on fiberendoscopic findings: a novel approach to therapeutic management. Int J Pediatr Otorhinolaryngol. 2005, 67:1503-9. 10.1016/j.ijpedit.2005.07.018
7. Petry C, Pereira MU, Petire PM, Jones MH, Stein RT: The prevalence of symptoms of sleep-disordered breathing in Brazilian schoolchildren. J Pediatr (Rio). 2008, 84:125-9. 10.2253/ped.1770
8. Abdel-Aziz M: Asymptomatic cardiopulmonary changes caused by adenoid hypertrophy. Craniofac Surg. 2011, 22:1401-5. 10.1097/SCS.0b013e31821ec354
9. Saedi B, Sadeghi M, Mojtabah M, Mahboubi H: Diagnostic efficacy of different methods in the assessment of adenoid hypertrophy. Am J Otolaryngol. 2011, 32:147-51. 10.1016/j.amjOTO.2009.11.003
10. Yadav SP, Dodeja OP, Gupta KB, Chanda R: Pulmonary function tests in children with adenotonsillar hypertrophy. Int J Pediatr Otorhinolaryngol. 2005, 67:121-5. 10.1016/s1165-5876(02)00551-8
11. Moghaddam VJ, Bavi SI, Ahvazian K: Do pre-adenotonsillectomy echocardiographic findings change postoperatively in children with severe adenotonsillar hypertrophy. J Saudi Heart Assoc. 2010, 23:51-5. 10.1016/j.jsaha.2010.10.003
12. Rogha M, Amini J, Raisi M: Pulmonary function after adenotonsillectomy. Iran J Otorhinolaryngol. 2016, 28:383-8.
13. Niedzielska G, Kotoski M, Niedzielski A: Assessment of pulmonary function and nasal flow in children with adenoid hypertrophy. Int J Pediatr Otorhinolaryngol. 2008, 72:533-5. 10.1016/j.ijpedit.2007.11.009
14. Dixit Y, Tripathi PS: Community level evaluation of adenoid hypertrophy on the basis of symptom scoring and its X-ray correlation. J Family Med Prim Care. 2016, 5:789-91. 10.4103/2249-4863.201156
15. Tatlıpınar A, Bitezek M, Merik M, Bayraktar Gİ, Tekkeşin Aİ, Gökçeer T: Adenotonsillar hypertrophy: Correlation between obstruction types and cardiopulmonary complications. Laryngoscope. 2012, 122:676-80. 10.1002/lary.22508
16. 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.
17. Rout MR, Mohanty D, Vijaylaxmi Y, Bobba K, Metta C: Adenoid hypertrophy in adults: A case series. Ind J Otolaryngol Head Neck Surg. 2012, 65:269-74. 10.1016/S1207-012-0549
18. Adegbija WA: Current trends of adenotonsillar hypertrophy presentation in a developing country, Nigeria. Int J Otorhinolaryngol Head Neck Surg. 2017, 3:501-5. 10.18203/issn.2454-5929/johns20173050
19. Yassen E, Khanmass A, Anbaky F: Adenoid enlargement assessment by plain X-ray and nasoscopy. Iraqi J Community Med. 2012, 1:98-90.
20. Sharifkashani S, Dahirmoghadam P, Kheirkhah M, Hosseinzadehnik R: A new clinical scoring system for adenoid hypertrophy in children. Iran J Otorhinolaryngol. 2015, 27:55-61.
21. Wormwald PJ, Prescott CA: Adenoids: comparison of radiological assessment methods with clinical and endoscopic findings. J Laryngol Otol. 1992, 106:342-4. 10.1016/s0022215100119449
22. Chisholm EJ, Lew-Gor S, Hajioff D, Caulfield H: Endoscopic findings: a novel approach to therapeutic management. Laryngoscope. 2012, 115:380-4. 10.1002/lary.22508
23. Yaseen E, Khanmass A, Anbaky F: Adenoid enlargement assessment by plain X-ray and nasoscopy. Iraqi J Community Med. 2012, 1:98-90.
24. Shariﬁkashani S, Dahirmoghadam P, Kheirkhah M, Hosseinzadehnik R: A new clinical scoring system for adenoid hypertrophy in children. Iran J Otorhinolaryngol. 2015, 27:55-61.
25. Wormwald PJ, Prescott CA: Adenoids: comparison of radiological assessment methods with clinical and endoscopic findings. J Laryngol Otol. 1992, 106:342-4. 10.1016/s0022215100119449
26. Chisholm EJ, Lew-Gor S, Hajioff D, Caulfield H: Adenoid size assessment: a comparison of palpation, nasendoscopy and mirror examination. Clin Otolaryngol. 2005, 30:59-61. 10.1111/j.1365-2273.2004.00003.x
27. Kubb H, Bingham BJ: Endoscopy in the assessment of children with nasal obstruction. J Laryngol Otol. 2001, 115:580-4. 10.1258/jlary.2001.101.09729
28. Mlynarek A, Tewfik MA, Hagr A, Manoukian JJ, Schloss MD, Tewfik TL, Choi-Rosen J: Lateral neck radiography versus direct video rhinoscopy in assessing adenoid size. J Otolaryngol. 2004, 33:560-5. 10.2510/j7070.2004.003704
29. Lourenço EA, Lopes Kde C, Pontes A Jr, Oliveira MH, Umemura A, Vargas AL: Comparison between radiological and nasopharyngolaryngoscopic assessment of adenoid tissue volume in mouth breathing children. Braz J Otorhinolaryngol. 2005, 71:237-7. 10.1016/s1808-8694(15)31280-5
30. Norte MC, Rocha SC: Adenotonsillar hypertrophy as cause of pulmonary hypertension. Int Arch Otorhinolaryngol. 2008, 2:133-136.
31. Yilmaz F, Gunduz H, Karaslan K, Arinc H, Cosgun M, Sessiz N, Uyan C: Holter analyses in children with adenotonsillar hypertrophy. Int J Pediatr Otorhinolaryngol. 2006, 70:1443-7. 10.1016/j.ijpedit.2006.05.005
32. Fasunla AJ, Onakoloye OA, Ogunkunle OO, Mhamb TT, Nwaogu OG: Routine electrocardiographic request in adenoidectomy: Is it necessary? J Otolaryngol Head Neck Surg. 2011, 65:350-5. 10.1007/s12070-011-0264-0
33. Rosensweig EB, Feinstein JA, Humpl T, Ivy DD: Pulmonary arterial hypertension in children: Diagnostic work-up and challenges. Prog Pediatr Cardiol. 2009, 27:4-11. 10.1016/j.ppedcard.2009.09.005
34. Maurizi M, Paludetti G, Todisco T, Dottorini M, Grassi V: Pulmonary function studies in adenoid hypertrophy. Int J Pediatr Otorhinolaryngol. 1980, 2:243-50. 10.1016/0165-5876(80)90049-x