



The impact of adenotonsillectomy on pulmonary arterial pressure in West African children with adenotonsillar hypertrophy



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ABSTRACT

Objectives: To assess the improvement or otherwise, in the mean pulmonary arterial pressure (mPAP) among children with adenotonsillar hypertrophy (ATH) after adenotonsillectomy (AT), and to examine the impact of timing of surgery as well as the patients' characteristics on the mPAP changes.

Methods: We included 39 children with ATH in this study. The adenoidal enlargement was evaluated radiologically with adenoid nasopharyngeal ratio (ANR) parameter from lateral neck radiograph, whereas the clinical assessment of adeno-tonsillar obstruction was conducted with the 'symptom score'. Pulmonary arterial pressure (PAP) measurement was performed noninvasively by Doppler echocardiography. All patients underwent adenotonsillectomy (AT). After 6 weeks, they were subjected again to clinical and echocardiographic assessments, and the mean pulmonary arterial pressures (mPAP) were then compared. The mPAP changes after AT were further related to the grades of ANR, symptom scores, tonsillar size, and timing of AT.

Results: The preoperative mPAP was 23.46 mmHg and was 18.98 mmHg post-operatively ($P = 0.003$). Seventeen of subjects (43.6%) had pulmonary hypertension (PH) (mPAP ≥ 25 mmHg) preoperatively, out of which 14 (82%) decreased to normal range 6 weeks after AT. Non-reversal of pulmonary hypertension was associated with ANR > 0.75 ($P = 0.043$), but was not related to the timing of surgery, tonsillar size, and symptom score. Significant reduction in mPAP was more likely with ANR ≤ 0.75 and pre-operative mPAP ≥ 25 mmHg. All the symptoms also improved significantly after AT.

Conclusion: Elevated PAP due to ATH in children was mostly reversible by AT irrespective of the timing of surgery, symptom severity, and tonsillar size, but gross enlargement of adenoids seem to be associated with non-reversal of PH.

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1. Introduction

Adenoid hypertrophy or adenotonsillar hypertrophy (ATH) remains the most common cause of upper airway obstruction and obstructive sleep apnoea (OSA) in children. Untreated ATH in some cases have resulted in wide range of cardiopulmonary changes including: pulmonary hypertension (PH) and cor pulmonale, cardiac failure, and systemic hypertension. Incidentally, most of the reported cases of severe cardiopulmonary complications are relatively rare and have been documented to resolve after adenoidectomy or adenotonsillectomy (AT) [1–4]. More recent reports have shown that majority of the affected children seem to belong to a

larger group that suffer partial upper airway obstruction on account of ATH, with asymptomatic milder cardiopulmonary changes [5,6]. These children who have milder cardiopulmonary sequel from obstructive ATH have not been investigated in details. These cardiopulmonary changes can be monitored effectively with doppler echocardiography [7]. The upper airway obstructive symptoms of ATH in general, including the sleep disordered breathing, have been shown to be effectively treated with AT [8,9]. However, the questions of whether milder forms of sleep disorder breathing in children are associated with cardiovascular changes and, what factors influence the reversal of cor-pulmonale after AT are yet to be examined in details.

This present study was designed to examine the improvements or otherwise, of the mean pulmonary arterial pressure (mPAP) after adenotonsillectomy in children with ATH, and to examine the impact of timing of surgery as well as the clinical and radiological characteristics of patients on the mPAP changes.

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2. Method

The study population consist of 39 consecutively recruited children (23 (59%) boys and 16 girls (41%) who underwent adenotonsillectomy (AT) or adenoidectomy on account of upper airway obstruction from ATH, in Otolaryngology department of the University of Nigeria Teaching Hospital Enugu during 32 months period ending February 2016.

2.1. Ethical consideration

Approval for the study was given by the institution ethical review committee.

Written informed consent was obtained from the parents or care givers of each of the children included in the study.

2.2. Recruitment of participants

Subjects included in the study were children with a clinical diagnosis of adenoid hypertrophy (AH) with or without tonsillar hypertrophy as the only cause of upper airway obstruction, those who completed the follow up schedule, and those that gave consent for the study.

Participants excluded had previous adenoidectomy/adenotonsillectomy (AT), or had other causes of upper airway obstruction (such as: nasal septal deviation, craniofacial abnormalities), or other cases of sleep apnoea (such as obesity or cerebral palsy). Participants were also excluded if they had co-morbidities that would otherwise explain PH (such as previous history of cardiac disease, or discovered during echocardiography, HIV infection, chronic obstructive lung disease, and sickle cell anaemia).

The following procedure was adopted for all the participants.

2.3. Pre- and post-operative assessment

At the point of first contact, history was obtained from the caregivers and physical examination performed on all the children including a general, ENT and cardiovascular assessment.

Assessment of the severity of the upper airway obstruction was carried out by grading the following symptoms: snoring, daytime stertor, mouth breathing during sleep, daytime mouth breathing, daytime hypersomnolence, obstructive breathing during sleep, nasal obstruction. The symptoms were graded with the following responses: 'absent', 'occasionally' (mild grade), and 'always' (severe grade). The responses of 'occasionally' and 'always' attracted scores of '1' and '2' respectively, whereas 'absent' answer was scored '0'. There was a maximum of 14 points. The overall symptom score was classified as either 'mild' (1–5 points), moderate (6–10 points), or severe (>10 points). No participant in this study had symptom score less than 6-points. The resolution/improvement or otherwise, of sleep disturbances and breathing difficulties after AT, was assessed by comparing the pre- and post-operative mean symptom scores.

The size of the tonsils was graded during oropharyngeal examination on a scale of 1–4 employing the Brodsky classification as previously described [7,10]: Grade 1 tonsils represented tonsils confined to the faucial pillars; grade 2 was tonsils that extended just outside the pillars; grade 3 - tonsils extending outside the pillars but not meeting in the midline; grade 4 – were tonsils meeting in the midline. All the patients in this study had a minimum of grade 2 tonsils.

Radiologic examination: included lateral plain x-ray of the nasopharynx. The size of the adenoid was assessed with the 'adenoid nasopharyngeal ratio' (ANR) parameter as previously described [11]. The adenoid hypertrophy was considered mild if AN ratio was ≤ 0.62 , moderate = $0.63–0.75$, gross = >0.75 . All the

children in this study had an ANR ≥ 0.63 .

Following routine cardiovascular examination, their cardiac functions were all evaluated pre-operatively using two-dimensional Coloured Doppler 'SONOSCAPE SSI-5000' Echocardiography, with 3.5 Hz transducer. It was equipped with the capability for M-mode, two-dimensional, 3-dimensional, pulsed wave, continuous wave and colour flow Doppler echocardiographic measurements. The mean pulmonary artery systolic pressure (mPAP) was determined by using the continuous wave Doppler to get the peak tricuspid regurgitation jet velocity and the pressure gradient. This was substituted in the Bernoulli Equation: $PASP = RVSP = 4 \times (V_{TR})^2 + RAP$. Pulmonary hypertension (PH) was defined by $mPAP \geq 25$ mmHg. The raised PAP was regarded as borderline for $mPAP \geq 20 \leq 25$ mmHg, and normal PAP ($mPAP < 20$ mmHg). All the children were subjected to similar echocardiographic examination at 6 weeks postoperatively. The obtained changes in mPAP post-operatively were compared with the tonsil grades, ANR and the overall symptom scores as well as the timing of AT (defined in relation to the Onset of symptoms/duration).

2.4. Operative procedure

- Adenoidectomy was carried out for all patients using conventional curettage technique. Tonsillectomy was by conventional dissection technique. Anaesthetic induction was done using isoflurane or halothane in oxygen to prevent sudden carbon dioxide wash and apnea.

2.5. Statistical analysis

The 'Statistical Package for Social Sciences' (SPSS) version 16.5 for Windows, was utilised in the analysis. The preoperative and postoperative echocardiographic parameters were compared with paired sample *t*-test, and the mPAP changes were compared among participants of different clinical and radiologic parameters with Fisher's exact test. Statistical significance was set at $P < 0.05$.

3. Results

Sixty one children who underwent adenoidectomy/AT were evaluated but 39 completed the follow up schedule and therefore met our inclusion criteria. All the children underwent adenotonsillectomy. Their mean was 4.6 years with a median of 3.4 years and 25–75 percentile range of 2.4–5.8 years. Table 1 shows the age distribution of the patients in relation to their mPAP. The mPAP was recorded highest among the 9 youngest participants aged <2 years, but the differences in the mPAP among the age groups were not significant ($P = 0.847$). In the pre-operative period, PH ($mPAP \geq 25$ mmHg) was observed in a total of 17 children which was distributed in 56%, 35%, and 46% of the age groups <2 years, 2–5 years, and >5 years respectively. In the post-operative periods, the mPAP of 14 of the children normalised below 25 mmHg, whereas it remained above 25 mmHg in 3 of children, giving 82.4% resolution of pulmonary hypertension following AT ($P = 0.000$).

Table 2 showed the mPAP before, and 6 weeks after AT. There was significant decrease in mPAP postoperatively (paired sample *t*-test, $P = 0.003$). The decrease in mPAP following AT/adenoidectomy was most remarkable among the children with pre-operative pulmonary hypertension with an average of 6.8 mmHg drop in mPAP ($P = 0.007$).

The recorded changes in the mPAP following adenoidectomy/AT among different pre-operative parameters were outlined in Tables 3 and 4. The reduction in the mPAP following surgery was

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