THE EFFECTS OF ADENOTONSILECTOMY ON PULMONARY PRESSURES AS SEEN IN THE ENT DEPARTMENT- KENYATTA NATIONAL HOSPITAL.

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Masters of Medicine, in Otorhinolaryngology Head and Neck Surgery.

A Dissertation Submitted In Partial Fulfillment of Requirements Of The University Of Nairobi For The Award Of The Degree Of Master's In Medicine In ENT, Head And Neck Surgery.

STUDENT'S DECLARATION

This is my original work and has not been presented for a degree in any other university'

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This Dissertation has been approved by Department of ENT, Head and Neck Surgery, University of Nairobi.

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ABSTRACT

Background

Patients with Adenotonsilar hypertrophy have been shown to have raised pulmonary artery pressures due to upper airway obstruction. Adenotonsilectomy reduces these pulmonary arterial pressures. There is some controversy as to when a post-operative Echocardiograph should be done so most studies adopt a timing of 6 months.

Broad objective

To find out whether there is any difference between pre and post-operative pulmonary artery pressures in patients with Adenotonsilar hypertrophy by 6 weeks post operatively.

Methodology

Patients were consecutively recruited from the ENT department KNH. A thorough history and clinical examination were done. The lateral soft tissue neck radiographs already done, were used to measure the adenoid ratio using the Cohen and Konak grading scale and confirm adenoid hypertrophy and physical examination was used to grade the tonsilar hypertrophy using the Brodsky grading scale. Patients were assessed for level of awake partial pressures of oxygen, weight, height and body mass index. The cardiovascular and respiratory systems were also assessed. Echocardiograms were done pre-operatively and at 6weeks post-operatively.

Results

There was a slight male preponderance with an age range of 2.5 years to 10 years. The age group of 2.5-4 years had the highest number of patients, while the symptom duration ranged from 1-9 years with the highest number of patients having symptoms of 1-3 years. The history of symptoms included snoring, mouth breathing and sleep disordered breathing with apneic attacks as seen by an observer (97.1%, 96.2% and 90.4%). The pulmonary pressures range 0 to 24mmHg, 87% below 20mmHg and 10% between 20-24mmHg. Pre-op and post-op mPAP changed significantly (P value of <0.001).

Conclusion

There was an association between adenoid size and the level of mPAP, but no association between tonsillar size, history of symptoms, symptom duration and mPAP. Pre- operative mPAP dropped significantly post- operatively for 87% of the patients.

1.0 CHAPTER ONE: BACKGROUND

1.1 Anatomy

The pharyngeal tonsil is a midline mucosal lymphoid tissue situated in the postnasal space inferior to the Sella turcica and posterior to the choanal aperture. It is also part of the Waldeyers ring together with the lingual, palatine and tubal tonsils. It is covered in pseudo stratified columnar epithelium. They are important as first line of defense in the aero-digestive tract. Adenoid hypertrophy refers to the hypertrophy of the postnasal space lymphoid tissue while tonsilar hypertrophy refers to enlargement of the palatine tonsil.

The palatine tonsils are a pair of lymphoid tissue lined by stratified squamous epithelium and located in the lateral aspect of the oropharynx encased in the tonsilar fossa. This fossa is bound anteriorly by the anterior pillar (palatoglossus muscle) and posteriorly by the posterior pillar (palatopharyngeus muscle). The tonsilar bed is formed by the superior constrictor muscle and styloglossus muscle. The tonsil is found within a tight capsule, however the capsule is loosely bound to the tonsilar bed and it is within this plane that tonsillectomy is done.

The adenoid has rapid growth in infancy but involutes from about age 8-10 and is rarely seen in adults. The tonsil on the other hand begins to involute during puberty.(1)

1.2 Pathophysiology

Adenoids and tonsils have physiological enlargement in childhood that subsides in adolescence and adulthood. Blockage by adenoids and tonsils reduces airflow. Adenoid hypertrophy and Adenotonsilar hypertrophy are known to cause pulmonary artery hypertension and eventually cor-pulmonale. This relationship was first noted and described in case reports in 1965 discussed by Farrehi and Menashe consisting of 2 patients(2).PAH associated with AH is classified under the 3rd group as per the 2015 ESC/ERS Guidelines for

the diagnosis and treatment of pulmonary hypertension and has not been noted to have a genetic component associated with it(3).

Under normal conditions during nasal breathing, the air column has laminar flow. This is disturbed by ATH causing obstruction in the nasopharynx and affects lung ventilation. This causes a raise in upper airway resistance in an attempt to raise air entry via the nasal route(4). If ventilation is inadequate, then mouth-breathing becomes obligatory. Mouth breathing causes turbulent flow. This flow is not sufficient to maintain patency of the nasopharynx and oropharynx especially when the patient is asleep and supine, the tongue also falls backwards due to gravity, all this causes alveolar hypoventilation. Hypoventilation causes hypoxia and hypercarbia when asleep(4)Hypoxia thus causes an increase in carbon-dioxide retention. This hypoxia also causes pulmonary vasoconstriction and increases capillary permeability(5). Of note is that some literature discusses the hypertrophy of the capillary muscular layer that causes vasoconstriction and a rise in mean pulmonary arterial pressures. The end point for all this leads to raised mean capillary pressure and right heart failure with the features associated with it.

1.3 Clinical presentation

A history provides symptoms associated with nasal obstruction, including: mouth breathing, snoring, sleep apnoea episodes, restless sleep with multiple awakening episodes, day-time hyper-somnolence for some patients while others have hyperactivity. There has also been an association with enuresis as well as poor feeding and poor weight gain for some. Post operatively there is weight gain in all children, those who are underweight and in those who are not(6).

The clinical symptoms of pulmonary artery hypertension are non-specific and mainly related to progressive right ventricular (RV) dysfunction. Initial symptoms are typically induced by

exertion. These include shortness of breath, fatigue, weakness, angina and syncope. Less commonly, patients may also describe dry cough and exercise-induced nausea and vomiting. Symptoms at rest occur only in advanced cases.

Physical signs of pulmonary artery hypertension include left parasternal lift, an accentuated pulmonary component of the second heart sound, a Right ventricular third heart sound, a pan-systolic murmur of tricuspid regurgitation and a diastolic murmur of pulmonary regurgitation. Elevated jugular venous pressure, hepatomegaly, ascites, peripheral edema and cool extremities characterize patients with advanced disease. Wheeze and crackles are usually absent.

1.4 Complications

The hypertrophy of these tissues is associated with complications such as: nasal obstruction (snoring, mouth breathing, dry lips, obstructive sleep apnea(7)), cardiopulmonary symptoms (pulmonary hypertension), right ventricular hypertrophy and cor-pulmonale, ear and paranasal sinus complications (frequent otitis media and sinusitis), voice changes (flat and toneless)(8), failure to thrive (due to anorexia poor intake, increased energy consumption from increased work during breathing and alterations in nocturnal growth hormone patterns (7)(9)).

1.5 Diagnosis of Adenotonsilar Hypertrophy

Adenoid and tonsilar hypertrophy may be diagnosed by history and clinical examination. This also helps to assess severity of upper airway obstruction, rule out co-morbidities and make a decision on further tests required. In history one expounds on sleep apnea episodes as witnessed by an observer (e.g. the parents), snoring, daytime hyper-somnolence, chronic or recurrent symptoms of tonsillitis associated with fevers(6). A point to note is that Polysomnography is the gold standard to assess for sleep apnea and features of upper airway obstruction however these facilities are not available in our setup(7).

In the physical examination, the grade of the tonsilar hypertrophy will be staged by the Brodsky (shown in the Appendix I) tonsilar grading scale. It is important to assess patients for adenoid fascies as well as other comorbidities like heart failure

Adenoid hypertrophy can be assessed using a soft tissue lateral neck radiograph and graded using the Cohen and Konak grading system (shown in the Appendix I).In a study done in 2012 that compared the various radiological methods to assess adenoid hypertrophy this method had been found to have 75% inter and intra- personnel reproducibility(10). Of importance is that this method was also seen to have the best comparable results to endoscopy as compared to other radiological assessment methods(11).

1.6 Diagnosis of Cardiac Complications

Polysomnography is the gold standard to assess for sleep apnea and features of upper airway obstruction however these facilities are not available in our setup(7).

PAH can be inferred using Electrocardiograms (ECG) as well as chest radiographs which are cheaper modalities than ECHO, however negative findings in these modalities do not exclude the presence of disease. This is because the symptoms are more likely to be seen in severe as opposed to mild disease.

Pulmonary Artery Hypertension is defined as mean pulmonary pressures ≥25mmHg at rest as assessed by cardiac catheterization(12). Normal pulmonary artery pressures fall within 14± 3mmHg range, with an upper limit of normal of approximately 20mmHg (7). The patients with mean pulmonary pressure ranges between 21 and 24mmHg may not have significant features of cardiopulmonary failure(12).Echocardiography has however been seen to be a useful noninvasive tool to diagnose pulmonary artery hypertension in comparison to cardiac catheterization which is the gold standard(12)(13)(14).

1.7 Literature Review

In our set up (KNH)a dissertation by Gathere S.A(15) in 2002 assessed the ECG and chest X- ray findings in children with adenotonsilar hypertrophy. He found that 10% of the

children had right ventricular hypertrophy while 11% had features of cardiomegaly on chest radiograph. He however was not doing a pre and post-operative comparison of the cardiac features found in patients with ATH. In 2011 D. Marangu et al did a cross sectional study to assess the prevalence of pulmonary artery hypertension in patients with adenotonsilar hypertrophy. That study found that 1 in 5 patients with adenotonsilar hypertrophy had raised mean pulmonary artery pressures (mean pulmonary artery pressure) ≥25mmHg, comprising a prevalence of 21.9%(16). In that study, 22.3% had borderline elevation of mean pulmonary artery pressure >20 mm Hg but <25 mm Hg while 41.5% had mean pulmonary artery pressure <20 mm Hg. The cardiovascular examination findings of mean pulmonary artery pressure <20 mm Hg were similar to the children with borderline elevated mean pulmonary pressures. This is relevant in that it shows there are still quite a number of patients who do not have overt PAH but have elevate mean pulmonary artery pressure in association with the ATH(16).

In Egypt there have been several studies done. In 2009, Ehab Soud, El Moneim(17)assessed the effect of adenoidectomy on right ventricular performance in children. The study had 30 children with adenoid hypertrophy aged between 2.5 and 12years. These children were examined using ECHO 1 day pre-operatively and a repeat done 1 month post-operatively. There was improvement in the right ventricular output and filling as well as reduction in pulmonary artery pressures. They assessed right ventricular output and mean pulmonary artery pressures with a focus more on the cardiac effects. This is because it was felt that the mean pulmonary artery pressure reduced by a small margin (4mmHg in their study and 6mmHg in studies previous to theirs) yet the study subjects markedly improved. That, according to the investigators, couldn't be explained by the small margin of mean pulmonary artery pressure improvement. Mosaad Abdel Aziz in 2011(18) did a study assessing asymptomatic cardiopulmonary changes caused by adenoid hypertrophy. They had 80patients

and assessed them using a chest radiograph and ECHO pre-operatively and the patients then had adenoidectomy with or without tonsillectomy. An ECHO was then used to assess them post operatively at 6 months. Their patients had mean pulmonary artery pressure ranging 22.7 mmHg with a standard deviation of 3.8 preoperatively and 17.2 mmHg standard deviation 2.1.

Turkey also has had a few studies regarding this topic. Yilmaz in 2005(19) looked at 52 subjects with ATH and had 33 controls. In this study, they used mean pulmonary artery pressure of 20mmHg as the cut off, lower than for most other studies. The patients had ASTS done and reviewed at 3 months and again at 6 months. It was found that 31 out of 52(59%) had OSA pre-operatively in the study group. Meanwhile in the control group, only 3 patients had snoring and difficulty in breathing, none had sleep apnea. At 3 months postoperatively, 18 of the 27 patients in the study group had normal values on ECHO while 9 still had raised mean pulmonary artery pressure (above 20mmHg but they were lower than the pre-operative values). These values normalized by 6 months post-operatively. They used Doppler ECHO and assessed mean pulmonary artery pressure as well as lateral neck radiographs. Their cut off value for pulmonary artery hypertension was 20mmHg this could explain their high prevalence of 59%.

While Koc et al in 2012 had a sample size of 27 subjects and no control group. In this study they were assessing tonsilar hypertrophy grade III and IV, using lateral neck x-rays and RV dysfunction as well as mean pulmonary artery pressure assessed using a Doppler ECHO. The children had a mean age of 8±2. They found that 44% had grade III and 56% had grade IV tonsilar hypertrophy. These patients were reviewed 3 months postoperatively using an ECHO. They found there were significant improvements in RV function as well as reduction in mean pulmonary artery pressure. Their sample size was small however and may not be a good representative of their population(1) .

Pac Asyenur in 2003 compared cardiac function and valvular damage in children with and without ATH. They had a study sample size of 28 pediatric patients with ATH and 35 healthy children matched for age and gender used as controls. The patients in the study arm (group I) were assessed at 1 month postoperatively. It was found that in the study group there was no statistically significant difference in pre and post- operative findings. This has been postulated to be due to the short post-operative duration of 1month unlike the other studies that assessed the post- operative period 3 months and beyond. However this has been challenged by the study done in Egypt(20).

A study done in Brazil by Martha et al in 2013(21) assessing the reversal of pulmonary hypertension in children after adenotonsilectomy, the study ages were from 1-16 years. These were patients that had already been referred for adenotonsilectomy due to ATH. The patients required to have tonsils graded as III or IV. Here they also evaluated for high arched palates, open bite or over bite. They had a sample size of 30 subjects based on their prevalence of 25% (though in the results obtained the prevalence was 36%)and had a control of 10 patients. Adenoid hypertrophy was assessed using video assisted nasal endoscopy and plain x-ray of the nasal cavities. ECHO was used to assess the systolic pulmonary artery pressure. A point to note was that the pulmonary artery hypertension decreased by 26% post operatively(21).

This year (2017) there is a study that has been published in West Africa by F.T. Orji et al(22). It was assessing the impact of adenotonsilectomy on pulmonary arterial pressure in West African children with adenotonsilar hypertrophy. They had a sample size of 39 children with ATH. They used adenoid nasopharyngeal ratio (ANR) to assess the AH on the lateral radiograph. Patients then had a pre-operative Echo and a post- operative Echo at 6weeks. In this study they were assessing the mPAP and used the cut off for PAH as mPAP $\geq$25mmHg. They found that Elevated PAP due to ATH in children was mostly reversible by adenotonsilectomy despite the timing of surgery, symptom severity, and tonsillar size. However, they found that gross adenoid enlargement of was associated with non-reversal of PH(22)

It has however been shown that adenotonsilectomy helps to reduce the pulmonary artery pressures.

1.7.1 Summary of Literature Review

Investigator	Year	Tests	Findings	Comments
Dr. Gathere	2002	ECG, CXR	10% RVH, 11% cardiomegaly Not a case control study	No pre and post op analysis.
Dr. Marangu	2011	Echo, ECG, lateral neck X-ray	mPAP >21mmHg, 21.9% prevalence, 22.3% borderline, 41.5% <20mmHg Not a case control study	No pre or post op analysis.

Investigator	Year	Tool	Results	COMMENTS
Pac Asyenur et al	2003	Echo, Lateral X-ray	No change at 1/12	Small sample size
Yilmaz et al	2005	Echo, Lateral X-ray	Prevalence 59%, change at 3/12 &6/12	mPAP cut off 20mmHg
EhabSoudElmonein	2009	Echo, Lateral X-ray	Reduced PAP, improved RV output	Small reduction in mPAP, focus on cardiac events
Mossad et al	2011	Echo chest X-ray	Significant improvement at 6/12	Noticed asymptomatic cardio pulmonary changes due to AH
Koc et al	2012	Echo, lateral X-ray	Improved RV function, reduced mPAP 3/12	Small sample size
Martha et al	2013	Echo, VNE, lateral X-ray	Prevalence 25%, 36% in study. PAH reduction 26%	
F.T. Orji et al.	2017	Echo lateral X-ray	mPAP≥25mmHg elevated PAP reversible by AT irrespective of symptom severity, TH but grossly enlarged AH associated with no-reversible PAH	sample size 39

2.0 CHAPTER TWO: JUSTIFICATION:

ATH is one of the most common ENT conditions with ASTS being one of the most frequent surgeries done. It has also been seen that PAH may be present but heart failure is seen as a late outcome thus ASTS is important to correct the raised mPAP. The prevalence of pulmonary artery hypertension is 1 in 5 patients with ATH in our set up. It is important to assess if by 6 weeks there is a significant reduction in the mPAP and the clinical findings found most commonly pre operatively.

2.1 Hypothesis

2.1.1 Null Hypothesis

Adenotonsilectomy does not reduce pulmonary pressures at 6weeks postoperatively

2.1.2 Alternative Hypothesis:

Adenotonsilectomy causes a reduction in pulmonary artery pressures postoperatively at 6 weeks.

2.2 Aims and Objectives

2.2.1 Broad objective

To find out whether there is any difference between pre and post-operative pulmonary artery pressures in patients with Adenotonsilar hypertrophy postoperatively.

2.2.2 Specific objectives:

- To assess the pre-operative mPAP.
- To assess the post- operative mPAP.
- To assess the radiological grade and the effect associated with raised pulmonary pressures in Adenotonsilectomy patients.
- To assess the association between clinical grade and its effect on PAH pre and post operatively.

3.0 CHAPTER THREE: MATERIALS AND METHODS:

3.1 Study Design

This was a prospective cross sectional study.

3.2 Study Area

The study was conducted at Kenyatta National Hospital (KNH) at the Ear Nose and Throat (ENT) out-patient clinic and ward 5C KNH.

3.3 Target Population

The study target population comprised of 113 consecutively recruited patients already selected for Adenotonsilectomy by the Otorhinolaryngologist in the ENT clinic or ward 5C in KNH. The ages ranged between 2 and a half years and 12 years.

3.4 Inclusion Criteria

This study included patients who had already been selected for Adenotonsilectomy and patient's whose parents/guardians consented to be involved in the study.

3.5 Exclusion Criteria

This included patients with: congenital anomalies that would cause UAO e.g. choanal atresia, Down's syndrome, muscular anomalies e.g. in patients with cerebral palsy, children who have chronic illnesses e.g. sickle cell disease, rickets, children with nasal obstruction due to other factors e.g. septal deviation, septal spurs, known cardiovascular diseases due to other factors other than ATH, tracheal malformations e.g. subglottic stenosis and patients who refused to participate.

3.6 Sampling Procedure and Sample Size Calculation

To calculate the sample size to help achieve the above objective, the following formula was used:

$$n = \frac{\left(Z_{.1-\alpha/2}\sqrt{P} + Z_{.1-\beta}\sqrt{P_{disc}} - P_{diff}^2\right)^2}{P_{diff}}$$

Where:

The significance level (α) is 5% and a power of 80% (from the t distribution tables $t\alpha/_2 =$ 1.96and $t_{.\beta} = 0.84$)

P_{diff}Is the difference in proportions of discordant pairs pre and post-operative artery pressure (29.2%)?

$P_{disc}$is the sum of proportions of discordant pairs pre and post-operative artery pressure (13.2%)?

Thus replacing the above parameters in the above formula, the sample size for this study was to be 101 patients(23)

3.7 Sampling Procedure

The sampling frame was patients being managed at the ENT out-patient clinic and in-patients from ward 5C in KNH selected for Adenotonsilectomy. The sample selection was done by consecutive sampling.

Recruitment, consenting and Data Collection Procedure

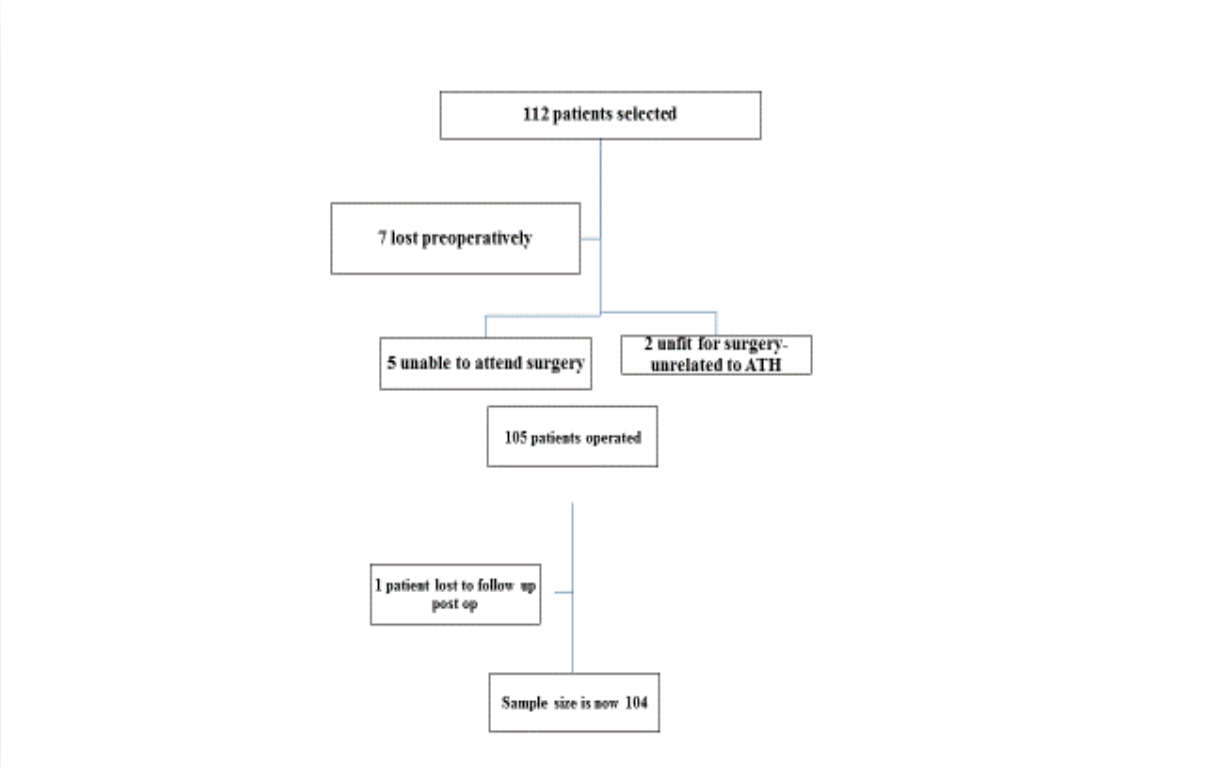
The principal investigator was available in the ENT outpatient clinic and ward for the recruitment of sample patients until the desired sample size was achieved.

For each identified sample patient, the following was done:

- Explanation of the study to the patient and obtaining of consent.
- Demographic and medical history taking and physical examination, lateral soft tissue radiograph and echocardiograms.

The study sample consisted of a cohort of 113 patients already diagnosed by qualified Otorhinolaryngologist in the ENT clinic with Adenotonsilar hypertrophy. Seven were lost preoperatively (5unable to attend surgery and 2 unfit for surgery), 1 lost to follow up post operatively bringing the sample size to 104.

Figure 1: Flow chart showing patient selection



3.8 Obtaining Informed Consent

The principal investigator introduced herself to the participants and explain the purpose of her study. After the study was explained to the participants, they were allowed to read through the participant information sheet and the consent form. Their concerns and questions were addressed before the consent was signed. Thereafter, parents or legal guardians for those below the age of 18, were asked to sign the consent forms if they agreed to participate in the study.

All participants were able to read and understand the consent in English and were able to go through the documents with the principal investigator and sign. No participant's parent or legal guardian was illiterate.

3.9 Data Collection Procedure

Pre-treatment demographic and clinical data was collected using questionnaires that contained closed and open ended questions, as well as information obtained from the ECHO results and lateral soft tissue radiograph.

Patients with Adenotonsilar hypertrophy selected for surgery were recruited from the ENT department KNH. The eligible patients were consecutively recruited into the study during the study duration and placed into: age groups, gender and grades according to the ATH they have.

These patients were already selected for Adenotonsilectomy by an Otorhinolaryngologist at the ENT department in KNH. The principal researcher then took a history and perform clinical examination. A general examination was done and that included assessing a patient's functional status, vital signs e.g. heart rate SPO_2, obtaining patient weight and height, a full head and neck examination, cardiovascular, respiratory and abdominal examination. Heart rate and respiratory rate will be counted using a stop watch and the SPO_2 was assessed using a Santa-Medical® pulsoximeter. The weight was measured using a SECA® digital weighing scale machine and a health-o-meter® manual height gauge found in the ENT ward. In the oral cavity examination, focus was on tonsilar examination (mainly the size). The cardiovascular and respiratory systems were assessed using a Litmann's stethoscope. This was used to assess heart sounds S1S2, including murmurs and lung crepitations. Hepatomegally was assessed during the abdominal examination. A lateral soft tissue neck radiograph was done as routine pre-operatively and used to measure the adenoid ratio using the Cohen and Konak grading scale (Appendix I) and confirm adenoid hypertrophy. Assessment of the radiograph was done using a ruler. Physical examination was used to grade the tonsilar hypertrophy using the Brodsky grading scale (Appendix I). A Doppler ECHO was also done pre-operatively and at 6 weeks post operatively in the Cardiology department. The cardiologist was blinded as to the severity of patient symptoms.

A Doppler ECHO was also done pre-operatively and 6weeks postoperatively by the same Cardiologist in the Cardiology department. All patients had baseline preoperative investigations of: height, weight, Body Mass Index, full haemogram and urea electrolytes and creatinine. A preformed questionnaire was used to collect data.

These patients then underwent Adenotonsilectomy under general anesthesia. Adenoidectomy was done by sharp curettage and tonsillectomy done by blunt dissection out of its bed both with cold steel instruments and hemostasis achieved by packing and/or bipolar diathermy. Post- operatively the patients received the same post-operative care. None of the patients developed complications postoperatively and they received the same care postoperatively i.e.

paracetamol and ibuprofen given at 10mg/kg three times daily, and Augmentin 25mg/kg twice daily orally for 5days.

3.10 Quality Control

Quality control was a continuous process throughout the study to maximize validity and reliability of the findings of the study.

3.10.1 Pre- Test

Pre-test of the study instrument was carried out to structure and modify the research instrument by clarifying grammar and language used so as to avoid bias and misinterpretations of the questions. The data collection instruments were also pre-tested for consistency, timing, accuracy and reliability. The pre-test was conducted among patients in the month of March 2017. The questions were then modified and rectified to remove those that were not relevant to the study or with ambiguous findings.

3.10.2 Validity and Reliability of Research Instrument

The research concentrated on content validity by performing a pre-test so as to adjust the research instrument to meet the required standards. The questionnaire was presented to the University for validation by the supervisors and research panelists before data collection.

3.11 Data Management

All data was entered in password secured computer databases. The databases were designed with checks for ranges, validity and consistency of data to minimize data entry errors. Data cleaning was conducted in SPSS version 18 through inspecting each variable for outlying or invalid values as appropriate, and inspecting ranges and means for continuous variables and using a paired t-test for non-independent samples. Categorical variables were analyzed by calculating percentage of patients with a level of the variable preoperatively and comparing this with the postoperative proportion using a McNemar's chi square test for dependent proportions.

3.12 Limitations

The radiographs were not done by one specific radiologist/radiographer.

3.13 Ethical Consideration

Approval to conduct the research was sought from KNH and University of Nairobi Ethics and Research committees. Permission to conduct research was also sought from the relevant

authorities at clinics and wards. Respondents were given consent to participate. They were informed that it was voluntary and informed of their right to accept or withdraw or refuse to participate. The researcher gave full information about what the research entails and ensure participants were competent to give consent. Full consent and explanation is in the Appendix. The questionnaires were administered after duly obtaining the consent of the participant. Participants' privacy was highly maintained by ensuring that they will not be exposed to the public when filling questionnaires. The researcher has ensured the anonymity of respondents by concealing their identity and keeping research data confidential for research purposes only. All concerns causing any sort of discomfort to respondents were resolved immediately and mitigation strategies put in place.

One participant was found to have (bilateral SNHL- moderate and severe) not associated with the ATH and was referred for further assessment and management. The one patient found to have CTGA with pulmonary stenosis was sent to the Cardiology clinic for further evaluation and management. There was no penalty for declining to participate in the study and the study results shall be published in reputable journals and periodical publications for the benefit of the medical fraternity, the study subjects and general population.

4.0 CHAPTER FOUR: RESULTS

4.1 Sample Characteristics

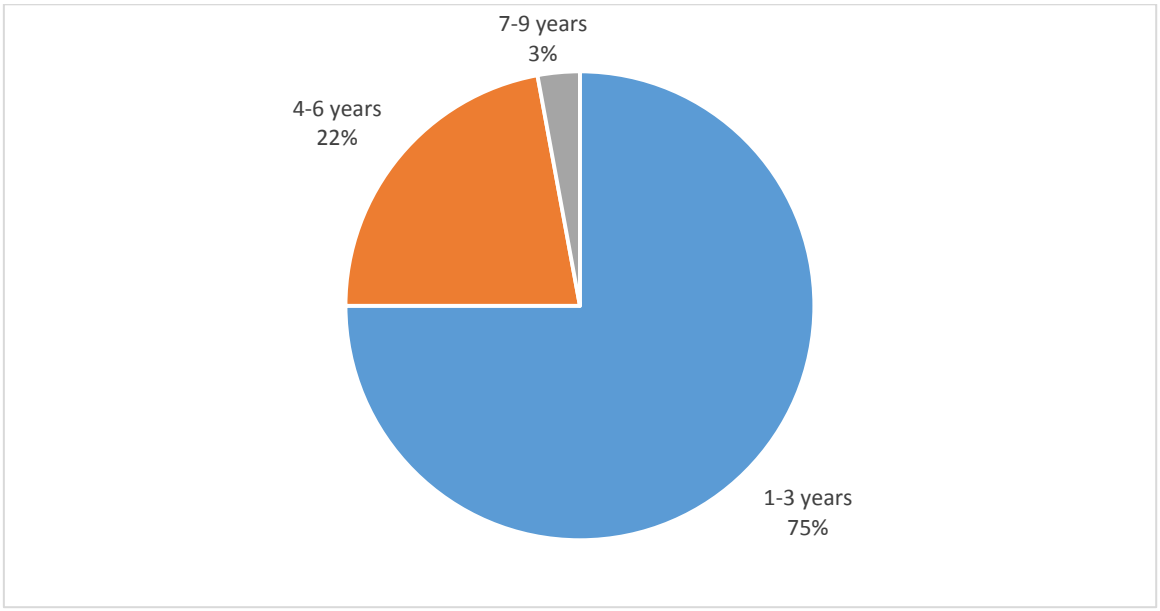
The sample comprised of a total of 104 patients of which there was a slight male preponderance as seen in the table below and a higher percentage of patients in the age group ranging from 2.5 to 4 years. Only 3 of these developed PAH (1 with moderate and 2 with mild PAH).

Table 1: Demographic characteristics of children with Adenotonsilar Hypertrophy in KNH

	Frequency	Percent
Sex		
Male	54	51.9%
Female	50	48.1%
Age		
2.5-4 years	60	57.7%
5-6 years	29	27.9%
7-10 years	15	14.4%

The symptom duration ranged from 1 to 9 years with the age group 1-3 years having the largest percentage.

Figure 2: Symptom duration in children undergoing Adenotonsilectomy in KNH



There was a positive correlation between duration of symptoms and preoperative mPAP (rho = 0.237, p < 0.001), table 2. Pre-operative mPAP also showed a strong positive correlation with post-op mPAP (rho = 0.676).

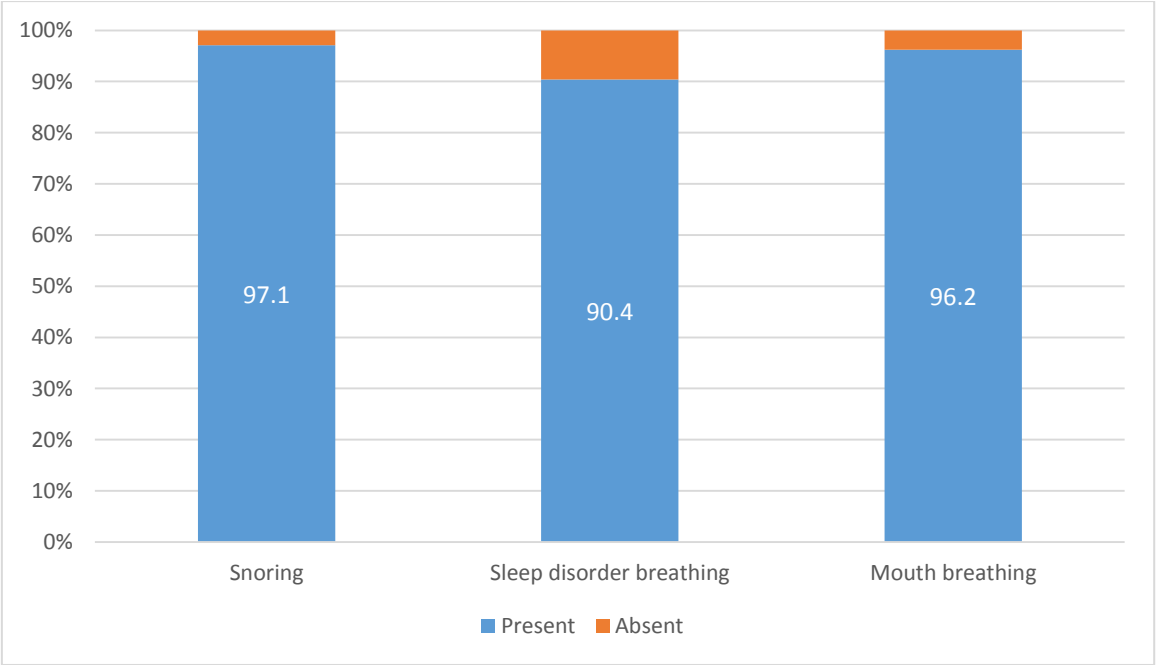
Table 2 : Spearman's rank co-relation co-efficient for mPAP values and duration of symptoms

		Symptom duration	Pre-op mPAP	Post-op mPAP
Statistic				
Coefficient	**Symptom duration**	1		
Coefficient	**Pre-op mPAP**	0.247**	1	
P value		0.012		
Coefficient	**Post-op mPAP**	0.117	0.676**	1
P value		0.237	<0.001	

In terms of the symptoms, snoring was found in up to 97.1% followed by mouth breathing in 96.2% and finally sleep disordered breathing with apneic attacks as seen by an observer was 90.4%.

Only one child presented with symptoms of tachypnea and tachycardia and required to be managed for heart failure and stabilization before Adenotonsilectomy.

Figure 3: History of symptoms in children with Adenotonsilar Hypertrophy in KNH



The weights of all the patients ranged from 10-40.6 kg with a mean of 17.8 and a SD of 5.4. We also assessed the BMI in this study. The mean BMI was 15.4 (SD ± 2.2) with a range from 10.2 to 21.93.

It was found that tonsillar hypertrophy grade 3 was the most common while moderately sized adenoids were also the highest in number. Those with small tonsillar size had more symptoms associated with chronic recurrent tonsillitis.

Table 3: Grading and size of Adenotonsilar Hypertrophy in Chidren in KNH

	Frequency	Percent
TH Grade		
Grade 2	32	30.8%
Grade 3	63	60.6%
Grade 4	9	8.7%
AH size		
Large	39	37.5%
Moderate	60	57.7%
Small	5	4.8%

Preoperatively, most children had normal pulmonary pressures (range within 0-24mmHg), with 87% below 20mmHg and 10% between 20-24mmHg. That is 97% were 24mmHg and below. Three percent had pulmonary artery hypertension (2 had mild pulmonary artery hypertension and 1 had moderate pulmonary hypertension).

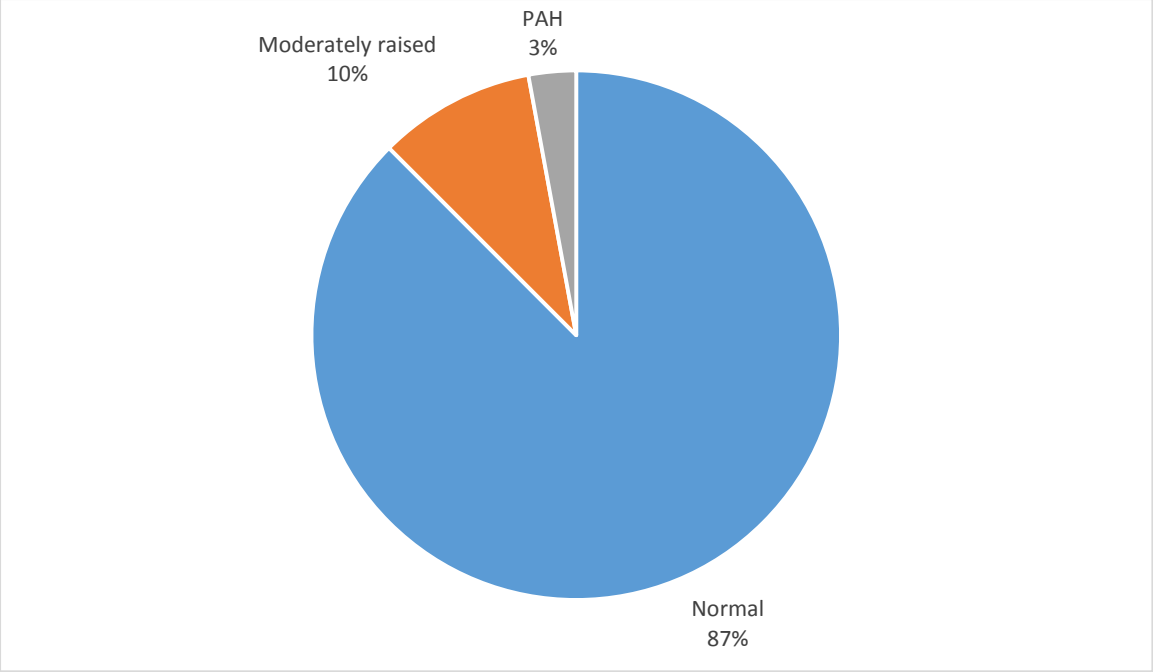


Figure 4: Pre-operative mean PAP in children with Adenotonsilar Hypertrophy in KNH

Mean pulmonary arterial pressures were 9.34 ± 8.52 preoperatively, and 2.91 ± 4.21 in the post-operative period. There was a statistically significant decrease in pressure levels postoperatively and a significant difference in the pre- compared to post-operative values (Paired *t*-test, $p < 0.01$).

Table 4 : Changes in Pulmonary Pressures after Adenotonsilectomy

	N	Mean	SD	95% CI		P value
Mean PAP preoperative	104	9.34	8.52	7.68	10.99	
Mean PAP post-operative	104	2.91	4.21	2.09	3.73	
Difference	104	6.42	7.44	4.98	7.87	< 0.001

The systolic PAP also had significant changes pre and post- operatively.

Table 5 : Changes in Systolic PAP after Adenosilectomy

	n	**Mean**	**SD**	**95% CI**		**P value**
Pre-op systolic PAP	104	13.5	12.50	11.07	15.93	
Post-op systolic PAP	104	4.7	6.12	3.54	5.92	
Difference	104	8.8	10.86	6.66	10.88	<0.001

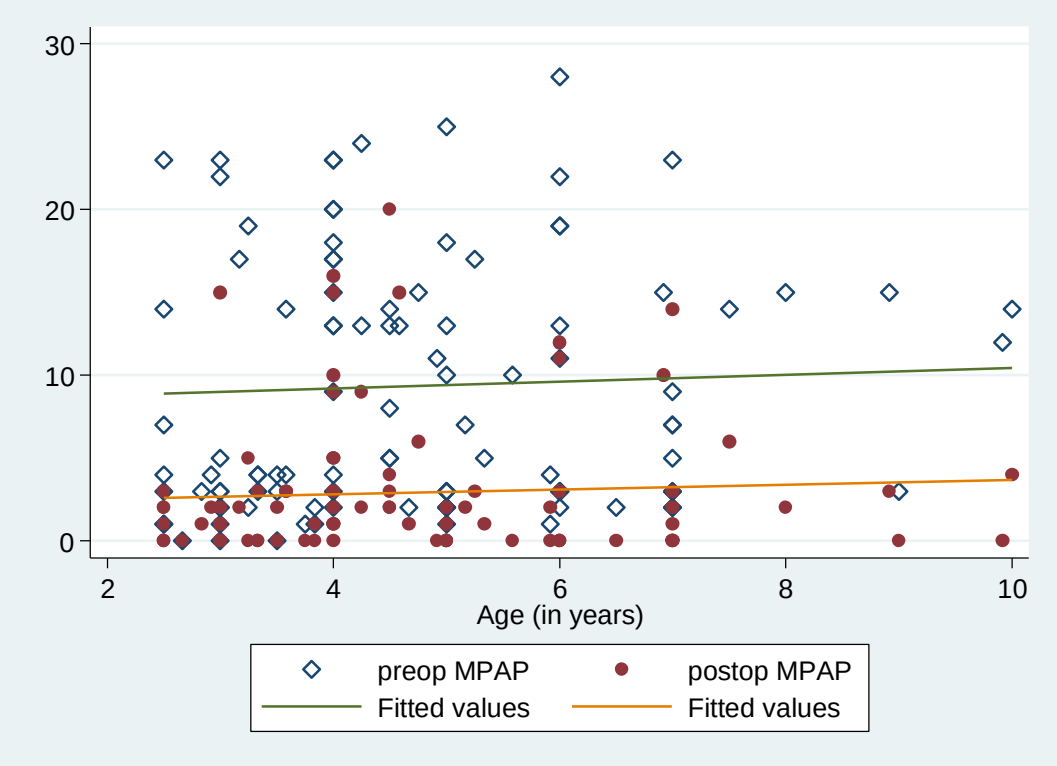
There was no relationship between age and the mean pulmonary arterial pressure changes.

Table 6 : Association of age and the mean pulmonary arterial pressures

	n	Mean MPAP change	SD
Age			
2.5 to 4 years	60	-6.33	8.35
5 to 6 years	29	-6.62	6.98
7 to 10 years	15	-6.40	3.92

This was seen even with evaluation using the Pearson correlation which showed a weak association. Pearson's correlation preoperatively mPAP and age 0.04, p = 0.68, Pearson's correlation postoperatively mPAP and age 0.06, p = 0.57.

Figure 5 : Graph showing Pearson's co-relation between Pre and Post-operative association of age an mPAP



Systolic PAP was highest in the age group 2.5 to 4 years as tabulated below.

Table 7 : Patient age and change in Systolic PAP

	n	**Mean systolic PAP change**	**SD**
Age group			
2.5 to 4 years	60	-9.17	12.90
5 to 6 years	29	-7.97	8.11
7 to 10 years	15	-8.73	5.68

History of symptoms was not statistically significant in association with the change in mean PAP.

Table 8 : History of Symptoms and Change in mean PAP

	Symptom present			
	No	Yes	Difference (95% CI)	P
Snoring	-6.5(±7.5)	-5.0(±6.2)	1.5(-5.8-8.7)	0.726
Sleep disordered breathing with apneic episodes as assessed by an observer	-6.7(±7.7)	-4.2(±4.4)	2.5(-0.7-5.6)	0.146
Mouth breathing	-6.5(±7.5)	-4.0(±5.5)	2.5(-3.0-8.1)	0.432

The symptoms were also not significantly associated statistically with the systolic PAP

Table 9 : History of symptoms and Change in Systolic PAP

	Symptom present			
	Yes	**No**		
	Mean (SD)	**Mean (SD)**	**Difference (95% CI)**	**P**
Snoring	-8.9(±10.9)	-6.0(±8.7)	2.9(-7.2-12.9)	0.631
Sleep disordered breathing with apneic attacks as seen by an observer with apneic episodes	-9.2(±11.2)	-4.8(±5.5)	4.4(0.3-8.5)	0.048
Mouth breathing	-8.9(±11.0)	-4.8(±7.5)	4.2(-3.5-11.9)	0.355

There was no association between tonsillar size and the mean PAP.

The BMI was not significantly correlated with either pre-op mPAP ($p = 0.862$) or post-op mPAP ($p = 0.609$) as seen in Table 11 below.

Table 10 : Spearman's rank co-relation co-efficient for mPAP values and BMI

		BMI	Pre-op mPAP	Post-op mPAP
Statistic				
Coefficient	**BMI**	1		
Coefficient	**Pre-op mPAP**	-0.017	1	
P value		0.862		
Coefficient	**Post-op mPAP**	-0.051	0.676**	1
P value		0.609	<0.001	

Table 11: TH and change in mPAP

	N	mPAP change	SD	P
TH size				
2	32	-6.1	5.9	0.323
3	63	-6.3	8.2	
4	9	-8.6	6.2	

There was also no association between tonsillar size and the systolic PAP.

Table 12 : TH and change in systolic PAP

	n	Systolic PAP change	SD	P
TH size				
2	32	-8.59	8.14	0.668
3	63	-8.41	12.39	
4	9	-11.89	7.66	

There was a statistically significant association between adenoid size and the mPAP with a p value of 0.0002. The systolic PAP was also statistically significant between adenoid size and systolic PAP with a p value of 0.001.

Table 13: AH size and change in mPAP

	n	mPAP change	SD	P
AH size				
Large	39	-10	9.7	0.0002
Moderate	60	-4.5	4.6	
Small	5	-1.2	1.3	

Table 14: AH size and change in systolic PAP

	n	Systolic PAP change	SD	P
AH size				
Large	39	-13.44	14.90	0.001
Moderate	60	-6.37	6.13	
Small	5	-1.20	1.64	

All three children with pulmonary artery hypertension (1 with moderate and 2 with mild), improved significantly post operatively. They all had normal values of pulmonary artery hypertension by the 6th week post-operatively.

It was also noted there was a statistically significant drop in mean PAP between pre and post-operative ECHOs a change from 9.3 to 2.9. a difference of 6.42. This gives a SD 95% and CI of 7.87 and P value <1.00.

Table 15: Changes in Systolic PAP after Adenotonsilectomy

	n	**Mean**	**SD**	**95% CI**		**P value**
Pre-op systolic PAP	104	13.5	12.50	11.07	15.93	
Post-op systolic PAP	104	4.7	6.12	3.54	5.92	
Difference	104	8.8	10.86	6.66	10.88	<0.001

The change in systolic PAP pre and post-operatively was also significant with a P value <0.001. There were three children with pulmonary artery hypertension. One had moderate PAH and required stabilization and management for heart failure prior to Adenotonsilectomy.

He had a large adenoid and a grade three tonsillar size. The second had mild PAH and had TH grade 4 and an AH graded as large. The third had mild PAH with a large AH but a grade 2 TH.

A point to note is that only ten patients did not have a drop in the mPAP from pre to post-operative periods, in fact one of these had an increase in the mPAP post operatively, one had no change, and 8 others had a very slight drop in mPAP and systolic PAP. These ten shall have follow up ECHOs again at 6months.

5.0 CHAPTER FIVE: DISCUSSION

In this study the total was 104 patients. Of these, we found there were 54 males (51.9%) and 50 females (48.1%). This is comparable to all the studies used in the literature review. For example in the study by Yilmaz et al in 2005(19) though the difference in gender was larger 16 girls (31%) and 36 boys (69%) and Koc et al in 2012(1) 17 (63%) males and 10 (37%) females. The age of the patients ranged from 2.5 years to 10 years despite the cut off being from 2.5 years to 12 years, with the patients aged 2.5 to 4 years being the highest with 57.7%. This is in keeping with the age of those mostly afflicted by ATH. As for Marangu et al, the mean age was lower (2.5 years), but her study assessed patients with AH and ATH as well.

The mean age was 4.5 years in this study which differed with Asyenur Pac et al(20) who had 7.3 - 2.9 (with a range of 3-14) years. F.T Orji et al(22) had a similar mean of 4.6 years with a median of 3.4 years. The age group 2.5- 4 years also had the largest change in mPAP as compared to other groups. For FT Orji(22) The mPAP was recorded highest among the youngest participants who were aged <2 years(9 patients in their study), but the differences in the mPAP among the age groups were not significant. That was a finding similar to our study as well. There was however no association between age and mPAP in this study as seen by a weak Pearson correlation.

Symptom duration ranged from 1-9 years with the symptom duration of 1-3 years being the largest percentage. This may also be associated with the largest age group of patients being 2.5- 4 years of age. Again, this is the age most afflicted by ATH. As for E.S. Abd El-Moneim et al(17) the duration of obstructive symptoms in years had a median of 2.2 and a range of 1.2–9.

In the history of symptoms, snoring was the most common followed by mouth breathing and then sleep disordered breathing Martha et al in 2013(21) found that all their subjects presented with snoring and oral breathing while 84.4% had restless sleep. She however went ahead to look for sialorrhoea 60.6%, nocturnal enuresis 12.1% and low school performance 12.1%. in the Marangu et al- mouth breathing was 74.8% mouth breathing and restless sleep- 88.5%(16).

The children in this study presented with snoring, mouth breathing and sleep disordered breathing with apneic attacks as seen by an observer coming to 97.1%, 96.2% and 90.4% respectively. Martha et al in 2013(21) found that all their subjects (i.e. 100%) presented with

snoring and oral breathing while 84.4% had restless sleep. She however went ahead to look for sialorrhoea, nocturnal enuresis, and low school performance which we didn't look into. Marangu et al found mouth breathing was present in 74.8%, while mouth breathing in association with restless sleep was 88.5%.

In this study, Grade 3 TH had the highest percentage and TH Grade 2 was the lowest grade observed while moderate AH had the highest percentage. This is similar to other studies e.g. for FT Orji whose study had a minimum TH of grade 2. However, other studies e.g. Martha et al only found TH of grade 3 and above. For Marangu et al, they had TH of grade 1 to 4 with grade 2 being the highest in number (16). This is perhaps because the age group was mainly of younger patients.

Our study found a statistically significant association between adenoid hypertrophy and mPAP pre-operatively and no significant association between tonsillar hypertrophy and mPAP. This differs from Marangu et al (16) and Martha et al(21) who found no association between the size of Adenotonsilar hypertrophy and the mPAP. However, for F.T Orji et al (22), they found an association between AH and mPAP change.

Preoperatively, most children had normal pulmonary pressures (range within 0-24mmHg), with 87% below 20mmHg and 10% between 20-24mmHg. That is 97% had 24mmHg and below. Three percent had pulmonary artery hypertension (2 had mild pulmonary artery hypertension and 1 had moderate pulmonary hypertension). Yilmaz et al(19) had raised mPAPs of 27 of the 52 subjects which was 51% of the children. It is important to note however that they used 20 mmHg as the cut off, above which was PAH in the study group preoperatively. Koc(1) also had significant drop in the mPAP pre and post- operatively more so observed after 3months than the 1month post- operative period. For Abd El Moneim(17), they found significant improvement in right ventricular output post- operatively. They felt the right ventricular output was the best measure for improvement post-operatively.

There was a statistically significant change in mPAP between pre and post-operative ECHOs as observed in this study with a pre-operative mean of 9.34± 8.52 to a post-operative mean of 2.91± 4.21. There was thus a change of 6.42 in the mean and this was found to be statistically significant with a P value of <0.001. In the study by E.S. Abd El-Moneim et al(17), they found a calculated mPAP ranging from 30.6 mmHg ± 8.8 pre-operatively to 26.2mmHg ± 6.8 postoperatively which was statistically significant with a p value of 0.007. Martha et al also

found a change in systolic PAP with a significant drop in pre and post-operative values which is also similar to our study.

In this study, the systolic PAP was associated with AH but was not associated with TH, symptom duration, age and BMI. This is similar to Martha et al(21) who had systolic PAP return to normal values after surgery. However, in their study, they had a reduced number of patients classified by the systolic PAP and this difference did not reach statistical significance.

5.1 Conclusion

There was an association between adenoid size and duration of symptoms, with the level of mPAP and systolic PAP. There was however no association between tonsillar size, BMI and mPAP. The complaint of snoring was the most common. Pre- operative mPAP and systolic PAP dropped significantly 6 weeks post- operatively except for two patients.

5.2 Recommendation

1. Patients with ATH who have an indication for Adenotonsilectomy should undergo the surgery as soon as possible as the effects on the heart will have resolved by 6weeks post-operatively.
2. Those with physical features of heart failure and a palpable P2 and heart murmurs should have ECHOs pre-operatively.
3. A larger study should be conducted with post- op assessment again at 3 and 6 months
4. A study should be done to assess if there is weight gain post op after ASTS

5.3 Strengths of the study

Since there are changes at 6 weeks for most then a review should be done at 6weeks and this will help to pick those who will need review for a longer duration of time.

5.4 Conflict of Interest

There was no conflict of interest. The ECHOs were funded by KNH Research and Programs.

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APPENDICES

Appendix I:

Appendix 1a) Cohen and Konak Radiologic Adenoid hypertrophy grading system

Air column (mm)	Distance between the posterior border of the soft palate 10mm away from the posterior nasal spine and the anterior curvature of the pharyngeal tonsil border
Air column/ Soft palate ratio (AC/SPR)	Between air column and soft palate
Soft palate (mm)	Thickness of the soft palate measured 10mm away from the posterior nasal spine
Small	Air column/soft palate ratio ≥ 1.0
Moderate	Air column/soft palate ratio ≥0.5 but < 1.0)
Large	Air column/soft palate ratio ≥ 1.0

Appendix 1b): The Brodsky Clinical Tonsilar hypertrophy grading system;

0-25% of the oropharynx (tonsil just outside the tonsilar fossa)
25-50% of the oropharyngeal width
50-75% of the oropharyngeal width
75-100% of the oropharyngeal width

Appendix II: Budget

Consideration	Quantity	Unit Cost (Ksh)	Total Cost (Ksh)
Biostatistician			20,000
Printing paper	40	400	20,000
Ethical committee			2000
Contingency			20,000
Echocardiograms	101(2per patient)	2500	505,500
TOTAL			567,000

Appendix III: General Patient Information and Consent Form

Study number _____

Study Title: -The Effects of Adenotonsilectomy On Pulmonary Pressures as Seen in The ENT Department- Kenyatta National Hospital.

Principal Investigator: Dr. Maria Gachambi Muthoka (Postgraduate student in Otorhinolaryngology and Head and Neck Surgery, University of Nairobi)

Supervisors: - Dr. Peter Mugwe

-Prof. Christine Jowi

-Dr. John Ayugi

Introduction

My name is Dr. Maria Gachambi Muthoka. I am a post graduate student in the University of Nairobi pursuing a Master's Degree in Otorhinolaryngology.

I am doing research on the effects of Adenotonsilectomy on pulmonary pressures as seen in the ENT department. Adenotonsilar hypertrophy has a negative effect on the heart and the lung causing the pressures to rise in the lung, which then affects the right side of the heart. Initially there are no symptoms but in late stages the child may develop heart failure. Not all children have heart failure because there is also an effect based on the size of the adenoid and tonsils. A previous study done in our population found 1 in 5 children with Adenotonsilar hypertrophy had raised pulmonary pressures. This study assesses the effect of Adenotonsilectomy on the raised pulmonary pressures at 1 month and at 3 months. The Adenotonsilar hypertrophy is assessed using history physical examination and an x-ray of the neck involving the space behind the nose. The effect on the heart is assessed using an echocardiograph pre-operatively and at 6 weeks post-operatively. The ECHOs will not be of extra cost to you.

It is not compulsory to participate in this study. If you choose not to participate in the study your treatment will continue as scheduled.

What is involved in this study?

Once consent is granted, the principal investigator will take a medical history from you about your child and an examination of the back of your child's mouth will be performed to assess the size of the tonsils. The principal investigator will also assess the soft tissue x-ray done of your child to assess the size of the adenoids. The principal investigator will record her findings in various documents. Your child will also have an assessment of the heart's function and the lungs using a noninvasive tool called the echocardiogram. The principal investigator will follow up on your progress after the surgery to remove the tonsils and adenoids and repeat an echocardiograph at 6 weeks after surgery.

Are there any risks involved?

There are no risks involved.

Will I be penalized for not participating?

No, participation in this study is voluntary you will receive the same attention and treatment as those who choose to participate.

What benefits will I get if I participate?

There is no direct benefit from participation in this study but the Information obtained from this study will help us understand more about this disease and be able to help more patients in the future.

What about confidentiality?

All the information we obtain from you will be kept confidential.

How much will it cost me?

No extra cost will be incurred

What are my rights as a participant?

Participation in the study is voluntary. Once inducted in the study, you can choose to discontinue at any time.

What do you do with the information you get?

The information will help us improve the care of our patients undergoing chemo-radiotherapy and like any other scientific information, we will seek to share our findings with other doctors in Kenya and the rest of the world.

Are you satisfied with the information given?

If yes and you are willing to participate, please fill in and sign the consent below.

If you have any questions or need further clarifications about the study contact

Consent for the Study

Participation in this study is voluntary

I……..……..……study no…… of ……..…… do hereby consent to be included in this study on:

The Effects of Adenotonsilectomy On Pulmonary Pressures As Seen In The ENT Department- Kenyatta National Hospital.

The nature of the study has been fully explained to me by Dr…… I have not been promised any material gain to participate.

Signed (or thumb print) ……. Date…….

I Dr.....confirm that I have explained to the patient the nature of the study. Date.....signed.....

CONSENT BY PATIENT'S PARENTS/GUARDIAN:

Participation of your child in this study is voluntary

I.....

Study number……. of.....hereby give consent for..... (Name of child) to be included in this study, on The Effects of Adenotonsilectomy On Pulmonary Pressures As Seen In The ENT Department- Kenyatta National Hospital.

The nature of the study has been fully explained to me by Dr…… I have not been promised any material gain to participate.

Signed…….

Date…….

I Dr.....confirm that I have explained to the patient the nature of the study. Date.....signed.....

The principal investigator:

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If you have any questions on your rights as a participant contact the *Kenyatta National Hospital Ethics and Research Committee (KNH-ERC)* by calling *2726300 Ext. 44355*

Ridhaa Maelezo

Nambari ya utafiti

Kiini cha utafiti: - Athari za upasuaji wa kutoa kiungo cha kuzuia maradhi na unyevu wa pumu kilicho nyuma ya mapua na koo na kutafiti shinikizo za pumu kwa mapafu vile huonekana kwa hospitali ya taifa Kenyatta, katika idara ya Masikio, Mapua na Koo.

Mtafiti mkuu: Dkt Maria Gachambi Muthoka (Mwanagenzi wa uzamili katika Chuo Kikuu cha Nairobi anayesoma kuhitimu kama daktari wa Masikio, Mapua na Koo).

Wasimamizi: -Dkt. Peter Mugwe

-Prof. Christine Yuko- Jowi

-Dkt. John Ayugi

Maelezo Zaidi kuhusuutafiti:

Kushiriki kwa utafiti huu ni kwa hiari yako

Lengo la utafiti huu ni kutathmini idadi ya wagonjwa walio katika matibabu ya kutolewa kiungo cha kujikinga na magonjwa kilicho kwa koo na nyuma ya mapua na athari zake kwa mapafu. Aidha utafiti huu utalenga kuona kama upasuaji wa kutoa hivyo viungo husaidia kupunguza madhara kwa kifua. Huu utafiti utasaidia kuongeza ujuzi wetu kuhusu ugonjwa huu ili tuweze kuwasaidia wagonjwa wengine siku za usoni.

Unapokubali kushiriki kwa utafiti huu, utaulizwa maswali mbali kuhusu ugonjwa ulionao. Walioko kwa utafiti wataangaliwa koo na picha ya x-ray itafanywa ilikupima viungo hivyo vimeziba njia ya kupumua kiasi gani. Pia roho na kifua vitaangaliwa kupitia kipimo cha echocardiogram ambacho kitafanywa kabla upasuaji kutoa viungo hivyo.

Wiki sita baada ya upasuaji kipimo cha roho na kifua (echocardiogram) kitafanywa tena na hutahitaji kulipa chochote. Mtafiti mkuu atawasiliana nawe kutoka kabla mtoto afanyiwe upasuaji hadi baada ya kufanyiwa upasuaji.

Madhara: -Hakuna madhara au gharama yoyote itakayo tokana na kushiriki kwako au mtoto wako katika utafiti huu.

Kushiriki ni kwa hiari yako na hautashurutishwa kwa njia yoyote. Una haki ya kukataa kushiriki au kutamatisha ushirikiano wako wakati wowote bila kuhujumiwa.

Hakuna malipo yoyote utakayopata ila shukrani kwa kukubali kushiriki katika utafiti huu. Ujuzi tutakao pata kwa utafiti huu utaweza kusaidia wagonjwa wengine siku za usoni.

Habari yote utakayo toa kukuhusu itawekwa kwa siri. Jina lako au la motto wako halitachapishwa popote bila idhini yako. Hata hivyo, majibu tutakayopata tutayajadili bila kutoa kitambulisho chako au cha motto wako kwa mtu yeyote.

Ikiwa una swali ama ungetaka kupata maelezo zaidi kuhusu utafiti huu, wasiliana na:

Kibali Cha Utafiti

Kushiriki kwako katika utafiti huu ni kwa hiari yako. ……..……

wa……. nambari ya utafiti…… nimekubali kuhusishwa katika utafiti huu unaoangalia: Athari ya upasuaji wa kutoa kiungo cha kuzuia maradhi kilicho nyuma ya mapua na koo na kuangalia shinikizo za pumu ya mapafu vile huonekana kwa Hospitali ya Taifa Kenyatta, katika idara ya Masikio Mapua na Koo.

Nimekubali baada ya kusoma na kufahamishwa na Dkt…… hakuna malipo nitapewa.

Sahihi……. Tarehe…….

Mimi Dkt ……. Nadhibitisha kuwa nimemwelezea mgonjwa yote yanayo husika na utafiti huu. Tarehe…….

Kibali Cha Utafiti Cha Watoto

Kushiriki kwa motto wako katika utafiti huu ni kwa hiari yako. Mimi ……..…… ……..

Mzazi / msimamiziwa ……. Nambari ya utafiti…… nimekubali motto wangu kuhusishwa katika utafiti huu unaoangalia

Athari za upasuaji wa kutoa kiungo cha kuzuia maradhi kilicho nyuma ya mapua na koo na kuangalia shinikizo za pumu ya mapua vile huonekana kwa Hospitali ya Taifa Kenyatta, katika idara ya Masikio Mapua na Koo.

Nimekubali baada ya kusoma na kufahamishwa na Dkt.…… hakuna malipo nitapewa.

Sahihi……. (mzazi / msimamizi) Tarehe ……

Mimi Dkt ……. Nadhibitisha kuwa nimemwelezea mgonjwa yote yanayo husika na utafiti huu.

Tarehe…….

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Ikiwa una swali kuhusu haki zako katika utafiti huu wasiliana na *Kenyatta National Hospital Ethics and Research Committee (KNH-ERC)* Nambari ya simu *2726300 Ext. 44355.*

Appendix IV: Questionnaire

EFFECTS OF ADENOTONSILECTOMY ON PULMONARY PRESSURES AS SEEN IN THE ENT DEPARTMENT

SERIAL NO: …….

BIODATA: …….

Proforma number …….

Gender: Male Female

Age: ……..

HISTORY

Duration of symptoms…….

History of snoring NO…… YES……

History of sleep disturbance as witnessed by an observer: NO…… YES……

History of mouth breathing NO…… YES……

CLINICAL EXAMINATION:

Weight (Kg)…….

Height (Meters)…….

BMI …….

Tonsilar hypertrophy Brodsky grading I…. II…. III…. IV….

Adenoid hypertrophy (lateral post nasal apace radiograph) Cohan and Konak grading

X-ray number…….

Size of air column (mm)……

Size of soft palate (mm)…….

Air column/ soft palate ratio…….

SMALL……. MODERATE……. LARGE……..

SPO_2.....

Respiratory Rate (breathes per minute) tachypnea YES…… NO……

Heart rate (beats per minute)- Tachycardia YES……. NO…….

Jugular venous distension YES…… NO……

Hepatomegaly YES…… NO……

Palpable P2 YES……. NO……

Heart murmurs YES…… NO…….

Heart murmur SYSTOLIC…. ….. DIASTOLIC…….

Heart murmur location. …….

ECHOCARDIOGRAPHY:

Mean PAP(mmHg) …….

Systolic PAP (mmHg) …….

Size of right ventricular chamber …….

Inter-ventricular septum shape ……

Right ventricular ejection fraction ……

Valvular insufficiency …...…….