PREVALENCE OF OTITIS MEDIA WITH EFFUSION IN CHILDREN WITH OBSTRUCTIVE ADENOID DISEASE COMPARED WITH NORMAL CONTROLS AT KENYATTA NATIONAL HOSPITAL.

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A study submitted in part fulfillment of the requirements for the degree of Master of Medicine in Ear, Nose and Throat- Head and Neck Surgery, at the University Of Nairobi.

DECLARATION

This dissertation is my original work and has not been presented for the award of a degree in any other university.

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APPROVAL BY SUPERVISORS

This dissertation has been presented with our full approval as supervisors.

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DEDICATION

To my beloved family: My parents Mr and Mrs Kiama Mbuthia,my wife Grace Ndungu, my children Ciru Mwaniki and Kiama Mwaniki.

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I would like to express my sincere appreciation to:-

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TABLE OF CONTENTS

CONTENTS

LIST OF TABLES

LIST OF FIGURES

ACRONYMS AND ABBREVIATIONS

OAD……Obstructive adenoid disease

OME……Otitis media with effusion

ENT-HN……Ear, Nose, Throat, Head and Neck

KNH……Kenyatta National Hospital

MEE…… Middle ear effusion

CSOM…… Chronic suppurative otitis media

TVP……Tensor veli palatini

ET……Eustachian tube

PET……Pressure equalization tubes

STUDY DEFINITIONS

1. **Clinician diagnosed adenoid hypertrophy -** Adenoid hypertrophy diagnosed by any clinician at the Kenyatta National Hospital.

2. **Radiologically confirmed adenoid hypertrophy-**Adenoid hypertrophy documented on a lateral neck radiograph by any radiologist at the Kenyatta National Hospital

ABSTRACT

Background. Otitis media with effusion (OME) is a common otological disease encountered in children**.** Diagnosis in children is often delayed as they cannot complain of hearing loss and this may result in speech impairment, inattention, poor performance in school and behavioral problems.

Objectives. To assess the association between OME and Obstructive adenoid disease (OAD) in children scheduled for adenoidectomy at Kenyatta National Hospital (K.N.H).

Study design. This was a Case control study carried out in children aged 1-8 years in the ENT and surgical outpatient departments of KNH. The study group had clinical and radiological features of chronic obstructive adenoid disease and the control group had no history suggestive of obstructive adenoid disease. Eligible patients were consecutively recruited into the study between June and September 2013. The patients were evaluated for symptoms, otoscopic findings and tympanometry. Lateral neck radiograph measurements was done for children in the study group.

Results: The prevalence of OME in children with adenoid hypertrophy at KNH was 67.3% and in the control group was 15.4 % (95% CI 4.4 to 29.3).

Conclusion and Recommendations*:* Six in every 10 children with clinician diagnosed and radiologically confirmed adenoid hypertrophy at KNH had OME. Clinical screening tympanometry evaluation and follow up is vital in preventing sequel associated with OME.

INTRODUCTION

Otitis media with effusion (OME) is an important and common condition in pediatric age group. Other terms commonly used to refer to the same process include secretory otitis media, non suppurative otitis media, serous otitis media and glue ear. Following a discussion at an international symposium the terms OME and middle ear effusion (MEE) were adopted by consensus (1).

OME was previously considered to be bacteriologically sterile. However positive bacterial cultures have been demonstrated in 40 percent of middle ear fluid. Streptococcus pneumonia and haemophilus influenza account for the majority of cases (2).

It is a common practice among otorhinolaryngologists to apply adenoidectomy as part of the treatment of medically resistant OME. Although some literature associates enlarged adenoid with OME, there are some studies questioning this relationship.

Although there are a large number of prevalence studies of OME in general population of children, there has been less research on its prevalence in children having adenoidal obstruction.

BACKGROUND

Otitis media with effusion

OME is defined as fluid in the middle ear without signs or symptoms of acute ear infection. OME is one of the commonest chronic otological conditions of childhood. Two third of children have had at least one episode of OME by the age of 3 years and in one third of them it is asymptomatic (3). Incidence varies according to geographical and race variation. The prevalence of OME is higher in Native Americans particularly Navajo and Eskimo people than in other races. The reason for the higher prevalence in these populations has been thought to be due to anatomic differences of skull base and Eustachian tube, biologic susceptibility and difference in socioeconomic status (4).Clinically the patient may present with mild to moderate hearing loss. Although the hearing loss is initially temporary and disease may resolve

by itself in a significant percentage of patients, the disease may continue to cause problems in 5 to15 % of children with persistent or progressive hearing loss, tinnitus, otalgia, and chronic suppurative otitis media (CSOM) (5).

Epidemiology

The prevalence of OME is bimodal with the first and largest peak of approximately 20% at 2 years of age with a second peak of approximately 16% at around 5 years of age (6). The prevalence rate then sharply declines in children older than 6 years. There are racial differences in prevalence of OME (4). A Nigeria urban population, the prevalence of OME in children aged 5-6 years using tympanometric studies was found to be 8% (7).The prevalence of otitis media with effusion in children in a black rural community in Venda (South Africa) is about 3.8% (8).

Studies done in Malaysia, report an overall prevalence rate of 13.8% of OME in preschool children aged between 5 and 6 years old and a prevalence of 7.26% in primary school children 7 to 12 years (9). Another study done in Malaysia found a higher prevalence in children in urban areas than rural areas (10). Tympanometric studies showed prevalence rates of 50% in 5-7 year age group in the United Kingdom (11), 30% in Danish children 2-4 years (12) and 26% in Danish 7 years (13).No significant difference exists between the sexes in terms of incidence or prevalence, although some findings suggest that males are more frequently affected than females (14).

Aetiology

The four main causes are Eustachian tube dysfunction, middle ear gas composition, nasopharyngeal disproportion and altered mucociliary system.

Eustachian tube dysfunction is the most important factor. The Eustachian tube has three physiologic functions with respect to the middle ear. These are protection of middle ear from nasopharyngeal secretion and pressure; clearance of middle ear contents and ventilation of

middle ear. It opens involuntarily during swallowing, yawning and valsalva maneuvers. The result of any tubal dysfunction is a decrease in intratympanic pressure (15).

In children the Eustachian tube is shorter and is predisposed to reflux. Its lumen being smaller is more vulnerable to obstruction by inflamed mucosa (secondary to allergy or infection). It lies more horizontally in infants with decreased efficiency in drainage of secretion. In addition, the cartilage is more compliant and collapses readily with negative pressure. The Eustachian tube achieves adult stiffness at about 6 years of age.

Children with anatomical defects such as cleft palate or craniofacial disorders have a higher incidence of OME (16-18). For children with cleft palate; the underlying defect causing tubal dysfunction is an abnormal mode of action of the tensor palati muscle. This is thought to be due to failure or abnormal insertion of the tensor veli palatini (TVP) muscle to the lateral paratubal cartilage resulting into failure of Eustachian tube to open (19).

Tubal dysfunction may result either from skull base abnormalities or where there are anatomical variations in the nasopharynx (20). These may be defined in relation to differences in the angle subtended by the floor of the anterior cranial fossa and basisphenoid with the level of the hard palate. Consequently otitis media with effusion is more common in craniofacial abnormalities such as Down's and Hurler's syndromes.

It is believed that with an increase in the vascularity of the middle ear cleft due to inflammation, there is an increase in gas diffusion into the blood, resulting in a decreased pressure in the middle ear cleft. Negative pressure in the middle ear cavity in turn results in serous fluid accumulation in the middle ear and retraction of the tympanic membrane (21).

Nasopharyngeal disproportion is also an important factor in the pathogenesis of OME. Children with adenoid hypertrophy and craniofacial disproportions have been shown to have increased risk of OME (22).

Jeans et al (23) showed the growth of the adenoids outstrips that of the nasopharynx between the age of 3 and 5 years of life with a reduction in the nasopharyngeal airway. The nasopharynx beyond 5 years starts to grow faster, while the adenoid size remains relatively unchanged.

Mucocilliary dysfunction can occur due to infection (nose, sinus, postnasal space, tonsils, and pharynx), allergy, immunological factors, surfactant deficiency, ultrastructural changes in cilia, fibrocystic disease, and hormonal factors among other factors (24).

Otitis media with effusion occurs more commonly with the immotile cilia syndrome, primary ciliary dyskinesia and particularly with that form of the condition which constitutes the Kartagener's syndrome (25).

Several risk factors have been associated with OME including previous acute otitis media, hereditary, parental smoking, attending day care centre's, bottle feeding and autumn season (26,27).

Diagnosis

Diagnosis can be made by taking history, otoscopic examination and audiological evaluation. Hearing loss is the most common presenting symptom. As children cannot complain of hearing loss, diagnosis is usually delayed for months or even years, resulting in impairment of speech, inattention, poor performance at school, psychosocial,cognitive and behavioral problems (28, 29). Older children and adults may complain of deafness, fullness in ear and tinnitus. On otoscopic examination, tympanic membrane is often cloudy with impaired mobility (30), and an air-fluid level or bubble may be visible in the middle ear. Pneumatic otoscopy combined with tympanometry improves the accuracy of diagnosis because many abnormalities of the eardrum and ear canal that might cause an abnormal tracing can be visualized. Determining the presence of obstructing cerumen in the canal, perforation or ventilation tubes in the tympanic membrane and characteristics of the tympanic membrane (e.g., color, mobility, position, and translucency) are helpful in correlating tympanometry findings with clinical disease. Congenital fixation of ossicular chain results to a non-progressive hearing loss with normal ear drum. Pneumatic otoscope and tympanometry are complementary tests and accordingly pneumatic otoscopy recommended as the primary test for the diagnosis of OME and tympanometry as a confirmatory test (31).Tympanometry is particularly useful in small children whose external auditory canals may be too small or too collapsible to permit adequate

visualization of the tympanic membrane. However, in children younger than 7 months, tympanometry is unreliable because of excessive compliance of the external auditory canal (32, 33). Tympanogram can be divided into four types: Type A: +200 to -99 mmH2O; Type B:flat traces without well defined maximum; Type C1:-100 to -199mmH2O and; Type C2:-200 to -400mmH2O (34, 35).(See Appendix 1).Type B trace can have a sensitivity and specificity of up to 93% (36) for detecting OME among cooperative children.

Tympanocentesis can serve as both a therapeutic procedure and a diagnostic procedure.The therapy consists of the removal of a middle ear effusion (MEE). However this form of therapy does not address the root cause of the effusion and is at best palliative.

The criterion standard for documentation of a middle ear effusion is myringotomy, which has the advantage of increased exposure and better suctioning relative to tympanocentesis. The primary disadvantage is a larger incision with a greater chance of persistent perforation or otorrhea.

Management

Management can be divided into conservative, medical and surgical management.

Conservative management includes risk factors modification and use of valsalva maneuvers.

Medical management comprises of use of antibiotics and steroid intranasal sprays. OME is a bacteria disease and is known to contain viable, pathogenic bacteria and this make antimicrobial therapy a logical choice (37). Several studies using various antibiotics combination showed that the clearance rates in the treated cases were significantly greater than in the control groups (38, 39, 40, 41).

For OME persisting more than 90 days in spite of adequate medical therapy, surgical treatment may be recommended.After a decision is made to treat the child surgically, a second decision about the type of procedure must be made. Myringotomy, adenoidectomy, tympanostomy tubes, and even tonsillectomy have been advocated.

Adenoid hyperplasia

The adenoid (pharyngeal tonsil) forms the uppermost part of the ring of lymphoid tissue surrounding the oropharyngeal isthmus, described in 1884 by von Waldeyer. It is located on the upper posterior wall of the nasopharynx adjacent to the choanal and auditory tube ostium. The adenoid is covered by respiratory epithelium that is rich in goblet cells and is plicated into numerous surfacefolds. Abundant lymphocytes are found within, especially on the crests of the folds.

The size of adenoids varies from child to child and also in the same individual as he/she grows. In general normal adenoids attain their maximum size between ages 3 and 7 years and then regress (1).The growth of the soft tissues of the postnasal space representing the adenoids outstrips growth of the nasopharynx from 3 to 5 years of age with the resultant reduction in the nasopharyngeal airway (22). Subsequently, growth of the nasopharynx increases while soft tissues remain relatively unchanged and thus the airway increases (42).

Clinical evaluation of adenoid size in young children is very difficult. History reported by parents of nasal obstruction, mouth breathing, nocturnal drooling and speech disorders suggest adenoid enlargement (43). Adenoids are not visible at direct inspection through anterior rhinoscopy. The value of posterior rhinoscopy, besides the technical difficulty in approaching young children, is controversial. Objective measures of adenoid hypertrophy are useful to provide information that may help deciding the need of surgery and subsequent outcomes evaluation and these include lateral neck x-ray and nasal endoscopy.

Cohen, Konai and Scott (44) support the idea that lateral x-ray of nasopharynx is an effective method to evaluate children with suspected adenoid hypertrophy, however, x-rays have some disadvantages, as they consist of irradiation on the child, the lack of standardization in technique and film evaluation, the two-dimensional image of nasopharynx rather than a three dimensional structure.

Wormald et al (45) report that, in doubtful cases, nasal endoscopy under local anesthesia provides a definitive evaluation of the nasal cavity and nasopharynx state.Difficulties involved

in submitting non-collaborative young children to endoscopy is a disadvantageous feature of this procedure.

Linder Aronson et al (46) stated that lateral radiographs provide a simple method of assessing the outline of nasopharynx and the soft tissue in relation to airway.

Obstructive adenoid disease and otitis media with effusion

Adenoids may become chronically infected and act as reservoir in upper airway and middle ear infection (47, 48). Other studies attribute the effect of adenoid to their size especially size in relation to nasopharyngeal dimension. Enlarged adenoids lead to Eustachian tube displacement or obstruction (49, 50). It has been demonstrated by radiological technique and pressure studies that adenoid can mechanically obstruct the Eustachian tube opening affecting middle ear aeration and adenoidectomy helps by relieving the obstruction (48, 51).

Adenoid tissue can also impede mucociliary drainage of the middle ear by the way of non ciliated metaplastic epithelium and fibrosis of connective tissue (52).

Eustachian tube dysfunction related to the adenoids may also have an allergy-related functional component. Allergic inflammation has been described for middle ear effusion (53, 54, 55), and some studies have reported that mast cells increase and allergic mediators release in adenoids as well. Berger et al (56) demonstrated large numbers of mast cells in the adenoids. These are capable of binding IgE and releasing histamine and other inflammatory mediators on antigen challenge. Adenoidectomy may reduce a potential source of inflammatory mediator from the vicinity of the Eustachian tube. However, in a study based on serum IgE levels, Maw (57) was not able to show any difference of outcome in cases with otitis media with effusion following treatment with adenoidectomy or by insertion of a ventilation tube, whether atopy was present or not.

Pulec et al (58) attribute the effect of adenoid to be due to lymphatic obstruction by inflamed and enlarged adenoids.

REVIEW OF LITERATURE

Many studies have been done in the past regarding OME and role of adenoid hyperplasia. Most of these studies assessed the cure rate of OME following adenoidectomy. Very few studies on prevalence of OME in adenoid hyperplasia exist in literature.

Gates et al (59) in a systematic review of three randomized controlled studies showed the efficacy of adenoidectomy in the treatment of chronic secretory otitis media. All three studies showed that the effect of adenoidectomy was independent of adenoid size. Prospective randomized studies by Maw (56, 60) showed that adenoidectomy alone produced significant clearance of middle ear effusion in 31.1% of cases of OME at 6 months and at 41.7% at 1 year judged by pneumatic otoscopy.

Van den Aardweg MT et al (61) conducted a systematic review of fourteen randomized controlled trials (2712 children). The effectiveness of adenoidectomy in children with otitis media was evaluated. The study showed a significant benefit of adenoidectomy as far as the resolution of OME is concerned.

Wright et al (62) in prospective survey collected data on 273 consecutive adenoidectomy patients. At the time of surgery, adenoid position in relation to the Eustachian tube (ET) orifice was recorded as well as concurrent procedures performed e.g. pressure equalization tubes (PET). Sixty percent of patients undergoing simultaneous PET insertion were found to have laterally hypertrophic adenoid tissue encroaching upon the ET orifice versus only 22% for those undergoing adenoidectomy alone. Takahashi et al (63) performed transnasal endoscopy of pharyngeal opening of Eustachian tube in 155 ears with OME and found compression of orifice by adenoid tissue in 52%.Bluestone and Berry in a study of 23 patients demonstrated radiologically retrograde obstruction of eustachian tube opening in relation to OME and enlarged adenoids (64).

Hibbert and Stell (65) in a study compared radiologically the size of adenoids in a series of children with OME with age and sex matched children who had sustained head injury. There was no significant difference in the size of adenoids in the two series of children.

A prospective study was carried out at a teaching hospital in Nepal from 15th December 2005 to April 2007. Study group comprised of 32 children with otitis media with effusion and control group of 28 children with clinically normal ear and nose. Rigid nasal endoscope was used for grading of adenoid in study and control group. In the study group 13 out of 32 children had grade 4 adenoid hypertrophy. This grade 4 adenoid hypertrophy was found to be statistically significant in children with otitis media with effusion ($P < 0.0002$). In control group 15 out of 28 had grade 1 adenoid hypertrophy which was significant in the same group ($P < 0.002$)(66).

Studies done by Liu and Sun as well as Ito and Rodger found adenoids to be hypertrophied in OME and middle ear diseases (67, 68, 69). The evaluation of adenoid sizes in these studies was not done using the adenoidal nasopharyngeal ratio and therefore was subjective. Hans et al in a study of 343 children with adenoid hypertrophy found a relationship between nasal symptoms of adenoid hypertrophy and OME (70).Pan H et al (71) conducted a prospective clinical study from February 2004 to October 2004 to evaluate the correlation between adenoidal-nasopharyngeal ratio and tympanogram/eustachian tube function in children. A total of 120 children with adenoids hypertrophy and 20 normal children were enrolled in the study. They found that the Middle ear pressures were negatively related to the AN ratio ($r = 0.41$, $P < 0.05$). The eustachian tube function of the children with adenoids hypertrophy was worse than the normal and the relation between the eustachian tube function and the AN ratio was not statistical difference.

Orji FT et al (72) in a prospective clinical study the incidence of OME among adenoidal patients was compared with its incidence in normal control. Of the adenoidal group 35% were found to have OME using type B tympanogram where as in the control group only 7 % were found to have OME.

Dong-dong and WANG Wu-Qing (73) in a study of 207 patients who were to undergo adenoidectomy 69.1% were found to have OME by tympanomery.

Farhad J ea al (74) found an incidence of 36.7% in children aged 3-12 years with clinical and radiological evidence of adenoid hypertrophy

STUDY JUSTIFICATION

Adenoid hyperplasia and OME are some of the commonest problems encountered by otolaryngologist. It is common practice among otolaryngologists to apply adenoidectomy as part of the treatment of medically resistant otitis media with effusion. Although some literatures associated adenoid hyperplasia with OME, there have been some studies questioning this relationship.(56,59,60,61,62,63,64,65).

In Kenya we neither have prevalence studies of OME in general population of children, nor its prevalence in children having adenoidal obstruction.

Because of the possible association between OAD and OME, and the known adverse effects of OME, the results of this study will inform the otorhinolaryngologist of need to look for possible presence of OME in children with OAD and may as well influence future approach to management of patients with OME and OAD in KNH.

STUDY QUESTIONS

Is there a difference in prevalence of OME between children with obstructive adenoid disease and those without?

NULL HYPOTHESIS

There is no difference in prevalence of OME in children with obstructive adenoid disease compared with those without.

AIMS AND OBJECTIVES OF THE STUDY:

Broad objective

To assess the association between OME and OAD in children scheduled for adenoidectomy at K.N.H.

Specific objectives

1. To determine the prevalence of OME in children with obstructive adenoid disease.

2. To determine the prevalence of OME in children without obstructive adenoid disease.

3.To determine the clinical and radiological factors associated with OME in children with obstructive adenoid disease.

MATERIALS AND METHODS

Study design-Case control study.

Study setting-This study was carried out within the ENT department and the surgical outpatient department of KNH.

Study population

The children were divided into two groups;

1. Study group.

2. Control group.

Study group

Inclusion criteria:

Children aged between 1 and 8 years with clinical and radiological features of chronic obstructive adenoid disease as the only cause of upper airway obstruction and scheduled for adenoidectomy.

Exclusion criteria:

- History of previous adenoidectomy.
- Nasopharyngeal tumor/mass other than AH.

- Neurological abnormalities. (E.g. Cerebral palsy)
- Genetic syndromes with craniofacial abnormalities. (E.g. Down syndrome)
- Other causes of airway obstruction (deviated septum, nasal polyposis, gross turbinate hypertrophy
- Active ear discharge.
- Cleft palate.
- Mucociliary disease.
- Parent/Guardian's refusal to consent

Control group

Inclusion criteria:

This comprised children aged between 1 and 8 years seen at dental and surgical outpatient clinics of KNH with no history suggestive OAD.

The children were matched for age and sex.

Exclusion criteria:

- Symptoms suggestive OAD.
- Cleft palate.
- Craniofacial abnormalities.
- Mucociliary disease.
- Parent/ Guardian refuse to consent.

Sample size

The main aim of the present study was to assess the role of OAD in the pathogenesis of OME by comparing the prevalence of OME between patients with OAD and those with no obstruction. There was no data on this subject in Kenya but a study in Nigeria (72) showed that the prevalence of OME in OAD and those with no obstruction were 35% and 7%,

respectively. Using this prevalence as the basis, the sample size was calculated using Kirk and Sterne (2003) formula below (75):

$$N = \frac{\left[u\sqrt{\pi_1(1-\pi_1)+\pi_2(1-\pi_2)} + v\sqrt{(\pi_1+\pi_2)\left(1-\frac{(\pi_1+\pi_2)}{2}\right)} \right]^2}{(\pi_1-\pi_2)^2}$$

Where $\pi_1 = 0.35$; $\pi_2 = 0.07$; N = minimum number of children in each group; μ = one-sided percentage point of the normal distribution corresponding to 100% less the power (95%) in this case 1.28 and; ν = percentage point of the normal distribution corresponding to the significance level of 5% (i.e. 1.96).

This formula gives a minimum (N) of 52 children in each group and hence a total of 104 children.

Sampling Method

All children who satisfied the inclusion criteria and had no exclusion criteria were enrolled into the study through consecutive sampling method.

PROCEDURE

Ethical approval was granted by the Kenyatta National Hospital Ethics and Research Committee .

Parents/ legal guardians of potential participants were approached and requested to participate in the study. A written informed consent was obtained (Appendix2). Exclusion criteria were validated during history taking and physical exam.

One hundred and four children were enrolled in the study, 52 children in the study group and 52 children in the control group. All the 104 children underwent tympanometry.

History

The principal investigator took pertinent history from the caregivers of the children recruited in the study on an individual basis. This included demographic data, history of chronic nasal

obstruction associated with snoring, and/or mouth breathing, and/or obstructive breathing during sleep and/or sleep disturbance. Otological history included history of otalgia, hearing loss and ear discharge.

Physical examination

The physical examination entailed a general exam and ENT evaluation with emphasis on otological examination. Otological examination involved assessing for any abnormality or disease in the external auditory canal and the middle ear. This was conducted by the principal investigator for each child recruited in the study.

Investigations

Radiologic findings

During the study period children recruited in the study group had lateral neck radiograph done as part of their routine workup at the patients cost. Only lateral neck radiography performed at the KNH radiology department was used because of standardization. Adenoid nasopharyngeal ratio (ANR) was measured by the principal investigator using a standardized technique proposed by fujioka et al (76) as shown in Figure 1 below. To make the measurements more objective, the AN ratio measurements obtained were graded using Sade J (1979) method as follows (77):

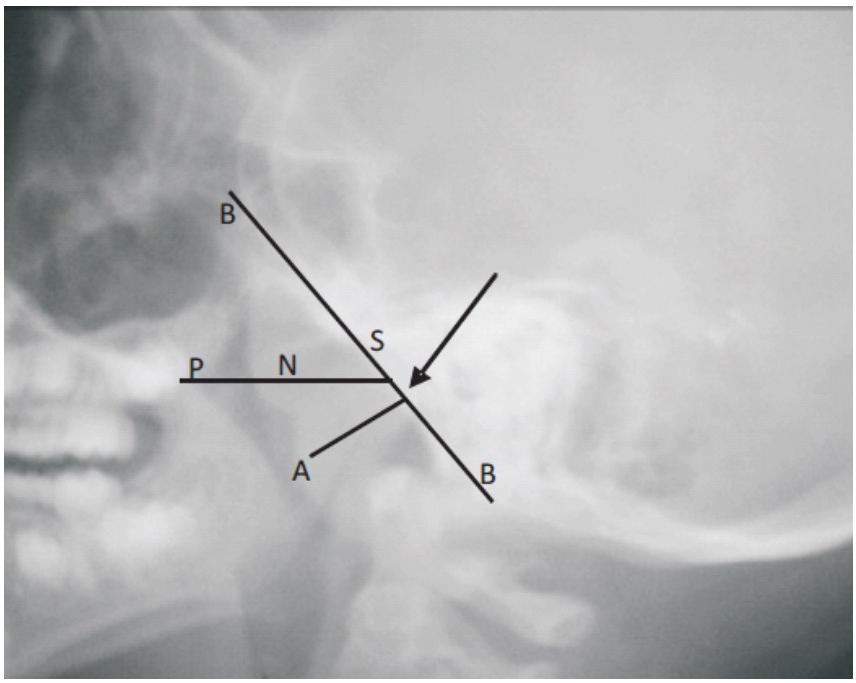
Grade 0 (0.0 – 0.25) no adenoid enlargement

Grade I (0.26 – 0.50) minimal enlargement

Grade II (0.51 –0.75) moderate enlargement

Grade III (0.76 – 1.00) gross enlargement

Figure 1: Lateral neck radiograph measurements as proposed by fujioka et al (76)



Photograph of postnasal x-ray of a patient illustrating the measurements for calculation of AN ratio. Line' B' is tangential to the basiocciput. The adenoidal measurement 'A' is obtained by drawing a perpendicular line to B at the point of maximal adenoidal tissue. The nasopharyngeal measurement 'N' is made between the posterior border of the hard palate and the antero-inferior aspect 'S' of the spheno-basiccipitalsynchondrosis (black arrowhead). When the synchondrosis is not visible, point 'S' is determined as the point on the anterior edge of the basiocciput which is closest to the intersection of the lines A and B

Tympanometry

Tympanometry was performed for both study and control groups. This was done by a qualified audiologist in the department of ENT at K.N.H. The machine model used was interacoustics impedance audiometerAT235. Serial number. 745338 in the department of E.N.T at K.N.H (see Appendix 3). The equipment use a probe tone frequency of 226 Hz, a probe tone intensity of 85 db SPL +/_ 1.5 db, compliance range of 0.1 to o.6ml and a positive and negative pressure sweep between +300 and 600 dapa.

QUALITY CONTROL

The patient proforma was pre tested before commencement of data collection and appropriate modification made. The patient history and physical examination was only done by the principal investigator who also entered the findings in the patient proforma.

Audiometric tests was done by an appointed qualified audiologist in both groups. Evaluation of the lateral cephalometric radiogram was done by the principal investigator. These measures were used to exclude interpersonal bias.

STUDY LIMITATIONS

It is possible that OAD and OME may be caused by similar etiological mechanisms.

ETHICAL CONSIDERATIONS

a. **Permission:** Permission to undertake this study was sought from Kenyatta National Hospital Scientific and Ethics Committee. A letter of protocol approval was obtained prior to the commencement of the study.

b. **Risks**: No invasive or experimental investigations or treatments were employed in this study.

c. **Benefits:** The study participants had tympanometry done by the investigator and significant findings were recorded in the patients file for follow up.

d. **Confidentiality**: Subject confidentiality was strictly held in trust by the investigator. The study protocol, documentation, data and all other information generated were held in strict confidence. No information concerning the study or the data was released to any unauthorized third party. Clinical information was released after permission by the subject when necessary to allow monitoring by ENT team.

e. **Informed consent:** Informed consent was obtained from the caregivers after explaining to them the objective of the study. The consent form described the purpose of the study and the procedure to be followed. The investigator conducted the consent

discussion and checked that the parent/caregiver comprehended the information provided and answered any question about the study. Consent was voluntary and free from coercion. No penalties were meted to patients who declined to join the study and study subjects had the option of refusing to participate or withdraw from the study.

DATA ANALYSIS

Data collection was confidential using a structured questionnaire and proforma tool. Filled questionnaires were solely utilized for this study and subsequently stored safely at the end of the study after entering the data in a Microsoft Access 2007 database. Data analysis was performed using Statistical Package for Social Sciences (SPSS). The population was described using age and sex summarized into mean (SD) and percentages respectively. The cases were further described using symptoms presented as proportions and the duration of symptoms presented as mean number of months. Prevalence of OME was calculated and presented as a proportion. Associations with categorical variables between cases and controls were tested using Chi square test with odds ratio to estimate risk. In addition, the differences in prevalence of OME across age groups and sex were also tested with Chi square test. Student- t test was used to compare mean duration of symptoms. All statistical tests were significant at a p value of 0.05 or less.

RESULTS

Patient characteristics

We had a total of 52 children in each group, 36 (69.2%) males and 16 (30.8%) females giving a male to female ratio of 2.25:1.The age range was from 12 months to 48 months with mean age of 26.0 and 24.1 in study and control groups respectively with most common age group being12-24 months 30 children (57.69%). Age group 25 - 36 months had 18 children (34.61%) and only 4 children (7.69%) in the age group 37 to 48 months.

Table 1: Patient characteristics

Variable	**Study group**	**Controls**	**P value**
Age: mean (SD)	26.0 (9.5)	24.1 (8.7)	0.302
Age groups			
12 – 24 months	30 (57.69%)	30 (57.69%)	
25 – 36 months	18 (34.61%)	18 (34.61%)	
37 – 48 months	4 (7.69%)	4 (7.69%)	
gender			
Male	36 (69.2%)	36 (69.2%)	1.000
Female	16 (30.8%)	16 (30.8%)	

Prevalence of OME

Out of all the 52 children with OAD 35 children had OME as compared with 8 children out of 52 in the control group giving an overall prevalence of 67.3% in the study group and 15.4% in the controls (95% CI 4.4 -29.3) as depicted in table 2 below.

Table 2: Prevalence of OME in study group and controls

Variable	Study group	Controls	OR (95% CI)	P value
OME				
Present	35 (67.3%)	8 (15.4%)	11.3 (4.4-29.3)	<0.001
Absent	17 (32.7%)	44 (84.6%)	1.0	

Patient characteristics associated with OME

Table 3 below depicts the prevalence of OME by age group. Children with OME in the study group were younger than those without although this was not statistically significant (p=0.279). However in the control group, OME was found in the older children but was not statistically significant (p =0.708). In the study group children below 24 months were 1.9 times more likely to have OME compared to those above 24 months OR 1.9 (0.6-6.2), p = 0.279 while in the control group children below 24 months were less likely to have OME compared with those above 24 months OR 0.7(0.2-0.3), p = 0.708. In both groups this was not statistically significant.

Table 3.Prevalence of OME by age group

Variable	Study group				Controls			
	OME Present (%)	No OME (%)	OR (95% CI)	P value	OME Present (%)	No OME(%)	OR (95% CI)	P value
Age group								
<24 months	22 (73.3)	8 (26.7)	1.9 (0.6-6.2)	0.279	4 (13.3)	26 (86.7)	0.7 (0.2-3.1)	0.708
>24 months	13 (59.1)	9 (40.9)	1.0		4 (18.2)	18 (81.8)	1.0	

Table 4 below shows propotions of children with OME according to gender. The odds of OME in children with OAD was 1.4 fold greater among male children compared to female but this was of no statistical significance OR=1.4(0.4-4.7), p=0.622. Similarly, in the control

group , the male child had a 1.4 fold increased risk of having OME but again this was of no statistical significance OR=1.4(0.3- 7.8), p=1.0).

Table 4.Prevalence of OME by gender

Variable	Study group				Controls			
	OME present (%)	**No OME(%)**	**OR (95% CI)**	**P value**	**OME present (%)**	**No OME(%)**	**OR (95% CI)**	**P value**
Sex								
Male	25 (69.4)	11 (30.6)	1.4 (0.4-4.7)	0.622	6 (16.7)	30 (83.3)	1.4 (0.3-7.8)	1.000
Female	10 (62.5)	6 (37.5)	1.0		2 (12.5)	14 (87.5)	1.0	

Symptoms

In the study group, nasal obstruction, mouth breathing and snoring was recorded in 52 (100%) children. Sleep fragmentation was reported in 44 (84.6%) children. The duration of symptoms ranged from 6 – 36 months with a mean of 15 months. No parent reported history of hearing loss, otalgia or ear discharge. Table 5 below indicates the mean duration, range and frequency of symptoms in the study group.

Table 5: Frequency of symptoms in the study group

Variable	Frequency
Duration of symptoms in months, mean (SD) Range (months)	15.0 (7.9) 6-36
Nasal obstruction (%)	52 (100.0)
Mouth breathing (%)	52 (100.0)
Snoring (%)	52 (100.0)
Frequent arousal/ sleep fragmentation (%)	44 (84.6)
Otalgia (%)	0 (0.0)
Hearing loss (%)	0 (0.0)
Ear discharge (%)	0 (0.0)

Table 6.Symptoms associated with OME in children with OAD

Variable	OME Present	No OME	OR (95% CI)	P value
Symptoms				
All four[1]	32 (72.7%)	12 (27.3%)	4.4 (0.9-21.5)	0.096
Three only[2]	3 (37.5%)	5 (62.5%)	1.0	

[1]Nasal obstruction, mouth breathing, snoring and frequent arousal/sleep fragmentation

[2]Frequent arousal/sleep fragmentation excluded

The number of symptoms present was not significantly associated with presence of OME. However, there was a 4 fold increased likelihood of OME among children with all the four symptoms than those with three symptoms as shown in table 6 above OR=4.4 (0.9-21.5), p=0.096.

Table 7.Duration of symptoms and OME

Variable	OME Present	No OME	OR(95%CI)	P value
Duration of symptoms in months				
Mean (SD)	14.8 (8.5)	15.4 (6.5)	-	0.815
Category				
6 to 12 months	22 (71.0%)	9 (29.0%)	1.0	
>12 to 18 month	4 (57.1%)	3 (42.9%)	0.5 (0.1-2.9)	0.477
>18 months	9 (64.3%)	5 (35.7%)	0.7 (0.2-2.8)	0.654

As shown in table 7 above, children with OME had a shorter mean duration of symptoms than those without, however, this difference was not statistically significant (p = 0.815).

Otoscopic and tympanometric evaluation

The frequency of otoscopic findings among children in the two groups is as shown in table 8 below. Abnormal findings in study group were more 29 children (55.8%) than in control 2 children (3.8%). The study group had 31.5 likelihood to have abnormal findings compared with the controls OR=31.5 (6.9-143.5) p<0.001.

Table 8. Otological findings

Variable	Study group	Controls	OR (95% CI)	P value
Otological findings				
Abnormal	29 (55.8%)	2 (3.8%)	31.5 (6.9-143.5)	<0.001
Normal	23 (44.2%)	50 (96.2%)	1.0	

Table 9 below shows association between otological findings and OME. Out of the cases with abnormal otological findings, all had type B tympanogram, while out of those with normal otological findings only 26% had type B tympanogram and this was statistically significant p< 0.001.

In the control group, out of 2 cases who had abnormal otological findings both had type B tympanogram while those with normal otological findinsonly 12% had type B and this was statistically significant p=0.001.

Table 9:Association between otological findings and OME

	Cases			**Controls**		
	OME (type B)	**No OME (type A&C)**	**P value**	**OME(type B)**	**No OME (type A&C)**	**P value**
Otological findings Abnormal Normal	29 (100.0%) 6 (26.1%)	0 (0.0%) 17 (73.9%)	<0.001	2 (100.0%) 6 (12.0%)	0 (0.0%) 44 (88.0%)	0.001

Tympanogram types in order of frequency were type A 14 children (26.9%), type B 35 children (67.3%), and type C 3 children (5.8%) in the study group and type A 42 children (80.8%), type B 8 children (15.4%), and type C 2 children (3.8%) in the control group. Children with OAD had 14.1 fold increased risk to have type B tympanogram compared with the controls and this was of statistical significance OR=14.1 (5.1-39.0), p<0.001. Study group were also more likely to have type C tympanogram compared with controls OR=5.0 (1.1-23.7), p=0.030.

Table 10 .Types of tympanograms

Variable	Study group	Controls	OR (95% CI)	P value
Tympanogram type				
Type A	14 (26.9%)	42 (80.8%)	1.0	
Type B	35 (67.3%)	8 (15.4%)	14.1 (5.1-39.0)	<0.001
Type C	3 (5.8%)	2 (3.8%)	5.0 (1.1-23.7)	0.030

Lateral neck radiograph findings

Measurement performed on the lateral neck radiographs was ANR. All the children had an ANR > 0.60 with range between 0.6 to 0.9 with a mean of 0.8 as shown in table 11 below.

Table 11.Mean AN Ratio

Variable	Mean (SD)	Range
A.N RATIO	0.8 (0.1)	0.6-0.9

No patient had AN ratio in the region of grade 0 or grade I. 16 (30.76%) of children in the study group had grade I I adenoid hyperplasia and 36 (69.23%) children had grade III adenoid hyperplasia. Type B tympanogram was recorded in 9 (56.3%) of children with grade I I nasopharyngeal obstruction and in 26 (72.2%) in children with grade III obstruction. There was no significant difference between children with OME and those with no OME in these two grades in terms AN ratio (p=0.257) as depicted in table 12 below. However children with grade III adenoid enlargement were two times more likely to have OME compared to those with grade II

OR= 2.0 (0.6-6.9), p = 0.257

Table 12.Presence of OME in relation to the Grades of AN Ratio

Variable	OME Present	No OME	OR (95% CI)	P value
A.N ratio				
Grade II	9 (56.3%)	7 (43.8%)	1.0	
Grade III	26 (72.2%)	10 (27.8%)	2.0 (0.6-6.9)	0.257

DISCUSSION

The prevalence of OME among children aged 12 to 48 months with OAD diagnosed clinically and radiologicaly at the KNH ENT clinic from june 2013 to september 2013 was 67.3%. The controls had a prevalence of 15.4% .OR 11.3 (95% CI 4.4 -29.3),p = 0.001. In the current study ,although were planned to evaluate children who were between 1 and 8 years, we only managed to enroll children aged between 1 and 4 years.This is because adenoid enlargement outstrips growth of nasopharynx from 3 to 5 years of age with resultantreduction of nasopharyngeal airway (22).

In this study, the prevalence of OME among children with OAD was significantly higher than its prevalence among the normal children. The results showed adenoid hypertrophy as a significant risk factor for OME. Children with OAD had more than 11 times the risk of developing OME (Odds ratio = 11.3) than the normal children.

There is only one African study conducted among Nigerian children available in the literature. In this study, Orji et al (72) found that of the 92 ears (46 patients) in children with adenoid obstruction, 35% (32 ears) were diagnosed with OME using type B tympanogram, whereas7%(36 ears) of the 540 ears (270 children) in the control group were diagnosed with OME. The difference in the proportions of OME in the two groups was significant ($p < 0.001$).

Our prevalence of OME therefore would almost be twice their prevalence of OME in both groups. Our children were relatively younger than the Nigerian group with mean age being 26.0 and 24.1 months for both study and control group respectively compared with 5.7 and 5.9 years for cases and control respectively for the Nigerian study.

Our children had a severe disease in regards to mean ANR of 0.8 compared to 0.7 for the Nigerian study.

Its worth noting that the prevalence of OME in the control group in the current study is higher than prevalence of OME in general population of African as quoted in the literatue. N.E

Okolugbo et al (7) found a prevalence of 8% in Nigerian urban population for children aged 5 and 6 years while Halama et al (8) found a prevalence of 3.8% in a black rural population in south africa in children aged below 15years. Enviromental factors such as urban versus rural setting and population characteristics such as age may determine the prevalence.

In a study of 207 children aged 2-7 with mean age 5.3 years scheduled for adenoidectomy due to OAD, Dong-dong and WANG Wu-Qing (73) found prevalence of 69.1% using type B tympanogram as the diagnostic criteria . The results in this study are almost similar to ours. The age group in this study compared well to that of our study. However in this study they did not have controls.

Farhad et al (74) evaluated 120 Children aged 3-12 years with clinical and radiological evidence of adenoid hypertrophy. 44 patients (36.7%) had OME, mean age was 6.5 years. Again our study found a higher prevalence than in this study possibly due to the fact that the mean age of our children was smaller.

Regarding gender distribution in the study group ,in the current study it was found to be slightly more in male (69%) than female (62%) although it was not statistically significant. This was similar to the result obtained by farhad et al (74) who found that that (55%) were male, and (45%) female and orji et al who found a prevalence of 36.53% in male and 32.5% in females .This difference may be because of growth difference or overall male predominance for childhood infection (78).

The number of symptoms present was not significantly associated with presence of OME. However a study done by Hans et al (69) found a relationship between nasal symptoms of OAD and OME.

Distribution of tympanogram types was type A 14 children (26.9%), type B 35 children (67.3%), and type C 3 children (5.8%) in the study group and type A 42 children (80.8%), type B 8 children (15.4%), and type C 2 children (3.8%) in the control group. Farhat et al (74) only found two types of tympanogram i.e. type B 70% and type C 30%. Orji et al (72) found type A in 43.47%, type B in

34.78% and type C in 21.73% in the study group and type A 84%, type B 6.66% and type C 9.25% in the control group.

Children in the current study presented with severe nasal obstruction compared to other studies (72). All children in the current study had an ANR in the range of grade II (30.76%) and grade III (69.23%). 9 out of 16 (56%) children with grade II adenoid hypertrophy and 26 out of 36 children (72.2%) with grade III adenoid hypertrophy had OME. This study however did not show a positive correlation between the degree of nasopharyngeal obstruction and the presence of OME when comparing grade II and grade III. However grade III adenoid enlargement was twice as likely to have OME as compared to grade II enlargement. OR 2.0 (0.6-6.9), p = 0.257. This was in contrast to other study by orji et al (72) who showed that the degree of obstruction was associated with OME.

In a different study by Pan H et al (70) found that the eustachian tube function of the children with adenoids hypertrophy was worse than the normal and the relation between the eustachian tube function and the AN ratio was not statistical difference

In a study assessing grades of adenoid hypertrophy in children with OME grade 4 adenoid hypertrophy was found to be statistically significant in children with otitis media with effusion ($P < 0.0002$) (65).In this study rigid nasal endoscopy was used for grading of adenoids.

CONCLUSION

The prevalence of OME among children aged 12 to 48 months with OAD diagnosed clinically and radiologicaly at the KNH was 67.3% in the cases and 15.4% in the controls(95% CI 4.4 - 29.3).

This study found adenoid obstruction as a significant risk factor for OME in children.

Gender,duration of symptoms and symptomatology are not significant risk factors for OME in children with OAD.

Children with OME may not present with history of hearing loss.

When comparing children with moderate to gross adenoid enlargement of adenoid tissue, the relative size of adenoid to that of nasopharynx (ANR) does not increase the risk of developing OME significantly.

RECOMMENDATIONS

1. Children with features of obstructive adenoid disease should be carefully examined for possible existence of OME.
2. This information should be availed to personnel's at public primary care units in Kenya.
3. The role of adenoid enlargement in the pathogenesis of OME can be determined by conducting further studies on adenoidectomy and their effect on OME.

REFERENCES

1.Jalisi M, Jazbi B. Chronic middle ear effusion. Current problems in otorhinolaryngology. *Pakistan Doctors Publication* 1991; 85-97.

2. Sriwardhana KB, Howard AJ, Dunkim KT. Bacteriology of otitis Media with effusion. *J laryngolotol* 1989; 103: 253-6.

3. Finkelstein Y, Talmi YP, Rubel Y et al. Otitis media with effusion as a presenting symptom of chronic sinusitis. *Journal of laryngolotol* 1989; 103: 827-832.

4. Chan KH, Swarts JD, Rudoy R et al. Otitis media in the republic of Palau.*Arch otolaryngol. Head Neck Surg* 1993; 119: 425-28.

5. David W, Jerome O, Benard R. Epidemiology of otitis media with effusion in children first seven years. a prospective cohort study. *Infect dis* 1999; 160: 83-94.

6. George Browning, Scott-Brown's Otorhinolaryngology-Head and Neck Surgery.7th edition. U.K: Hodder Arnold; 2008, vol – 1, pg-880.

7. N.E. Okolugbo, M. Ugwu. Prevalence of secretory otitis media amongst primary school children in Benin city Nigeria. *Continental J. Medical Research 2009;* 3: 12 –15.

8. Halama AR, Voogt GR, Musgrave GM. Prevalence of otitis media with effusion in children in a black rular community in South Africa.*Int J PediatrOtorhinolaryngol* 1986; 11: 73-7.

9.Saim A, Saim L, Saim S et al. Prevalence of otitis media with effusion amongst preschool children in Malaysia. *Int J PediatrOtorhinolaryngol* 1997; 41: 21-28.

10.Elango S, Purohit G, Hashim M et al. Hearing loss and ear disorders in Malaysian school children. Int J *PediatrOtorhinolaryngol* 1991; 22: 75-80.

11. Brooks D. School screening for middle ear effusion. *Ann OtolRhinol* Laryngo1 1976; 85: 223-29.

12. Tos M, Poulsen G. Tympanometry in 2 year old children, seasonal influence on secretory otitis media and tubal dysfunction. *Ann OtolRhinolLaryngol* 1979; 41: 1-10.

13. Lous J, Nikolajsen F. Epidemiology of middle ear effusion and tubal dysfunction. A one year prospective study comparing monthly tympanometry in 387 non selected 7 year old children.*IntPaedOtorhinolaryngol* 1981; 3: 303-17.

14.Paparela M, Jung T, Goycoolea M. Otitis Media with Effusion :Paparella and Shumrich - Otolaryngology, London, *Saunders 1990; 2: 1317- 1342.*

15. Richard A. Otitis media with effusion(Glue ear) in scott browns otolaryngology.paediatric otolaryngology.London,Butterworths1987; 6: 159-176.

16. Randall P, Estool S. Unexpected otitis media in infants with cleft palate. *Cleftpalate j* 1967; 4: 99-103.

Paradise J, Bluestone C, Felder H. The universality of otitis media in 50 infants with cleft palate**.***Pediatrics* 1969; 44: 35-42.

18. Lokman S, Loh T, Said H et al. Incidence and management of middle ear effusion in cleft palate patients. *Med Malaysia* 1992; 47: 51-55.

19.Matsune S, Sando I, Takahashi H. Insertion of the tensor velipalatini muscle into the eustachian tube cartilage in cleft palate cases.*Ann OtolRhinolLaryngol* 1991; 100: 439

20.P.m brown;GTR lewis;AJ Parker et al, the skull base and nasopharynx in down syndrome in relation to hearing impairement ; clin otollaryngo.1989 ; 14: 241-246

21.Juhn SK, Jung TT. Inflammatory changes reflected in middle ear effusion in otitis media. *AurisNasus Larynx* 1985; 12: 63-6.

22.Acharya K, Bhusal CL, Guragain RP**.** Endoscopic grading of adenoid in otitis media with effusion. *J Nepal Med Assoc* 2010; 49: 47-51.

23. Jeans W, Fernando D. A longitudinal study of the growth of the nasopharynx and its contents in normal children. *British Radiology* 1981; 54: 117-21.

24.Ohashi Y, Nakai Y. Current concepts of mucociliary dysfunction in otitis media with effusion. *ActaOtolaryngol*1991; 486: 149-61.

25. Ernstson S, Afzelius BA, Mossberg B. Otologic manifestations of the immotile-cilia

syndrome. *ActaOtolaryngol*1984; 97: 83-92.

26. Daly K. Epidemiology of otitis media. *Otolaryngologic clinics of NorthAmerica* 1991; 24: 775– 82.

27.Ahlo O, Oja H, Koivu M et al. Risk factors for chronic otitis media with effusion in infancy. *Arch Otolaryngol Head Neck Surg* 1995; 121:839-43.

28.Bennet K, Hoggard M, Silva P et al. Behaviour and developmental effects of otitis media with effusion into the teens. *Arch Dis Child* 2001; 85: 91-5.

29.Augustsson I, Engstrand I. Otitis media academic achievement**.** *IntJPaediatricOtorhinolaryngol* 2001; 57: 31-40 .

30.Karma PH, Penttila MA, Sipila MM et al. Otoscopic diagnosis of middle ear effusion in acute and non-acute otitis media. The value of different otoscopic findings.*Int J PediatrOtorhinolaryngol*1989; 17: 37–49.

31.Stewart MG, Manolidis S, Wynn R et al. Practice patterns versus practice guidelines in pediatric otitis media. *Otolaryngol Head Neck Surg* 2001; 124: 489–95.

32.Johansen EC, Lildholdt T, Damsbo N et al. Tympanometry for diagnosis and treatment of otitis media in general practice. *FamPract*2000; 17: 317–22.

33.Potsie WP, Shott SR. The ear. Rudolph AM. Rudolph's Pediatrics. 20th ed. Stamford, Conn.: Appleton & Lange 1996; 939–52.

34.Fiellau, Nikolajsen M. Tympanometry and secretory otitis media. Actaotolaryngologica 1983; 399: 1-3.

35.EdwardOnusiko, Tympanometry. *Am Fam Physician*2004 ;70:1713-1720

36.Dempster J H**,** Makensie K. Tympanometry in detection of hearing impairment associated with otitis media with effusion.*clinical otolaryngology* 1991; 16: 157-159.

37. Liu YS, Lim DJ, Lang RW et al. Chronic middle ear effusions*:* Immunochemical and bacteriological investigations. *Arch Otolaryngol 1975; 101: 278-86*.

38. Gates GA. Medical treatment of chronic otitis media with effusion (secretory otitis media). *Otolaryngol Head Neck Surg* 1986; 94: 350-354.

39.Mandel EM, Rochette HE, Bluestone CD et al. Efficacy of Amoxillin With and Without Decongestant antihistamine for Otitis Media with Effusion in Children. Results of Double Blind-Randomized Trial.*TheNew England Journal 1987; 316: 432 – 437.*

40. Thomsen J. Antibiotic treatment of children with secretory otitis media**.***Arch Otolaryngol Head Neck Surg* 1989; 115: 447-451.

41.Healy GB. Antimicrobial therapy of chronic otitis media with effusion. *Int J PediatrOtorhinolaryngol* 1984; 8: 13-17.

42.Jeans. Longitudinal study of growth of nasopharynx and its contents in normal children.*British journal of radiology* 1981; 54: 117-121.

43. Jeans WD, Fernando DC, Maw AR. How should adenoidal enlargement be measured? A radiological study based on interobserver agreement. *ClinicalRadiology* 1981; 32: 337-40.

44. Cohen ML, Konai PJ, Scott JR. Lateral cervical radiographs and adenoid size: Do they correlate? *ENT Journal* 1992; 71: 638-42.

45. Wormald PJ, Prescott CA. Adenoids: comparison of radiological assessment methods with clinical and endoscopic findings. *The Journal of Laryngology andOtology* 1992; 106: 342-34.

46. Linder Aronson S. Radiocephalometric analysis of anteroposterior nasopharyngeal dimension in 6 to 12 years old mouth breathers compared with nose breathers. *journals of otorhinolaryngology* 1973; 35:1 9-29.

47. Tomonaga K, kurono Y, chaen R et al. Adenoid and otitis media with effusion. nasopharyngeal flora. *Am j otolaryngol*1989; 10: 204-207.

48. Gates GA. Adenoidectomy for otitis media with effusion. *Ann OtolRhinolLaryngol* 1994; 163: 54-58.

49.Bluestone CD. Eustachian tube in cleft palate.*Annals Of Otologyrhinologylaryngology* 1971; 80: 1-25.

50.Di Francesco R, Paulucci B, Nery C et al. Craniofacial morphology and otitis media with effusion in children. *Int J PediatrOtorhinolaryngol* 2008; 72: 1151-1158.

51. Blue stone C D. Obstructive adenoids in relation to otitis media.A*nnals ofOtology, Rhinology, Laryngology*1975; 84: 44-48.

52.Yasan H, Dogru H, Tüz M et al. Otitis media with effusion andhistopathologicproperties of adenoid tissue. *Int J PediatrOtorhinolaryngol*2003; 67: 1179-83.

53.Corey JP, Adham RE, Abbass AH et al. The role of IgE-mediated hypersensivity in otitis media with effusion.*Am J Otolaryngol*1994; 15: 138-144.

54. Maw AR, Bawden R. Factors affecting resolution of otitis media with effusion in children. *ClinOtolaryngol*1994; 19: 125-130.

55.Nsouli TM, Nsouli SM, Linde RE et al. Role of food allergy in serous otitis media. *AnnAllergy* 1994; 73: 215-219.

56. Berger G, Ophir D. Possible role of adenoid mast cells in the pathogenesis of secretory otitis media.*Ann OtolRhinolLaryngol* 1994; 103: 632-5.

57. Maw AR. Chronic otitis media with effusion and adeno-tonsillectomy, a prospective randomized controlled study.*Int J PediatrOtorhinolaryngol*1983; 6: 239-46.

58.Pulec J l, Tomokazu K, Malcom D. Eutachian tube lymphatics. *Annals ofOtology,Rhinology and Laryngology* 1975; 84: 483-492.

59. Gates GA, Muntz HR and Gaylis B. Adenoidectomy and otitis media. *Ann OtolRhinolLaryngolSuppl.*1992;155:24-32.

60.May AR. Age and adenoid size in relation to adenoidectomy in otitis media with effusion. *American journal of otolaryngology* 1985; 6: 245-248.

61.Van denAardweg MT, Schilder AG, Herkert E et al. Adenoidectomy for otitis media in children. *Cochrane Database Syst Rev* 2010; 1: CD007810.

62.Wright ED, Pearl AJ, Manoukian JJ. Laterally hypertrophic adenoids as a contributing factor in otitis media.*Int J Pediatr Otorhinolaryngol.*1998; 45:207-14.

63. Takahashi. Endoscopic findings at the pharyngeal orifice of Eustachian tube in effusion. *Eur-Arch-Otorhinolaryngo* 1996; 253: 42-44.

64.Bluestone CD, Berry QC. Concepts on the pathogenesis of middle ear effusions.*Ann. Otol. Rhinol. Laryngol* 1976; 85: 182—187.

65.Hibbert J, Stell PM. The role of enlarged adenoids in the aetiology of serous otitis media.*Clinotolaryngol*1982;7:253-256.

66.Acharya K, Bhusal CL, Guragain RP.Endoscopic grading of adenoid in otitis media with effusion.*JNepal Med Assoc.*2010; 49: 47-51.

67. Liu Y, Sun Z, Li Z et al. Relationship between adenoid hypertrophy and secretory otitis media (in Chinese). *Liu Chuang Er Bi yanHouKeZaZhi* 2004; 18: 19-20

68.Ito H. Radiographic examination of adenoids (high voltage technique). *Journal of Otolaryngology ofJapan* 1968; 71:96.

69.Rodger TR. Treatment of chronic suppurative otitis media. *Journal of laryngology andotology*1993; 48: 55.

70. Hans H, Elverland, Olav K, ET et al. Adenoidectomy and Secretory Otitis Media.*ActaOto-laryngologica*1982; 93: 134-136

71. Pan H, Li L, Liang Z, et al. Relationship between adenoids hypertrophy and tympanogram/eustachian tube function in children. *Lin Chuang Er Bi Yan HouKeZaZhi.*2005 ; 19: 1015-6.

72. Orji FT, Okolugbo NE, EzeanolueBC,et al . The role of adenoidal obstruction in the pathogenesis of otitis media with effusion in Nigerian children. *Niger J Med* 2010; 19: 62-8.

73.Dong-dong, Wang Wu-qing. Assessment of middle ear effusion and audiological characteristics in young children with adenoid hypertrophy.*Chin Med J 2012; 125: 1276-1281.*

74.Farhad J. Khayat Lana Sh. Dabbagh Incidence of otitis media with effusion in children with adenoid hypertrophy.Zanco J. Med. Sci., Vol. 15, No. (2), 2011

75. Kirkwood BR, Sterne JA. Essential of Medical Statistics.2nd Edn.London: Blackwell Scientific Publications, pp 131- 137, 413-428.

76. Fujioka M, Young L W, Girdang BR. Radiographic evaluation o adenoid size in children. American journal of radiology 1979;133:401-404.

77. Sade J. secretory otitis media and its sequele.Newyork:curchilllivingstone;1979. Pp 1-2.

78.Maw AR. Otitis media with effusion;Evans JNG ed. Scoots browns otolaryngology.5th ed.Butterworth int ed.1987;159-172.

APPENDIX 1: TYPES OF TYMPANOGRAMS

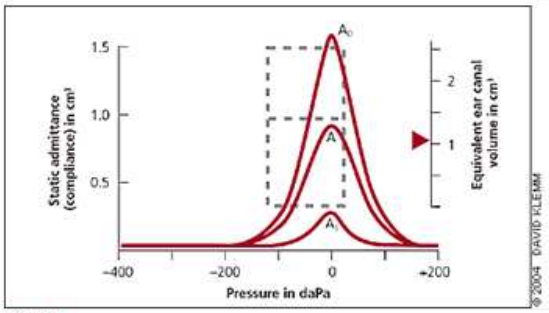
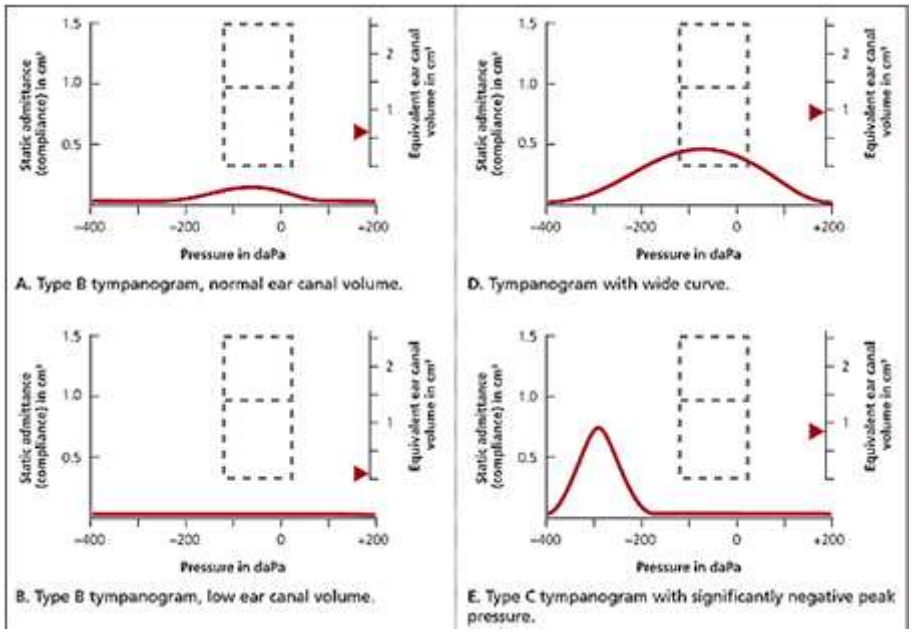


Figure 1
Type A tympanogram. Type A_D has a high peak height. The middle curve is normal. A_S reduced peak height.



Courtesy of American journal of family physicians(34)

APPENDIX 2: GENERAL PATIENT INFORMATION AND CONSENT FORM.

Greeting, my name is Dr. Anthony. M. Kiama, I would like to seek your consent for your child participation in a study aimed at assessing the prevalence of middle ear effusion in children with adenoid enlargement seen at KNH. The information gathered will be used to improve the management of children with adenoid enlargement.

There is no harm or risk anticipated in this study.

The only additional test that will be carried out is tympanometry which will be at no extra cost to you. Tympanometry is a safe non invasive test.

Benefits of the study include early detection of any middle ear effusion which will mean early intervention or treatment.

Participation in this study is out of your own will. Medical care will not be denied in case you decline to participate in the study. You may terminate participation at any time with no consequences whatsoever. All information will be treated with confidentiality.

Consent Form

I hereby consent to my child participation in the study to determine prevalence rate of middle ear effusion in children with adenoid enlargement as explained to me by Dr Anthony. M. Kiama.

Name of parent Signature Date

Name of researcher Signature Date

CONTACTS

1. Dr Anthony Kiama
   Tel: 0721550604
   Email: kantonymwaniki@yahoo.com
2. Prof.M.L. Chidia
   Secretary KNH/UON Ethics and Research Committee
   Tel: 2726300 ext 44102

MAELEZO KWA MGONJWA NA MAKUBALIANO YA KUSHIRIKI KATIKA UTAFITI.

Jambo, mimini Daktari Anthony. M. Kiama. Naomba ruhusa yako kushiriki katika utafiti unaochunguza ukubwa washida ya kuwepo kwa maji katika sehemu yakati yasikio kwa watoto ambao wamegojeka na ukubwawa adenoid katika hospitali kuu ya Kenyatta. Matokeo ya utafiti itakuwa muhimu katika kuboresha kufuatiriwa kwa watoto wenye ukubwa w aadenoid .Hakuna madhara au hatari inayotarajiwa kwakushiriki katika utafiti huu. kipimo cha ziada nje ya yale kawaida kwa matibabu itakayofanywa ni ya tympanometry. Hakuna gharama yoyote ya ziada itatokana kwa ajili yakushiriki katika utafiti. Kipimo cha tympanometry niuchunguzi ambao hauna madhara. Moja yao ya manufaa yahii utafiti ni kuwa kukiwa nashida ya maji katika sehemu yakati ya sikio inaguduliwa mapema na hii inamaanisha matibabu itaanza mapema.

Kushiriki kwa utafiti huu ni kwa hiari yako na hauwezi kushurutishwa. Utahudumiwa ata kama utakataa kushiriki kwa utafit ihuu. Una uhuru kutamatisha kuhusika wakati wowote bila madhara yoyote. Habari zozote utakavyo toa zitawekwa kwa siri na jina lako halitachapishwa popote.

KIBALI

Mimi Bw/Bi/Binti----------nimesoma maelezo yanayo husu utafiti huu kama nilivyoelezwa na DaktariA.Kiama na nimekubali kushiriki katika utafiti huu. Sahihi yangu nidhihirisho ya ridhaa yangu. Sijapatiwa fedha wala nyenza yoyote ilinishiriki katika utafiti huu.

Jina la mzazi SahihiTarehe

Jina la DaktariSahihiTarehe

MAELEZO YA ZIADA

1. Dr Anthony Kiama
   Nambariyasimu: 0721550604
   Baruapepe: kantonymwaniki@yahoo.com
2. Prof M.L Chidia
   KatibuKNH/UON Ethics and Research Committee
   Nambariyasimu: 2726300 ext 44102

APPENDIX 3:

TYMPANOMETRY MACHINE

InteracousticsimpedanceaudiometerAT235 (Interacoustics A/S-Assens, Denmark).

APPENDIX 4: PATIENT INFORMATION QUESTIONNAIRE.

INITIALS

IP NO/ENT NO

AGE

GENDER

DURATION OF SYMPTOMS (months)_____

MEDICAL HISTORY

SYMPTOM	PRESENT	ABSENT
Nasal obstruction		
Mouth breathing		
Snoring		
Frequent arousals/sleep fragmentation		
Otalgia		
Hearing loss		
Ear discharge		

OTOSCOPY FINDINGS

LEFT EAR: NORMAL ☐

ABNORMAL ☐

Specify _____

RIGHT EAR: NORMAL ☐

ABNORMAL ☐

Specify _____

RADIOLOGIC FINDINGS

AN ratio	

TYPE OF TYMPANOGRAM

Type A ☐

Type B ☐

Type C ☐

KENYATTA NATIONAL HOSPITAL
APPROVED
08 MAY 2013
ETHICS & RESEARCH COMMITTEE

KENYATTA NATIONAL HOSPITAL
05 JUL 2013
E.N.T, NOSE & THROAT CLINIC
P. O. Box 20723 NAIROBI

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KNH/UON-ERC
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Website: www.uonbi.ac.ke
Link:www.uonbi.ac.ke/activities/KNHUoN

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Telegrams: MEDSUP, Nairobi

8th May 2013

5/7/2013
Approved to collect data

Dr. Anthony Mwaniki Kiama
Dept.of Surgery
School of Medicine
University of Nairobi

Dear Dr. Kiama

RESEARCH PROPOSAL: PREVALENCE OF OTITIS MEDIA WITH EFFUSION IN CHILDREN WITH OBSTRUCTIVE ADENOID DISEASE COMPARED WITH NORMAL CONTROLS AT KENYATTA NATIONAL HOSPITAL (P25/01/2013)

This is to inform you that the KNH/UoN-Ethics & Research Committee (KNH/UoN-ERC) has reviewed and **approved** your above revised proposal. The approval periods are 8th May 2013 to 7th May 2014.

This approval is subject to compliance with the following requirements:

a) Only approved documents (informed consents, study instruments, advertising materials etc) will be used.
b) All changes (amendments, deviations, violations etc) are submitted for review and approval by KNH/UoN ERC before implementation.
c) Death and life threatening problems and severe adverse events (SAEs) or unexpected adverse events whether related or unrelated to the study must be reported to the KNH/UoN ERC within 72 hours of notification.
d) Any changes, anticipated or otherwise that may increase the risks or affect safety or welfare of study participants and others or affect the integrity of the research must be reported to KNH/UoN ERC within 72 hours.
e) Submission of a request for renewal of approval at least 60 days prior to expiry of the approval period. (*Attach a comprehensive progress report to support the renewal*).
f) Clearance for export of biological specimens must be obtained from KNH/UoN-Ethics & Research Committee for each batch of shipment.
g) Submission of an *executive summary* report within 90 days upon completion of the study This information will form part of the data base that will be consulted in future when processing related research studies so as to minimize chances of study duplication and/or plagiarism.

For more details consult the KNH/UoN ERC website www.uonbi.ac.ke/activities/KNHUoN

Protect to Discover

Yours sincerely

PROF. M. L. CHINDIA
SECRETARY, KNH/UON-ERC

c.c. Prof. A.N. Guantai, Chairperson, KNH/UoN-ERC
The Deputy Director CS, KNH
The Principal, College of Health Sciences, UoN
The Dean, School of Medicine, UoN
The Chairman, Dept.of Surgery, UoN
The HOD, Records, KNH
Supervisors: Prof. H.O. Oburra, Dr. M. Omutsani