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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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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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 is 67.3% and in the control group is 15.4% (95% CI 4.4 to 29.3).

Conclusion and Recommendations: 6 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

Adenoid enlargement has traditionally been considered a factor in otitis media with effusion (OME). 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 to 15% 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). In Nigeria urban population, the prevalence of OME in children aged 5-6 years using tympanometric studies was found to be 8% (7). 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 incidence 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 valsala 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. Mucociliary 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 -199 mmH2O and; Type C2: -200 to -400 mmH2O (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 tympanometry.

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):

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

Where \(\pi_1 = 0.35; \pi_2 = 0.07; N = \) minimum number of children in each group; \(\mu = \) one-sided percentage point of the normal distribution corresponding to 100% less the power (95%) in this case 1.28 and; \(\nu = \) 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):

24
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-basioccipital synchondrosis (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 audiometer AT235. 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 0.6ml 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 being 12-24 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

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

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

<table>
<thead>
<tr>
<th>Variable</th>
<th>Study group</th>
<th>Controls</th>
<th>OR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>OME</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>35 (67.3%)</td>
<td>8 (15.4%)</td>
<td>11.3 (4.4-29.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Absent</td>
<td>17 (32.7%)</td>
<td>44 (84.6%)</td>
<td>1.0</td>
<td></td>
</tr>
</tbody>
</table>

### 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 stastically significant.

### Table 3. Prevalence of OME by age group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Study group</th>
<th>Controls</th>
</tr>
</thead>
</table>

29
Table 4 below shows proportions 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

<table>
<thead>
<tr>
<th>Variable</th>
<th>Study group</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OME present (%)</td>
<td>No OME (%)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>25 (69.4)</td>
<td>11 (30.6)</td>
</tr>
<tr>
<td>Female</td>
<td>10 (62.5)</td>
<td>6 (37.5)</td>
</tr>
</tbody>
</table>

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

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

Table 6. Symptoms associated with OME in children with OAD

<table>
<thead>
<tr>
<th>Variable</th>
<th>OME Present</th>
<th>No OME</th>
<th>OR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All four&lt;sup&gt;1&lt;/sup&gt;</td>
<td>32 (72.7%)</td>
<td>12 (27.3%)</td>
<td>4.4 (0.9-21.5)</td>
<td>0.096</td>
</tr>
<tr>
<td>Three only&lt;sup&gt;2&lt;/sup&gt;</td>
<td>3 (37.5%)</td>
<td>5 (62.5%)</td>
<td>1.0</td>
<td></td>
</tr>
</tbody>
</table>

<sup>1</sup>Nasal obstruction, mouth breathing, snoring and frequent arousal/sleep fragmentation
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

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

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

<table>
<thead>
<tr>
<th>Variable</th>
<th>Study group</th>
<th>Controls</th>
<th>OR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
</table>


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 findings only 12% had type B and this was statistically significant p=0.001.

Table 9: Association between otological findings and OME

<table>
<thead>
<tr>
<th>Otological findings</th>
<th>Cases</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OME (type B)</td>
<td>No OME (type A&amp;C)</td>
</tr>
<tr>
<td>Abnormal</td>
<td>29 (100.0%)</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Normal</td>
<td>6 (26.1%)</td>
<td>17 (73.9%)</td>
</tr>
</tbody>
</table>

Tymanogram 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**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Study group</th>
<th>Controls</th>
<th>OR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tympanogram type</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type A</td>
<td>14 (26.9%)</td>
<td>42 (80.8%)</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Type B</td>
<td>35 (67.3%)</td>
<td>8 (15.4%)</td>
<td>14.1 (5.1-39.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Type C</td>
<td>3 (5.8%)</td>
<td>2 (3.8%)</td>
<td>5.0 (1.1-23.7)</td>
<td>0.030</td>
</tr>
</tbody>
</table>

**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**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.N RATIO</td>
<td>0.8 (0.1)</td>
<td>0.6-0.9</td>
</tr>
</tbody>
</table>
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

\[ \text{OR} = 2.0 \ (0.6-6.9), \ p = 0.257 \]

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

<table>
<thead>
<tr>
<th>Variable</th>
<th>OME Present</th>
<th>No OME</th>
<th>OR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. N ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade II</td>
<td>9 (56.3%)</td>
<td>7 (43.8%)</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Grade III</td>
<td>26 (72.2%)</td>
<td>10 (27.8%)</td>
<td>2.0 (0.6-6.9)</td>
<td>0.257</td>
</tr>
</tbody>
</table>
DISCUSSION

The prevalence of OME among children aged 12 to 48 months with OAD diagnosed clinically and radiologically 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 resultant reduction 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, whereas 7% (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.

It's 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 literature. 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 15 years. Environmental 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 radiologically 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


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


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


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

![Diagram of tymanograms](image)

Figure 1: Types of tymanograms. Type A demonstrates high peak height, Type B shows normal peak height, Type C indicates low peak height, Type D represents wide curve, and Type E shows significantly negative peak pressure.

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.


KIBALI

Mimi Bw/Bi/Binti------------------- nimesoma maelezo yanayo husu utafiti huu kama nilivyoelezwa na Daktari A. 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
   Baruuapepe: kantonymwaniki@yahoo.com

2. Prof M.L Chidia
   KatibuKNH/UON Ethics and Research Committee
   Nambariyasimu: 2726300 ext 44102
APPENDIX 3:

TYMPANOMETRY MACHINE

Interacoustics impedance audiometer AT235 (Interacoustics A/S-Assens, Denmark).
APPENDIX 4: PATIENT INFORMATION QUESTIONNAIRE.

INITIALS

IP NO/ENT NO

AGE

GENDER

DURATION OF SYMPTOMS (months)

MEDICAL HISTORY

<table>
<thead>
<tr>
<th>SYMPTOM</th>
<th>PRESENT</th>
<th>ABSENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasal obstruction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mouth breathing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Snoring</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frequent arousals/sleep</td>
<td></td>
<td></td>
</tr>
<tr>
<td>fragmentation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Otalgia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hearing loss</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ear discharge</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

OTOSCOPY FINDINGS

LEFT EAR: NORMAL

ABNORMAL

Specify ____________________________

RIGHT EAR: NORMAL

ABNORMAL

Specify ____________________________
RADIOLOGIC FINDINGS

<table>
<thead>
<tr>
<th>AN ratio</th>
</tr>
</thead>
</table>

**TYPE OF TYMPANOGRAM**

- Type A: ☐
- Type B: ☐
- Type C: ☐