PREVALENCE OF MIDDLE EAR EFFUSION AMONG CHILDREN WITH ADENOID HYPERTROPHY AT MUHIMBILI NATIONAL HOSPITAL

Faustine M.C.Bukanu, MD

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By

Faustine M.C.Bukanu, MD

A Dissertation Submitted in Partial Fulfillment of the Requirements for the Degree of Master of Medicine (Otorhinolaryngology) of Muhimbili University of Health and Allied Sciences

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CERTIFICATION

The undersigned certify that they have read and hereby recommend for acceptance by Muhimbili University of Health and Allied Sciences a thesis/dissertationentitled,**Prevalence of middle ear effusion among children with adenoid hypertrophy at Muhimbili National Hospital, April 2012 – December 2012,**in partial fulfillment of the requirements for the degree of Master of Medicine (Otorhinolaryngology) of Muhimbili University of Health and Allied Sciences.

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Dr. H. Swai

(Supervisor)

Date: _____

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Prof. N. Moshi

(Supervisor)

Date: _____

DECLARATION AND COPYRIGHT

I, **Faustine M.C.Bukanu**, declare that this **dissertation** is my own original work and that it has not been presented and will not be presented to any other university for a similar or any other degree award.

Signature……. Date…….……..

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I would like to cordially address my thanks to God for His redeeming love which gives us the joy of carrying out the life of sure destiny.

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"May each one of you find through this work, the fruit of the unforgettable rendered service".

DEDICATION

To Almighty God

To my beloved wife Nelly Mwalongo

To my dear Daddy

To all my family

To all my teachers

To all my friends and colleagues,

This work is dedicated.

ABSTRACT

Introduction: Middle Ear Effusion is a common disorder in children which may either resolve spontaneously or cause undesirable complications especially if associated with persistent hearing loss.

Middle Ear Effusion contributes to hearing loss, poor speech acquisition and learning difficulties. There has been no study done to show the magnitude of middle ear effusion in children with adenoid hypertrophy and its complications in our country.

Aim: This study aimed at determining the prevalence of middle ear effusion among children with adenoid hypertrophy at Muhimbili National Hospital

Materials and Methods: This study was conducted at MNH, Dar-es-salaam in Tanzania among 420 children aged 9 years or less. All children with adenoid hypertrophy attending MNH were included in the study. Diagnosis of middle ear effusion was reached by findings of the ear drum with exclusion of features of acute otitis media. Tympanometry was conducted by research assistant and type B curve was regarded as diagnostic for middle ear effusion.

SPSS computer program 16.0 wasused to analyze the data and relationships were tested at 5% tolerable error.

Results; Adenoid hypertrophy is a significant risk factor for MEE. About 61.7% of children with adenoid hypertrophy presented with MEE. On the other hand males were more affected with MEE as compared to females and the age of over 2years to 6 years had more cases of MEE of about 67.9%.

Conclusion; Adenoid hypertrophy is a significant risk factor for MEE and the most affected age is over 2 years to 6 years with male preponderance.

DEFINITIONS AND ABBREVIATIONS

Abbreviations

AHCPR - American Health Care Policy and Research.

AOM - Acute otitis media

CHL - Conductive hearing loss

COM - Chronic otitis media

dB - Decibel

EAC - External auditory canal

ET - Eustachian tube

ETD - Eustachian tube dysfunction.

HL - Hearing loss

MEE - Middle ear effusion

OM - Otitis media

OME - Otitis media with effusion

ORL - Otorhinolaryngology

SNHL - Sensorineural hearing loss

TM - Tympanic membrane.

URTI - Upper respiratory tract infection

DEFINITIONS

- Hearing impairment refers to any degree of hearing loss from mild to severe.
- Atelectasis is medial retraction of pars tensa of the tympanic membrane (TM) as a result of otitis media with effusion (OME).
- Decibel (dB) is a unit used in measuring sound pressure levels.
- Tympanosclerosis is sub epithelial deposition of calcarious plaques in the middle ear or tympanic membrane (TM).
- Adhesive otitis media is fibrous adhehesion between TM and medial wall of middle ear.

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1.INTRODUCTION AND LITERATURE REVIEW

1.1. INTRODUCTION

Middle ear effusion (O.M.E.) is the commonest problem among young children. It is characterized by accumulation of semi sterile serous or mucoid secretion in the middle ear cleft. It results from either an alteration of the Eustachian tube function or the mucociliary system or both. The disease is also known as secretory otitis media, middle ear effusion, middle ear catarrh, non suppurative otitis media and glue ear.[1]

Adenoids are aggregates of non capsulated lymphoid tissue located in the roof and posterior aspect of thenasopharynx. They are largest in children between 2 to 6 yr. Enlargement may be physiologic or secondary to viral or bacterial infection, allergy, irritants, and, possibly, gastro esophageal reflux[1]. Other risk factors include enrollment in day care centre. Severe hypertrophy can obstruct the Eustachian tubes (causing Otitis media with effusion), posterior choanae (causing sinusitis), or both.[2]

Symptoms include nasal obstruction, snoring during sleep, mouth breathing, sleep disturbances, and hearing loss. Diagnosis depends mainly on history and investigation which include flexible fiber opticnasopharyngoscopy and Lateral X-Ray of nasopharynx. Treatment often includes intranasal corticosteroids, antibiotics, and, for significant nasal obstruction or persistent recurrent acute otitis media or middle ear effusion, adenoidectomy[2].

Myringotomy and insertion of tympanostomy tube if the above form of therapy fails to resolve the effusion.

Some authors have hypothesized that the adenoid may compress or obstruct the Eustachian tube lumen, thereby causing negative middle ear pressure and subsequently middle ear effusion.[3]

Adenoids infection and Eustachian tube dysfunction have been frequently associated with the incidence of middle-ear effusion[4].

1.1.1 ANATOMY

Nasopharynx

The nasopharynx is that part of the pharynx located behind the nose. Air inhaled through the nose enters the nasopharynx on its way to the lower respiratory tract.

Structures in the nasopharynx include:

- Openings of the Eustachian tubes – these are tubes connecting the ear cavities with the pharynx. Their purpose is to ventilate the middle ear and equalize middle ear pressure with atmospheric pressure for optimal hearing.
- Fossa of rosenmuller – This is a small but deep cavern, often the starting point for cancer in these parts.
- Adenoids – a collection of lymphoid tissue located in the roof and posterior wall of the nasopharynx. It is part of the immune system and helps protect the body from infective organisms. Adenoids are usually only present in children. They regress in size and gradually disappear as the child grows older.
- Tubal tonsils – also lymphoid tissue, located around the Eustachian tube openings[5]

Middle ear

The **middle ear** or **tympanic cavity** is an irregular, laterally compressed space within the temporal bone. It is filled with air, which is conveyed to it from the nasal part of the pharynx through the auditory tube. It contains a chain of movable bones, which connect its lateral to its medial wall, and serve to convey sound vibrations from the tympanic membrane to the internal ear.

The tympanic cavity consists of three parts: the **tympanic cavity proper(mesotympanum),**medial to the tympanic membrane, the **attic** or **epitympanum,**above the level of the membrane,and **hypotympanum** below the tympanic membrane. The vertical and antero-posterior diameters of the cavity are each about 15 mm. The transverse diameter measures about 6 mm. above and 4 mm. below; opposite the center of the tympanic membrane it is only about 2 mm. The tympanic cavity is bounded laterally by the tympanic membrane; medially, by the lateral wall of the internal ear; it communicates with the mastoidantrum and through it with the mastoid air cells, and the auditory tube[5].

The **auditory tube** (*Eustachian tube*) is the channel through which the tympanic cavity communicates with the nasopharynx. Its length is about 36 mm in adult and 21mm in children.[6] , Its direction is downward, forward, and medially, forming an angle of about 45 degrees (in adults) with the sagittal plane and one of from 30 to 40 degrees with the horizontal plane. It is formed partly of bone, partly of cartilage and fibrous tissue .There is slight anatomical variation between adults and children in terms of length and angulations.

The **tympanic membrane** separates the tympanic cavity from the bottom of the external acoustic meatus. It is a thin, semitransparent membrane, nearly oval in shape, somewhat broader above than below, and directed very obliquely downward and inward so as to form an angle of about fifty-five degrees with the floor of the meatus. Its longest diameter is downward and forward, and measures from 9 to 10 mm; its shortest diameter measures from 8 to 9 mm (in adults)[5].

1.1.2 EPIDEMIOLOGY

The last two decades of the 20th century saw a dramatic rise in OME, largely due to increased pollution and increased use of early childhood day care (where children are exposed to many respiratory infections)[7].

Rosenfield in 1994 noted that about a quarter of OME cases are discovered incidentally during well-child examinations. About 60 percent of children would have OME by the age of two years and 80 percent before school entry [8].

The Agency for Healthcare Research and Quality 1994 OME guideline reported that in one study of children two to six years old in children day carecentre, 53 percent had at least one episode of OME during the first year of the study, 61 percent had at least one episode during the second year of the study, and 30 percent had recurrent OME [8].

Otitis Media with Effusion has a retarditive effect on mental development since it impairs the ability to hear and learning. Therefore more effective strategies to encounter this effect in areas where OME is present need to be established.

MEASUREMENT OF ADENOID NASOPHARYNGEAL RATIO

The radiographic measurement of adenoid hypertrophy was from the lateral view X-rays of the nasopharynx in which the Adenoidal-Nasopharyngeal Ratio(AN Ratio) of 0.0-0.25 was taken as no obstruction and a ratio of 0.26-1.0 was considered as obstruction as demonstrated by Fujioka et al[9] in the figure below.

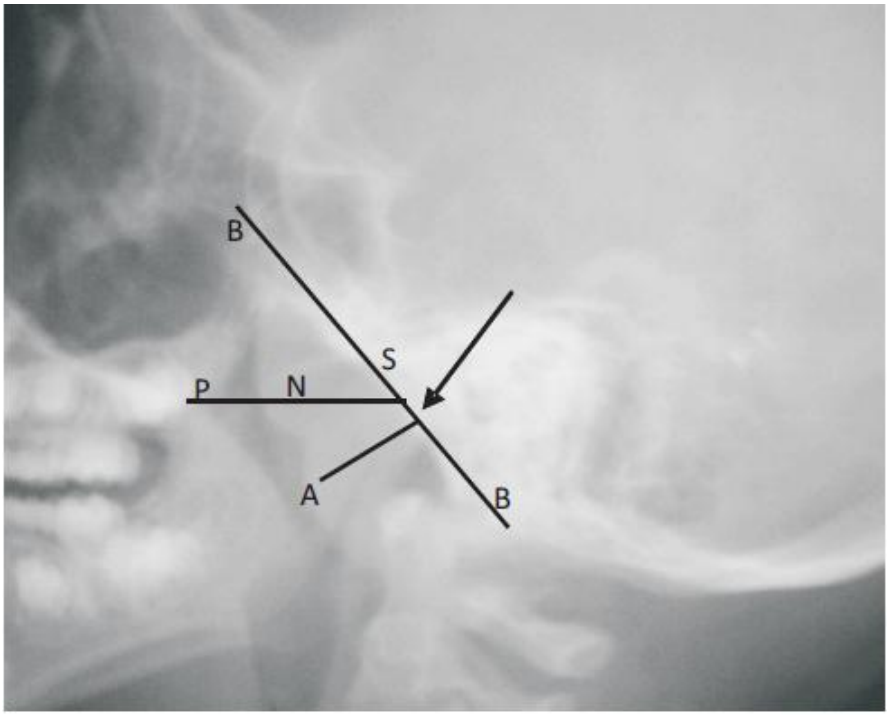

Figure 1.It shows a photograph of postnasal x-ray of one of the patients illustrating the measurements for calculation ofANratio. Line' B'is tangential to the basiocciput. The adenoidal measurement 'A' is obtained by drawing a perpendicular line to B at the pointofmaximal adenoidal tissue. The nasopharyngeal measurement 'N' is made between the posterior border of the hard palateand the antero-inferior aspect 'S' of the spheno-basiccipitalsynchondrosis (black arrowhead). When the synchondrosis is notvisible, point 'S' is determined as the point on the anterior edge of the basiocciput which is closest to the intersection of thelinesAand B .

1.1.3 DIAGNOSTIC TESTS FOR OME

Various methods have been proposed for the diagnosis of OME. These include pneumatic otoscopy and tympanometry. The OME guideline panel drew several conclusions regarding diagnosis of OME[7]. They recommended the use of tympanometry as a confirmatory diagnostic method.

TUNING FORK TESTS

These have limited value in diagnosing OME. They are also unreliable in children

TYMPANOMETRY

The handheld tympanometer is a device that provides quantitative information on the function of structures and the presence of fluid in the middle ear. The graphic display of this data is the tympanogram.

Tympanometry is performed by inserting into the ear canal a probe that emits a tone and measures the amount of sound energy reflected from the tympanic membrane as a function of ear canal air pressure.

The instrument may or may not be handheld. The output of tympanometric measurement may be qualitative, that is, tympanogram patterns, or quantitative, for example static admittance, equivalent ear volume, tympanometric width, tympanometric peak pressure, or acoustic reflex.

Tympanomety has an important place in the evaluation of children with middle ear disorders. Ease of use, acceptance by patients, reproducibility of results and availability of low cost machines has led to wide use of tympanometry in physicians' offices.

By plotting the amount of acoustic energy reflected from the TM as the pressure in the EAC is varied from -400daPa to -100daPa,a tympanogram is obtained, the shape of which provides considerable information about the status of the middle ear.

The flat or type B tympanogram is believed to be associated with the presence of middle ear effusion. The type atympanogram is believed to indicate normal middle ear status. The relationship of the type C tympanogram to middle ear status is less clear [8]

When the tympanogram is completely filled or impacted with effusion, compliance is low and the tympanogram is flat, which signify that reflected energy does not vary with the pressure change. This shape was labeled type B by Jerger[10].

Type B curves may be completely flat or have rounded compliance peak, which is often below -300daPa.

*Figures 2 and 3*depict various tympanogram tracings based on variations of the original Liden and Jerger classifications[11]. The middle curve in *Figure 2*is from a normal ear. The tympanogram curve has a normal maximum height that occurs at a pressure close to zero and the width of the curve is normal. This is referred to as a type A tracing. In this figure, the ear canal volume is normal. *Figure 2*also has a curve that demonstrates a high peak height, labeled as type AD. High static admittance can result from an overly mobile tympanic membrane caused by disarticulation with the bony structures of the middle ear, or a tympanic membrane that has healed over a perforation but is thinner and more compliant thanexpected[10.] The lowest curve in *Figure 2*is a type AS tracing, with a reduced peak height, recorded from middle ears with some fluid or ossicular fixation that partially decreases mobility [13]

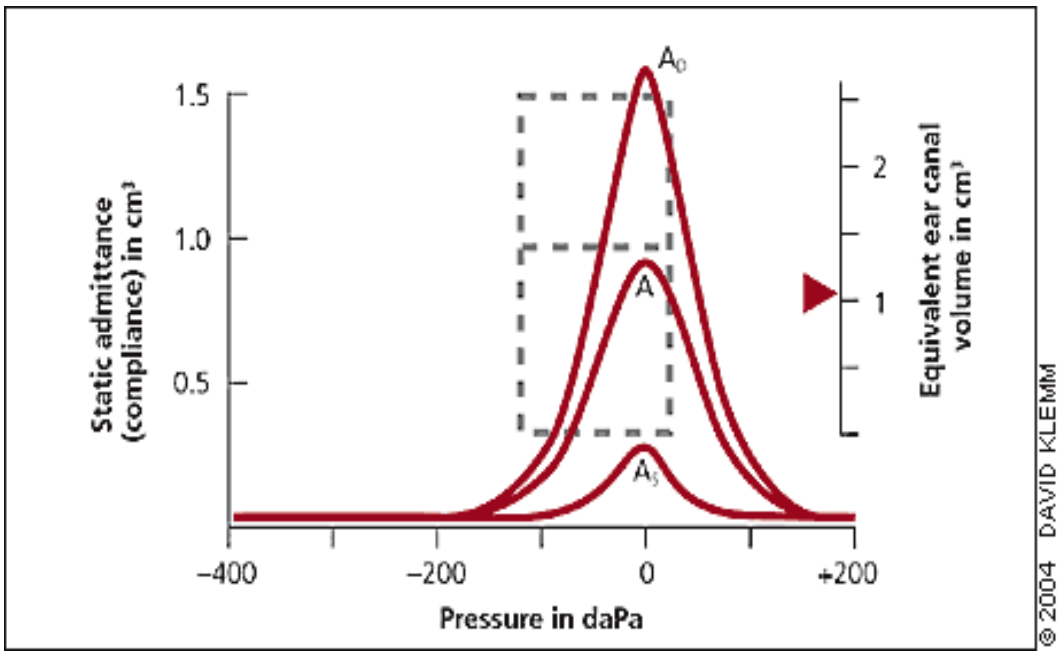

Figure 2. Type atympanogram. Type AD has a high peak height. The middle curve is normal.Type AS has a reduced peak height.

The right vertical axis represents the equivalent ear canal volume measured in cm3 and is indicated with a triangle in each tympanogram. The left vertical axis represents the staticadmittance (or compliance) measured in cm3. Static admittance and peak pressure are normal for adults (older than 10 years) if the curve crosses into either of the two stacked interrupted-line rectangles. They are normal for children if the curve crosses into the lower of the two rectangles.

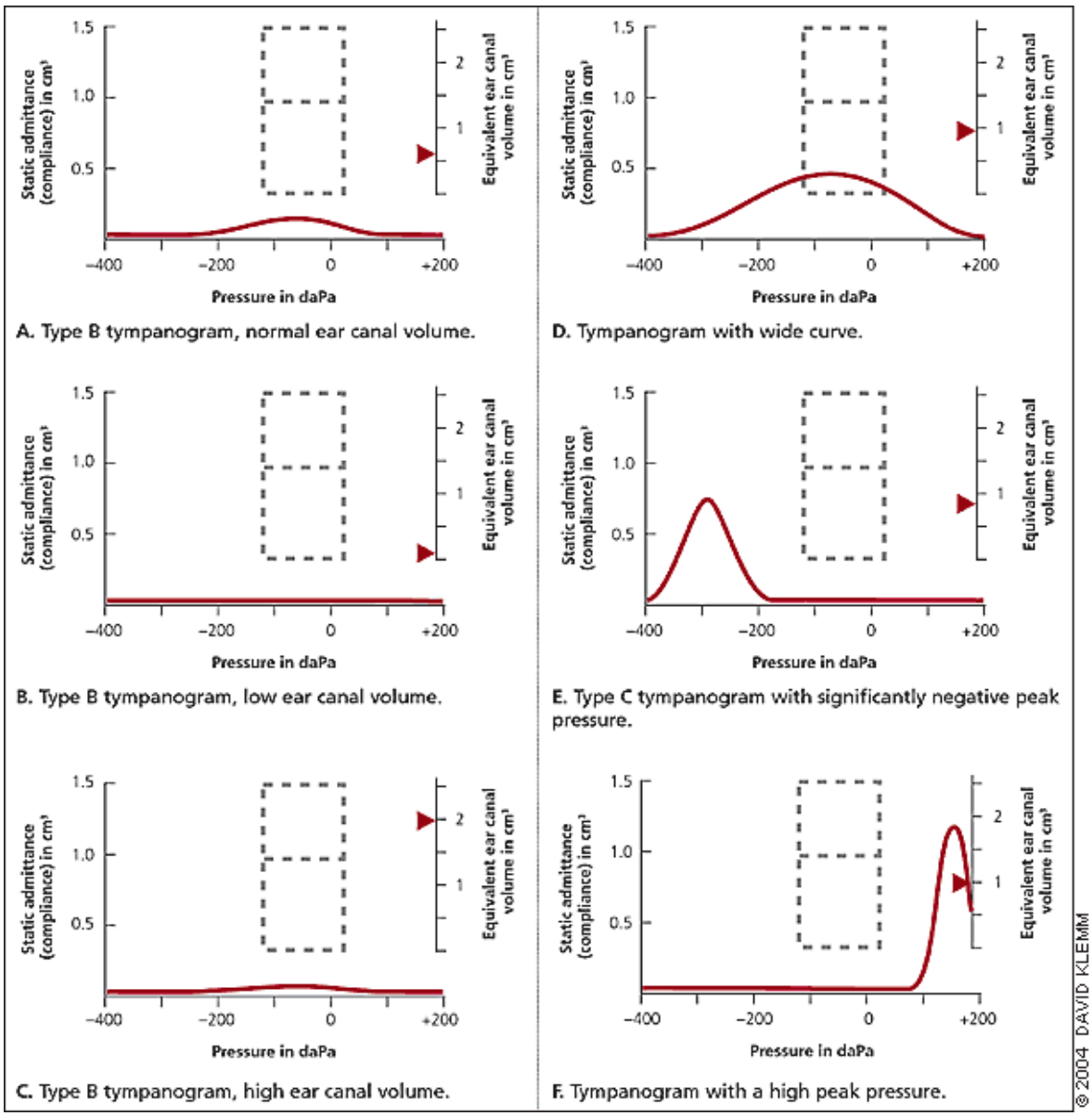

Figure 3.Ais a flattened or type B tracing, with a low static admittance. The ear canal volume is normal. The most common cause of this pattern is decreased mobility of the tympanic membrane secondary to middle ear fluid (OME). Other causes are increased stiffness of the eardrum (from scarring), tympanosclerosis (the formation of dense connective tissue around the auditory ossicles), cholesteatoma, or middle ear tumor [14]. When evaluating the efficacy and clinical usefulness of tympanometry, many studies consider only a type B tracing as definitely abnormal [15].

Bilateral tympanograms will be recorded by specially trained audiometrist for each child selected using a calibrated Kamplex AT2 tympanometer with a 220-Hz probe tone. The lower pressure limit of this machine is − 300 daPa. The tympanogram traces were categorized by an audiological scientist using Fiellau-Nikolajsen's modification of Jergers classification: [16].

A = Peak at + 100 daPa to – 100 daPa1 C = Peak at – 101 daPa to – 200 daPa 2 C = Peak at – 201 daPa to – 300 daPa B = Flat (compliance < 0.2 ml)U = Unclassified (e.g. child refused test, incomplete trace, ear blocked by wax) methodology

PURE TONE AUDIOMETRY

Effusion that completely fills the middle ear cleft usually results in a moderate conductive hearing loss, however small amount of effusion may not alter hearing sensitivity.

Audiometry is impractical in children less than 2 years old but it is recommended in decision algorithm for electing surgical drainage of middle ear. It is recommended by the Agency of American Health Care Policy and Research (AHCPR) guidelines [16].

The acoustic middle-ear muscle reflex, either ipsilateral or contra lateral, may also be measured by acoustic emittance instruments and represents the contraction of the stapedius and tensor tympani in response to sound stimulation. Its absence may be related to the presence of middle ear effusion depending on the clinical situation[8].

ACOUSTIC REFLECTOMETRY

Acoustic reflectometry is performed using a handheld instrument that measures the response of the tympanic membrane to a frequency-sweep sound spectrum. The spectral gradient angle, which is a function of the frequency and amplitude, may be related to middle ear effusion presence [8].

Distinguishing features between AOM and OME is that children with OME present no evidence of acute inflammation despite visible fluid or reduced mobility on pneumatic

otoscopy.The ear is not acutely painful, but the child may have ear discomfort and/or hearing loss.

Children with AOM present with combination of ear pain (otalgia) loss of landmarks and an opaque bulging, inflamed TM on direct otoscopy.

1.1.4 RISK FACTORS

A study done by Skull et al in Australia found that the risk factors for MEE are younger age,family history of ear infection, previous history of tympanostomy tubes,ethnicity, and day care centre attendance[7]

The incidence declines with age except for slight upward trend between 5 and 6 years since this is the age where most of children start kindergarten.

The age specific incidence of OME parallels that of acute infection; the peak was at age of 6 to 13 months in Boston children and 10 to 12 months in Nashville children. Persistence effusion of the middle ear was more likely in young children i.e. between 2 to 5 years of age [6].

Reton et al found that approximately 50% of children of 2 years of age or younger had effusion that lasted for 4 weeks only, 20% of children older than 2 years had OME of this duration. Similar findings were noted in Danish surveys of healthy children [17]. OME and high negative pressure were more frequent in children 1 – 4 years of age than in children aged 7 years and older.

In Danish studies of healthy children the findings were similar with high incidence of OME between 1 to 4 years than in those older than 7 years [18].

Studies done in Boston revealed that about 50% of children aged 2 years or less had OME secondary to AOM, another study was done by Pelton et al which showed that 20% of children older than 2 years had OME which persisted for 1 month or more [19,17] .

SEX:

Males have a higher incidence of AOM than females. In the Boston study, males had significantly more single and recurrent episodes [20]. In Finnish males had significantly more episodes than did female in the study done June 1978[21].

Another Finnish study done showed that males had more episodes than females[22].More males undergo myringotomies and tympanoplasties than females a fact that shows that chronic or severe infections of ME are common among males[23].

RACE:

Eskimos, America Indians and Hispanic children have higher incidence than America white, Blacks have a lower incidence of OME than whites. These differences may be due to anatomical differences in ET in term of length and width [24]

SOCIAL/ECONOMIC CONDITION

OME is common in low socioeconomic classes. Factors like Crowding, poor hygiene,indignant nutrition, delay in medical services and poor compliance with treatment increase the occurrence of OME[6].

In some studies done in Pittsburgh children revealed that children living in urban areas have higher incidence of OME than those leaving in rural areas. The reason is considered to be the higher density of population in urban area especially those with day care centers.[6]

Cambon et al noted a strong association between OME and poor social economic conditions among native Americans of British Colombia.[25]

In the Boston study children living in overcrowded household were more likely to have OME than the children living in household with few members [6].

DAYCARECENTER

The setting for child day care in the preschool years (child's home, other home, family based care or large center facility) is an important factor in the incidence of OME. The more the respiratory pathogens the higher rate of respiratory infection including AOM which is a precursor of OME

Dannish studies revealed that children cared outside the home have higher incidence of OME than those cared at home [26].
Early placement of the child out of home, group day care may discourage breast feeding hence increasing the risk of OME

Current estimates are that more than 11 Million children receive full or part- time day care. More than 50% of mother who have children < 6 years of age work outside the home [6].

Day care centers vary in size and others are crowded and poorly ventilated. Therefore there is opportunity for spread of respiratory infection among the children attending day care than those receiving care at homes.
Day care attendance has increased substantially in the last 25 years [6].

SEASON

Studies done in Pittsburgh revealed OME occurring in winter months persisted longer than effusion occurring in summer months[6].

SMOKING

Smoke exposure can result in structural and physiological changes in respiratory tree such as goblet cell hyperplasia and mucus hypersecretion, ciliostasis and decreased mucociliary transport.[27]

Etzel et al demonstrated high incidence of AOM and increased duration of OME in children exposed to smoke.[28]

ALTERED HOST RESPONSES AND UNDERLYING DISEASES

These include immunodeficiency (congenital or acquired), Drugs which suppress immunity,Anatomical defect, Cleft palate ,cleft uvula, Sub mucus cleft,PatulousEustachian tube,Down syndrome[68].

ANTIBIOTICS

Wide spread use of antibiotics has been implicated, resulting in either incomplete resolution of AOM,or a change in virulence of organisms. It has been postulated that antibiotics interfere with local Immunoglobulin M production in the middle ear[68].

GENETIC FACTORS

Genetic predisposition to ME infection as well as OME has been documented. This is related to the genetic anatomical abnormalities in skull,nasopharynx,Eustachian tube and immune response.[29]

BREAST FEEDING

Breast feeding has preventive factors against URTI including AOM and therefore OME.[30]

RECURRENT ATTACKS OF AOM

OME may follow recurrent attacks of AOM. Merchant et al (1984) in a study of 70 babies followed from birth to one year showed that 54% had one or more episodes of AOM,10 of them developed bilateral middle ear effusions.

VIRAL RESPIRATORY INFECTION

Acute OME is increased in children with history of viral respiratory infection. The viruses which have been implicated are;influenza virus,adenovirus,respiratory syncyntial virus and rarely parainfluenza and rhinovirus.

1.1.5 AETIOLOGY

The etiology and pathogenesis of OME are not yet fully understood, however, multiple factors and complex interaction of biochemical, immunologic and inflammatory mediators in the middle ear cavity have been documented in various studies. Among these factors abnormal function of the ET mucosal changes, presence of microorganisms, the effect of inflammatory cells seem to have the most influence on the etiology and pathogenesis of OME [31,32].Eustachian tube dysfunction is also caused by adenoid hypertrophy and subsequently causing OME[33]

EUSTACHIAN TUBE

The ET has at least three physiologic functions with respect to the ME which are:

1. Protection from nasopharyngeal secretions and pressure. Experimental studies done revealed that there is surfactant protein in the middle ear cleft which is similar to the one found in the alveoli and have immunoreactive properties [22].

2. Clearance of secretion from ME into the nasopharynx[22,34].

3. Ventilation of the ME to equilibrate air pressure with atmospheric pressure [35,36]

The factors which may affect eustachian tube may be considered under three main groups.

i. **Factors causing basic Eustachian tube malfunctio**n.e.g cleft palate. This is related to abnormal patency of Eustachian tube followed by unwanted nasopharyngeal secretions which may enter the middle ear, with or without negative pressure.

ii. Factors leading to altered mucociliary systems

Examples are infections of the sinuses, nose, postnasal spaces, tonsils and pharynx, allergy; altered immunological factors; surfactant deficiency; ultrastructural changes in cilia; fibrocystic disease; hormonal factors such as high oestrogen levels

and hypothyroidism. These lead to poor clearance of middle ear fluid with resultant accumulation of fluid in the middle ear[39].

iii. Factors leading to nasopharyngeal disproportion

These include craniofacial abnormalities as found in Down's syndrome, Hurler syndrome and Hunter's syndrome, and tumor of the postnasal space. These pose a mechanical disadvantage to clearance of the middle ear fluid. Adenoid hypertrophy has been incriminated[39].

1.1.6 PATHOGENESIS

Infection or allergic reaction resulting in congestion of the respiratory mucosa of the nose, nasopharynx and ET obstructs the narrowest portion of the tube, the isthmus. This obstruction causes negative middle ear pressure followed by OME.

Anatomic or physiologic abnormality of the ET can cause recurrent episodes of AOM or persistent OME.

The hypothesis that abnormal function of ET is important factor in the pathogenesis of ME disease was first suggested more than 100 years ago by Politzer.[37]

Studies done revealed that inflammation due to infection or allergy may cause intrinsic mechanical obstruction of the ET.[31,32]

A much smaller proportion of children have mucosal disease of middle ear as a result of allergy itself.[38,39]

The structural TM damage which can occur is due to the reason that effusion contains leukotrienes, prostaglandins andarachidonic acid metabolites that invoke a local inflammatory response [40]. Reactive changes may occur in the adjacent TM and mucosal linings. A relative under ventilation of the middle ear produce a negative pressure that predispose to focal retraction pockets, generalized atelectasis of the TM and cholesteatoma.

After blockage of ET, reaction in the mucosal lining develops in phases as follows:

1. Lack of ventilation creates a “hydrops ex vacuo”.Fluid is sucked into the tympanic cleft. The mucosa become swollen, hyperaemic and oedematous. The basal membrane thickens and its fiber network is altered. Therefore the middle ear is filled with effusion. Anaerobic conditions, due to oxygen resorption by the mucosa, favour epithelial metaplasia.The flat epithelium becomes higher and ciliated.Secretory elements and goblet cells develop, and gland-like structures are build up.Mucous is produced as an important component of the effusion.[6]
2. Blocked ET with intact ear drum membrane prevents the clearance of effusion away from the tympanum. This brings to the whole elaboration of the middle ear content to the mucosa. Finger like projections and connective tissue buds plunge into the effusion, starting a tissue proliferation. Various granulomas are built up, for example cholesterol granulomas.Enzyme production helps to digest this mass of fatty degenerated or mucoid discharge. A lot of microphages can be seen, overloaded with debris, like lipophagocytes or foam cells.[41]
3. The reticulohistiocytic system of the middle ear lining also proliferates. A mononuclear diffuse cellular infiltration can be seen locally around the vessels.Focus – like aggregations are seen, similar to lymphocytic nodules.[41]
4. Epitheloid cells and giant cells surrounded by lymphocytes can be seen, which is a sign of special irritation. Without restoration of tubal function, the abilities of the mucosa are overcharged and defective healing results. Effusion under organization. Fatty deposits, hyalinization, dystrophic calcification and strands of scar tissue narrow the tympanic cleft. Also there is proliferation of collagen fibers in the submucous connective tissue. These will end up in the atelectasis of the middle ear.[6]

MICROBIOLOGY

In the past OME was thought to be non infective condition.[22]Recently Rainsanem, Stenfors and Senturia et al identified bacteria by means of smear and culture and by Polymarase Chain Reaction (PCR) in about 42 % of children with OME.[42]

Bacteria,mycoplasma and viruses have all been shown to be present in some samples.[43]

The type of microorganisms isolated are similar to those isolated in AOM predominantly B- hemolytic streptoccocci and H.infuenza.

1.1.7 CLINICAL PRESENTATION

OME in its quiescent phase has none of the symptoms and signs attributed to the infection of the middle ear. Therefore the most frequent presentation is fluctuant, latent or overt hearing loss. When latent in infants and young children, it may present with impaired speech and language development. There may be behavioral difficulties and scholastic retardation. Children rarely complain of hearing difficulty. In the western world it is frequently first detected on routine screening tests, either clinically, audiometrically or by impendence studies. Commonly found are indirect symptoms such as shouting, insularity, increasing the volume of the television or delay in reading development.

Recurrent otalgia usually results from secondary infection of the middle ear fluid and so frequently coincides with a cold or URTI, sinusitis or an episode of allergic rhinitis or following swimming.

Tinnitus is another feature of OME. The patient may complain of presence of flies and bees in the ear.

Dizziness is another reported symptom. OME is the commonest cause of vertigo in children. The affected child is noted to hold onto his seat momentarily during a spell of vertigo.

1.1.8 DIAGNOSIS

Diagnosing OME correctly is fundamental to proper management; moreover OME must be differentiated from AOM.

OME may be asymptomatic or patients may experience ear discomfort, hearing loss, tinnitus, possibly vertigo, feelings of ear fullness most of these vague presentation can be noted and expressed by adult but in young children the features frequently noted are hearing loss, unstable gait and language problems.

Often the child with OME is so accustomed to reduced hearing sensitivity that parents become aware of the problems only after the child turns up the volume of the radio or television or is not attentive during conversation.

1.1.9 COMPLICATIONS OF OME

A. CONDUCTIVE HL

Persistent or fluctuating HL is present in most of children who have OME usually mild to moderate in the range of 20 and 30 dB where the softer speech sound and voiceless consonants may be missed.

The HL is influenced by quantity and quality of fluid in the middle ear, i.e. ears with thin fluid are not as impaired as ears with glue like fluid and ears that are partially filled with fluid have less hearing impairment than ears that are completely filled with fluid.

Permanent CHL can occur secondary to irreversible changes that result from recurrent acute a chronic inflammation (e.g. adhesive OM or ossicular discontinuity).

B. SENSORINEURAL HL

This can be attributed to effect of increased tension or stiffness of round window membrane. Permanent SNHL may occur presumably due to spread of infection or products of inflammation through round window membrane.[36,38,30]

C. LINGUISTIC AND LEARNING EFFECTS

Impaired cognitive, language and emotional development in children. When effusion is chronic, surgical intervention should be considered especially when antimicrobial therapy fails.

Untreated OME, especially if it is bilateral, has been associated with delayed language development and poor learning. It is especially significant in children with many episodes of OME during the language formative years and is due to the prolonged periods of hearing loss. Those children with recurrent episodes have been found to perform poorly compared to their counterparts. In contrast to adults, a 25 dB hearing loss is catastrophic for children's speech development and learning. Fluctuating hearing loss is also confusing to children.

D. ATELECTASIS, RETRACTION AND CHOLESTEATOMA

Atrophy of the tympanic membrane can follow long standing negative middle ear pressure. The atrophy can be localized or generalized. Since the lamina propria is very thin, even minor pressure can cause retraction.

Localized atrophy frequently occurs in combination with tympanosclerosis. Perforation of the atrophic region may occur following a slap or blow to the ear, ear syringing, diving, or following episode of acute suppurative otitis media. Following perforation the chances of spontaneous healing are minimal.

Retraction pockets may occur on the retracted TM. In the attic, the formation of attic cholesteatoma may occur.

In diffuse atrophy, there may be large perforation of the TM that does not heal. A tensa retraction may progress to cholesteatoma involving the incus and malleus, and atelectasis. The atelectasis is extremely difficult to arrest or correct and may lead to erosion of the outer attic wall and ossicles.

E. TYMPANOSCLEROSIS

Long standing OME may be associated with tympanosclerosis. This has been associated with TM perforation and interferes with hearing. However it is commoner following ventilation tube insertion.

1.1.10 MANAGEMENT OF OME

Management of OME is still controversial. Correct management approach remains to be defined, although there are many forms of treatment for this condition. However we now have some evidence based information to make some important decision regarding treatment or no treatment and which therapeutic options are effective and which ones are not [44].

Management of the effects of the effusion on hearing thresholds varies according to the duration and severity of the hearing loss. Many cases of OME resolve spontaneously without treatment, while a significant number require intervention.

Since this condition shows seasonal variation, and that relapses and remissions may occur several years after the prescribed treatment has been completed, it is therefore difficult to formulate an empirical approach to management of this condition.

OME in most children will need treatment if it persists for two or 3 months, but treatment may be indicated in some children because there are possible complications and sequelae associated with this condition [45].

Since impairment of hearing of some degree usually accompanies OME [46], treatment may be warranted when long standing HL is present . This is because such a loss may impair cognitive and language developmentfunction and result in disturbances in psychosocial adjustment

Important factor which should be considered when deciding to treat or not to treat are:

- Significant conductive HL
- Occurrence in young infants, since they are unable to communicate about their symptom and may have suppurative disease
- An associated acute suppurative URTI
- Concurrent permanent conductive or SNHL
- Presence of speech or language delay associated with effusion or HL.
- Alteration of TM such as retraction pocket.
- Middle ear changes e.g adhesive OM or osscicular involvement
- Previous surgery for OM e.gTypanostomy tube placement or adenoidectomy
- When episodes occur frequently
- When effusion last for 3 months or longer both ears or 6 or more months in one ear

MEDICAL TREATMENT

Use of decongestants and antihistamine have failed to demonstrate efficacy in eliminating OME in children [22,47] .However they can be used in children with recurrent episodes with evidence of upper respiratory allergy.

Inflation of the ET middle ear using Politzer's method or the valsava maneuver has been advocated for more than a century for OME however studies by Chan and Bluestone [47],found lack of efficacy and therefore not recommended except in adults with barotrauma.

Of the medical treatment advocated antimicrobial agent can be helpful in those children who have not received antibiotic recently.

There is no convincing evidence to recommend the use of steroids in the management of OME.

A study reported by Rosenfeld and Post [48], confirmed efficacy, particularly when the effusion is chronic,surgical intervention should be considered especially when antimicrobial therapy fail.

Recent guideline has recommended either antimicrobial therapy ortympanostomy tube insertion for bilateral, chronic effusion (i.e. 3-4 months)associated with HL [49].

SURGICAL TREATMENT

Myringotomy with tympanostomy tube placement

The potential benefits of prolonged ventilation of the middle ear were first recognized by Politzer (1867) and Hinton (1860) who created a permanent perforation by burning a hole in the TM using galvano-cautery,and inserting foreign materials such as catgut or silver canulae to maintain patency[38].There efforts had little success until 1954 when Amstrong succeeded in maintaining a plastic tube in position.

The ventilation tube acts as temporary substitute for the eustachian tube. The ventilation of the middle ear therefore improves and the epithelium gradually reverts to normal. Glandular activity is reduced and ciliary activity resumes to normal. During this period the restoration of normal ET should be the objective, failure of which will result into recurrence of effusion when the ventilation tube ceases to function.

A neat radical incision is made through TM in the anteroinferior or anterosuperior quadrant. The fluid is aspirated and the ventilation tube is inserted where it is found necessary to prevent early closure of the incision.

Immediate hearing improvement is achieved by this procedure. The mean duration of the survival of functioning ventilation tube is around 6 to 12 months. This depends on many factors including the epithelial migration of the TM and external auditory meatus, the tube

design, size of myringotomy incision and the size of middle ear cavity on the face of atelectatic tympanic membrane.

Ventilation tubes are associated with more complications compared to myringotomy alone .These includes short-lived otorrhoea, scarring and tympanosclerosis of the TM and rarely the persistent TM perforation. Extrusion of the tube is also a complication which requires re-insertion of the ventilation tubes on one or more occasions until spontaneous resolution occurs.

ADENOIDECTOMY AND ADENOTONSILLECTOMY

Myringotomy and adenoidectomy with or without tympanostomy tube insertion have been demonstrated to be effective in children with chronic OME who are unresponsive to antibiotic.

Adenotonsillectomy as practiced to date is based on indications other than OME.A study done by Bluestone et al demonstrated radiographically retrograde obstruction of the eustachian tube in relation to OME and adenoids hypertrophy [23].

Paradise et al carried out a well controlled prospective study which confirmed improvement in recurrent otitis media and OME following adenoidectomy [50].Other studies demonstrated disagreement.

OTHER SURGICAL MEASURES

Surgical intervention can be done in the other obvious cause affecting the eustachian tube function, example earlier closure of cleft palate which results to improvement of aural condition. Other measures such as turbinate resection may reduce obstruction due to hypertrophic turbinates in indicated cases.

SUMMARY OF MANAGEMENT OF OME

i. OME without significant hearing loss, which is hearing of more than 20 dB in speech frequencies, requires no intervention. These cases require watchful waiting and close follow up.

ii. Bilateral OME with significant hearing loss require observation and follow up for three months. If no spontaneous recovery myringotomy and aspiration of fluid is indicated at the first instance. If fluid recurs, myringotomy with ventilation tube insertion is done. This may be repeated if needed.

iii. Unilateral OME with or without significant hearing loss,surgical intervention is deferred after special consideration of other factors such as speech, language development and learning.

iv. If there is specific underlying cause of OME, example cleft palate should be managed accordingly.

v. Adenoidectomy should only be done selectively after careful investigations.

vi. Tonsillectomy is not recommended unless there are very specific indications for tonsillectomy in children with OME.

vii. Parents should be made aware of the possibility of ventilation tube extrusion after 6 to 12 months which may need re-insertion in about 25% of cases.

viii. The status of the TM with respect to the degree of atelectasis will indicate the need for bilateral tube insertion. Limitation of tube insertion to one ear for bilateral cases greatly reduces the complication rates.

Approximately 2.2 million diagnosed episodes of OME occur annually in US.[52]

OME may occur spontaneously because of poor ET function or as an inflammatory response following AOM.

Approximately 90% of children (and 98% of individual ear) have OME at some time before school age, most common between 6 month and 4years.[51]

In the 1st year of life, >50% of children will experience OME, increasing to >60% by 2 years [51].

Many episode resolve spontaneously within 3 months, but 30% - 40% of children have recurrent OME and 5% - 10% of episodes last 1 year or longer.[51]

Children get more OME than older children or adults due to several reasons.

-The Eustachian tube is shorter, more horizontal and straighter (quick andeasy trip for the bacteria)

-The tube is floppier with a tinier opening (easily to block)

-Young children get more colds and takes time for immature immune systemto be able to recognize and ward off cold viruses.

Many children with OME do not have past histories of AOM but it is suggested that in them there may have been a sub clinical infection [51.]

Otitis media with effusion is among the most common pediatric health problems especially among young children[52]
It appears to be the most common precursor of chronic OM and may lead to SNHL [10]

From the studies done in one third of children with AOM were found to have OME that persisted for 4 or more weeks [24].

Another study done in Boston children showed persisted OME after AOM which showed persistent OME in 10% of children after 3 months following episode of AOM [6]

In a study done in Boston, persistent OME in the middle ear was found in 70% of children 2 weeks after 1st episode of OM, 40% at 1 month, 20% at 2 months and 10% at 3 months [6].

Studies done by Weichbold and Rohrer in Germany in 2004 in forty seven nursery schools where total of 183 children were screened for the presence of OME,63% aged between 3 to 5

years and among them 64% had temporary CHL due to external or middle ear problems (glue,ETD, OM or cerumen).[50]

A study done in Nigeria by Orji F T,Okolugbo N E,Ezeanolue B C showed the prevalence of OME of 35% in patients with adenoid hypertrophy[53]. It also showed that the proportions of males and females diagnosed with OME were not significantly different 69% by 33%.

In 1998 Lyn and Jadusingh studied 2202 Jamaican children aged 5 to 7 years old. 4.9% had hearing impairment and OME was present in 2%, which was less than in developed countries [54].

Another study done by Kramer AH,McCullonghamong 126 Inuit children the prevalence of OME was studied.4% had OME causing significant HL,17% had OME with minimal or no detectable HL. Therefore prevalence of OME was between 4% to 21% [55].Total of 284 children between 3 to 8 years of age in Southern India were studied in 1997 for the overall prevalence of otological abnormality (excluding wax)which was 21.5%.Hearing impairment was detected in 34 children (11.9%).Otitis media with effusion was noted in 17.6 %.Therefore the importance of including tympanometry as part of screening protocol was highlighted [56].

In the study done at Turkey in Kindergartens, day care centers in rural and central areas of Trabzon revealed that prevalence of OME was 11.14% and the risks factors noted were low socioeconomic factors, URTI,AOM and recent antibiotic use.

Another study done in hospitalized patients in Denmark in 1988 revealed a prevalence of 68 % of OME in children 0-6 yrs of age [18].

Study done in Malaysia revealed prevalence of OME among preschool children between 5-6 yrs was 13.8% [18].

The study done in China by REN Dong and Wang Wu qing related MEE and hearing loss in which 5.2% had severe to profound hearing loss and 55.8% had slight moderate hearing loss[57] Children found to have no hearing loss were 29.7%.

The study done by Paparela et al showed the peak age for developing MEE is at 2 years of age[58]

The study done by Sorensen et al in children 4 years old in post winter season showed prevalence of bilateral MEE by tympanometry to be 7%. Left MEE of 14.5%, right MEE of 12.9% and MEE in at least one ear of 20.4%.[59]

In 1985, 480 children between 0 to 23 months were studied in Venda South Africa for the presence of ME pathology by pneumatic otoscopy,tympanometry and contralateral acoustic reflex. Prevalence of OM was 8.2%.Among those with OM 3.8% had OME which was different from the study done in Nigeria.

Study done at Lagos teaching hospital in Nigeria between January 2002 to June 2003 among children aged 6 months to 15 years those presented in ENT clinic , Among 184 patients HL was documented in 120 (65.2%) children, speech disorders in 56 (30.4%) ,of those with HL, 70% had delayed speech and language. Etiological factors recorded for communication disorders were among others OME contributed 4.3%.Others were meningitis, seizures, congenital etc. Early detection and follow up was recommended in early years of life[60].

In a study done at Maputo among children 3-7 years revealed 49.2% of children with Otitis media with effusion which is strongly associated with history of adenoiditis and/or Eustachian tube dysfunction[4]

A study done in Istanbul, Turkey showed no difference between tympanogram findings with adenoid hypertrophy[61]

Astudy done in Israel by Greenfeld et al showed the peak age for adenoid andtonsillarhypertrophy is 3- 6 years.[62]

1.2 STATEMENT OF PROBLEM

OME appears to be most common precursor of chronic otitis media and may lead to SNHL[63].

The magnitude of MEE in children with adenoid hypertrophy in Tanzania is not well understood.

OME has been noted to cause deafness at critical time in a child's language and speech development [52]

The hearing loss, language and speech delay problem is evident in some of the children in Tanzania although the etiological factors are not clearly known.

There are very few studies which have been done to show the magnitude of MEE in most of developing country such as Tanzania. Adenoid hypertrophy is a common clinical disorder in Tanzania and surgery is often indicated because of its obstructive effects in nasal passage and ET, but no studies have been done in this country to find out its association with MEE.

This study will therefore be the beginning of other studies in Tanzania which will initiate strategies of screening children for presence of MEE and its complications and therefore promote early intervention to prevent complications which follows unintervenedMEE among children with adenoid hypertrophy.

1.3 STUDY RATIONALE

This study was intended to evaluate the magnitude of MEE in children with adenoid hypertrophy at MNH in DSM which will sensitize the clinicians and other health care providers to develop the habit of screening the children at risk and therefore early intervention (in indicated children) so as to prevent the possible complications.

Monitoring and intervention of persistent MEE is important so as to prevent the complications which may follow MEE such as HL, language development and impaired cognitive perception.

This study will also be done as a requirement of the partial fulfillment of the master of medicine degree in ORL at Muhimbili University of Health and Allied Sciences

2.OBJECTIVES OF THE STUDY

2.1Broad objective

To determine the prevalence of MEE amongchildren with adenoid hypertrophy at MNH in Dar-es-salaam.

2.2 Specific objectives

1.To determine the prevalence of MEE among children with adenoid hypertrophy according to age and sex.

2.To determine the symptoms associated with adenoid hypertrophy in children aged 9 years or less.

3.To determine the laterality of MEE among children with adenoid hypertrophy.

3. METHODOLOGY

3.1 Study Location

This study was done at MNH at the ORL department.

3.2 STUDY POPULATION

The study populations included all children with adenoid hypertrophy aged nine years of age or less attending at MNH in which a care provider was asked about the history of the child and then proceed to do examination in order to meet the following criteria.

Inclusion criteria

- All children aged nine years or less.This age was considered important as it was seen in pilot study which was done by a researcher at MNH
- All children with adenoid hypertrophy that had nasopharyngeal narrowing of 0.26 or more on lateral view X-Ray of nasopharynx
- All children with two or more of the following symptoms; mouth breathing, rhinorrhea, snoring or sleep apnea.
- Children whose parents consented for the study were included.

Exclusion criteria

- Children older than nine years.
- Children with craniofacial malformations and those with Down's syndrome.
- Children with symptoms and signs of AOM or COM.
- Children whose caretakers did not consent.
- Children with no symptoms of adenoid hypertrophy.

3.3 STUDY DESIGN

This was a hospital based descriptive cross sectional study.

3.4 STUDY DURATION

The study duration was 9 months, from April 2012 to December 2012

3.5 SAMPLING METHOD AND SAMPLE SIZE DETERMINATION

All children with adenoid hypertrophy attending MNH wereincluded in this study. The formula used to calculate the sample size was developed by the Creative Research system [www.surveysystem.com/sscalc.htm

$$n=\frac{t^2 \times p(1-p)}{d^2}$$

Description:

n = required sample size
t = confidence level at 95% (standard value of 1.96)
p = estimated prevalence of adenoid obstruction in children with OME=32%[53]
d = margin of error at 5% (standard value of 0.05)

n=1.96*1.96*0.32(1-0.32)/0.05*0.05=334

3.6 DATA COLLECTION TECHNIQUES AND TOOLS

After obtaining the relevant research clearance, the investigator started collecting the data

Equipment

i. Diagnostic set which included an otoscope with various sizes of aural specula.
ii. Tympanometer
iii. Cotton wool, spirit and Boric acid ear drop
iv. Cerumen hook
v.

Hearing assessment

This was done by asking the care provider on how the child behaves on hearing sound.

Otoscopic examination:

Each ear was examined by the researcher who is a resident in ORL.Otoscopic examination was done using battery hand handled otoscopy and the wax or foreign bodies which were encountered were removed.

Tympanometry

Tympanometry was done to each ear by the research assistant who is a special trained audiometrist .A flat, non peakingtympanogram image with low static compliance was evaluated as type B tympanograms according to Jergers classification. This was diagnostic for MEE.The findings were analyzed and documented for both ears.

3.7 DATA COLLECTION FORM

Data collected (demographic,otoscopic,and tympanometry)was entered into data collection form by principal investigator.

3.8 DATA PROCESSING AND ANALYSIS

Data were entered after cleaning for accuracy,completeness and internal consistency using SPSS 16.0 computer program. P values of <0.05 was considered significant.

3.9 ETHICAL CONSIDERATION

Ethical clearance was obtained from MUHAS Ethical Committee.

3.10 POSSIBLE CAUSES OF BIAS

- Since this was a hospital based study, only children whose care givers brought them to hospital were included. Some parents do not bring their children to hospital.
- Hearing assessment was done by asking the care provider

3.11 STUDY LIMITATIONS

- There were only few studies done on this topic to show their relationship and therefore difficult in comparing the findings from one country to another.
- The study duration was short and therefore only few children were considered who attended ENT clinic on that particular time.
- The children taken were only those that attend at MNH and therefore cannot be a representative of all children in Dar-es-salaam because majority of care givers have no knowledge on the condition.

4. RESULTS

The results of this study are summarized in the following tables;

Table 1.Population Distribution by Age and Sex

Sex	Age Groups(Years)					Total (%)
	0-2 (%)	>2-4 (%)	>4-6 (%)	>6-8 (%)	>8 (%)	
Female	39 (9.3)	89 (21.2)	55 (13.1)	14 (3.3)	5 (1.2)	202 (48.1)
Male	44 (10.5)	104 (24.8)	51 (12.1)	14 (3.3)	5 (1.2)	218 (51.9)
Total	83 (19.8)	193 (46)	106 (25.2)	28 (6.6)	10 (2.4)	420 (100)

In table 1 above about 51.9% of the children with adenoid hypertrophy attending MNH were males while females were 48.1%. The most affected age group was >2-4 years which accounted for 46% in which males were 24.8% and females were 21.2% with the p value of 0.908.

Table 2.Clinical Features Associated with Adenoid Hypertrophy

Status	Sleep apnea (%)	Snoring (%)	Mouth breathing (%)	Rhinorrhea (%)
No	172 (41)	9 (2.1)	18 (4.3)	156 (37.1)
Yes	248 (59)	411 (97.9)	402 (95.7)	264 (62.9)
Total	420 (100)	420 (100)	420 (100)	420 (100)

In table 2 about 59% of children with adenoid hypertrophy presented with sleep apnea,97.9% presented with snoring, 95.7% presented with mouth breathing and 62.9% presented with rhinorrhea.

Table 3.Prevalence of Middle Ear Effusion by Sex

Sex	No middle ear effusion (%)	Middle ear effusion (%)	Total (%)
Females	77 (18.3)	125 (29.8)	202 (48.1)
Males	84 (20)	134 (31.9)	218 (51.9)
Total	161 (38.3)	259 (61.7)	420 (100)

In table 3 about 61.7% of all children with adenoid hypertrophy had MEE in at least one ear in which males were31.9% and females were 29.8% with the p value of 0.00.

Table 4. Prevalence of Middle Ear Effusion by Age

Age group (years)	No middle ear effusion (%)	Middle ear effusion (%)	Total (%)
0-2	15 (3.6)	68 (16.2)	83 (19.8)
>2-4	86 (20.5)	107 (25.5)	193 (46.0)
>4-6	37 (8.8)	69 (16.4)	106 (25.2)
>6-8	23 (5.5)	5 (1.2)	28 (6.7)
>8	0 (0)	10 (2.4)	10 (2.4)
Total	161 (38.3)	259 (61.7)	420 (100)

In table 4 majority of children with middle ear effusion 25.5% are from age >2-4 years, however this age group shows a large number of population 46% with the p value of 0.00.

Table 5. Prevalence of Middle Ear Effusion by Age and Side

Age group(years)	MEE Bilateral (%)	MEE Left Ear (%)	MEE Right Ear (%)	Total (%)
0-2	48 (18.5)	15 (5.8)	5 (1.9)	68 (26.3)
>2-4	98 (37.8)	4 (1.5)	5 (1.9)	107 (41.3)
>4-6	60 (23.2)	5 (1.9)	4 (1.5)	69 (26.6)
>6-8	5 (1.9)	0 (0)	0 (0)	5 (1.9)
>8	5 (1.9)	0 (0)	5 (1.9)	10 (3.9)
Total	216 (83.4)	24 (9.3)	19 (7.3)	259 (100)

In table 5 about 83.4% of all children with adenoid hypertrophy with MEE had bilateral MEE, 9.3 had MEE on left ear and 7.3 had MEE on right ear. The age group >2-4 was more affected by bilateral MEE by 37.8%.The group 0-2 was more affected by MEE on the LT ear by 5.8%. The RT ear was affected almost equally in all age groups with the p value of 0.00.

Table 6. Prevalence of Hearing Loss in Children with Middle Ear Effusion

Hearing loss	Middle ear effusion (%)
Yes	4 (1.5)
No	255 (98.5)
Total	259 (100)

In table 6 above 1.5% of the children with MEEpresented with hearing loss and about 98.5% had no hearing loss. P value 0.282

Table 7.Prevalence of Tonsillitis in Children with Adenoid Hypertrophy by Age

Age(years)	Tonsillitis		Total (%)
	No (%)	Yes (%)	
0-2	74 (17.6)	9 (2.1)	83 (19.8)
>2-4	100 (23.8)	93 (22.1)	193 (46.0)
>4-6	15 (3.6)	91 (21.7)	106 (25.2)
>6-8	0 (0)	28 (6.7)	28 (6.7)
>8	5 (1.2)	5 (1.2)	10 (2.4)
Total	194 (46.2)	226 (53.8)	420 (100)

In table 7 above about 53.8% of children with adenoid hypertrophy presented with tonsillitis. The most affected age is from over 2-4 years by 46% with the p value 0.00.

5. DISCUSSION

Adenoid hypertrophy can partially or completely obstruct the nasopharyngeal airway. The airflow passing through in the relatively narrower lumen will cause a negative pressure which will induce tubal dysfunction in the most severe cases, and this can only be eliminated by means of adenoidectomy.[64, 65-66]However, the main clinical problem for some children with symptoms of OME is occult and is easily neglected by the parents, in terms of the poor expression and communication skills of the children. If the ear canals are narrow, it is very difficult to examine the tympanic membrane, and thenOME can easily be neglected.

Wang et alidentified 63 cases in China (77.8%) with OME of 81 cases of severe adenoid hypertrophy[67], this correlate with the finding of my study which shows the prevalence of MEE among children with adenoid hypertrophy of about 61.7%.The difference in percentage can be due to difference in sample size in which my sample size is about four times the sample of that study. It can also be due to race difference in which blacks have a lower incidence of OME than whites. These differences may be due to anatomical differences in ET in term of length and width.[24]

The results of my study is also different from the study done in Nigeria by Orji F T,Okolugbo N E,Ezeanolue B C which showed the prevalence of OME among 46 patients with adenoid hypertrophy of about 35%[53].The difference in findings can be due to a small sample size used by the researcher.

Both males and females were almost equally affected by adenoid hypertrophy regardless of a small difference which is notsignificant of about 52% by 48% respectively, this is different from the study done by REN Dong-dong and WANG Wu-qing which showed that males were more affected by 63% and females were affected by 37%.[57]

With regard to MEE the results of this study shows that males were significantly affected by 31.9% as compared to females which were affected by 29.8% with the p value of 0.00.This finding is different from the study done in Nigeria by Orji F T**,**Okolugbo N E,Ezeanolue B C which showed no significant difference in proportions between males and females (0.69 by 0.33 respectively).[53]

The age group of over 2 years to 6 years was most affected by adenoid hypertrophy (71.2%). This can be due to attendance to nursery and primary school which is normally started at this age. This finding correlates with the study done by Sasaki,CT which showed that the age group between 2-6 years has largest size of adenoids.[2]

It also correlate with the study done in Israel by Greenfeld et al which showed the peak age for adenotonsilar hypertrophy at the age of 3-6years.[62]

Each of the children included in this study had post nasal airway narrowing in which the Adenoidal-Nasopharyngeal Ratio of 0.26-1.0 on radiographic findings was considered as adenoid hypertrophy. Other symptoms which were observed in these children were snoring which was seen in 97.9% of all children, mouth breathing in 95.7% of all children, rhinorrhea in 62.9% of all children and sleep apnea in 59% of children as reported by care providers. These findings were similar to the study done in Israel by Greenfeld et al which showed that snoring was the most common finding which was seen in 100% of infants with adenotonsillarhypertrophy, sleep apnea in 72% and mouth breathing in 62% of the children.[62]

This study also shows that middle ear effusion is not significantly related to hearing loss.Only 1.5% of the children with middle ear effusion presented with the complain of hearing loss while 98.5% of all children with middle ear effusion has no complain of hearing loss as reported by their care providers (p value 0.282) this correlates with the study done by Kramer AH,McCullonghamong 126 Inuit children which showed a prevalence of 4% with OME causing significant hearing loss[55].In this study the hearing loss recorded was from the complain of care provider of the child, so most of the children with mild to moderate hearing loss were missed and only those with severe to profound hearing loss could be peaked by their care providers.

Another study done in China by Ren Dong-Dong and Wang Wu qing[57] among children with MEE who presented with adenoid hypertrophy had similar findings in which 5.2% of children had severe to profound hearing loss.[61]The researcher used AC-ASSR(Air Conduction-Auditory Steady State Response) to assess the hearing, he found 29.7% had normal hearing,

55.8% had slight mild hearing loss. So thepercentage which could not be recognized by care providers for hearing loss was 85.5% which is close to the finding of my study of 98.5%

The study also shows that Majority of children with middle ear effusion 58.1%% are from the age of 0-6 years, this correlate with the study done in Denmark by Takata et al which showed the prevalence of OME of 68% between 0-6 years.[18] In my study the prevalence of MEE in the age of over 2-6 years was 41.9% which correlates with the study done in Germany by Weichbold et al which showed the prevalence of Otitis Media with Effusion of 63% between 3-5 years.[50]

The study also shows that 51.4% of all children with adenoid hypertrophy presented with bilateral Middle Ear Effusion, 5.7% presented with Middle Ear Effusion on left ear, 4.5% presented with Middle Ear Effusion on right ear and in general about 61.7% presented with Middle Ear Effusion in at least one ear. This is different from the study done by Sorensen et al[59] which showed the prevalence of bilateral Middle Ear Effusion of 7%, left Middle Ear Effusion of 14.5%, right Middle Ear Effusion of 12.9% and 20.4% in at least one ear, this can be explained either by the age included in the study in which in my study I considered children with 9 years or less while Sorensen considered 4 years old children alone. It can also be due to season at which the study was done, my study included all seasons while Sorensen considered only post winter season.

The age group >2-4years was more affected by bilateral Middle Ear Effusion by 37.8%.The age group 0-2years was more affected by Middle Ear Effusion on the LT ear by 5.8%. The RT ear was affected almost equally in all age groups with the p value of 0.00.This results correlate with the study done by Paparela et al which showed the peak age of Middle Ear Effusion at 2years of age[58].

Tonsillitis was also seen to be common among children with adenoid hypertrophy.About 53.8% of children with adenoid hypertrophy presented with tonsillitis. This can be due to recurrent upper respiratory tract infections which occur in these children. The most affected age was the age from over 2 years to 6 years which correlate with the study done by Greenfeld et al which showed the peak age for adenotonsilar hypertrophy to be between 3-6 years.[62]

6.CONCLUSION AND RECOMMENDATION

CONCLUSION

Adenoid hypertrophy is a risk factor for middle ear effusionand the most affected age is over 2 years to 6 years with male preponderance.About 83.4% of all children with adenoid hypertrophy had bilateral MEE.

About 59% of children with adenoid hypertrophy presented with sleep apnea, 97.9% presented with snoring, 95.7% presented with mouth breathing and 62.9% presented with rhinorrhea.

RECOMMENDATIONS

- Practitioners should pay much attention to the middle ear condition and be aware of a possible development of severe to profound hearing loss during the course of MEE in adenoid hypertrophy in young children.
- It is important to perform the middle ear examination and audiological assessments for children with adenoid hypertrophy before surgery.
- Follow up is very important after surgery in order to know the improvement of MEE.
- The otoscopy and tympanometry can make a more accurate diagnosis of pediatric MEE in the adenoid hypertrophy children with parental suspicion of hearing impairment.
- Hearing assessment should include use of ABR or Play Audiometry which will help not to miss majority of children with mild and moderate hearing loss.
- More studies should be done to assess the effect of other risk factors which can contribute to the formation of MEE.

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APPENDICES

Appendix i: Data collection Form

General information:

1. Number……

2. Residence…….

4. Sex……

5. Age (yrs)……...

6.TYMPANOMETRY

Rt Lt

Type A & C …… …….

Type B ……. …….

7. Findings on nasopharynx X-Ray lateral view

Yes No

Post nasal airway narrowing …… ……

8. Otoscopic findings

RT LT

Wax ……. ……..

TM retracted ……. ……

Dull TM ……. ……..

Air bubbles …….. …….

9. Yes No

Tonsillitis …….. …….

10. Symptoms associated with adenoid hypertrophy

	Yes	No
Sleep apnea	……	……..
Mouth breathing	……	……
Snoring	……..	……..
Rhinorrhea	……	……..

Hearing lossas reported by parent or care giver RT …… ……..

LT …….. ……..

Appendix ii

Informed Consent Form(English Version)

MUHIMBILI UNIVERSITY OF HEALTH AND ALLIED SCIENCES

SCHOOL OF MEDICINE,

Department of ORL

INFORMED CONSENT FORM

ID-NO……

Consent to participate in a research study

- Greetings. I am DrBukanuFaustine, a postgraduate student, doing masters of medicine in ORL at Muhimbili University of Health and Allied Sciences. Iam doing research with the objective of services at Muhimbili National Hospital, Dar es Salaam

Purpose of the study: To determine the prevalence of MEE among children with adenoid hypertrophy attendingMuhimbili National Hospital, Dar es Salaam.

Participants of the study

All children with adenoid hypertrophy will be included in this study.

Confidentiality

All the participants who will join the study their names will not be required but will be identified by use of number. The information obtained during data collection will be kept under strict locked environment where it is only the researcher will have access and will be destroyed after the dissertation have been submitted and accepted for the award of my postgraduate degree.

Risk

No harm is expected to occur because of joining the study.

Benefits

The results of this research will help to improve the quality of care of these patients.

Right to withdrawal

Joining in this study is completely your choice. You can withdrawal at any particular moment even after signing the consent form. You can even refuse to respond to any question in the questionnaire or review guide.

Whom to contact

In case of any concern or question about the study you can contact the researchers, DrBukanuFaustine, Dr Henry Swai or Prof. Moshi Ndesarua at Muhimbili University, P.O. BOX 65001, Dar es Salaam. You can also contact Prof M. Aboud the Chairperson of the Muhimbili University Senate Research and Publications Committee, P.O.BOX 65001, Dar es Salaam, for any matters concerning ethical violation of the study.

I ……..have read the contents in this form. My questions have been answered and I agree to participate in this study.

Signature of participant……..

Signature of researcher/research assistant…….

Appendix iii

Informed Consent Form (Swahili Version)

Salaam, miminiDrBukanuFaustine,mwanafunziwashahadayauzamiliyaudaktari,Idaraya ENT,chuokikuu cha AfyaMuhimbili.Nafanyautafitikuangaliakiwango cha majikatikamasikioyawatotowenyematatizoyanyamazakwenyepuakatikahospitaliyataifaMuhimbili.

Lengo la utafiti

Kuangaliakiwango cha majikatikamasikiokwawatotowenyenyamakatikapuakatikahospitaliyataifaMuhimbili.

Usiri

Majinayawashirikiwautafitihuuhayatahitajikabadalayakezitatumikanambazautambuzi. Habarizotezitakazokusanywawakatiwautafitizitatumikakwawahusikawautafititunazitaharibiwa baadayaripotiyautafitikuwaimekubalikakwaajilikutunukiwashahadayauzamili.

Washirikiwautafiti

Wagonjwawotewenyenyamazakwenyepuawatahusishwa. Washirikiwatafanyiwauchunguziwamasikiowakawaidanakwakutumiavifaaambavyohavihatarishihaliyamasikio.

Madhara

Hakunamadharayanayotarajiwakwawashirikiwautafiti.

Faida

Matokeoyautafitihuuyatasaidiakuboreshahudumazamasikiokwawagonjwawenyenyamazakwenyepuanajamiikwaujumla.

Hakiyakujitoa

Ushirikikatikautafitiniwahiyari, mshirikiyoyoteanahakiyakuamuakujitoakatikautafitiwakatiwowotekujitoahakutaathirikiwango cha hudumakwamgonjwa.

Mawasiliano

Kama kunashidayoyote au maswalikuhusuutafitihuuunawezakuwasilianawatafiti Dr Bukanu Faustine, Dr Henry Swaina Prof. Moshi Ndesaruawa Chuo Kikuuch Muhimbili S.L.P. 65001, DSM. Piaunawezakuwasilianana Prof M. Aboudmwenyekitiwakamatiyautafitinamachapisho Chuo Kikuu cha Muhimbili, S.L.P.65001, DSM.

Mimi ……nimesomanakuelewamaelezoyaliyokwenyefomuhiinama swaliyanguyamejibiwananinakubalikushirikikwenyeutafitihuu.

Sahihiyamshiriki……..

Sahihiyamtafiti/mtafitimsaidizi…….