HISTOLOGICAL EVIDENCE OF ADENOID MAST CELLS INDICATING A ROLE IN OTITIS MEDIA WITH EFFUSION (OME)

By

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PENGENALAN

'Otitis media with effusion'(OME) ialah radang telinga tengah di mana terdapat koleksi cecair di telinga tengah sedangkan gegendang telinga tidak berlubang. Penyebab otitis media berkemungkinan pelbagai termasuk hipertropi adenoid, jangkitan (virus atau bacteria), alahan, alam sekitar dan faktor sosial. Hubungan antara adenoid dan OME telah lama dikaitkan tetapi mekanisma sebenar terus dibahaskan. Kami mengkaji peranan sel mas adenoid dalam penyebab OME.

OBJEKTIF

Menkaji taburan dan peranan sel mas adenoid sebagai penyebab 'otitis media with effusion'(OME).

METODOLOGI

Kajian prospektif keratan rentas telah dijalankan di Klinik Otorinolaringologi, Jabatan Otorinolaringologi, USM dari Jun 1999 sehingga September 2001. Sejumlah 50 kes di kaji. 25 pesakit telah menjalani pembedahan adenoidektomi sementara 25 pesakit lagi telah menjalani adenoidektomi dan miringotomi bersama kemasukan tiub ventilasi. Kesemua adenoid telah diambil

dan diwarnakan dengan 'toluidine' biru dan dikira menggunakan mikroskop cahaya. Keputusan dianalisis dengan perisian SPSS versi 10.0.

KEPUTUSAN

Sebilangan besar pesakit dengan kronik 'adenoiditis' mengadu berdengkur sementara 'otitis media with effusion'(OME) mengalami kurang pendengaran. Audiometri menunjukkan sebilangan besar dengan OME mendapat kurang pendengaran pertengahan jenis konduksi. Populasi sel mas adenoid dalam OME lagi padat dari pesakit tanpa OME (nilai p = 0.000). Tidak ada beza antara umur dalam kedua-dua kumpulan. Tidak ada kaitan antara kaum dan jantina dengan keputusan.

KESIMPULAN

Bilangan tinggi sel mas dalam pesakit mengidap kronik 'adenoiditis' dengan 'otitis media with effusion' mengesyorkan radang mempunyai peranan sebagai penyebabnya.

ABSTRACT

INTRODUCTION

Otitis media with effusion (OME) is an inflammation of the middle ear in which a collection of liquid is present in the middle ear space while the tympanic membrane is intact. The pathogenesis of otitis media is multifactorial which includes factors such as adenoids hypertrophy, infection (viral or bacteria), allergy, environment and social factors. The association between adenoid and OME has long been noted but the exact mechanism is still much debated. We studied the role of adenoid mast cells in the causation of OME.

OBJECTIVE

To study the distribution and a role of adenoid mast cells in the causation of otitis media with effusion.

METHODOLOGY

A cross-sectional prospective study was carried out in Otorhinolaryngologic Clinic, Department of Otorhinolaryngology (ORL), Science University of Malaysia (USM) from June 1999 to September 2001. A total number of 50 cases were studied. 25 of the patients studied underwent adenoidectomy while another 25 patients underwent adenoidectomy and myringotomy with ventilation tube insertion. The adenoid specimens from all patients were taken

and studied for the number of adenoid mast cell using toluidine blue as the staining agent and light microscopy to count the number of mast cell. The results were analysed using SPSS version 10.

RESULTS

The majority of the patients with chronic adenoiditis had snoring while most of the patients having otitis media with effusion had hearing impairment. Audiometry showed most patients having otitis media with effusion had moderate conductive hearing loss. The population of adenoid mast cell of children with OME was significantly denser than without OME(p value = 0.000). There is no significant difference between age in the two groups. No statistically significant correlation was found between race and gender with outcome.

CONCLUSION

The increased number of adenoid mast cells in patients having otitis media with effusion suggest that inflammation may play a role in their pathogenesis.

CHAPTER 1

INTRODUCTION

1:INTRODUCTION

Otitis media effusion (OME) is an inflammation of the middle ear in which a collection of fluid is present in the middle ear space while the tympanic membrane is intact. The presence of a relatively asymptomatic middle ear effusion has many synonyms, such as secretory, nonsuppurative or serous otitis media but the most acceptable term is otitis media with effusion. Since the effusion may be serous (transudate) the term secretory may not be correct in all cases. Likewise, the term nonsuppurative may not be correct, since asymptomatic middle ear effusion may contain bacteria and may even be purulent. The term serous otitis media is appropriate if an amber or bluish effusion can be visualized through a translucent tympanic membrane; however, the most frequent otoscopic finding is opacification of the tympanic membrane, which makes assessment of the type of effusion (ie serous, mucoid or purulent) not possible (Bluestone and Klein, 1995).

Pneumatic otoscopy frequently reveals either a retracted or convex tympanic membrane in which the mobility is impaired. However, fullness or even bulging may be visualized. In addition, an air-fluid level or bubbles, or both may be observed through a translucent tympanic membrane. The duration (not the severity) of the effusion can be acute (less than three weeks), subacute (three weeks to two to three months) or chronic (longer than two to three months). The pathogenesis of otitis media is multifactorial which includes factors such as adenoids hypertrophy, infection (viral or bacteria), allergy, environment and social factors (Bluestone and Klein, 1995).

Socioeconomic factors such as overcrowding, poor diet and lack of health care may contribute to the development of otitis media. Certain ethnic groups notably Native Americans have a high prevalence of OME presumably due to differences in the anatomy of their eustachian tube and skull base. Differences in OME rates by race may also reflect differences in access to medical care, biologic susceptibility and socioeconomic status. Other risk factors include male gender, bottle feeding and position of feeding (children fed while supine are at greater risk for OME than are children held upright).

Cigarette smoking in the home appears to be a risk factor for OME. Children in day care have more OME than do children reared at home because of endemic upper respiratory infections in groups. Similarly, children in the early school grades have an increased frequency of OME. It has been well established that antecedent upper respiratory tract infections is a major risk factor for OME (Daly, 1991; Gates, 1998).

The association of allergy and OME continues to be a subject of debate. Epidemiologic studies have made it difficult to draw significant conclusions and the results of immunologic studies in humans are equivocal (Juhn, Tolan et al 1991). The mechanism by which allergy might cause otitis media remains hypothetical and controversial. One or more of following mechanisms might be involved (Bluestone 1978):

1. Middle ear mucosa functioning as a "shock (target) organ".
2. Inflammatory swelling of the mucosa of the eustachian tube.

3. Inflammatory obstruction of the nose.

4. Aspiration of bacteria-laden allergic nasopharyngeal secretions into the middle ear cavity.

After studying middle ear mucosa for IgE, Philips et al (1974) postulated that allergy may play a role in the pathogenesis of OME after finding increased number of IgE in patients with OME. Yamashita and colleagues (1980) observed formation of middle ear effusion that contained eosinophils in addition to other inflammatory cells following ovalbumin injection into the middle ear cavity of guinea pigs previously sensitized to this antigen. Changes in the middle ear mucosa and proximal eustachian tube showed inflammatory infiltration. In contrast, those sensitized animals that underwent nasopharyngeal challenge did not show involvement on the middle ear or eustachian tube but rather inflammatory infiltration in the nasal cavity and pharynx.

Doyle and colleagues (1984) demonstrated that nasal antigenic challenge is capable of inducing a functional eustachian tube obstruction in the chinchilla and as a consequence felt that prolonged changes of this nature could lead to OME. Bernstein and coworkers (1985) investigated the role of IgE-mediated hypersensitivity in 100 children with recurrent otitis media who were divided into non-allergic and allergic groups, based on their history and physical examination, prick testing for selected antigens, and total IgE and specific IgE radioallergosorbent test (RAST) analysis. Following aspiration of the children's middle ears and testing for IgE they concluded that 35% of the 100 children

may have had IgE-mediated allergy as a cause of their effusion and that in 8 % of the children the middle ear was a possible target organ. In the other 27 % they postulated that the eustachian tube might have been the target organ.
To evaluate the role of type 1 hypersensitivity reactions in the pathogenesis of OME, Doyle and colleagues (1985) used a passively sensitized rhesus monkey model. There was no otomicroscopic, tympanometric or histologic evidence that antigen (pollen) was capable of inducing middle ear inflammation with effusion suggesting that local allergic reactions do not contribute to the pathogenesis of OME.

Most researchers consider OME a sequelae of eustachian tube dysfunction related to the adenoid due to the proximity of the adenoid to the pharyngeal opening of the tube and the improvement of OME following adenoidectomy. However, the exact role of adenoid in the pathogenesis of OME has not been fully elucidated.

In a prospective study by Maw (1983) on the effects of adenoidectomy and adenotonsillectomy in established otitis media with effusion showed that adenoidectomy alone confer a significant therapeutic benefit compared with control. However, the addition of tonsillectomy did not give any increased benefit from adenoidectomy alone.

Gates et al (1987) studied the effect of adenoidectomy on OME in four groups of children. Two groups had myringotomy or myringotomy and ventilation tubes. Two groups had adenoidectomy combined with either myringotomy or

myringotomy and ventilation tubes. The two groups treated with adenoidectomy did significantly better than the others.

Paradise, Bluestone et al (1990) studied the efficacy of adenoidectomy in 213 children who had received ventilation tubes because of persistent or recurrent otitis media. Ninety-nine of the children were assigned randomly to either an adenoidectomy group or a control group.114 children whose parents withheld consent for randomization were assigned according to parental preference (non-randomized). In both trials, adenoidectomy group outcomes were more favorable than control group.

Further more, children with OME who have smaller palatal airway than the mean for normal population, clearance of effusion is greater following adenoidectomy than in those children not submitted to pharyngeal surgery (Parker and Maw, 1989 ; Maw and Parker ,1993).

Hearing loss affecting children with OME can be cured or markedly reduced by adenoidectomy (Maw and Herrod, 1986). The improvement in the hearing might be related to the middle ear pressure. Tuohimaa and Palva (1987) carried a study on the effect of adenoidectomy on the intratympanic pressure. The study was carried out in a group of 67 children who underwent adenoidectomy and /or tonsillectomy because of either recurrent otitis media or upper respiratory tract infection by documenting the tympanometry preoperatively and postoperatively. They demonstrated that enlarged adenoids

preoperatively cause highly increased negative middle ear pressure as compared to children without adenoid problem.

It has been suggested that the adenoid plays an important role in OME by mechanical and functional obstruction of eustachian tube and by functioning as a source of bacteria antigens due to an inadequate handling of bacteria during upper respiratory tract infection (Gates et al, 1988).

Similarly, several authors have hypothesized that the adenoid may compress or obstruct the eustachian tube lumen causing middle ear under-pressures and subsequent effusion formation (Cantekin et al ,1977; Elverland et al,1981; Casselbrandt et al, 1988).

On the other hand, several studies had shown the value of adenoidectomy in OME is not related to the adenoid size (Maw, 1983 ; Gates et al, 1987). Other authors observed that there is no correlation between adenoid size and OME (Roydhouse, 1980;Gates et al, 1988). Gerwat (1975) compared the weight of adenoids removed from 39 patients suffering from OME with that from 29 controls and found no difference between them. Interestingly, many children with OME were found to be devoid of adenoids or had previously undergone adenoidectomy (Dawes 1970; Mawson and Fagan, 1972; Kokko, 1974).

Further more, the obstructive role of adenoids in the pathogenesis of OME is lacking as demonstrated by Sade and Luntz (1989). Their histological study of the eustachian tube lumen in OME and acute otitis media failed to show any

obstruction of the lumen or a significant difference between the size of the lumen in OME, acute otitis media and non-pathological specimens.

Although bacteria may be present in humans and experimental animals with chronic middle ear effusion, it is not known whether bacteria play a role in the evolution of chronic OME. The rationale for pharyngeal therapy in OME is removal of a focus of infection and improvement in nasal airflow (van Cauwenberge et al,1995). Most episodes of acute OME follow an upper respiratory tract infection-induced bacterial colonization of the nasopharynx with secondary colonization of the middle ear. Removal of the adenoid, but not the tonsils, appears to interrupt that process (Maw, 1983).

Several investigators have found that bacteria can be cultured from chronic middle ear effusion. Liu et al (1975) found evidence of bacteria in effusion by culture or gram stained smears in 81% of 102 chronic effusions although the percentage of culture positive mucoid effusion (36 %) was approximately one half that of serous effusions (63%). Riding et al (1978) cultured 119 chronic effusions from patients not receiving antibiotics and 55% yielded bacteria; an equal percentage of purulent, serous and mucoid effusions were culture positive. Giebink et al (1979) found that 51% of serous and 50% of mucoid effusions were culture positive in 140 cases of chronic middle ear effusions.

Healy and Teele (1977) reported isolating bacteria from 36 % of 96 ears with chronic effusions; the frequency of culture positive serous and mucoid effusions was identical. Two thirds of their patients under 3 years of age had culture

positive effusion compared with one third of the patients older than 3 years. Liu et al (1975) also reported a higher frequency of culture positive effusions in patients less than 6 years old compared to the older group. Giebink et al (1982) summed up their study by suggesting that bacteria contribute to the pathogenesis of chronic otitis media with effusion by increasing the number of inflammatory cells in the middle ear cleft.

Other researchers however could not find the role of bacteria in OME. Linder et al (1997) studied 60 children from 2 to 12 years old by dividing them into 3 groups; group 1 consisted of patients with a history of recurrent otitis media with effusion or chronic otitis media effusion; group 2 were patients with a history of recurrent or chronic otitis media without effusion at myringotomy; group 3 those with adenoidal enlargement with symptoms of obstruction acting as control. They found no overgrowth of pathogenic organisms in children with recurrent or persistent ear disease. Forsgren, Samuelson et al (1993) also found the number of pathogenic organisms in both the adenoidal hypertrophy and OME group was similar.

The contradictory and divergent data in the literature may be explained by the different sampling techniques (nasopharyngeal swabs versus quantitative bacteriology performed on adenoidal tissue, conventional culture techniques versus polymerase chain reaction) and different patient cohort (Linder et al, 1997).

Collins et al (1985) suggested another possible mechanism "the adenoid mediator release" theory. They have shown that children with fluid present in both ears have increased amounts of histamine in their adenoid tissue compared to those with no signs or symptoms of OME. They postulated that the release of this powerful mediator of inflammation from the adenoid tissue is responsible for initiating and maintaining a local inflammatory reaction in the eustachian tube that may lead eventually to middle ear effusion.

It has been shown by Dennis et al (1976) that histamine induces vasodilatation, increased vascular permeability and edema of the eustachian tube and middle ear mucosa. These inflammatory changes may represent indirect evidence that histamine contributes to eustachian tube dysfunction and OME.

Palva et al (1991) and Berger and Ophir (1994) in two separate researches found increased adenoids mast cell and histamine in patients with OME. Nakata et al (1992) demonstrated high level of histamine in middle ear effusion of chinchillas induced by the introduction of immune complex. Kiroglu et al (1998) performed electron microscopic examination of adenoids in 28 children who underwent adenoidectomy for OME while adenoids taken from 10 children who were operated for foreign bodies in the airway act as control. They noted mast cell was found in abundance in the adenoids of patients with OME compared to control.

The possible reasons for degranulation of mast cells in the adenoids and middle ear are diverse and include not only allergy but also infection and complement reactions (Drake-Lee et al, 1994).

This study is performed to find out whether there is a role of adenoid mast cell in the pathogenesis of otitis media with effusion by examining the histology of the adenoids of patients having otitis media with effusion and comparing it to those not having otitis media with effusion.

1.1 ANATOMY

1.1.1 ADENOID

The adenoid is a collection of lymphoid tissue also called nasopharyngeal tonsil and is found in the mucous membrane overlying the basisphenoid. It has an oblong, rectangular shape, extending from the roof of the nasopharynx. The anterior edge of this block of tissue is vertical and in the same plane as the posterior nasal aperture .The posterior edge gradually merges into the posterior pharyngeal wall. It is covered by epithelium which is thrown into numerous folds separating the lymphoid follicles.

Detectable at around the fourth month of embryonic development embedded in the mucosa at the junction of the roof and posterior nasopharyngeal wall, the adenoid is poorly developed at birth. It is not visible on plain radiographs in infants under the age of one month but is clinically identifiable by the fourth month. It is radiologically demonstrable in all infants by the age of 6 months. By the age of 2 years, hypertrophy and hyperplasia of the adenoid occurs. Rapid growth occurs from 3 to 5 years with a consequent decrease in the nasopharyngeal airway.

The adenoid receives a blood supply from the branches of the ascending pharyngeal artery arises from the medial of the external carotid artery. The veins of the adenoid are arranged in an internal submucous and external

pharyngeal plexus with numerous communication branches. Eventually it drains into the internal jugular veins.

1.1.2 EUSTACHIAN TUBE

The eustachian tube lies at an angle of 45 ^{0} in relation to the horizontal plane. The tube length varies from 31 to 38 mm. The posterior third is osseous while the anterior two thirds is composed of membrane and cartilage. The osseous eustachian tube lies completely within the petrous portion of the temporal bone and is directly continuous with the anterior wall of the superior portion of the middle ear .The junction of the osseous tube and the epitympanum lies 4 mm above the floor of the tympanic cavity. The course of the osseous tube is linear anteromedially, following the petrous apex and deviating little from the horizontal plane. The lumen is roughly triangular measuring 2 to 3 mm vertically and 3 to 4 mm along the horizontal base. The healthy osseous portion is open at all times, in contrast with the fibrocartilaginous portion, which is closed at rest and opens during swallowing or when forced open, such as during the Valsalva maneuver.

The osseous and cartilaginous portions of the eustachian tube meet at an irregular bony surface and form an angle of about 160 ^{0}. The cartilaginous tube then courses anteromedially and inferiorly, angled in most cases 30 to 40 ^{0} to the transverse plane and 45 ^{0} to the sagittal plane. The tube is closely applied to the basal aspect of the skull and is fitted to a sulcus tubae between the

greater wing of the sphenoid bone and the petrous portion of the temporal bone.

The cartilaginous tube is firmly attached at its posterior end to the osseous orifice by fibrous bands and usually extends some distance (3mm) into the osseous portion of the tube. At its inferomedial end it is attached to a tubercle on the posterior edge of the medial pterygoid lamina. The cartilaginous tube has a crook-shaped mediolateral superior wall. It is completed laterally and inferiorly by a veiled membrane which serves as the site for the attachment of the fibers of the dilator tubae or tensor veli palatini muscle. Tubal cartilage increases in mass from birth to puberty. The tubal lumen is shaped like two cones joined at their apices. The juncture of the cones is the narrowest point of the lumen and has been called the "isthmus", and its position is usually described as at or near the juncture of the osseous and cartilaginous portions of the tube. The lumen at this point is 2 mm high and 1 mm wide. From the isthmus, the lumen expands to 8-10mm in height and 1-2 mm in diameter at the pharyngeal orifice. The lumen has a constant height and there are only small differences between the height of the isthmus in the child and that in adult.

The cartilaginous eustachian tube does not follow a straight course in the adult but extends along a curve from the junction of the osseous and cartilaginous portions to the medial pterygoid plate, approximating the cranial base for the greater part of its course. The eustachian tube crosses the superior border of the superior constrictor muscle immediately posterior to its terminus within the nasopharynx. The thickened anterior fibrous investment of the medial cartilage

of the tube presses against the pharyngeal wall to form a prominent fold, the torus tubarious which measures 10 to 15 mm in thickness.

The torus is the site of origin of the salpingopalatine muscle and is the point of origin of the salpingopharyngeal muscle which lies within the inferoposteriorly directed salpingopharyngeal fold. The mucosal lining of the lumen of the eustachian tube is continuous with that of the nasopharynx and middle ear and is characterized as respiratory epithelium. Structural differentiation of this mucosal lining is evident; mucous glands predominate at the nasopharyngeal orifice and there is graded change to a mixture of goblet, columnar and ciliated cells near the tympanum.

1.1.3 THE INFANT EUSTACHIAN TUBE

The eustachian tube in the infant is about half as long as that in the adult; it averages about 18 mm. The cartilaginous tube represents somewhat less than two thirds of this distance whereas the osseous portion is relatively longer than and wider in diameter than it is in the adult. The height of the pharyngeal orifice of the infant eustachian tube is about one half that of the adult but the width is similar. Since the infant (and young child) has a shorter eustachian tube than the older child and adult, nasopharyngeal secretions may reflux more readily into the middle ear through the shorter tube.

The ostium of the tube is more exposed in the infant than it is in the adult, since it lies lower in the shallower nasopharyngeal vault. The direction of the tube

varies from horizontal to an angle of about 10^0 to the horizontal and the tube is not angulated at the isthmus but merely narrows. In infants the medial cartilaginous lamina is relatively shorter since there is less tubal mass and stiffness in the infant tube than there is in that of the older child and adult. The tensor veli palatini muscle is less efficient in the infant.

1.1.4 MIDDLE EAR

The middle ear is an irregular, laterally compressed air filled space lying within the petrous portion of the temporal bone between the external auditory canal and the inner ear. This cavity can be considered to be divided into three parts superoinferiorly in relation to the tympanic membrane. The epitympanum or attic refers to that space lying above the superior border of the tympanic membrane. The mesotympanum lies opposite the membrane and the hypotympanum lies below the membrane. At birth the cavity and associated structures are of adult size. The vertical and anteroposterior diameters measure about 15 mm whereas the transverse diameter measures 4 mm at the epitympanum, 2 mm at the mesotympanum and 6 mm at the hypotympanum. The mucous membrane of the middle ear and mastoid is continuous with that of the nasopharynx via the eustachian tube. This membrane covers all structures within the middle ear including the ossicles, vessels and nerves. Examination of cells of the mucous membrane within the tympanic cavity reveals a gradual change from tall, columnar cells with interspersed goblet cells to shorter cuboidal cells at the posterior portion of the promontory and aditus ad antrum.

1.2 PHYSIOLOGY OF THE EUSTACHIAN TUBE AND THE MIDDLE EAR

The eustachian tube has at least three physiological functions with respect to the middle ear:

(1) Protection from nasopharyngeal sound pressure and secretions.
(2) Drainage and clearance into the nasopharynx of secretions produced within the middle ear.
(3) Ventilation of the middle ear to equilibrate gas pressure in the middle ear with atmospheric pressure.

Ventilation of the middle ear is the most important function of the eustachian tube since hearing is optimum when the middle ear gas pressure is relatively the same as the air pressure in the external auditory canal. Clearance of secretions from the middle ear is provided by the mucociliary system of the eustachian tube and some of the middle ear mucous membrane. In ideal tubal function, intermittent active opening of the eustachian tube due only to contraction of the tensor veli palatini muscle during swallowing maintains nearly ambient pressures in the middle ear. The levator veli palatini muscle is not involved in active tubal function but is involved in velopharyngeal function.

1.3 MAST CELL AND HISTAMINE

Mast cells are widely distributed in most tissues and are particularly numerous adjacent to small blood vessels. Mast cells have large basophilic cytoplasmic vesicles (secretory granules) which can be discharged ('degranulation') by various stimuli for example various causal agents of acute inflammation, releasing histamine and other vasoactive compounds form the cell (Anderson, 1988).

Mast cells bear membrane Fc receptors for IgE. Mast cells are triggered to degranulate when their Fc receptors are cross-linked by the binding of antigen to antigen-specific IgE. Mast cell activation can also be produced by other stimuli such as anaphylatoxins and chemicals such as compound 48/80, codeine and synthetic ACTH. Mast cell degranulation involves an influx of calcium and activation of cylic AMP as a second messenger (Maran and Lund, 1990).

Mast cell mediators fall into 2 groups:

1. Those which are preformed and exist within granules packaged with glycosaminoglycan. These include histamine, heparin, proteolytic enzymes such as tryptase and B-glucosaminidase and chemotactic and activating factors-eosinophil chemotactic factor, neutrophil chemotactic factor and platelet activating factor.

2. Newly synthesized membrane –associated mediators. These result from changes in the plasma membrane associated with activation which allows phospholipase A2 to release arachidonic acid which is metabolized by lipoxygenase or cycloxygenase enzymes. The newly synthesized mediators include prostaglandin A2, thromboxane A2 and leukotrienes eg LTC4 and LTD4 which are the slow reacting substances of anaphylaxis and LTB4 which is a chemotactic molecule.

Histamine is present in inactive form in mast cells. It can be released from the mast cell by inflammatory stimuli. Histamine causes active hyperaemia and increased vascular permeability (Hurley, 1988).

CHAPTER 2

STUDY OBJECTIVES

2:OBJECTIVES

GENERAL OBJECTIVE:

To study the distribution and a role of adenoids mast cells in the causation of otitis media with effusion (OME).

SPECIFIC OBJECTIVES:

1. To count the number of mast cells in enlarged adenoids of patients with or without otitis media with effusion.

2. To obtain a correlation between the number of mast cells in adenoids and the causation of otitis media with effusion.

CHAPTER 3

METHODOLOGY

3: METHODOLOGY

3.1 STUDY DESIGN

A cross sectional observational-comparative study.

3.2 POPULATION, SETTING AND TIME

Study population: Patients who were diagnosed to have enlarged adenoids with or without otitis media with effusion in Otorhinolaryngology Clinic, USM Hospital (HUSM).

Period of study: June 1999 to September 2001.

Place of study: Otorhinolaryngology Clinic of Department of Otorhinolaryngology, and Histopathology Laboratory of Department of Pathology, School of Medical Science, Universiti Sains Malaysia (USM).

3.3 SAMPLE SIZE

Total number of patient	50 cases
Chronic adenoiditis	25 cases (control)
Chronic adenoiditis and otitis media with effusion	25 cases

3.4 SELECTION CRITERIA

3.4.1 INCLUSION CRITERIA

1. All patients between the age of 3 years to 12 years.
2. Patients having chronic adenoiditis without otitis media with effusion for at least 6 weeks to serve as control group.
3. Patients having chronic adenoiditis with either unilateral or bilateral otitis media with effusion to serve as the active group.

3.4.2 EXCLUSION CRITERIA

1.Patients with history and /or on examination to have otitis externa.
2.Examination showing tympanic membrane perforation with or without infection.
3.Absence of symptoms and signs of OME for more than 12 months.

3.5 CONSENT

Consent was taken from the parents for the respective operation(s) in the usual manner.

3.6 METHODS

Only patients in the age range of 3 to 12 years old were selected in this study. Their demographic data were recorded in the data collection form.

SYMPTOMS (Collins et al, 1985; Brodsky, 1989; Berger and Ophir, 1994):

Every patients and their parents were asked specifically about the symptoms.

In the case of **chronic adenoiditis**, the symptoms specifically asked were long term nasal blockage, nasal discharge, mouth breathing, snoring and findings confirming obstructive sleep apnea.

In **otitis media with effusion** the symptoms sought were ear blockage, otalgia, tinnitus and hearing impairment.

EXAMINATION (Collins et al, 1985; Hibbert, 1987; Brodsky, 1989 ; Van Cauwenberge et al ,1995; Bluestone, 1998) :

All the patients were examined by an otorhinolaryngologist or a senior otorhinolaryngology medical officer. Examination findings were noted as following.

The appearance of the patient specifically for adenoid facies(opened mouth, broad upper lip, short nose, prominent upper teeth or triangular face). Anterior

rhinoscopy in all cases and posterior rhinoscopy (where possible) were performed to confirm the presence of enlarged adenoids and exclude other obstructive causes such as deviated nasal septum or nasal polyp.

Examination of the ears on both sides was performed either by using otoscope or operating microscope. The character of the tympanic membrane was determined as appearance whether normal, dull or retracted, presence of fluid in the middle ear as air bubbles or fluid level and the colour of the tympanic membrane that is yellow, grey, blue or amber.

The tuning fork tests, Rinne's and Weber's tests were performed using 512 Hertz tuning fork whenever possible or reliable and the findings were recorded.

3.6.1 METHOD OF IMAGING

All the patients were then subjected to a soft tissue lateral neck x-ray (true lateral view). The patient was positioned in the erect position and the head fixed with a wall-mounted cephalostat and oriented with the Frankfort horizontal plane. The cassette of the film was centred 1 inch below the level of the external auditory meatus. The tube cassette distance was 180 cm. The central ray was directed perpendicularly to the midpoint of the film. The film was taken during inhalational phase of quiet nasal breathing to ensure filling the passages with air. We documented the size of the adenoids according to the system used and describe by Cohen et al (1992).

Radiologically determined size of the adenoids:

1+ : normal sized adenoids for a child of that age along with a normal nasopharyngeal airway.

2+ : moderate enlargement of the adenoids with moderate narrowing of the airway.

3+ : marked enlargement of the adenoids with an airway nearly occluded.

4+ : massive enlargement of the adenoids along with total airway occlusion.

3.6.2 METHODS OF HEARING ASSESSMENT

Hearing assessment was done by certified audiologists by using tympanometry in all ages from 3 to 12 years old while play audiometry was performed for those between 3-6 years old and pure tone audiometry for patients between 7-12 years old.

Tympanometric measurement was made using the middle ear analyzer model GSI 33 version 2.0 serial A 1354 (Garson-Stadler Inc., USA). It is provided with multiple probes and printer. Standardization is carried out periodically or when needed.