

## Introduction

Otitis media with effusion is a leading cause of conductive hearing loss in children. It is defined by a chronic effusion in the middle ear cavities, behind an intact tympanic membrane without acute infection. (*Portier et al., 2001*).

Otitis media with effusion is characterized by a non specific inflammation of the middle ear mucosa and secretory transformation of the epithelial layer, resulting in fluid accumulation in the middle ear space (*Ovesen et al., 2000*).

Many sequential studies have reported that between 20% and 50% of children will have an episode of otitis media with effusion at some time between the ages of 3 and 10 years (*Browning, 1997*).

The pathogenesis of otitis media with effusion is multi- factorial which includes factors such as infection (usually viral or bacterial), eustachian tube dysfunction, immunological status, environmental and even social factors (*Bluestone and Klein, 1985*).

Dang et al., 1998 found that the organic Sequale of otitis media with effusion are changes in the tympanic membrane such as atrophy, retraction, and tympanosclerosis and changes in the mastoid, middle ear and inner ear.

Prolonged otitis media with effusion carries the risk for further progression into adhesive otitis media, chronic suppurative otitis media, and cholesteatoma, which cause serious hearing defects later in life (*Dang et al., 1998*).

The pneumatization of mastoid begins on 33<sup>rd</sup> wk embryologically and continue up to 8-9 years of age. The mastoid doesn't properly develop and has a sclerotic structure in patients with cholesteatoma or chronic

suppurative otitis media, it is not clear whether the chronic middle ear disease leads to inadequate development of the mastoid or vice versa (*Bayramoğlu et al., 1997*).

*Tos et al., 1984* found that the environmental influence as disturbance of the genetically governed process of the pneumatization, which may be arrested by inflammation and infection of the middle ear mucosa resulting in a reduced air cell system.

The size of air cell system is thus the result of the genetic and the environmental factors on the process of pneumatization. The development of mastoid air cell system taking place during the first years of life, is disturbed by episodes of acute otitis media and secretory otitis media and the result is hypocellularity, which, accordingly, must be regarded as a sequel to pathologic influence on the middle ear. (*Tos et al., 1984*).

*Aoki et al., 1998* found that the growth of the mastoid cells and the middle ear transmucosal gas exchange function are closely affected by the sub epithelial histopathologic changes in middle ear mucosa and these two factors recover by the treatment of otitis media with effusion.

It's well known that most patients with chronic suppurative otitis media, and cholesteatoma have reduced pneumatization of mastoid process, but it's still a subject of controversy whether hypocellularity is the cause or the result of chronic otitis media (*Sadé and Hadas 1979*).

*Bayramoğlu et al., 1997* suggested that the mastoid pneumatization might be considered as a prognostic indicator in secretory otitis media and the estimated prognosis is poor when the mastoid pneumatization is poor.

Negative middle ear pressure is known to produce otitis media with effusion (*Makoto et al., 1998*).

Middle ear pressure is regulated by transmucosal gas exchange as well as by Eustachian tube (*Hergils and Magnuson 1988*) .

The gas exchange function through mastoid mucosa was correlated well with the mastoid pneumatization degree and that loss of the function was found to be related to pathogenesis of several middle ear diseases (*Tanabe et al., 1997* ).

Previous studies suggested several techniques for measurement of mastoid pneumatization (*Chatterjee et al., 1990*) In order to predict the prognosis of otitis media with effusion in patient treated with ventilation tube insertion (*Rock 199*).

Recently *Görür et al., 2006* suggested the use of post operative tympanometric follow up of patients with otitis media with effusion.