Introduction

Tympanosclerosis is a degenerative healing process of the middle ear structures by hyalinization and calcification, and it often develops after inflammation when the process is limited to the tympanic membrane alone, the term myringosclerosis is preferred ***(Cüneyd Uneri et al., 2006)***.

The etiology and pathogenesis of tympanosclerosis is not completely understood yet. It has been suggested that tympanosclerosis is caused by immunological reaction, infection, inflammation, genetic tendency or trauma ***(A. Ko Ç, C. Uneri, 2002)***.

Ventilation tube is the accepted surgical treatment of chronic otitis media with effusion ***(D.N. Riley et al., 1997)***.

Myringosclerosis often occurs in patients who underwent tympanostomy and had ventilation tube insertion, the incidence of tympanosclerosis after ventilation tube insertion differs between 25 and 35% ***(Johnston LC, 2004)***.

Recent studies have established the relationship between the reactive oxygen species and the development of myringosclersis after tympanic membrane trauma or ventilation tube insertion ***(K. Gorur et al., 2002)***.

The main treatment for patients with tympanosclerosis is surgery but recurrences are common and there is also a risk for iatrogenic sensorineural hearing loss associated with this kind of surgery ***(Karlidag T et al., 2004)***.

Free oxygen radical have been implicated as important pathologic mediators in many diseases, under physiological conditions they are part of normal regulatory circuits and neutralized by anti-oxidants ***(Yariktas M et al., 2004)***.

Vitamin E is a potent chain-breaking antioxidant that affects on development of myringosclerosis after ventilation tube insertion ***(Cüney D Uneri et al., 2006)***.