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ORIGINAL ARTICLE

## How does childhood otitis media change the radiological findings of the temporal bone?

TULAY ERDEN HABESOGLU<sup>1</sup>, MEHMET HABESOGLU<sup>1</sup>, SEMA ZER TOROS<sup>1</sup>,  
ILDEM DEVECI<sup>1</sup>, MEHMET SURMELI<sup>1</sup>, SHAHROUZ SHEIDAEI<sup>1</sup>, AHMET BARAN<sup>2</sup>  
& EROL EGELI<sup>1</sup>

<sup>1</sup>Department of Otolaryngology - Head and Neck Surgery and <sup>2</sup>Department of Radiology, Haydarpaşa Numune Education and Research Hospital, Istanbul, Turkey

### Abstract

**Conclusion:** Inflammatory changes in the middle ear mucosa since childhood may affect mastoid pneumatization without a change in the position of the sigmoid sinus. Also, despite the fact that recurrences of inflammatory middle ear disease lead to bone destruction, we did not see any relationship between the childhood otitis media and bone destruction. **Objective:** To assess radiological findings in temporal bone in both healthy ears and diseased ears in patients with unilateral chronic otitis media since their childhood. **Methods:** Twenty-five patients who had unilateral otological symptoms, such as recurrent otalgia, purulent otorrhea or hearing loss since their childhood were included in the study. Assessment of radiological parameters was performed using a quantitative digital image processing computed tomography program. **Results:** Mastoid volume values in the chronic otitis media group were significantly smaller when compared with those of a healthy group ( $p < 0.05$ ). In healthy and diseased mastoid groups, there were no significant differences between groups when we assessed Henle spine-sigmoid sinus (HS-SS) distances. There was a significant correlation ( $p = 0.001$ ) in both the groups when we compared mastoid volume values with HS-SS distances. There was ossicular discontinuity in two cases and in one patient tegmen tympani was not intact in diseased ears.

**Keywords:** Mastoid volume, middle ear effusion, Henle spine-sigmoid sinus distances

### Introduction

In general, otitis media (OM) is the most common diagnosis in pediatric patients who visit physicians because of illness [1]. Chronic otitis media with effusion (OME) is characterized by the presence of a middle ear effusion in the middle ear space for 3 months or longer. Children with persistent middle ear effusion also frequently have difficulties with recurrent acute OM [2].

Chronic otitis media (COM), which is usually associated with purulent otorrhea and hearing loss, is an inflammatory disease of the middle ear and mastoid. COM has been shown to be related to recurrent acute OM or OME in childhood [2].

Middle ear infections in childhood may lead to inflammatory changes in the middle ear mucosa and in the development of temporal bone [3], according to the environmental theory, which emphasizes that the extent of mastoid pneumatization is determined by environmental factors. In contrast, according to the genetic theory, the extent of mastoid pneumatization is determined genetically [4]. On the basis of this association, a controversy as to whether the underdeveloped mastoid is a genetic trait or follows an acute OM in childhood has continued for many years [5].

Therefore, this study aimed to assess mastoid volumes and Henle spine-sigmoid sinus (HS-SS) distances in diseased and healthy ears in patients

who had experienced otological symptoms since their childhood and to discuss whether the changes seen were caused by the infection or were the cause of the infection. In addition, since chronic inflammation in the middle ear may lead to ossicular damage, we also evaluated radiological bony findings such as the ossicles, tegmen tympani, scutum, lateral semicircular canal, and facial canal.

## Materials and methods

Twenty-five patients with unilateral COM, who had experienced otological symptoms such as recurrent otalgia, purulent otorrhea or hearing loss since their childhood, were included in the study. The contralateral ears of the patients were healthy. The diagnoses were made according to otomicroscopy, audiometry, and computed tomography (CT). The patients had no history of previous surgical intervention for otological diseases or trauma. When we diagnosed cholesteatoma or adhesive otitis media on otoscopic examination, these patients were excluded. All patients were informed about the purpose of the study and their informed consents were obtained.

In total there were 16 female and 9 male patients with ages ranging from 16 to 34 years. CT scans of the temporal bones of the subjects were obtained with a Hitachi-Pronto AR HP spiral scanner. Contiguous sections with 1 mm thickness were obtained parallel to the orbitomeatal line. The following assessments were made using the measurement properties of the Adobe Acrobat Professional Programme.

- (1) Using a quantitative digital image processing program on temporal bone CT examination, the air cell area of the mastoid bone was calculated. The CT program was adjusted so that it could specifically measure the areas of air or soft tissue densities within selected borders. Selection of the soft tissue areas was made in an effort to include the air cells that had been attacked by the inflammatory process, so that the actual air cell area could be measured (Figure 1). The volume of the mastoid bone was estimated using the morphometric method of Cavalieri. This method is based on the principle developed by the Italian mathematician Bonaventura Cavalieri in 1635, and subsequently updated [6]. The CT images of the temporal bones were projected on a screen for magnification, and the volume of mastoid bone was measured according to Cavalieri's principle [7].
- (2) The shortest distance between the Henle spine and the sigmoid sinus was measured [8].

Also, we evaluated commonly used radiological bony findings such as ossicular discontinuity, tegmen tympani, scutum, lateral semicircular canal, and facial canal erosions.

SPSS (Statistical Package for the Social Sciences) 10.0 for Windows was used for statistical analysis. The resulting data were analyzed using Student's *t* test and Pearson correlations to compare the relationship between the mastoid volume and HS-SS distance in both groups.

## Results

### *Mastoid volume and position of the sigmoid sinus*

In healthy and diseased groups, median values of mastoid volumes were  $4.63 \pm 3.29$  and  $6.75 \pm 4.06$ , respectively. Mastoid volume values in the COM group were significantly smaller when compared with those in the healthy group ( $p < 0.05$ ) (Table I). Also, in the healthy and diseased mastoid groups, median values of HS-SS distances were  $1.05 \pm 0.37$  and  $1.02 \pm 0.38$ , respectively, there being no significant difference between groups ( $p > 0.05$ ).

In both the groups, when we compared mastoid volume values with HS-SS distances, there was a significant correlation ( $p = 0.001$ ) (Figure 2).

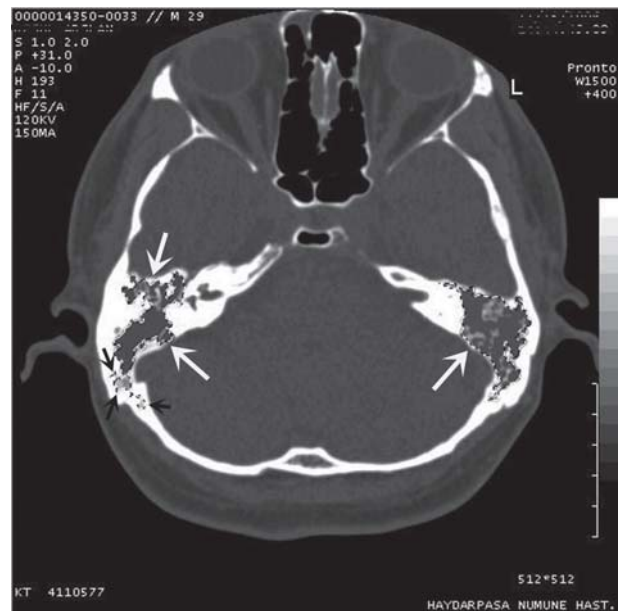


Figure 1. Air cell area of the mastoid bone. White arrows, selection of total air cell area; black arrows, air cells attacked by inflammatory process.

Table I. Comparison of mastoid volume and HS-SS distances among groups.

Parameter	Control group Mean ± SD	COM group Mean ± SD	p value	Student's t test
Mastoid volume (cm <sup>3</sup> )	6.75 ± 4.06	4.63 ± 3.29	0.049*	2.023
HS-SS distance (cm)	10.56 ± 3.73	10.26 ± 3.85	0.775	0.288

HS-SS, Henle spine-sigmoid sinus.

\*p < 0.05.

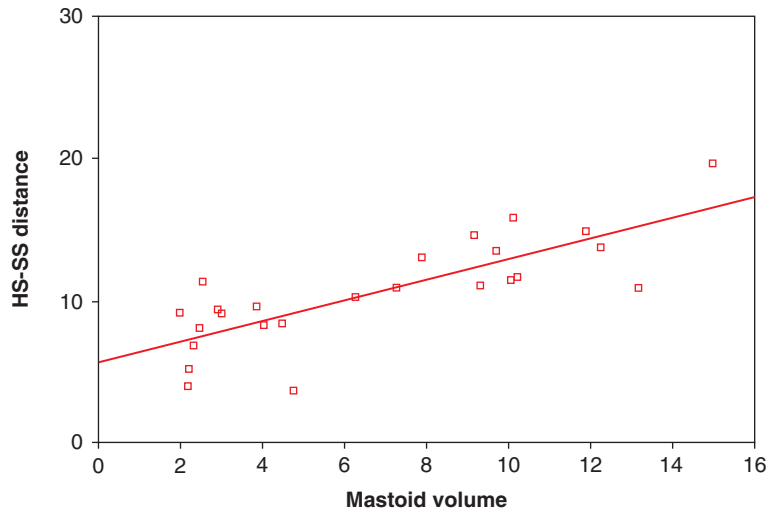


Figure 2. Relationship between mastoid volume and Henle spine-sigmoid sinus (HS-SS) distance.

*Other findings*

There was ossicular discontinuity in two cases and tegmen tympani was not intact in one patient in the diseased group. The ossicles and tegmen tympani were intact in the healthy group. In all of the cases the scutum, lateral semicircular canal, and facial canal were intact.

**Discussion**

Repeated recurrences of inflammatory middle ear disease, when not interrupted by effective aeration, will lead to irreversible damage to the temporomastoid system [9]. Determination of the possible changes in the middle ear and mastoid is significant for surgeons, such as mastoid volume, position of sigmoid sinus, erosion of lateral semicircular canal, facial canal, tegmen tympani, scutum, and ossicles, and disease in the sinus tympani and facial recess [10].

The size of the mastoid is of clinical interest, since ears with the various forms of ‘otitis media syndrome’, i.e. chronic secretory otitis media, atelectatic ears, chronic otitis media, and cholesteatomas, mostly

develop in ears with little or no pneumatization [11]. It is said that the more severe the ‘otitis’, the smaller the mastoid pneumatization. On the basis of this association, a controversy as to whether the underdeveloped mastoid is a genetic trait or follows an acute OM in childhood has continued for many years [7]. Thus, the subject of temporal bone changes/ small mastoid cells and infections in childhood is not new, but remains unsolved as yet, like the question as to which comes first, the chicken or the egg.

Inflammatory changes in the middle ear mucosa or genetic predisposition have been shown to affect mastoid pneumatization [12]. So, evaluation of the mastoid air cell system development is necessary in the diagnosis and treatment of middle ear diseases. Numerous different methods have been described for the measurement of mastoid air cell pneumatization [13]. Digital image processing programs can be used to measure mastoid pneumatization on CT examination. Here, we first measured mastoid volume according to Cavalieri’s principle [7] in both the diseased group and the healthy group. Sirikci et al. [14] also measured mastoid volume according to Cavalieri’s principle and they concluded that mastoid volume in the healthy group was greater than in the diseased mastoids, as we also observed in our study.

The sigmoid sinus develops in fetal life, but pneumatization of the mastoid continues after birth until puberty. The distance of the sigmoid sinus from the external ear canal is one of the factors that could determine to what extent the mastoid bone and its air cells develop after birth. However, middle ear infections in childhood that block mastoid air cells through inflammation may further contribute to the arrest of mastoid pneumatization [3]. Previous studies have shown a relationship between mastoid pneumatization and sigmoid sinus position [15]. Turgut and Tos [15] reported that there was no significant correlation between the degree of pneumatization and HS-SS distance. However, they studied unselected adult temporal bones, and not patients with COM. But Graham [16] found that the development of sigmoid sinus was influenced by mastoid pneumatization. In our study, when we included both healthy and diseased ears, there was a significant correlation between mastoid volume – which reflected mastoid pneumatization – and HS-SS distance. But when we measured HS-SS distances in the diseased and healthy groups, there were no significant differences.

On the basis of our and Graham's [16] study findings, we would support a new hypothesis that, when the decrease in mastoid volume is due to genetic predisposition, this decrease may lead to the changes in the localizations of landmarks on the mastoid bone such as the decrease in HS-SS distance. But, when the decrease in mastoid volume is due to inflammatory conditions, since mastoid volume was affected locally, the localizations of the landmarks on the mastoid bone may not change. So, according to our results, we could claim that chronic inflammation of the middle ear in children suppresses the development of pneumatization in the temporal bone, without any change in the position between the sigmoid sinus and external auditory canal.

In addition, chronic inflammation in the middle ear may lead to ossicular damage [17]. Browning [18] mentioned that bone destruction may occur as long as inflammation was in contact with the bone. Browning [18] also mentioned that the ossicles are frequently eroded because of their location. Thomsen et al. [19] observed that the long process of the incus and the stapes suprastructure are the most frequently effected ossicles. They also observed that bone erosion is more prevalent in cholesteatoma ears than in noncholesteatoma ears. In our study, since we excluded ears with cholesteatoma, we only detected one patient with bony erosion, of tegmen tympani. In all of the cases the scutum, lateral semicircular canal, and facial canal were intact. Chole and Choo [20] reported that granulation tissue can lead to bone erosion. Perforation edges adhering to the promontory may confine the

granulation tissue and inflammation products in a small, dead space, therefore inducing further ossicular discontinuity. In our study, there was ossicular discontinuity in two cases in the diseased group.

## Conclusion

Our results demonstrated that chronic inflammation of the middle ear in children suppresses the development of pneumatization in the temporal bone, without any significant change in the position between the sigmoid sinus and the external auditory canal. Also, despite the fact that recurrences of inflammatory middle ear disease lead to bone destruction, such as ossicle, bony labyrinth, facial canal and tegmen tympani, we did not see any important relationship between the childhood otitis media and bone destruction.

**Declaration of interest:** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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