PARANASAL SINUS VOLUMES AND CHRONIC OTITIS MEDIA: ANY RELATION?

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Abstract

Abstract: Objective: Investigate the relationship between the mastoid pneumatization and the paranasal sinus volumes in patients with unilateral chronic otitis media (COM) and make a contribution to understanding of COM etiopathogenesis. Methods:101 patients were divided into two groups: group 1, COM with isolated tympanic membrane perforation, consists of 51 patients and group 2, COM with cholesteatoma/retraction pocket, consists of 50 patients. Mastoid and sinus volumes were evaluated on preoperative temporal bone High-Resolution Computed Tomography (HRCT). Results: Sphenoid sinus volumes were significantly lower at the diseased side ($p< 0.05$), while there is no statistically difference in the maxillary and frontal sinus volumes. Also, the sphenoid sinus volumes significantly lower at the side of the ear with sclerotic mastoid than others ($p<0.001$). Conclusions: Long-standing childhood COM might restrict to pneumatization of the same sided sphenoid sinus considering the delayed development of the sphenoid sinus compared with other sinuses.

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Abstract:

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Design: 101 patients were divided into two groups: group 1, COM with isolated tympanic membrane perforation, consists of 51 patients and group 2, COM with cholesteatoma/retraction pocket, consists of 50 patients. Mastoid and sinus volumes were evaluated on preoperative temporal bone High-Resolution Computed Tomography (HRCT).

Results: Sphenoid sinus volumes were significantly lower at the diseased side ($p< 0.05$), while there is no statistically difference in the maxillary and frontal sinus volumes. Also, the sphenoid sinus volumes significantly lower at the side of the ear with sclerotic mastoid than others ($p<0.001$).

Conclusions: Long-standing childhood COM might restrict to pneumatization of the same sided sphenoid sinus considering the delayed development of the sphenoid sinus compared with other sinuses.

Key Points:

- The development of the mastoid cells and paranasal sinuses are affected by environmental factors, genetic diseases, and previous infections.
- Due to the similar developmental pattern, close anatomic localization, and mucosal continuity of the mastoid cells and paranasal sinuses, an interplay of these structures at the development stage can be expected.

- There are studies in the literature investigating the relationship between mastoid cell pneumatizion and paranasal sinus volumes, but none of them investigate the relation between mastoid pneumatizion and paranasal sinus volumes in patients with unilateral COM.
- Our study's attractive finding is that although there is no statistically significant difference in the maxillary and frontal sinus volumes, sphenoid sinus volumes were significantly lower at the affected side of patients with unilateral COM.
- This can be explained by the close neighborhood of the mastoid cells and the sphenoid sinus and mucosal lining contiguity. Also, delayed development of the sphenoid sinus in comparison with other sinuses may increase exposure to various pathological conditions.

Keywords: Chronic otitis media; Cholesteatoma; Paranasal sinus radiology; Pneumatization

Background

Chronic otitis media (COM) is a disease characterized by the recurrent and persistent inflammation of the mastoid cavity, middle ear, and tympanic membrane [1]. While the incidence of the disease is below 1% in the USA, the rates rise to 4% in developing countries, leading to significant morbidity and mortality[2]. Even though the etiology and pathophysiology of the disease are not completely revealed; low socioeconomic status, inadequate nutrition, passive smoking, insufficient mastoid pneumatization , and sinonasal diseases have been found pertinent to the progression of the disease [1,3].

The middle ear, mastoid cells, and paranasal sinuses are the largest air-filled cells with close anatomic localization in the skull and have similar embryologic characteristics [4]. Both the paranasal sinuses and the mastoid cells are formed by pneumatization of the related bones, and their surfaces are lined with the upper respiratory tract epithelium, which is originated from endoderm. The development of mastoid cells, maxillary and frontal sinuses start in the embryological period and continue until puberty [4,5], while the pneumatization of the sphenoid sinus starts approximately in the 1st year of life and continue until the end of the 3rd decade [5,6]. Due to the similar developmental pattern, close anatomic localization, and mucosal continuity of the mastoid cells and paranasal sinuses, an interplay of these structures at the development stage can be expected.

The association between mastoid pneumatization and COM has been long investigated, and the relationship between chronic inflammatory middle ear disease and poor mastoid pneumatization has been shown in several studies [7,8]. Some studies show that sinonasal disease increases the risk of middle ear disease [3,9,10]. However, to the best of our knowledge, only one radiologic study investigated the interrelation between the measurements of the paranasal sinuses and mastoid pneumatization in patients with COM and suggested a hypothesis that chronic rhinosinusitis during childhood may play a role in the development of cholesteatoma [11].

Material and Methods

2.1. Subjects

The STROBE statement for cross-sectional studies was adhered to. 179 patients with retrospective chart analysis unilateral COM with isolated tympanic membrane perforation (group 1) and cholesteatoma/retraction pocket (group 2) between 2015-2019 in our tertiary clinical was performed. The demographic data, preoperative temporal bone High-Resolution Computed Tomography (HRCT), and operation notes of each patient were evaluated.

The inclusion criteria were (1) being over 18 years of age, (2) having unilateral middle ear disease, and (3) visualization of all paranasal sinuses in axial, coronal, and sagittal planes of preoperative HRCT. The patients were excluded from the study in the presence of any pathological findings in the contralateral ear and the sinonasal region in physical or radiological examination. That exclusion had two purposes: To create an internal control group with a healthy side and to eliminate possible effects of ongoing sinonasal disease on the sinus volumes.

The paranasal sinus volumes and the mastoid pneumatization of the diseased and healthy sides of the patients were further evaluated for statistical analysis.

2.2. Radiologic Evaluations and Calculations

HRCT examinations had been performed with multidetector CT system (Activision 16-row CT scanner; Toshiba Medical Systems, Otawara, Japan) at 120–400 mA and 80–160 kV, and CT slides 0.5 mm in thickness; a matrix size 512 × 512 were obtained, and approximately 120–300 images per CT were evaluated.

Mastoid pneumatization was evaluated by using the criteria Diamant[12], Doland [13], and Valvassori et al.[14] described: fully pneumatized, diploic (partial pneumatized), or sclerotic (non-pneumatized) **(Figure 1)** . Petrous apex and perilabyrinthine pneumatization were evaluated as present or absent.

Paranasal sinus volumes were calculated by using the formula defined by Barghouth et al. in 2002 [15]. The greatest depth, width and length of the sinuses were calculated respectively by using axial and coronal sections, and the Sinus volume index (SVI) was found by the equation: SVI = 1/2. A x B x C **(Figure 2)** . Due to the complex anatomy and vague borders of the sinuses, ethmoid sinus volumes were not calculated.

Two otolaryngologists blindly evaluated all CT scans. The mean values of the measurements obtained by the two physicians were used for analyses of the sinus volumes. For the evaluation of mastoid, petrous apex, and perilabyrinthine cell pneumatization , the results of the two otolaryngologists were compared, if any conflict was present, the CT scan was further evaluated by an associated professor (B.P).

2.3. Statistical Analysis

Chi-square test of homogeneity (Rx2) was performed to determine if the proportions of pneumatized and non-pneumatized petrous apex and perilabyrinthine cells and the proportions of fully pneumatized, diploic and sclerotic mastoid cells were statistically significantly different between healthy and diseased-side for group 1 and group 2.

The assumption of normality for volumes of paranasal sinuses was not satisfied with the healthy-side and diseased-side of group 1 and group 2, as assessed by Shapiro-Wilk's test. ($p<0.05$) Therefore, A Mann-Whitney U test (as a non-parametric test) was run to determine if there were differences in volumes of paranasal sinuses between healthy-side and diseased-side for group 1 and group 2. Kruskal Wallis H test was performed to compare the paranasal sinus volumes between the related ears with grouped based on their mastoid pneumatization. Dunn's procedure was performed, and adjusted p-value (Bonferroni correction) was calculated for posthoc analysis.

Statistical significance was accepted at $p< 0.05$.

Results

A total of 179 patients were screened. Thirty-one patients were excluded due to age, 35 due to bilateral ear disease, 7 due to insufficient HRCT images, and 5 due to sinonasal disease. Remaining 101 patients were included in statistical analysis.

Group 1, COM with isolated tympanic membrane perforation, consists of 51 patients, of which 26 (51%) were female and 25 (49%) male. The average age was 37.1(+- 14.75).

Group 2, COM with cholesteatoma/retraction pocket, consists of 50 patients, of which 27 (54%) were female and 23 (46%) male. The average age was 38.8 (+-13.73).

3.1. Comparison of the mastoid pneumatization in the healthy and the diseased ears

There were no significant differences in pneumatizion of the petrous apex, perilabyrinthine, and mastoid cells between the healthy and diseased-side for group 1. However, statistically, significant differences were seen in the aeration of the three regions between the healthy and diseased-side for group 2. Proportions and p-values are given in **Tables 1 and 2** . Post-hoc analysis was done to compare the pneumatization degree of mastoid cells for group 2, using multiple z-tests of two proportions with a Bonferroni correction.

Statistical significance was accepted at p< 0.016667. Although there were statistically significant differences in proportions of fully pneumatized (p=0.012) and sclerotic (p<0.001) mastoid cells, no difference was seen in proportions of diploic (p=0.211) mastoid cells between healthy and diseased-side.

3.2. Comparison of the paranasal sinus volumes of the healthy and the diseased ear sides

Distributions of the frontal, maxillary and sphenoid sinus volumes for healthy and diseased-side of group 1 and group 2 were similar. Median volumes of paranasal sinuses and p-values are given in **Table 3** . The only statistically significant difference was seen in the volumes of sphenoid sinus between the healthy and diseased-side for both groups.

3.3. Comparison of the paranasal sinus volumes of related-ears grouped based on their mastoid aeration

There were no statistically significant differences in frontal (p=0.287) and maxillary (p=0.073) sinus volumes between the ears with fully-pneumatized, diploic, and sclerotic mastoid cells observed. However, the sphenoid sinus volumes significantly lower at the side of the ear with sclerotic mastoid than others. (p<0.001) We also observed smaller sphenoid sinus volumes at the diploic mastoid side in group 1 (p=0.003) and at the sclerotic mastoid side in group 2 (p<0.001), separately. The median (IQR) values of the parameters and posthoc analysis were given in **Table 4** .

Discussion

To our best of knowledge, this is the first study radiologically assessing the association of the paranasal sinus volumes and mastoid, petrous apex, and perilabyrinthine pneumatization in patients with COM. A study [11] investigating the association of the length, height, and weight of the paranasal sinuses and mastoid pneumatization in COM patients was published while we were in the data-gathering stage of our study. We included only the patients with unilateral middle ear disease, creating an internal control group while they included both unilateral and bilateral disease and an external control group, including healthy participants. Although the methodology of the studies was different, both have reached similar outcomes. In our study, the volumes of sphenoid sinus were significantly smaller in COM (with isolated tympanic membrane and with cholesteatoma/retraction pocket) side than the contralateral healthy side. There was no difference in volumes of other sinuses. Similarly, Arai et al.[11] found that the anterior-posterior length of the sphenoid sinus was smaller in cholesteatoma patients than control subjects. However, there was no difference in width. In contrast, while the width was smaller in patients with COM without cholesteatoma, no difference was seen in the length. Both studies also agreed to that poor mastoid cell pneumatization is associated with lower sphenoid sinus in patients with cholesteatoma. The pneumatization of the sphenoid sinus might be restricted more than other sinuses by childhood chronic rhinosinusitis, as it needs a longer time to complete its development[16]. Arai et al.[11] hypothesized that childhood chronic rhinosinusitis might play an essential role in the etiopathogenesis of the acquired cholesteatoma based on their results. However, it is logical to expect that bilateral COM and bilateral smaller sphenoid sinus length should have been observed, considering the chronic rhinosinusitis is a disease that generally affects all mucosa of the nasal cavity and paranasal sinuses. However, they observed smaller sphenoid sinus only in patients with unilateral cholesteatoma. The outcome of our study seems to support their hypothesis. However, we included patients with unilateral COM. The reduced volume of sphenoid sinus on both sides would be expected if the cholesteatoma was caused by childhood chronic rhinosinusitis in these patients. Therefore, we propose another hypothesis suggesting that long-standing childhood COM and middle ear ventilation problems might restrict to pneumatization of the same sided sphenoid sinus. This restriction might be explained the changing of the bone resistance against pneumatization due to the inflammatory mediators triggered by adjacent chronic inflammatory tissue. Unfortunately, our study did not have enough data to prove both hypotheses.

Mastoid pneumatization starts primarily in the mastoid antrum at 21^{st}-22^{nd} weeks during the embryogenesis, and by the 34^{th} week, the pneumatization of the antrum is nearly completed [17]. The development of the mastoid bone continues until puberty, thus influenced by genetic and environmental factors [18]. For more than half a century, clinicians have been exploring the association between mastoid aeration and middle ear disease.

In a cadaveric study in 1940 by Diamant[19], the mastoid cell system of the sides with COM was determined to be smaller and underdeveloped. In 1959, Tumarkin suggested that mastoid bone hypocellularity is one of the most important risk factors for COM [20]. After that, many studies have been done on this subject. The study of Tos et al.[7] with 79 patients between the ages of 2-7 in 1985; and Sade and Fuch's study[8] in 325 patients with cholesteatoma in 1994 are the striking ones which state the insufficient mastoid aeration increases the risk of otitis. Aria et al. [11] and our study showed that mastoid aeration is reduced in cholesteatoma patients, comparable to literature [7,8]. However, the mastoid pneumatization was normal in COM without cholesteatoma in both studies. This can be explained by the possible presence of a single attack of acute otitis media-related perforation and traumatic tympanic membrane perforation in this group. Besides, effects on COM with or without cholesteatoma to adjacent structures may vary at the molecular level. Also, mastoid bones with low pneumatization are thought to have limited ability to buffer pressure changes, resulting in atelectasis, retraction pocket, or a cholesteatoma [21].

The development of the mastoid cells and paranasal sinuses are affected by environmental factors, genetic diseases, and previous infections. The interplay of mastoid cell and paranasal sinus development can be explained as [3,5,10]:

- *The anatomical contiguity* : the drainage pathway of the mastoid cells and paranasal sinuses have similar routes. Furthermore, petrous apex cells and sphenoid sinuses are in close contact.
- *Similar physiological characteristics* : both structures are lined with respiratory epithelium.
- *Similar embryological development:* as stated above, the embryologic development of the mastoid cells and paranasal sinuses are similar.

The connection between sinonasal diseases and ear diseases has been interest. The first large scale study has been published by Van Cauwenberge et al. [7] in 1983, which stated that in children with septum deviation and allergic rhinitis, the risk for otitis media with effusion is increased. In the following years, studies indicating that septum deviation increases the risks of COM have been published. Gopalakrishnan and Kumar [22] observed that in patients with COM between 18-49 of age, septum deviation is present in 73%. Moreover, in the study of Sajitha et al.[10], at least one sinonasal pathology was found in 82 of 100 patients with COM; thus, routine endoscopic nasal cavity examination was suggested. One possible explanation for sinonasal diseases and increased COM incidence is secondary eustachian tube dysfunction and consecutive mastoid aeration disruption.

The relation between the mastoid and paranasal sinus pneumatization has also been investigated. In 2005 Karaskas and Kavaklı[23] published their study showing the strong correlation between the right, left, and total paranasal sinus volume and right, left, and total mastoid volume, respectively. However, no correlation between any specific sinus and the mastoid bone was determined. Lee et al.[4] described the correlation between age and the development of the mastoid cells and paranasal sinuses in their study in 62 pediatric patients with an age average of 13.4. Similarly, they also failed to show any correlation between any specific sinus and the mastoid bone. On the other hand, Kim et al.[24] and Hindi et al. [25] found a correlation between the sphenoid sinus and mastoid pneumatization.

In our study, unlike the above studies, the relation between mastoid aeration and paranasal sinus volumes was investigated on patients with suffering unilateral COM. This can be explained by the close neighborhood of the mastoid cells and the sphenoid sinus and mucosal lining contiguity. Also, delayed development of the sphenoid sinus in comparison with other sinuses may increase exposure to various pathological conditions. Both Aria et al. [11] and us suggested it could be the reason for the developmental insufficiency of the sphenoid sinus in patients with COM. However, in our study, reliable history of childhood sinonasal and middle ear diseases were not present. Besides, no molecular and histopathological data of affected structures were available to prove these hypotheses. Therefore, further prospective cohort studies with a large population, and animal studies, modeling COM and chronic rhinosinusitis, should investigate these diseases' effect on the related anatomical structures.

Conclusion

There are studies in the literature investigating the relationship between mastoid cell pneumatizion and paranasal sinus volumes, but none of them investigate the relation between mastoid pneumatizion and paranasal sinus volumes in patients with unilateral COM. Our study's attractive finding is that although there is no statistically significant difference in the maxillary and frontal sinus volumes, sphenoid sinus volumes were significantly lower at the affected side of patients with unilateral COM.

Further studies with larger groups are needed to obtain more information about the relation between sphenoid sinus pneumatization and COM.

Conflict of Interest:

The authors declare no conflicts of interest.

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Table 2: Comparison of the mastoid pneumatization in the healthy and the diseased ears in the group 2.

Group 1		
Healthy-side (n=51)		
Diseased-side (n=51)		
p-value		

Group 1: COM with isolated tympanic membrane perforation **Table 2: Comparison of the mastoid pneumatization**

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Group 2	Mastoid cells	Mastoid ce
	Fully-pneumatized	Diploic
Healthy-side (n=50)	48%	42%
Diseased-side (n=50)	24%	30%
p-value	<0.001	<0.001

Group 2: COM with cholesteatoma/retraction pocket	Group 2: COM with cholesteatoma/retraction pocket	Group 2: C

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Table 3: Comparison of the paranasal sinus volumes of the healthy and the diseased ear sides in both groups.

	Group 1 (n=51)	Group 1 (n=51)	Group 1 (n=51)	Group 2 (n=50)	Group
Volume of Paranasal Sinuses(mm3)	Healthy-side	Diseased-side	p-value	Healthy-side	Diseas
Frontal Sinus	1357.67(1288.44)	1245.73(888.13)	0.383	1413.64(1479.15)	1534.8
Maxillary Sinus	12470.41(7870.15)	10927.24(5451.6)	0.202	10987.06(5433.58)	9751.1
Sphenoid Sinus	3909.40(1784.4)	2409.81(1679.06)	<0.001	3509.41(4136.65)	2174.3

Group 1: COM with isolated tympanic membrane perforation, Group 2: COM with cholesteatoma/retraction pocket, mm3: cubic millimeter. (COM:Chronic otitis media)

Data are presented as Median (Interquartile Range).

Six and four cases were excluded while comparing the volumes of frontal sinuses because of frontal sinus agenesis for group 1 and group 2, respectively.

Table 4: Comparison of the paranasal sinus volumes of related-ears grouped based on their mastoid aeration.

Paranasal sinus volumes
Total (n=202)
F-Volume
M-Volume
S-Volume
Group 1 (n=102)

S-Volume
Group 2 (n=100)
S-Volume
Group 1: COM with isolated tympanic membrane perforation, Group 2: COM with cholesteatoma/retraction pocket. (CO

Figure Legends:

Figure 1: Axial view of the temporal HRCT showing; **A**fully pneumatized, **B** diploic (partial pneumatized) and**C** sclerotic (non-pneumatized) mastoids on the right side of different patients.(HRCT: High-Resolution Computed Tomography)

Figure 2: Paranasal sinus computed tomography sections;**A** length of the evaluated sinus in an axial section, **B**width of the evaluated in coronal section and **C** height of the evaluated sinus in coronal section.

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