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Temporal bone erosion in patients with chronic suppurative otitis media

E. Yorgancilar[1], *Z. Akkus*[2], *R. Gun*[1], *M. Yildirim*[1], *S. Bakir*[1], *V. Kinis*[1], *F. Meric*[1] *and I. Topcu*[1]

[1]Department of Otorhinolaryngology and Head and Neck Surgery, Dicle University School of Medicine, Diyarbakir, Turkey; [2]Department of Biostatistics, Dicle University School of Medicine, Diyarbakir, Turkey

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Abstract. *Temporal bone erosion in patients with chronic suppurative otitis media. Objectives*: To analyse temporal bone erosion sites (including scutum, labyrinth, facial canal, mastoid tegmen, posterior fossa dural plate and sigmoid sinus plate) in patients with chronic suppurative otitis media (CSOM).
Methodology: Retrospective case review in a tertiary referral centre. Medical records were reviewed from 905 patients (121 complicated; 784 non-complicated) who received a mastoidectomy as a minimum intervention for the treatment of CSOM.
Results: All types of temporal bone erosion were found to be more frequent in patients with complicated CSOM. Erosion in the scutum, mastoid tegmen, posterior fossa dural plate and labyrinth was observed significantly more frequently in complicated-CSOM patients with a cholesteatoma. Granulation/polyp tissue invaded the sigmoid sinus and facial canal at a rate similar to cholesteatoma.
Conclusions: Our study demonstrates that bone erosion is more frequent in complicated-CSOM patients. Temporal bone erosion can be seen in both cholesteatomatous and non-cholesteatomatous CSOM patients. Granulation/polyp tissue was as important as cholesteatoma in the erosion of the facial canal and sigmoid sinus plate.

Introduction

Chronic suppurative otitis media (CSOM) is defined as the chronic inflammation of the middle ear and mastoid mucosa in which the tympanic membrane is not intact and otorrhea is present.[1] Chronic suppuration can occur with or without cholesteatoma. A cholesteatoma results from the abnormal migration of epidermal tissue into the middle ear and mastoid. One of the characteristic features of a cholesteatoma is bone destruction. Bone destruction by a cholesteatoma can be explained by the pressure of the matrix of the cholesteatoma, enzymes, and mediators.[2-4] Non-cholesteatomatous CSOM is a persistent and insidious infectious process of the middle ear and mastoid air cells. This condition may be associated with chronic changes of the middle ear and mastoid cell mucosa, such as the formation of granulation tissue or polyps. Granulation tissue adjoining temporal bone may produce several enzymes and mediators that can accelerate bone resorption.[3] This may also lead to destructive changes of the adjacent bones.

CSOM may lead to complications. These complications can be classified as extracranial (EC; mastoiditis, petrositis, labyrinthitis, and facial nerve paralysis) and intracranial (IC; extradural abscess, brain abscess, subdural abscess, sigmoid sinus thrombophlebitis, otic hydrocephalus, and meningitis).[5,6] These complications are related to bony destruction, which may lead to serious and potentially life-threatening conditions.

There are many studies in the literature describing ossicle erosion patterns in patients with CSOM. However, to the best of our knowledge, there are only a few reports examining temporal bone erosion in patients with CSOM. This retrospective study evaluated temporal bone erosion in CSOM patients in our centre over a 10-year period.

Materials and methods

The study was approved by the local research and ethics committee of the Medical School, Dicle University (246/20.10.2011). The charts of patients diagnosed with CSOM and scheduled for middle ear surgery between January 2000 and October 2011 in the Department of Otolaryngology of the Dicle University Faculty of Medicine, Diyarbakir, Turkey were reviewed. The patients who underwent mastoidectomy as a minimum intervention for the treatment of CSOM were included in this study.

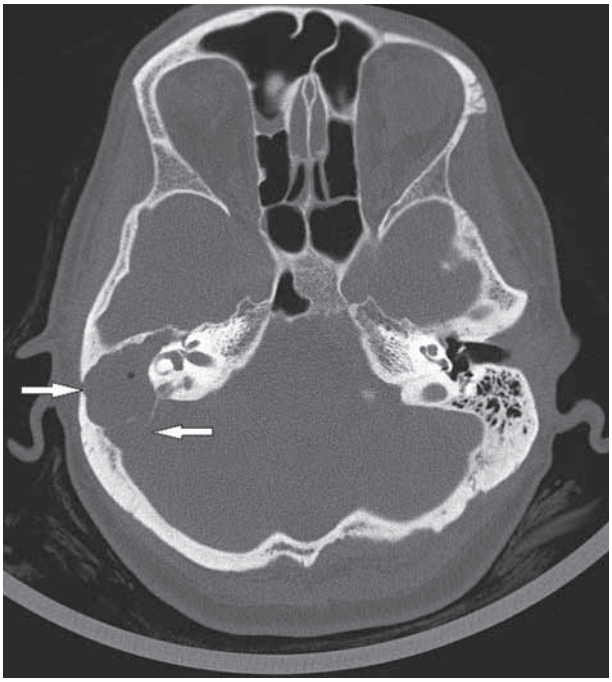

Figure 1
Computed tomography image showing erosion of the mastoid cortex, sigmoid sinus plate and posterior fossa dural plate (*white arrows*).

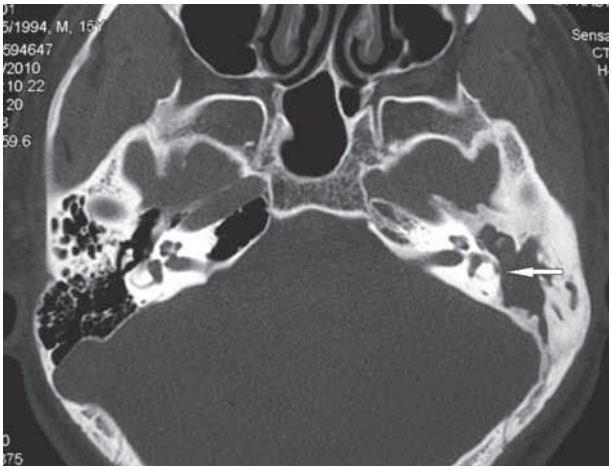

Figure 2
Computed tomography image showing bone erosion in the lateral semicircular canal (*white arrow*).

All patients underwent radiological studies that included at least contrast-enhanced computed tomography (Figures 1, 2). The operation theatre logbooks were also examined to identify intra-operative findings.

Patients with revision surgeries, tympanosclerosis, complications of acute suppurative otitis media, and tympanoplasties without mastoidectomy were excluded from this study.

Intra-operative middle ear findings, including pathological tissue and gross observations of temporal bone erosion, were noted. Temporal bone erosion sites included the scutum, facial canal, labyrinth, mastoid tegmen, posterior fossa bone plate and the sigmoid sinus plate. Iatrogenic bone defects were excluded.

A total of 905 patients were selected for this study and divided into 'complicated' and 'non-complicated' CSOM patients. Complicated-CSOM patients were defined as the patients with CSOM-related EC and/or IC complications. The patient who underwent a mastoidectomy as a minimum intervention for the treatment of CSOM and who had no CSOM-related complications was defined as a non-complicated-CSOM patient.

All data were analysed using SPSS software (ver. 15.0; SPSS, Chicago, IL, USA). Pearson's chi-square test, Yates' correction for continuity, and Fisher's exact test were used to evaluate the level of significance, and P values < 0.05 were considered statistically significant.

Results

There were 121 complicated-CSOM (13.4%), and 784 non-complicated-CSOM (86.6%) patients. The patients were aged between 4 and 72 years, with an average age of 29.7 ± 13.1 years. The male to female ratio was 1.16.

Of the 121 patients with CSOM complications, 94 (78%) had a cholesteatoma. Granulation and/or polyp tissues were found in only 27 (22%) of these patients. An EC, IC, or combined EC and IC complication occurred in 58 (48%), 50 (41.3%), or 13 (10.7%) patients respectively.

A total of 244 bone erosions were observed in 121 patients with complications (Tables 1, 2, Figure 3). Bone erosion was found significantly more frequently in patients with a cholesteatoma than in those with granulation/polyp tissues ($p < 0.05$). Erosion in the scutum, labyrinth, mastoid tegmen and posterior fossa was observed significantly more frequently in patients with a cholesteatoma ($p < 0.05$). There were no significant differences between cholesteatomatous and non-cholesteatomatous patients with respect to erosion of the facial canal ($p > 0.05$).

Table 1

Distribution of temporal bone erosion in complicated chronic suppurative otitis media (*Several patients had more than one bony erosion*)

	Scutum	Sigmoid Sinus Plate	Mastoid Tegmen	Posterior Fossa Plate	Labyrinth	Facial Canal	Total
Cholesteatoma(+)	67†	30	28†	16†	17†	18	176
Cholesteatoma(-)	25	21	4	1	0	17	68

† Statistically significant (P<0.05).

Table 2

Distribution of temporal bone erosion in complicated chronic suppurative otitis media (*Several patients had more than one bony erosion*.) EC: extracranial complication, IC: intracranial complication

	Scutum	Sigmoid Sinus Plate	Mastoid Tegmen	Posterior Fossa Plate	Labyrinth	Facial Canal	Total
EC	42	4	4	7	13†	17	87
IC	48†	36†	16†	9	3	12	124
EC+IC	6†	11	7	2	1	6	33
Total	96	51	27	18	17	35	244

† Statistically significant (P<0.05).

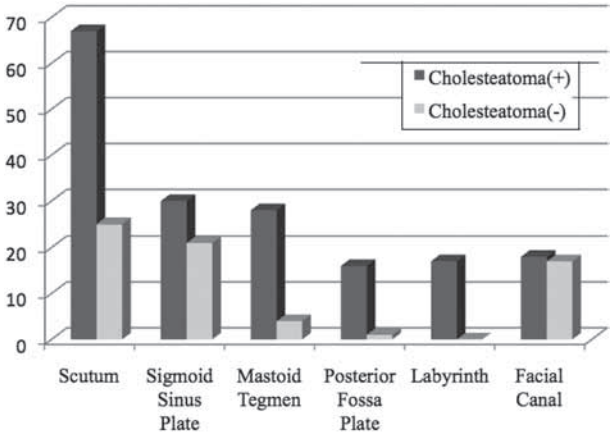

Figure 3
Distribution of temporal bone erosion in complicated chronic suppurative otitis media.

All of the sigmoid sinus thrombophlebitis and/or perisinusal abscess patients had sigmoid sinus plate erosion and no statistical difference was found between cholesteatomatous and non-cholesteatomatous patients with respect to sigmoid sinus plate erosion (p>0.05).

Of the 784 non-complicated-CSOM patients, 491 (63%) had a cholesteatoma and 293 (37%) had a granulation/polyp tissue. Of these 784 patients, 80 (10%) had temporal bone erosion. Of these 80 patients, 67 (84%) had a cholesteatoma. Thirteen (16%) patients had granulation/polyp tissue. Erosion in the facial canal was observed in 18 patients (2.2%) and in the mastoid tegmen in 16 patients (2%). There was no statistical difference between patients with cholesteatoma and granulation/polyp tissue with respect to all types of erosion. The distribution of temporal bone erosions in non-complicated-CSOM patients is shown in Table 3 and Figure 4.

Comparison of complicated-CSOM and non-complicated-CSOM patients determined that erosion of the scutum, facial canal, labyrinth, mastoid tegmen, posterior fossa bone plate and sigmoid sinus plate was found significantly more frequently in patients with complicated-CSOM than non-complicated-CSOM (p<0.05) (Table 4, Figure 5).

Discussion

Although much has been written about the general clinical features and ossicular erosion patterns of CSOM, little is apparently known about temporal bone erosion. The present study differs from previously published reports of CSOM in comparing the extent of bone erosion in different pathologies and complications.

Table 3

Distribution of temporal bone erosion in non-complicated chronic suppurative otitis media (*All patients had one bony erosion*)

	Scutum	Sigmoid Sinus Plate	Mastoid Tegmen	Posterior Fossa Plate	Labyrinth	Facial Canal	total
Cholesteatoma(+)	27	4	17	4	4	16	67
Cholesteatoma(-)	6	1	1	0	0	2	13
Total	33	5	16	4	4	18	80

† Statistically significant (P<0.05).

Table 4

Comparison of temporal bone erosion in complicated and non-complicated chronic suppurative otitis media (*Several patients had more than one bony erosion*)

	Scutum	Sigmoid Sinus Plate	Mastoid Tegmen	Posterior Fossa Plate	Labyrinth	Facial Canal	Total
Complicated	96†	51†	27†	18†	17†	35†	244
Non-complicated	33	5	16	4	1	18	80

† Statistically significant (P<0.05).

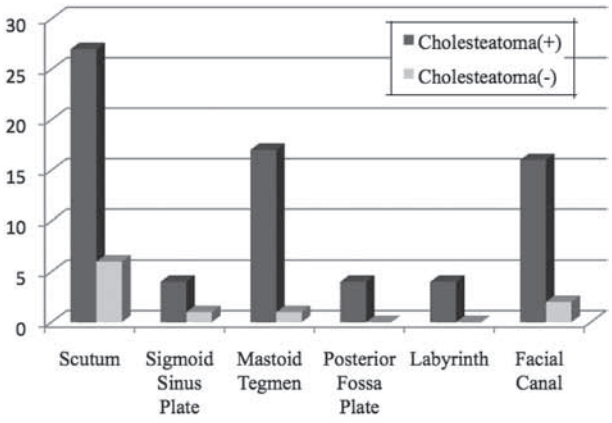

Figure 4
Distribution of temporal bone erosion in non-complicated chronic suppurative otitis media.

The mechanisms of bone erosion in both cholesteatomatous and non- cholesteatomatous CSOM are well documented. In cholesteatoma, excess keratin debris accumulates in the middle ear and mastoid cavity, resulting in the destruction of surrounding bony structures.[7] The accumulation of the keratin debris diminishes the blood supply and causes bone resorption.[8] The other mechanism is osteoclastic bone resorption. In normal bone, homeostasis is achieved by maintaining a balance between bone-resorbing osteoclasts and bone-forming osteoblasts. In patients with CSOM, this synergy is disrupted, resulting in the osteoclastic resorption of bone.[3,9,10] The osteoclastic resorption of adjacent bones in CSOM patients is initiated by a complex interaction of inflammatory factors.[3,4,11-14] Chronically inflamed tissue (activated granulation tissue) adjacent to bone is capable of causing eventual bone resorption.[3] The infiltration of inflammatory cells and secretion of cytokines in the cholesteatoma and/or granulation tissue results in bone resorption. In our study, bone erosion was observed more frequently in cholesteatomatous CSOM. Cholesteatomas may involve cranial bones, including the hardest bone of the body, the optic capsule, and this demonstrates their destructive potential for bone. Our findings are consistent with the bone erosion potential of cholesteatoma. However, we also observed bone erosion in non-cholesteatomatous patients. We advise surgeons to be very alert to the possibility of bone erosion, even in non-cholesteatomatous CSOM surgery.

There are four different routes for the spread of infection.[15] These include: preformed anatomical pathways, bony erosion, thrombophlebitis, and the haematological route. In our study, there was a statistically significant preponderance of bone erosion in complicated-CSOM patients. This finding reflects the importance of direct bone erosion in the aetiology of these complications.

Scutum erosion plays an important role in the diagnosis of attic cholesteatoma. This type of bone erosion is clear to see in computed tomography images.[16,17] We evaluated this erosion pattern intra-

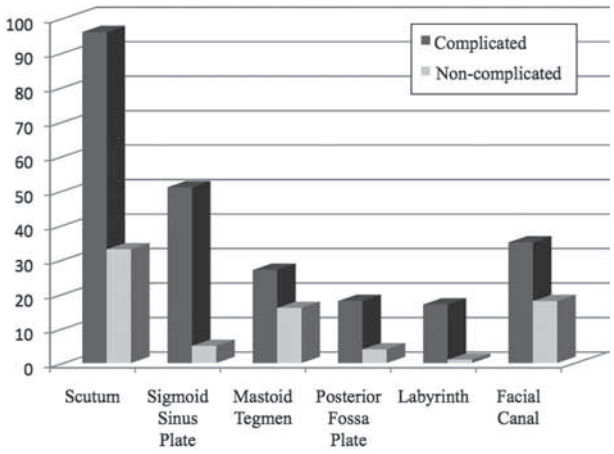

Figure 5
Comparison of temporal bone erosion in complicated and non-complicated chronic suppurative otitis media (*x axis: type of erosion; y axis: no. of patients*).

operatively and we found that scutum erosion was observed more frequently in cholesteatoma and complicated CSOM. We suggest that this finding reflects the importance of attic cholesteatomas in the aetiology of complicated CSOM. However, this finding requires further study.

Labyrinth fistulas are usually caused by the erosion of the lateral aspect of the horizontal semicircular canal. In the literature, the labyrinthine fistula rate is higher in cholesteatoma than in other chronic ear diseases.[18] Our study is consistent with this finding.

The pathological process that causes destruction in the temporal bone can affect the facial canal. Previous studies focusing on facial canal erosions showed that the frequency of intra-operative facial canal dehiscence varied from 0.5% to 74%.[19,20] However, the determination of facial canal dehiscence frequency due to the erosion of CSOM is very difficult since facial canal dehiscences can be seen in normal populations. In a study of 1000 temporal bones, Moreano *et al*.[21] reported that one-third of the temporal bones observed had a microdehiscence of the facial canal. Li and Cao[22] reported an 11.4% incidence of facial canal dehiscence in 1465 stapes operations. Bayazit *et al*.[23] reported dehiscence incidences of 8.9% in surgical studies of tympanoplasty with mastoidectomy. Wang *et al*.[18] presented mastoid findings from cholesteatoma surgeries, and found a 29.7% incidence of facial canal dehiscence. In this study, we found a higher incidence of facial canal erosion and a higher risk of facial nerve injury during complicated chronic otitis media surgery. Our study also demonstrated that granulation tissue frequently eroded the facial canal. Savic and Djeric[24] concluded that the spread of infection and inflammation along the nerve tissue, rather than compression atrophy, is the major factor in most facial nerve paralyses resulting from CSOM. Our findings are consistent with this report.

It is well known that the local spread of mastoid infection to the cranium is associated with mastoid tegmen erosion. In our study, mastoid tegmen erosion was seen more frequently in the patients with IC complications. This finding demonstrates that these types of erosion area are more important for the intracranial local spread of mastoid infections. Sigmoid sinus thrombophlebitis may result either from the direct spread of infection from the erosion of the sigmoid sinus plate or septic thrombophlebitis of the emissary veins of the middle ear.[25] In our study, there were no significant differences between cholesteatomatous and non-cholesteatomatous patients with respect to the erosion of the sigmoid sinus bony plate. Our findings demonstrated that granulation tissue is as important as cholesteatoma in the aetiology of sigmoid sinus thrombophlebitis.

Conclusion

The growth of either a cholesteatoma or granulation/polyp frequently involved the adjacent bony surfaces. The incidences of bone erosion were found with greater frequency in complicated-CSOM patients than in non-complicated CSOM patients. Complicated-CSOM patients suffered from severe bone erosion, and the frequency of observed scutum, labyrinth, mastoid tegmen and posterior fossa dural plate erosion was higher in CSOM patients with a cholesteatoma. Granulation/polyp tissue tended to invade the sigmoid sinus and facial canal. Knowing that these kinds of erosion can be seen makes surgeons more aware during procedures and prevents possible complications.

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Ediz Yorgancilar
Dicle University Faculty of Medicine
Department of Otolaryngology
21280 Diyarbakir, Turkey
Tel.: +904122488001-4543
E-mail: edzyrg@hotmail.com