The Journal of Laryngology & Otology (2016), ***130***, *66–68.*

doi:10.1017/S0022215115003047

MAIN ARTICLE

A study of mastoid pneumatisation and the presence of cholesteatoma in 393 patients

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Abstract

Objectives: This study aimed to evaluate the relationship between cholesteatoma formation and the degree of mastoid pneumatisation, and to assess the relationship between the location of cholesteatoma and the degree of mastoid pneumatisation.

Methods: Data on all patients undergoing mastoid exploration for cholesteatoma between 1993 and 2011 were collected prospectively. Basic demographics, the degree of mastoid pneumatisation and cholesteatoma site were recorded.

Results: A total of 393 patients (222 males and 171 females) underwent surgery for cholesteatoma. Patients' mean age was 37 years (range, 6–79 years). Pneumatisation of the mastoid was sclerotic in 23 per cent ($n = 90$), diploic in 16.7 per cent ($n = 66$) and cellular in 60.3 per cent of cases ($n = 237$) ($p < 0.001$). Atticoantral disease was present in 88.9 per cent of sclerotic, 95.4 per cent of diploic and 91.1 per cent of cellular mastoids. Middle-ear cholesteatoma was present in 54.4 per cent of sclerotic, 56 per cent of diploic and 51.9 per cent of cellular mastoids.

Conclusion: The findings demonstrate the presence of cholesteatoma in well-pneumatised mastoids. It is hypothesised that a well-pneumatised mastoid may lead to cholesteatoma formation.

Key words: Cholesteatoma; Mastoid; Middle Ear

Introduction

There are various theories describing the pathophysiology of cholesteatoma formation.[1–3] The widely accepted view is that poorly pneumatised mastoids lead to negative middle-ear pressure. This causes hypopneumatisation of the air cell system, negative middle-ear pressure and inflammation, and over time may lead to formation of a retraction pocket.[1] The result is development of cholesteatoma.

This study aimed to evaluate the relationship between cholesteatoma formation and the degree of mastoid pneumatisation in our subset of patients. We also assessed the relationship between the location of cholesteatoma and the degree of pneumatisation.

Materials and methods

The study was performed at a district general hospital serving a population of approximately 400 000. Data on all patients undergoing mastoid exploration for cholesteatoma were collected prospectively as part of an ongoing audit of surgical outcome from 1993 to 2011. Basic demographics (age and sex), date of surgery, type of surgery performed, the degree of mastoid pneumatisation, cholesteatoma site and side, hearing outcomes, and need for revision surgery were amongst many details recorded. Data were entered into a Microsoft Excel® spreadsheet. Mastoid pneumatisation was classified as pneumatic, diploic or sclerotic (Table I).

All of the patients were operated on and graded by the same primary surgeon (CW). There is no agreed classification system for mastoid pneumatisation. The terms used in this ongoing database were therefore adopted in consensus with operative and radiological descriptions.

A total of 533 patients were treated. Patients undergoing revision surgery and atticotomy were excluded as the degree of pneumatisation could not be fully assessed. Statistical analysis was performed using StatsDirect statistical software (StatsDirect, Altrincham, UK).

TABLE I
CLASSIFICATION OF MASTOID PNEUMATISATION

Sclerotic	Pneumatisation absent
Diploic	Pneumatisation partial
Pneumatic	Pneumatisation complete

Accepted for publication 9 September 2015 First published online 23 November 2015

Ethical considerations

The study was approved by the Darlington Memorial Hospital Research and Audit Department, which is equivalent to an institutional review board.

Results

A total of 393 patients were included in the study. Of these, 222 of the patients were male and 171 were female. Patients' mean age was 37 years (range, 6–79 years). The side of cholesteatoma was left in 203 and right in 190 patients.

Pneumatisation of the mastoid was sclerotic in 23 per cent ($n = 90$), diploic in 16.7 per cent ($n = 66$), and pneumatic in 60.3 per cent of cases ($n = 237$) ($p < 0.001$). Atticoantral cholesteatoma was present in 88.9 per cent of sclerotic, 95.4 per cent of diploic and 91.1 per cent of pneumatic mastoids. Middle-ear (i.e. sinus tympani, anterior tympanum, posterior tympanum and eustachian tube) cholesteatoma was present in 54.4 per cent of sclerotic, 56 per cent of diploic and 51.9 per cent of pneumatic mastoids. The results demonstrate the presence of cholesteatoma in well-pneumatised mastoids ($p < 0.001$ (chi-square test)).

Discussion

Although studies have found congenital cholesteatoma to be associated with well-pneumatised mastoids, this is not always the case in acquired cholesteatoma.[4]

The formation of cholesteatoma is multifactorial. Genetics, otitis media, eustachian tube function and mastoid pneumatisation are aetiopathogenic factors. The well-described theories highlighting hypopneumatisation of the mastoid air cell system, which leads to cholesteatoma, cannot explain our findings. The relatively high proportion of well-pneumatised mastoids in patients with cholesteatoma initially seemed difficult to explain.

The eustachian tube plays a vital role in normalising middle-ear pressure. Dysfunction leads to a process of negative middle-ear pressure and inflammation.[5] One study investigating recurrent cholesteatoma found that eustachian tube dysfunction is closely related to recurrence.[6]

It is thought that a well-pneumatised mastoid may function as a reservoir of air to buffer the effects of intermittent eustachian tube obstruction on middle-ear pressure and so provide a level of protection from the development of retraction pockets (Figures 1 and 2). This may be the case if eustachian tube function is impaired. However, with a more prolonged period of eustachian tubal obstruction, the buffering effect of a greater volume of air is eventually exhausted.

If we consider two ears, one sclerotic and the other well-pneumatised, both ears are subject to periods of eustachian tube closure, resulting in the development of negative middle-ear pressure. Equilibration between middle-ear pressure and atmospheric pressure can only occur by the inflow of air through the eustachian tube.

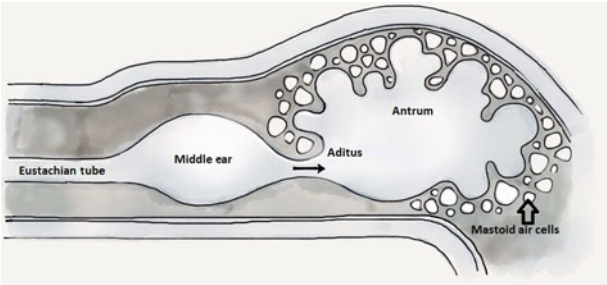

FIG. 1

Illustration demonstrating normal airflow (arrow) in the mastoid system.

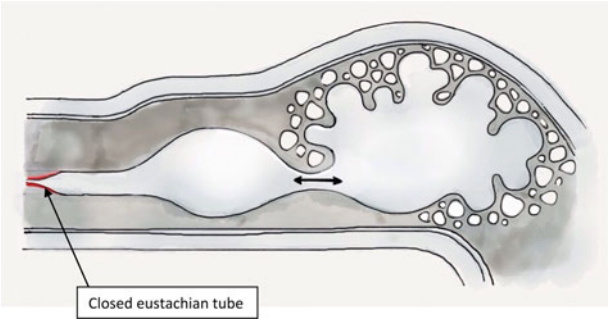

FIG. 2

Illustration demonstrating equilibrium of airflow (double-headed arrow) between the mastoid air cells and middle ear when the eustachian tube is closed.

In turn, equilibration of pressure between the mastoid air cells and the middle ear depends on the passage of air through the attic and aditus. The well-pneumatised mastoid requires a larger volume of air than the sclerotic mastoid to fill, and therefore it may potentially take longer for pressure to equilibrate. If eustachian tubal function is impaired, there may be insufficient time for this to occur. Furthermore, any disease process (such as adhesions, oedema or residual exudates from previous middle-ear effusion) that restricts the flow of air through the attic and aditus from the middle ear to the mastoid air cells may compound this effect.

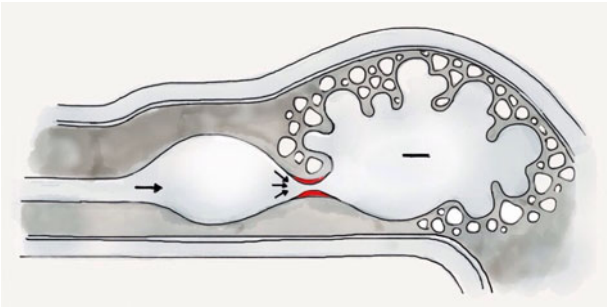

FIG. 3

Eustachian tube is open, airflow between the middle ear and mastoid antrum is impeded (arrows) because of disease in the aditus, leading to a negative pressure in the antrum relative to the middle ear.

https://doi.org/10.1017/S0022215115003047 Published online by Cambridge University Press

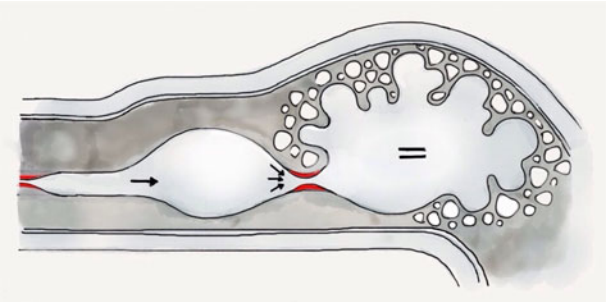

FIG. 4
Eustachian tube is closed; further relative negative pressure causes air to flow into the middle ear (arrows) and create a suction effect, which may also affect eustachian tube opening.

With regard to impaired eustachian tube function, we postulate that in some circumstances there may exist a relative pressure gradient between the middle ear and mastoid air cells. The prolonged negative pressure on the tympanic membrane may then lead to retraction formation and the subsequent development of cholesteatoma (Figures 3 and 4). In these circumstances, it is the well-pneumatised mastoid that never fully equilibrates which has an even greater potential to exert prolonged negative pressure on the middle ear. If this theory is correct, the development of cholesteatoma may occur quite rapidly, as the potential risk factors of a restricted aditus and a period of eustachian tubal dysfunction coincide.

- **In this study of 393 patients, pneumatisation of the mastoid was sclerotic in 90, diploic in 66 and pneumatised in 237 ($p < 0.001$)**
- **The study demonstrates the presence of cholesteatoma in well-pneumatised mastoids**
- **Cholesteatoma formation may in part be due to a pressure gradient created between the middle ear and mastoid air cells**
- **This may be more likely to occur in a well-pneumatised mastoid**

Any study based on a large series of patients with cholesteatoma demonstrates the very wide age range of patients at presentation. In this series, there are several patients who underwent mastoid surgery after their children, and others who developed cholesteatoma after having what appeared to be a stable retraction pocket over a period of years. Furthermore, some patients experienced late onset of cholesteatoma, and others developed second-sided cholesteatoma, at a much later date, in what was previously an apparently normal ear. Our theory may also explain the not uncommon finding of cholesteatoma in an ear which appears to have relatively normal eustachian tubal function at the time of surgery.

Limitations

A potential weakness in this study is that assessment of the degree of pneumatisation is subjective. However, all patients were graded by the same surgeon as part of a wider data collection of operative findings, therefore eliminating any subjective variation in the grading or observer bias.

Conclusion

This study demonstrates the presence of cholesteatoma in well-pneumatised mastoids. We hypothesise that a well-pneumatised mastoid may lead to cholesteatoma formation as a result of a pressure gradient created between the middle ear and antrum. Undoubtedly, the development of cholesteatoma is complex, but this theory may explain some of the unusual features of the presentation of the condition.

Acknowledgement

All illustrations are by Dr Morium Howlader (Northern Deanery, Victoria Road Health Centre, Washington, UK).

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Miss I Z Iqbal takes responsibility for the integrity of the content of the paper
Competing interests: None declared