The Journal of Laryngology and Otology
February 1992, Vol. **106**, pp. 162–163

Myringitis bullosa haemorrhagica: clinical course influenced by tympanosclerosis

JOHN K. S. WOO, F.R.C.S., C. ANDREW VAN HASSELT, F.C.S.(SA), M.Med., (Otol), PAUL G. C. GLUCKMAN, F.R.C.S.
(Hong Kong)

Abstract
Observations based on two contrasting cases of myringitis bullosa haemorrhagica indicate that tympanosclerosis involving the tympanic membrane may have a significant effect on the clinical course of the disease. The evidence suggests that the bullae associated with the condition arise from the epidermal layer of the eardrum.

Introduction

Myringitis bullosa haemorrhagica is a painful, self-limiting condition of the ear of obscure aetiology. A bullous eruption may be seen on the tympanic membrane of the involved ear. The bullae may involve the external auditory canal (Pulec, 1980). Most patients will present at this stage as the associated otalgia reaches a climax. Alternatively, if a patient presents at a later stage, the bullae may have ruptured givng rise to bleeding and serosanguinous discharge, leaving only the outline of the blebs visible on otoscopy. When uncomplicated, hearing loss is usually transient, conductive and mild although neurosensory hearing loss has been reported following the condition (Hoffman and Shepsman, 1983; Lashin *et al.*, 1988; Hariri, 1990). The diagnosis of the myringitis bullosa haemorrhagica is a clinical one with histological appearances not having been described in the literature. Nevertheless, the bullae appear to involve only the superficial epidermal layer of the tympanic membrane (Browning, 1987). Indirect supportive evidence of this observation comes from the histological appearance of other bullous dermatological conditions which arise from or affect essentially the epidermis (Haber, 1980).

The aetiology of the condition is obscure though it has long been attributed to viral infection. In the majority of cases, however, a virus cannot be isolated (Roberts, 1980). The only evidence that this condition may be of viral origin is the common association of a preceding upper respiratory tract infection (Browning, 1987; Lashin *et al.*, 1988).

The recent experience of two such cases leads to the following interesting observations which may be of clinical significance.

Case reports

Case 1

A 37-year-old gentleman presented with severe progressive left otalgia over a two day period to the accident and emergency department. This was the most severe pain ever experienced by the patient. There was no otorrhoea and hearing was subjectively normal. He gave no history of previous ear disease but there was a preceding flu-like illness two weeks prior to the onset of pain. On examination, there were two large haemorrhagic bullae on the anterior and inferior part of the tympanic membrane obscuring almost the whole eardrum (Fig. 1). The Rinne test was positive on both sides but the Weber test lateralized to the diseased ear. The patient was seen three days later, the bullae having ruptured, by that time the pain had almost completely subsided. The

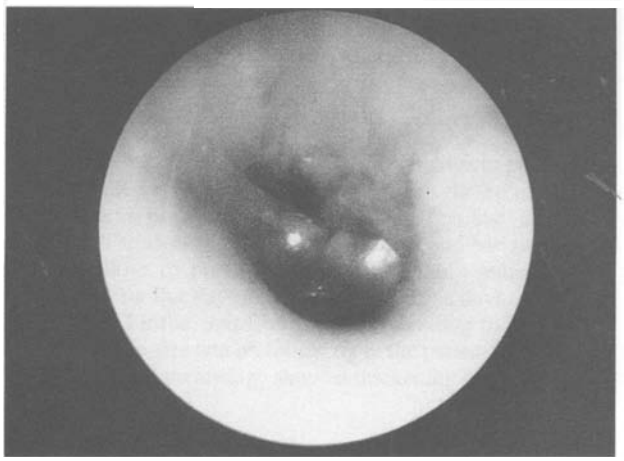

FIG. 1
Myringitis bullosa haemorrhagica of a previously normal eardrum. Note the almost complete involvement of the ear drum.

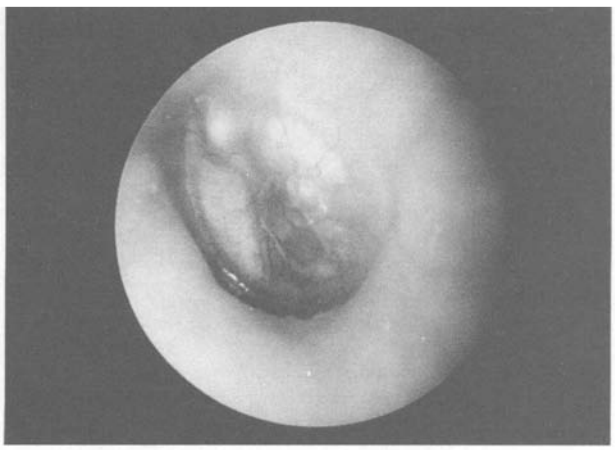

FIG. 2
Myringitis bullosa haemorrhagica of an ear drum previously involved by tympanosclerosis. The outline of a ruptured bulla is shown. Note the obvious sparing of the sclerotic area of the drum.

Accepted for publication: 9 October 1991.

tympanic membrane was intact and the base of the bullae were clearly seen. When seen two months later, the ear drum was normal in appearance and the Weber test was central.

Case 2

A 49-year-old housewife presented to the Otolaryngology outpatient clinic complaining of left otalgia of three days duration. She had a history of recurrent otorrhoea but had been free of discharge for the previous year. She had noticed no change in hearing. There had been no preceding upper respiratory tract infection. Examination revealed a haemorrhagic bulla over the antero-inferior aspect of the eardrum sparing the antero-superior and the postero-superior part which was heavily involved by tympanosclerosis. The other ear was completely normal. Pure tone audiometry showed a moderate conductive hearing loss on the diseased ear. A clinical diagnosis of bullous myringitis was made and the patient was given paracetamol for pain relief. At review the following day, it was apparent that the bulla had ruptured spontaneously so that only its outline remained visible (Fig. 2). The pain was not excessive but took two weeks to subside completely. When seen again six weeks later, no sign of previous bullous myringitis was evident, and the repeated pure tone audiogram showed no significant change when compared to the earlier assessment.

Discussion

The normal eardrum is made up of three layers with the exception of the pars flaccida. The outer squamous epithelial layer is separated from the inner mucosal layer by a middle fibrous layer. The outer epithelial layer, which is continuous with the epithelial lining of the external canal, is richly innervated by pain fibres so that the middle ear is protected from self-inflicted injury. In general, any acute lesion involving the epithelial layer of the eardrum is extremely painful. The severe pain of bullous myringitis and the occasional involvement of the external canal by the bullae leads one to believe that the bullae arise from, or, at least involve the epithelial layer of the eardrum. Histological evidence for this is however lacking, as there is no justification for the taking of a biopsy (which must be full thickness) of the eardrum from patients with the condition. A review of the literature reveals no histological description of this disease.

The difference in the site and extent of involvement between the two cases described, suggests that the bullae of myringitis bullosa haemorrhagica appear to affect only the 'anatomically normal' ear drum or the 'normal part' of a previously abnormal ear drum. This observation is in keeping with the belief that the bullae develop from the superficial (epidermal) layer of the ear drum. Tympanosclerosis involving the tympanic membrane affects the middle fibrous layer resulting in fusion of the different layers (Gibb, 1979) and thus may limit the development and progression of the bullae associated with the condition. It is interesting to reflect on the difference in the severity of pain experienced by the two patients. It would be logical to assume that the pain experienced by the patient would be proportional to the area of the drum affected. In the first patient, the extensive involvement by bullae was associated with severe pain. The presence of tympanosclerosis of the tympanic membrane in the second patient theoretically limited the progression of the bulla causing rupture when still small. The pain experienced by the second patient was mild. After one attack of myringitis bullosa haemorrhagica, there would inevitably be some degree of fusion of the different layers of the tympanic membrane making it less likely for the same disease process to recur in the same ear. A literature search failed to find any documented case of recurrent myringitis bullosa haemorrhagica in the same ear. However, one patient did have each ear involved separately, on two different occasions (Hariri, 1990). In Hoffman's series, two patients developed a second attack of bullous myringitis, but no mention was made whether this happened on the same or contralateral ear (Hoffman and Shepsman, 1983). The fact that the second patient took over two weeks for the pain to subside may be due to the poor blood supply of a tympanosclerotic eardrum thus prolonging the time for the ruptured bulla to heal.

Conclusion

The differences in the clinical features observed in these two cases of myringitis bullosa haemorrhagica support the proposition that the bullae affect the superficial layer of the ear drum. It seems likely that tympanosclerosis restricts the extent of involvement of the tympanic membrane by bullae and hence limit the pain associated with the condition.

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Address for correspondence:
C. Andrew van Hasselt,
Senior Lecturer and Chief,
Division of Otolaryngology,
Chinese University of Hong Kong,
Prince of Wales Hospital,
Shatin, Hong Kong.

Key words: Ear deformities, acquired; Tympanic membrane