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THE ROUND WINDOW NICHE AND MEMBRANE IN CHRONIC SUPPURATIVE OTITIS MEDIA

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ABSTRACT

This study evaluated the histopathologic changes in the round window niche and membrane in cases of chronic suppurative otitis media. The pathologic process was found in all subjects. Fibrocystic or fibrous tissue was most commonly seen, while active granulation was less developed. The round window membrane was thicker and inflamed throughout its layers. Inflammatory invasion of the inner ear through the membrane is very rare, because the thickened membrane has a protective role for spreading of infection.

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Keywords: Round window niche, Membrane, Chronic suppurative otitis media.

INTRODUCTION

Pathological changes in the round window niche or membrane may occur in various otological diseases such as congenital malformations, otosclerosis, acute and chronic otitis, trauma and middle ear injuries.[1] Previous studies have shown that an inflammatory process and its products may spread through the round window membrane and produce suppurative labyrinthitis.[2] It was found that chronic middle ear infection may induce sensorineural hearing loss by producing toxins which penetrate the round window membrane and damage sensory cells in the lower cochlear basal turn.[3] In chronic otitis media the pathologic process is localized in the round window niche in about 45.5% of cases.[4]

The aim of this report is to describe histopathological changes in the round window niche and membrane in chronic suppurative otitis media and to suggest their clinical importance.

MATERIAL AND METHODS

The study was done on temporal bones collected from the Massachusetts Eye and Ear Infirmary in Boston. 26

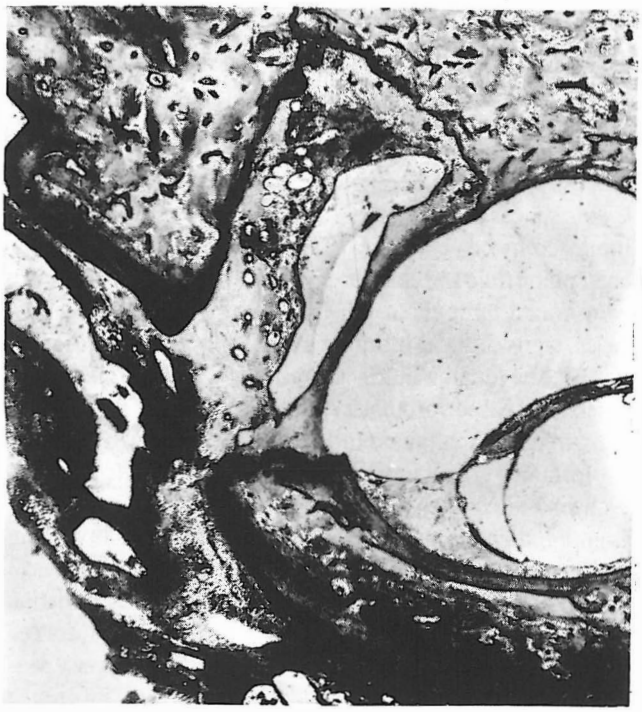

Fig. 1. Fibrocystic tissue occupies the round window niche. Inflammatory infiltrates are present in the low niche part. A similar process is seen throughout the middle ear space (H&E, ×120).

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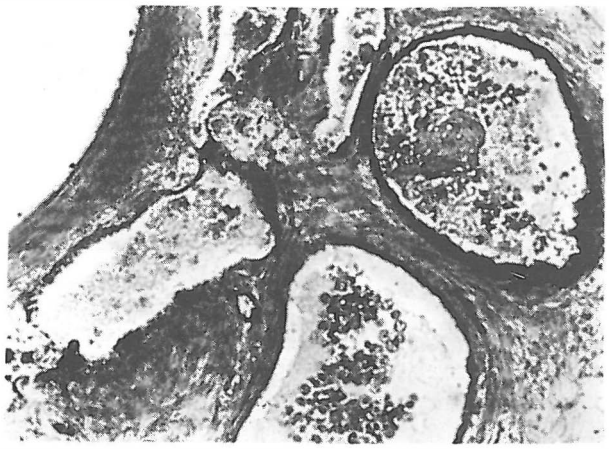

Fig. 2. Fibrocystic tissue is attached to the round window membrane. Thickening of the membrane is marked with diffuse infiltration throughout its layers (H&E, ×200).

Fig. 3. Active inflammatory tissue partly occupies the round window niche. Marked inflammation and vascularity are seen. The round window membrane is involved by inflammation (H &E, ×120).

temporal bones were chosen from subjects who, prior to dying, had chronic otitis media, but histopathologically, fibroproliferative or granulation processes had been observed within the middle ear spaces. The specimens of the temporal bones were processed by standard laboratory procedures for hematoxylin and eosin stain.

RESULTS

In all studied cases the round window niche was involved by the pathological process. The pathology of this region showed the following characteristics:

A fibrocystic or fibrous process more or less occupied the round window niche in the majority of cases. This process was often adherent to the round window membrane. Cystic spaces of various size were observed throughout the loose connective tissue. These spaces contained an acidophilic fluid and cellular debris. Inflammatory infiltrates were found throughout the fibrous niche tissue. In some cases, signs of focal inflammation were observed (Figs. 1 and 2). The round window membrane was thickened in all layers. Increased vascularity and invasion of inflammatory cells were seen in the connective tissue layer.

Inflamed granulation tissue involved the round window niche in only four cases. The inflammatory process spread from the niche to the round window membrane involving the portion of the epithelium facing the middle ear (Figs. 3 and 4). All membrane layers were thickened but inflammation was more developed in the outer and middle layers. Marked inflammatory cell invasion of the perilymphatic space was obvious in two cases. The membrane layers were infiltrated severely and could not be distinguished (Fig. 5). The inflammatory granulation tissue of the round window niche showed direct extention as an identical process in the posterior tympanic spaces as well.

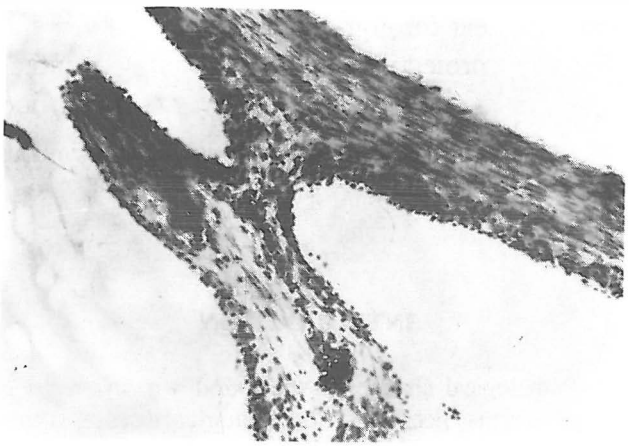

Fig. 4. Higher magnification of Fig. 3. The inflammatory process has spread into the membrane mostly in the outer layer (H&E, ×120).

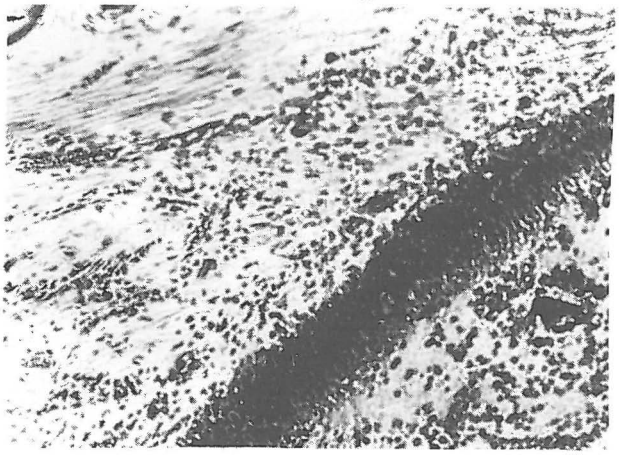

Fig. 5. Inflammatory process spreading from the round window niche (arrow) into the inner ear (scala tympani). All layers of the membrane are severely infiltrated (H&E, ×300).

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DISCUSSION

The round window membrane is the only soft tissue structure separating the middle ear from the inner ear. It consists of an outer, middle and inner layer. The outer layer is divided into an epithelial layer, which has epithelial cells contiguous with the mucous membrane of the promonotory, and a subepithelial connective tissue layer which lies between the outer and middle layer. The middle layer consists of connective tissue which contains mainly fibroblasts, collagen and elastic fibers. The inner membrane layer is basically a continuation of the mesothelial cells lining the scala tympani of the cochlea. The cell junctions are usually loose and the intercellular spaces are wide.

The round window membrane is permeable to various biological substances which may pass through the membrane and cause inner ear damage and is a very important structure when considering the inner ear pathology of sensorineural hearing loss associated with otitis media. Previous reports have shown that penetration of substances into the inner ear is largely dependent on the morphological status of the round window membrane, the concentration of the substances in the niche, exposure time, and local resorption and elimination from the niche and middle ear.[5]

In chronic otitis media, bacterial toxins may pass into the cochlea and cause hearing loss. It was found that staphylococcal exotoxin caused marked morphological changes in the round window membrane in experimentally induced otitis.[6] Sensorineural hearing loss as a complication of chronic otitis media due to the passage of inflammatory products through the round window membrane has been reported by numerous authors. Paparella et al.[7] described that the round window membrane is thicker in chronic otitis media and postulated that it protects the cochlea much better than when it is thin and permeable. The authors have also suggested that children with acute inflammation and a thin round window membrane have a greater risk of inflammatory cell invasion than adults with a membrane thickened due to chronic otitis. Inflammatory cell invasion of the inner ear may cause labyrinthitis.

Our findings correlate with other studies and suggest the protective role of the round window membrane in chronic otitis media. Pathologic changes in the round window niche, occurred in all cases of chronic suppurative otitis media. The niche is most often occupied by fibrocystic tissue showing more or less inflammatory infiltration spreading to the round window membrane. Thickening of the membrane is present in all cases, while in some it is notably inflamed. Inflammatory invasion of the inner ear through the membrane is very rare, since the thickened membrane protects the inner ear from inflammation.

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