Pathology of Secretory Otitis: A Temporal Bone Report

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During the past 10 years, the collection and microscopic examination of human temporal bones has provided the opportunity to correlate clinical symptomatology and findings with histopathology. The case findings in this report support the clinical suspicion of glandular mucosal disease in some forms of secretory otitis media.

Chronic secretory otitis media (serous or mucous) is one of the most frequent and frustrating problems in otology. Hall has attributed the growing number of such cases in recent years to increased use of antibiotics. Hoople and Suehs have pointed out that some of this apparent increase may be due to greater otologic interest in secretory otitis as a cause of persistent hearing impairment.

Numerous opinions have been expressed on the etiology of middle ear fluid, ranging from pure pressure considerations following eustachian tube occlusion to pure mucosal disease with production of fluid. As early as 1883, Politzer described middle ear fluid consisting of a serous or mucous exudate. He felt this was caused by an inflammatory process of the mucosa, but was favored by simultaneous rarefaction of the air in the middle ear due to tubal obstruction. This well-known theory is based on the absorption of oxygen from an isolated volume of air. Middle ear pressure becomes negative in relation to the atmospheric air and "hydrops ex vacuo" develops, sometimes accompanied by "hydrops ex vacuo."

In 1931, Blegvad opposed this theory. In his opinion the observations used as conclusive evidence of tubal occlusion had been misinterpreted. He alleged that the tube was not hermetically closed and that there is no such thing as negative middle ear pressure, but a positive pressure which causes inflammatory exudate instead of transudate.

Ruttin believed effusion in the middle ear was due to lymphatic and venous stasis, caused by a mass in the nasopharynx or mucosal swelling in the eustachian tube or its orifice.

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Zöllner found the tube was often clinically patent in the presence of middle ear fluid, and therefore regarded the condition as an exudate due to inflammation.

Bendek biopsied 40 cases of transudatory-secretory otitis media and found secretory epithelium with or without glandular structures in half the cases. Ten of 15 tested showed positive mucicarmine staining, indicating the presence of mucus. He also found secretory epithelium in three of ten control patients with otosclerosis.

Friedman stated that the normal healthy ear exhibits secretory columnar epithelium only in the neighborhood of the eustachian tube orifice, and thus the middle ear lumen is not a true mucosa. But he found mucosal changes, including glandlike columnar structures and columnar epithelium, both in purulent and secretory otitis media. He felt that, once established by eustachian tube obstruction or inadequately treated otitis media, these mucosal changes act as a source for middle ear fluid whether or not the eustachian tube is patent.

Recently Sade pointed out that prolonged decalcification of temporal bones will in most cases destroy cilia and mucus in the middle ear. With reduction of bony bulk and shorter decalcification, he found the normal middle ear to have a true mucosa possessing cilia and secreting mucosa over a wide area. He interpreted the active mucous glands in ears with secretory otitis media as an exaggeration of the normal state.

As is suggested from these reports, the histopathology and pathogenesis of chronic secretory otitis media are not clearly understood, with a multitude of relevant studies resulting in conflicting opinions. One difficulty has been the paucity of suitable temporal bone specimens.

It is the purpose of this report to present the pathological findings of an interesting pair of human temporal bones considered compatible with a diagnosis of long-standing secretory otitis media and mastoiditis.

Case Report

A 75-year-old white woman was first admitted to Henry Ford Hospital on March 12, 1963, with general symptoms suggesting metastatic tumor. When a pulmonary consolidation with pleural effusion was found, she was referred to the ENT Department for evaluation and bronchoscopy.

The ENT consultation elicited a history of a progressive hearing loss for four or five years, but no hearing aid use. She had no history of ear infections, toxic drug exposure, or familial hearing loss. Examination revealed bilateral nasal polyps. Both tympanic membranes appeared scarred but intact. On tuning fork testing, the Weber test was in the midline and the Rinne test was negative in each ear. Audiometric examination showed a combined loss in each ear with decreased discrimination.
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CASE D. L.

![Audiogram](image)

RT LT
PB.MAX 36% 60%
S. R. T. 45 35

Figure 1

Audiogram taken five months before patient's death. Right ear line — X; Left ear line — O.

(Fig. 1). The audiologist felt her responses suggested CNS involvement and noted the patient's comment of "trouble thinking what I want to say."

Bronchoscopy and subsequent biopsy revealed a well differentiated adenocarcinoma in the right lower lobe. Skull x-rays showed calcified parietal density, interpreted as either an old subdural hematoma or meningioma. EEG showed disturbance of the entire left hemisphere.

The patient died five months later. Significant autopsy findings included metastases to both parietal lobes of the brain and meningioma, left temporal-parietal area.

Temporal Bone Histopathology:

Since the two ears were very similar, they will be described together. The mastoid cells of both ears were completely filled with dense, fibrous tissue with numerous cuboidal epithelium-lined glands with eosinophilic secretion in the lumina (Figs. 2 and 3). PAS (Periodic-Acid-Schiff) stain for mucus was strongly positive in the glandular secretions. A thin layer of positive staining secretion was also seen overlying portions of the middle ear mucosa. There were a few scattered collections of lymphocytes present. Areas of bone erosion and new bone formation and many vascular channels were seen.
Figure 2a
Low-power view of left ear. Mastoid cells filled with dense fibrous tissue containing numerous glands. (S) stapes footplate, (F) facial canal, (L) lateral canal.

Figure 2b
High-power view of glands from insert area. The secretions were strongly positive staining with PAS (Periodic-Acid-Schiff).
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Figure 3a
Lower-power view right ear. Mastoid cells also showing fibrous tissue with glands. (L) latera canal, (F) facial nerve, (S) stapes footplate.

Figure 3b
High-power view of insert area mucus containing glands.
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The middle ear mucosa was very thick and fibrous and contained many of the same glands. In the right ear the space between the oval window and tympanic membrane was completely filled with fibrous tissue which surrounded the incus and the remnant of the largely destroyed stapes superstructure. The tympanic membrane was intact, but thick and fibrous (Fig. 4). The long process of the left incus was missing and a retracted pocket of the otherwise intact tympanic membrane was in contact with the stapes head (a spontaneous type III tympanoplasty), with no evidence of cholesteatoma. The remainder of both middle ear spaces was air-containing, and the tympanic orifices of both eustachian tubes were patent (Fig. 5). The facial canals were intact.

Figure 4
Right ear showing thick tympanic membrane (TM) and fibrous tissue from malleus (M) to stapes footplate (S).
Figure 5
Right ear with air-containing middle ear (ME) and patent tympanic orifice eustachian tube (E).

Figure 6
Mid-modiolar section right cochlea.
In the cochlea the organ of Corti was present in all turns of both ears (Figs. 6 and 7). Because the organ was shrunken in the right ear, individual hair cells could not be distinguished but appeared to be present. In the left ear hair cells were present in all turns. There was a mild degree of hydrops in the apex of the right ear but none in the left. The stria vascularis had patchy areas of atrophy which were greater in the right ear. Both ears had a moderately severe loss of spiral ganglion cells in the lower basal turn, less severe in the upper basal turn, slightly greater in the right ear.

The maculae of the saccule and utricle were grossly normal but seemed to have some missing hair cells. The semicircular canal cristae appeared normal. The auditory nerves and internal auditory meati appeared normal on both sides. No endolymphatic sac abnormalities were seen.

Discussion

This pair of adult temporal bones represents an interesting case with unusual pathology. Of particular interest is the bilateral presence of marked mucous gland formation throughout the mastoid air cell system and middle ear mucosa in the presence of air-containing middle ears and apparently patent eustachian tubes, without evidence of active infection.

These findings give pathological support for the clinical impression that at least one form of secretory otitis can occur in spite of a patent eustachian tube. Clinically a few patients are seen who can readily autoinflate their ears, but continually accumulate gluelike mucoid secretions. In this form of secretory otitis the persisting disease could be mainly mucosal, although the underlying etiology may have been eustachian tube dysfunction or infection.

It is somewhat remarkable to note the small amount of air bone gap on the audiogram (Fig. 1) in view of the degree of middle ear pathology. The presence of a mobile tympanic membrane with a spontaneous type III tympanoplasty and an air-containing middle ear makes this feasible in the left ear. With the absence of most of the right stapes superstructure, the dense fibrous connection from the tympanic membrane to the stapes footplate must have provided excellent sound conduction to result in a conductive loss this small.

Although there is no single striking pathological finding in the inner ear to explain the sensorineural component of the patient’s hearing loss, both cochleas do show several types of mild pathology (ie, stria atrophy, ganglion cell loss, right apical hydrops) which taken together are compatible with the audiogram, the findings being more severe in the right ear.

Summary

The histopathological findings of an interesting pair of adult temporal bones have been presented. Both ears show extensive mucous gland formation and fibrosis.
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Figure 7
Mid-modiolar section left cochlea.

of the mastoid and middle ear mucosa in spite of air-containing middle ears and absence of active infection.

The conductive portion of the patient's hearing loss was remarkably small considering the middle ear pathology.

Theories of etiology of secretory otitis media are reviewed. These bones give evidence for a mucosal form of this disease.

REFERENCES


