

Acute otitis media. Histopathological changes: a post mortem study on temporal bones

F. SALVINELLI, F. GRECO, M. TRIVELLI, F.H. LINTHICUM JR*

Institute of Otolaryngology, "Campus Bio-Medico" University - Rome, Italy

*Department of Histopathology, "House Ear Institute" - Los Angeles, CA, USA

Abstract. – A classification of otitis media is proposed and the histopathological changes of otitis media with effusion are reviewed post mortem on two temporal bones. A case of adhesive otitis media is observed on temporal bone slides. The clinical and therapeutic aspect are examined and treatment guidelines are proposed.

Key Words:

Secretory otitis media, Eustachian tube.

Introduction

Infection of the middle ear causes not only generally known inflammatory changes but also others peculiar to the site.

Otitis media is one of the most common of all diseases, particularly in young children. The clinical forms of the acute and chronic conditions correspond to the pathological changes, but intermediate or mixed states are frequent. The presence or absence of perforation of the tympanic membrane accounts for two of the three subgroups. The third is that of an intact tympanic membrane but with an effusion in the middle ear (Table I).

These conditions may each be acute, subacute or chronic.

Materials and Methods

We have studied the histopathological changes of two cases of otitis media with effusion, one in an early stage and the other in a late stage of illness and a case of adhesive otitis media. These patients were donors and

agreed during their life to donate post mortem their temporal bones to the House Ear Institute Los Angeles, CA, USA as a contribution to a better knowledge of temporal bone diseases.

We have removed the temporal bones in a standard way.

The usual method of approaching the temporal bone at post-mortem involves prior removal of the skull cap and brain¹. In doing so the dura should be treated carefully and left adherent to the temporal bone in order not to damage the endolymphatic sac. The seventh and eighth cranial nerves should be cut at the orifice of the internal auditory meatus so as to leave portions of the nerve trunks within the temporal bone specimen. A vibrating electric saw is satisfactory for removing the petrous temporal bone. A triangular blade is better than the more commonly employed circular blade. Three vertical and one horizontal cuts are made with the saw.

a) The first cut is set medial to the internal auditory meatus and extends vertically through the petrous temporal bone at right angles to the superior and posteromedial surfaces to a depth of approximately 5 cm.

b) The second cut is made parallel with the first and at least 5 cm posterolateral to it at the lateral end of the temporal bone. It also passes vertically to a depth of > 5 cm. This cut leaves out most of the mastoid air cell system.

c) The third vertical cut is made connecting the forward ends of the two previous cuts, approximately parallel with the free anterior end of the petrous temporal bone at the posterior extent of the middle cranial fossa.

d) A fourth cut is made behind the petrous temporal bone and parallel with it, at the anterior extent of the posterior fossa.

Table 1. Classification of otitis media.

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| <ol style="list-style-type: none">1. Otitis media without effusion or perforation of the tympanic membrane2. Otitis media with effusion, but without perforation of the tympanic membrane (serous otitis media)3. Otitis media with perforation of the tympanic membrane |
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The block is removed by gently “rocking” and cutting the ligamentous structures on its inferior surface. So removed it includes a portion of the ear canal, the tympanic membrane, the middle ear, the labyrinthine structures and the petrous portion of the seventh and eighth cranial nerves.

Slicing method

The temporal bone is removed at post-mortem as described above. Fixation take place for a minimum of 4 days. The bone is then mounted with molten dental wax on a glass plate measuring 6 × 5 cm with a thickness of approximately 0.5 cm. The surface to be presented for slicing is arranged perpendicular to the glass plate. The glass plate, with the surface of the temporal bone that is to be cut to the front, is now mounted on the metal plinth attached with dental wax to the inner end of the lever of the slicing machine (Microslice 2 Precision Annular Saw, available from Cambridge Instruments Ltd., Rustat Road, Cambridge, CBI 3QH, England). This is a cutting machine with a circular steel blade which is bolted to the machine at 16 point to prevent lateral vibration. Cutting proceeds around a circular inner opening where the blade is tipped with diamond. The cutting edge is lubricated by a continuous jet of cold water. The speed of the rotatory motor may be adjusted from low up to 1200 rpm by the left hand knob on the front of the machine. The right hand knob advances the lever with the specimen by the required length before each slice is made so that the thickness of the specimen can be regulated. Slices of 1, 2 or 3 mm thickness may be prepared. Slicing is carried out by gently lowering the weighted left-hand counterpoised end of the lever so that the specimen rotates up and is applied against the cutting edge. With this system the specimen backs

away from the blade when a particularly hard area is encountered, so avoiding excessive mechanical and thermal stresses. The slices adhere together and are removed from the machine after the whole temporal bone has been treated. Each slice is N-rayed with a laboratory X-ray machine (such as 4380 5N X-ray, Faxitron system, made by Hewlett Packard)².

Discussion

In the acute phase of otitis media *Streptococcus pneumoniae* and *Haemophilus influenzae* are the most common causative organisms. Epidemiological studies have indicated certain respiratory viruses as possible agents in the early phases of the illness³. In the chronic phase Gram negative organisms, particularly *Proteus* and *Pseudomonas* are found, although *Staphylococcus pyogenes* and beta-haemolytic streptococci are sometimes isolated from the discharging pus of chronically inflamed ears. Although much less frequent than the above organisms, *Mycobacterium tuberculosis* may be the causative agent of chronic inflammation of the middle ear. In such cases the inflammatory reaction is quite distinct.

General pathological changes

The acute phase is characterized by a severe congestion of the mucosa of the middle ear and the tympanic membrane. It is not generally realized that congestion of the mucosa is frequently also a marked feature of chronic otitis media. The fluid portion of blood, plasma, may leave a deposit of fibrin in the tissues. A fluid exudate in the middle ear cavity is frequently a prominent component of the inflammatory reaction - a specific form of the disease known as otitis media with effusion. In these cases mucus may be secreted by newly formed glands in the middle ear mucosa and may contribute to the fluid “exudate”. In acute inflammation neutrophils are prevalent. In chronic inflammation histiocytes (derived from monocytes of the blood), lymphocytes and plasma cells (derived from lymphocytes), are the characteristic infiltrate. Organisms are seen very rarely in histological sections of acute or chronic in-

flammation of the middle ear. In newborn infants an inflammatory reaction may be the result of the contamination of the middle ear by inhaled amniotic squames. In these cases the histiocytes reacting to the foreign material fuse to form giant cells. Haemorrhage is a common result of the congestion of otitis media. It may lead to cholesterol granuloma. Local tissue cells frequently react to the inflammatory process by dissolution or proliferation. Necrosis may occur, as is characteristic of perforation of the tympanic membrane or rarefying osteitis of the ossicles. Several processes may produce the necrosis. It is likely that rupture of the tympanic membrane takes place as a result of ischaemic necrosis caused by pressure at a focal point. Ossicular loss may, on the other hand, be caused by substances such as collagenase produced by the inflammatory connective tissue on the surface of the ossicle⁴. At the same time as the process of necrosis, proliferative activity of middle ear tissue occurs and may represent an important part of the pathological picture. The columnar epithelium of the middle ear has in the presence of inflammation the remarkable property of invaginating itself to produce glands, which often develop luminal secretion. The glandular transformation of the middle ear mucosa may be seen in any part of the cleft, including the mastoid ear cells. The secretion of the glands contributes to the exudate in otitis media with effusion. Fibrous tissue proliferation may also occur in combination with glandular transformation - a process which Schuknecht⁵ has called fibrocystic sclerosis. Squamous cell epithelium may likewise proliferate in the middle ear - a process known as cholesteatoma. A specific form of reparative reaction following inflammation is the development of granulation tissue. In this process the endothelium of blood vessels and fibroblasts are the newly formed cells. Mononuclear inflammatory cells usually accompany the latter. Fibroblasts and collagen are abundant in the terminal phase of the reparative stage. A normal degree of cellularity in the fibrous reaction is seen in adhesive otitis. A peculiar form of scar tissue production occurs in the middle ear, in which the collagen is poorly cellular and hyalinized. This condition, known as tympanosclerosis, is characterized also by deposition of calcium salts in the hyaline fibrous tissue. The bony

walls of the middle ear frequently react to the inflammatory process by a new formation of bone. This is woven in the early stages and lamellar later.

Incidence and clinical features

The incidence of acute otitis media as seen in hospital practice in developed countries has declined over the past 25 years because of the ready availability of antibiotics and improved socio-economic conditions. Children are more often affected than adults. The clinical features are general signs of infection, pain, particularly in the mastoid area, tenderness and swelling in the postauricular region and oedema of the posterosuperior wall of the external auditory meatus. The tympanic membrane is initially hyperaemic and then bulges as more pus collects in the middle ear, until eventually it may burst.

Pathological appearances

The appearances of the middle ear mucosa as seen in the bone chips removed at mastoidectomy have been described by Friedmann⁶. The mucosa of the mastoid air cells is congested and oedematous. Haemorrhage may be severe and the mucosa and air cells are filled with neutrophils. Pus destroys bone, the actual dissolution being carried out by osteoclasts. At the same time new bone formation takes place, commencing as osteoid, later becoming woven and finally lamellar. Fibrosis may also be active even in the acute stage. Acute inflammatory changes are also prominent in other parts of the middle ear. The tympanic membrane shows marked congestion, the dilated vessels distending the connective tissue layer. Pus cells fill the middle ear cavity. The acute inflammation may spread deep into the temporal bone as osteomyelitis.

Secretory otitis media

Synonyms commonly in use for secretory otitis media are: serous otitis media (SOM), otitis media with effusion, catarrhal otitis media and tubotympanitis. It has attracted much attention in recent years because of its frequency as a cause of hearing loss in children. It is defined by Sadè⁷ as a "condition in which an effusion is present behind an intact drum in the absence of frank symptoms of acute in-

fection". The lesion is said to affect 5-10% of all children, in many of whom there are no symptoms. Adults may complain of shifting fluid in the ear. The importance of adenoid enlargement as a cause has been disputed and the question of Eustachian tube obstruction as an important precursor is still open. The pathological changes that have been associated with the clinical syndrome of secretory otitis media read like a review of the alterations described above. Cholesterol granuloma, chronic inflammatory granulation tissue, ossicular destruction and tympanosclerosis are all important features and the tympanic membrane itself often changes with the progress of the disease, becoming "atelectatic" (i.e. adherent to the medial wall of the middle ear) or even forming a retraction pocket (Figures 1, 2 and 3).

Cholesteatoma may develop behind the intact tympanic membrane or with the formation of a small perforation in the pars flaccida. Chronic otitis media is an extremely common malady. In a pathological study of 123 temporal bones with chronic otitis media 46% of active lesions and only 19.5% of inactive lesions showed a perforation⁸. Secretory otitis media is one form of chronic otitis media without perforation.

Conclusion

We suggest more attention to the causes of otitis media with effusion, which are mainly

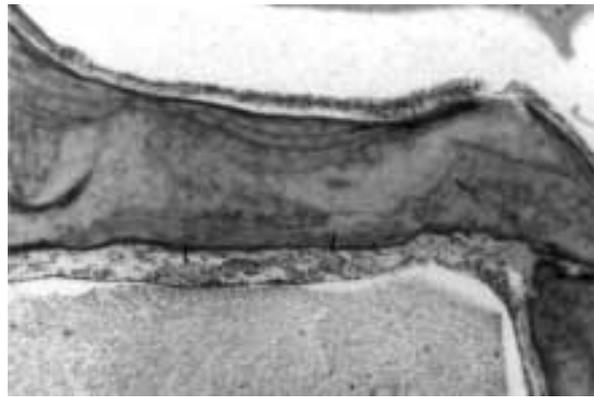


Figure 2. Otitis media with effusion, late stage. Submucosa is hyperplastic and hyperemic. Note dilated blood vessels. $\times 110$

alterations of nose, rhinopharynx and Eustachian tube. The careful evaluation of these regions allow the correct treatment of the causes of ear, nose and throat obstruction, which are the causes of recurrent and chronic. As visible in Figure 1, the early stage of is rich of fluid and devoid of pus cells. In this cases a general antinflammatory and nasal topical decongestant therapy is mandatory, but no antibiotic is necessary. With the recovery of a normal Eustachian tube function, the acute inflammation withdraws. The persistence of Eustachian tube dysfunction cause a negative pressure in the middle ear cavity, with development of an adhesive otitis media: the tympanic membrane is sucked persistently down towards the cochlea, with permanent transmissive

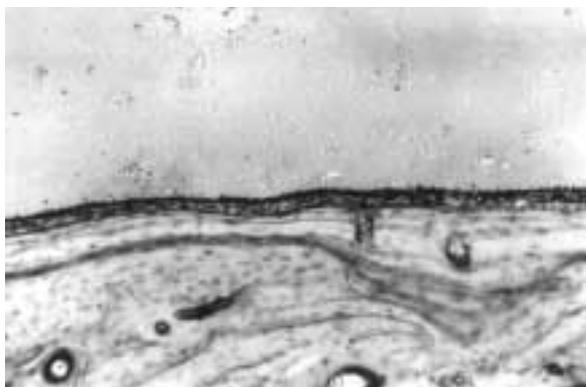


Figure 1. Otitis media with effusion, early stage. A few pus cells are scattered in the fluid in the middle ear space adjacent to the bone of the promontory. $\times 110$

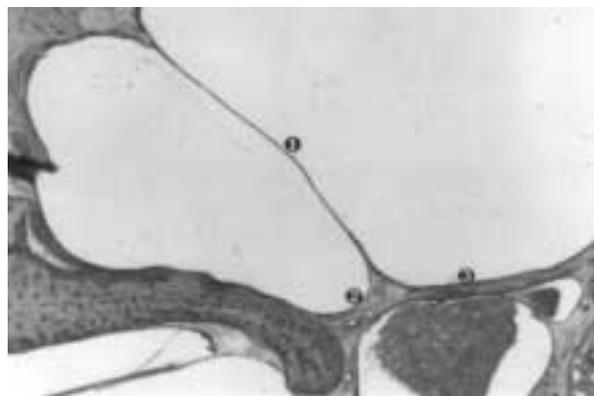


Figure 3. Adhesive otitis media. Atrophic ear drum (1) is adherent to the promontory by a fibrous band (2). The posterior third of the ear drum is of normal thickness (3). $\times 15$

hearing loss (Figure 3). In cases of suprainfection, antibiotics can be used, but always in synergism with local and general anti-inflammatory therapy. The current epidemic-like frequency of OME may be the result of the indiscriminate treatment of acute otitis with antibiotics in recent years^{9,10}.

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