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OTOLOGY

Vascularization of the long process of the malleus: surgical implications

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Abstract Knowledge of the vascularization of the malleus may help to better understand some unwanted effects occurred in those surgical techniques using the handle as an attachment site of ossicle-replacement prostheses. Our hypothesis is that vascular damage occurred in the long process of the malleus during tympanic membrane detachment before fastening a malleovestibulopexy (MVP) prosthesis promotes the lysis of this ossicle at the attachment site. To confirm this premise, we carried out an evolutive and descriptive study of ten MVP procedures as well as a morphological analysis of ten cadaveric mallei using both light and scanning electron microscopies. The arterial supply of the manubrium comes from two main sources: vascular branches that reach this ossicle from the site of insertion of the tensor tympani tendon and small perforating branches which penetrate the bone from arteries of the tympanic membrane that, following the periosteum/perichondrium, they arrange parallel to the bone. The detachment of the tympanic membrane to fasten a MVP prosthesis causes a damage in the periosteal/perichondral vascular supply of the handle as well as micro-trauma on it, which can promote bone resorption at the point of anchorage by osteoclast stimulation. Respect periosteal vascularization of the manubrium may be important to prevent damage in this ossicle caused by some middle ear reconstruction prostheses.

Keywords Malleus · Vascularization · Long process · Manubrium · Handle · Malleovestibulopexy

Introduction

Schucknecht [1] published a series of 40 cases in which a prosthesis fastened to the manubrium was used to address conductive hearing loss due to damage in both incus and stapes at the same time. Posteriorly, Fish [2] supported the attachment of a prosthesis from the malleus to the inner ear when—in otosclerosis surgery—there is a fixation of the incus or this ossicle is damaged by a previous surgery. This technique, the malleostapedotomy as described by Fish—or malleovestibulopexy (MVP)—has been proved itself a useful procedure with satisfactory hearing results [3].

The functional failures of malleovestibulopexy are related to the prostheses displacement or its extrusion. Consequently, different alternatives have been suggested: enclosing the complete manubrium by a conventional prosthesis [1] or using nitinol prosthesis to better achieve the attachment [4]. Recently, some works have highlighted the biological benefits of fastening the prosthesis to the neck of the malleus, avoiding the detachment of the tympanic membrane [5].

Like other authors [6] we have noticed that in some occasions, and in a variable period of time after a MVP, the prosthesis is extruded due to erosion or breakage of the manubrium at the site where it was fastened. This circumstance was observed in those cases when intratympanic pressure was altered (primary acquired cholesteatoma) as well as those when the middle ear

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pressure was normal (stapedectomy failures due to erosion of the long process of the incus). By this reason, it is not only a negative intratympanic pressure but also other little known factors, which contribute to those undesired situations.

As well as there are disruptions of the long process of the incus due to vascular deprivation in stapes surgery, inflammatory damage induced in bone [7] or rubbing by a not well-adjusted prosthesis [8], we consider—as initial hypothesis—that similar situations might occur on the manubrium of the malleus and would be the origin of some malleovestibulopexy failures. The damage suffered by any of the middle ear ossicles when they are partial or total deprived of their vascular supply should be considered with the previous hypothesis. That vascular damage would modify bone turnover in the ossicles and promote its fracture.

The description of the ossicle's vascular supply was done on the second half of the twentieth century [9]. Some of those works emphasized the vascularization of the long process of the incus due to its surgical implications [10], but no one deeply studied the blood supply of the manubrium of the malleus.

The malleus is vascularized by two arterial branches arising from the anterior tympanic artery. The superior branch goes over the mucosal creases of the malleus head, where it enters by a *foramina nutricia*. The other branch travels in the anterior ligament to the malleus neck, where it access providing small vessels along the manubrium [11].

As demonstrated Anson, the middle ear ossicles do not have a typical haversian structure and their vascular architecture does not follow the standard distribution observed in other bones [12]. The vascularization of the middle ear ossicles derived from vessels that go through the mucosa arriving from tendons and ligaments that give support to those ossicles [13], as well as small vessels that penetrate deep into the bone from the periosteum. The outer surface of the tympanic membrane receives vascular supply from the deep auricular artery that springs from the maxillary artery. It branches into small radial vessels for the tympanic membrane and one or more descending vessels for the manubrium.

The deprivation of this vascular supply, joined to the stimulation of the osteoclastic activity triggered by the prosthesis friction, the surgical trauma and the decrease of nitric oxide production from the damaged vascular endothelium, can explain the arising of fractures in the manubrium after the use of malleovestibulopexy prostheses. By this reason, we considered the necessity of studying the vascular supply of the manubrium, to know if the vascular deprivation could play a role in the failure of some malleovestibulopexy procedures.

Materials and methods

Clinical descriptive study

Data from 10 consecutive patients who underwent a malleovestibulopexy during a 3-year period were collected. In those cases, attention was focused on the permanence of the prostheses and their position, the presence of erosion in the long process of the malleus and the endotympanic pressure.

Microscopical study

(a) Five fresh cadaveric mallei were studied using optical microscopy. The tympanic membrane was not detached from the ossicle to preserve the microvascular structure (Fig. 1). After the extraction, all the specimens were submerged in 4 % paraformaldehyde, and later decalcified with a 10 % EDTA and 40 % NaOH (9:1) solution with a pH value of 7.5. After 30 days of treatment, the samples were cleaned and embedded in paraffin. Several 7 μm sections of each sample were stained with hematoxylin–eosin and Masson trichrome before the inspection with microscope.

(b) Another different 5 mallei were studied with scanning electron microscopy. In this case, the specimens were obtained during middle ear surgery and were separated from the tympanic membranes. The samples were cleaned and submerged in a 3 % hydrogen peroxide and 0.9 % saline solution (1:1) during 10 days in order to detach the mucosal layer. After desiccant-drying at room temperature, they were introduced in the vacuum chamber of a FEI—Quanta 200FEG environmental scanning microscope for observation.

Results

Clinical descriptive study

Data of the studied patients are presented in Table 1. In five of the ten cases, the prosthesis extruded spontaneously leaving some kind of damage—erosion or complete fracture (Fig. 1)—of the long process of the malleus. In the other five patients, the prostheses are in their original surgical position, but showing different grades of erosion at the fastening site in the manubrium (Fig. 2).

In all cases, to varying degrees, an abnormal macroscopic distribution of the vascular support was observed in the route from the tympanic membrane to the handle of the

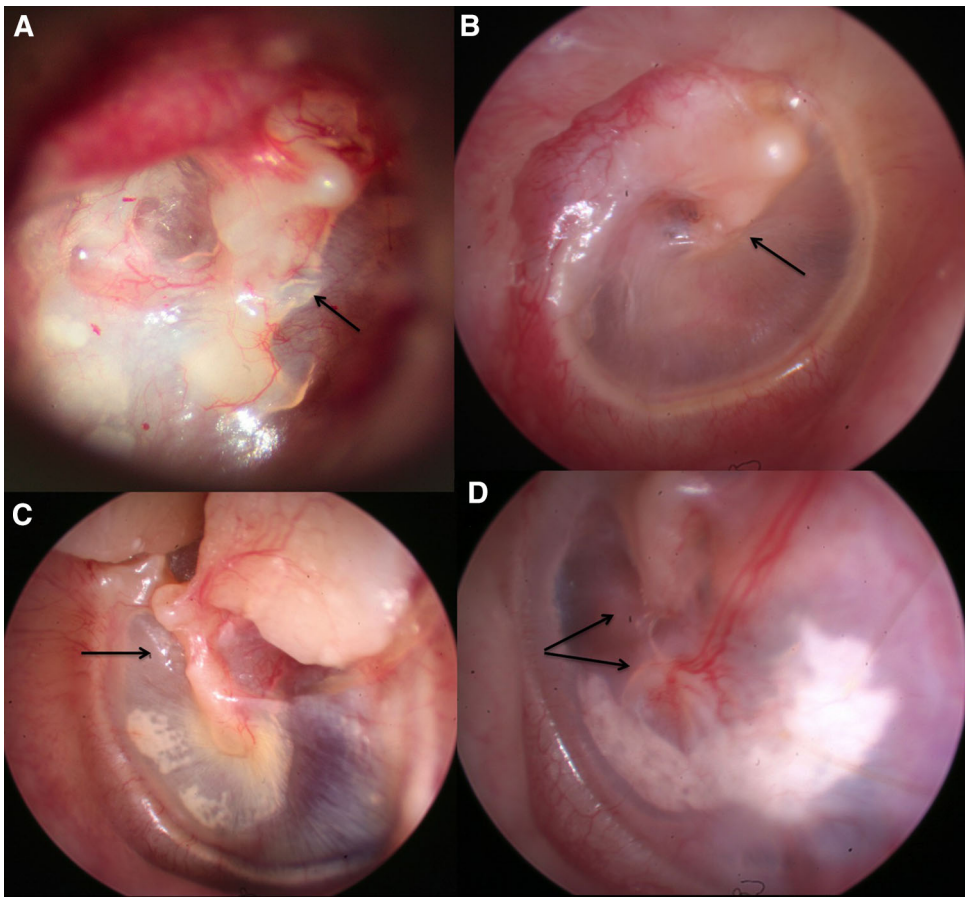

Fig. 1 Images of some of the patients where malleovestibulopexy failed: **a** patient 3. Right ear notch in the middle third of hammer handle (*arrow*). Absence of the prosthesis used. Disruption of normal vascularization that runs through the handle. **b** Patient 5. Right ear *arrow* shows the notch on the manubrium after extrusion of the prosthesis. **c** Patient 1. Left ear *arrow* points a notch in the hammer handle below its short process. Absence of prosthesis. Endotympanic pressure was negative in this patient (he underwent surgery for cholesteatoma). **d** Patient 9. Left ear the undamaged parts of the handle are indicated; between them, where prosthesis was tied, a portion of bone is missing. The vascular supply that runs to the umbo is altered

malleus. These changes include different modifications in the normal course of the vessels (Figs. 1d, 2b), even complete interruption of the vascularization (Fig. 1a).

Microscopical study

Studying the stained slices, arterioles, capillaries and venules are identified in the samples. They travel along vascular channels in the inside of the manubrium (Fig. 3) and they seem to proceed from the vascular support arising from the sustaining ligaments and muscular tendon, as well as from the periosteum of the ossicle. The vessels located in the middle tympanic layer that travel along the handle give vascular support to its interior (Fig. 4) until they reach the deeper vascular lakes, developing a complex vascular network between the surface and the deep portion of the manubrium.

The vascular foramina observed along the surface of the ossicles with scanning electron microscopy are allocated in two different sites:

In the head and the neck of the malleus, the vascular orifices are situated in the surroundings of the ligaments and tensor timpani tendon insertions (Fig. 5).

Along the handle, there is a high variability of vascular foramina. Most of them spread out the lateral—or tympanic—surface of the manubrium, while they are difficult to find in the medial aspect of the process (Fig. 6).

Discussion

Different hypotheses have been suggested to explain the damage in the ear ossicles where prosthesis is attached: by osteoclastic stimulation due to chronic irritation induced by the loop of the prosthesis [14], by post-surgical inflammatory phenomena [7] or caused by bone cell reduction at the surroundings of the anchoring points [15]. Another aspect that can be involved in the malleovestibulopexy prostheses dislocation is the negative intratympanic pressure that may induce a tympanic membrane retraction and promote the displacement or even the extrusion of the prosthesis. An important fact must be considered as well: the damaged induced to any of the middle ear ossicles when they are partial or total deprived of their vascular support. This vascular damage can change the bone turnover of the ossicle and elicit to its fracture.

Table 1 Clinical characteristics and findings of the individual patients included in the first part of the study

Patient	Age	Sex	Cause of surgery	Anchoring site	Endotympanic pressure	Type of prosthesis	Otoscopic examination	Follow-up time (months from surgery)
1	48	F	Cholesteatoma primary surgery	Superior third	Negative	Modified Cause fluoroplastic	Absence of prosthesis. Erosion of the superior third of the handle. Dehiscent jugular gulf	13
2	39	F	Revision stapes surgery	Middle third	Normal	Teflon platinum wire	Prosthesis in place. Erosion of the handle	19
3	52	F	Revision tympanoplasty	Middle third	Normal	Teflon platinum wire	Absence of prosthesis. Erosion of the middle third of the handle	8
4	56	F	Cholesteatoma primary surgery	Middle third	Normal	Teflon platinum wire	Prosthesis in place. Erosion of the handle	22
5	53	M	Revision tympanoplasty	Middle third	Normal	Modified Cause fluoroplastic	Absence of prosthesis. Erosion of the middle third of the handle	5
6	69	F	Revision stapes surgery	Middle third	Normal	Teflon platinum wire	Absence of prosthesis. Erosion of the middle third of the handle	9
7	66	F	Revision stapes surgery	Inferior third	Normal	Teflon platinum wire	Prosthesis in place. Erosion of the handle	16
8	59	F	Revision stapes surgery	Inferior third	Normal	Teflon platinum wire	Prosthesis in place. Erosion of the handle	30
9	63	F	Revision tympanoplasty	Middle third	Normal	Modified Cause fluoroplastic	Absence of prosthesis. Fracture in the middle third of the handle	16
10	56	F	Revision stapes surgery	Middle third	Normal	Teflon platinum wire	Prosthesis in place. Erosion of the handle	21

Maher [16] showed in a dog model how the anterior and posterior auricular divisions of the deep auricular artery form a vascular network in the periosteum that covers the handle of the hammer, and how branches of this plexus extend into the fibrous layer of the tympanic membrane radially. This study also demonstrates how a small posterior tympanic artery (division of the stylomastoid artery) forms a plexus on the annulus it out radially vessels leading to the manubrium. These vessels will end in an anastomosis with the radial ones coming from the periosteal plexus of the handle. This tympanic vascularization is only evident in the human under conditions that cause vasodilation (inflammation or irritation).

In the cadaveric ossicles (both by optical and electronic microscopy) we observed small branches that penetrate, from the tympanic membrane, inside the handle of the hammer. These perforating vessels are disrupted when detaching the tympanic membrane of the handle to place malleovestibulopexy prosthesis, and could alter the vascular supply of the handle portion in which the prosthesis is anchored, increasing erosion or rupture risks.

Bone consists of a metabolically active tissue that undergoes continuous remodeling processes, which give its regenerative capacity and functional adaptation. Mechanical forces that are induced on the bone are identified by osteocytes, interconnected by a canalicular system, so that bone micro-trauma induces apoptosis of osteocytes in adjacent areas, followed by resorption of the affected sites [17].

How does micro-trauma may influence normal bone turnover? Several local, paracrine and autocrine factors have been blamed influencing the process of bone remodeling. Source of those factors is located at bone cells, blood cells (such as monocytes and macrophages), endothelial cells and bone marrow cells. Both systemic and hormonal factors and bone matrix elements released during the resorption process (such as collagen fragments) are involved in its regulation [18].

The mechanism by which bone tissue detects and translates physical stimuli, and how these signals result in gene expression and biological signals are not entirely known, although the important role of osteocytes in this process has recently highlighted. In the proximity of micro fractures, the amount of osteocytes expressing apoptotic molecules (such as Bax molecular effect) increases [19], unlike what happens with osteocytes away from mechanical damage, which express anti-apoptotic molecules (Bcl-2). This author concludes that metabolic signals that guide the process of bone resorption to micro-damage positions are not only chemical signals from apoptotic cells but also signals from neighboring cells. The reduction in osteocytes/osteoclasts relation at micro fracture sites tilts the metabolic balance towards bone resorption by osteoclast activation.

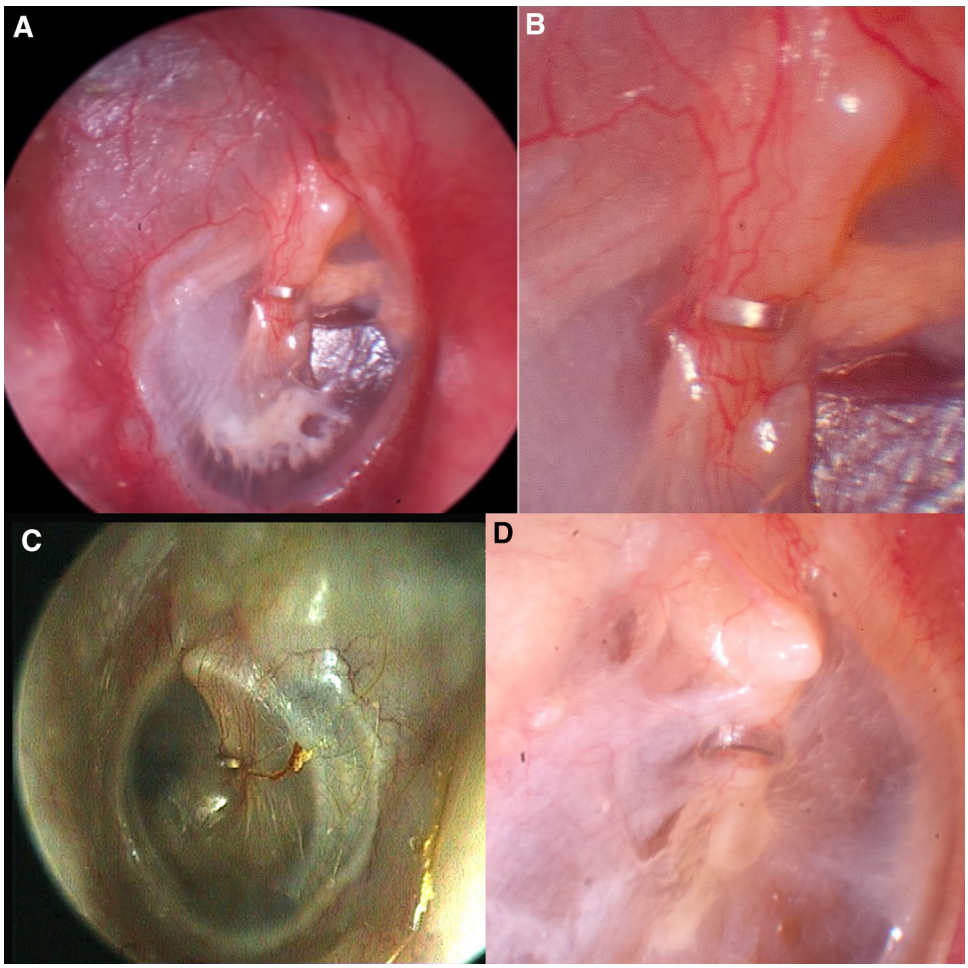

Fig. 2 Images of some of the patients in which surgery remains successful, keeping the prosthesis anchored in the handle of the hammer but with varying degrees of bone erosion. **a** Patient 4. Right ear. The metal prosthesis anchored the middle third of the handle is identified. **b** Patient 4. Right ear. Same case, with more resolution, where bone erosion by the prosthesis and vascular damage can be seen. **c** Patient 7. Left ear. Metal prosthesis anchored near the distal end of the handle. **d** Patient 10. Right ear. Metal prosthesis the middle third of the handle with a bone notch in the point of attachment

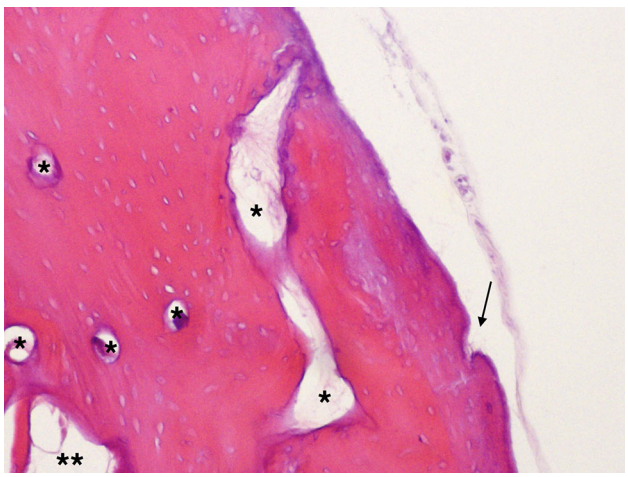

Fig. 3 Tympanic membrane observed under light microscopy (hematoxylin–eosin 10×) as a thin sheet under which the handle of the hammer is observed. The arrow points at a vessel entering the bone tissue, where capillaries in vascular channels (*) surrounded by bone lamellae can be seen heading deep vascular lacunae (**)

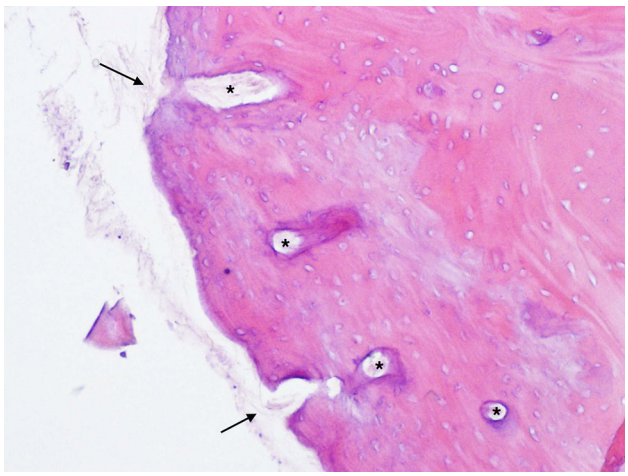

Fig. 4 Light microscopy preparation (HE 10×) where bone can be observed under a thin film of tympanic membrane. *Arrows* indicate the entrance of various vessels from the membrane into the handle. Vascular paths inside the bone are also shown (*)

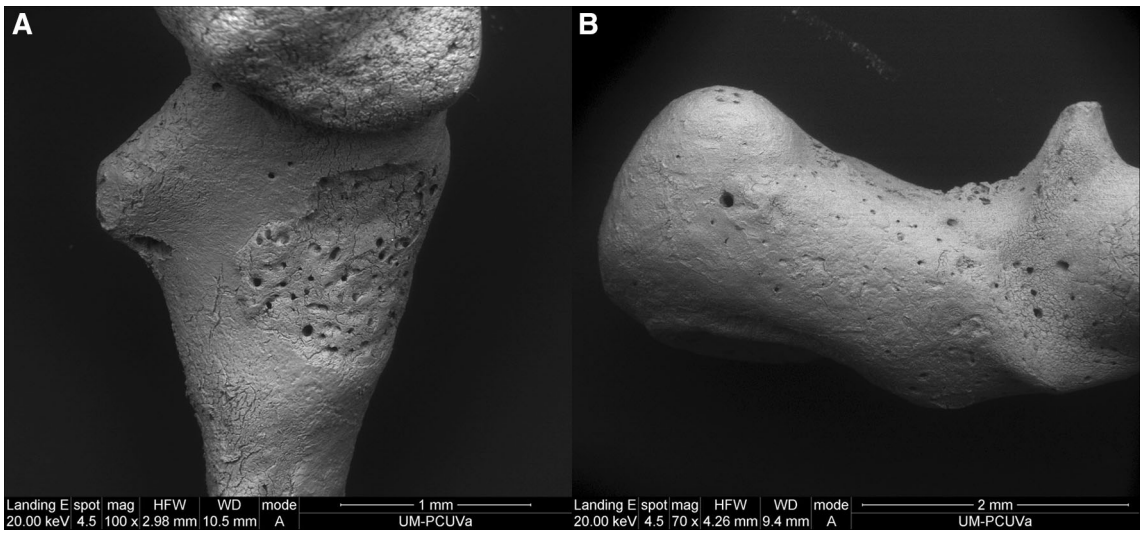

Fig. 5 Scanning electron photomicrographs corresponding to two different specimens. **a** Many vascular foramina in the insertion of the tensor tympani. **b** Vascular perforations both in the head and neck of the hammer

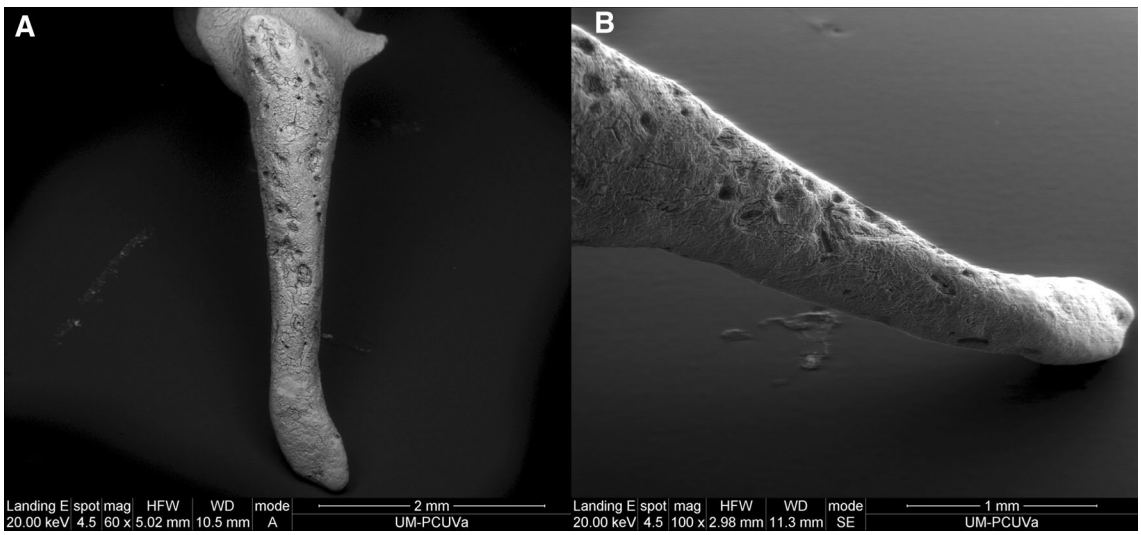

Fig. 6 Several vascular foramina observed in the tympanic face of the manubrium (**a**). Note how its frequency is reduced in the lateral portions of the handle (**b**)

On the other hand, in the micro-trauma area, the so-called multicellular Basic Osteon Units increase in number. Their function then is to eliminate the histological damage of bone tissue, promoting its resorption [20]. The osteoclastic activity is the stimuli that guide the action of those osteon units [21]. Some authors [22] have shown how osteoclasts cultured in media with healthy osteocytes barely have bone resorption ability; by contrast, osteoclasts cultured in media with apoptotic osteocytes (resulting from mechanical or thermal micro-damage) show high bone resorptive activity. Thus they concluded that healthy osteocytes prevent bone resorption.

Finally, the role of nitric oxide in the bone remodeling process has to be noted. In recent years, it has gained prominence as a regulator of bone remodeling, as it is a mediator between the anabolic effects of estrogen and the mechanical response of bone, and also affects the osteoclast inhibition [18]. A major source of nitric oxide in the blood is vascular endothelium.

Despite the small series studied, histologic changes observed in all the patients—with or without displacement of the prostheses—must have an important signification. Some of these observations are not perceived but with detailed analysis, especially with a context of clinical success. The probable multifactorial phenomenon of bone

damage at the site of the prostheses and—especially—the equilibrium of bone turn-over must have the key in the failures of the procedure, which cannot be explained with other clinical data. We think that damage induced by the prosthesis in the handle is exacerbated by the vascular deprivation. Unlike what is observed in the long process of the incus by electronic microscopy [23], where basal bone remodelation is very intense, in the manubrium a bone turnover stability is supposed. But we think that vascular damage—as well as structural modifications during the fastening—plays an important role in the histological changes observed at the site of the prostheses, inducing a poor bone tissue substitution facing a higher osteoclastic activity provoked by the micro-trauma of the prosthesis.

Malleovestibulopexy prostheses in the market have been designed to be anchored to the handle of the hammer with the subperichondrial detaching of the tympanic membrane that covers the ossicle [24–26]. The placement of these prostheses causes:

1. Surgical bone micro-damage in the hammer handle that stimulate osteoclast activity by favoring the presence of apoptotic osteoclasts in the focus damage.
2. Interruption of the vascular supply that reach the handle from the tympanic membrane; this vascular damage causes a reduction in nitric oxide production of endothelial origin inhibiting osteoblasts and stimulating osteoclast activity.

Both situations provide an explanation for the presence of bone erosions and extrusions observed in the clinical follow-up of patients who underwent a malleovestibulopexy.

For this reason, we believe that new malleovestibulopexy prosthesis designs are needed that avoid the detachment of the tympanic membrane and whose placement minimize micro-trauma induced in the handle of the hammer.

Conclusions

Displacement/extrusion of prostheses used in clinical malleovestibulopexy is due to multiple factors. We believe that the release of the tympanic membrane from the handle during the insertion favors the discharge of the prosthesis as osteoclastic bone resorption is promoted.

Design of new malleovestibulopexy prostheses for whose fastening detachment of the tympanic membrane is avoided is essential, reducing the induced damage into the handle of the hammer.

Compliance with ethical standards

Conflict of interest All the authors declare that they have no conflict of interest.

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