European Archives of Oto-Rhino-Laryngology (2019) 276:367–373
https://doi.org/10.1007/s00405-018-5246-3

OTOLOGY

A new theory interprets the development of a retraction pocket as a natural self-healing process

Karl-Bernd Hüttenbrink[1]

Received: 8 August 2018 / Accepted: 8 December 2018 / Published online: 12 December 2018
© Springer-Verlag GmbH Germany, part of Springer Nature 2018

Abstract

Purpose The thesis that cholesteatoma evolves from a retraction pocket is widely accepted today. Yet, its prime etiology, the question of what triggers the invagination of healthy skin, still remains unclear despite centuries of investigations into the origin of cholesteatoma. A new idea interprets the horizontal migration of skin into the middle ear cavities as a self-healing process, curing an underlying inflammation in the tympanic cavity, through the overgrowth and contact with immunologically active tissue.

Methods A retrospective analysis of the interrelation of retraction pockets and underlying granulation tissue was conducted in 209 second-look cholesteatoma surgeries over the last decade.

Results A stable tympanic membrane over aerated, healthy middle ear mucosa was found in 71.3% of cases. In 11%, small retractions with air in other parts of the middle ear cleft (epitympanic, sinus or anterior mesotympanum) were described. In 6.2%, granulations under a retraction were found. Only 3.8% of the reports revealed air behind a retraction or did not provide enough information on the mucosa situation behind the drum membrane.

Conclusions A new hypothesis interprets the origin of a retraction pocket—the precursor of a cholesteatoma—as a natural attempt by the body to cure an underlying inflammation in a cavity. Analogous phenomena exist, e.g. the migration of the omentum towards a local inflammation in the abdomen. This idea, which is supported by the findings in our 209 second-look surgeries, is the first explanation of the origin of retraction pockets that is compatible with the various characteristics of original or recurrent cholesteatoma. A prophylaxis against a recurrent cholesteatoma might be attained by securing free drainage of the mucosa into the tubal orifice with the use of thin silicone foils in an attempt to prevent any granulation in the middle ear cleft, similar to the principles of modern rhinosinusoidal surgery with its emphasis on unblocked mucosa clearance. This allows gas production in the healed middle ear mucosa to recover, reducing the risk of a recurrent retraction.

Keywords Retraction pocket · Cholesteatoma · Pathogenesis · Self-healing

Introduction

A wide variety of theories have been proposed over the last centuries to explain the pathomechanism of the development of acquired cholesteatoma: why keratinizing squamous epithelium starts growing from the outer layer of the tympanic membrane into the cavity of the middle ear [1–4].

The immigration theory [5], which assumes that squamous epithelium grows over the rim of a perforation, has the serious shortcoming that the majority of cholesteatomas in fact start from an intact tympanic membrane [4]. The metaplasia theory of mucosal cells transforming into keratinizing squamous epithelium cells [6] seems relevant only for the primary (genuine) cholesteatoma [7]. The papillary ingrowth hypothesis is based on the stimulus of an inflammation, e.g. embryonic tissue in the epitympanon, with active deep growth of the basal keratinocytes penetrating the basilar membrane in depth [8, 9]. In recent publications [10], this papillary ingrowth theory is discussed in combination with a retraction pocket [11]. But this theory lacks supporting evidence, as it is based on the unexplained migration of epithelial cells medially rather than laterally [3], whereas the typical concept of the self-cleaning of the tympanic membrane in the external ear canal is the horizontal migration of the

✉ Karl-Bernd Hüttenbrink
k-b.huettenbrink@uni-koeln.de

[1] Department of ORL, University Clinic Cologne, Kerpenerstr. 62, 50937 Cologne, Germany

squamous epithelium [12]. Furthermore, this theory cannot explain all aspects of the varying clinical manifestations of cholesteatoma [3].

The most widely accepted theory today is the inwards displacement of the tympanic membrane in the form of a local retraction pocket as the precursor to a cholesteatoma. The decisive proof was given by Tos in his extensive long-term cohort studies in the 1980s in which he examined 1100 children's ears over a period of 3–14 years. He was able to show that the primary pathology is the retraction pocket, in combination with a local atrophy of the tympanic membrane in the attic, the sinus, or the pars tensa, for example after a chronic secretory otitis media [13].

Yet, the conversion of a prospective into a manifest cholesteatoma, with its progression of an unsuspicious small retraction pocket into the depth, is not predictable [14]. Tos (1984) found harmless unsuspicious retractions in the pars flaccida in 34% of 527 children's ears after a chronic secretory otitis media. In a 14-year follow-up study after cholesteatoma surgery in 224 children, he observed a further invagination of retraction pockets only in 15% of patients. Only 6.3% of these cases developed further into a manifest recurrent cholesteatoma [15].

When the self-cleaning with the lateral migration of the epithelium from the depth of the retraction pocket becomes obstructed, with a decrease in the opening at the neck of the pouch, an accumulation of keratin occurs. The ensuing foreign-body reaction results in the bone-destructive growth inwards. This sequence of the "late phase" of cholesteatoma development has been examined extensively in its pathological course using histochemical, molecular, biological, and genetic approaches [1, 2, 11, 16, 17].

Yet, explanations for the "prehistoric" genesis of a cholesteatoma, why a local partition of the tympanic membrane will form a pouch and grow inwards, either in the epitympanon or in the mesotympanon, remain inconsistent and not generally accepted.

Many otologists have considered a dysfunction of the Eustachian tube with a resulting vacuum in the tympanic cleft, which pulls a weakened tympanic membrane inwards, as being the origin of the development and inward growth of a retraction pocket into depth [4, 18]. This e-vacuo theory, which also holds this mechanism responsible for epitympanic retraction [19, 20], is based on both clinical studies and the results of animal experiments, yet with contradictory results [9]. A grommet cannot prevent the development of a cholesteatoma either in animals [21] or in humans [22, 23]. Only an incomplete closure of the Eustachian tube could induce the development of a cholesteatoma in the attic via an inflammation of the middle ear mucosa [24].

The clinical tests of Eustachian tube function reveal a hypopatency only in 40% of cases [13]. Hyperpatency of the Eustachian tube, coupled with habitual sniffing [25], was not convincing either as cause, due to the low incidence of cholesteatoma development [3].

The concept of a transtubal aeration of the tympanic cavity is furthermore dubious due to the finding of gas production in the mucosa [26]. A closed Eustachian tube overnight will result in a slight overpressure, but not underpressure in the tympanic cleft in the morning [27].

Another hypothesis therefore mentioned scar tissue strands which pull the tympanic membrane inwards. This idea is based on the findings in clinical and experimental observations of an inflammation in the middle ear cleft, which was correlated to the development of a cholesteatoma [28].

A recent theory correlates the mucosal migration and the mucociliary cleaning in the tympanic cavity with the pathogenesis of cholesteatoma [3]. This hypothesis, however, was also contradicted [29, 30], as it depends on a preexisting retraction (for a contact of the undersurface of the tympanic membrane with the mucosa) and can explain only its further deepening. Furthermore, a newly developing retraction pocket in a recurrent cholesteatoma cannot be explained, due to a lack of mucosal contact with absent ossicles. The ciliary beating stagnates in an inflammation, which is the typical cofactor in cholesteatoma [31].

In conclusion, all theories presented on this evolution up till now lack a convincing explanation why the tympanic membrane should invade the middle ear. A key element seems to be the middle ear mucosa: An inflammation in the middle ear cleft is considered to be a trigger for cholesteatoma development [32] according to experimental [21, 24] and clinical [4, 8, 9, 33] experience. Meyerhoff demonstrated that an invagination of the tympanic membrane with its further development into a cholesteatoma arose after the induction of a local inflammation in the middle ear cleft, even with a concomitant insertion of a grommet [21].

Knowledge of the correlation of a local inflammation of the middle ear mucosa with the evolution of a cholestatoma might be helpful to prevent its occurrence in children. Furthermore, it can be a basis for surgical therapy after the eradication of the manifest cholesteatoma, to prevent a recurrent retraction. Until now, only the obliteration of the middle ear cleft (by eliminating persisting inflammation) seems to reduce the reappearance of a recurrent cholesteatoma [34, 35].

A new idea that has not been mentioned in the accessible literature until now as an explanation for this mysterious behaviour of the tympanic membrane is that it perhaps stems not from a pathological phenomenon, but from a biologically intended active defence mechanism. It is a biological principle that nature tries to heal an inflammation in a cavity by covering it with immunologically active tissue. In the abdomen, for example, the natural attempt to cure a local inflammation, i.e. appendicitis is seen in the active migration of the

omentum majus towards the inflammation to cover and cure the underlying disease [36, 37]. In the middle ear cleft, the tympanic membrane can migrate towards the inflammation.

As a consequence of this theory of a local healing mechanism, it could be expected that the distant regions of the middle ear would be healthy and without pathological changes. To observe this evolution in its early stage, it would be necessary to look behind the tympanic membrane, thus correlating the condition of the mucosa with the position of the tympanic membrane. Unfortunately, the feasibility of taking such a glance behind the tympanic membrane is limited by the problem of the requisite surgical opening and the fact that many healthy ears have an unsuspicious retraction pocket [22]. Perhaps a high-resolution MRI with specification of mucosal disease will enable noncontact observation in future.

A second-look control after cholesteatoma surgery, however, offers a unique opportunity to examine the condition of the mucosa behind the tympanic membrane in a relatively short time after the reconstruction of a stable tympanic membrane and tympanic cleft anatomy, and to correlate this condition with a recurrent retraction.

To test this self-healing hypothesis as the starting point of a tympanic membrane retraction, a retrospective study in a large number of cholesteatoma second-look surgeries was performed with the goal of correlating the position of the tympanic membrane with the condition of the underlying middle ear mucosa.

Methods

239 planned second-look surgeries were performed after 1291 cholesteatoma ear surgeries at the Ear, Nose and Throat Clinic of the University Hospital Cologne in the years 2005–2015. The control surgery was indicated during the first, cholesteatoma-eradication surgery, only if there was uncertainty of the complete eradication of squamous epithelial cells during the first surgery, mostly in the oval niche, the inner side of the tympanic membrane, in the hypotympanon, or on the dura. Non-EPI-DW-MRI was not applied, due to the lack of reliable experience with this radiological technique in that period and its insensitivity to thin-layer, not pearl-shaped residuals. The surgical reports of the first and the control surgery were extracted retrospectively from the patients' charts along with the postoperative results of pure tone audiometry (for information on aeration).

The reconstruction of the tympanic membrane was always performed with thin slices of cartilage and perichondrium [38], as well as the reconstruction of the posterior meatal wall. In a radical cavity procedure, the mastoid cavity was obliterated in our technique with bone dust and covered with cartilage plates [34]. The eradication surgery therefore resulted in middle ear clefts stabilized by cartilage plates and with unimpeded drainage towards the Eustachian tube.

The following data were extracted from the surgical reports of both surgeries:

Status and position of the tympanic membrane; localization of the cholesteatoma or a retraction pocket in the epitympanon, sinus tympani/pars tensa; aeration of the middle ear cleft with condition of the mucosa or mucosal inflammation in the epitympanon, the sinus tympani/pars tensa; mucous or scar tissue in the tympanic cleft; recurrent or residual cholesteatoma.

Results

Details of the data collection are reported in Tables 1 and 2.

The data for the primary (or eradication) surgery were obtained from 183 patients with 190 surgeries in whom second-look surgery was performed within 12 months. 43 ears (22.6%) had been referred from other clinics.

Surgery was performed in 83 cases (43.7%) on the right ear, in 107 cases (65.3%) on the left ear, and in 7 cases (3.7%) on both ears. For eradication, a "through the canal" or "inside-out" technique was performed in 106 ears (55.8%), a canal wall-up technique with mastoidectomy in 62 cases (32.6%), and a radical cavity with obliteration using bone dust and cartilage coverage in 22 ears (11.6%). The average age was 33.1 years; the youngest patient was 4-year-old, and the oldest 82-year-old.

An epitympanic cholesteatoma was found in 108 cases (56.8%). Of these, 88 (81.5%) presented with an air-filled mesotympanon with intact pars tensa tympanic membrane.

Table 1 Primary (eradication) cholesteatoma surgeries in 2005–2015

183 patients, 190 surgeries
Mean age 33.1 years (4–82)
Right ear 83 (43.7%)
Left ear 107 (56.3%)
Both 7 (3.7%)
108 (56.8%) cholesteatomata in the epitympanon with 88 (81.5%) air-filled tympanic cleft
113 (59.5%) cholesteatomata of the pars tensa with 60 (54.1%) air-filled anterior tympanic cavity
40 (21.1%) cholesteatomata both epi- and mesotympanic
9 (4.7%) genuine cholesteatoma
37 (19.5%) granulation tissue under the cholesteatoma
4 (2.1%) healthy, air-covered mucosa under the cholesteatoma
Surgical technique
106 (55.8%) inside-out (pursuit) technique
62 (32.6%) intact canal wall technique
22 (11.6%) radical cavity

Table 2 Second-look surgeries in 2005–2015

Within 12 months, mean 7.4 months
209 ears, 168 patients
Right ear 87 (41.6%)
Left ear 122 (58.4%)
22 (10.5%) third control surgery
6 (2.9%) fourth control surgery
1 (0.5%) fifth control surgery
1 (0.5%) sixth control surgery
Air in tympanic cavity, healthy mucosa, no retraction 149 (71.3%)
Granulation tissue under the retraction 13 (6.2%)
Epitympanic retraction with air in the tympanic cavity 12 (5.7%)
Retraction posterior pars tensa, air-filled anterior cavity 11 (5.3%)
Fluid and scar tissue in the cavity without retraction 16 (7.7%)
Manifest recurrent cholesteatoma 27 (12.9%)
Residual cholesteatoma 20 (9.5%)

In 113 cases (59.5%), a cholesteatoma was localized in the posterior pars tensa. 60 of these cases (54.1%) had an air-filled anterior mesotympanon with normal tympanic membrane. 40 cases (21.1%) had an extensive cholesteatoma in the epitympanic and in the mesotympanic space. In 9 cases (4.7%), a genuine cholesteatoma had been operated in the first surgery.

209 planned second-look surgeries in these patients were analysed. 7.4 months was the mean interval between eradication and second-look surgery.

Surgery had been performed in 87 cases (41.6%) on the right ear and in 122 cases (58.4%) on the left ear, by a total of 11 surgeons. In 22 cases (10.5%) a third, in 6 cases (2.9%) a fourth, and one-fifth and one-sixth planned second-look surgeries were documented.

The surgical reports described an air-filled cavity covered with healthy mucosa in 149 cases (71.3%). In 12 cases (5.7%) with an epitympanic retraction, an air-filled healthy mesotympanon was described. 11 cases (5.3%) with a retraction of the posterior pars tensa were accompanied by a healthy mucosa in the anterior mesotympanon. 13 ears (6.2%) presented with granulation tissue under the retraction. In 16 cases (7.7%) the mesotympanon was filled with fluid or scar tissue without a retraction. From the remaining eight revision surgeries, two surgical reports gave no significant description of the condition of the middle ear. Only in three ears (2 of them as revisions after a genuine cholesteatoma surgery with extensive bone removal of the anterior lateral attic wall) was an air-filled cavity with healthy mucosa described behind a deep retraction. Three further cases showed a shallow retraction above healthy mucosa.

27 ears (12.9%) had already developed a recurring cholesteatoma, and in 20 cases (9.5%) a residual pearl was found. The ears with recurrent cholesteatoma had shown a significant inflammation in the first (eradication) surgery in one-third of the cases (9 ears); in seven ears, the surgeon had reported an aggressive, tapestry-like growth with cholesteatosis.

Discussion

Many theories have been developed in the long history of cholesteatoma research to explain the pathogenesis of this mysterious disease. Although the most widely accepted theory today is its development from a retraction pocket [3, 13], the basic mechanism why an intact tympanic membrane deepens into the depth of the tympanic cavity at all is still unclear. This lack of convincing evidence and knowledge has given rise until today to repeated assumptions (e.g. the e-vacuo theory based on tubal dysfunction) [16] or newly presented hypotheses (mucosal migration) [3]. Yet, these ideas apply only to some aspects of the observed behaviour, but not to all characteristics of the acquired cholesteatoma, which also encompass recurrence (see Table 3). As a consequence, no generally accepted theory on the origin of a retraction pocket has yet been presented [1, 3].

A remarkable factor is that the pathogenesis of cholesteatoma is always associated with a mucosal inflammation in the middle ear spaces [4, 21, 24, 32, 33]. But what is the connection between a local inflammation in the middle ear

Table 3 Peculiarities in the clinical experience of a localized cholesteatoma

A cholesteatoma develops from a retraction pocket of the epitympanic and posterior pars tensa of the tympanic membrane
In the epitympanic cholesteatoma, the pars tensa is unsuspicious, with an air-filled cavity and healthy mucosa
In a retraction of the posterior pars tensa (sinus cholesteatoma), the anterior tympanic membrane as well as the epitympanic space are air-filled and unsuspicious
The retraction of the tympanic membrane can occur above an epitympanon/antrum/posterior pars tensa, filled with granulation tissue, which cannot develop an underpressure
A grommet will not stop the progression of an epitympanic cholesteatoma
A retraction pocket with sufficient self-cleaning, without inflammation of the skin of the tympanic membrane, can remain stable above healthy mucosa without further deepening and development of a cholesteatoma
The retraction of a recurrent cholesteatoma can develop despite a stabilization of the reconstructed tympanic membrane with cartilage

and the retraction of the tympanic membrane? Is this correlation perhaps the key to an explanation of cholesteatoma origin?

The new hypothesis presented here is intended to present a new way of looking at the retraction pocket. It challenges the former concept of a pathological development. On the contrary, this theory is based on the physiological reaction of the body when fighting inflammation in a cavity. This self-healing involves covering the inflammation with immunologically active tissue. This is a natural mechanism seen in the migration of the omentum majus onto a localized infection in the abdomen, for example towards an inflamed appendix, gall bladder, etc. [36, 37]. Surgical practice also applies this effect in the drainage of an abscess and advancement of the walls of the cavity. In the thorax and pleura cavity, a cavern and inflammation are treated by deliberate collapsing of the cavity [39].

In the middle ear with its bony walls, the tympanic membrane is the only "movable" structure. Moreover, the skin of the tympanic membrane has the remarkable ability to migrate laterally, unique in the body with the exception of the nail folds. It can be assumed that the immunologically active skin with its remarkable accumulation of Langerhans cells [40] sprouts onto the inflammation, blocks it, and defeats it.

Yet, proof of this hypothesis of a self-healing mechanism is difficult to obtain. Otologists discover a commencing, unsuspicious retraction pocket only by chance. In the manifest cholesteatoma, the inflammation with its increased molecular genetic markers, its genetic up-regulations, etc. can point to an enhanced migration [1], and thus might be seen as an attempt at self-healing. But these properties can also represent a reaction to the inflammation of the skin in this "late" stage. This is the classic "chicken and egg" problem: The question of whether the inflammation in the tissue surrounding a cholesteatoma is the origin or a consequence of the displaced skin cannot be answered with our current knowledge of molecular markers [1] in the manifest, inflamed cholesteatoma.

We therefore studied the findings in controlled second-look surgeries after a cholesteatoma eradication surgery within 12 months after reconstruction of a solid tympanic membrane to find arguments for this new self-healing hypothesis. Opening the tympanic cavity gives us the unique chance to judge the condition of the mucosa behind the early stage of a retraction of an intact tympanic membrane, before the formation of an inflamed cholesteatoma.

It is interesting to note that literature on second-look surgery has focused mainly on postoperative hearing, the rate of recidivism, etc. But there have rarely been reports, and more rarely comprehensive studies, on the condition of the mucosa behind the reconstructed tympanic membrane, with the goal of discovering pathological causes for newly developed retractions as potential precursors of a recurrent cholesteatoma. This paper therefore was intended to shed light on the hypothesis of self-healing with the help of a retrospective analysis of 209 second-look surgeries performed within 12 months after eradication surgery over the last 10 years.

The first (eradication) surgery rebuilds a generally intact middle ear by removing all pathological tissue and reconstructing a stable coverage of the tympanic cavity using the cartilage plate technique [38, 41]. However, these thin cartilage plates of 200–500 µm thickness may lose their firmness and even melt away if chronic inflammation persists.

The goal of this evaluation was to demonstrate that a new retraction would only occur onto a mucosal inflammation behind the tympanic membrane. The distant parts of the tympanic cavity would thus be covered by healthy mucosa and be air-filled. A stable, air-cushioned tympanic membrane without retraction should not be associated with an inflammation in the tympanic cavity.

This correlation could be found in 185 (88.5%) of the second-look surgeries performed after a mean of 7.4 months. The remaining ears presented in 16 cases (7.7%) a tympanic cavity filled with scar tissue and fluid without tympanic membrane retraction, which can also be considered as not contradicting inflammation as a cause for retraction. For the other eight surgeries (3.8%), two reports remained vague, without a precise description of the mucosa. In three surgeries, a shallow indentation above innocent mucosa was described, which can be interpreted as a status after healing of an inflammation of the mucosa in the first postoperative period. A deep retraction above innocent mucosa, which would oppose this hypothesis, was reported only in three revision surgeries. Two of these ears, however, had shown a huge genuine cholesteatoma in the primary event, which forced an excessive removal of the anterior attic wall. Perhaps, the reconstruction with thin cartilage plates had not remained stable enough. This interpretation is based on the unusual localization of the retraction in the anterior superior quadrant of the tympanic membrane. Only in one ear was a deep retraction of the posterior quadrant described above innocent mucosa. It is not clear retrospectively whether the heavy inflammation of the mucosa as reported in the first surgery had persisted for a prolonged period postoperatively. It might be that it was then covered by the retracted tympanic membrane and had healed at the time of the control surgery. Thus, the retraction had reached its intended goal of curing the underlying disease.

The retrospective design of this study with 11 surgeons who composed the surgical reports in the 11-year period has a deficiency, as they could not consider the relevance of the status of the mucosa under the retraction during that time. However, their descriptions were clear enough in 96.2% of the cases, and they support the assumption that at least after

a successful cholesteatoma eradication surgery without persistent mucosal inflammation, the tympanic membrane will not retract again over air-filled and healthy cavities, similar to a healthy middle ear. Instead, the stimulus of a mucosal inflammation in the middle ear seems responsible for a retraction of the tympanic membrane even in the short interval after a stable reconstruction with cartilage plates. The data from this study on the importance of an inflammation in the middle ear mucosa correspond to former experiences [9, 21, 24, 32, 33] and is now also depicted for the early state of retraction pocket development in humans with an intentional analysis of the status of the underlying mucosa. Its findings are not contradictory, but are compatible with the new self-healing hypothesis of the origin of a retraction pocket.

Therefore, if further, prospectively designed studies can confirm this theory, the pathogenetic sequence can be imagined as follows.

A persistent inflammation of the mucosa in the middle ear causes a defence reaction in the adjacent tympanic membrane. The tympanic membrane, softened in the pars tensa through disintegration of the lamina propria or directly in the pars flaccida [4], moves actively into the depth towards the inflammation by lateral migration and covers it. A healing of the inflammation by this natural defence mechanism will result in an unsuspicious and stable retraction pocket of the tympanic membrane, either hanging free or lying on the now-healthy mucosa, sometimes partly adherent.

Such an unsuspicious retraction pocket or even an adhesion of the complete tympanic membrane can remain silent lifelong. If, however, the self-cleaning mechanism of the squamous epithelium in the retraction deteriorates, with accumulation of keratin debris, a manifest cholesteatoma will develop [3, 4, 14]. Now, all the manifold components of the pathomechanism that have been described extensively [1] will commence and the inflammation will spread to the mucosa in the distant surroundings.

This new theory of self-healing as the origin of a deepening of the tympanic membrane towards a source of mucosal inflammation is compatible with all aspects of localized cholesteatoma behaviour listed in Table 3.

Retractions and consecutive cholesteatoma development are independent of the tubal function, but associated with restricted drainage and ensuing inflammation of the mucosa. Inflammations of embryonic tissue in the epitympanon result in the typical high rate of epitympanic cholesteatoma development in children [4, 8, 14]. In these cases, an obstruction of the drainage of the epitympanon anteriorly towards the tubal orifice by membranous or bony obstacles ("tensor fold") can nearly always be treated successfully by surgical removal [19, 20]. The tympanic membrane above the anterior mesotympanon and often also the hypotympanon, with their unimpeded drainage towards the tubal orifice, are usually unsuspicious, with typical air bubble and thin, healthy mucosa there [14]. Therefore, a grommet insertion has no influence on the epitympanic retraction [21–23].

A localized inflammation in the mesotympanon nearly always develops in the posterior part, in the narrow niches around the incus and the stapes [3] with their reduced mucociliary clearance [42]. This can explain the frequent retraction of the posterior tympanic membrane towards the narrow sinus tympani. A former explanation was attempted by considering a thinner lamina propria in the tympanic membrane with a weaker anulus fibrosus here and in the pars flaccida [14]. However, this is not convincing as the sole pathogenetic factor. Why should a tympanic membrane, even if weakened by a chronic infection, pull inwards? A negative pressure can only develop in a gas-filled cavity, but not if the skin rests directly on the granulation tissue [3]. It practically never bulges outwards, as it would be expected above a subcutaneous inflammation in other regions of the body.

If prospective studies can confirm this new hypothesis, the accidental discovery of an unsuspicious retraction pocket of the tympanic membrane in an otherwise healthy middle ear can be interpreted as a sign of the successful self-healing of a former local mucosal inflammation in the middle ear and not as an active pathologic disease. An external circumstance, e.g. water filling or keratin accumulation, could however induce a progression into a manifest cholesteatoma [14]. In this early stage of cholesteatoma development, a stabilization surgery, for example with cartilage plate underlay, may be adequate [41].

In eradication surgery for cholesteatoma, a renewed blockage of the drainage of the middle ear mucosa should be prevented by covering the mucosa with thin silicone sheeting. Rhinosurgery already discovered the exceptional importance of unimpeded drainage of mucosa-lined cavities for preventing recurring inflammation several decades ago, resulting in a radical change in surgical techniques [43]. The success of drainage of the nasal sinus through endonasal procedures has replaced the former external techniques of frontal or maxillary sinus surgery nearly completely. In middle ear surgery, this new way of looking at the pathomechanics of local inflammation might foster the approach with less destruction of principally healthy tissue in the mastoid, but focusing on establishing a stable drainage in the middle ear cleft towards the tubal orifice. Another idea is that intensive antibiotic therapy as already established in rhinosurgery for re-establishing drainage in operated sinus orifices [44] may perhaps ward off local inflammation of the middle ear mucosa [45] and thus counteract the progression of a recurrent retraction, with the goal of preventing a recurrent cholesteatoma. This will be evaluated in future prospective studies.

Funding No support or funding was received for this work.

References

1. Kuo CL (2014) Etiopathogenesis of acquired cholesteatoma: prominent theories and recent advances in biomolecular research. Laryngoscope 125:234–240
2. Soldati D, Mudry A (2001) Knowledge about cholesteatoma, from the first description to the modern histopathology. Otol Neurotol 22:723–730
3. Jackler RK, Santa Maria PL, Varsak YK, Nguyen A, Blevins NH (2015) A new theory on the pathogenesis of acquired cholesteatoma: mucosal traction. Laryngoscope 125(Suppl. 4):1–14
4. Tos M (1981) Upon the relationship between secretory otitis in childhood and chronic otitis and its sequelae in adults. J Laryngol Otol 95:1011–1022
5. Habermann J (1898) Zur Entstehung des Cholesteatoms des Mittelohres. Arch Ohrenheilkunde 27:42–50
6. Gruber J (1888) Das Cholesteatom (Perlgeschwulst). Lehrbuch der Ohrenheilkunde. Berlin. C. Geralds Sohn
7. Michaels L (1986) An epidermoid formation in the developing middle ear: possible source of cholesteatoma. J Otolaryngol 15:169–174
8. Escher F (1979) Die Therapie des Mittelohrcholesteatoms. HNO 27:145–149
9. Steinbach E, Pusalkar A, Heumann H (1988) Cholesteatoma—pathology and treatment. Adv Oto-Rhino-Laryngol 39:94–106
10. Karmody CS, Northrop C (2011) The pathogenesis of acquired cholesteatoma of the human middle ear: support for the migration hypothesis. Otol Neurotol 33:42–47
11. Sudhoff H, Tos M (2007) Pathogenesis of sinus cholesteatoma. Eur Arch Oto-Rhino-Laryngol 264:137–143
12. Deong KK, Prepageran N, Raman R (2006) Epithelial migration of the postmyringoplasty tympanic membrane. Otol Neurotol 27:855–858
13. Tos M (1988) Incidence, etiology and pathogenesis of cholesteatoma in children. Adv Oto-Rhino-Laryngol 40:110–117
14. Wayoff M, Charachon R, Roulleau P, Lacher G, Deguine C (1987) Surgical treatment of middle ear cholesteatoma. In: Advances in Oto-Rhino-Laryngology, vol 36. Karger, Basel
15. Tos M, Lau T (1988) Attic cholesteatoma. Recurrence rate related to observation time. Am J Otol 9:456–464
16. Louw L (2010) Acquired cholesteatoma pathogenesis: stepwise explanations. J Laryngol Otol 124:587–593
17. Hüttenbrink KB (1984) Die chronische Otitis media. In: Nauman H, Helms J, Herberhold C, Kastenbauer E (eds) Oto Rhino Laryngologie in Klinik und Praxis, Band 1 Ohr, G. Thieme, Stuttgart, pp 601–632
18. Bezold F (1891) Über das Cholesteatom des Mittelohres. Z Ohrenheilkunde. 21:252–271
19. Marchioni D, Alicandri-Ciufelli M, Molteni G, Artioli FL, Genovese E, Presutti L (2010) Selective epitympanic dysventilation syndrome. Laryngoscope 120:1028–1033
20. Morimitsu T, Nagay T, Nagay M, Ide M, Makino K, Tono T (1989) Long term results of anterior tympanotomy for cholesteatoma. In: Tos M, Thomssen J, Peitersen E (eds) Cholesteatoma and mastoid surgery. Kugler, Amsterdam, pp 875–880
21. Meyerhoff WL, Wright CG, Gerken GM (1990) Effects of middle ear ventilation on cholesteatoma development in experimental animals. Acta Otolaryngol 110:279–285
22. Tos M, Stangerup SE, Holm-Jensen S, Sørensen CH (1984) Spontaneous course of secretory otitis and changes of the eardrum. Arch Otolaryngol 110:281–289
23. Spilsbury K, Ha JF, Semmens JB, Lannigan F (2013) Cholesteatoma in cleft lip and palate: a populations-based follow-up study of children after ventilation tubes. Laryngoscope 123:2024–2029
24. Heumann A, Steinbach E, Koneberg F (1981) Untersuchung zur Cholesteatomentstehung durch Verschluss der Tube bei Kaninchen. Arch Oto-Rhino-Laryngol 231:813–815
25. Buckingham RA (1988) Patent eustachian tube in the underaerated middle ear: a paradox. Ann Otol Rhinol Laryngol 97:219–221
26. Sadé J, Luntz M, Levy D (1995) Middle ear gas composition and middle ear aeration. Ann Otol Rhinol Laryngol 104:369–373
27. Gaihede M, Dirckx JJ, Jacobsen H, Aernouts J, Sovso M, Tveteras K (2010) Middle ear pressure regulation-complementary active actions of the mastoid and the Eustachian tube. Otol Neurotol 31:603–611
28. Wullstein HL, Wullstein SR (1980) Cholesteatoma. Etiology, nosology and tympanoplasty. J Otorhinolaryngol 42:313–335
29. Chole RA, Lim DJ (2015) In reference to a new theory on the pathogenesis of acquired cholesteatoma: mucosal traction. Laryngoscope 126:E129–E130
30. Kuo CL (2016) Letter to the editor in reference to a new theory on the pathogenesis of acquired cholesteatoma: mucosal traction. Laryngoscope 126:E132
31. Yeger H, Minaker E, Charles D, Rubin A, Sturgess JM (1988) Abnormalities of cilia in the middle ear in chronic otitis media. Ann Otol 97:180–191
32. Schwartz M (1984) Die genetischen Faktoren des Mittelohrcholesteatoms. HNO 32:59–60
33. Ruedi L (1978) Pathogenesis and surgical treatment of the middle ear cholesteatoma. Acta Otolaryngol 361(Suppl):1–45
34. Beutner D, Helmstaedter V, Stumpf R, Beleites D, Zahnert T, Lüers JC, Hüttenbrink KB (2010) Impact of partial mastoid obliteration on caloric vestibular function in canal wall down mastoidectomy. Otol Neurotol 31:1399–1403
35. Vercruysse JP, van Dinther JJ, De Foer B, Casselman J, Somers T, Cremers CC, Offeciers E (2016) Long-term results of troublesome CWD cavity reconstruction by mastoid and epitympanic bony obliteration (CWR-BOT) in adults. Otol Neurotol 37:698–703
36. Kaufmann E, Staemmler M (1956) Lehrbuch der speziellen pathologischen Anatomie. Band 1; 1. Hälfte. De Gruiter, Berlin
37. Rost F (1925) Pathologische Physiologie des Chirurgen. F.C.W. Vogel, Leipzig 3. Aufl. 1925
38. Beutner D, Hüttenbrink KB, Stumpf R, Beleites D, Zahnert T, Lüers JC, Helmstaedter V (2010) Cartilage plate tympanoplasty. Otol Neurotol 31:105–110
39. Shields TW (2009) General thoracic surgery, 7th edn. Lippincott Williams & Wilkins, Philadelphia
40. Veldman JE (1985) The Langerhans' T cell microenvironment in aural cholesteatoma. Kugler, Amsterdam, pp 69–79
41. Hüttenbrink KB (2018) Eingriffe am Ohr. In: Rettinger G, Werner JA, Hosemann W, Hüttenbrink KB (eds) HNO-Operationslehre. Thieme, Stuttgart
42. Shimada T, Lim DJ (1972) Distribution of ciliated cells in the human middle ear. Electron and light microscopic observations. Ann Otol-Rhinol-Laryngol 81:203–211
43. Lund V (2002) The evolution of surgery on the maxillary sinus for chronic rhinosinusitis. Laryngoscope 112:415–419
44. Elliott KA, Stringer SP (2006) Evidence based recommendations for antimicrobial nasal washes in chronic rhinosinusitis. Am J Rhinol 20:1–6
45. Takahashi H (2012) The middle ear. Springer, Berlin