

# Assessment and management of retraction pockets

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## ABSTRACT:

This manuscript intends to review types, pathogenesis, associated risk factors, and potential methods of prevention and treatment of the retraction pockets in adults and children. The importance of retraction pockets (RP) lies in loss of original histological and anatomical structure which is associated with development of ossicular chain erosion, cholesteatoma formation and potentially life threatening complications of cholesteatoma. The trans-mucosal exchange each gas in the middle ear (ME) is towards equalizing its partial pressures with the partial pressure in the environment. MEs that have abnormalities in the volume and ventilation pathways in the epitympanic may be more susceptible to retraction pockets. Sustained pressure differences and/or inflammation leads to destruction of collagen fibers in the lamina propria. Inflammatory mediators and cytokines lead to release of collagenases result in viscoelastic properties of the lamina propria. The process of changes in the tympanic membrane structure may evolve to the cholesteatoma formation. There are many different staging systems that clinicians prioritize in their decision making in the management of RP. The authors discuss the management possibilities in different clinical situations: RP without and with ongoing or intermittent evidence of Eustachian Tube Dysfunction (ETD), presence of adenoid hypertrophy or re-growth of adenoids, presence or absence of effusion, invisible depth of RP without effusion, invisible depth of RP with effusion, ongoing RP after VT insertion, and finally suspicion of cholesteatoma in a deep RP with ME effusion. A decision algorithm regarding the management of TM retraction and retraction pockets is provided.

## KEYWORDS:

Retraction pocket, middle ear, tympanic membrane, Eustachian tube

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- Abbreviation list:
- TM - Tympanic membrane

- ME – Middle ear
- ET - Eustachian tube
- MEP – Middle ear pressure
- MEPR - Abnormal middle ear pressure regulation
- MACS – Mastoid air cell system
- ME-MACS – Middle ear and mastoid air cell system
- ETD – Eustachian Tube dysfunction
- OM – otitis media
- ETF - Eustachian Tube function
- VT – Ventilation tube
- RCT - Randomized controlled clinical trial
- RP – Retraction Pocket
- PCNA - Proliferating Cell Nuclear Antigen

## INTRODUCTION

Tympanic membrane (TM) separates the middle ear (ME) and the external ear canal. There are a number of functions and effects of the TM. This separation protects the ME from entry of the foreign material, cerumen, water and pathogens. In addition, by creating a ME air cushion, it reduces the risk of secretions and pathogens being refluxed through the Eustachian tube (ET) from the nasopharynx into the ME. It has an important function of having a surface that vibrates with the sound waves reaching into the ear canal and transmitting this vibration to the manubrium of malleus. In addition, having a much larger surface area compared to the oval window, the vibration is proportionally enhanced. Also, by prevents the sound waves travelling into the ear canal from reaching the round window at the same time that they reach the oval window, therefore, prevents the potential sound wave cancelling in the inner ear and that would otherwise cause a decrease in the hearing. The TM also displaces in and out changing the ME volume, therefore, reducing the pressure fluctuations from either changes in the environmental pressure or changes in the MEP from increase or decrease in the ME gas volume. Lastly, in the presence of a ME infection, increased pressure from the purulent effusion is dampened by the fluctuant nature of the TM.

Histologically, TM consists of an outer keratinizing squamous epithelial layer, an inner cuboidal mucosal layer, and a middle fibrous connective tissue layer between the tympanic annulus scutum at the notch of Rivinius and the manubrium of malleus. In a normal TM, in its pars tensa, lamina propria is made of densely packed collagen fibers. Fibers are highly organized in outer radial and inner circular layer. Between fibers there is a considerable volume of extracellular matrix. In pars flaccida, lamina propria consists of loose connective tissue.

In a normal physiological status, when the ME pressure (MEP) is equal to the environment, the TM has a very slight

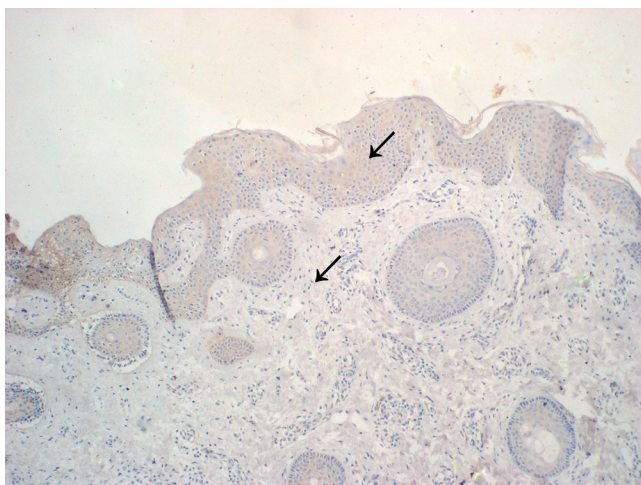
convexity between the manubrium and the annulus, with the cross sectional shape of a seagull appearance. When there is an increased MEP compared to the environment, the TM becomes more convex. When there is negative MEP, first the convexity disappears, and TM becomes more flat, and then, the TM is stretched in, creating a concave TM surface. This condition is called TM retraction. If part of the TM is stretched in more than the rest of the TM, forming a distinct second (or multiple) concavity, this segment of TM is named as a retraction pocket. Development of retraction pocket presumes a change in the histological structure of the TM, typically, loosening or loss of the elastic fibers in the middle layer, and reduction of thickness and tensile strength and its ability to maintain the shape of the TM or ability to return to its original thickness, strength or position of the TM. Retraction pockets are considered sequelae of the abnormal MEP regulation (MEPR), however, inflammation or earlier perforations may contribute to the development of retraction pockets.

The importance of retraction pockets lies in loss of original histological and anatomical structure, therefore inability to sustain the functions outlined above. More importantly, this sequelae is associated with development of other sequelae and complications, including ossicular chain erosion and cholesteatoma formation, and serious and potentially life threatening complications result from cholesteatoma.

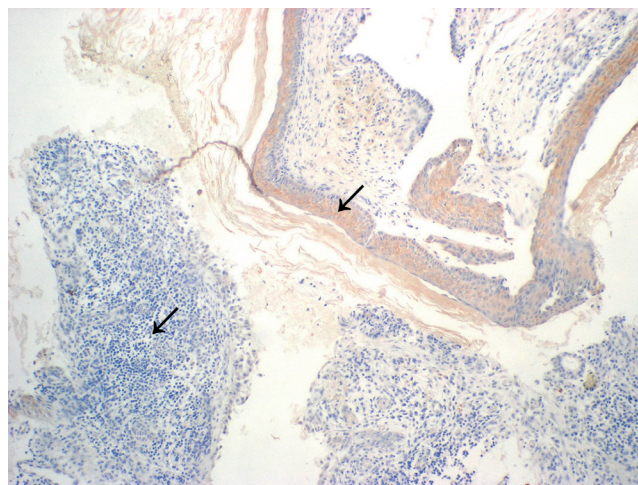
This manuscript intends to review the types, pathogenesis, associated risk factors, and potential methods of prevention and treatment of the retraction pockets in adults and children. Although there are no anatomical or histological differences between the retraction pockets in adults and children, focusing on the retraction pockets in the pediatric population have several advantages. First of all, retraction pockets are in much more mature and stable form in adults, such as formation of stable ME atelectasis or adhesive otitis. On the other hand, early phases of the retraction pocket formation and gradual worsening of the retraction pocket and evolution into pre-cholesteatoma and cholesteatoma are much more likely to be observed and noticed in children. Therefore, although most of the content of this manuscript is applicable to the adults as well, the main emphasis will be on the pediatric retraction pockets.

## MIDDLE EAR PRESSURE REGULATION AND THE TYMPANIC MEMBRANE

Tympanic membrane has a natural elasticity allowing its position and shape to change with the variation of differences



**Fig. 1.** Matrix metalloproteinase-9 (MMP-9) is shown in perimatrix of cholesteatoma (strong immunoreactivity) and within matrix (weak reactivity). MMP-9 positive cells are marked with arrows. Labeled streptavidin peroxidase method. Magnification x 200.



**Fig. 2.** Matrix and subepithelial connective tissue of retroauricular skin layers show weak positive reaction with MMP-9 (arrows). Magnification x 200.

between the middle ear (ME) and the ambient pressures. This change, in fact, results in change in the ME and mastoid air cell system (ME-MACS) volume. Outward and inward displacement of the TM results in an increase and a decrease in the ME-MACS volume, which as a result, decreases the pressure difference between ME and the ambient [1, 2]. This dampening effect is greater, if the MACS volume is smaller. Same amount of TM displacement would result in far less MEP change in a well pneumatized MACS [3].

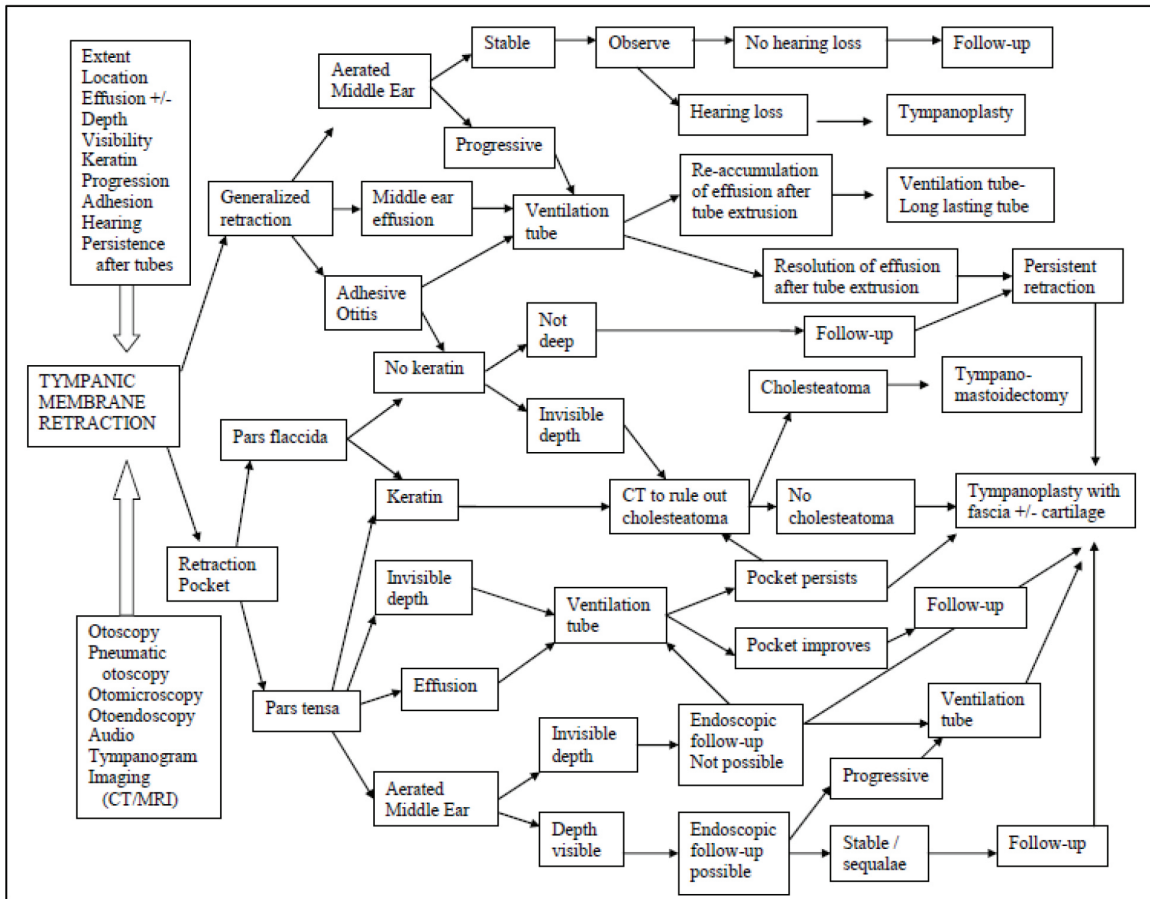
Normal TM has a certain capacity to buffer the pressure changes [4]. While in and out displacement of pars flaccida may result in 50 microliter volume change, pars tensa displacement may result in 200-400microliters. When there is a pressure gradient that exceeds the TM's buffer capacity, a negative or positive MEP develops. Typically, the increased degree of pressure difference is corrected by passive or active opening of the ET, resulting in partial or total correction of this pressure difference.

Although changes in the environmental pressures due to flying or passing through high elevations can result in positive MEP, the physiologic trend of MEP change is towards developing negative pressure. The trans-mucosal exchange each gas in the ME is towards equalizing its partial pressures with the partial pressure in the environment [5]. Since sum or partial pressure of gasses in the ME is greater than the air, the gradual equilibration results in negative MEP. If the ET does not open, the end result of MEP equilibration would be approximately -600daPa.[6] This degree of negative MEP is not sustainable. The negative MEP decreasing below -200 to

-400daPa results in transudation and exudation of fluids into the ME. Moreover, being subjected to prolonged significant negative MEP, TM elastic fibers start weakening, resulting in higher degree of displacement. This continued or recurred MEP difference from environment will only create a vicious circle, making the increased elasticity and loosening of fibers result in further decrease in thickness and strength of the TM and progression and deepening of the retraction pocket. Middle ears that have abnormalities in the volume and ventilation pathways in the epitympanic may be more susceptible to retraction pockets.[7-9]

## ASSESSMENT OF EUSTACHIAN TUBE FUNCTION

Traditionally Eustachian Tube dysfunction (ETD) is diagnosed by history and physical examination, mostly with the assumption that ETD is the underlying problem when there is otitis media (OM), ME effusion, TM retraction, retraction pocket, cholesteatoma, ear pain when there is no infection, barotrauma with flying or diving.[10-12] Otoscopy shows the presumed clinical consequences or tympanogram may show negative pressure but these don't measure the ETF, or validate the ETD. The recently developed ETDQ-7 questionnaire is non-specific.[13] The ETS-7, a scoring system is developed by assigning a score for ETF, combining physical exam findings, ability to do Valsalva and normal Tubomanometry results.[14] The Tubomanometer, essential for the ETS-7 scoring system, is used only in few clinical research centers, therefore, ETS-7 is not practically useful. There



**Fig. 3.** Example of decision algorithm regarding the management of tympanic membrane retraction and retraction pockets. Note that this is only one decision tree, based on a set of important variables and priorities, and is not applicable to all clinical presentations. A combination of variables in a presentation changes the order of priorities and result in a different algorithm. It is not possible to have a single set of decision tree applicable to all variables. This manuscript therefore attempts to dissect the variables and discusses their degree of importance in various combinations of other variables.

are more sophisticated ET function testing methods, such as sonotubometry, inflation-deflation test, forced response test, pressure chamber tests, but they are available in only few research centers.[15] Technically Bluestone's 9-step test is not sophisticated, needs only a tympanometer and an audiologist to spend time to get the results. Practically, this test is not in routine use even in the center that created this test, because of concerns in its accuracy.

Many tympanometer brands have built-in software to test the Eustachian Tube function (ETF). Every otolaryngology department has access to a tympanometer and an audiologist. However, it is rare that ETF test is requested in addition to the audiometric and tympanometric testing. The tympanometry machines come with manuals that describe

the test options, including the ETF test. This testing is done with the option of non-intact and intact TM settings, and essentially, the tympanometer gradually increases the external ear canal pressure with the sealed ear probe in place, and it is expected that the ET passively opens at a specific pressure level. Opening pressures of 150-300daPa may be considered within normal range. Right after this passive opening tympanometer software stops increasing the pressure and pressure is expected to stabilize at that or a little lower level. While the probe continues to be sealed in the ear canal, the patient is than expected to swallow, and in a patient with normal ETF, the pressure is expected to drop with most swallows. If one single swallow drops the pressure difference more than 50%, or in 5 consecutive swallows, of >80% of the pressure difference is corrected, it would be safe

to say that this patient has pretty normal ETF. If the pressure that ET opens is less than 50daPa, the ET may be patulous or semi-patulous. If the ET opens <20daPa, it is likely patulous. Whether or not the patient hears his or her own voice or breathing should be questioned. When semi-patulous or patulous ET is suspected, it is recommended that TM is observed with otoscope, microscope or otoendoscope to see if the ET moves in and out synchronous with breathing. To manifest this more clearly, patient should be asked to pinch one nostril and breath more deeply and forcefully.

A similar simple method, if the tympanometer does not have built-in software to test, is to have the ear canal pressure manually decreased to -200 daPa, which makes the MEP relatively higher at +200daPa, and then ask the patient to swallow, to see how much the patient would correct in 1 and 5 swallows. This test is then to be repeated with the ear canal pressure is manually increased to +200daPa, making the MEP -200 daPa, which is relatively more difficult for the patient to correct. If a patient is able to correct both the positive and negative MEP's with swallows, it is reasonable to assume that ETF is normal.

One other simple method, that requires also just a tympanometer is to measure the baseline MEP, than ask the patient to perform a Valsalva, and measure again to see if the MEP has increased. If properly done, and if the patient is able to generate significant pressure, the ET should open. If the ET does not open despite proper Valsalva maneuver, there may be ETD. If successful, then the patient is asked to swallow and correct the MEP difference. Achievement of similar degree of correction with 1 and/or 5 swallows should indicate normal ETF.

It is important to be cautious in calling an ear having ETD. Assigning the label of normal ETF, if these test results are normal, it is easier, however, abnormal test results are not always accurate in showing ETD. Especially if the ear has a normal TM, normal MEP, any abnormal test results should not assign abnormal ETF to that ear. Opening of the ET periodically is essential in maintaining an aerated ME within normal MEP range. Theoretically or practically, it is not possible to have an aerated ME, if the ET does not open at least with some frequency. On the other hand, ETF is not considered a static uniform ability to open and equilibrate the pressure differences between the ME and the environment. This ability is variable, which is affected by the body position, altitude, rate of changes in environmental pressure, illnesses and conditions that affect the rate and efficiency of ET opening, such as viral upper respiratory tract infections, sinusitis, allergy exacerbations, driving up and down

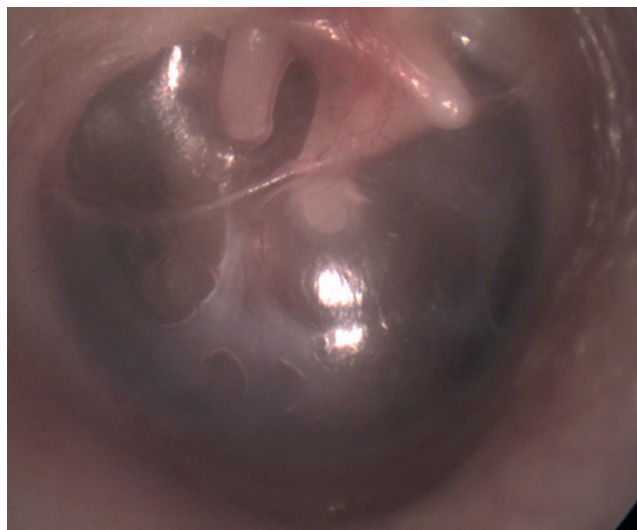


Fig. 4. An aerated middle ear and a stable posterior-superior retraction pocket.



Fig. 5. Very thin TM as the sequelae of previous ETD may form a retraction pocket even without significant or persistent negative ME pressure.

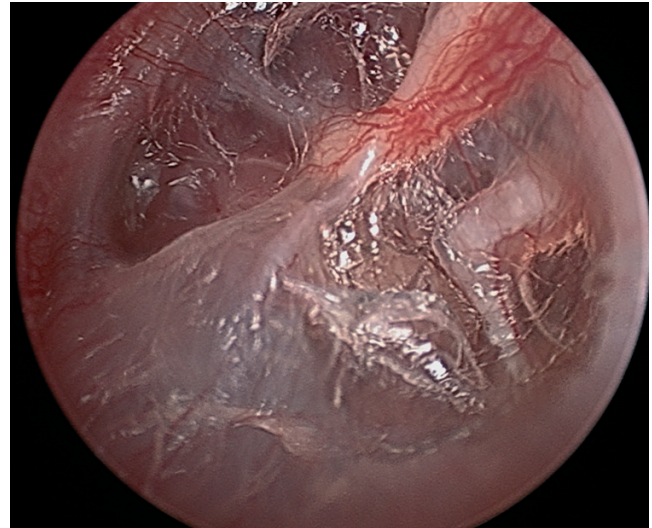
high altitudes, flying or diving. This may be confusing to the patient and the clinician, because at the time of assessment MEP, ear findings and test results may be perfectly normal, however, when challenged with any of these conditions, when the ET opening efficiency drops, the supply of air into the ME would decrease temporarily, or even seasonally, like during an allergy season, and unless the patient is assessed and tested under those circumstances, it would not be possible to document an abnormal ETF.

## INFLAMMATION AND RETRACTION POCKET FORMATION

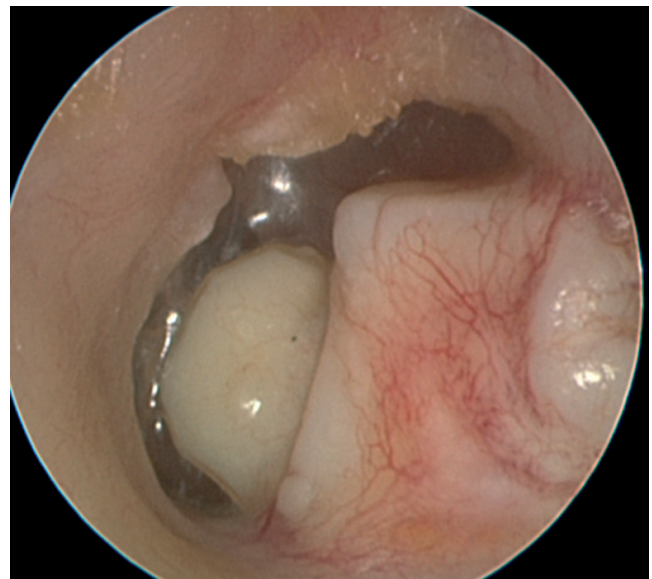
Inflammation affects the TM structure in several mechanisms. Simple mechanism is the effect of positive pressure in the ME in the condition of acute (OM). Even if it does not result in a perforation, this bulging does affect the TM, similar to the negative pressure retracting the TM. Inflammation of the ME is not limited to an acute infection, and otitis media with effusion, without any signs or symptoms of an infection is also a risk factor. Middle ear effusion often has silent bacteria and definitely is very rich in inflammatory mediators and cytokines.

There may be a pressure difference between the mesotympanum and the epitympanum, resulting in higher pressure differences that TM needs to withstand in the pars flaccida and posterior superior quadrant, due to the folds and membranes within the ME, limiting the communication between the mesotympanum and the epitympanum, possibly blocking the anterior and posterior isthmus around the ossicles, especially in the presence of inflammation and edema. This could result in more thinning in these locations compared to the rest of the pars tensa. Moreover, any perforation, if that were to happen, would weaken the TM further. Similar mechanism of pressure differential subjects the posterior superior quadrant to higher risk for perforation, and resulting weakening, and proneness to retraction pocket formation. The other mechanism is the damage on the tissues from the inflammatory cytokines and mediators. While these factors may result in tympanosclerosis, the stiffened TM that reduces the risk of retraction in these locations, it also can result in loss of elastic fibers in the middle layer of TM.

Another risk factor is trauma to the TM, from a traumatic perforation, tympanocentesis, myringotomy or ventilation tube (VT) insertion. Long term tubes are associated more with the atrophic TMs and retraction pockets, but this does not mean that long term tubes directly cause the atrophy or retraction pocket. It is true that long term tubes increase the

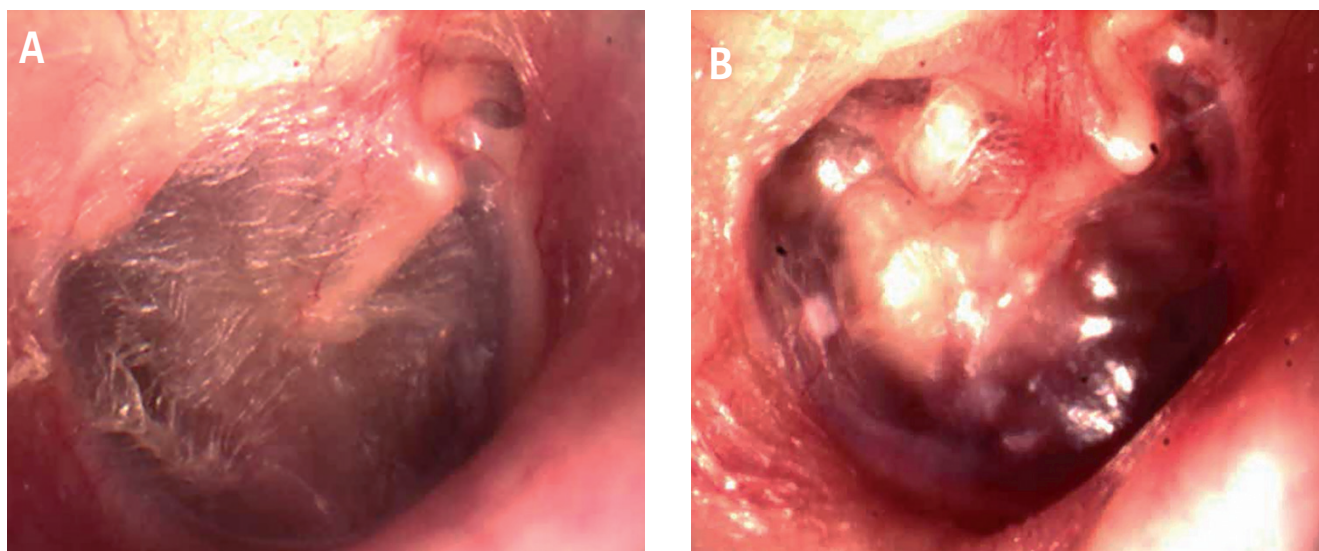


**Fig. 6.** Very thin segments of the TM may stay in neutral position. However even a minor reduction of ME pressure can manifest itself as collapse of this atrophic TM.



**Fig. 7.** If there is ongoing ETD, parts of the TM that is not supported by cartilage may develop retraction pocket.

risk of perforation, and may indirectly increase the risk of atrophy also, however the association between the long term VT and the atrophy and retraction pocket is mostly because of the ear with such conditions needing long term tubes, first, due to anticipated longer duration of help in ventilation of the ME, second due to atrophy, reduced duration of regular tube retention. Tubes are placed to prevent progression of TM retraction. However VTs can cause retraction pockets



**Fig. 8.** Very thin TM can be seen in a) neutral position or b) retracted and atelectatic state when there is a barochallenge, or with worsened ETF during colds of allergy exacerbations

or cholesteatoma. Atrophy or retraction risk is 25.5% for the short term VTs versus 23.3% for long term tubes, and cholesteatoma risk is 0.8% and 1.4%, and chronic perforation risk is 2.2% vs. 16% respectively.[16]

The main risk factors for the TM atrophy, retraction and retraction pockets are the factors that cause ETD. These include Down's and other syndromes, craniofacial abnormalities, some indigenous races, cleft palate, sub-mucous cleft palate, conditions that cause nasal obstruction, adenoid hypertrophy, adenoiditis, nasal allergies, recurrent or chronic sinusitis, gastroesophageal reflux disease. One other important risk factor is sniffing habit. Patulous ET is another risk factor. Continuous inward and outward movement of TM synchronous with breathing by itself is a risk factor to thin the TM. Especially, if this is more forceful, the nasopharyngeal pressure variations would directly be transmitted to the ME and the TM. Sniffing habit is also a risk factor, especially if the ET is patulous or semi-patulous, the sniffing would pull the retraction pocket further and further in.

## **PATHOGENESIS OF TYMPANIC MEMBRANE RETRACTION POCKET**

Atrophy of the lamina propria never happens suddenly or in isolation. Sustained pressure differences and/or inflammation leads to destruction of collagen fibers in the lamina propria. Inflammatory mediators and cytokines lead to release of collagenases and the rupture of the disulfate bridges. As a re-

sult, changes occur in the viscoelastic properties of the lamina propria. Through this process generalized or localized TM thinning develop.

Thinned TM loses its resistance to pressure changes. TM can move in and out much more, and can buffer larger pressure changes. However it becomes easier for the TM without its original strength to lose more fibers and get thinner. After the initial insult and loss of strength, inflammation is no longer necessary for progression of the TM retraction. Similarly, significant pressure differences between ME and the environment is not necessary either. Even relatively small pressure differences can displace the TM to its limits, and stretch more and lead to progression or retraction pocket.

The process of changes in the TM structure may evolve to the cholesteatoma formation.[17-25] Pars tensa retraction is more frequently associated with ossicular erosions, especially the long process of incus while pars flaccida (attic) retraction predisposes to acquired cholesteatomas. The keratinocyte migration within layers of cholesteatoma epithelium, keratinocytes proliferation and angiogenesis within matrix and perimatrix of cholesteatoma are involved in the development of cholesteatoma.[17, 18, 21] Proliferation theory assumes that the ability of endothelial cells of cholesteatoma to proliferate depends on ongoing inflammation that occurs below the epithelium. Molecular markers of epidermal hyperproliferation, such as interleukin 1 (IL-1), transforming growth factor  $\alpha$  (TGF- $\alpha$ ), epidermal growth factor receptor (EGFR), nuclear antigen of cellular proliferation

(PCNA), Ki-67 antigen and cytokeratin are used to explain the specific behavior of keratinocytes in cholesteatoma. The immune response is critical for the primary defense against middle ear infections. The DNA-binding high-mobility box 1 (HMGB1) which plays a crucial role in molecular responses to cell damage/necrosis via activation of the nuclear factor kappa B transcription factor and subsequent induction of pro-inflammatory cytokines, IL-11, TNF- $\alpha$ , is released extracellularly in response to stress or necrosis and behaves as a cytokine, promoting inflammation. It is possible that keratinocyte proliferation is promoted by HMGB1 via its interaction.[23]. The process of keratinocyte proliferation within cholesteatoma epithelium is important not only for understanding the complex mechanisms of the pathogenesis of this disease, but also to anticipate the possible development or recurrence of cholesteatoma.[18, 21] Extracellular matrix proteins (ECM) and the basement membrane of the epithelium affect the process of proliferation, migration and differentiation of keratinocytes, among others, by the activity of metalloproteinases (MMPs) in the cell matrix. (Figure 1 and Figure 2). It is believed that the process of apoptosis of epithelial keratinocyte of cholesteatoma contributes to cyst enlargement due to the accumulation of residues of the keratin.[20, 25] MMPs affect both inhibitory and stimulating effect on cell apoptosis. Angiogenesis in cholesteatoma is induced in two ways: first by monocytes, macrophages and leucocytes present within cholesteatoma perimatrix which are capable of producing angiogenic factors, such as: fibroblast growth factor, vascular endothelial growth factor, transforming growth factor alpha. On the other hand, keratinocytes themselves stimulate the release of angiogenic factors. Enhanced vascularization in cholesteatoma may also cause a continuous and pathologic growth of cholesteatoma mass. An increased rate of dead cells formed during the differentiation of keratinocytes may likely be related to apoptosis observed in cholesteatoma.[20, 24]

Further studies investigated the effects of fiber distribution in the human ear drum through biomechanical investigation. [26] Histological analysis of pars tensa retraction pockets were consistent with these results.[27, 28]

## INCIDENCE

In a cohort of children followed from age 4 to 16 years with annual examinations, otomicroscopy and tympanometry, the incidence of atrophy increased from 4% at age 4 years to 11% at age 16 years.[29] During the same period, the point prevalence of atrophy with retraction decreased from 4 to 0%, whereas the prevalence of atrophy with pexy increased from

0.3 to 3.2% at age 16 years. In total, the incidence of atelectasis was about 5.7% at school age, when the ET function is usually normalized.

There is an association between OM and retraction pockets, and VT insertion with retraction pockets.[30] While atrophy of the TM, which is a precursor of the retraction pocket in ears without history of OM is seen in 6% and of children at age 8 that resolved 10 years after. In children with history of OM, the rates of atrophy were 28% and 63% at age 8, which improved to 7% and 20% at age 18 for the ears that did not and did receive VT respectively. In this same group the prevalence of pars tensa retraction pockets were 0%, 0% and 3% at age 8, which worsened to 0%, <1% and 5% at age 18 for the ears that did not have history of OM, did not receive and did receive VT respectively. On the other hand, the prevalence of pars flaccida retraction pockets were 6%, 33% and 58% at age 8, which improved to 0%, 8% and 20% at age 18 for the ears that did not have history of OM, did not receive and did receive VT respectively.

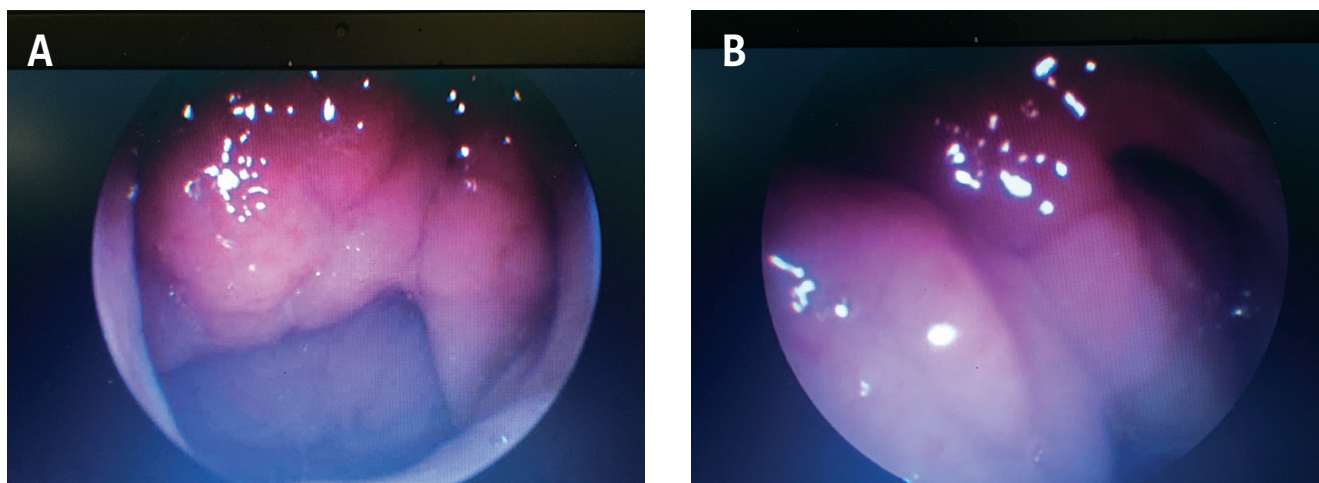
## CLASSIFICATION AND STAGING

The TM retraction pockets are classified based on three criteria: topographic, quantitative and qualitative. Topographic criteria refer to the location of the TM retraction pocket, indicating whether it is at pars tensa or pars flaccida. The location could be more specific by describing the involved quadrant. Quantitative criteria refers to the dimension whether it is partial or general. Typically, general retraction or atelectasis is not named as retraction pocket, unless some specific sections are more retracted.

One other important criteria used with respect to depth of the retraction pocket is whether or not the depth is visible with otoscopy or otomicroscopy. Increased use of endoscopes in the clinical setting has added a new criteria to the description and classification of retraction pockets, i.e., whether the depth can be visualized with an endoscope.

Qualitative criteria for retraction pocket classification refers to the behavior of the pocket, specifically whether the retraction pocket is self-cleaning or not. Loss of self-cleaning ability is considered as criteria for pre-cholesteatoma or cholesteatoma. Another qualitative criterion is whether or not there is bone erosion. This erosion can manifest itself as thinning and/or erosion of long process of incus, stapes superstructure, and/or erosion and receding of scutum. One other qualitative criteria is whether or not, there is fixation of the TM retraction pocket onto the ME ear structures.





**Fig. 9.** Adenoid tissue that is partially obstructing the nasopharynx, but more importantly that is impinging the Torus Tubarius by limiting the posterior rotation of the cartilage during swallows is visible with a) 0 degree b) 45 degree endoscope.

Earliest and most well-known staging system belongs to Sade.[31] In his classification, the degree of retraction is graded by its contact to incus, promontory or adhesiveness. Tos proposed a staging system focused on pars flaccida.[32] Adhesion, degree of invisibility and erosion were the criteria in staging. Charachon highlighted the visibility, erosion and self-cleaning ability.[33] Dornhoffer's staging was similar to Sade except Stage 4 was assigned to retraction pockets with invisible depth.[34] While there are several other classifications and staging systems, each of them prioritize some aspects as the important variables, and modify the stages accordingly.

The concept of staging assumes that each stage is distinct from other stages due to the difference as per the described variable. And that, that distinct feature is a prognostic indicator, determining the distinct fate of that ear, different than the ears that don't fall under that stage criteria. Therefore, a specific treatment modality for that should be applied to the ears under that stage to achieve the best outcome. This is the concept behind staging and classification. However, unfortunately, none of the staging systems have clearly distinct treatment methods, specific to each of the stages. Instead, as it has been summarized in the review conducted by Alzahrani, the same treatment methods are assigned to more than one stage by the creators of these staging systems for retraction pockets.[35] First of all, the purpose of staging system should be questioned, if staging does not help the clinicians decide the treatment protocol as per the proposed system. Second, if there are so many staging and classification systems, and none of these systems have a specific treatment method for any given stage distinct from the other stages, there must be some other factors that the cli-

nicians use while deciding on treatment method, and these factors may be too many to fit under a simple staging and classification system. One key element is the association of certain type or severity of retraction pocket in the development of acquired cholesteatoma, however, there is no consensus existing staging systems on retraction pockets or cholesteatoma in serving this purpose.[36] There must be many variables. Decisions and outcome are determined not only as per one or two more prominent ones, but a combination of these, with varying weight of importance not fixed and certain for each ear, but variable based on how each of the variable interacts with the set of other variables present in that specific ear. Based on this logic, the right approach is not to adhere to a specific staging system, but assess each ear as per the combination of these factors, and learn to prioritize the factors, in stepwise approach to decision making in the management of retraction pockets.

## VARIABLES AND DECISION MAKING IN THE MANAGEMENT OF RETRACTION POCKETS

The reason for having many different staging systems in the literature is because of the different variables that authors prioritize in their decision making.[37] None of these staging systems are applicable to management of all ears with retraction pockets. However, each and every variable are important, and may more important in some ears than the other ears. Each ear may have a combination of important variables that would have individualized set of priorities. Unfortunately there is lack of randomized controlled clinical trials (RCT). A recent Cochrane review identified only 2 RCT's on total of 71 patients, which concluded that in those

two trials, there was no evidence of significant benefit from cartilage graft tympanoplasties or ventilation tubes.[38] Under the current level of evidence, it is not possible to strongly support or refute any treatment modality or staging for the management of retraction pockets. On the other hand every ear surgeon follows a path of decision making in his/her approach to retraction pockets, based on the knowledge of the literature, school of training they underwent and past experience from their or their group's practice. The following section attempts to summarize the important variables and how they affect the decisions in management of retraction pockets. The decision algorithm outlined on Figure 3, is only an example of common variables and paths of decisions, however, it is not possible to include all variables in a single order of priority, because, a set of other variables may easily shift the order of importance.[39-41] Some variables that play a role in decision making in the management of retraction pockets are discussed below.

**Retraction Pocket without Evidence of Ongoing Eustachian Tube Dysfunction.** An important factor in decision making for the management of retraction pockets is whether or not there is ongoing ETD. Any patient that has TM retraction pocket should be evaluated for the presence of ETD. Although ETD may have played a significant role in weakening and thinning of the TM and development of retraction pockets, the patient may have outgrown of the ETD, but may continue to have a problem due to the sequelae that previous sustained MEP has caused (Figure 4). The TM may be extremely thin and it may have collapsed onto the promontory or towards sinus tympani or to epitympanum. Even though there may be no longer ETD, the ear may not be safe, and may develop cholesteatoma (Figure 5). Retraction pocket may progress because of being very thin and having very weak elastic layers. Even though ET may be opening periodically and significant negative MEP may not be developing, even normal pressure variations and slight negative MEP may be adequate to keep the TM retracted, even to the extent that the full depth may not always be visible. This kind of retraction pocket may lose its self-cleaning ability and start building up keratin. Based on the ETF testing methods explained earlier, if an ear has reasonably normal ETF, a VT should not be placed, and instead, the retraction pocket should surgically be managed with a tympanoplasty.[42] If it is pretty certain that the ETF is normal, there is no need to place a cartilage graft. A tympanoplasty with temporalis fascia should provide enough support to the TM, practically returning the TM to its original condition.

**Retraction Pocket with Ongoing Eustachian Tube Dysfunction.** A significant proportion of older children, teenagers and adults



**Fig. 10.** Tympanic membrane with geographic scar bands, and a retraction pocket touching the long process of incus and promontory. Note that the contact surface on the promontory may look like effusion, even though may just be from contact of two moist surfaces.

grow out of ETD but some may still have symptoms and findings of ongoing ETD that manifests itself as TM retraction (Figure 6). The presence of ETD would manifest itself with negative MEP, inability to correct the MEP with normal swallowing. If the significant negative MEP is not corrected with the repeated swallowing, then one can conclude that there is the likely condition of ETD. Performing a tympanometry would more clearly demonstrate the negative pressure. Any negative MEP less than -200 daPa implies that ET does not open frequent enough to correct the pressure difference between the ME and the environment. The ETD can be assessed and confirmed with more sophisticated tests. With or without ME effusion, if there is evidence that the patient has ETD, the risk factors should be assessed and managed, if possible. An important risk factor is family history, and even though that is not manageable, knowing that there is this important risk factor, decisions for management may be made based on the assumption that ETD may continue to be present. Other risk factors include exposure to viral respiratory tract infections, applicable mostly to infants and children in daycares and schools, environmental allergies, recurrent acute otitis media, recurrent sinusitis, acid reflux. Rare conditions like nasopharyngeal or parapharyngeal space tumors can also manifest themselves with ETD, and if the masses grow slowly, retraction pockets may develop.

Management options for the ongoing ETD include elimination of risk factors, if possible; use of periodic Valsalva maneuver

to inflate the ME and equilibrate the MEP to the environment; use devices that facilitate the increase in the nose and nasopharynx such as Otovent<sup>®</sup> **nasal balloon device**; **nasal pressure delivery devices such as EarPopper<sup>®</sup>**; or **performing balloon dilation of the ET**. [43, 44]

Surgeons should consider some preferential techniques in ears with evidence or suspicion of ongoing ETD. In general, medial grafts have higher risk of failure. Especially in ears that have retraction extending to the anterior TM, or if during the tympanoplasty very thin or adhered parts of TM tears and the condition turns into a perforation, there is a need to place the graft far anterior, securing the anterior edge of the graft from medializing. Techniques may include: making sure that there is sufficient support in the ME with the Gelfoam packing to push the graft laterally; denuding the medial part of the annulus and the anterior ME wall to prepare for better attachment of the graft; extending the annulus elevation to the anterior canal wall on the more severely retracted or perforated side, so that the graft can be laid on part of the anterior canal wall, still a medial graft by definition, because medial to the annulus; or creating an 2 mm anterior canal tunnel starting 5-7 mm lateral to the annulus and extending into the middle ear under the annulus, to be able to pull a tail of the fascia graft with a small right angle pick to create like hammock like suspension of the graft under the TM.

Lateral graft is in general better for definitely ongoing ETD, however, technically more difficult to perform, and has higher risk of blunting, lateralization and cholesteatoma formation if not done in experienced hands. Moreover, there is another problem with the lateral graft technique, because of the need for completely removing lateral layer of the TM. This is usually not possible to do in retraction pockets with thin or absent middle layers. Therefore, if lateral graft technique is used, it is better to remove the retraction pocket completely.

One other surgical technique is abundant use of cartilage to cover large areas of TM, definitely complete support of the retraction pocket. [45-47] A surgeon needs to be aware and should inform the patient that once the retraction pocket area is secured, other parts may start retracting, which may require repeat surgeries (Figure 7). There is the option to cover the entire TM with a cartilage graft, keeping in mind that, even though studies show no significant conductive hearing loss from this complete cartilage graft, sometimes there may be.; The assessment of ME status will become difficult as well as the placement of a VT, if not impossible. In a case that there is even a small gap between the ear canal and the cartilage, a tiny retraction, like in pars flacci-

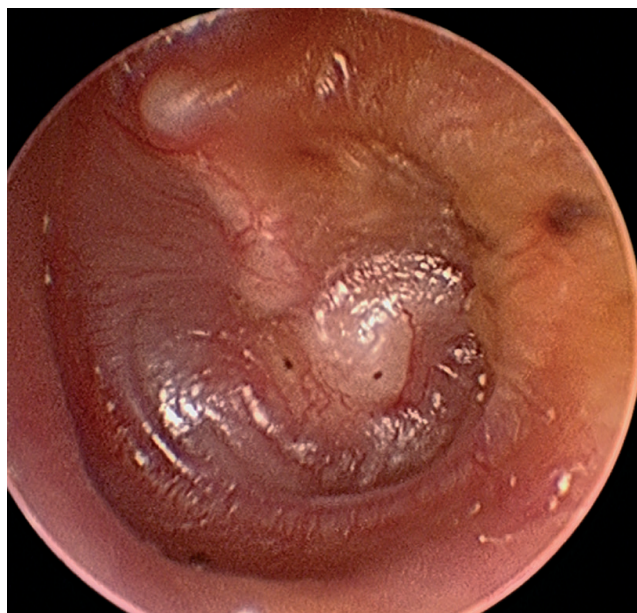


Fig. 11. Retraction pocket with ME effusion.



Fig. 12. Deep retraction pocket without ME effusion

da retraction pockets, may expand in long years to form a cholesteatoma, while this process will be difficult and too late to notice. One option is to insert a VT at the time of tympanoplasty if ETD is suspected. [48]

Retraction Pocket with Intermittent Eustachian Tube Dysfunction. There could be baseline well aerated ME with normal pressure under normal conditions. [49] But when ETF is challenged, with the reduced frequency or efficiency of

ET opening, or increased need for ET opening, there may be negative MEP, and even intermittent, can result in the development of retraction pockets (Figure 8a, 8b). This occurs when the ET opening frequency and efficiency is not able to keep up with the increased demand, due to increased gas absorption into the blood, expected with the increased blood flow, or rapid changes in the environmental pressures, or conditions that are related to the infections and allergy seasonal or exposure related exacerbations and would result in intermittent decreases in MEP, and development or worsening of the retraction pockets.[50, 51] It is critical to find out if the retraction pocket is visible or concerning only when the ear is exposed to pressure differences or when temporarily the ETF is worsened, and at other times the ear is safe. In such ears, options may include avoidance or prevention of such risk factors, or monitoring the ear after the exposure period to assess if there is persistence or progression of the retraction pocket. If the other measures don't work, balloon dilation of the ET may be considered instead of VT insertion in such ears.

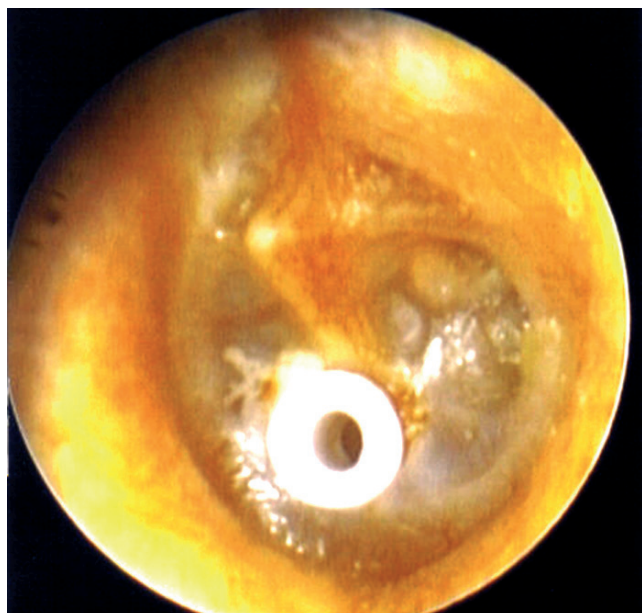
Presence of adenoid hypertrophy or re-growth of adenoids. Adenoid tissue is a risk factor for ETD and development and progression of retraction pockets. This is applicable mostly to young children, however, teenagers and young adults may have large adenoid tissue in general, or right posterior to the ET cartilage, affecting the ET with bacterial colonization and impingement of the ET cartilage rotation with the swallowing, therefore limiting the widening of the ET orifice that prepares the orifice by widening for complete opening. Even though a child has had a previous adenoidectomy, there is a need to pay attention to the re-growth of adenoid tissue, especially the peri-tubal lymphoid tissue posterior to the ET cartilage, limiting the posterior rotation of the ET cartilage and helping to open better the ET orifice.[52, 53] Especially in children between 4-8 years of age, adenoidectomy should be considered in the presence of retraction pocket, even if the adenoids are not too large or obstructing. In children younger than 4 and older than 8, an endoscopic examination can reveal large and obstructing adenoids.[54-56] Special attention should be paid on the adenoid tissue next to the ET cartilage (Figure 9a and 9b). Video-endoscopic evaluation during swallowing should focus on whether the adenoid tissue is impinging the posterior rotation of the ET cartilage with the elevation of soft palate with Levator Veli Palatini muscle contraction. Even though adenoid tissue is not too large or obstructive, this tissue may be large enough in the fossa of Rosenmuller to limit the posterior rotation of the cartilage and impinge the ET opening.[52] Video-recording of the endoscopic evaluation during swallow with a ideally 45°, if not available with a 30° or 70° endoscope allows the



Fig. 13. Deep retraction pocket with effusion

detailed evaluation of the changes in the peri-tubal structures swallow, the assessment of normal and abnormal patterns of swallow, and structures affecting the ET orifice widening.[53, 57] The effect of adenoid tissues on the ETF is not limited with previously non-operated adenoids. There is a known risk of residual or re-growth of adenoid tissues after adenoidectomy. There have been reports that majority of the residual or regrowth of adenoids is observed in the lateral parts of nasopharynx, therefore potentially affecting the ET cartilage rotation during swallow and associated impingement of the ET cartilage rotation and ETF.[58, 59] It is also important to note that, while adenoid tissue typically is a distinct tissue from the torus tubarius, it is not unusual that adenoid-like lymphoid tissue extends onto the torus, and becomes a part of the problem affecting the ETF. Such tissue needs to be carefully, cleaned with special attention to the lymphoid tissues within the fossa of Rosenmuller and on the Torus Tubarius. Use of suction Bovie at low settings under endoscopic visualization will increase the precision of tissue removal while protecting the important ET structures. It is also possible to consider a light superficial touch up of the surface of the Torus Tubarius, without using the open end of the Bovie,

Presence or absence of effusion. This is an important factor in decision making in the management of retraction pockets, because, it is very likely that there is ongoing risk factors for otitis, and very likely ongoing ETD. However sometimes



**Fig. 14.** After VT insertion, although there can not be negative ME pressure, adhesions may prevent a retraction pocket to resume its neutral position

it may not be easy to diagnose effusion when there is geographic appearance of sclerotic and atrophic areas, and when parts of the TM is touching the promontory (Figure 10). The normally moist mucosal surfaces at the interface between the medial surface of TM and the promontory will have a meniscus of very thin fluid layer and may be mistaken as effusion. Lifting this area with pneumatic otoscope will reveal absence of effusion. It should be also noted that ME may temporarily have effusion after an OM or even a viral infection. Therefore decisions should not be made based on incidental finding of effusion, instead of confirming chronic ME effusion.

It should be noted that, if the ME is filled with effusion, it is not possible to test the ETF (Figure 11). If there is chronic effusion, and there is a need to place a tube, this could withhold any decision for tympanoplasty, until status of ear is assessed after the tube insertion. It would not make sense to perform a tympanoplasty with or without cartilage, and find out that the ear is developing ME effusion, and decide to place a tube. This is especially valid, if a VT (standard or a T-tube) would bring the TM to completely visible and safe position. If a VT going to stabilize the ear, and make the ear safe, it should be the first choice. Presence of effusion is more likely in children, however, adults present with effusion and retraction pockets may need tubes.

**Invisible Depth Of The Retraction Pocket Without Effusion.** It may be difficult to tell if there is effusion. Every effort

needs to be made to visualize full depth of the retraction pocket to assess the severity of retraction pocket, whether there is adhesions, early keratin build-up or cholesteatoma formation (Figure 12). Key diagnostic tool is pneumatic otoscopy. This is an essential routine examination tool, to only differentiate the ears with or without effusion in the ears with retraction pockets, but also to assess if the retracted TM will come out, and become visible, or whether it continues to be retracted and invisible, with or without an adhesion. Otomicroscopic exam with Brunning's or Siegel's pneumatic otoscope will also be helpful, with better visualization to assess if the retracted TM comes out. If this pneumatic otoscopic methods don't work, clinician should try to insufflate air into the ME, to see if this retracted TM would be pushed out. Valsalva or Toynbee maneuvers may be successful in delivering air into the ME and push the TM out to make it visible. If this examination and assessment was done under anesthesia, using Nitrous oxide, and giving some time for the inhaled Nitrous oxide gas to dissolve into the blood and then exchange into the middle ear space, creating a relative positive pressure that pushes the TM out. Endoscopes have expanded our ability to visualize the depths of retraction pockets.[60] A 0 or 30 degree endoscopes are most commonly utilized and are capable of seeing the entire depth of a retraction pocket in most cases. Cartilage and perichondral graft shapes and sizes can be tailored to the need of individual ear. A large variety of cartilage grafts are described in the literature.[61]

**Invisible Depth of the Retraction Pocket with Effusion.** When the depth of the retraction pocket is invisible and if there is chronic effusion, it is best to place a VT (Figure 13). There is no point in visualizing the depth of retraction pocket better, prior to the insertion of VT. An ear with chronic ME effusion will need a treatment. The first and most common treatment method is VT insertion, however, recently new treatment methods are becoming popular. Although to date, there are no randomized clinical trials showing efficacy of the balloon dilation, in experienced hands, this procedure is considered safe, therefore, if this procedure were to be tried in some patients, this group that has a retraction pocket with invisible depth and ME effusion would represent relatively worse clinical presentations of the retraction pockets, making this balloon dilation trial option reasonable, more than the other presentations.

**Ongoing Retraction Pocket After VT Insertion.** A persistent retraction pocket after the VT insertion implies either adhesion bands keeping the TM in the retracted position, or non-functional VT, with blockage, or not being able to equilibrate the MEP in the entire ME segments, possibly due to the

scar bands or folds blocking the aeration pathways within the ME (Figure 14). If the retraction pocket depth remains invisible, there is a need to assess if there is a cholesteatoma with an endoscope, CT scan or both and perform tympanoplasty with or without removal of cholesteatoma.

**Suspicion of Cholesteatoma in a Deep Retracted Pocket with Middle Ear Effusion.** Suspicion of cholesteatoma requires a CT scan. However, a CT scan will not be helpful when there is ME effusion, unless there is apparent bony erosion that could be visualized with the CT scan. Effusion and an early non-erosive cholesteatoma would look the same on the CT scan. Therefore, prior to ordering a CT scan, it is better to place a VT, let the ME dry, see if the retraction pocket comes out, depth becomes visible, and retraction pocket becomes safe. However, if, after the VT placement, the depth of the retraction pocket remains invisible, a CT scan will then be helpful in identifying soft tissue after the resolution of effusion. Alternative to the CT scan, despite the disadvantage of not being able to show bony erosion and not being sensitive to pick small cholesteatoma is-use of MRI in high-risk retraction pockets.[62]

**Location Of Retraction Pocket.** Even not too large or deep any retraction pocket at the posterior superior quadrant is of higher risk and importance.[63] On contrary, a tube related retraction pocket at the anterior inferior quadrant is not risky, and can be followed for a long time. While the depth of the retraction pocket can be more or less guessed from the curve and direction of the retraction pocket, this is much harder and unreliable to manage when the retraction pocket is located at the pars flaccida (Figure 15). Pars tensa retraction pockets at the posterior superior quadrant are more common whereas pars flaccida retraction pockets are rare in the pediatric population. Pars flaccida retraction pockets may be seen in adults, with more advanced state, after they develop cholesteatoma.

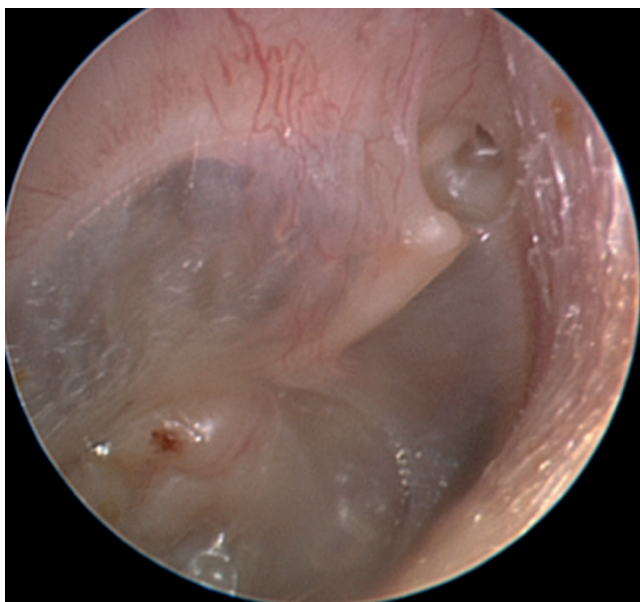
The pars tensa retraction pockets at the anterior inferior quadrant are usually related to the previous VT insertion, and they are relatively safe, since it is easier to visualize the full depth, and if progresses there is less risk for dangerous cholesteatoma formation. One option for such small retraction pockets may be excision and patch myringoplasty, hoping that the small perforation will close with a better and thicker TM, and retraction pocket will not recur. Another option is to excise these small retraction pockets and insert a new VT.[64, 65]

**Progression.** Whether the retraction pocket is in its final sequelae stage or it is gradually worsening with ongoing risk

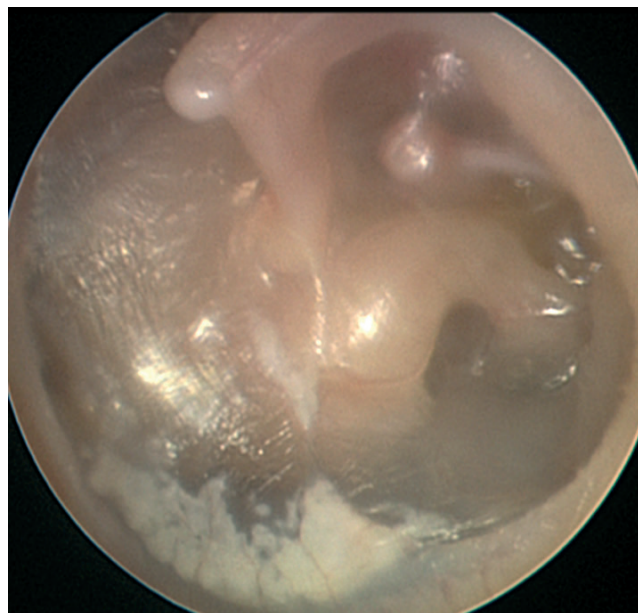
factors matters. As stated earlier, it is mostly assumed that progression is secondary to ongoing ETD, however, very thin TM can progress with relatively normal ETF. This requires closer follow-up and more timely treatment, but at the same time choosing more conservative methods such as VT insertion, instead of a tympanoplasty, as there would be a high chance of recurrence. Even a cartilage shield graft covering the posterior superior TM, or even majority of TM may not prevent the development of retraction pockets which is not supported by the cartilage between the ear canal and the cartilage.[66, 67]

Progression of retraction pockets can be followed without VT insertion, if it is possible to visualize the entire depth of the retraction pocket. Even if full depth may not be directly visible, if it is possible to pull the pocket out with pneumatic otoscopy, follow-up may still be safe, before a decision is made for more definitive treatment method. Endoscopic follow up allows visualization of the full depth of retraction pockets in most cases, however, office endoscopy may not be safe in patients that cannot be fully compliant with keeping still, which applies to all young children, and some teenagers and adults.

**Erosion of the Ossicles.** Ossicular erosion is classically associated with cholesteatoma, however, this is sometimes seen in the absence of classical cholesteatoma with keratin build-up (Figure 16). It is more commonly seen at the long process of incus, at a location closer to the incudo-stapedial joint. Even in the absence of keratin development or progression of the retraction pocket, in retraction pockets that is continuously in contact with some adhesion, it is not rare to see gradual thinning of the long process of incus, gradually turning into a fibrous band between the long process remnant and the head of the stapes.[68] This condition has clinical and surgical implications. The incudostapediopexy status, the status of retraction pocket laying on both incus and stapes, and when this condition progresses, laying only on the stapes head does not present with significant hearing loss. However, when the TM is no longer in contact with the stapes head (if there is no adhesion) with the improvement of retraction pocket, with resolution of ETD risk factors or VT insertion, suddenly maximal conductive hearing loss may develop. Similarly, if a tympanoplasty does not address the ossicular continuity and communication between the TM and the footplate, outcome again could be maximal conductive hearing loss. During tympanoplasty, contact between the stapes head and the TM must be achieved, by laying cartilage shield graft on the stapes head (may require folded, double layer cartilage placement), incus interposition graft, or partial ossicular reconstruction prosthesis placement (typically



**Fig. 15.** Retraction pockets are seen at both pars flaccida and pars tensa. Full depth of pars flaccida pocket is not visible, therefore should be considered more at risk for cholesteatoma formation.



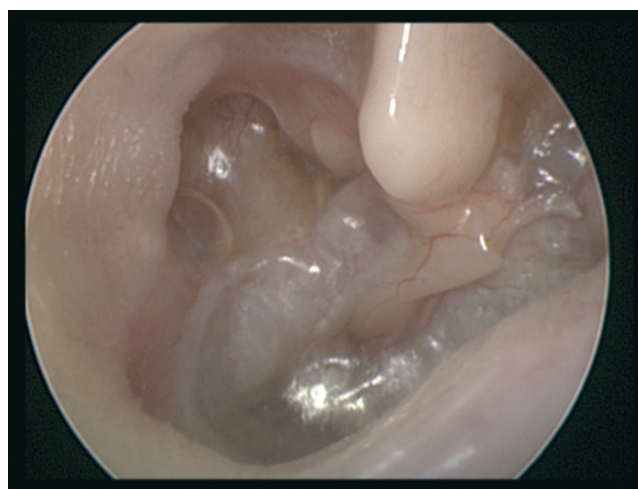
**Fig. 16.** Retraction pocket without any keratin build-up but with erosion of long process of incus.

covered with cartilage graft).[69] Use of endoscope is useful in surgical management of retraction pockets in attic and in the sinus tympani.[70]

**Status of the Other Ear.** In infants and young children, it is more likely that both ears of a child have similar findings. As children grow, a difference between the two may become apparent. In teenagers and adults, it is not uncommon to find problems of ETD, retraction pocket even cholesteatoma on one side while the other is essentially normal. Having a normal other ear may allow the surgeon and the patient to take more risk, either by observing without any surgical intervention even though the ear may progress, or performing reconstructive surgery. Even though the treatment may not resolve the underlying ETD and retraction may recur.

If the other ear also has similar or worse process of retraction pocket, cholesteatoma and/or hearing loss, decisions are made more conservatively. Priorities would be making ear safe with the most conservative treatment options, such as VT insertion.

**Signs of Decreased Self-Cleaning Ability of the Retraction Pocket.** Signs like thickened layer within the retraction pocket limiting its mobility with pneumatic otoscopy, or appearance of a cast like wax formation within the retraction pocket, or formation of Herodion, a path of keratin tracking from the edge of retraction pocket and extending laterally on the ear



**Fig. 17.** Ongoing ETD after a tympanoplasty may result in chronic effusion and recurrence of the retraction pocket.

canal implies that even though there is ongoing self-cleaning, there is a possibility that this is not adequate, and there may be early backing up of keratin. There is a transitional state between a retraction pocket and a cholesteatoma, and this transition is not sudden, one day the ear has retraction pocket and the next day it turns to a cholesteatoma. This pre-cholesteatoma state of ear will have some features of retraction pocket and cholesteatoma, without full manifestation of cholesteatoma. Technically, this term also implies that it is a reversi-

ble condition, and with proper management, cholesteatoma formation may be prevented. In the presence of ETD, these methods include auto-inflation techniques, balloon dilation of ET, and VT insertion. If the ear no longer has ETD, then tympanoplasty would be warranted. Adhesion. The retraction pocket may be sitting on the ossicles and promontory and even may have the appearance of adhesion, however, this appearance may be misleading. The TM may be pulled off the surfaces by the techniques described earlier. However, there may be adhesion bands keeping the TM attached to the visible and invisible surfaces. Moreover, these adhesion bands from infections can gradually be pulling the retraction pocket deeper, even in the absence of ETD or negative pressure. Apparently, this implies an irreversible condition, and therefore will need lysis of these attachments, and measures to prevent re-formation of these adhesion bands. A tympanoplasty with cartilage graft should have the nude surface facing the ME to reduce the risk of re-formation of adhesions, while the surface with perichondrium should be placed facing lateral, under the TM remnant. Ideally, the perichondrium can be made larger than the cartilage itself, allowing larger parts of the TM covered under with this extra “skirt” of the perichondrium, and part of the perichondrium laid on the external ear canal and if present on the manubrium, for the stability and viability of the cartilage graft. It is best to harvest the cartilage with perichondrium on both sides, and elevate one side, but still keep it attached to the cartilage and trim the cartilage (but not the perichondrium) to the desired size, resulting in an island graft with relatively smaller cartilage surrounded by a large perichondrium. Naturally, tragal cartilage is more suitable for this purpose, if the superior free edge of the tragal cartilage and perichondrium is kept intact.

Previous myringoplasty or tympanoplasty. If previously repaired TM perforation retracts, the depending on whether or not effusion is present, a ventilation tube could be placed or a cartilage shield graft would be considered. If there is no effusion, but TM is retracting, it is better to use measures that help ETE, try auto-inflation methods, or place a cartilage graft. If there is effusion that persists, it is better to place a VT. Placement of long lasting tubes like T-tubes need to be considered. On the other hand, these tubes have higher risk for TM perforation after extrusion or when removed, there-

fore a sub-annular tube may be considered. Although cholesteatoma formation from tracking of ear canal skin along the shaft of the T-tube under the annulus into the ME is not a concern of most surgeons who prefer sub-annular tubes, this risk should be carefully monitored. One other caution is the location of the groove, if drilling in the ear canal under the annulus is preferred for the location of this sub-annular tube. The posterior inferior quadrant of the ear canal may be the easiest place to create a tympano-meatal graft, but if drilling is to be performed, a surgeon should keep in mind that inferiorly, facial nerve may track lateral to the annulus.

Retraction pockets may develop after removal of cholesteatoma, irrespective of the type or extent of the disease (Figure 17). Intact canal wall tympanoplasty increases not only the risk of residual but recurrent cholesteatoma as well. This often starts with development of a retraction pocket towards attic and mastoid.[71]

## CONCLUSIONS

Retraction pockets are caused and worsened by a large number of factors. Retraction pockets have the risk of progressing into a cholesteatoma, therefore risk factors for this progression needs to be carefully assessed and eliminated if possible. Retraction pockets with signs of progression should be closely and carefully monitored. Management of retraction pockets start with the treatment of these risk factors, assisting the pressure equalizing ability of the ET, and bypassing this function by VT insertion. Making the ear with retraction pocket safe is the priority. While goals of management remains to make the ear safe, if there are ongoing risk factors, primarily persistent ETD, priority is to make the ear safe by stabilizing the retraction pocket, and stopping the progression. If, on the other hand, the risk factors are no longer present and the retraction pocket is a sequelae, permanent solutions with tympanoplasty with or without cartilage support is preferred. Irrespective of status of the TM and which treatment method is preferred, caution should be taken by close follow-up, to notice any sign of recurrence of retraction pocket in the same or other locations, and not to miss a developing cholesteatoma.

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