Chronic Otitis Media Without Cholesteatoma

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Introduction

Otitis media is defined as an inflammatory process, infectious or not, focal or generalized, located in the middle ear cleft [1]. Because of its high prevalence and multiple clinical presentations, otitis media generates high social costs and gets directly or indirectly expensive. In the same way, the family suffering caused by the otitis media is absolutely immeasurable—the pain, the discomfort, the hearing loss, the moral damage of an ear with chronic fluids and foul smell, the psychological trauma of a child who is unable to enjoy the fun of a single dive (or at least a single bath unsupervised), the complications, and unfortunately, the death, are still problems faced by otorhinolaryngologists in their daily office routine. Thus, even with extreme scientific advances, otitis media contradicts all expectations and is, still in the third millennium, a global health public problem far away from being equated [2].

Definitions

Chronic otitis media (COM) can be defined under different aspects: the clinical, the temporal, and the histopathological [3].

From the eminent *clinical* point of view, it is characterized as an inflammatory condition associated with wide and persisting perforation of the tympanic membrane (TM) and otorrhea.

Chronologically, it refers to an inflammatory process of the middle ear, which does not last less than 3 months.

Histopathologically, it has been defined as an inflammatory process of the middle ear associated with irreversible tissue changes. Still, from the histopathological aspects, this inflammatory process may even dispense from tympanic perforation and chronic otorrhea for its characterization. However, it will always be associated with irreversible clinical tissue damage.

While acute otitis media (AOM) presents an inflammatory process that develops abruptly, and the resolution comes quickly and thoroughly, COM is usually associated with insidious, persistent, and, mainly, destructive inflammatory situations. It is subclassified into two major groups: non-cholesteatomatous chronic otitis media (NCCOM) and cholesteatomatous chronic otitis media (CCOM) [4, 5]. As their names reveal, the fundamental difference between these two groups is the presence of the cholesteatoma, which on its own would place a degree of increased aggressiveness to this condition. The present chapter describes and discusses only the NCCOM and its variations.

Etiology

Some of the main situations that trigger the installation of a chronic inflammatory process in the middle ear are recurrent otitis media**,** acute necrotizing otitis media (ANOM), and trauma. In addition, social-economical and geographical factors also influence the incidence of the disease.

Repeated Otitis Media

The NCCOM may originate from a repeated outbreak of acute otitis media (AOM) [6–9], either because of inappropriate treatment or conditions that ease the re-infection. Risk factors for repetitive AOM are selective immune deficiency, allergy, and eustachian tube dysfunction, among others. Understanding these factors and the correct treatment is vital to prevent the process from becoming chronic. In case the outbreak happens many times, the possibility of weakening

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of the TM increases, with the consequent manifestation of degenerative mucous changes, such as epithelial dysplasia and interstitial fibrosis. This cycle will make it easy to establish the chronicity of the infection or the functional and morphological consequences [10–12].

Acute Necrotizing Otitis Media (ANOM)

The NCCOM may also develop after an outbreak of necrotizing AOM [13]. Most of the time, the ANOM affects children and young people with a systemic acute or toxic disease like scarlatina, pneumonia, measles, or any other acute and feverish disease. The otologic infection often goes unnoticed until we have otorrhea, which is usually profuse and requires regular microscopic aspiration. If this is not done, the diagnosis will be postponed until the next appointment with the physician. A typical characteristic of this entity is the erosion of the TM in the early stages of the disease, happening at the same time the patient begins to complain about pain and the beginning of the otorrhea. The otoscopy will reveal, very soon, a perforation of great dimensions different from that seen in common suppurated AOM with small and little granulation tissue around.

Trauma

Another possible etiology for a tympanic perforation, and thus the characterization of an NCCOM, is the trauma over the TM. The most common traumas are barotrauma (atmosphere pressure variation), explosion, and foreign bodies (cotton swabs, keys, and pens). Most often, the damaged TM heals spontaneously, mainly in children. However, in substantial losses, the healing may not happen and result in a perforation. Thus, an open door for contamination will be formed, from the external auditory canal into the middle ear.

Upper Airway Infections

Repeated upper airway infections, associated or not to allergic conditions, are a possible risk factor for COM [14]. Chronic tonsillitis can collaborate with impaired middle ear homeostasis, usually associated with eustachian tube dysfunction. Adenoid hypertrophy can promote obstruction of the ostium of the eustachian tube and can be associated with contamination of the region, mainly in children [15, 16]. Allergic and infectious rhinitis and sinusitis have also been associated with generating favorable conditions for middle ear contamination through mechanisms similar to those in tonsillitis.

Fungal Otitis Externa

Especially in countries with a tropical climate and also considering a predilection for certain seasons of the year, external fungal otitis can be quite frequent. In some cases, they can lead to partial necrosis of the TM and generate a tympanic perforation, often permanent. Prompt recognition of this condition and, consequently, appropriate treatment may reduce the risk of irreversible damage to the TM.

Social-Economical and Geographical Factors

Malnutrition, inadequate hygiene, low quality of living, and high population density are factors that, from the epidemiological point of view, are associated with a higher incidence of middle ear infection. The incidence of COM is higher in countries with a low development level.

Clinical Forms

While CCOM is unequivocally characterized due to the presence of squamous epithelium in the middle ear (or other pneumatized areas of the temporal bone), NCCOM presents a greater variety of clinical presentations [4].

Although it is impossible to subdivide with absolute precision the clinical phases of a disease as complex as COM, it is necessary to establish some parameters for didactic and research purposes to organize the study. Thus, the clinical entities described below as separate diseases may often present points of intersection between them or simply coexist in the same ear. For example, a retraction may course with effusion, or a tympanic perforation may result from a retraction.

Retractions

It is proposed that TM retractions result from the middle ear and mastoid deventilation. Starting in a dysfunctional eustachian tube, the sustained negative pressure in the middle ear ends up collapsing the most fragile wall of the system, that is, the TM [11, 16–18]. Retractions are classified according to their location and degree.

The study of retractions should also consider the presence of effusion, involvement of the ossicular chain, or other bone erosions (posterosuperior wall, lateral wall of the attic). In the clinical evaluation, in addition to the otoscopic findings at the first consultation (degree and location), it is essential to analyze the hearing status and to follow evolution over time. The instability of the retraction, with recurrent granulation or otorrhea, is also crucial in deciding on the (surgical) management of the retraction.

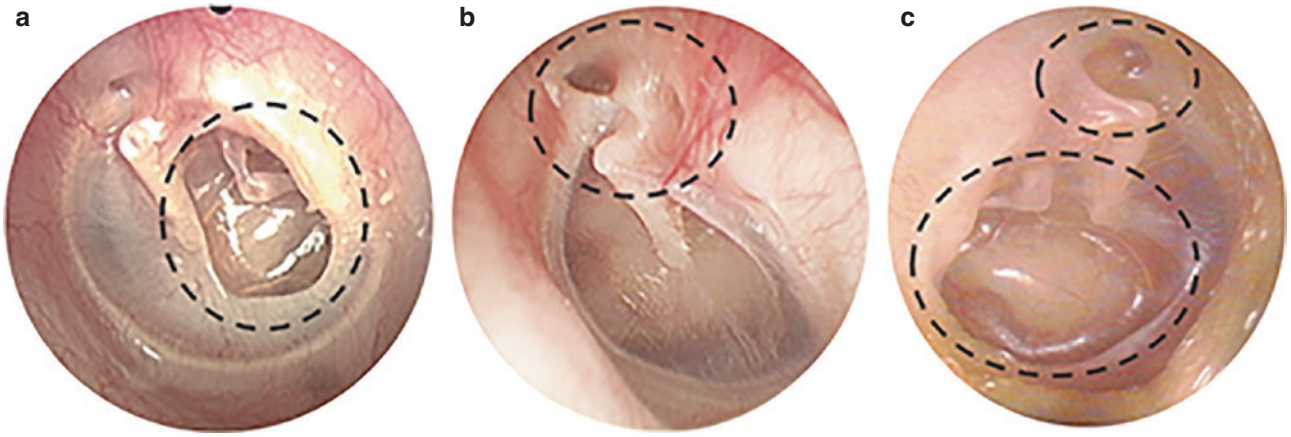

Fig. 38.1 Retractions according to localization. (**a**) *Pars tensa* retraction. (**b**) *Pars flaccida* retraction. (**c**) Two-route retraction

We suggest the reader refer to the specific chapters about TM retractions available in this book. In Fig.38.1, we illustrate the three types of retractions according to localization: *pars tensa*, *pars flaccida*, and two-route retractions.

Perforations

Tympanic membrane perforations are defined as a lack of integrity of the tympanic membrane (TM), which can result from different etiologies and require different treatments. They are part of a spectrum of diseases known as chronic otitis media (COM). Historically, tympanic perforations are divided into *central*, that is, if there are TM borders in all 360° of the perforation, and *marginal*, if there is a lack of any of the perforation edges [4, 5].

In our group, after many years of studies and publications on this subject, we currently prefer to classify perforations as ***outside-in*** (or retraction-perforation) or ***inside-out*** (without associated retraction or explosive), as we will see below. We strongly recommend that the reader to refer to chapter **Tympanic Membrane Perforations and Tympanoplasty: New Profiles, New Strategies**.

In short, the ***inside-out*** are the ones not associated with retraction, that is, an "explosive" perforation. This group of perforations, whose proposed etiology is related to acute events (acute suppurative otitis media, traumatic perforations, and external otitis with partial necrosis of the tympanic membrane), is understood as an "accident." They are previously healthy middle ears, which possibly have suffered aggression at a specific moment, be it traumatic, inflammatory, or infectious, generating tympanic perforation as a permanent sequel. Disruption of intratympanic homeostasis, caused by an acute mechanism, will modulate a series of histological transformations in the mucous lining of the middle ear, which would change permanently, characterizing COM. Figure 38.2 illustrates an *inside-out* perforation.

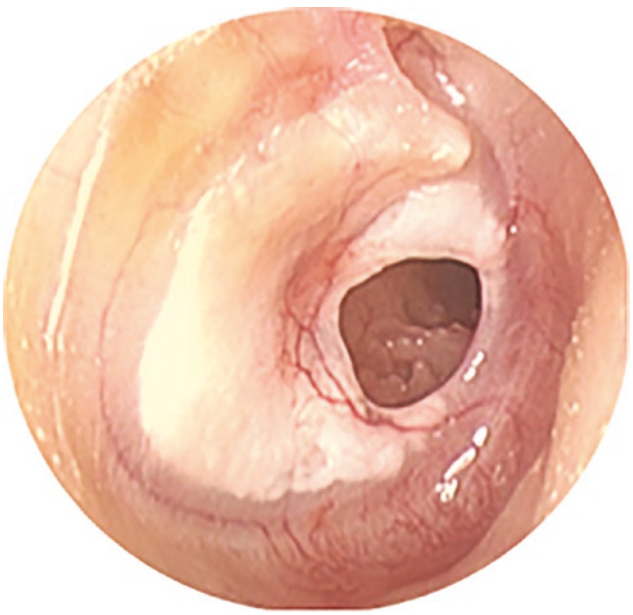

Fig. 38.2 Inside-out perforation, without signs of TM retraction

On the other hand, the ***outside-in*** perforations (Fig.38.3) refer to the ones associated with retraction signs. Those signs are (a) medialization of the malleus handle, (b) tympanic remnants over the promontory, (c) tympanic remnants over the ossicular chain, and (d) erosion of the ossicular chain. It represents an alternative pathogenesis model in which a progressive retraction would lead to perforation. The pressure self-generated by the retracted TM against the structures of the middle ear (ossicles, promontory) induces ischemia of the capillaries of the membrane and, consequently, the necrosis of a part of it. It is reaching the apex of this process that the retraction will turn into a perforation.

Thus, the pathogenesis proposed for this group of perforations is very similar to that of tympanic retractions, involving a negative pressure mechanism (probably from auditory

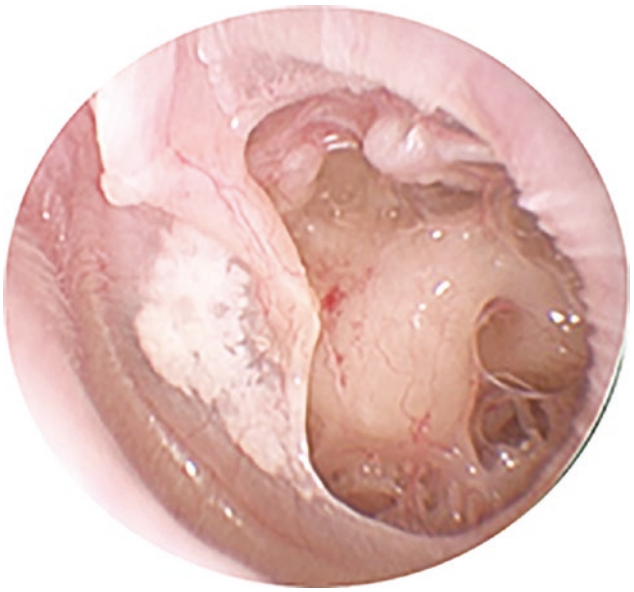

Fig. 38.3 Outside-in perforation, with signs of TM retraction

tube dysfunction) and an entire altered middle ear homeostasis, which would be responsible for the progressive retraction until perforation.

Chronic Silent Otitis Media

Although NCCOM is traditionally associated with tympanic perforations and otorrhea, histopathological studies in animals and human beings are unanimous in demonstrating pathological tissue with irreversible inflammatory changes in the middle ear despite the presence of perforations in the tympanic membrane. Based on evidence like this, Paparella et al. [19] created the term *chronic silent otitis media,* referring to the inflammatory pathologies in the middle ear with veiled and disguised progression. Thus, it is important to highlight that the presence of a normal TM does not exclude, in many cases, the detection of otitis media.

This way, aiming to avoid the inappropriate use of the term chronic silent otitis media (CSOM), it was divided into two variants: *undetected* and *undetectable*. This distinction is crucial since the *undetected* CSOM may be due to a primary failure during the otolaryngologic investigation; on the other hand, the *undetectable* CSOM is a challenging diagnosis to the physician, who must be aware of the fact that an asymptomatic ear, apparently normal, may shelter an active and focal pathological process.

In 1991, Costa studied 144 human temporal bones with irreversible inflammatory changes (granulation tissue, ossicular changes, cholesteatomas, cholesterol granuloma, tympanosclerosis, and tympanic membrane perforations) [2]. The temporal bones were divided into two groups: those with intact tympanic membrane (group I) and those with tympanic membrane perforations (group II). The more frequent findings in these temporal bones were granulation tissue (in 97.9%), ossicular changes (in 91.6%), tympanosclerosis (in 24.3%), tympanic membrane perforations (in 19.4%), cholesterol granuloma (in 13.8%), and cholesteatomas (in 10.4%). There were tympanic membrane perforations in 28 temporal bones (19.4%), while 116 bones presented intact membranes. Surprisingly, the comparative analysis of the temporal bones with or without tympanic membrane perforations indicated that the groups were similar: granulation tissue, ossicular changes, and cholesterol granuloma were found in both groups, and the differences in frequencies were not meaningful. However, cholesteatomas and tympanosclerosis were identified more frequently in temporal bones with tympanic membrane perforations (group II).

Clinical Implications and Physiopathogenesis Evolved from the Concept of the Chronic Silent Otitis Media

Frequencies and Distribution of the Granulation Tissue

The high frequencies and generalized distribution of the granulation tissue throughout the entire middle ear cleft in temporal human bones with or without tympanic perforations seem to be very important [3, 15, 20–22]. A lot has been written about the CCOM. However, a few publications mention the problem of some patients who, although do not present cholesteatomas, complain of profuse and continuous purulent otorrhea. The origin of this otorrhea is the granulation tissue that practically fills up the middle ear and, not rarely, extends to the mastoid. This type of otitis cannot be considered a synonym of the *simple* chronic otitis media (a term widely used in medicine to name an otitis media case that is not associated with cholesteatomas), since it carries a morbidity similar to the one in the cholesteatomas itself. In a panel about mastoidectomies, Paparella, Caparosa, and Glasscock were unanimous in pointing to the granulation tissue as the primary pathologic condition that most frequently conducted this procedure.

Lau and Tos [23] named this condition granulomatous NCCOM, recommending, in these situations, the tympanomastoidectomy whenever an aggressive conservative therapy (with frequent cleaning, local and systemic antibiotic therapy) does not succeed in controlling the inflammatory process after three months. Proctor [24] recommended tympanomastoidectomy in patients with chronic otorrhea, who are resistant to the clinical treatment, despite the pres-

ence of cholesteatoma in the middle ear. Paparella et al. [25] and Costa and Cruz [26] claim that the presence of small masses of focal granulation tissue strategically distributed in the middle ear cleft (mainly in the region of the round window niche) is responsible for hearing loss.

In conclusion, the granulation tissue is an almost unique characteristic of the histopathological profile of chronic otitis media. Its clinical importance originated from the possibility of generating, even in isolation, a series of signals and symptoms like otorrhea, osteitis, ossicular necrosis, conductive and sensorineural hearing loss, and disturbs related to the posterior labyrinth. Besides that, its pathogenicity does not end itself since the presence of granulation in transitional areas of the middle ear cleft (protympanum, isthmus and *aditus ad antrum*) interrupts the airflow. Thus, a vicious cycle of additional pathological changes is established.

Interaction Between Middle and Inner Ear

Various studies show the connection between chronic otitis media and cochlear damage. Paparella et al. [27], English et al. [28], Dumich and Harner [29], Walby et al. [30], and Paparella [1, 31] all of them indicated the relationship between chronic otitis media and sensorineural hearing loss. Paparella et al. [27] investigated the pathology of sensorineural hearing loss caused by otitis media. Also, Goycoolea et al. [32] and Walby et al. [30] published this subject. Their work points to the round window membrane as responsible for transmitting the pathological process to the labyrinth. The anatomy and localization of the round window niche predispose to the accumulation of pathology in the presence of otitis media.

Dysventilation of the Middle Ear Cleft and the Development of Otitis Media

Several studies point out that dysventilation is the most important factor in the pathogenesis of otitis media. Therefore, the regions deprived of O_2 tend to suffer pathological alterations more frequently. To corroborate this hypothesis, studies with patients and with temporal bone dissections have also led us to believe that sites of ventilation obstruction (in certain sections or compartments of the middle ear) may influence chronic otitis media pathogenesis [16, 33–35]. Those obstructions might be due to anatomic, physiologic, or even pathologic variations that result in compartmental pathologic processes [16].

With this information, we can more appropriately investigate, understand, and work with some otological symptoms that seem, at first sight, obscure, and inexplicable. Neglecting those concepts would be a tremendous medical mistake, which suggests that we are more interested in provoking or sealing perforations in the tympanic membrane rather than diagnosing and treating pathologic conditions of the middle or inner ear.

Ossicular Pathology of the Chronic Otitis Media

The hearing loss associated with otitis media may appear early or late during the process, varying considerably regarding the type and degree. As for the type, the hearing loss may be conductive, sensorineural, or mixed [36]. As for the degree, depending on how aggressive and the extension of the pathological process, they can vary from mild or moderate losses (usually associated with the conductive type) in the majority of cases. However, some cases may course with severe and profound losses (sensorineural).

As mentioned before, the mechanisms involved in sensorineural hearing losses have been explained based on the biological behavior of the round window, mainly, and the oval one during the inflammatory processes of the middle ear [37, 38]. This way, they can be understood as gateways for toxins from the middle ear toward the inner ear, characterizing the interaction between these two compartments. Furthermore, several studies demonstrate an association of the chronic otitis media to cochlear damage, again pointing to the round window as the structure responsible for the transmission of the pathological process to the labyrinth.

On the other hand, the conductive losses regarding COM include a series of changes in the tympano-ossicular system. Concerning the tympanic membrane, the loss of substance (perforations), the atrophy (and the consequent retraction), and the increase of its rigidity (because of tympanosclerosis plaque) are some of the most often causes of hearing loss. The ossicular system, however, can also be compromised by ossicular erosion (with the interruption of the sound transmission as a consequence), as ossicular fixation, due to tympanosclerosis or fibrosis.

Finally, the effects of mass in the middle ear cleft (caused by liquid, cholesterol granuloma, cholesteatomas, or granulation tissue) may damage the ossicles and compromise the vibratory capacity of the tympano-ossicular system. Thus, conductive hearing loss is generated or majored.

Considering the dynamic nature of COM, all those changes may present themselves isolated or in combinations at the middle ear. For instance, as usual, gross erosions of the long incus process follow the atrophy and retraction processes of the posterosuperior quadrant of the tympanic membrane. Likewise, the oversized perforations, the cholesteatomas, and the granulation tissue may cause ossicu-

lar destructions and, consequently, significant hearing damage [39–41]. The long-term active inflammatory processes [42] of the middle ear may course with tympanosclerosis with fibrosis of the ossicular chain.

Costa et al. [43] studied specifically the ossicular pathology of 132 temporal human bones with COM and pathologies in 91, 6% of the subjects. These changes varied from simply the erosion of the ossicular cortical to complete destruction. According to the authors, the ossicular damage caused by the pathological process was incus (91%), followed by the stapes (69, 4%) and the malleus (54, 2%).

Surprisingly, the comparative analysis of the temporal bones with and without perforations of the tympanic membrane indicated that the groups were similar concerning the ossicular pathology (prevalence, localization, and mainly damaged ossicles). These findings again call our attention to the large number of COM cases that might be underdiagnosed. In this context, the correct diagnosis can only be confirmed through an exploratory tympanostomy. This procedure is an irreplaceable tool for these patients once all irreversible changes in the middle ear are not evident.

Thus, the exploratory tympanostomy is added to the propaedeutic and therapeutical resources of the otologist as a minor surgical procedure [44]. It is safe and extremely useful because it allows the direct visualization of the middle ear, revealing, confirming, and frequently treating pathological conditions. The accurate knowledge of the nature and extension of the lesions will then determine the following surgical steps.

The numbers are still similar when we compare these findings with clinical studies concerning the ossicular changes. Austin [45] noticed the interruption of the ossicular chain in 50% of his patients that were submitted to tympanoplasty because of COM. Tos [46] found interruption in the ossicular chain in 50% of the ears that underwent surgery dry and in 80% of the surgery patients with otorrhea. Sadéand Halevy [37], when revising their cases of tympanoplasties and tympanomastoidectomies, observed lesions in the ossicular chain in 84% of the ears with CCOM and 83% of the ears with NCCOM. We must emphasize that these studies, as eminently clinical, functionally classified the ossicular pathologies. Small erosions in the ossicular surfaces were probably not considered. Thus, only ossicular in discontinuity, destroyed, or amputated were considered pathological.

We believe the ossicular defects result from active processes of bone reabsorption and not ossicular necrosis. This theory presupposes the presence and participation of immune cells in the mechanisms of demineralization, erosion, and destruction of the bone. An ossicle without blood supply can remain in situ for several years without suffering the reabsorption process, as we can testify in the ossicular reconstruction techniques. The mechanism through which bone reabsorption occurs in the chronic otitis media is not entirely understood [47].

Ruedi [12] and Tumarkin [48] suggested that bone reabsorption would happen because of the pressure done by the cholesteatomas over the ossicular surface. Sadé and Berco [38] and Thomsen [49] noticed that the eroded ossiculars were invariably surrounded by an inflammatory reaction and suggested that the inflammation was the cause of the ossicular reabsorption. It has demonstrated that the tissue of the granulation adjacent to the ossicles is capable of producing a variety of enzymes and mediators that accelerate the ossicular reabsorption. These include lysosomal enzymes that accelerate ossicular reabsorption. However, the dominant cell in the process of bone reabsorption is still controversial. While some studies reveal the presence of osteoclasts in bone destruction, others point to the mononuclear cells as the responsible ones for the situations. There is evidence that mastocytes function in the mechanisms of bone reabsorption because these cells increase in several pathologies like, for example, osteoporosis and chronic periodontal destructive disease.

Pathogenesis of Non-Cholesteatomatous Chronic Otitis Media

Considering such a complex disease, with so many faces and a high level of complexity, searching for the only trigger of this series of events, its pathological results, and multiple clinical consequences seems an unachievable task. In other words, our opinion is that searching for a unique explanation for a disease with so many facets and nuances could be seen as exaggerated optimism or scientific naiveness [5].

On the other hand, a brief (but careful) review of the medical literature shows that this theme has been approached two ways equally unsatisfying: the unsuitable simplification (which ignores controversial evidences and elects the acute necrotizing otitis media as the starting point of all of those processes) and the inconsistent generalization (which hides a number of frailties and many hypotheses incompletely tested under the broad name “multifactorial”).

Perhaps, many restrictions to the discussion about the pathogenesis of this disease are imposed by vices originated by either, and in this case, objective but distorted definitions or excluding didactic classifications (also known as “working” classifications). An additional complicating factor in this sense is the difficulty of selecting specific clinical and pathological characteristics in this complex realm that allow the development of representative subgroups of the active disease, sequels, and complications. Besides that, the natural history of the disease conspires against our intention to dispose of it in those subgroups and transits freely among those stages, going through courses that are not necessarily unidirectional. Thus, as the active disease relapses for long periods, the silent sequel may emerge in outbreaks that get more and more acute and, at times, very close to complications.

Some proposed triggers for developing the chronic inflammatory process in the middle ear are the suppurative acute otitis media (repetitive or inappropriately treated), the necrotizing ones, the temporal traumas, and the insidious infections of the upper airways. It is widely spread that these situations can be molded negatively by various unfavorable social, geographical, and economic co-factors.

Whatever origin they might have, the pathology of the TM acts as open doors to middle ear cleft contamination. These ways of contamination include fundamentally one intrinsic route (nasopharynx—Eustachian tube—middle ear) and another extrinsic (external ear, middle ear cleft—middle ear). In the first model, leaking and loss of middle ear pneumatic space (usually guaranteed by the integrity of the tympanic membrane) facilitate the aspiration of contaminated secretions of the nasopharynx (through the eustachian tube) to the middle ear. In the second one, the direct infection of the middle ear through externally contaminated fluids is a joint event and obviously facilitated by small and medium perforations.

Contrariwise to what has been exposed in earlier paragraphs, daily practice shows us that these triggering situations (acute otitis media, recurrent acute otitis media, and trauma) are infrequent events. Nevertheless, otorhinolaryngological clinics care daily for many patients with tympanic perforations. In other words, it seems that the mechanisms traditionally related to the origin of those perforations (that is, causes) are identified in a much smaller proportion daily than the number of tympanic perforations presented to us (that is, the consequences). Given this paradox, it is possible to conclude that there must be other mechanisms responsible for the generation of these alterations, or we are faced with a bizarre "missing link" in otitis media pathogenesis.

Our studies supply sufficient information to reject missing links, considering additional explanations in the pathogenesis of NCCOM. This way, the transition between acute and chronic cases has been explained through predominantly "explosive" mechanisms, that is, any sudden inflammatory or traumatic events that befall a primarily rigid structure. The rupture of the intratympanic homeostasis will mold a series of histological transformations in the mucosa layer of the middle ear, which will modify proportionally according to future aggressions.

Adopting this acute-chronic express or "direct" model of pathogenesis leaves out a series of situations commonly witnessed by the otorhinolaryngologist. Historically, some of these events (tympanic retractions, atelectasis, and adhesive otitis media) have been inexplicably set aside in this discussion. Still, the chronic silent otitis media (along with its long list of clinical and pathological explanations) have been disregarded, not due to negligence, but due to a lack of information and technological resources for correct identification and analysis. New studies focused on the specific characteristics of these conditions may provide the necessary information for elaborating more realistic and operational concepts of NCCOM. For many years, we have seen two entities being studied as if they were different conditions: otitis with effusion (serous, secretory, and acute) and cholesteatoma. Studies have brought light to several questions related to etiopathogenesis, diagnosis, and treatment of those conditions. Notwithstanding, it is necessary to establish the right connections between these two extremes.

Since 1987, we have spent much of our time researching the pathological profile and the pathogenesis of NCCOM. The results of our studies, allied to practice, point to NCCOM as a reference in the chapter related to inflammatory processes in the middle ear. Perhaps, it acts as a "link" between the most incipient and advanced forms of otitis media. In short, at one extremity (the beginning), there are acute, serous, and secretory otitis media cases. On another end, there are cholesteatomas. Finally, we can understand all the pathology described in this chapter, conceptually as NCCOM, being the link between both.

The tympanic perforation in that context is merely one detail more in the realm of the irreversible tissue alterations that comprehend this prevailing disease. We point out that if a simple tympanic continuity solution (always) represented a severe medical problem, we, "diligent" and zealous doctors, would undoubtedly prevent our patients from being introduced, all over the world, millions of ventilation tubes every year. So, one more time, an apparent paradox takes us to one more question: what is the actual morbidity of the tympanic perforations, since a great deal of them is caused by us? The answer to this question involves a series of considerations about the dimension and the localization of the perforation, the degree of the hearing loss, and the intratympanic instability directly related to it, and obviously to its impact on the quality of life of the patient in question.

Analyzing this subject more technically, Costa et al. [2], while studying temporal bones of humans who had chronic otitis media, noted that besides the activity and aggressivity of the pathological findings revealed in the interior of the middle ear cleft, regardless of the tympanic integrity, some of the temporal bones with perforated tympanic membrane presented inflammatory profiles less salient and with well-established sequels (tympanosclerosis and bone neoformation). In light of these findings, the authors stated that, in these cases, the tympanic perforation might have worked as a natural ventilation tube, allowing the aeration of the middle ear and, thus, collaborating with the deceleration and stabilization of the inflammatory process.

A broad view of this scenario (on one extremity, the ventilation tubes and the small perforation in the injured ear, and on the other, the secretory ears and fast lanes to migration of

epithelial tissue to the middle ear), we conclude the existence of two types of perforation: the solution perforation and the problem perforation. The former soothes, decelerates, or solves the intratympanic inflammatory events, while the latter clearly emphasizes them.

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