Otitis Media with Effusion / Secretory Otitis Media

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Definitions

The inflammatory processes of the middle ear mucosa can be named in several ways. Denominations are frequently confused and the understanding of the pathophysiological processes can be compromised if a definition is not very well established.

Regarding to the chronic inflammation of the mucosa with an intact tympanic membrane (TM), two terms are generally used: *otitis media with effusion* (OME) and *secretory otitis media* (SOM). Superficial analysis can interpret these as two terms as a definition of the same situation. In fact, they have a light but significant difference.

Otitis media with effusion is defined by the *Clinical practice guideline: otitis media with effusion* of 2004 as the presence of fluid in the middle ear without signs or symptoms of acute infection. This definition does not indicate the type of pathological condition or any time limit.

On the other hand, (according to the Brazilian consensus on otitis media,1999) secretory otitis media is the term used to define the chronic inflammatory process of the middle ear mucosa characterized by the persistence of fluid in the tympanic cavity for more than 3 months. Thus this term refers to a state of hypersecretory metaplasia that produces seromucous secretion that fills the middle ear lumen in conditions of hypoventilation of the mucosal lining.

From the first definition, we admit that the serous fluid that persists in the middle ear a few days after an acute episode of otitis media will be seen in the same context as a mucoid secretion that is produced by a chronically inflamed mucosa and lasts for several months. In fact, even an acute episode, if it is asymptomatic, could be interpreted as a case of OME. So, it is reasonable to say that the definition of OME can relate to quite different inflammatory stages of otitis media, with different therapeutic approaches.

Generally speaking, with exception of the acute symptomatic episode, the only situation of fluid presence in the middle ear that demands any concern is, as we will see later, when the effusion lasts for more than 3 months (**Figure 1)**. This is because most of the time, this precludes a chronic and hardly reversible inflammatory state of the mucosal lining, with functional relevance. For this reason, we prefer to deal with the label SOM and its definition before mentioned.

However, in this chapter, since the term OME is accepted worldwide, we will use both concepts in different occasions. The graphic below represents the differences in evolution behavior of the inflammatory process in different types of otitis media according to their commonly accepted definitions.

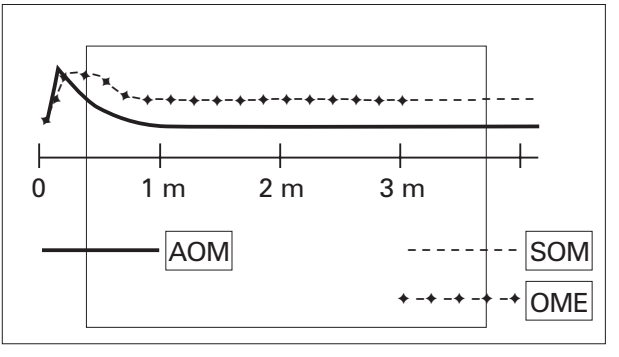

Figure 1. Evolution in time (months) of inflammatory process in different types of otitis media

Epidemiology

OME is a quite prevalent disease in younger children. This prevalence can vary according to age (greater between 2 and 5 years of age), season of the year (more frequently seen in winter), race (Indians, Africans, aboriginals and Eskimos are more prone), day care, breast-feeding and genetic factors.

Pathogenesis

The mucosal lining of the middle ear is of respiratory type. It has a *lamina propria* with submucous glands lined by columnar pseudo-stratified ciliated epithelium that contains also globet cels. This mucous architecture can be appreciated only in the Eustachian tube and the far most anterior aspect of the tympanic cleft, the rest being covered by an almost monolayered non-ciliated mucoperiosteum. This respiratory epithelium is responsible for gases exchanges between the blood and the air. The gas exchanges is fundamental for the cellular integrity and the air that fills the middle ear lumen is provided by the Eustachian tube that opens periodically by action of the palatal muscles during deglutition, thus communicating the nasopharynx to the middle ear space.

The opening of the Eustachian tube permits a good gas exchange by the mucosa and also equilibrates the middle ear air pressure with the external medium one. As already_mentioned, it is essential for the integrity of the epithelium, but also for a perfect function of the tympano-ossicular chain. When the vibratory sound energy arrives to the external surface of the tympanic membrane (TM), it begins to vibrate in consonance with that energy. For the TM to vibrate adequately, there must be a perfect equilibrium between the air pressure of its both sides. Any difference between these two pressures can lead to displacement and stretching of the membrane, and reduce its movements, altering the sound transmission mechanisms.

In addition, the lack of a good aeration of the middle ear causes inflammatory

changes in the mucosal lining. First, edema and transudation is caused by the action of the negative pressure on the mucosal surface, filling the middle ear spaces with serous fluid. This is followed by a neutrophilic infiltration and exsudation. If the mechanisms that promoted the Eustachian tubal dysfunction have a chronic nature, the inflammatory process also tends to be chronic, characterized by a lymphocitic and monocytic infiltrate,and metaplasia of the mucosa, with proliferation and hyperfunction of the globet cells and submucosal glands. If these changes last for more than 3 months, they determine the picture of SOM.

The Eustachian tube dysfunction is the most implicated factor in the genesis of otitis media. The most common example of this is the blockage by adenoid hypertrophy. However, several other factors can contribute to the onset or the persistence of this pathologic condition, as depicted in the diagram below. Allergy, for example, can be implicated either directly or indirectly as a pathogenic agent in SOM/OME. Some papers point to the presence of elements of the allergic response in the fluid collected from OME patients as an indicator of the role of the middle ear as generator of an allergic reaction. On the other hand, allergic rhinitis can contribute to Eustachian tube blockage by edema, thus playing an indirect role in the origin of the middle ear inflammation. Besides, the so called Toynbee effect can be provoked by deglutition when the nose is obstructed by allergic rhinitis or other cause of nasal obstruction, promoting, instead of equilibration of pressure, the suction of air from the tympanic cavity, aggravating even more the condition of negative pressure. Regardless if whether allergy is a direct or indirect causative factor of OME/SOM, it is certain that atopic children are much more predisposed to otitis media than non-allergic.

Etiopathogenesis of otitis media

Infection is another factor frequently implicated. Even though the studies show that OME or SOM fluids are sterile in the majority of cases, the finding of bacteria in some instances can justify, for some authors, the theory that the chronic state is maintained by this infection. Finally, gastro-esophageal reflux has been demonstrated to possibly have an important role in the pathogenesis of some cases of OME/SOM. The already classic works of Tasker et al. in 2002 showed the presence of pepsin and pepsinogen protein in the middle ear content in cases of OME. It is doubtful if passive smoking and supine position during feeding can participate in the genesis of these inflammations.

Various inflammation markers are involved in the pathologic processes that originate OME/SOM, like tumor necrosis factor, superoxide dismutase, interleukins 1, 6, 8 and 10, nitric oxid and other mediators. They seem to participate in various stages of the inflammatory reaction, stimulating metaplasia, mucin production and cell proliferation.

Signs and symptoms

Many cases of OME/SOM are asymptomatic, and the diagnosis is made by otoscopy during otolaryngologic examination for other purposes. When any symptom is present, the main one is hearing loss, although this complaint can be unnoticed in small children. The other clinical manifestation, less present, but

more noticeable is recurrent acute otitis media, with episodes of ear pain with or without hyperthermia. Besides the otologic symptoms, there may be respiratory complaints related more to pathogenic factors than to the otitis itself, like nasal obstruction, snoring, oral breathing, etc.

Diagnosis is usually made by physical examination. Otoscopy shows varying degrees of TM retraction and fluid in the middle ear. Pneumotoscopy may be essential in small children. According to the definition of OME, these findings are sufficient for diagnosis, regardless of the duration. For the label of SOM, at least 3 moths of observation are necessary.

Audiometry is fundamental if one is to evaluate the degree of functional damage, which can be decisive in establishing the therapeutic measures. This function disorder can vary widely, from no loss at all to a moderate degree conductive deficit, more pronounced at the low frequencies. Impedanciometry confirms Eustachian tube dysfunction (tympanogram type C) or the presence of middle ear effusion (tympanogram type B). However, it is not generally needed for the diagnosis, even though, in difficult otoscopy cases, it can be quite useful.

Other examinations (radiological, cytological, serologic, endoscopic etc) can be used to evaluate predisposing factors.

Treatment

Three types of measures can be used to manage OME/SOM: those directed to the predisposing factors, those that aim to revert the inflammatory reaction of the mucosa, and those destined to mechanically drain and ventilate the middle ear.

Among the first type, many different therapeutic choices are applicable depending on the predisposing factor involved. In the case of adenoid hypertrophy, adenoidectomy is indicated, with very good results. When gastro-esophageal reflux exists, one must control it by means of postural cares, diet and/or drugs. Allergic rhinitis must be treated as well as food allergy, when present. Attention shall be paid to passive smoke and other environmental factors.

To act directly on the inflammation, the most used treatment is oral steroids, normally administrated for 14 to 21 days in a decreasing scheme, despite the lack of definitive scientific basis for its employment and its low benefit in a long-term basis. Cortico-steroids reduce not only the edema but also the metaplasia, prostaglandins production and the viscosity of the effusion. The resolution of edema in the region of the Eustachian tube, facilitates its opening and, therefore, the natural drainage and ventilation of the tympanic cavity. Antibiotics are used by some practitioners as a way to combat mucosal infection, with some good short-term responses, but, again, it has little or no long-term effects.

Finally, the ventilation tube insertion through tympanostomy is indicated when the clinical measures fail. It can be done with or without adenoidectomy depending on the participation of the adenoids on Eustachian tube obstruction. There are various types of tubes, which are built for short- or long-term permanence on the TM. The decision of what type is to be used is based on several factors, including type of secretion, previous tube insertions, social and psychological conditions and individual preferences.

The indication of any kind of treatment is based on the two major clinical

manifestations: hearing loss and recurrent acute otitis media. If there are not frequent acute episodes and the audiometry shows no important deficit, no treatment is indicated other than periodic and rigid observation until the effusion disappears spontaneously. In this respect, there is a fundamental difference between OME and SOM. As the term SOM applies to an established chronic inflammation, since there is any relevant symptoms, there is a need for treatment. Conversly, OME can represent either sub-acute or chronic disease, or even a resolution stage of an acute infection. It is clear that these distinct situations deserve distinct therapeutic approaches. Sub-acute or post-acute otitis resolution phase does not need any treatment even if symptomatic. According to the *Clinical Practice Guideline*, treatment is needed only after the minimal period of 3 months of effusion persistence (when the process is thought to be chronic). Thus, one can say that OME is of clinical relevance only if it turns into SOM.

These guidelines propose a very practical protocol of treatment of OME that can be summarized as follows: once the diagnosis is made, one should observe the patient for 3 months during which no treatment is needed. After that period, hearing is tested. If there is no important hearing loss, a 3- to 6-month interval re-examination must be conducted until the effusion is no longer present, any significant hearing loss appears or any structural changes in the TM is identified. In those cases, ventilation tube insertion should the first choice treatment, together with adenoidectomy if there is significant hypertrophy of the adenoids. If OME recurs after tube elimination, another tube should be inserted and adenoids removed regardless their size. A permanent tympanostomy tube may be needed in more refractory cases, when no perspective of Eustachian tube function recovery exists. This is the case, for example, in cleft palate patients.

Recommended readings

1. Rosenfeld RM, Culpepper L, Doyle KJ et al. Clinical practice guideline: otitis media with effusion. Otolaryngol Head Neck Surg 2004;130(5 suppl.):S95-118.
2. Campos CH, Oliveira JÁ, Endo L et al. Consenso sobre otites médias. Rev Bras ORL 1999;65(1 suppl.):14.
3. Caldas Neto S. Otite média secretora. In: Caldas N, Caldas S, Sih T, eds. Otologia e Audiologia em Pediatria. Rio de Janeiro, Revinter, 1999:58-62
4. Tasker A, Dettmar PW, Panetti M, Koufman JA, Birchall JP. Reflux of gastric juice and glue ear in children. Lancet 2002;359:493.
5. Tasker A, Dettmar PW, Panetti M, Koufman JA, Birchall JP. Is gastric reflux a cause of otitis media with effusion in children? Laryngoscope 2002;112:1930-4.
6. Ryan AF, Jung TT, Juhn SK, Li JD, Andalibi A, Lin J, Bakaletz LO, Post CJ, Ehrlich GD. Recent Advances in Otitis Media. 4B. Biochemestry. Ann Otol Rhinol Laryngol 2005;194:50-5.