SUMMARY

Chronic otomastoiditis is an inflammatory disorder of the antro-mastoidian cell type system with an evolution over three months. This pathological entity has a higher frequency in populations with a low socio-economic level and a poor medical system, but although it is becoming increasingly rare with the overall development of modern society, the nature of the injuries ranging from loco-regional events to functional manifestations with impaired hearing function and severity of complications, requires knowledge of major clinical, paraclinical diagnostic elements, and to proceed with the initiation of a proper treatment which prevents their installation. The authors intend to look over the main anatomoclinical forms of the chronic otomastoiditis and to underline some essential clinical and paraclinical elements important in the diagnosis of this pathology, as well as highlighting the complications that shadow modern therapeutic developments that led to a decrease in the incidence of chronic mastoiditis.

INTRODUCTION

Otomastoiditis represents the inflammation of the middle ear mucosa and mastoid cells. Most often, this occurs as a complication of a defectively treated medium chronic otitis. Before the era of antibiotics usage, about 50% of the medium otitis evolved towards acute otomastoiditis, associated or not with different intracranial complications which often required surgery [1]. Mastoiditis is divided in terms of clinical evolution in acute with an evolution under three weeks, subacute if the
evolution varies between three and twelve weeks and chronic, the latter representing the persistent or recurrent inflammation of the middle ear and mastoid area for a minimum of twelve weeks [3].

Mastoiditis appears as the extension of an inflammatory process of the middle ear area in the pneumatic mastoid cellular system through aditus ad antrum. The main contributing factors include marked pathogen virulence of the germ which causes otitis, for example the pneumococcc mucous secretion being credited as being extremely osteophile, the insufficient drainage of the purulent collection from the eardrum level, the insufficient treatment of the acute otitis, the patient’s age and the history (muscular dystrophy, diabetes, AIDS) and the level of pneumatization of the mastoid that can facilitate its rapid infection.

It has been noted in patients with a history of suppurative otitis media that they present a higher presence of sclerotic mastoiditis with a lower level of pneumatization degree in comparison to healthy subjects. This observation is based on two possible theories: the hereditary theory provides that patients with a low pneumatized mastoid bone are susceptible to suffer from a suppurative otitis media and the environmental theory which attests that suppurative otitis media leads to allow pneumatization of the mastoid [4, 5].

There is plenty of evidence to support the idea that chronic inflammation during childhood can induce bone formation at middle ear level and mastoid bone, with the decrease in size of the pneumatized cells, so Shatz and Sade have thus measured the distance between the lateral sinus and the external auditory canal, discovering a lower value in patients with sclerotic mastoid bone[6].

From an anatomopathologic point of view, acute mastoid bone lesions start with the hyperemia of the mucosa, serous mucosa at mastoid cells level, thickened mucous congestion, vasodilatation and congestion of the bone vessels. Osteitis occurs subsequently, which may lead to the destruction of the intercellular septums and to the formation of bone attachment in the necrotizing forms. An abscess or mastoid empyema most frequently occurs, and if not drained, it may extend endocranial or externally.In the chronic otomastoiditis form we find antroatical lesions of the rarified osteitis type with colestetoma or bone attachment, fungal or bacterial collections, sometimes polyps or condensing osteitis often located at the periphery. These lesions represent a dangerous form of defense by the presence of superficial osteocondensant lesions and deep bone lesion, with the risk of spreading the infection towards the skullbase by crossing the internal cortex.

Pathological lesions can leave their mark on the middle ear structures and their surrounding areas so that we can find tympanic membrane lesions characterized by the presence of healed perforations observed by the presence of a cicatrical membrane or by areas with sclerotic material deposits of the eardrum or calcifications in its thickness. We can also find free tympanic perforations that in certain conditions may lead to the adhesion of its edges to the medial wall of the tympanic cavity creating spontaneous tympanoplasty or rarely marginal total tympanic perforations [4].

The suppurative process also leads to lesions relating to the continuity or mobility of the ossicular chain. In terms of its continuity, the most frequent lesion is represented by the osteolysis of the descending branch of the incus, which is incriminated by its poor structure and vascularization [7]. Ossicular chain mobility is affected by fibrous-like attic lesions with incudomalleolar joint immobilization, the presence of fibrous clamps between the tip of the lower part of the handle of the malleus and promontory of the tympanic cavity, fibrous lesions which may block the oval window niche and stapes or even block the entire ossicular chain through its absorption in a fibrin-rich exudate which is strengthening among ossicular chain part and on the walls of the tympanic cavity [4].

Postotitic sequelae include lesions of the middle ear mucosal passing through various stages from congestion and swelling of the mucous membrane associated with the presence of a fibrin-rich exudate at middle ear level. This process is often reversible, with restitutio ad integrum in case of a functional Eustachian tube.

The following stage consists into the appearance of a neoformation conjunctive tissue with infiltration of inflammatory-type and partial necrosis of the mucosa associated with a process of fibrous conjunctive reorganization of the middle ear exudate. It is a long time process, with no tendency of healing. The last stage is the tympanosclerosis, with the mucosa reverting to normal and the development of the sclerotic retractile lesions, atrophy and calcification of the submucosa. There were some cases of bone destruction and bone neoformation with metaplasia of the cylindrical epithelium of the mucosa in the squamous epithelium keratinized, with a tendency to desquamation and formation of small cholesteatoma [8].

Last but not least Eustachian tube lesions have an increased importance in chronical ear affections with closed eardrum whose final result is fibro-adhesive otitis.

From a functional point of view in case of middle ear suppurations we find a conductive hearing loss and also a sensorineural hearing loss [9].

**Materials and Method**

Suppurative otomastoiditis is an inflammatory disorder of the antromastoidian cell type system through a chronic bacterial process.

It can be a complication of a suppurated medium otitis or constitutes the chronicity of an acute otomastoiditis.

The antromastoidian region can be infected through three ways: tubal-the most common way of spreading, through the tympanic membrane (trauma or pre-existing eardrum perforations) and the hematogene path which is seldomly encountered.

Chronic otomastoiditis may be poor in terms of clinical signs. Anatomic-clinical forms are represented by hyperergic forms which consist of chronic otomastoiditis in which we find the disappearance of the mastoid cells, with the preservation of a small antrum or chronic otomastoiditis with a
tumoral aspect in which we find a hyperplastic extensively granulomatous process [10].

The erosive type implies a normal mastoid pneumatic system or excessively developed prior to the supplicative process, a local hypoergic reaction and a bacterial virulence with histolytic action [9]. Given the topographic location of the inflammatory process, we find clinic forms of antritis, affecting exclusively the antrum, respectively the antro-cellulite which affects the entire cellular mastoid system.

The symptomatology of the chronic mastoiditis is clinically "deleted" and englobes the otorrhea with purulent or sanguineous ear discharge, fetid, ecdrum perforation at the level of the Shrapnell membrane or in the posterosuperior part, marginal, which can assign atical wall erosion. Ear secretions aspiration leads to their easy recurrence, representing the mastoid reservoir sign [9].

Pure tone audiometry shows a conductive hearing loss or mixed hearing loss. Sensorineural hearing loss designates the complication through the labyrinthine fistula.

Hematologically there are no significant changes, but when they exist, they may indicate a specific infection, acute exacerbation of a chronic inflammatory process or the presence of complications. Bacteriology identifies common germs, more common Pseudomonas aeruginosa and Colibacillus.

From an imagistic point of view, the options include radiological examinations in various projections - Schuller for mastoid, Chausse III for the tympanic cavity and Stenvers for the temporal bone - as well as computed tomography which exposes osteitic lesions including multiple cell groups, with the formation of the geodes. Cellular septums which disappear have an irregular aspect. The edges of the osteolysis centres are irregular, and sometimes may present small attachments on the inside.

The topography of the middle ear at skull base level explains the type, extension and the complications encountered in the suppurations in these areas [9].

According to the direction of propagation of complications, they classify into external, cervical, intra-bone (intra-temporal or intrapetrous), endocranial and general ones.

External complications include external otitis, retroauricular adenitis, retroauricular abscess, Gelle fistula. Retroauricular abscess represents the most common complication of mastoiditis [11]. The infection spreads from mastoid level into the subperial space through direct extension or due to the erosion of the mastoid cortex or through venous extension. Soft tissues related to the abscess are callous, congested, fluctuant. Tissue edema and abscess push the ear’s pavilion inferior and laterally. In early stages when the abscess fluctuation is not obvious can used ultrasonography or imaging examination of retroauricular fluctuation to highlight the presence of air or abscess within its capsule. This complication involves the immediate drainage of the collection, with the association of mastoidectomy [11]. Gelle fistula or the transepal fistulation is characterized by the appearance of a fistula at the level of the upper posterior meatal wall. This can be evidenced by the retropavilion compression which produces the pus evacuation from the fistula. The collapse of the postero-superior wall of the tube is suggestive for mastoid osteitis. The cervical complications include Bezold cervical mastoiditis which is the apical exteriorization of a purulent collection along the sternocleidomastoidian muscle sheath or beneath it, the Mouret jugulo-gastric otomastoiditis, which has its starting point at subternal cells level and evolves in a laterocervical mode into the sheath of the posterior belly of the digastic muscle. This collection is associated with torticolis and can be expand to the level of the posterior foramen lacerum (affecting nerves IX, X, XI), erodes the jugular bulb and it can determine meningitis at the level of the posterior cranial fossa. The posterior cervical or occipital exteriorization is a serious complication with possibility of thrombosis of the mastoidian emissary vein, then to the sigmoid sinus and perisinusal abscess [9].

There are multiple complications which can include:
- Osteitis or osteomyelitis of the temporal bone;
- Temporo-zygomatic otomastoiditis, due to the presence of a cellular group in the posterior zygomatic root. The supplicative process can extend in depth under the aponeurosis of the temporal muscle. Clinically, we find trismus in case of deep involvement or superficial inflammatory edema and palpebral edema in the superficial form;
- Facial paralysis that may result from chronic mastoiditis with or without cholesteatoma. The infection affects the facial nerve due to a congenital splitting of the Fallopian bone tube or due to the erosion of the granulomatous tissue or to the cholesteatoma; the persistence of the inflammatory edema can lead to axonotmesis; in case of damage of the nerve within the chronic otomastoiditis without cholesteatoma, usually, the damage occurs in the horizontal portion of the facial nerve, near the stapes, in which case the clinic evolution of paralysis can be long with a stepwise clinical progression [12]; in the case of cholesteatoma, the erosion of horizontal Fallopian channel is extensive. An erosive cholesteatoma can expose the facial nerve anywhere at the temporal bone level, the installation paralysis being gradual in this case;
- Cochlear fistula with sensorineural hearing loss;
- Acute supplicative labyrinthitis whose diagnosis is clinically and includes tinnitus and dizziness which progresses rapidly in vertigo, pallor, diaphoresis, nausea and vomiting;
- Petroitis which occurs in case of hyperpneumatized mastoid starting spontaneously or after the curettage of the perilabyrinthine posterior cells; they are frequently encountered after surgery and postoperatively is characterized by persistent subfebrility, weight loss, headache and large amount of otorrhea. Certification of deepen osteitic lesions at the level of the temporal bone is evident in the appearance of Gradenigo syndrome, consisting in the paralysis of nerve VI (convergent strabismus with diplopia), trigeminal neuralgia (algea dental and maxillary) and otorrhea.
Mastoid Chausse III radiography highlights the bone reduction on an hyperpneumatized apex. The prognosis of this complication is severe due to the difficult recognition, the risk of meningitis and difficult surgical approach [9];

- Petrous apicitis which appears at the petrous apex level is rarely due to low prevalence of the pneumatization at this level. In terms of pathophysiology we find intercellular septum destruction due to an infectious process or we can find granulomatous tissue and osteitic lesions found in chronic mastoiditis;

- In seldom cases the cholesteatoma can be found also in apex. CT and MRI examinations support the diagnosis, MRI being able to make the difference between marrow, spinal fluid and mucus, in case of opacification of the apex. Clinical manifestations include the syndrome described by Gardenigo in 1904, namely retroorbital pain, nerve VI paralysis and otorhea [13];

- Endocranial complications consist of meningitis (the most frequent endocranial complication), the lateral sinus thrombosis, extradural abscess (located between the dura and the cortical inner), subdural abscess (located between the dura and the arachnoid), brain suppuration or brain abscess, cerebellum abscess and otitic hydrocephalus.

Endocranial complications are manifested by a symptomatology which consists in Bergmann syndromes, as intracranial hypertension syndrome (with headache, vomiting, tachycardia, drowsiness, seizures, signs of meningal irritation, mental disorders, respiratory, ocular), suppurative syndrome clinically manifested by motor disorders (seizure crisis paralysis, paresis, impaired sensory and sensitive locator syndrome clinically manifested by motor disorders and laboratory signs manifested by fever, fatigue, mood disturbances, gastrointestinal disorders and laboratory signs manifested by the erythrocyte sedimentation rate increase, leukocytosis with polymorphonuclear, left transfer of the Arneth formula and locator syndrome clinically manifested by motor disorders (seizure crisis paralysis, paresis, impaired sensory and sensitive or obvious radiological signs to PEG, cerebral angiography, CT, MRI [9]).

General complications include septicemia with otitic origin.

**DISCUSSION**

Chronic otomastoiditis is a disease with a long, latent evolution, most often with a clinically "deleted" symptomatology manifested by otoreic or suppurrative syndrome. Note that in the aspiration we find the sign of the mastoid reservoir through which purulent secretion are easily recollected after the procedure, this fact pleading for chronic otomastoiditis. First, patients often feel hypoacusia or its aggravation and the pure tone audiogram will highlight a conductive hearing loss without additional sensorineural component. In case of cochlear involvement we find sensorineural hearing loss. Cochlear functional status is appreciated through the status of the bone threshold -known as the so called cochlear reserve-considered good when bone transmission is situated between 0 and 30 dB in pure tone audiogram [9]. Cochlear impairment methods include the following mechanisms:

- The enhancement or complication of the inflammatory process which may lead to spontaneous unilateral cochlearization;
- Intracochlear hemorrhage;
- Perilymphatic fistulization;
- Iatrogenic - secondary to the local antibiotherapy treatment (ipsilateral impairment) or generally (with bilateral impairment);
- Fibrosis or cochlear cicatricial ossification with the Corti organ degeneration.

An important clinical sign which warns us about cochlear lesion is the deflection of Weber test. Imagering is very important in the diagnosis of chronic otomastoiditis and consists of radiological examination in various projections, the most used being Schuller which appreciates the mastoid size, its pneumatization degree and also the position of the lateral sinus.

Computed tomography shows osteolytic, osteonecrotic lesions, with geode formation and sequestrum, as well as increased transparency images (bone rarefaction) in case of suspicion of a cholesteatoma. Imagistic exploration also reveals precious information of the position of dura mater and the lateral sinus.

Another important element for diagnosing a chronic otomastoiditis is the therapeutic sample, the lack of answer in the local therapeutic treatment pleads for chronic otomastoiditis and not for otitis.

Some unclear cases require surgical exploration and this represents the last sequence to confirm or rule out the otomastoiditis.

There are many discussions in literature about clinical forms and complications, so that antritis and antrocellulitis are considered to be clinical forms of otomastoiditis and osteomyelitis of the temporal bone, tempozygomatic mastoiditis, Bezold abscess, Mouret jugulodigastric mastoiditis, occipital lobe mastoiditis are considered complications, not clinical forms because they extend beyond the anatomic limits of the antromastoidian region.

**CONCLUSION**

Considered today as "endangered" in many specialty publications, especially in well developed countries, where the incidence of this pathological entity is low, chronic otomastoiditis should not be neglected just because of the parsimonious character of this disease, which may have clinical manifestations with unprecise evolution, but which produces major destructions at antromastoid topography level and increases the risk of complications, some of them with high mortality and morbidity. Also should not be neglected the overall functional hearing impairment, often the only symptom that is relevant for the patient which involves additional costs in terms of therapeutical management and social reintegration.
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