

# Chronic otitis media

## Complications of otitis media

### Definition:-

Otitis media (OM) is broadly defined as inflammation from any cause of the middle ear. This may involve any of the contiguous pneumatized process portions of the temporal bone such as the mastoid or petrous apex.

**chronic otitis media” (COM).** term refers to intractable pathology of greater than 3 months duration within the middle-ear system in the setting of a permanent TM defect.

If persistent otorrhea is present, the term chronic suppurative otitis media (CSOM) is applied

**TM defects include:** - retraction pockets, atelectasis, and perforations secondary to infection, trauma, or surgery, for example, tympanostomy tubes.

COM can be further subclassified into those cases with or without aural cholesteatoma

### **RISK FACTORS OF COM**

**General risk factors for otitis media include**

**mechanical obstruction of the eustachian tube** (eg, sinusitis, adenoid hypertrophy, nasopharyngeal carcinoma),

**immunodeficiency** (primary or acquired)

**ciliary dysfunction,**

**congenital midfacial anomalies** (eg, cleft palate, Down syndrome)

**gastroesophageal reflux.**

**Environmental factors** such as number of hours spent in child daycare, passive exposure to smoke, lack of breastfeeding in infancy

**Significant specific risk factors for COM include**

- history of recurrent acute otitis media
- parents with histories of COM
- Allergy has been implicated as a risk factor.
- Genetic predisposition

## MICROBIOLOGY OF COM

In CSOM isolates, aerobic and anaerobic bacteria are involved, coexisting in half of cases. The most common aerobic bacteria isolated are *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and other gram-negative bacilli, for example, *Escherichia coli*, *Proteusspecies*, and *Klebsiella species*. *Bacteroides* spp. And *Fusobacterium* spp. are the most common anaerobic bacteria isolated. Furthermore, fungi are frequently found within CSOM samples, specifically *Aspergillus*spp. and *Candida* spp. There is some speculation that fungi may result as overgrowth after initial treatment with antibiotic drops.

## PATHOGENESIS OF COM

COM is characterized by pathological findings consistent with irreversible inflammatory changes within the middle ear and mastoid. Undoubtedly, dysfunction of the Eustachian tube plays a prominent role in both AOM and COM.

Under physiologic conditions Eustachian tube is responsible for **clearance of middle ear secretions into the nasopharynx, prevention of nasopharyngeal secretions from refluxing into the middle ear, and pressure equalization between the middle ear and the external environment.**

Obstruction of the Eustachian tube results in lowering of the normally negative middle ear pressure due to increased nitrogen absorption into middle ear subepithelial mixed venous blood. This results in transudation of serous fluid into the middle ear cleft. Also, nasopharyngeal bacteria are more readily introduced into the middle ear upon tubal opening as they are in the settings of a shorter tube (eg, infant) or a perforated TM.

Once bacteria enter the middle ear via the nasopharynx or a TM defect, bacterial replication ensues within a serous effusion, an immune response is triggered, releasing immune and inflammatory mediators into the middle ear space. The hyperemia and polymorphonuclear leukocyte-dominated acute inflammatory phase gives way to a chronic phase, characterized by a shift toward mononuclear cellular mediators (eg, macrophages, plasma cells, lymphocytes), persistent edema, and granulation tissue. Furthermore, metaplasia of the middle ear epithelium may occur, converting cuboidal epithelium to a pseudostratified columnar epithelium capable of increased mucoid secretion. Granulation tissue

becomes increasingly fibrotic, eventually forming adhesions to important structures within the middle ear. This can disturb aeration of the antrum and mastoid by decreasing space between the ossicles and mucosa which separate the middle ear from the antrum. Chronic obstruction leads to irreversible changes within both bone and mucosa of these structures.

### **MIDDLE EAR ATELECTASIS AND ADHESIVE OM**

Middle ear atelectasis is a variant of COM and refers to TM retraction onto the promontory and ossicles of the middle ear. The most likely reason for this phenomenon is increased negative middle ear pressure from Eustachian tube dysfunction. It is potentially reversible and does not involve changes to the lining middle ear mucosa. Should a medially retracted TM completely obliterate the middle ear space and irreversibly replace the normal mucosa by adhering to the bony promontory, the term adhesive OM is applied.

### **CHOLESTEATOMA**

Epidermal inclusion cysts within the pneumatized portions of the temporal bone. As such, keratinizing stratified squamous epithelium is found ectopically within the middle ear, an area typified by low cuboidal epithelium. The squamous epithelium comprises the “matrix” of the cholesteatoma which rests above the “perimatrix” that contains inflamed fibrous tissue. The epithelium of cholesteatoma, while not neoplastic, is hyperproliferative, in addition to chronic inflammation these 2 factors lead to cholesteatoma growth. In established cases of COM with or without cholesteatoma, bone erosion is almost invariably present and represents the major source of morbidity. Pressure necrosis or proteolytic factor secretion by components of the cholesteatoma matrix, increased bone erosion lead to more morbidity. Cholesteatoma can be classified as either congenital or acquired. Acquired cholesteatomas are further divided into primary or secondary forms.

**Congenital Cholesteatoma.** When cholesteatoma arises behind an intact TM without history of otorrhea, the term congenital cholesteatoma is used. Congenital cholesteatomas arise from keratinizing epithelium within the middle ear cleft.

**Acquired Cholesteatoma.** Primary acquired cholesteatoma refers to cholesteatoma that arises from simple retraction of the pars flaccida. Secondary acquired cholesteatoma refers to cholesteatoma that arises in the setting of TM perforation, usually in the posterosuperior quadrant of the middle ear.

**There are 4 main theories proposed to account for acquired cholesteatoma etiopathogenesis:**

(1) **TM invagination**; the TM becomes retracted further medially into the middle ear secondary to increased negative middle ear pressure. This has been established for COM in general eustachian tube dysfunction, inflammation, TM atrophy, and poor mastoid pneumatization.

(2) **migration of epithelium through a TM perforation**; Squamous epithelium of the external auditory canal and the outer margin of the TM has the ability to migrate into the middle ear across TM perforations.

(3) **basal cell hyperplasia**; keratinizing epithelial cells of the pars flaccida could invade the normally inaccessible subepithelial space to form attic cholesteatomas.

(4) **squamous metaplasia** Chronically infected or inflamed tissues are known to undergo metaplastic transformation. The cuboidal epithelium of the middle ear may undergo transformation into keratinizing epithelium.

**Diagnosis of COM**

The diagnosis of COM with or without cholesteatoma is usually made based on:-

**History and physical examination.** 1- Evaluation should elicit prior history of middle ear disease and surgical interventions. Symptoms including hearing loss, otorrhea, otalgia, nasal obstruction, tinnitus, and vertigo may prompt the patient to seek medical attention. Of these presenting symptoms, hearing loss and otorrhea are by far the most common. CSOM presents with a profuse, viscous otorrhea that is intermittent in nature. On the other hand, patients with infected cholesteatoma present with small amounts of foul-smelling, purulent otorrhea. Otalgia usually represents a secondary external Otitis. Alternatively, pain may represent an intracranial sequela of cholesteatoma. Other symptoms of possible sequelae include bloody otorrhea in advanced disease, vertigo from a labyrinthine fistula, facial nerve paralysis, or neurologic symptoms from intracranial spread.

2- The diagnosis of COM and aural cholesteatoma can usually be made on otomicroscopic examination given that the ear is properly cleansed of debris and drainage. It is also important to evaluate the nasopharynx in

these patients, as eustachian tube dysfunction is a common cause of COM in many cases. In regard to the ear, the microscope will allow visualization of the drumhead to identify perforations, retraction pockets, cholesteatoma, and granulation tissue.

If a TM perforation is present and large enough, middle ear mucosa may be assessed for evidence of inflammation and infection; furthermore, some or all of the ossicles may be visible and assessed for erosion, fixation, or disruption. Granulation tissue is the most common finding associated with COM and is a consequence of inflammation. Sometimes polyps herald the presence of cholesteatoma; they represent granulation tissue at the junction between the cholesteatoma and eroding bone and may be seen extending as far as the external meatus in advanced cases. A positive fistula test characterized by vertigo and nystagmus suggests inner ear involvement and need for urgent intervention

**Audiometric evaluation** including air and bone thresholds as well as speech discrimination testing is imperative

**Imaging** Although imaging is usually unnecessary for uncomplicated cases of COM or cholesteatoma due to eventual surgical exposure, high-resolution computed tomography (CT) and magnetic resonance imaging (MRI) of the temporal bones may provide supplementary information

## **Management of COM**

**Medical.** Most infected perforations can be managed conservatively with topical antibiotics and regular aural toilet. A clean external meatus is needed to ensure proper drug penetration into the middle ear mucosa. The antibiotics chosen should have efficacy in eradicating *Pseudomonas aeruginosa* and *Staphylococcus aureus*, the most common pathogens in COM. In those with recurrent or chronic infections, cultures should be obtained to direct antimicrobial therapy; Systemic antibiotics may be administered according to culture and sensitivity results.

**Surgical Considerations.** When conservative medical management has failed to control COM an individual often becomes a candidate for a surgical procedure. If a surgical procedure is required, the choice of the surgical procedure depends on the nature and extent of disease. In COM without cholesteatoma, the procedure should be designed to

provide aeration of the middle ear, attic, antrum, and mastoid air cell spaces, as well as closure of the TM (**cortical mastoidectomy with tympanoplasty**)

When a surgical procedure is chosen for COM with cholesteatoma, the nature and extent of the cholesteatoma must also be kept in consideration. Cholesteatomas extending into the antrum and mastoid and those extending medially past the ossicular heads are most appropriately handled with a complete mastoidectomy (**modified radical or radical mastoidectomy**)

## **COMPLICATIONS OF COM AND CHOLESTEATOMA**

**The most common complication of COM** is

1- conductive hearing loss, typically ranging from 20 to 60 dB. This may be caused by one of many noninfectious sequelae of COM, namely TM perforation, middle-ear atelectasis, tympanosclerosis, ossicular disruption, and cholesteatoma, CSOM

2- Although conductive hearing loss predominates, infectious and inflammatory components may also be transmitted to the inner ear via the round window resulting in cochlear damage and hearing loss

3- Other important noninfectious sequelae include facial paralysis and cholesterol granuloma.

4- Infectious complications include subperiosteal abscess, mastoiditis, labyrinthitis, petrositis, and intracranial infection, eg, encephalitis, meningitis, brain parenchymal abscess, subdural empyema, sigmoid sinus thrombophlebitis, extradural abscess.

**The main complications accounting for the morbidity of cholesteatoma**

- 1- destruction of nearby bony structures. These include the ossicles, the otic capsule, facial nerve canal, tegmen tympani, and tegmen mastoideum.
- 2- Infections of cholesteatomas are also a common complication and tend to be recurrent. This results in purulent otorrhea and inflammatory damage to structures.
- 3- Conductive hearing loss typically results from erosion of the incus.

- 4- Erosion of the otic capsule, most commonly involving the lateral semicircular canal, can result in labyrinthine fistula, vertigo, or infectious sequelae such as suppurative labyrinthitis.
- 5- Fistula, labyrinthitis or cochlear erosion may result in sensorineural hearing loss.
- 6- Facial nerve paralysis may result from nerve invasion after erosion through the facial nerve canal or from infectious involvement of cholesteatoma tissue that abuts the facial nerve
- 7- Cerebrospinal fluid leakage and brain herniations can result from erosion of either tegmen.