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Cochlear Affection in CSOM: Review Article

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ABSTRACT

Background: Chronic suppurative otitis media (CSOM) refers to a long-standing inflammatory condition affecting the middle ear and/or mastoid cavity. It is also known by several other terms, including chronic otitis media, chronic mastoiditis, and chronic tympanomastoiditis. The clinical hallmark of CSOM is recurrent or persistent aural discharge (otorrhoea) lasting between two to six weeks, typically occurring through a perforated tympanic membrane. The condition often originates in childhood as a sequela of unresolved acute otitis media (AOM) with tympanic membrane rupture. Otoscopic examination may reveal a thickened, granulated middle-ear mucosa and the presence of mucosal polyps. In some instances, CSOM may be accompanied by a cholesteatoma within the middle ear space. It is important to distinguish CSOM from chronic otitis media with effusion, a separate entity characterized by the accumulation of middle ear fluid in the absence of tympanic membrane perforation or active infection. CSOM is broadly categorized into two subtypes: The tubotympanic (benign) form and the atticoantral (malignant) form—the latter, also referred to as the "dangerous type," involves the attic and posterosuperior regions of the middle ear and is associated with more serious complications**.**

Objective: This review article aimed to evaluate effect of CSOM on cochlear health.

Methods: We used Google Scholar, Science Direct, PubMed, and other internet databases for Cochlear affection, CSOM and Hearing loss. Additionally, the writers combed through relevant literature for references, however they only included researches covering the years from 2015 to 2024. Due of lack of translation-related sources, documents in languages other than English were excluded. Also, works in progress, unpublished publications, abstracts from conferences, and dissertations that did not form part of broader scientific investigations were excluded.

Conclusions: CSOM continues to represent a prevalent chronic infectious condition among children globally, transcending racial and cultural boundaries in both low- and high-income regions. This condition is associated with substantial morbidity and carries the potential for both extracranial and intracranial complications. Hearing impairment in CSOM is primarily attributed to three mechanisms: Disruption of the tympano-ossicular system's impedance-matching function, diminished protective baffle effect at the round window, and underlying cochlear pathology.

Keywords: Cochlear affection, CSOM; Hearing loss.

INTRODUCTION

The middle ear is an air-filled cavity located between the outer and inner ear, functioning primarily to transmit sound waves via the ossicles—malleus, incus, and stapes—to the inner ear. Proper aeration and pressure regulation within the middle ear are essential for its normal function and are maintained through the Eustachian tube. Eustachian tube dysfunction, seen in over 70% of patients undergoing middle ear surgery, disrupts this equilibrium, contributing to the development of middle ear diseases. One such condition is chronic suppurative otitis media (CSOM), characterized by persistent otorrhea through a perforated tympanic membrane due to chronic middle ear infection. Common risk factors for CSOM include recurrent acute otitis media, upper respiratory infections, trauma to the tympanic membrane, and poor socioeconomic conditions [1].

The pathogenesis of CSOM involves bacterial invasion of the middle ear mucosa through the perforated membrane, leading to chronic inflammation, edema, and fibrosis. Though viruses are common in otitis media, bacterial infections—often polymicrobial—predominate in CSOM. Staphylococcus aureus (particularly MRSA) is the most frequent isolate, followed by Pseudomonas aeruginosa, Proteus, Klebsiella, Bacteroides, Fusobacterium, and occasionally fungal pathogens such as Aspergillus and Candida in immunocompromised patients. CSOM can lead to multiple complications, including polyps, tympanosclerosis, labyrinthitis, and intracranial abscesses, with hearing loss being the most frequent and significant outcome. While, conductive hearing loss is typical, prolonged infection can cause toxin absorption into the cochlea, damaging hair cells and resulting in sensorineural hearing loss (SNHL) [2].

This review aimed to evaluate the effect of CSOM on cochlear health.

1. Structure and physiology of human Ear

Sound travels as longitudinal waves through physical media—gas, liquid, or solid—consisting of alternating compressions and rarefactions. These waves cause particles in the medium to vibrate parallel to the direction of wave propagation, highlighting the longitudinal nature of sound. When produced by a speaker or any sound source, these vibrations are captured by the ear and translated into electrical signals processed by the brain. Hearing enables perception, communication, and awareness of environmental cues. The human ear, a paired organ, detects sound via the cochlea—a coiled, snail-shaped structure responsible for transducing vibrations into nerve impulses through specialized hair cells. The ear operates within a range of 20–20,000 Hz via air conduction and can detect even higher frequencies via bone conduction. Anatomically

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and functionally, the ear comprises three main parts: The outer ear, middle ear, and inner ear. The inner ear is further divided into the vestibular labyrinth and the cochlea, which work together to ensure normal hearing function **(Figure 1)**.

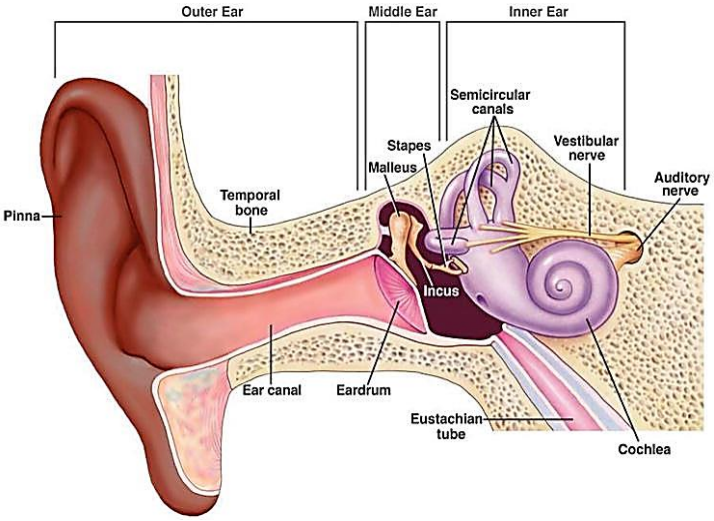

Figure (1): Structure of the mammalian ear.

2. Chronic suppurative otitis media

CSOM is defined as a prolonged inflammatory process involving the mucosal lining of the middle ear and mastoid cavity, persisting for more than two months. It is typically marked by tympanic membrane perforation and either continuous or intermittent otorrhoea, which may present as serous, mucoid, or purulent discharge from the external auditory canal. When associated with a cholesteatoma, CSOM often features a marginal or attic perforation, which may extend to involve the bony structures of the middle ear, thereby increasing the risk of serious and potentially life-threatening complications. CSOM is still a major health problem, especially in developing countries [3].

❖ Etiology of CSOM

CSOM is influenced by multiple factors including upper respiratory infections, low immunity, diabetes, poor nutrition, and low socioeconomic status. It is commonly polymicrobial (52.5%), with Proteus mirabilis and Klebsiella pneumoniae being frequent isolates. Gram-negative bacteria (59.7%) predominate, while Staphylococcus species are the most common among Gram-positive. Pseudomonas aeruginosa is the main pathogen in CSOM with cholesteatoma (66%). Risk factors include Eustachian tube obstruction, congenital anomalies, recurrent AOM, allergies, and genetic susceptibility [4].

❖ Incidence and epidemiology

CSOM frequently has its onset in early childhood and may persist into adulthood, with an estimated global burden ranging between 65 and 330 million individuals—most of whom reside in developing countries. Approximately 31 million new cases are reported each year, with children under the age of five accounting for about 22.6% of these. The condition is particularly prevalent among certain indigenous populations, such as Inuits, American Indians, and Australian Aborigines, where prevalence rates range from 7% to 46%. Intermediate prevalence levels, ranging from 1% to 6%, have been observed in areas including Africa, India, and South Korea. In Greenland, studies have shown a 14% incidence among children younger than four years.

Earlier studies in Greenland found even higher rates (19–20%) among children aged 3–8, with onset typically before age one. Risk factors include day-care attendance, maternal history of ear discharge, smoking in the home, frequent upper respiratory infections, and Inuit ethnicity. Despite ongoing cases in developed nations, data remain limited [5].

❖ Chronic suppurative otitis media (CSOM)

CSOM is a prolonged inflammatory disorder involving the middle ear and/or mastoid cavity, most frequently developing as a sequela of unresolved acute otitis media with tympanic membrane perforation. The condition is typically associated with continuous or episodic ear discharge persisting for 2 to 6 weeks. It is also referred to as chronic otitis media or chronic mastoiditis. Clinical manifestations may include mucosal hypertrophy, the development of polyps and, occasionally, cholesteatoma—a pathological squamous epithelial growth within the middle ear or mastoid spaces. CSOM differs fundamentally from chronic otitis media with effusion, where fluid is retained behind an intact eardrum in the absence of infection.

The global prevalence of CSOM ranges from 65 to 330 million, with 60% experiencing significant hearing loss. Cholesteatoma, often secondary to CSOM, is detected by otoscopy and is usually acquired (95%), though congenital forms exist. Its incidence is about 9 per 100,000 people and affects both children and adults equally [6].

❖ Types of CSOM

CSOM presents in several forms, each with distinct clinical and pathological features. Tubotympanic CSOM, also known as “safe” CSOM, is the most common type and involves a central perforation of the tympanic membrane, allowing drainage and reducing the risk of serious complications. Symptoms include intermittent mucoid discharge and mild to moderate hearing loss. It typically responds well to treatment. In contrast, Atticoantral CSOM, or “unsafe” CSOM, affects the attic and mastoid regions and is often associated with cholesteatoma and bone erosion. It can cause purulent discharge, poor aeration, and more severe hearing loss, often requiring surgical management.

Cholesteatomatous CSOM involves abnormal skin growth within the middle ear or mastoid, capable of eroding bone and damaging nearby structures. Surgical removal is essential to prevent serious

complications. Mucosal CSOM is marked by chronic inflammation of the middle ear mucosa, sometimes without perforation, and may lead to recurrent infections and hearing issues. Finally, bilateral CSOM, though less common, affects both ears and requires comprehensive evaluation due to its greater impact on hearing and quality of life. Each type of CSOM demands a tailored approach for effective management and prevention of complications [7].

❖ **Microbiology**

The leading etiology of OM is bacterial infection within the middle ear cavity. In cases of AOM, the predominant pathogens include Streptococcus pneumoniae, Haemophilus influenzae, and Moraxella catarrhalis. In contrast, Pseudomonas aeruginosa and Staphylococcus aureus are the most frequently identified aerobic organisms in CSOM, followed by other isolates such as Proteus vulgaris and Klebsiella pneumoniae **(Table 1)**.

Table 1: A list of micro-organisms isolated from CSOM patients [3]

Organisms	**Percentage isolation**
Aerobic bacteria	
Pseudomonas aeruginosa	**22–44**
Staphylococcus aureus	**17–37**
Klebsiella pneumoniae	**4–7**
Proteus mirabilis	**3–20**
Proteus vulgaris	**0.9–3**
Escherichia coli	**1–21**
Streptococcus pneumoniae	**1–3**
Acinetobacter baumanii	**1–3**
Enterobacter aerogenes	**0.9–4**
Anaerobic bacteria	
***Bacteroides* spp.**	**4–8**
***Clostridium* spp.**	**3–6**
***Prevotella* spp.**	**1–3**
Fusobacterium nucleatum	**3–4**
Fungi	
Aspergillus niger	**3–15**
Aspergillus flavus	**3–20**
Candida albicans	**0.9–23**
Candida krusei	**2–3**

Epidemiological research across various countries has revealed regional differences in the microbiological profile of CSOM. In nations such as India, Nepal, Singapore, and Nigeria, Pseudomonas aeruginosa has emerged as the most common causative agent, typically followed by Staphylococcus aureus. However, studies from Pakistan, Iran, and Saudi Arabia have reported a higher prevalence of S. aureus in CSOM cases.

These variations likely reflect geographic and population-based differences. Advanced analyses using 16S rRNA sequencing in pediatric cases revealed Pseudomonas spp. as dominant in the middle ear, while Streptococcus spp. were more abundant in tonsils. P. aeruginosa thrives in the middle ear, evading immune defences by damaging epithelium and reducing local blood flow, enabling tissue destruction and antibiotic resistance. Other anaerobic bacteria such as Bacteroides, Clostridium, and Fusobacterium spp. may also contribute to CSOM [8].

❖ **Pathophysiology of acquired cholesteatoma**

Acquired cholesteatoma typically results from Eustachian tube dysfunction and differs from congenital cholesteatoma, which is present at birth. Several theories explain its development. The most accepted is the retraction pocket theory, where negative pressure in the middle ear pulls the pars flaccida inward, forming a pocket that traps keratinizing epithelium—eventually leading to cholesteatoma. Chronic retraction and blockage of the epitympanum may result in accumulation of mucus and granulation tissue. In contrast, non-retraction pocket theories include migration of squamous epithelium through a perforated tympanic membrane (migration theory), transformation of middle ear mucosa into keratinizing epithelium due to chronic inflammation (metaplasia theory), and basal cell hyperplasia where keratinocytes proliferate and invade subepithelial spaces. Diagnosis is based on clinical signs with or without tympanic membrane perforation, otorrhea, hearing loss, and radiological findings such as ossicular or mastoid destruction on CT or MRI [9].

❖ **Classification CSOM**

CSOM is broadly categorized into two clinical types: The tubotympanic (benign or safe) type and the atticoantral (malignant or dangerous) type. The tubotympanic form predominantly involves the anterior-inferior section of the middle ear cleft and is typically associated with a central perforation of the tympanic membrane. This variant is considered less aggressive and is seldom linked to serious complications. In contrast, the atticoantral type affects the attic and posterosuperior regions of the middle ear and is marked by marginal or attic perforations. It is often complicated by cholesteatoma, granulation tissue, or osteitis, which may contribute to ossicular destruction and a heightened risk of intracranial or extracranial sequelae. Complications that arise from malignant CSOM are quite dangerous, one of the complications of malignant CSOM is facial nerve paresis, due to the growth of tympanic cholesteatoma, which is progressive, destructive, and is a hallmark of malignant CSOM [10].

❖ **Diagnosis of CSOM**

Diagnosing CSOM, especially with cholesteatoma, involves a thorough history, physical examination, and targeted investigations. Patients often present with persistent or intermittent foul-smelling, purulent or mucoid discharge and hearing loss.

Otoscopic evaluation helps identify the type and location of tympanic membrane perforation, granulation tissue, or white mass typical of cholesteatoma, particularly in the pars flaccida or anterosuperior quadrant. Inspection of the external ear, ear canal, and pre-/retroauricular areas is essential. Nasal and nasopharyngeal assessment is also important due to their anatomical connection with the ear.

Imaging studies such as CT scan of the temporal bone provide detailed information on mastoid pneumatization, ossicular erosion, and extent of cholesteatoma. MRI, especially diffusion-weighted imaging (DWI), offers high sensitivity for detecting cholesteatoma based on keratin debris restricting water diffusion. Swab cultures of ear discharge guide appropriate antibiotic therapy by identifying aerobic and anaerobic pathogens. Audiometry is used to assess hearing loss, which is typically conductive in CSOM but may also be mixed or sensorineural in advanced cases [11, 12].

Management of CSOM

Management of CSOM includes both medical and surgical approaches. Medical treatment involves aural toilet and the use of systemic and topical antibiotics targeting Pseudomonas aeruginosa and Staphylococcus aureus. Common systemic antibiotics include ciprofloxacin, amoxicillin-clavulanic acid, and cefixime, while topical options such as ciprofloxacin and ofloxacin ear drops have proven effective in reducing symptoms and improving hearing [13].

Surgical intervention is the definitive treatment, especially in cases with cholesteatoma, aiming to eradicate disease, dry the ear, and restore function. Procedures include canal wall down (CWD) and canal wall up (CWU) mastoidectomy. CWD is preferred in extensive disease with ossicular destruction and complications, creating a single cavity by removing the posterior canal wall. CWU preserves the canal wall and is suited for limited cholesteatoma with intact ossicles and good drainage. The choice of technique depends on disease extent, anatomy, and hearing status, with CWD being more common in advanced cases [14].

❖ Complications of CSOM

The emergence of complications in CSOM is influenced by multiple variables, including the anatomical site of infection, the mode of its spread, and the inherent nature of the disease. Accurate knowledge of these aspects is crucial for anticipating and managing adverse outcomes. The core pathogenic process involves the interaction between the etiological pathogen and the host's defence mechanisms. The host will respond by forming tissue edema in the narrow space between the mesotympanum and the epitympanum and in the aditus between the epitympanum and the mastoid antrum that inhibits normal aeration pathways and reduces oxygenation and vascularity [15].

Complications associated with CSOM are classified into two major groups: Extracranial (intratemporal) and intracranial. Intratemporal sequelae include mastoiditis, petrositis, inflammation of the labyrinth, facial nerve weakness, and the formation of labyrinthine fistulae. Intracranial complications may involve extradural abscesses or granulation, thrombosis of the sigmoid sinus, brain abscesses, otitic hydrocephalus, meningitis, and subdural abscesses. When such complications arise, they typically present with a rapid onset of symptoms, necessitating prompt medical intervention. Fever indicates an intracranial infectious process or extracranial cellulitis [16].

3. Effect of CSOM on hearing

❖ Hearing loss

The most prevalent consequence of CSOM is hearing impairment, which may manifest as either conductive or SNHL. Conductive hearing loss in CSOM typically results from interference with the passage of sound vibrations from the middle ear to the inner ear, often due to the presence of inflammatory exudate or pus. This fluid accumulation obstructs sound conduction pathways, and the degree of conductive loss has been found to correlate positively with the volume of middle ear effusion. CSOM is characterized by the presence of tympanic membrane perforation, which can hinder the conductance of sound to the inner ear [17].
Hearing deterioration in CSOM is closely linked to the severity of anatomical damage sustained by the middle ear components. In advanced or chronic cases, irreversible changes within the auditory cleft—such as fibrotic tissue remodelling or ossicular chain destruction—may culminate in permanent auditory deficits. Chronic infection of the middle ear causes oedema of the middle ear lining and discharge, tympanic membrane perforation and possibly ossicular chain disruption, resulting in CHL ranging from 20 to 60 dB [18].

Histopathological studies in patients with CSOM have revealed degeneration of both outer and inner hair cells, particularly within the basal turn of the cochlea. SNHL associated with CSOM is predominantly unilateral and affects high-frequency thresholds. Recent evidence suggests that bacterial toxins present in the middle ear during active infection may infiltrate the cochlear structures, contributing to inner ear damage and subsequent auditory dysfunction. These bacterial toxins can be exotoxins (proteins) produced by both Gram-positive and Gram-negative bacteria, or endotoxins (LPSs of the outer membrane of Gram-negative bacteria) [19].

Toxins produced during infection in CSOM may exert direct cytotoxic effects on cochlear hair cells, particularly in the basal turn where high-frequency sound detection occurs. Histological examinations have demonstrated considerable degeneration of both outer and inner hair cells, along with marked atrophy of the stria vascularis in this region, highlighting the potential

for significant sensorineural damage in affected individuals. The basal turn of the cochlea also demonstrated severe pathological changes that were consistent with the high-frequency SNHL in CSOM patients [20].

In CSOM, audiometric findings commonly reveal sensorineural deficits, evidenced by increased bone conduction (BC) thresholds. A threshold difference of 20 dB or more between the diseased and the normal ear has been observed at each frequency assessed, underscoring the presence of significant cochlear involvement. In a multi-centre study, 58 % of 874 patients with unilateral CSOM presented with SNHL of more than 15 dB in the affected ear [21].

An age-related trend has been observed in CSOM patients, with the prevalence of elevated bone conduction (BC) thresholds increasing progressively with age. Moreover, both the location and extent of tympanic membrane perforation have been linked to the severity of hearing impairment. Perforations situated posteriorly are associated with greater hearing loss, likely due to the diminished shielding of the round window membrane, allowing direct exposure to sound pressure waves. It was suggested that all measures for an early cure, including surgery, should be considered promptly to prevent hearing loss in CSOM patients [22].

While CSOM is capable of causing severe and even fatal complications, hearing impairment—particularly of the conductive type—remains the predominant clinical concern. This form of hearing loss has been extensively reported in existing literature. However, the potential link between CSOM and SNHL remains inconclusive. Some researchers affirm the frequent occurrence of SNHL in affected individuals, whereas others argue that the evidence supporting such an association is weak or absent. Ironically, despite the continued high prevalence of CSOM in low-resource settings, limited data are available regarding its effect on cochlear function. Addressing this gap through targeted research is essential to clarify the disease's impact on inner ear structures [23].

CSOM refers to chronic inflammation affecting the middle ear or mastoid space, distinguished by ongoing or intermittent ear discharge via a tympanic membrane perforation. Treatment strategies primarily involve medical management targeting causative aerobic and anaerobic microorganisms. When pharmacologic approaches prove ineffective, surgical procedures may be indicated to control the disease and prevent complications [4].

While several investigations have explored the relationship between CSOM and SNHL, agreement on the clinical relevance of this association remains lacking. A retrospective study examined patient records to assess the correlation between SNHL and factors including patient age, duration of ear discharge, and the presence of cholesteatoma or ossicular erosion. The study revealed a statistically significant increase in bone conduction thresholds at speech frequencies (500, 1000, 2000, and 4000 Hz) in the diseased ear compared to the unaffected side.There was a greater difference in the high frequencies than in the low frequencies (7.00 dB at 500 Hz increasing to 9.71 dB at 4000 Hz) [24].

CONCLUSION

CSOM is recognized as one of the leading chronic infectious diseases in children worldwide, with a broad impact across ethnically and geographically diverse populations in both resource-limited and industrialized settings. It is associated with notable morbidity and carries the potential for serious complications affecting both extracranial and intracranial structures. The pathophysiology of hearing loss in CSOM is multifactorial, involving impaired sound conduction due to tympano-ossicular dysfunction, reduced acoustic insulation of the round window, and possible cochlear involvement.

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