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Chronic Suppurative Otitis Media

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Introduction

Chronic suppurative otitis media (CSOM) is a common problem in developing countries and among ethnic minorities of the world. It is the commonest cause of childhood hearing loss in these populations.[1-4] It is also a disease with a propensity for causing potentially fatal intracranial complications. Early diagnosis and treatment are, therefore, essential. The condition usually follows an episode of acute suppurative otitis media following which there has been persistent ear discharge through a perforation with spread of infection to the entire middle ear cleft. It can also occur when persistent negative middle ear pressure due to Eustachian tubal block causes the tympanic membrane to form a retraction pocket which allows accumulation of desquamated epithelium with bone erosive properties.

Patients with CSOM can be diagnosed with otoscopy or otomicroscopy. Subsequently, they should be evaluated by audiometry to assess the degree and type of hearing loss and radiology. Most cases of CSOM may be treated initially with medical management. Failures of medical therapy as well as those cases with complications should be treated surgically.

In the following review, we describe the classification, epidemiology, pathogenesis, pathology, clinical features, diagnostic evaluation and treatment of CSOM.

Definition

Chronic suppurative otitis media(CSOM) is defined as a chronic inflammation of the middle ear and mastoid cavity of at least 6 weeks 'duration causing purulent ear discharge. The tympanic membrane exhibits either a perforation or a retraction pocket of the pars tensa and/ or pars flaccida.

Classification

Two popular classifications are considered here (**Table 1**). The old classification into tubotympanic and atticoantral varieties of CSOM is still used widely. A recent classification favours division into mucosal and squamous disease, with both types being either active or inactive[1]. Mucosal disease is characterized by the otoscopic appearance of a perforation of variable size involving the pars tensa of the tympanic membrane (central perforation) (**Figure 1**). In active disease, the mucosa of the middle ear appears congested and moist. Mucoid or mucopurulent ear discharge spilling over from the middle ear into the ear canal may be present.

Squamous disease is characterized by perforations or retraction pockets of the pars flaccida (attic perforation/retraction) or pars tensa (posterosuperior retraction pocket or central perforation) with squamous debris and / or cholesteatoma and granulations **(Figure 2).** Either of the 2 categories could be active (discharge present) or inactive (discharge absent). This type of disease has a tendency to erode bone and produce complications.

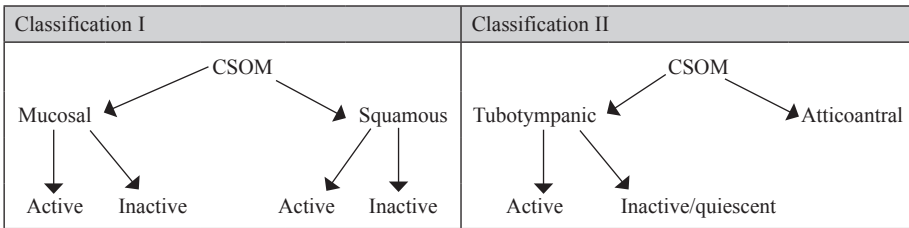**Table 1.** Two classifications of CSOM

Classification I	Classification II
CSOM Mucosal ← → Squamous Active ↓ ↓ Inactive Active ↓ ↓ Inactive	CSOM Tubotympanic ← → Atticoantral Active ↓ ↓ Inactive/quiescent

Figure 1. Otoscopic appearance of central perforation typical of mucosal CSOM.
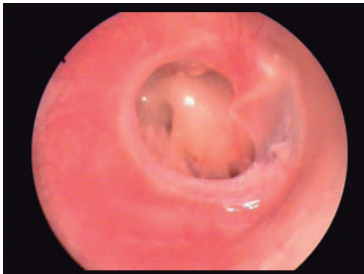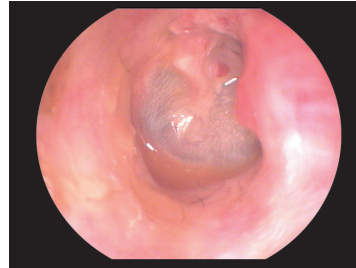

Figure 2. Otoscopic appearance of posterior superior and attic retraction pocket with granulatiosn and cholesteatoma typical of squamous active disease.

Cholesteatoma , a bag like cystic structure with white, flaky or cheesy material which has bone erosive properties is often associated with atticoantral disease or squamous disease (**Figure 2**). The classification of cholesteatoma helps to distinguish congenital cholesteatoma from the 2 types of acquired types of cholesteatoma **(Table 2)**. Congenital cholesteatoma , which may occur as rests at various locations within the temporal bone, is often asymptomatic and diagnosed incidentally on otoscopy as a white mass behind an intact tympanic membrane. When infected, there is associated otorrhoea. Acquired cholesteatoma may be primary acquired (in association with a pars tensa or pars flaccida retraction pocket) or secondary acquired (in relation to a preexisting, usually long- standing central perforation). Children are more likely to have primary acquired than secondary acquired cholesteatoma. Granulations, which are a manifestation of underlying osteitis, are more frequently seen in patients with atticoantral disease than tubotympanic disease.

Table 2. Classification of cholesteatoma

Congenital	Acquired
	Primary acquired Secondary acquired

Epidemiology
a) *Global burden of disease*

The prevalence of childhood CSOM is clearly much greater in developing countries and among ethnic minority populations of the world[2,3]. The former include countries like India, Bangladesh, most countries of the African continent, Thailand, Phillipines, Malaysia, Vietnam and Korea. The latter include the Australian

aborigines, Maoris of New Zealand, Inuits of Greenland and natives of Alaska.

According to the World Health organization, a prevalence of a disease of >4% in a country, suggests that the disease is a massive public health problem that requires urgent attention[2]. In many Western countries, the prevalence of CSOM is very low (<1%).

Reports from developing countries suggest that the prevalence of CSOM may show a decline with time[4-6]. The experience from a rural population in South India showed that concomitant with improved immunization coverage, better health indices (reduced infant mortality rate, reduced maternal mortality rate) and higher incomes, the prevalence of otitis media had halved and the prevalence of CSOM had reduced to 1/3 the prevalence in 1997over a decade[4,6].

b) *Risk factors for CSOM*

Risk factors reported for CSOM include low socioeconomic status,[7-10] supine bottle-feeding,[8-10] malnutrition[8], persistent rhinorrhoea[6], parental smoking and household smoke,[8] daycare attendance, [8-10] nasopharyngeal colonization[11] and overcrowding.[8-10] Some authors have found no relationship with socioeconomic status ,[6,12,13]and parental smoking, [6,8] however. Improvements in poor housing, hygiene and nutrition have been shown to halve the prevalence of CSOM in Maori children over a decade.[14]

Pathogenesis

Mucosal CSOM usually results from persistent discharge following an episode of acute suppurative otitis media[3]. Occasionally, it may result from a persistent perforation following removal or extrusion of a ventilation tube. External trauma due to a blow to the ear or insertion of a sharp object into the ear canal can result in a traumatic perforation. Infection occurs when the ear gets wet during bathing without ear protection or when cleaning the affected ear without aseptic precautions.

Squamous CSOM, in contrast, develops progressively, commencing as a retraction of the pars tensa or pars flaccida in response to Eustachian tubal block. With persistent negative intratympanic pressure, a retraction pocket with retained debris forms which ultimately becomes a cholesteatoma. Secondary infection leads to purulent discharge and the presence of granulations around the pocket suggests associated osteitis.

Pathology

In active mucosal CSOM, the mucosa shows features of chronic inflammation with edema, submucosal fibrosis, hypervascularity and infiltration with lymphocytes, plasma cells and histiocytes.[1] There may be areas of ulceration with proliferation of blood vessels, fibroblasts and inflammatory cells. Ossicular erosion may occur in mucosal CSOM. Typically, the tip of the handle of malleus and the long process of incus are the most affected and to an almost equal extent (16%).[15] Cholesterol granuloma, a small collection of cholesterol crystals surrounded by giant cell reaction, is seen in some cases.

In active squamous CSOM, cholesteatoma is the hallmark. It consists of a squamous epithelial lining called "matrix" subjacent to which lies a layer of subpeithelial connective tissue with chronic inflammation[1]. Bone erosion is much

more common in these cases. Ossicular erosion is seen in 70% of patients and incus erosion in 75%.[16]

Bacteriology

Classically, there is mixed infection with *S.aureus, S. pyogenes, Pseudomonas* species, *Proteus* species,*E. coli* and *Klebsiella* species. One study showed that in mucosal disease*, Pseudomonas* species were isolated from 84% of patients, enteric gram-negative bacilli from 32%, *S. aureus* from 20%, streptococci from 14%, and *H. influenzae* from 15%. [17] Some authors have found that S.*aureus* was more commonly isolated in children and *Pseudomonas* species more commonly in adults. [18] A small percentage (11%)of anaerobic bacteria like *Peptostreptococcus, Bacteroides* and *Propionibacterium* may also be present .[19]

In the presence of complications of CSOM, pus culture may reveal a high proportion of *Proteus* species (34%) and anerobes (21.3%).[20]

Clinical features

a) Mucosal disease

Symptoms

Patients with mucosal disease typically have profuse mucoid or mucopurulent ear discharge. There is usually no blood staining of the discharge and it is not foul smelling. Hearing loss is common and can vary from mild to moderately severe or profound hearing loss. The presence of earache or vertigo suggests acute exacerbation of disease or a complication.

Patient with mucosal disease typically have a central perforation (**Fig. 1**). This is a perforation of variable size which involves the pars tensa of the tympanic membrane. In mucosal active disease the ear is actively discharging and the middle ear mucosa may be pink or congested and edematous. In mucosal inactive disease the perforation is dry. Some authors describe an entity called *permanent perforation syndrome* which refers to a condition in which a patient has an asymptomatic, dry central perforation which has not discharged for several years. This condition is not considered under the broad diagnosis of CSOM because there has ostensibly been no suppuration for a long period of time.

Signs

Central perforations may be described as pinhole, small central, medium central, large central and subtotal perforations based upon their size (**Fig. 1**). Subtotal perforations have no pars tensa remnant and have only the circumferential annulus present. Ossicular erosion in mucosal disease is less common than in squamous disease. In one study, 16% cases of mucosal CSOM had malleus handle necrosis and 16.7% had incudal necrosis.[15] The same study showed that significant risk factors for incudal necrosis were middle ear granulations (p=0.04; OR=3.161; C.I.=1.087-9.196) and moderately severe hearing loss (p=0.03; O.R.=1.720; C.I.=1.064-2.782).

b) Squamous disease

Symptoms

Patients with squamous disease typically have scanty foul- smelling discharge which may be blood –stained, particularly if there are granulations. Patients with dry, self cleaning pockets may deny any history of ear discharge.

The presence of earache, headache and vertigo suggests the onset of a complication.

Signs

Otoscopic findings in atticoantral or squamous disease include the presence of a pars tensa or pars flaccida retraction pocket with cholesteatoma and/or granulations (**Figure 2**). If the retraction pocket is dry and self cleansing, the fundus of the pocket may be seen.
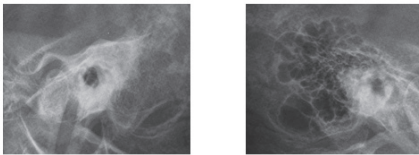
Diagnosis

The diagnosis of CSOM is largely dependent on otoscopic findings. However, otomicroscopic examination is essential to confirm findings, particularly in those with actively discharging ears. A swab may be taken for pus culture and sensitivity at this juncture although this need not be done for every case and children who are diabetic or immunosuppressed, who have suspected methicillin resistant staphylococcus aureus (MRSA) infection or who have persistent ear discharge despite conventional therapy would be ideal candidates for this test.

Pure tone audiometry is done to ascertain the nature and severity of the accompanying hearing loss. In CSOM, the hearing loss is usually conductive. However, mixed hearing loss or severe to profound sensorineural hearing loss may be present in those in whom there is cochlear damage due to diffusion of toxins through the round window membrane or ototoxicity. In very young children, auditory thresholds may be obtained with brainstem evoked response audiometry (BERA). Ossicular erosion may be predicted to some extent by the degree of hearing loss. Studies have shown that incudal necrosis is associated with a hearing loss of 45-60dB.[15]

X-ray of the mastoids, Laws view, may be performed to ascertain the status of mastoid pneumatisation (**Figure 3**) and the integrity of the tegmen and sinus plates. High resolution CT scan of the temporal bone (**Figure 4**) is preferred when information regarding the integrity of the ossicles, course of the facial nerve and status of the labyrinth are required. This is particularly useful for children with squamous disease on otoscopy, suspected congenital cholesteatoma and

Figure 3. X-ray of mastoid, Laws view, showing sclerosis of ipsilateral mastoid due to CSOM and normal, contralateral, aerated mastoid

Figure 4. High resolution CT scan of the temporal bone in a patient with bilateral CSOM showing bilateral sclerotic mastoids with soft tissue thickening both middle ears; ossicular remnants are also seen
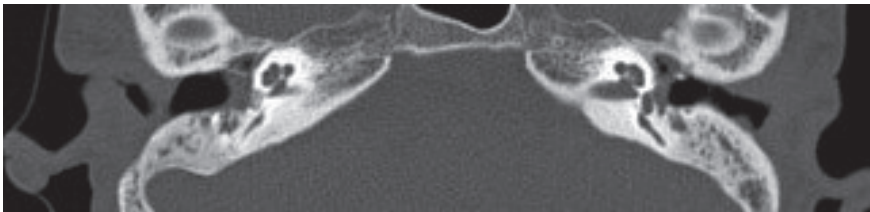
those who are scheduled to undergo revision surgery. In children with suspected intracranial complications, contrast enhanced CT of the brain is essential (**Figure 5**).

Figure 5. Contrast enhanced CT of brain showing a large, right sided cerebellar abscess

Complications of CSOM

Complications are usually associated with atticoantral or squamous disease with cholesteatoma. However, complications secondary to tympanomastoid disease (central perforations with granulations alone in the middle ear and mastoid) are not unknown.[20,21] Complications are broadly classified as being either extracranial or intracranial (**Table 3**). The commonest extracranial complication is mastoid abscess (**Figure 5**), while meningitis is the commonest intracranial complication. Mastoid abscesses occur at the sites of pneumatisation of the temporal bone and are named based on the site affected. Thus, we have the classic mastoid abscess which overlies the mastoid bone, Luc's abscess which overlies the zygoma, Citelli's abscess which overlies the digastric ridge and Bezold's abscess which overlies the upper part of the jugular vein.

Table 3. Classification of complications of CSOM

Extracranial	Intracranial
Mastoid abscess Facial palsy Labyrinthitis Petrositis	Extradural abscess Brain abscess - temporal lobe abscess - cerebellar abscess Meningitis Lateral sinus thrombosis Subdural abscess Otitic hydrocephalus

Figure 6. Patient with fullness in postaural groove suggestive of left mastoid abscess; note depression indicating site of previous incision and drainage

An intracranial complication should be suspected in a child who has had a history of ear discharge with recent onset of headache, fever, not feeding well, lethargy, earache, ataxia, vomiting, seizures or focal neurological deficit.[22] Such children require early imaging in the form of contrast enhanced CT scan of the brain and HRCT of the temporal bone to establish a diagnosis. For those with suspected meningitis, a negative scan and positive CSF microscopy and culture are diagnostic. An important dictat in the management of children with suspected intracranial complications is that radiological evaluation to exclude an intracranial pus collection like brain abscess (**Figure 6**), extradural or subdural abscess *should precede* the performance of lumbar puncture for CSF analysis.[22] Ignoring this important step could

result in fatality as the brain could herniate through the foramen magnum (coning) with the release of CSF pressure.

Treatment

a) Medical

1. Mucosal disease

Children with active mucosal disease should be given a course of topical antibiotics like quinolone preparations (Ciprofloxacin or Ofloxacin ear drops) or antiseptics like 1% acetic acid or 5% povidone –iodine drops after otomicroscopic aural toilet with a small suction or dry mopping for at least 10 days. A Cochrane review[23] has shown that aural toilet combined with topical antibiotics is more effective than aural toilet alone with a 57% reduction in otorrhoea compared to 27% with aural toilet alone(OR=0.31;95%CI=0.19-0.49). The same review also showed that topical antibiotics were as effective as systemic antibiotics in clearing ear discharge (OR =1.71; 95% CI=0.88-3.34).

The topical antibiotic chosen should be effective against *Pseudomonas* species and *S. aureus*, the two most common pathogens encountered in CSOM. A Cochrane review[23] showed that topical quinolones can reduce ear discharge better than non- quinolone antibiotics or antiseptics (OR=0.36; 95% CI=0.22-0.59). Once the ear has become dry, the parent is advised to ensure that the ear is protected from getting wet with ear plugs. If the ear remains dry for at least 6 months, there is a good chance that perforation may heal spontaneously, particularly if it is small.

2. Squamous disease

Children with small retraction pockets which are self cleansing and no hearing deficit may be kept on regular follow up. In those who develop localized infections and granulations, a trial of topical and systemic antibiotics with cautery of the granulations may be tried. In practice, most children with evidence of otorrhoea and granulations and squamous disease will require mastoid surgery or at the very least, cartilage tympanoplasty, eventually.

3. Children with complications of CSOM

This entity is considered separately because of the difference in management. Culture driven antibiotics are preferred in these children and specimens may be obtained from the ear canal or CSF (if there is meningitis). In developing countries, where funding and resources are scarce, it is advisable for children to be given cost- effective, parenteral antibiotics. The antibiotics selected should have adequate Gram positive, Gram negative and anerobic cover. Crystalline Penicillin in meningitis doses, chloromycetin and metronidazole given intravenously have been found to be extremely useful in containing the infection.[24] Intravenous antibiotic therapy should be given for at least 2 weeks and oral antibiotics for a further 4 weeks to completely eradicate infection and prevent recurrence in the postoperative period.

Surgical

When medical measures fail to either stop otorrhoea or heal the perforation and restore hearing in both mucosal and squamous disease, surgery is indicated. Early surgery prevents repeated middle ear infections due to contamination of the

middle ear from organisms in the external canal. Surgery is also indicated, often on an emergency basis, in all patients who develop complications of CSOM.

a) Mucosal disease

The role of surgery in mucosal disease is to stop further otorrhoea and improve hearing. Type 1 tympanoplasty or myringoplasty is adequate in most cases. In the case of small perforations a pop-in technique may be used. In the case of a large or subtotal perforation, it is preferable to raise a tympanomeatal flap either through a transcanal or endomeatal incision and inspect the ossicles. Concurrent ossiculoplasty may be performed if the ear is dry and ossicular erosion present. If the tympanic membrane is very thinned out, cartilage reinforcement of the fascial graft or use of a perichondrium-cartilage graft is useful.
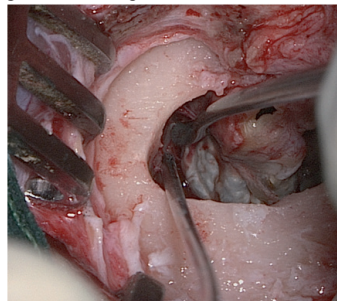
One of the dilemmas in management of children with mucosal CSOM is determining whether cortical mastoidectomy should be combined with tympanoplasty. Some authors have found no difference in outcomes when both procedures are combined.[25] The advantage of exploring the mastoid is that granulations filling the mastoid and blocking the aditus can be cleared and aditus patency restored. Preoperative assessment of aditus patency may be ascertained by high resolution CT scanning of the temporal bone. However, CT scanning of the temporal bone is not a routine procedure for children with mucosal CSOM as it is expensive and results in radiation exposure. Studies have shown that the inflation- deflation test of Eustachian tubal function may be predictive of aditus patency preoperatively.[26] In children with suspected mastoid reservoir because of recurrent or persistent otorrhoea, unresponsive to topical and systemic antibiotics, mastoid exploration with tympanoplasty should be performed.

b) Squamous disease

Children with squamous disease will require mastoid exploration in all cases with cholesteatoma (**Figure 7**). However, while those with limited disease (grade 2 or 3 retraction pockets with debris) may be treated with canal wall up procedures like cortical mastoidectomy and tympanoplasty, those with cholesteatoma involving the mastoid or even reaching upto the aditus, will require a canal wall down procedure like modified radical mastoidectomy **(Figure 8)** to provide adequate drainage and to facilitate surveillance.

Figure 7. Removal of cholesteatoma sac after mastoid exploration in a patient with squamous disease

Some authors have found intact canal wall procedures to be useful even in pediatric cholesteatoma involving the mastoid, despite a high recidivism rate of 41%.[27]These children required planned revision surgery ("second look surgery")and indefinite follow up. Preservation of hearing thresholds of < 30dB was found to be the chief benefit of this protocol. However, even these authors preferred a canal wall down procedure in the presence of an only hearing ear.
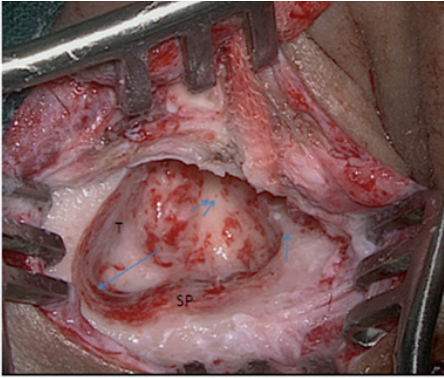
Figure 8. Modified radical mastoidectomy; note drilled out posterior canal wall (short arrow), bulge of lateral semicircular canal (double arrow), tegmen (T), sinus plate(SP) and sinodural angle (long arrow)

Non echo planar diffusion weighted MRI scans of the mastoid have been shown to have a relatively high sensitivity and specificity in the diagnosis of recurrent or residual cholesteatoma in those who have undergone canal wall up procedures for cholesteatoma.[28] However, doubts about the possibility of missing foci of cholesteatoma using this modality have been cast by some authors.[29] The consequences of missing postoperative recurrent / residual cholesteatoma in children by imaging alone could be disastrous and hence the decision to perform a canal wall up procedure should be judiciously made. In developing countries, where follow up is poor and resources are scarce, performing a canal wall down procedure for pediatric cholesteatoma is preferred as it helps to ensure early detection of recidivism and prevention of complications.

c) Children with complications of CSOM

Children with extracranial complications should undergo treatment of the complication along with mastoid exploration simultaneously. Mastoid abscesses should be drained and modified radical mastoidectomy or cortical mastoidectomy performed, depending on whether the underlying disease is squamous or mucosal .[24] In those with cholesteatoma, modified radical mastoidectomy is required. In those with granulations alone intact canal wall mastoidectomy is sufficient. All affected cells should be exenterated. Facial nerve decompression may be performed simultaneously for those with facial nerve palsy. Full recovery of the palsy may be expected in most cases if operated early.

In tertiary care centres where neurosurgical facilties are available, children with intracranial complications could undergo concomitant craniotomy and surgical excision of the intracranial abscess collection with modified radical mastoidectomy. In all other instances, the mastoid focus should be addressed within days to weeks of treatment of the intracranial complication to prevent recurrence.

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