Chronic Suppurative Otitis Media

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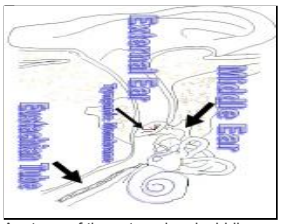
Background

Chronic suppurative otitis media (CSOM) is a perforated tympanic membrane with persistent drainage from the middle ear (ie, lasting >6-12 wk).[1, 2] Chronic suppuration can occur with or without cholesteatoma, and the clinical history of both conditions can be very similar. The treatment plan for cholesteatoma always includes tympanomastoid surgery with medical treatment as an adjunct.

CSOM differs from chronic serous otitis media in that chronic serous otitis media may be defined as a middle ear effusion without perforation that is reported to persist for more than 1-3 months. The chronically draining ear in CSOM can be difficult to treat.[3] McKenzie and Brothwell demonstrated evidence of chronic suppurative otitis in a skull found in Norfolk, United Kingdom, which is thought to be from the Anglo-Saxon period.[4] Radiologic changes in the mastoid caused by previous infection have been seen in a number of specimens, including 417 temporal bones from South Dakota Indian burials and 15 prehistoric Iranian temporal bones.[5, 6]

Anatomy

The middle ear cleft can be thought of as a 6-sided cube. Its lateral boundary, the tympanic membrane, separates it from the outer ear. Its medial boundary is formed by the promontory, which denotes the basal turn of the cochlea. Anteriorly, it is related to the tendon of tensor tympani superiorly and the opening of the eustachian tube inferiorly. Posteriorly, it is related superiorly to the aditus, which connects the middle ear cavity with the mastoid antrum, and inferiorly to the facial ridge. The roof of the middle ear cavity is formed by the tegmen tympani, and the floor of the middle ear cavity lies in close relation to the jugular foramen. (See the image below displaying anatomy of the ear).



Anatomy of the external and middle ear.

The anterior and posterior malleolar folds, which originate at the level of the lateral process of the malleus, form the boundary between the epitympanum and mesotympanum, which lie above and below it, respectively. Atticoantral disease predominantly affects the pars flaccida, and tubotympanic disease affects the pars tensa.

The middle ear cavity also consists of the ossicular chain (malleus, incus, and stapes). The ossicular chain connects the tympanic membrane, in which the handle of the malleus is embedded, to the oval window, on which sits the footplate of the stapes. In atticoantral disease, the ossicular chain is frequently affected by cholesteatoma, thereby causing hearing loss. Removal of the malleus and or incus may be necessary if they are extensively involved by cholesteatoma. In these cases, a planned second-stage reconstruction is often appropriate.

Pathophysiology

CSOM is initiated by an episode of acute infection. The pathophysiology of CSOM begins with irritation and subsequent inflammation of the middle ear mucosa. The inflammatory response creates mucosal edema. Ongoing inflammation eventually leads to mucosal ulceration and consequent breakdown of the epithelial lining. The host's attempt at resolving the infection or inflammatory insult manifests as granulation tissue, which can develop into polyps within the middle ear space. The cycle of inflammation, ulceration, infection, and granulation tissue formation may continue, eventually destroying the surrounding bony margins and ultimately leading to the various complications of CSOM.[7, 8]

Common bacteria

Pseudomonas aeruginosa, Staphylococcus aureus, Proteusspecies, Klebsiella pneumoniae, and diphtheroids are the most common bacteria cultured from chronically draining ears. Anaerobes and fungi may grow concurrently with the aerobes in a symbiotic relationship. The clinical significance of this relationship, although unproven, is theorized to be an increased virulence of the infection. Understanding the microbiology of this disease enables the clinician to create a treatment plan with the greatest efficacy and least morbidity.

P aeruginosa is the most commonly recovered organism from the chronically draining ear. Various researchers over the past few decades have recovered pseudomonads from 48-98% of patients with CSOM.

P aeruginosa uses pili to attach to necrotic or diseased epithelium of the middle ear. Once attached, the organism produces proteases, lipopolysaccharide, and other enzymes to prevent normal immunologic defense mechanisms from fighting the infection. The ensuing damage from bacterial and inflammatory enzymes creates further damage, necrosis, and, eventually, bone erosion leading to some of the complications of CSOM. Fortunately, in the immunocompetent individual, the infection rarely causes serious complications or disseminated disease. Pseudomonal infections commonly resist macrolides, extended-spectrum penicillins, and first- and second-generation cephalosporins. This can complicate treatment plans, especially in children.

S aureus is the second most common organism isolated from chronically diseased middle ears. Reported data estimate infection rates from 15-30% of culture-positive draining ears. The remainder of infections are caused by a

large variety of gram-negative organisms. *Klebsiella* (10-21%) and *Proteus* (10-15%) species are slightly more common than other gram-negative organisms.

Polymicrobial infections are seen in 5-10% of cases, often demonstrating a combination of gram-negative organisms and *S aureus*. The anaerobes (*Bacteroides*, *Peptostreptococcus*, *Peptococcus*) and fungi (*Aspergillus*, *Candida*) complete the spectrum of colonizing organisms responsible for this disease. The anaerobes make up 20-50% of the isolates in CSOM and tend to be associated with cholesteatoma. Fungi have been reported in up to 25% of cases, but their pathogenic contribution to this disease is unclear.

Etiology

The diagnosis of CSOM requires a perforated tympanic membrane. These perforations may arise traumatically, iatrogenically with tube placement, or after an episode of acute otitis media, which decompresses through a tympanic perforation.[3, 9, 10, 11]

The mechanism of infection of the middle ear cleft is postulated to be translocation of bacteria from the external auditory canal through a perforation into the middle ear. Some authors suggest that the pathogenic organisms may enter through reflux of the eustachian tube. The data supporting this theory are inconclusive. Most of the pathogenic bacteria are those common to the external auditory canal.

The risk of developing otorrhea (but not necessarily CSOM) through a ventilation tube is reportedly 21-50%. Annually, more than a million tubes are placed in the United States for recurrent otitis media and otitis media with effusion. Studies have reported that 1-3% of patients with ventilation tubes develop this disease.

The risk of developing CSOM increases with the following circumstances[12]:

- A history of multiple episodes of acute otitis media
- Living in crowded conditions
- Day care facility attendance
- Being a member of a large family

Studies trying to correlate the frequency of the disease with parental education, passive smoke, breastfeeding, socioeconomic status, and the annual number of upper respiratory tract infections are inconclusive.

Patients with craniofacial anomalies are special populations at risk for CSOM. Cleft palate, Down syndrome, cri du chat syndrome, choanal atresia, DiGeorge syndrome, cleft lip, and microcephaly are other diagnoses that increase the risk of CSOM, presumably from altered eustachian tube anatomy and function.

Epidemiology

The larger the tympanic membrane perforation, the more likely the patient is to develop CSOM. Some studies estimate the yearly incidence of CSOM to be 39 cases per 100,000 persons in children and adolescents aged 15 years and younger. In Britain, 0.9% of children and 0.5% of adults have CSOM. In Israel, only 0.039% of children are affected.[13]

Certain population subsets are at increased risk for developing CSOM. The Native American and Eskimo populations demonstrate an increased risk of infection. Eight percent of Native Americans and up to 12% of Eskimos are affected by CSOM. The anatomy and function of the eustachian tube play a significant role in this increased risk. The eustachian tube is wider and more open in these populations than in others, thus placing them at increased risk for nasal reflux of bacteria common to acute otitis media and recurrent acute otitis media and leading to more frequent development of CSOM.

Other populations at increased risk include children from Guam, Hong Kong, South Africa, and the Solomon Islands. The prevalence of CSOM appears to be distributed equally between males and females.Exact prevalence in different age groups is unknown; however, some studies estimate the yearly incidence of CSOM to be 39 cases per 100,000 in children and adolescents aged 15 years and younger.[12]

Prognosis

Patients with CSOM have a good prognosis with respect to control of infection. The recovery of associated hearing loss varies depending on the cause. Conductive hearing loss can often be partially corrected with surgery. The goal of treatment is to provide the patient a safe ear.

Much of the morbidity of CSOM comes from the associated conductive hearing loss and the social stigma of an often fetid fluid draining from the affected ear. The mortality of CSOM arises from associated intracranial complications. CSOM itself is not a fatal disease. Although some studies report sensorineural hearing loss as a morbid complication of CSOM, other evidence conflicts with this claim.

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