



Evaluation of bacteriological profile in patients suffering from chronic suppurative otitis media (CSOM) from nalanda medical college and hospital

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Abstract

There are two main varieties of CSOM *viz.* tubotympanic type and atticofacial CSOM. The later type of CSOM can cause severe adverse effects like intra and extracranial complications which can be life threatening. The complications of CSOM can be prevented to a greater extent by the judicious use of antibiotics. Due to the recurrent nature of the disease and the development of drug resistant pathogenic organisms, the control of infection poses a great therapeutic challenge. The knowledge of bacteriological profile helps in appropriate management of the cases. Hence this study was carried out to know the bacterial etiology of CSOM and their antibiotic susceptibility pattern.

The present study was planned in the Department of Microbiology, Nalanda Medical College and Hospital, Patna, Bihar from Jan 2018 to July 2018. Total 100 patients presented with ear discharge and tympanic membrane perforation/retraction with or without cholesteatoma were enrolled in the present study. Direct culture material was seeded on Blood agar, Mac Conkey's agar and on Chocolate agar plates. All plates were incubated aerobically at 37°C and evaluated at 24 hours, 48 hours and 72 hours and the plates were discarded if there was no growth. The specific identification of bacterial pathogens was done based on microscopic morphology, staining characteristics, cultural and biochemical properties using standard laboratory.

The knowledge of the etiological agents causing CSOM and their antimicrobial susceptibility pattern is of great importance for an effective treatment and prevention of complications. As higher incidence of CSOM was seen among children, educating parents and guardians on possible risk factors of the disease may be a preventive strategy that might reduce disease occurrences.

Keywords: chronic suppurative otitis media, CSOM, aerobic bacteriological profile, etc

Introduction

Chronic suppurative otitis media (CSOM) is a chronic inflammation of the middle ear and mastoid cavity that is characterised by discharge from the middle ear through a perforated tympanic membrane for at least 6 weeks. CSOM occurs following an upper respiratory tract infection that has led to acute otitis media. This progresses to a prolonged inflammatory response causing mucosal (middle ear) oedema, ulceration and perforation. The middle ear attempts to resolve this ulceration by production of granulation tissue and polyp formation. This can lead to increased discharge and failure to arrest the inflammation, and to development of CSOM, which is also often associated with cholesteatoma. There may be enough pus that it drains to the outside of the ear (otorrhea), or the pus may be minimal enough to be seen only on examination with an otoscope or binocular microscope. Hearing impairment often accompanies this disease.

People are at increased risk of developing CSOM when they have poor eustachian tube function, a history of multiple episodes of acute otitis media, live in crowded conditions, and attend paediatric day care facilities. Those with craniofacial malformations such as cleft lip and palate, Down syndrome, and microcephaly are at higher risk^[1].

Chronic suppurative otitis media (CSOM) is a perforated tympanic membrane with persistent drainage from the

middle ear (ie, lasting >6-12 wk)^[2, 3]. 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^[4]. 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^[5]. 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^[6, 7].

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. (A study by Wang *et al.*

suggested that in CSOM, T-cell-mediated cellular immunity plays a role in the formation of granulation tissue [8]. 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 [9, 10].

Pseudomonas aeruginosa, *Staphylococcus aureus*, *Proteus* species, *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.

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 [11, 12].

A study by Jensen *et al.* of two groups of children in

Greenland found that among those children with CSOM, 91% suffered permanent hearing loss of greater than 15 dB HL (decibel hearing level). The groups were followed up for 10 and 15 years [13].

A study by Aarhus *et al.* of hearing loss in various types of otitis media found that childhood hearing loss from CSOM is associated with adult hearing loss, with the effect on hearing thresholds being greater in middle age (age 40-56 years) than in young adulthood (age 20-40 years). The same held true for recurrent acute otitis media [14].

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Methodology

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All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Results & Discussion

CSOM is defined as chronic inflammation of the middle ear and mastoid cavity which presents with recurrent ear discharges or otorrhoea through a tympanic membrane perforation. It is classified into 2 types tubotympanic and atticofacial depending on whether the disease process affects the pars tensa or pars flaccida of tympanic membrane. Tubotympanic is called as a safe type or benign type as there is no serious complication whereas atticofacial is called as the unsafe or dangerous type because of associated complications. Infection can spread from middle ear to vital structures such as mastoid, facial nerve, labyrinth, lateral sinus, meninges and brain leading to mastoid abscess, facial nerve paralysis, deafness, lateral sinus thrombosis, meningitis and intra cranial abscess. Of all the complications, hearing loss associated with chronic ear discharge is nearly always significant reported in 50% of cases and tending to be more severe than those reported in other types of otitis media [17].

Table 1: Culture pattern in various age groups

Age group	Single bacteria isolated	Multiple bacteria isolated	Sterile culture	Total
1-15	22	0	5	27
16-30	20	1	3	24
31-45	24	3	3	30
46-60	10	1	2	13
61-75	5	0	1	6
Total	81	5	14	100

Table 2: Organisms isolated in the study (M =multiple organisms).

Organism	Frequency
<i>Acinetobacter</i>	6
<i>Acinetobacter, CONS (M)</i>	1
<i>Citrobacter</i>	3
<i>CONS</i>	5
<i>E. coli</i>	5
<i>E. coli, CONS (M)</i>	1
<i>E. coli, Pseudomonas (M)</i>	2
<i>Enterococci</i>	2
<i>Klebsiella</i>	2
<i>MRSA</i>	5
<i>No growth</i>	15
<i>Pneumococci</i>	1
<i>Proteus mirabilis</i>	1
<i>Proteus vulgaris</i>	2
<i>Pseudomonas</i>	28
<i>Pseudomonas, Staph. aureus (M)</i>	1
<i>Schwanella species, CONS (M)</i>	1
<i>Schwanella</i>	3
<i>Staph. aureus</i>	19
<i>Staph. aureus, Pseudomonas (M)</i>	1
<i>Total Staphylococcus</i>	20
<i>Total Pseudomonas</i>	29

The frequency of *Staphylococcus aureus* in the middle ear infections can be attributed to their ubiquitous nature and high carriage of resistant strains in the external auditory canal and upper respiratory tract. The organisms like *Pseudomonas* spp. and *Proteus* spp. were considered mostly as secondary invaders from external auditory canal gaining access to the middle ear via a defect in tympanic membrane resulting from an acute episode of otitis media. Organisms like *E. coli* and *Klebsiella* spp. become opportunistic pathogens in the middle ear when resistance is low.

Group B *Streptococcus* (GBS) was not isolated in this study, unlike western, developed countries where it is the major agent of neonatal septicemia. This may be attributed to low prevalence of GBS colonization of pregnant women in this area or possibly, to the presence of strains with low virulence [18]. Since a sizeable number of culture specimens were negative by aerobic culture, the possibility of infection by anaerobes must be entertained and anaerobic culture can be performed routinely in cases of neonatal sepsis [19]. However, the feasibility, logistics and cost-effectiveness of routine anaerobic culture for neonatal sepsis need to be explored further.

The antibiotic sensitivity testing showed that most of the gram negative isolates were sensitive to meropenem followed by chloramphenicol, ciprofloxacin, gentamicin and amikacin. They were not sensitive to the commonly used antibiotics like penicillin, ampicillin etc. The antibiotic sensitivity testing of the gram positive isolates showed that they were maximally sensitive to linezolid, netilmicin and vancomycin. This is comparable to the study done by P.

Jyothi *et al.* in which maximum sensitivity was observed in imipenem and linezolid [20]. Netilmicin and Amikacin was found to be highly sensitive in the study done by Agnihotri *et al.* for *Staph. Aureus* and gram negative isolates respectively [21]. While the study by Mathur *et al.* showed Gentamicin to be sensitive in gram cases. 6 many studies also reported cefotaxime to have shown maximum sensitivity [22] but it could not be compared as some of the samples from this study had not undergone the sensitivity testing for cefotaxime. Besides the antimicrobial sensitivity patterns differs in different studies as well as at different times in the same hospital. This is because of emergence of resistant strains as a result of indiscriminate use of antibiotics. Thus, Meropenem and Linezolid were found to be the most sensitive drugs for gram negative and gram positive respectively, but these two drugs should not be used indiscriminately and kept as reserve drugs, otherwise resistance to these drugs may develop, thereby threatening the treatment.

As the strains of bacterial isolates responsible for CSOM are still found to be responsive to first line drugs, at least in our area, the treatment of CSOM should be tailored according to the pattern in the microbiological flora of each discharging ear. As far as we know, this is the first study from this region regarding the common bacterial pathogens isolated in cases of CSOM and their antibiogram in this region. More such studies are urgently required in this context, as they will help the clinician in selecting the optimum presumptive therapy in these cases.

It was not possible to follow up all patients included in this study after the antibiotic therapy was initiated. The patients who could be followed up responded well after initiating specific therapy. This study cannot recommend one single drug for therapy as the antibiotic susceptibility was variable depending on the organism. The antibiotic susceptibility also varies depending on the region. Empiric therapy is also not recommended as blind therapy without a culture and the antibiotic sensitivity report may lead to prolonged duration of therapy and huge burden to the patient, if the bacteria isolated is resistant to the antibiotic used. Hence, culture and antibiotic sensitivity must be done before initiating the therapy. Studies have to be done on regular intervals in different regions to determine the most common isolates and their susceptibility.

Conclusion

The knowledge of the etiological agents causing CSOM and their antimicrobial susceptibility pattern is of great importance for an effective treatment and prevention of complications. As higher incidence of CSOM was seen among children, educating parents and guardians on possible risk factors of the disease may be a preventive strategy that might reduce disease occurrences.

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