Multidrugs Resistant Bacteria Associated with Human Otitis Media

Gram's negative bacteria were the commonest microorganisms;it comprises (60 %). Pseudomonas aeruginosa was common causative agent (19.04%), followed by Staphylococcus aureus (16.7%) and Klebsiella spp.(14.3%). Mixed infection was found in high percent (74%), in which Pseudomonas aeruginosa and other microorganisms were more common . The antibiotic sensitivity pattern showed that Pseudomonas aeruginosa was sensitive to Ciprofloxacin, amoxicillin + clavulanic acid and gentamicin, while other is appeared resistant, Staph. aureus was sensitive to ciprofloxacin, amoxicillin + clavulanic acid, erthomycin, cephalexine and it is resistant to pencillin and ampicillin, klebsiella species were sensitive to ciprofloxacin, amoxicillin + clavulanic acid, gentamicin, while resistant to tetracycline. Geographical distribution, 100 (84%) of patients with CSOM living in urban area, 20(16.7) living in rural area . Smoking and passive smoking, 30(25%)smoker patients, 80(75%) non smoker, while 63(52.5%) patients with CSOM suffering from passive smoking. Distribution of feeding type among infants and children with CSOM 20(16.66%) breast feeding, while 10(8.33%) bottle feeding.

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Multi Drugs Resistant Bacteria Associated With Chronic Suppurative Otitis Media

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Summary

One hundred twenty (120) patients with chronic suppurative otitis media (CSOM) in Basrah ,65(54.2%) males and 55(45.8%) females ,with male: females ratio (1.2:1) and 60 individual without otological problems as control group were included in this study, which done during the period between March 2009 and January 2010.

1. **Clinical study** :

This include history , address of patients, tuning fork examination (Rinne's and Weber's) test, examination of the ear using otoscopy, audiological investigation including pure tone audiometery and tympanometery.

The result of clinical study showed that CSOM cause various type of hearing loss. Most common type was conductive hearing loss (50%) followed by senserineural hearing loss(29.17%) and mixed hearing loss(20.9%).

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It is found that CSOM was most common among infants and children (60%) .

There was no significant variation in the prevalence of CSOM between patients according to sex .P>0.05

There was significant variation in the prevalence of CSOM between urban area (84%) and rural (16%).P<0.01

2. **Microbiolgical study** :

This Include collection of aural swab samples ,culturing of samples ,identification of causative agents species and antibiotic sensitivity.

Gram's negative bacteria were the commonest microorganisms;it comprises (60 %).*Pseudomonas aeruginosa* was common causative agent (19.04%),followed by *Staphylococcus aureus* (16.7%) and *Klebsiella* spp.(14.3%).

Mixed infection was found in high percente (74%),in which *Pseudomonas aeruginosa* and other microorganisms were more common .

The antibiotic sensitivity pattern showed that *Pseudomonas aeruginosa* was sensitive to Ciprofloxacin ,amoxicillin +clavulanic acid and gentamicin ,while other is appeared resistant ,*Staph.aureus* was sensitive to ciprofloxacin , amoxicillin+clavulanic acid ,erthomycin ,cephalexine and it is resistant to pencillin and ampicillin,*klebsiella* species were sensitive to ciprofloxacin ,amoxicillin +clavulanic acid ,gentamicin,while resistant to tetracycline.

Geographical distribution ,100 (84%) of patients with CSOM living in urban area ,20(16.7) living in rural area .

Smoking and passive smoking ,30(25%)smoker patients ,80(75%) non smoker, while 63(52.5%) patients with CSOM suffering from passive smoking.

Distribution of feeding type among infants and children with CSOM 20(16.66%) breast feeding, while 10(8.33%) bottle feeding.

Overcrowding among patients with CSOM 80(66.66%).

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List of Abbreviations

AOM	**Acute otitis media**
CSOM	**Chronic suppurative otitis**
CHL	**Conductive Hearing Loss**
dB	**Deci Bell**
daPa	**Deca pascal**
E.T	**Eustachian tube**
E.N.T	**Ear,nose and throat**
H.influenzae	***Haemophilus influenza***
Hz	**Hertz**
MXHL	**Mixed Hearing Loss**
M.catarrhalis	***Moraxella catarrhalis***
N.G	**NO.Growth**
OM	**Otitis Media**
OME	**Otitis media with effusion**
O.R.L	**Oto-rhino-laryngology**
Ps.aeruginosa	***Psedomonase aeruginosa***
PHL	**Profound Hearing Loss**
P.T.A	**pure tone audiometric**
SOM	**Secretory otitis media**
Staph.aureuse	***Staphylococcus aureuse***
Strept.pneumoniae	***Streptococcus pneumonia***
SNHL	**Senserineural Hearing Loss**
T.F.E	**Tuning fork examination**
T.M	**Tympanic membrane**
U.R.I	**Upper respiratory tract infection**
W.H.O	**World Health Organization**

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Chapter One

Introduction and literatures Review

1.1 Introduction:

Otitis media is inflammation of the middle ear. This is most commonly caused by the build up of fluid behind the ear drum, as a result of a blockage to the Eustachian tube.Otitis media is more common in children, as their Eustachian tube is shorter and more horizontal than adults and is made up of more flaccid cartilage, which can impair its opening. Otitis media can cause a mild to moderate hearing loss, due to the fluid interfering with the transmission of sound through to the inner ear. It can often affect the tympanic membrane causing it to retract or become inflammed. The fluid can cause the tympanic membrane to bulge and become inflamed and occasionally the tympanic membrane will perforate .There are three common types of otitis media, acute purulent otitis media, otitis media with effusion and chronic suppurative otitis media. **(Berman , 1997)**.

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The most frequent causative agent of acute Otitis media (AOM) was *Streptococcus pneumoniae* (40%), followed by *Haemophilis influenzae* (25%), *Moraxella catarrhalis* (10%), *Group A Streptococcus* (2%) and *Staphylococcus aureus.***(Stephenson,(1992)**.

The emergence of viral identified and presumably are pathogens bacterial strains that are increasingly resistant to antimicrobial agents is a growing concern in Canada and worldwide. In appropriate use of antibiotics for viral upper respiratory tract infections has been a major contributor to antimicrobial resistance Currently, 25% of *Haemophilus influenza**e*** and 90% of *Moraxella catarrhalis* produce β-lactamase

enzymes which will inactivate penicillin and amino penicillins. Recently, there has been a dramatic increase of multiple antibiotic resistant *Streptococcus pneumonia*.**(American academy, 2004)**

Chronic otitis media with effusion (persistent middle-ear effusion) occurs with continuing negative pressure within the middle ear: the effusion becomes " trapped" in middle ear due to the special anatomy, and impairment of both the mucociliary system and the pumping action of tubal opening and closure .Chronic perforation of the tympanic membrane may develop after an acute perforation fails to heal, resolution of active CSOM, or during the course of chronic otitis media with effusion, CSOM occurs when acute drainage through a non-intact tympanic membrane persists for 2 weeks to 3 months or longer. If there is a chronic perforation, reinfection may occur by reflux through the Eustachian tube of nasopharyngeal secretions containing the bacteria seen in acute otitis media (eg *Streptococcus pneumoniae, and Hemophilus influenzae*).Organisms such as *Pseudomonas aeruginosa and Staphylococcus aureus*, may then enter the middle ear directly from the external ear canal by bathing and swimming which results in secondary infection, chronic otorrhea, and chronic osteitis of the middle ear cleft.**(WHO,2000)**

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Cholesteatoma is a non-neoplastic lesion consisting of keratinizing stratified squamous epithelium and desquamating epithelium of keratin within the middle ear or other pneumatized portions of the temporal bone. Cholesteatoma can be congenital or acquired; in the latter case it is associated with COM and related conditions .**(Peltonen, 2008)**

Children and adults who have chronic suppurative otitis media appear to be protected from developing an attic or posterosuperior type of cholesteatoma. This may explain the low incidence of cholesteatoma in racial groups that have a high rate of chronic perforation , deafness,

tinnitus and discharge are common sign and symptom of cholesteatoma.**(Choa , 1982)**

The effects of hearing, conductive hearing loss usually accompanies CSOM; it results from blockage of the external auditory canal by pus and perforation of the tympanic membrane. The average hearing loss is usually worse than that caused by otitis media with effusion. A hearing loss greater than 40dB may indicate fixation or discontinuity of the ossicular chain as well. Sensori-neural hearing loss may also occur, probably due to infiltration of infectious or inflammatory agents through the round window to produce a serous labyrinthitis. **(Howard ,2007)**

1.2 Literatures review:

1.2.1 Otitis media :

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Otitis media is inflammation of the middle ear cause hearing loss in children around the world, this is most commonly caused by the buildup of fluid behind the ear drum, as a result of a blockage of the Eustachian tube, because it is shorter and more horizontal than adults and is made up of more flaccid cartilage, which can impair its opening ,otitis media can cause a mild to moderate hearing loss, due to the fluid interfering with the transmission of sound to the inner ear. It can often affect the tympanic membrane causing it to retract or become inflammed. The fluid can cause the tympanic membrane to bulge and become inflamed and occasionally the tympanic membrane will perforated.(**Bluestone *et al*., 1992)**

1.2.2 Chronic suppurative otitis media (CSOM)

CSOM, for the purposes of this document, defined as a chronic inflammation of the middle ear and mastoid cavity, which presents with recurrent ear discharges or otorrhoea through a tympanic perforation. The disease usually begins in childhood as a spontaneous tympanic perforation due to an acute infection of the middle ear, known as acute

otitis media which presents with a rapid onset of signs and symptoms, such as pain, fever, irritability; a red bulging ear drum and middle ear effusion.(**Jahn, 1991)**

A sequel of less severe forms of otitis media (e.g. secretory OM). The infection may occur during the first 6 years of a child's life, with a peak around 2 years. The point in time when AOM becomes CSOM is still controversial generally, patients with tympanic perforations which continue to discharge mucoid material for periods of from 6 weeks to 3 months, despite medical treatment, are recognized as CSOM cases .(**Kenna *et al*., 1994)**

The WHO definition requires only 2 weeks of otorrhoea but otolaryngologists tend to adopt a longer duration, more than 3 months of active disease the ultimate fate of the tympanic perforation is still largely unclear. Thus, both the start and the end of the disease process are difficult to define . **(Smith,et al.,1996 and Goycoolea,1991)**

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The perforation in the safe type is central no matter how large, there is always arim of drum or it sannulus around the edge. In contrast, the perforation in the unsafe variety extends to the bony edge of the drum, where chronic necrosis of the bone is often associated with the production of granulation tissue. This marginal perforation is usually posterior or in the attic. Discharge in the safe variety comes from the inflammed and secreting mucosa of the middle ear, and is mucoid. It may be intermittent, with activity provoked by water and by blockage of the Eustachian tube. In the unsafe variety, the discharge is often scanty and foul smelling and there are no periods of quiescence. This discharge usually comes from the infected debris accumulating within the cholesteatoma sac . **(Mawson and Pollack,1988)**

Often, the perforation heals imperfectly with areas of retraction and scarring in the eardrum which do not vibrate in response to sound, as well as normal areas. The episodes of otorrhoea are often provoked by upper respiratory infections. This is particularly common in children. Soiling of the middle ear from swimming or bathing also leads to intermittent and unpleasant discharges.Adecidedly smaller group of patients,particularly those who have not been treated, develop life threatening complications. **(Brobby ,1992)**.

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1.2.3 Anatomy of the ear.

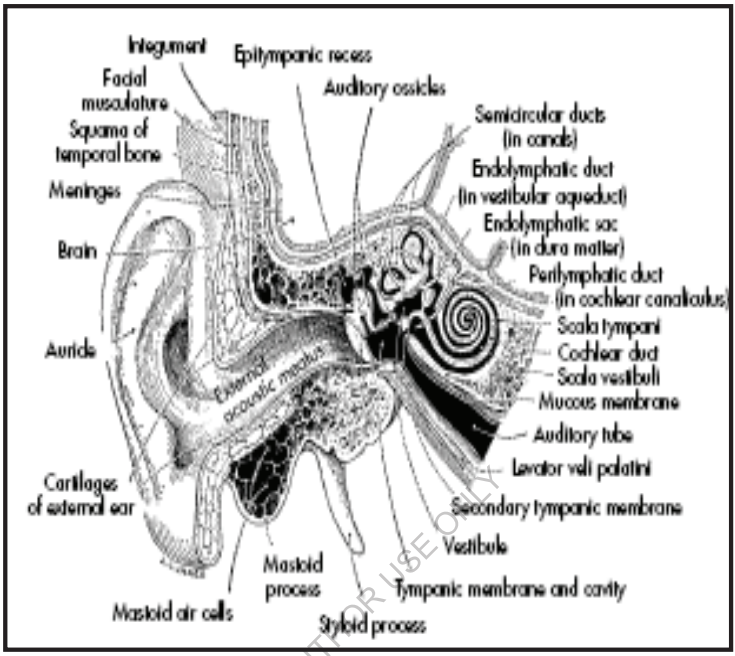

Figure (1-1) Anatomy of the Auditory and Vestibular Systems.(Anson, 1981).

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The ear consist of three part: outer part (external ear),consists of the pinna and external auditory canal , the pinna acts to funnel sound waves from the outside environment into the ear canal, while auditory canal act to pass sound wave to middle ear and secreted cerumin (ear wax) for protection from forgien bodies is composed of fibro elastic cartilage covered by skin**.(Canter,1997).**

Middle part(middle ear),consists of the tympanic membrane ,middle ear cavity, ossicular chain and Eustachian tube. The tympanic membrane (eardrum) has two functions: it gathers sound like the membrane of a microphone, and it provides sonic shielding of the round window

membrane. Sound waves that directly impinge on the round window can counteract the peri lymphatic fluid displacement induced by the stapes, reducing the sensitivity of the cochlea Several conditions can diminish the mobility of the small bones (ossicles) in the middle ear. Otitis media, an infection in the middle ear, occurs when fluid cannot escape into the throat because the eustachian tube is blocked. **(Mair,1994)**

The fluid (pus or mucus) that accumulates prevents the ossicles from moving as efficiently as they normally do, thus dampening the sound waves, the middle ear cavity connected to the nasopharynx via the eustachian tube, there are three ossicles the malleus, the incus, and the stapes in the middle ear, the Eustachian tube act to ventilates the tympanic cavity and air cells, equalizes pressure differences between the tympanic cavity and the atmosphere,drains the middle ear spaces and creates a barrier for ascending infection. **(Haben, 2005)**

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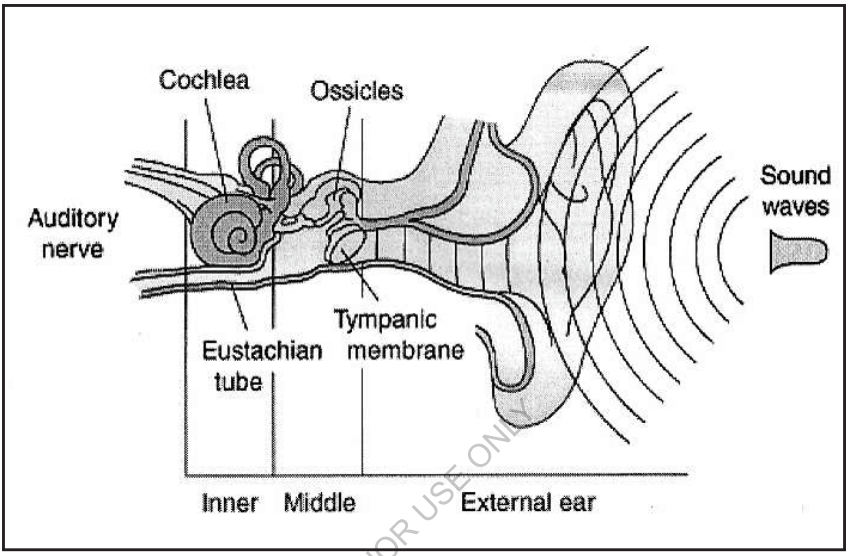
Inner part(inner ear): consists of cochlea and vestibule , audutory nerve, cochlear duct, perilymphatic duct ,semicircular canal. There are two fluid compartments of the inner ear, the endolymph and the perilymph .endolymph is a fluid that has a similar ionic composition to intra cellular fluid and fills the membranous auditory and vestibular labyrinth endolymph is formed by secretory cells in the stria vascularis and by dark cells near the ampullary ends of the semicircular ducts and the walls of the utricle,the endolymph is thought to be absorbed in the endolymphatic sac, endolymph composition is characterized by a high potassium level and a low sodium level, the membranous labyrinth is suspendedwithin the bony labyrinth by a fine trabecular net-work in a space that is filled with perilymph. **(Lalwani, 2009)**

The external ear collects sound pressure waves and funnels them toward the tympanic membrane. The middle ear ossicles transmit the sound waves to the inner ear (cochlea), it also match the impedance

difference between the air of the external environment to the fluid within the cochlea. This permits maximal sound transmission. **(Mair,1994)**



Figure(1-2)Anatomy &Physiology of the ear. (Lalwani,2009).

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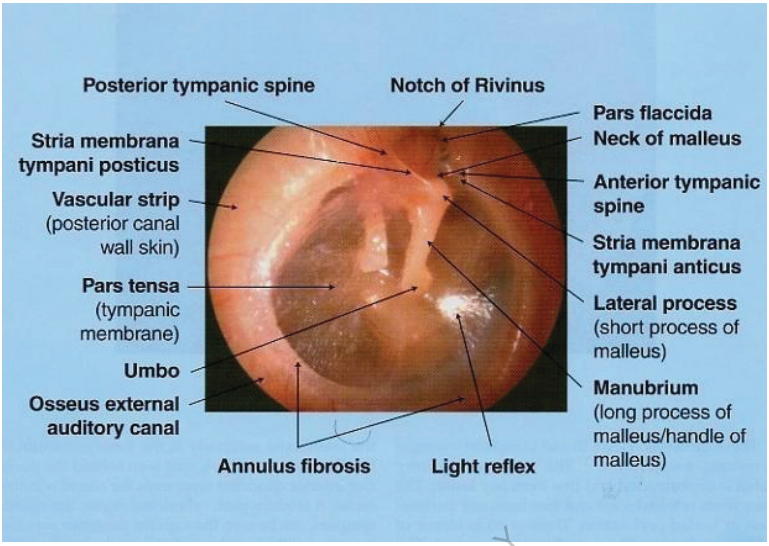
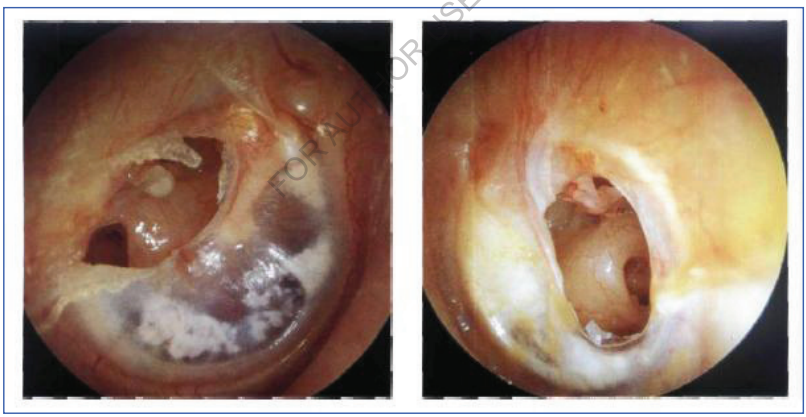


Figure(1-3) Normal tympanic membrane (normal ear).,(Jack *et al*.,2001)



Figure(1-4) Poreferated tympanic membrane CSOM.,(Mario *et al.* ,1999)

1.2.4 Classificationof COM.

Otitis media is a broad subject which could be classified according to:

duration in acute otitis media and chronic otitis media,nature of fluid/discharge suppurative and non suppurative otitis media.Otitis media with effusion and aero otitis media.Causative organism bacterial otitis media (common) and specific otitis media e.g. Tuberculous and syphilitic otitis media(less common).**(Browning , 2008)**.

1.2.4.1.Suppurative :

1.Acute suppurative otitis media .

2.Chronic suppurative otitis media .

1.2.4.2.**Non –Suppurative** :

1.Acute non-suppurative otitis media .

2.Chronic non-suppurative otitis media.

1.2.4.3.**Specific** :

1.Tuberculous otitis media.

2.Syphilitic otitis media.

3.Mycotic otitis media.

4.Viral otitis media.

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1.2.5 Epidemiology:

Chronic suppurative otitis media most often occurs in the first 5 years of life. Furthermore, it is most common in developing countries, in special populations (e.g., in children with craniofacial anomalies), and in certain racial groups. Prevalences of CSOM in children range from less than 1% up to 46% worldwide, being lowest in highly developed countries. **(Verhoeff, *et al.*, 2006)**.

Adhesive otitis media may develop as a result of long-lasting chronic middle ear infection in a small percentage of the patients.(**Cummings, *et al.*, 1998 and Bluestone, *et al.*,1995**) .

There appear to be four groups of populations based upon the prevalence of the disease with certain disadvantaged ethnic groups having some of the highest prevalence. However, there is a still shortage of accurate, standardized data with which to compare the size of the problem between different parts of the world. In developed countries, since the advent of antimicrobial therapy, the incidence and prevalence of chronic suppurative otitis media has markedly decreased, and that of otitis media with effusion has increased,in Britain 0.9% of children and 0.5% of adult have chronic suppurative otitis media with no difference between the sexes. **(Vanderveen *et al*., 2006)**

High rates of chronic otitis media have been attributed to overcrowding, inadequate housing, poor hygiene (these factors through transmission of the pathogens by physical contact with a contaminated individual, inhalation of infected droplets, or contact with an infected surface), lack of breastfeeding, poor nutrition, passive smoking, anecdotally to wood-burning smoke, high rates of naso-pharyngeal colonization with potentially pathogenic bacteria, and inadequate or unavailable health care.**(Lasisi *et al*.,2007)**

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Poverty is a major risk factor in developing countries and certain neglected populations. Since Chronic suppurative otitis media begins with an acute onset of otitis media, either acute otitis media or otitis media with effusion, risk factors associated with acute otitis media may also be initially involved (viral and bacterial infection); eustachian tube dysfunction; young age and immature or impaired immunologic status; upper respiratory allergy; familial predisposition; presence of older siblings; male sex; bottle feeding; day-care attendance; passive smoking.**(WHO, 2000)**

The prevalence of CSOM was as following in various countries : Highest inuits 12-46 % Australian Aboriginals 12-25 % Native

Americans 4-8 % High S Pacific Islands 4-6 % Africa 3-6 % Low Korea 2 % India 2 % Saudi Arabia 1.4 % Lowest USA <1 % UK <1. **(WHO, 2004 and Ologe, *et al.*,2002)**

1.2.6.**Etiology and pathogenesis** :

Several identified risk factors exist for development of CSOM, including a history of acute and recurrent otitis media, parental history of otitis media, . The pathogenesis of CSOM is multifactorial, and both environmental and genetic factors contribute to the likelihood of the disease. In addition, pathology in the anatomy and physiology of the Eustachian tube is involved.**(Seibert,2006).**

Bacteria isolated from CSOM were most typically *Pseudomonas aeruginosa,Staphylococcus aureus,Proteus* species among the aerobes, and *Bacteroides* species among the anaerobes. **(Aslam, 2004)**

They can enter the middle ear either through the ET or from the external auditory canal (EAC) through a perforated tympanic membrane ,fungi may also be present. It is still unclear whether the bacteria play a role in the CSOM development or represent secondary contamination in the inflamed middle ear. With growing evidence for bacteria being a major cause of resistant, chronic infections on tissue or implant surfaces, the existence of these bacteria in CSOM and infected cholesteatomas has become obvious the eventual role of the biofilms in these diseases is under investigation and is yet to be clarified.**(Post, 2001)**

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1.2.7.Complications of CSOM

CSOM produces chronic mastoiditis by contiguous spread. Erosion of the walls of the middle ear and mastoid cavity, which is rare, leads to exposure of the facial nerve, jugular bulb, lateral sinus, membranous labyrinth and temporal lobe dura. This in turn leads to such complications

as facial nerve paralysis, lateral sinus thrombosis, labyrinthitis, meningitis and brain abscess. Contiguous or haematogenous spread of infection to the brain produces similar, permanently disabling and potentially fatal complications. **(Vikram *et al*., 2008)**

1.2. 8.Diagnosis of C S O M:

1.2.8.1 History taking:

History taking should be carried out to elicit the symptoms of ear pain, ear discharge, ear tugging or crying when the ear is touched, all of which suggest an ear problem. A history of previous ear discharge, especially when accompanied by episodes of colds, sore throat, cough or some other symptom of upper respiratory infection, should raise the suspicion of CSOM A history of vigorous ear cleaning, itching or swimming that could traumatize the external ear canal suggests acute otitis externa (AOE), and not usually CSOM ,The presence of a central perforation with or without discharge usually makes the diagnosis quite straight forward a history of ear pain suggests AOE or AOM, not usually CSOM in the case of AOM, the ear is only painful until the eardrum perforates, relieving the pressure thus, if the main symptom is painless otorrhoea the duration of otorrhoea will help distinguish AOM from CSOM, every patient most be questioned about the three symptoms of an inner ear disorder(diminished hearing, tinnitus and vertigo) . **(WHO , 2004)**

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1.2.8.2 Otoscopy.

On direct otoscopy the only specific sign of acute otitis media is a bulging, inflamed ear drum. in the absence of bulging, the eardrum must demonstrate acute inflammation and draining. The diagnosis of CSOM rests on the verification of a discharging tympanic perforation. This is

only possible by removing any obstructing wax, ear discharge, debris or masses in the external auditory canal and visualizing the eardrum and, if possible, the middle ear through the perforation. Such an examination requires adequate illumination through a head mirror, head light, otoscope or otomicroscope, suction apparatus . Acute otitis externa and acute otitis media can produce both ear pain and ear discharge. However, tragal tendernessis found in otitis externa, mastoid tenderness in otitis media. The discharge in otitis externa is less profuse and foul-smelling and there is no mucus, as can be tested with a cotton mop by the tendency to form mucus threads. Fever is also higher in otitis media than in otitis externa. CSOM produces painless mucoid otorrhoea without fever, unless accompanied by otitis externa or complicated by an extracranial or intracranial infection.**(Probst *et al.*, 2006)**

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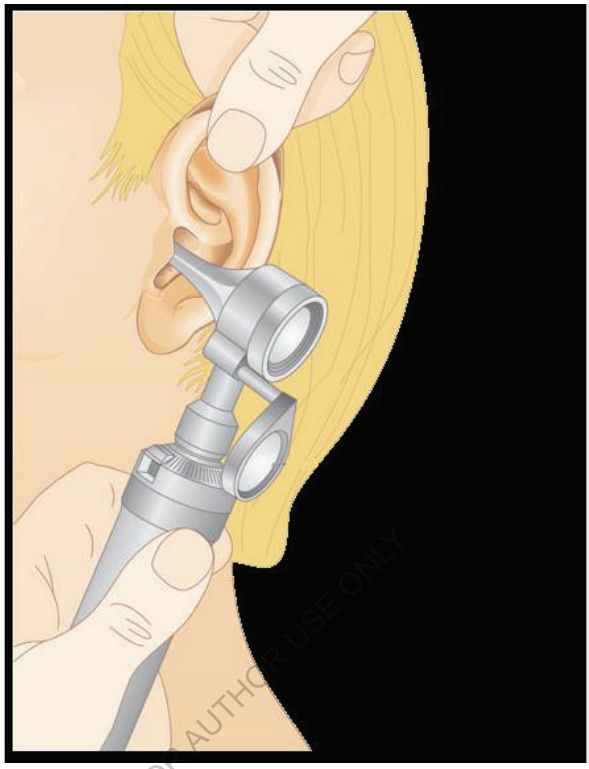

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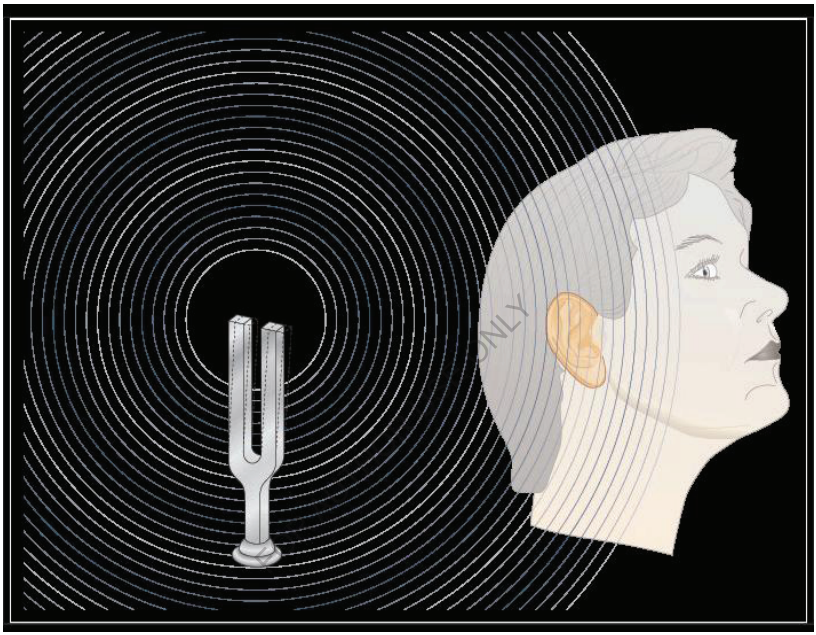
Figure(1-5) Auroscopic examination. (Probst,2006)

1.2.8.3 Tuning fork Examination:

The goal of tuning fork tests is to differentiate between conductive and sensorineural hearing loss. Tow tests are adequate for this purpose: the Weber's test and the Rinne's test.**(Probst *et al*.,2006)**.



Figure(1-6) Tuning Fork Exame to patient with CSOM.

(Probst *et al.*, 2006)

1.2.8.3.1 Rinne's test:

In positive test the tuning fork vibration is transmitted to the cochlea better by air conduction than by bone conduction.this presenst in normal person and those with sensorineural hearing loose ,When conductive hearing loss is present, the sound is perceived as louder on the mastoid than outside the ear canal, the result is negative Rinne's test ,when sensorineural hearing is better on one side than the other, it is necessary to mask the opposite ear before performing the rinne test.**(Yueh, 2003)**

1.2.8.3.2 Weber's test technique:

The tuning fork is placed in the midline of the skull, usually on the vertex or the forehead the vibrations are transmitted by bone conduction to the cochlea.

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When hearing is normal, the vibrations are perceived as equally loud on both sides, and so the sound is heard midway between the ears. In an abnormal test, the sound will be lateralized to one side or the other .If the patient has sensorineural hearing loss, the tuning fork is lateralized to the better- hearing ear.If the patient has a conductive hearing loss, the tuning fork is lateralized to the affected ear because the vibrational energy is more poorly transmitted from the cochlea through the middle ear and it is more difficult for ambient sounds to reach the cochlea (less masking). **(Yueh, 2003)**

1.2.8.4 **Audiological investigations**

1.2.8.4.1 Tympanometry:

Tympanometry is an objective test ,produce apeak when the pressure in the external ear canal equal that of the middle ear ,while varying the pressure in the external ear ,the tympanometery is able to provide information on the status of the middle ear . figure 7 ,showed that type A tympanograms have normal peak height and pressure, two variations of the type A tympanogram also are normal in pressure, but may be shallow (AS), while otosclerosis or middle ear effusion, or peaked very high (AD). Type B, tympanogram is flat in appearance, indicating lack of compliance. The volume measurement that is simultaneously performed with tympanometry helps to differentiate between a flat tympanogram suggesting an intact eardrum with middle ear effusion and a perforated eardrum or patent ventilating tube.Type C, tympanogram has negative peak pressure.was also stated that the majority of tympanometry results, that suggested fluid, were in agreement with a medical finding of fluid**. (Lalwani , 2009)**

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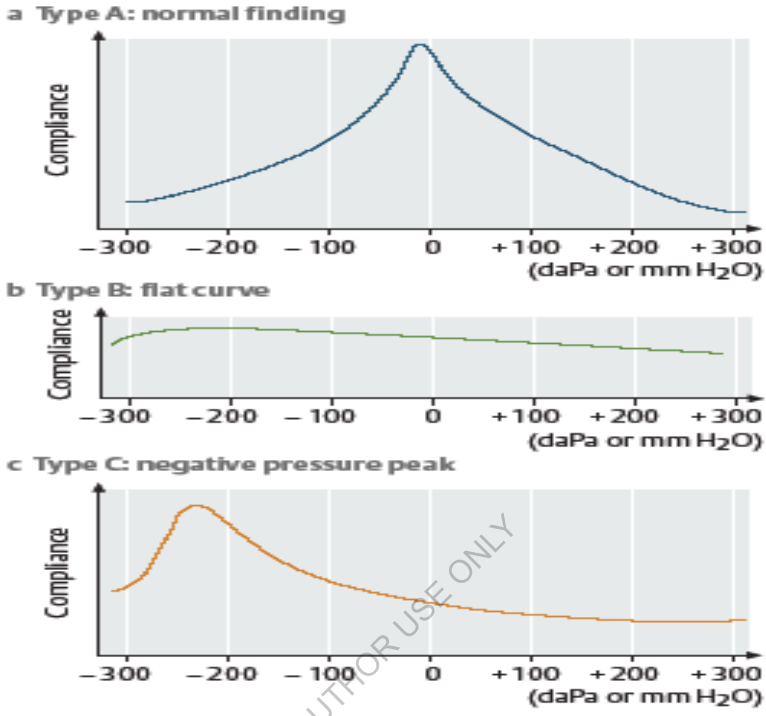

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Figure(1-7) Tympanogram Clearfied normal and abnormal hearing .

(Probst,2006)

daPa=deca Pascal

1.2.8.4.2 Pure Tone Audiometer

An electronic device called an audiometer is used to generate pure tones of varying frequency and loudness , provides an assessment of the severity of hearing losses, frequencies below 125Hz are difficult to distinguish from vibratory sensations; and with tones higher than 8 KHz, the sound pressure level cannot be secretly calibrated with ordinary headphones **.(Howard ,2007 and Ellison,2005)**

Table(1-1) Classification of hearing loss according to severity.(Probst, 2006)

Severty of infection	Hearing loss in dB	Hearing loss in %
Normal hearing	<20 dB	0-20%
Mild hearing loss	20-40 dB	20-40%
Moderate hearing loss	40-60 dB	40-60%
Severe hearing loss	60-90 dB	60-80%
Profound hearing loss	90-110 dB	80-95%
Deafness	>110 dB	100%

dB=deciBell

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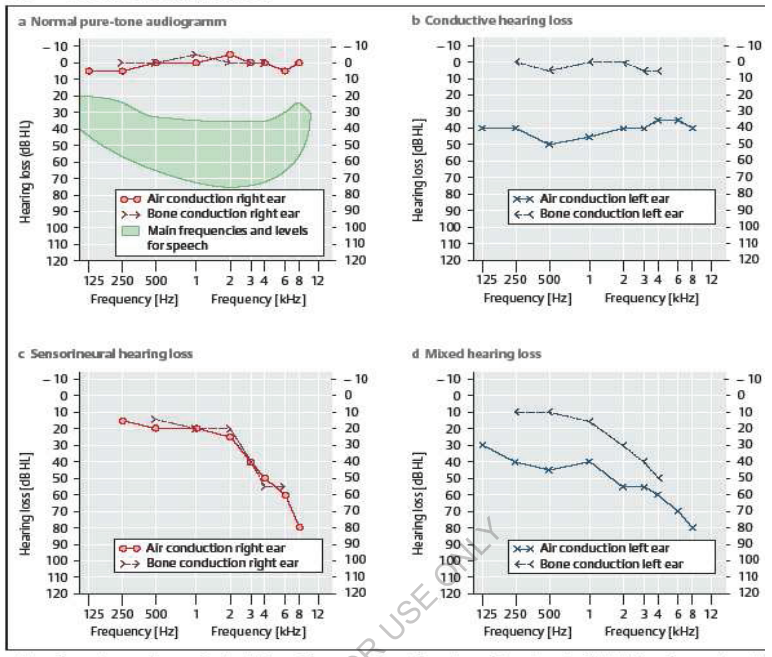

a Normal pure-tone audiogram for the right ear. The range of frequencies and levels that typically occur in conversational speech is shown in green.
b Conductive hearing loss in the left ear.
c Sensorineural impairment with high-tone loss on the right side.
d Mixed conductive and sensorineural hearing loss on the left side.

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Figure (1-8) Pure tone audiogram to identifying type of hearing loose.(Probst *et al.*, 2006)

1.2.8.5. **Bacterial cultures:**

In CSOM, bacterial culture of middle ear secretion is a good diagnostic tool when planning treatment with antibiotics. **(Verhoeff *et al,*. 2006)**

In places where bacterial cultures are available, used to aid in diagnosing CSOM Bacterial cultures may not be needed t o establish the diagnosis of CSOM since exhaustive studies have established that 90–100% of chronic draining ears yield two or more isolates consisting of both aerobic and anaerobic bacteria Also,treatment may eradicate middle ear bacteria but this does not guarantee non-recurrence of otorrhoea or complete resolution of the CSOM.**(Broby,1992)**

Isolated *Pseudomonas aeruginosa* from draining ears in the pretreatment and in the recurrent stage. Some would argue that perforated drums might develop discharge from time to time even without the presence of bacteria and that this does not constitute CSOM which must be treated. In practice, however, patients with draining ears do expect some treatment regardless of culture results. Since topical treatment is often effective and seldom harmful, most experts would start with a wide spectrum antibiotic on an empiric basis and make are quest for cultures if drug resistance is suspected. **(Leiberman *et al*.,1992)**

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1.2.9 Bacteriology of otitis media:

1.2.9.1 Acute otitis media:

The most frequent causative agent of AOM is *Streptococcus pneumoniae* (40%), followed by non type able *Haemophilis influenzae* (25%), *Moraxella catarrhalis* (10%), Group A *Streptococcus* (2%) and *Staphylococcus aureus* (2%).(**Stephenson, 1992**).

Up to 15% of middle ear fluid cultures reveal 2 organisms and findings from the left and right ear may differ. About 20-30% have no

bacterial pathogens identified and presumably are viral in etiology. The emergence of bacterial strains that are increasingly resistant to antimicrobial agents is a growing concern in Canada and worldwide.**(Doern and Simor,1996)**

In appropriate use of antibiotics for viral upper respiratory tract infections has been a major contributor to antimicrobial resistance. (**Mainous , 1992)**

Currently, 25% of *Haemophilus influenzae* and 90% of *Moraxella catarrhalis* produce β-lactamase enzymes which will inactivate penicillin and amino penicillins. Recently, there has been a dramatic increase of multiple antibiotic resistant *Streptococcus pneumoniae*. Currently, approximately 8% of *Streptococcus pneumoniae* isolates in Alberta demonstrate in vitro resistance to penicillin, with 1.4% of these isolates exhibiting high level resistance. In Alberta, resistance to macrolides for *S. pneumoniae* is approximately 9%.**(Block , 1997)**

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1.2.9.2 Acute suppurative otitis media:

The acute form if not recognized early is commonly characterized by suppuration from the middle ear following perforation of the tympanic membrane, Otalgia followed by otorrhea are the commonest symptoms prior to presentation in the hospital, aural drainage is a frequently encountered otolaryngological complaint ,it may be due to a variety of causes

including myringitis ,otitis media with perforation ,infected cholesteatoma**. (Iseh *et al*.,2004)**.

Astudy of 41 patients with acute suppurative otitis media ,found that *Staphylococcus aureus* was 18 (46.2%) *Escherichia coli* was 9 (23.1%) *Proteus species* was 5 (12.8%) *Pseudomonas species* was 5 (12.8%) and *Klebsiella* was 2 (5.1%) were isolated . **(Iseh *et al*.,2004)**

Table (1-2) Type of bacteria in acute suppurative otitis media.

(Iseh *et al*.,2004).

Microorganism	No. (%)
Staphylococcus aureus	18 (46.2)
Escherichia coli	09 (23.1)
Proteus species	05 (12.8)
Pseudomonas species	05 (12.8)
Klebsiella	02 (5.1)

1.2.9.3 Chronic suppurative otitis media:

CSOM can also be differentiated from AOM on bacteriological grounds. In AOM the bacteria found in the middle ear include *Streptococcus pneumoniae*, *Staphylococcus aureus, Haemophilus influenzae* and *Micrococcus catarrhalis*. These are respiratory pathogens that may have been insufflated from the nasopharynx into the middle ear through the Eustachian tube during bouts of upper respiratory infections. In CSOM the bacteria may be aerobic (e.g. *Pseudomonas aeruginosa, Escherichia coli, S. aureus, Streptococcus pyogenes, Proteus mirabilis, Klebsiella* species) or anaerobic (e.g. *Bacteroides, Peptostreptococcus, Proprionibacterium*) .**(Brook, 1996)**

The bacteria are infrequently found in the skin of the external canal, but may proliferate in the presence of trauma, inflammation, lacerations or high humidity. These bacteria may then gain entry to the middle ear through a chronic perforation. Among these bacteria, *Pseudomonas aeruginosa* has been particularly blamed for the deep-seated and progressive destruction of middle ear and mastoid structures through its toxins and enzymes. **(WHO , 2004and WHO , 2000).**

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1.2.9.4.Tuberculous Chronic Otitis Media:

Tuberculous otitis media: is uncommon. However, following global re-emergence of tuberculosis the trend may be on the rise again. The first documentation of the clinical features of this disease was made in 1853 whereas the first isolation of the organism *Mycobacterium tuberculosis spp* from ear discharge was in 1883. It is usually characterized by painless ear discharge on a multi-perforated tympanic membrane and affects all age groups especially children and young adults (84% of cases the diagnosis is confirmed by isolation of the organism from the otorrhoea and treatment is with anti-tuberculous therapy**.(Prasad, 2007).**

1.2.9.5 Syphilitic otitis media:

Syphilitic caused by a spirochete(*Treponema pallidum*) which affects both the cochlear and semicircular canal. Therefore it is usually characterized by severe Sensorineural hearing loss and vertigo. Diagnosis is made through a dark field microscopic examination of the ear discharge. (**Lukehart, 2005)**

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1.2.9.6 Viral Otitis Media:

Viral otitis media is a frequent complication of the common cold. Serous otitis media may occur from Eustachian tube and Eustachian tube lymphatic obstruction of a cold but the virus in many cases may invade the mucosa of the tympanium to produce a secretory otitis media. A special type of viral otitis media involve the development of bullous myringitis. This condition is often complicated by the simultaneous invasion of the tympanum by *hemolytic streptococci*, *pneumococci*,or by *Respiratory syncytial virus*(*R.S.V.*), *influenza* and *parainfluenza virus*, *coronavirus*, *adenovirus*, *enterovirus* . **(Pitkäranta,1998)**

Viral otitis media may be associated with a simultaneous viral labyrinthitis with vertigo, sensorineural hearing loss, and tinnitus. Acute bacterial otitis media can be differentiated from an complicated viral otitis media by the presence of fever and a positive culture that occur with the bacterial infection in the presence of an intact tympanic membrane , the most likely source of the middle ear pathogens is the nasopharynx. Respiratory viruses may potentiate the possibility of nasopharyngeal colonization with bacteria, further increasing the incidence of otitis media . **(Peter, 2009)**

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In previous studies 25 to 30% of middle ear fluid cultures are negative for bacteria, some of which are positive for viruses, including *rhinovirus*, *adino virus*, *influenza virus* , *parainfluenza virus*, and *respiratory syncytial virus* (RSV). Acute OM has been documented in 20 to 50% of hospitalized patients with laboratory-confirmed respiratory viral infection. (**Heikkinen *et al*, 1999**),

(Uhari *et al*.,1995) found that *respiratory syncytial virus* is the most commonly identified virus, being found in 74% of the middle ear isolates,

followed by *parainfluenza virus* and *influenza virus*, also it has been shown that vaccination against influenza virus decreased the incidence of acute otitis media in infants and children**.**

1.2.9.7 Otomycosis:

Otomycosis is used to describe mold or yeast infection of the external auditory canal ,*Aspergillus* and *Candida* more common species isolates of patients with otomycosis.**(Chen,1999 and Loh,1998)**.

Whenever fungi are found in the ear,they present in colonization or clinically relevant infection,persisting discharge with maceration of the meatal epithelium may support fungal colonization of the external ear in patients with otitis media ,the demonstration of conidiophores (*Aspergillus* head) in the auditory canal is consistent with the hypothesis that mucous discharge severs as nutrient .Clinically relevant infection are accompanied by inflammation . **(Vennewald *et al.*, 2003**)

A primary infection may arise from oropharynx via the auditory tube .Infection by mold ,in contrast to bacteria and yeasts ,is only possible after perforation of ear drum .**(Borkowski,2000)**

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1.2.10 Mangement of CSOM.

Management are the eradication of infection and the closure of the tympanic perforation. Both are important. While the abiding presence of pathologic bacteria within the middle ear and mastoid cavities accounts for the mortality and severe morbidity associated with CSOM, the persistent tympanic perforation represents unrelieved hearing loss and the constant threat of microbial invasion of the middle ear. The use of topical vasoconstrictors/ nasal decongestants to reduce otorrhoea the oedema in the Eustachian tube, nasal and the middle ear mucosa and thus encourage aeration of the middle ear and the use of appropriate antibiotics to treat

underlying infections are recommended.**(Verheoff, 2006 ,McCracken ,1998 and Lundy *et al*., 1993)**

1.2.10.1 Primary Ear care.

Primary ear care and case management programmes for CSOM in high-risk populations in developing countries have been shown to be associated with a reduction in frequency of CSOM and mastoiditis COM can be prevented by treating AOM well. The current WHO treatment recommendations for a 2 month to 5 year old child with an acute ear infection (ear pain and/or pus draining from the ear for less than 14 days) are to give an appropriate antibiotic for 5 days.**(Mustafa ,2008 and Hassmann, 2007).**

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1.2.10.2 Ear toilet.

Ural toilet is no better than no treatment,there is no consensus among general and specialist physicians with regard to themedical management of CSOM.However,there is general agreement that auraltoilet must be part ofthe standard medical treatment for CSOM.Cleaning the ear of mucoid discharge could reduce,even if temporarily,the quantity ofinfected material from the middle ear and could facilitate middle ear penetration oftopical antimicrobials.(**Dhooge *et al.*,2005, Brobby, 1992 and Ludman ,1980**)

1.2.10.3 Role of antiseptics and antibiotics .

Antiseptic ear drops (2% acetic acid with 0.5% prednisolone) have been found to be effective against *pseudomonas* and fungi and less painful than 50% spirit drops. Drops containing neomycin, polymyxin and a steroid are often available but may not be effective. The newer 4-quinolone antibiotics (ofloxacin, norfloxacin, or profloxacin) are likely to

become the most effective topical antibiotics**.(Verhoeff, 2006 and Acuin,2004).**

1.2.10.4 .Surgical Interventions:

When conservative management is not sufficient for the resolution of the problem, then surgery is indicated in from of Tympanoplasty. (**Matrin *et al* , 2007 and Acuin,2004).**

1.3 AIMS OF THE STUDY

1.Evaluate the hearing ability on patients with CSOM.

2.To study the epidemiological(demographical) factors associated with CSOM.

3.Identify the bacterial pathogens associated with CSOM.

4. Study the antibiotic susceptibility pattern of antibiotic against bacterial pathogen.

5.Determined the mode of bacterial isolation and multi drugs resistant bacteria.

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Chapter Two

Material and Method

2.1.Patiens.

A total of 120 patients with CSOM were included in this study .The clinical study include history-taking ,otoscopic examination ,tuning fork examination,audiological investigation (pure tone audiometry and tympanometry .While, Microbiological investigation include(culture ,identification of causative agents and antibiotic sensitivity.The study was carried out in Basrah General hospital out patients E.N.T. clinic under supervision of specialists of ENT,during the period from March 2009 January 2010,

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2.2 Grouping of patients with CSOM.

The studied patients were divided into five groups according to **(Charman &William,2002).**These group are

1. Infantil group:less than two years.
2. Childhood group from 2 to<11 years.

Adulthood group :more than 11years.and then subdivided into.

3. 11 to <20 years.
4. 20 to<30 years
5. over than 30 years.

2.3 Control goupe.

A total of 60 individuals without otological problems ,30 males and 30 females in various age group,they were regarded as a control group .

2.4 Clinical and epidemiological (demographical) studies .

Various clinical and demographical parameters were included in this study these includs:

1. Age.
2. Sex.
3. Family history:
4. Degree of hearing loos. Conductive, enserineural, ixed,). **Probes. 006)**
5. Degree of severity: (mild, modrate,sever) .(Abdel-Hamid,2007)
6. Type of feeding (breast and bottle feeding) .
7. Mode of smoking.
8. Living modes of patients.

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2.5.Clinical examination techniques.

1. Otoscopical examination . is necessary to differentiate CSOM from another types(e.g acute otitis media ,otitis media with effusion,otitis external) .**(Uhari *et al.*,1995)**
2. Tuning fork examination by using . **(** Rinne's test & Weber's test). **(Finitzo *et al.*,1992)**
3. Audiological investigation by using , Pure tone audiometry and tympanometry . **(Finitzo *et al.*,1992)**

2.6 .Bacteriological study:

2.6.1 **Sampling.**

Two groups were included in this study:

Group(1) 120 aural swabs were taken from infected ear of CSOM patients .

Group(2) 60 aural swab taken from a control group

Swabs were taken under sterile condition and transfer immediately to the laboratory by brain heart broth for aerobic bacteria ,thioglycollate broth for anaerobic bacteria ,and cultured on suitable media at 37^0C for 24- 48 hours.

Primary isolation on (Blood agar, chocolate agar, nutrient agar), accor then on selective media identification and biochemical characterization were carried out according to standard routin techniques. (Fingole and Baron,2002).

2.6.2 Microbiological Media (culture media).

1-Blood agar base. (HI media).

Suspend 40gm in 1000ml D.W.,heat to boiling to dissolve the medium completely, after sterilization the media cooled to 45- 50^0c and aseptically added of 5% sterile blood then mix well and pour into sterile petridishs.

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2-Macconkey agar. (Hi media).

Suspend 51.5gm in 1000ml D.W.

3-Mannitol salt agar. (Hi media).

Suspend 111gm in 1000ml D.W.

4-Muller-Hinton agar. (HI media).

Suspend 38gm in 1000ml D.W.

5-Sabouraud's dextrose agar. (HI media).

Suspend 65gm in 1000ml of D.W.

6-Kligler iron agar . (HI media).

Suspend 52gm in 1000ml D.W.

7-Brian heart infusion. (HI media).

Suspend 37gm in 1000ml ,

8-Thioglycolat infusion. (HI media).

Suspend 29gm in 1000ml D.W. boiling to dissolve the medium completely ,pouring in screw cap tube (5ml for each tube).

Note: All media sterilized by autoclave (121^0c under 15 Ibs pressure for 15 minutes)

9-Rabbit plasma (Biomerieux).

Commercial supplied used for coagulase test

2.6.3 Antibiotics disc.

1-penicillin G 10mg (Bioanalyse).

2-Erythromycin 15mg (Bioanalyse).

3-Tetracycline 30mg (Bioanalyse).

4-Ciproflaxin. 5 mg (Bioanalyse).

5-Gentamicine 10mg (Bioanalyse).

6-Ampicillin 10mg (Bioanalyse).

7-Augminten 20 mg (Bioanalyse).

8-Trimethoprim 25mg (Bioanalyse).

9-Streptomycin 10mg (Bioanalyse).

10-Lincomycin 2mg (Bioanalyse).

11-Optochin disc.(Ethyl-hydrocupreine hydrochloride). 5mg (Bio analyse).

Used for diagnosis of *Streptococcus pneumonia* .

12-Bacitracin disc . (Bio analyse).

Used for diagnosis *Streptococcus viridance*.

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2.6.4.-Stains.

2.6.4.1.Gram stain. (Al-Hadithi&Al-Saimary,1993)

Stock crystal violet(solution A).Crystal violet 10g, Ethanol (95%) 100ml, dilute the stock crystal violet solution in 1:10 with D.W.

Ammonium oxalate (solution B) , 1g in 100 ml D.W.

Working solution :mixed solution A with solution B in 1:4 volum solution.and stored in dark bottle.

2-Lugol's iodine solution: Iodine 1g,Potassium iodide 2g. Dissolve completely in 300 ml D.W ,

3-Decolorizer.Ethanol (95%)

4-Counter stain: Safranin (2.5g), Ethanol (95%) 100ml

Dilute stock safranin 1:10 with D.W.

2.6.4.2.Zhiel-Nelson stain.

Carbol-fuchsin solution:(5gm of basic fuchsine dissolve in 95ml absolute alcohol,add to 45gm of phenol dissolve in 950ml of D.W.).mix well the stored in dark bottle.

Decolorizer: 20%H2SO4(20ml concenterated H2SO4 mixed with 80ml D.W.).

Counter stain: methylene blue (1-2gm dissolve in 1000ml D.W.) **. (Baily and Scotte.2002).**

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2.6.4.3.Albert stain.

Albert solution 1(Toludine blue 0.15gm,malachite green o.2gm,these are dissolved in 2ml of 95% alcohol ,then add to 100ml of D.W. containing 1ml of glacial acetic acid ,filtered and become ready for use).

Albert solution 2(iodine 2gm,potassium iodide 3gm,300ml D.W.)

2.6.5 Biochemical test.

The following biochemical test were done according to procedures of Mackey &MacCartney,(2005), Fingold & Baron ,(2002)

2.6.5 .1.Catalase test:

Add 0.5ml of 3%H2O2 solution to bacterial growth transferred from blood agar plate to slid . Bubbles of gas were appear when bacteria gave catalase , (positive reaction)

2.6.5 .2.Coagulase test:

A loopful of culture or 2drops of broth culture with 0.5ml of plasma,let stand up to three hours in the incubator at 37^0C then examine for clotting .

2.6.5 .3-Oxidase test:

Indicator(1% tetramethyl-p-phenylene diamine-oxidase reagent). converted culture colour to dark purple color within (2-5)seconds.Used to diagnosis of *Pseudomonas spp.*

2.6.5 .4. Indol test:

Inoculate tryptophane broth with 1 drop of culture ,incubate for 48hour,add 0.5ml kovacs reagent.This test done for genus of Enterobacteriaceae.

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2.6.5 .5.Kligler's iron test:

Acid and gas formation as bubbles in the medium formed by fermentative gas producing bacteria,black color precipitate synthesis by H2S producing bacteria such as *Proteus* spp**.**

2.6.5 .6.Citrate utilization test:

The slant of Simmon's citrate agar medium was inoculated with culture for (24) hours at 37^oc.The both blue color and growth indicated positive result.

2.6.5 .7.Methyl red test:

MRVP medium was inoculated culture for 24 hrs, then 5drops of methyl red was added. A red color indicated positive reaction .

2.7 Statistical analysis:

In order to determine the statistical significance among different variables, SPSS program(statistical program for social sciences) ver.11,was used for this purpose.

The following statistical testes were performed:

Chi-square (x^2)test, univariate and multivariate logistic regression analysis and the difference between two proportions by T-test were used to assess the significance of difference between groups .P-value less than 0.05 was considered as statistically significant (S),P-value<0.01 as highly significant .(HS), P-value <0.001 as extremely significant (ES). P-value more than 0.05 was considered as statistically not significant (NS).

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Chapter three

Results

Atotal number of (120) patients with chronic suppurative otitis media (C.S.O.M) were atteneded to the Basrah General Hospital (out patients clinic of E.N.T), 120 aural swabs were taken from all patients . Patients were distributed according to age and sex, There were 65(54.16 %) males and 55(45.83%) females,and males to females ratio was 1.2:1 ,the age of patients was between (1-60) years and 60 individuls regarded as control without otological problems were also studied .

3.1 Distribution of the patients with CSOM according to age.

Table (3-1) shows that the majority of our patients belong to age group 2-11 years (25.38%) ,followed by age group 20-30 years 26 patients (21.2%) was a higher than adult patients over than 30 years (14.16%) . P<0.01.

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Table (3-1) Distribution patients with CSOM according to age groups.

Age groups	Male Patients No.(%)	Female Patients No.(%)	Total
Infantile group< 2 years.	*12(10)	8(6.66)	20
Childhood group from 2 to<11 years.	16(13.33)	15(12.5)	31
Adulthood group 11 to< 20 years	15(12.5)	11(9.16)	26
Group 20 to<30 years(adulthood)	11(9.16)	15(12.5)	26
Group over than 30 years	11(9.16)	6(5)	17
Total	65(54.16)	55(45.83)	120

P<0.01

3.2 Distribution of the patients with CSOM according to sex .

Table (3-2) illustrated that the majority of patients with CSOM was 65(54.16%) males belong to age (1-9) years , and females was 55(45.83%) ,statistically there was no difference between patients with CSOM at same ages (P>0.05).

Table (3-2) Distribution of patients with CSOM according to sex.

Age (years)	NO of patients (%)	Male (%)	Female (%)
1-04	20 (16.66)%*	12(10)%	8(6.66)%
05-09	29(24.16)%	14(11.66)%	15(12.5)%
10-14	22(18.33)%	13(10.83)%	9(7.5)%
15-19	8(06.66)%	5(04.16)%	3(02.50)%
20-24	10(08.33)%	3(02.50)%	07(05.83)%
25-29	8(06.66)%	3(02.50)%	5(04.16)%
30-34	4(03.33)%	3(02.50)%	1(00. 83)%
35-39	4(03.33)	3(02.50)%	1(00. 83)%
40-44	4(03.33)%	1(00.83)%	3(02.50)%
45-49	4(03.33)%	3(02.50)%	1(00. 83)%
50-54	4(03.33)%	3(02.50)%	1(00. 83)%
55-60	3(02.50)%	2(01.60)%	1(00. 83)%
Total	**120(100)%**	**65(54.16)%**	**55(45.83)%**

X^2=0.64 , *: p>0.05.

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3.3 Geographical distribution of Patients with CSOM .

The prevalence of patients with CSOM at peripheral area from Basra was 25% ,while at central area of Basra was 59% ,While in rural the prevalence of disease was 16.7% which less than peripheral and central area .Table (3- 3) illustrate that frequency of CSOM patients in central area of urban was highly statistical differences from that of peripheral and rural area (P<0.01).

Table(3-3) Prevalence of patients with CSOM according to geographical area .

Geographical area		**No. of patients**	**(%)**
Urban	Peripheral area	30*	25
	Central area	70	59
Rural		20	16.7
Total		120	100

*(**P<0.01**)

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3.4 Clinical features .

Table(3- 4) shows that the majority of clinical feature were otorrhoea (100)%.Otalgia 50(41.7%),Pad odor from ear 30(25%),ear itching 45(38%), ,tinnitus 60(50%) ,vertigo 30(25%) and fever 50(41.8%).

Table(3-4) Clinical features of patients with CSOM.

Clinical Feature	No. of patients %
-Otorrhea.	120(100)*
-Otalgia	50(41.8)
-Pad odor from ear	30(25)
-Ear itching	45(38%).
- (tinnitus)	60(50)
- (vertigo)	30(25)
-fever	50(41.8)

***P<0.001**

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3.5 Tuning fork examination .

Including Rinne's test and weber's test . Table 5 shows that the frequency of Rt ears was significantly higher than Lt ear and conductive hearing loss more than senserineural and mixed hearing loss among CSOM patients (25-31%) patients Rt ear +Ve,while (18.33-25%)of patients Lt ear +ve.

In Rinne test 18.3% of left ear patients were normal hearing , 31.7% of right ear patients were normal hearing and 25% of both patients right and left ears normal hearing, while in Weber test ,20.9% of patients have sound (vibration) of tuning fork shifted to left side it means effect in left ear ,25% of patients have centeral (sound of tuning fork don't shifted to right or left ear) it means normal right and left ears ,and 29.2% of patients have sound of tuning fork shifted to right side it means effect in right ear. In another way Rinne test was useful to detecte the type of hearing loss by evaluation the difference between air and bone conduction in each ear,if patient still heared the sound ,but bone conduction is better than air conduction (negative Rinne)the patient has conductive deafness.If no longer hears the sound (positive Rinne) so,has either normal hearing or asensory deafness. Also Weber test useful to detected type of hearing loss ,The sound immediately lateralizes to the blocked ear,the weber test tends to lateralize towards the side of aconductive loss and away from sensorineural loss.

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Table(3- 5) Tuning fork examination results for patients with CSOM.

Rinne's test	NO. of patients (%)	Weber's test	NO. of patients (%)
Rt -ve test	22* (18.33)	Shifted + Lt	25*(20.83)
Lt -ve test.	38 (31.66)	Shifted + Rt	35 (29.16)
Rt+Lt +ve test	30 (25)	centeral	30(25)
Non applicable	30 (25)	Non applicable	30(25)
Total	120 (100)	Total	120 (100)

Non applicable =(children under 5 years).
*** Rinne's test x^2=1.28 P>0.05**

*** Weber's test x^2=1.3 P>0.05**

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3.6 Unilateral and bilateral hearing loss .

Hearing loss either unilateral or Bilateral in our study for 80% of patients was unilateral, while for 20% was bilateral ,the frequency of right ear hearing loss was 60% and 40%for left ear hearing loss. It means that right ear more affected than left ear in our study tuning fork and PTA use for diagnosis.

Table (3-7) shows that the frequency of unilateral hearing loss was higher than bilateral hearing loss (P<0.01).

Table(3-6) Unilateral and bilateral hearing loss

Age group	Bilateral H.L. (Rt andLt ear discharged) No. ofpatients (%)	Unilateral H.L. No. of patients(%)	
		Rt ear discharged	Lt ear discharged
01-04	10 (8.4)**	15 (18.9)*	10 (12.4)
05-14	8 (6.7)	7 (8.8)	8 (10.0)
15-24	8 (6.7)	6 (07.4)	4 (05.0)
25-34	6 (5.0)	8 (10.0)	2 (02.5)
35-44	4 (3.4)	4 (5.0)	4 (05.0)
45-54	2 (1.7)	4 (5.0)	2 (02.5)
55-65	2 (1.7)	3 (3.8)	3 (03.8)
Total	**40 33.4**	**47 39.2**	**33 27.4**

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**** P<0.01**

3.7 Normal flora.

Table (3-8) show results of isolated bacterial from(60) healthy persons. The following bacteria were isolated ,*Staphylococcus epidermidis* 20 isolates (40%),flowed by *Corynebacterium* species 15 isolate (30%) .Other types distributed accoding to species in table 3-8. 10 samples gave negative result for bacteria culture (16.66%).

Table(3-7) Normal flora

Microorganisms	**No.of isolates**	**(%)**
Klebsiella spp.	2*	4
Strept. Spp.	3	6
E.coli spp.	2	4
Bacillus spp.	8	16
Staph.epidemedis.	20	40
Corynebacterium spp .	15	30
No growth	10	16.66
Total	**60**	**100%**

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3.8Pathogenic Bacteria Isolated From Patients With CSOM.

The occurance of difference bacterial isolate among CSOM patients presented *Pseudomonas aeruginosa* was more frequently isolates 40(19.41%),while *Staph. aureus* followed *Pseudomonas* 35 (16.99%), *Klebsiella* 30(14.56%) ,*Br. catarrhalis* 20 (9.70%), *Proteus* 20(9.70%), *H.influenzae* 20 (9.70%), *strept.* spp 15(7.28%), *E.coli* 10(4.85%), *Corynebacterium* 8 (3.88), and *Bacillus* 8 (3.88).

Table(3-8) Bacterial type isolated from patients with CSOM

Caustive agents	No .of isolates	(%)
Ps.aeruginosa.	40*	19.41
Staph.aureus.	35	16.99
Klebsiella spp.	30	14.56
Br.catarrhalis.	20	9.70
Proteus spp.	20	9.70
H.influenzae.	20	9.70
Streptococcus spp.	15	7.28
E.coli spp.	10	4.85
Corynebacterium spp.	08	3.88
Bacillus spp.	08	3.88
Total No. of isolates	**206**	**100**

** x^2=49.8 P<0.01

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3.9 Bacterial caustive agents and hearing loss

The occurance of various caustive agents isolates among CSOM patients in four different hearing loss is presented in table(3-10).*Ps. aeruginosa* was more frequently isolated in senserineural and profound hearing loss (25-26.2%),while in conductive and mixed hearing loss (16.7-20.4%) *Staph.aureus* isolates , appeared more frequently among CSOM patients with conductive and mixed hearing loss(20.4-25%) than in senserineura and profound hearing loss (12.5-15%) *Klebsiella* species and the other organisms isolated in verious percentage from these four hearing loss.

Table(3-9) Relationship between caustive agents and hearing loss.

Causative agent	NO. of Patients with Hearing Loss.			
	No. of isolates	CHL	SNHL	MXHL
Ps.aeruginosa	40	5	12	١٠
Staph .aureus	35	6	١٠	٩
Klebsiela spp	30	٤	١٠	٨
B.catarrhalis	20	٥	٨	٣
Proteus spp	20	٨	٦	٣
H.influenzae	20	٦	٤	٥
Strept.spp	15	٤	٦	٢
E.coli	10	٣	٤	١
Corynebacterium spp	8	٣	٣	١
Bacillus spp	8	٢	٣	١

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Table(3.10) Standard antibiotic susceptibility test according to diameters of inhibition zone supplied by Bio analysis Company.

Antimicrobial agent	symbol	Conc. mcg	Zone diameter (mm).	
			Sensitive	Resistant
Ciprofloxacin.	(cip).	10mcg.	20 or less.	29 or more.
Amoxicillin+clavulanic acid.	(AMC).	20mcg.	19 or less.	20 or more.
Gentamicin.	(CN).	10mcg.	10 or less.	15 or more.
Vancomycin.	(VA).	30mcg.	9 or less.	12 or more.
Lincomycin.	(L).	2mcg.	9 or less.	15 or more.
Cephalexin.	(CL).	30mcg.	14 or less.	18 or more.
Penicillin.	(p).	10mcg.	11 or less.	22 or more.
Erythromycin.	(E).	15mcg.	13 or less.	18 or more.
Ampicillin.	(AM).	10mcg.	(11-21)or less.	(14-30) or more.
Tetracycline.	(T).	30mcg.		19 or more.
Streptomycin.	(s).	10mcg.	14 or less.	15 or more.
Trimethoprim+	(SXT).	1.25mcg	11 or less.	16 or more.
sulphamethoxazole.		23.75mcg.	10 or less.	

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3.11 Antibiotic sensitivity of *Psedomonas aeruginosa*.

Table (3-11) Show that the frequency of Ciprofloxacin, Amoxicillin + clavulanic acid (Augmentin) and Gentamicin.W ere statistically significantly higher than other types of antibiotics.P<0.01 in percentages of sensitivity between (50- 75%).(P<0.01),while 88% of *Ps.aeruginosa* isolates was resist trimethoprim ,85% to Streptomycin,and 80% to Vancomycin,while other pattern of resistance were between 25-78% of various antibiotics.P<0.01.

Table (3-11) Antibiotic susceptibility pattern of *Psedomonas aeruginosa* .

Drugs type	No. of isolated	Sensitive (%)	Resistant(%)
Ciprofloxacin	40	30*(75)	10 (25)
Augmentin	40	21(52.5)	19(47.5)
Gentamicin	40	20(50)	20(50)
Vancomycin	40	8(20)	32(80)
Lincomycin	40	9(22.5)	31(77.5)
Cephalexin	40	11(27.5)	29(72.5)
Penicillin	40	10(25)	30(75)
Erythromycin	40	12(30)	28(70)
Ampicillin	40	14(35)	26(65)
Tetracycline	40	13(32.5)	27(67.5)
Streptomycin	40	6(15)	34(85)
Trimethoprim	40	5(12.5)	35(87.5)

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X^2=25 P<0.01

3.12 Antibiotic sensitivity of *Staph.aureus* .

Table (3- 12) shows that in each drugs group the frequency sensitivity of Ciprofloaxacin,Augmenten,Cephalexin and Pencillin,(57-80%) were statistically significantly higher sensitive than other antibiotic . (P<0.01),while 83% of *Staph.aureus* isolates was resist trimethoprim ,83% to Streptomycin,and 83% to Vancomycin,while other pattern of resistance were between 20-77% of various antibiotics.P<0.01.

Table(3-12) Antibiotic susceptibility pattern of *Staph.aureus*

Drugs type	No. of isolated	Sensitive (%)	Resistant (%)
iprofloxacin	35	20*(57.15)	15(42.85)
Augmentin	35	20(57.15)	15(42.85)
Gentamicin	35	15(42.85)	20(57.15)
Vancomycin	35	6(17.14)	29(82.86)
lincomycin	35	8(22.85)	27(77.15)
Cephalexin	35	20(57.15)	15(42.85)
Penicillin	35	12(34.28)	23(65.72)
Erythromycin	35	28(80)	07(20)
Ampicillin	35	10(28.57)	25(71.43)
Tetracycline	35	10(28.57)	25(71.43)
Streptomycin	35	6(17.14)	29(82.86)
Trimethoprim	35	6(17.14)	29(82.86)

T=6.9 P<0.01

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3.13 Antibiotic sensitivity of *Klebsiella* spp .

Table(3-13)shows that in each drugs group the frequency of sensitivity of Ciprofloxacin and Augmentine (67-70%)were statistically significantly higher than other type of antibiotic drugs. (P<0.01),while 73% of *Klebsiella spp* isolates was resist trimethoprim ,70% to Erythromycin,and 80% to Vancomycin,while other pattern of resistance were between 30-73 % of various antibiotics.P<0.01.

Table(3- 13) Antibiotic susceptibility pattern of *Klebsiella spp*

Drugs type	No of isolated	Sensitive (%)	Resistant(%)
iprofloxacin	30	20* (66.66)	10 (33.34)
Augmentin	30	21(70)	9(30)
Gentamicin	30	16(53.33)	14(46.67)
Vancomycin	30	06(20)	24(80)
lincomycin	30	08(26.66)	22(73.34)
Cephalexin	30	12(40)	18(60)
Penicillin	30	10(33.34)	20(66.66)
Erythromycin	30	09(30)	21(70)
Ampicillin	30	11(36.66)	19(63.34)
Tetracycline	30	10(33.34)	20(66.66)
Streptomycin	30	10(33.34)	20(66.66)
Trimethoprim	30	08(26.66)	22(73.33)

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X^2=25 P<0.01

3.14 Antibiotic sensitivity *Branhamella catarrhalis*.

Table(3-14) shows that in each drugs group Ciprofloxacin, Augmentin, Cephalexin, Ampicillin, Gentamicin were statistically significantly higher sensitivity (50-75%) against *Branhamella* spp than other type of antibiotic . (P<0.01),while 75% of *Branhamella* spp isolates was resist trimethoprim ,70% to Streptomycin,and 70% to Vancomycin, while other pattern of resistance were between 25-60of various antibiotics. P<0.01.

Table(3-14) Antibiotic susceptibility pattern of *Branhamella spp*

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Drugs type	No . of isolated	Sensitive (%)	Resistant(%)
Ciprofloxacin	20	15* (75)	05 (25)
Augmentin	20	15(75)	05(25)
Gentamicin	20	10(50)	10(50)
Vancomycin	20	06(30)	14(70)
lincomycin	20	08(40)	12(60)
Cephalexin	20	12(60)	08(40)
Penicillin	20	10(50)	10(50)
Erythromycin	20	09(45)	11(55)
Ampicillin	20	11(55)	09(45)
Tetracycline	20	10(50)	10(50)
Streptomycin	20	06(30)	14(70)
Trimethoprim	20	05(25)	15(75)

X^2=25 P<0.01

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3.15 Antibiotic sensitivity *Proteus* spp .

Table (3-5shows that in each drugs group the frequency of Ciprofloxacin ,Augmentin,Gentamicin and Trimethoprim were statistically significantly higher effective against *Proteus spp* than other type of Antibiotics, (60-70%) sensitive . (P<0.01),while 75% of *Proteus* spp isolates was resist Ampicillin ,70% Erythr omycin,and 75% to Penicillin,while other pattern of resistance were between 30-60% of various antibiotics.P<0.01.

Table(3- 15) Antibiotic susceptibility pattern of *Proteus spp*

Drugs type	No. of isolated	Sensitive (%)	Resistant(%)
Ciprofloxacin	20	14*(70)	06(30)
Augmentin	20	12(60)	08(40)
Gentamicin	20	12(60)	08(40)
Vancomycin	20	10(50)	10(50)
lincomycin	20	10(50)	10(50)
Cephalexin	20	08(40)	12(60)
Penicillin	20	05(25)	15(75)
Erythromycin	20	08(40)	12(60)
Ampicillin	20	05(25)	15(75)
Tetracycline	20	10(50)	10(50)
Streptomycin	20	08(40)	12(60)
Trimethoprim	20	10(50)	10(50)

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X^2 =25 P<0.01

3.16 Antibiotic sensitivity *Heamophilus influenzae*

Table(3-16) shows that in each drugs group the frequency of Ciprofloxacin,Augmentin,Gentamicin ,Vancomycin and Lincomycin (50-75%) were statistically significantly higher sensitive drugs against *Heamophilus influenzae* than other . (P<0.01),while 80% of *Heamophilus influenzae* isolates was resist to Streptomycin,70% Tetracyclin,and 70% to Ampicillin,while other pattern of resistance were between 25-65% of various antibiotics.P<0.01.

Table(3-16) Antibiotic susceptibility pattern of *Heamophilus spp*

Drugs type	No. of isolated	Sensitive (%)	Resistant(%)
iprofloxacin	20	15* (75)	5 (25)
Augmentin	20	12(60)	8(40)
Gentamicin	20	12(60)	8(40)
Vancomycin	20	10(50)	10(50)
Lincomycin	20	10(50)	10(50)
Cephalexin	20	8(40)	12(60)
Penicillin	20	8(40)	12(60)
Erythromycin	20	10(50)	10(50)
Ampicillin	20	6(30)	14(70)
Tetracycline	20	6(30)	14(70)
Streptomycin	20	4(20)	16(80)
Trimethoprim	20	7(35)	13(65)

X^2= 25 P<0.01

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3.17Antibiotic sensitivity *Streptococcus* spp.

Table (3-17) shows that in each drugs group the frequency of Ciprofloxacin ,Augmentin,Pencillin,Erythromycin and tetracycline were statistically significant higher sensitive(67-80%) than other type of Antibiotics.(P<0.01),while 60%, of *Streptococcus* spp isolates was resist to Trimethoprim , Streptomycin,53.33%,and 46% to Ampicillin ,while other pattern of resistance were between 20-40% of various antibiotics.P<0.01..

Table(3-17) Antibiotic susceptibility pattern of *Streptococcus spp*

Drugs type	No. of isolated	Sensitive (%)	Resistant(%)
Ciprofloxacin	15	12* (80.00)	3(20.00)
Augmentin	15	10(66.66)	5(33.34)
Gentamicin	15	10(66.66)	5(33.34)
Vancomycin	15	9(60.00)	6(40.00)
Lincomycin	15	9(60.00)	6(40.00)
Cephalexin	15	10(66.66)	5(33.34)
Penicillin	15	10(66.66)	5(33.34)
Erythromycin	15	10(66.66)	5(33.34)
Ampicillin	15	8(53.33)	7(46.67)
Tetracyclin	15	9(60.00)	6(40.00)
Streptomycin	15	7(46.67)	8(53.33)
Trimethoprim	15	6(40.00)	9(60.00)

X^2=10.8 P<0.01

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3.18 Antibiotic sensitivity *E. coli*.

Table(3-18) shows that in each drugs group the frequency of Ciprofloxacin,Augmentin, Gentamicin ,L incomycin and Cephalexin were statistically significantly higher sensitive drugs (60-80%)against *E.coli* than other type of drugs . (P<0.01),while 80%, of *E.coli* spp isolates was resist to Streptomycin,60% to Trimethoprim , and 60% to Ampicillin ,Erythromycin and penicillin ,while other pattern of resistance were between 20-40% of various antibiotics.P<0.01..

Table(3-18) Antibiotic susceptibility pattern of *E.coli* .

Drugs type	No. of isolated	Sensitive (%)	Resistant(%)
Ciprofloxacin	10	8* (80)	2(20)
Augmentin	10	8(80)	2(20)
Gentamicin	10	8(80)	2(20)
Vancomycin	10	6(60)	4(40)
Lincomycin	10	6(60)	4(40)
Cephalexin	10	6(60)	4(40)
Penicillin	10	4(40)	6(60)
Erythromycin	10	4(40)	6(60)
Ampicillin	10	4(40)	6(60)
Tetracyclin	10	4(40)	6(60)
Streptomycin	10	2(20)	8(80)
Trimethoprim	10	4(40)	6(60)

X^2=25 P<0.01

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3.19 Antibiotic sensitivity *Corynebacterium spp.*

Table(3-19) shows that in each drugs group the frequency of Ciprofloxacin,Cephalexin,Erythromycin,Ampicillin and Penicillin were statistically significantly higher sensitive drugs (75%)against Corynebacterium spp. (P<0.01),while 63%, of ***Corynebacterium*** spp isolates was resist to Lincomycin ,63% to Vancomycin and 50% to Gentamicin &Cephalexin ,while other pattern of resistance were between 25-38% of various antibiotics.P<0.01..

Table(3-19) Antibiotic susceptibility pattern of *Corynebacterium spp*

Drugs type	No . of isolated	Sensitive (%)	Resistant(%)
Ciprofloxacin	8	6* (75.0)	2(25.0)
Augmentin	8	6(75.0)	2(25.0)
Gentamicin	8	4(50.0)	4(50.0)
Vancomycin	8	3(37.5)	5(62.5)
Lincomycin	8	3(37.5)	5(62.5)
Cephalexin	8	4(50.0)	4(50.0)
Penicillin	8	6(75.0)	2(25.0)
Erythromycin	8	6(75.0)	2(25.0)
Ampicillin	8	6(75.0)	2(25.0)
Tetracycline	8	4(50.0)	4(50.0)
Streptomycin	8	5(62.5)	3(37.5)
Trimethoprim	8	5(62.5)	3(37.5)

X^2=45.4 P<0.01

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3.20 Antibiotic sensitivity *Bacillus spp* .

Table(3-20) shows that in each drugs group the frequency of Ciprofloxacin, Erythromycin, Ampicillin, and Trimethoprim were statistically significantly higher sensitive drugs (75%) against *Bacillus spp* than other type of drugs . (P<0.01),while 50%, of ***Bacillus*** spp isolates was resist to Lincomycin, 50% to Vancomycin and 50% to Cephalexin, Penicillin and Streptomycin, while other pattern of resistance were 25-37.5% of various antibiotics. P<0.01.

Table(3-20) Antibiotic susceptibility pattern of *Bacillus spp*

Drugs type	No. of isolated	Sensitive (%)	Resistant (%)
Ciprofloxacin	8	6 *(75)	2(25)
Augmentin	8	4(50)	4(50)
Gentamicin	8	6(75)	2(25)
Vancomycin	8	4(50)	4(50)
Lincomycin	8	4(50)	4(50)
Cephalexin	8	4(50)	4(50)
Penicillin	8	4(50)	4(50)
Erythromycin	8	6(75)	2(25)
Ampicillin	8	6(75)	2(25)
Tetracycline	8	5(62.5)	3(37.5)
Streptomycin	8	4(50)	4(50)
Trimethoprim	8	6(75)	2(25)

T=1.26 P>0.05

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3.21 Type of infection according to number of causative agent.

Table(3-21) show that the frequency of double causative agents (55 isolates ,45.83 %) was statistically significantly higher than single causative agent (38 isolates 31.66%),three causative agents (18 isolates,15%)and more than three(9 isolates, 7.5%).there was no difference between male and female in the frequency of various types of mode of isolates .

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Table (3-21) Modes of isolation of the bacterial pathogens among patients with CSOM

Modes of isolated	Male	Female	Total
	No . of patients (%)		
Single causative agent	18*(15.00)	20 (16.66)	38(31.66)
Double causative agent	30(25.00)	25 (20.83)	55 (45.83)
Three causative agent	12 (10.00)	6 (05.00)	18 (15.00)
More than three	5(04.16)	4 (03.33)	9(07.50)
Total	65(54.16)	55(45.83)	120(100)

P < 0.01

3.22 Bacterial agents and antibiotics.

Table(3-22) shows that in each isolates group the frequency of susceptibility to antibiotic. *Ps.aeruginosa* was statistically significantly higher resistance than other bacterial isolates (10.19%) flowed by *Staph.aureus* (8.73%), *Klebsiella* (7.76%), *Br. catarrhalis*, *Proteus*, *H.influenza* (6.97%), *Strept*.spp(4.85%), *Corynebacterium* (0.9%)and *Bacillus* spp(0.9%). P<0.01

Table (3-22) Relationship between causative agents and antibiotics

Bacterial isolate Type	No. of Isolate	Susceptiblity to drugs								Total	
		(1) drug		(2) drugs		(3) drugs		Morethan (3)drugs		R/S ratio	
		R	S	R	S	R	S	R	S	R	S
Ps.aeruginosa	40	1	2	٢	3	٨	6	١0	8	21(10.19)	19(9.22)
Staph. aureus	35	2	3	2	4	٦	٥	٨	5	18(8.73)	17(8.25)
Klebsiela	30	1	٣	٢	٣	5	4	٨	4	16(7.76)	14(6.79)
Br.catarrhalis	20	٢	1	٣	١	4	2	٥	٢	14(6.97)	6(2.91)
Proteus spp	20	2	1	٣	1	4	2	5	2	14(6.97)	6(2.91)
H.influenza	20	2	1	3	1	4	2	5	2	14(6.97)	6(2.91)
Strept..spp	15	2	1	2	1	3	2	3	1	10(4.85)	5(2.42)
E.coli spp	10	2	1	1	2	1	1	2	0	6(2.91)	4(1.94)
Corynebacterium spp	8	١	2	0	١	١	١	0	٢	2(0.9)	6(2.91)
Bacillus spp	8	١	2	0	2	1	١	0	1	2(0.9)	6(2.91)

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3.23 Smoking and patients with CSOM.

Smoking result in three forms of tobacco smoke :mainstream smoke was directly inhaled by the smoker through a burning cigarette ,Exhaled mainstream smoke the mainstream smoke breathed out by a smoker and Side stream smoke the smoke from the end of a burning cigarette , figure (3-1) shows that in each group of patients with CSOM the frequency of smoking patients the relation of smoking and patients with CSOM,it is found that 30 (25%) patients with CSOM were smoking and 90(75%) were non smoking patients.

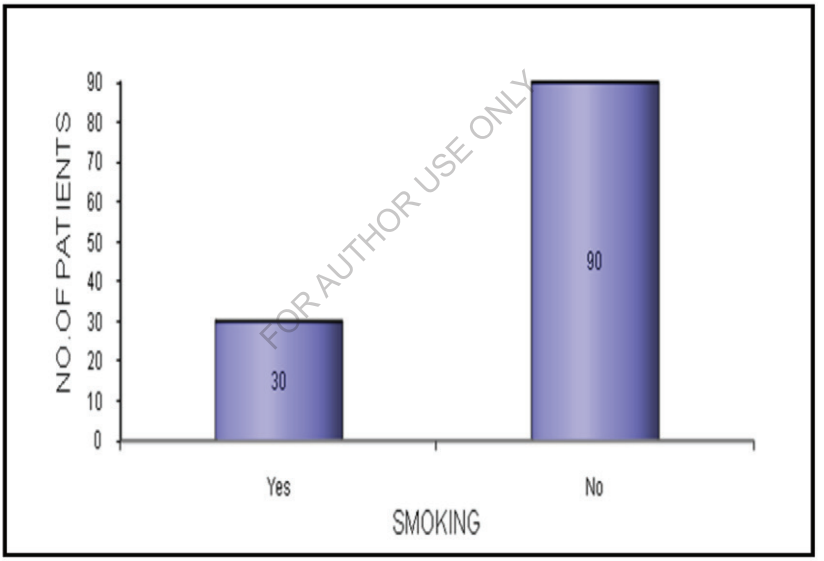

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Figure (3-1) Relation of smoking with patients with CSOM

P<0.01

3.24 Passive smoking and patients with CSOM

Passive smoking is the breathing of environmental tobacco smoke (ETS),.Figure (2-3) shows that in each patients group the frequency of passive smoking 63(52.5%) was highest among patients with CSOM.

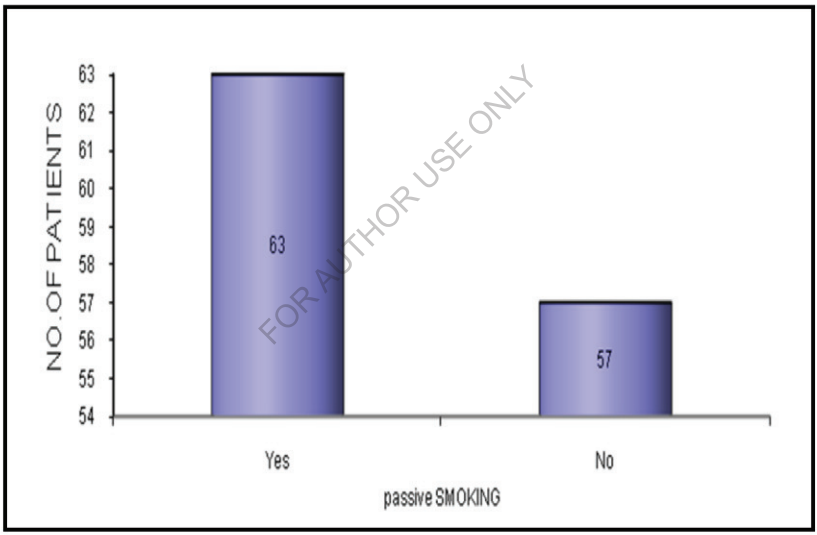

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Figure (3-2) Prevalence of passive smoking among patient with CSOM

X^2=0.36 P>0.05

3.25 Distribution of type of feeding among infant and children with CSOM

Figure (3- 3) shows that the frequency of usual feeding 90 (75%) was higher than breast 20 (16.66%) and bottle feeding 10(8.33%).

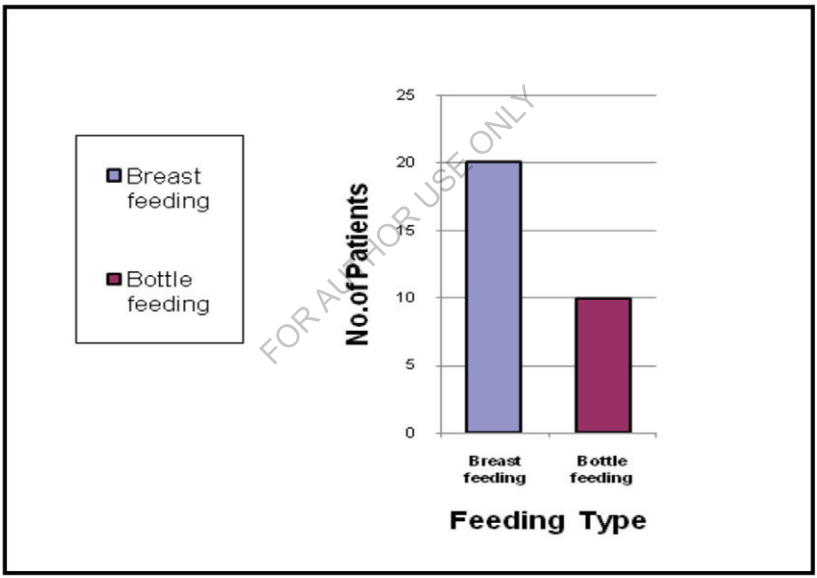

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Figure (3- 3) Type of feeding in patients with CSOM .P<0.01

3.26 Overcrowding patterns among patients with CSOM .

Overcrowding was the presence of three persons or more in a single room.(Ministry of planning ,2004). Figure 4 shows that in most socioeconomic group in Basra the frequency of crowding was 80(66.66%) of patients with CSOM .P<0.01

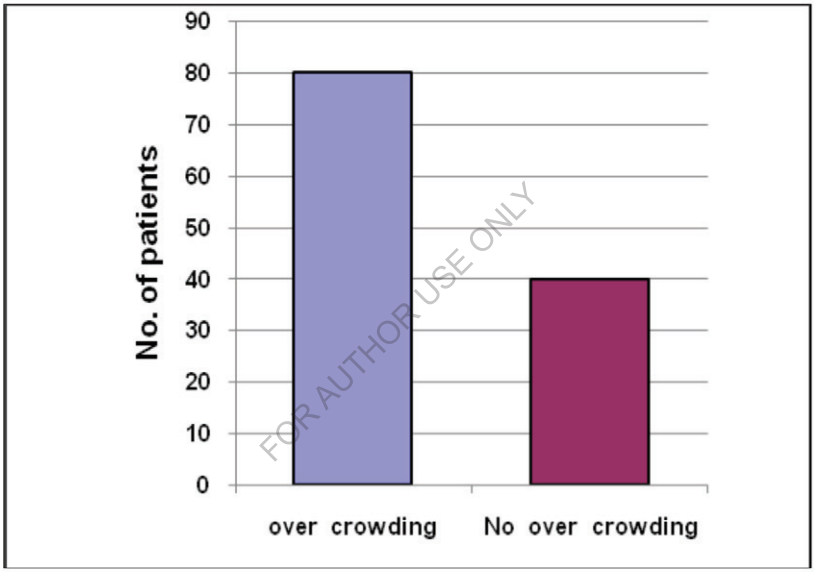

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Figure (3- 4) Over crowding patterns among patients with CSOM.

P<0.01

Chapter four

Discussion

4-1 Association of the patients with CSOM and age.

The majority of patients with CSOM were children (59.2%)from patients while (40.8%) were adults ,our result goes with studies done by (**Verhoeff, 2006)** he stated that the middle ear disease more common between children age group in poor and developed countries .

(Niskar *et al*.,1998 Canter,1997 and Bluestone, 1974) found that children are more prone due to anumber of reasons such as susceptibility to upper respiratory tract infection ,more horizontal nature of Eustachian tube ,and immaturity of immune system.

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4-2Association of the patients with CSOM with sex.

It is found that 65 (54.16 %) males, while 55 (45.83) females. This finding suggests that sex is not affecter in the development of CSOM in the ears. Our results approved with the results of (**Vanderveen *et al.,* 2006**), while not agreed with the study of .(**Arnold, 1996)**.

(**Vanderveen *et al.,* 2006**) stated that no difference between the sexes in the patients with CSOM in age .

(Arnold, 1996), found that the middle ear disease is more common in boys than in girls.

4-3 Clinical Examination Techniques.

Results showed that Rinne test in Lt ear was positive in 22% ,30% right and left ear positive ,38% right ear positive and 30% no applicable(under 5 years old), while in Weber test we show that 25% shifted to right side ,30% centeral,35% shifted to left side and 30% non applicable.

(**Abdel-Hamid *et al*., 2007)** found that the frequency of right ear hearing loss was 86.7% and left ear hearing loss was 89.2% out of the 641 subjects diagnosed with hearing loss.

4.3.1 Tuning fork examination (TFE).

This examination included two type of test(Rinne and Weber) test.In these tests used to identified what kinds of hearing loss is present, a bone conduction hearing test is administered in this test ,a vibrating tuning fork is placed behind the ear, on the mastoid ,when the patient can no longer feel(hear the vibration),the tuning is held in front of the ear ; if the patient cannot able to Hearn sound ,there is conductive hearing loss in that ear. additionally, the tuning fork is placed on the fore head ,the patient asked if the sound localized in the center or shifted to right or lift side .if the sound louder to the effected ear it means conductive hearing loss or if quieter on the affected ear it means sensorineural hearing loss. (**Yueh *et al* ., 2007)**

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4.3.2 Pure Tone Audiometry (PTA).

In our result type of hearing loss proportional with age and severity of infection , conductive hearing loss (CHL)associated with mild and modrate infection in 50 patients,(55.68 %) of patients that have (CHL) suffering from mild to modrate infection ,while senserineural and mixed

proportional with sever and profound , 25 patients ,(20.9 %) of patients have senserineural and 15 patients ,(12.5 %) suffered from mixed hearing loss .

Audiological assessment was done by the same investigator at Basra general hospital , department of audiology and consisted of pure tone audiometry for air and bone conduction by .produce tones at specific frequencies and set volume levels to each ear independently ,the graph have frequency on the x-axis and loudness on the y-axis = (hearing loss) [-10 -120]decibel hearing loss,while x-axis =frequencies between 250 and 8000 Hz (air hearing) and between 250 and 4000 Hz (bone conduction) . **(Probes et al.,2006)**

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4.4 Relationship between type of isolates bacteria and type of hearing loss.

Our result goes with study which done by **(Guo,1994)and(Engel ,1998)**, that show most patients with CSOM infected by more than one pathogenic bacteria leading to hearing loss , about 40 patients ,(33.4%) of patients with CSOM suffered from bilateral hearing loss,while (80 patients ,66.6)of patients with CSOM have unilateral hearing .

(Guo *et al*., 1994) studied found the effect of endotoxic damage to the stria vascularis and concluded that lipopolysaccharide induced strial ototoxicity produced ion imbalance ,causing changes in endolymph composition and energy failure in the middle and inner ears organ explaining the pathogenesis of hearing loss in CSOM.

(**Engel *et al*.,1998)** studied the passage of streptolysin-O and albumin through the round window membrane and proposed that the passage of macromolecule ,such as protease,from apurulent middle ear

effusion may be facilitated by pore forming toxins,resulting in middle and inner ear organs damage and hearing loss.

The infection causes a buildup of fluid in the middle ear .The pressure exerted by this fluid can build up to the point where the ear drum perforated .The fluid buildup and ear drum perforation inhibit the transmission or conduction of sound through the ear .**(Howard , 2007)**

4.5 Types of Hearing Loss.

1. Conductive hearing loss(CHL)

In our study 50 patients with CSOM ,(55.68 %) have conductive hearing loss in mild and modrate infection.The diagnosis done by pure tone audiogram ,

(Howard, 2007) found that (CHL) occur due to impairment of the transmission of sound impulses before they enter the inner ear

(Hogan and Moor, 2003) found that childhood middle ear disease contributes to a secondary condition problem with the processing of auditory information .The persistent partial sensory deprivation that result from the conductive hearing loss associated with middle ear disease .

Many studies stated that CSOM cause middle ear disease lead to conductive hearing loss. **(Morris *et al*.,2007& Bowd, 2005**).

2. Senserineural hearing loss.

In our result, 25 patients which comprises ,(20.9%) CSOM suffering from senserineural hearing loss .Most of them were adult patients.

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Sensorineural hearing loss (SNHL)is caused by dysfunction in the cochlea or the cochlear nerve. Sometimes a cochlear loss is referred to as a sensorineural .

(Blakley, 1998) , **(Kaplan *et al*.,1996)** studied 127 diseased ears in children and reported that CSOM has little effect on cochlear function .

(Kholmatov , 2001) ,showed that when the duration of disease was compared with the incidence of SNHL, a progressively increase incidence of SNHL was found as the duration of disease increased .

3. Mixed hearing loss.

In our result 15 patients which comprises ,(12.5%) of patients with CSOM suffered from mixed hearing loss.

Mixed hearing loss occurs when both sensorineural and conductive hearing loss are evident. (**Kamaijit *et al*., 2003)**

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4-6 Clinical Feature of Patients with CSOM

In the Present study it is found that 120 patients were presented with otorrhoea(100%), followed by otalgia . (40%) ,ear iteching (30%) , , tinnitus 60% and vertigo 30% and fever 50(41%). Our result agreed with study done by (**Hwang *et al*, 2002)** that otorrhoea was the most common symptom in patients with CSOM ,while disagreed by result (**Bardanis, 2003)** who stated that otorrhoea commnly found in otitis externa.

Otorrhea is a common symptom and sign of patients with CSOM**,** when discharge contain mucus ,it must have arisen from glands within the middle ear cleft, passing into the external ear through a tympanic membrane perforation. **(Roland., 2000)**

(Bardanis *et al*.,2003) found that otitis externa form the common cause of purulent otorrhoea .

There is no consensus on the duration of otorrhea to be termed chronic(**WHO ,2000**)

4.7 Bacteriology.

Chronic suppurative otitis media develops from a chronic bacterial infection . However the bacteria that caused the initial episode of acute otitis media with perforation are usually not those isolated from the chronic discharge when there is a chronic infection in the middle ear and mastoid infection usually polymicrobial and secondary in nature, derived from the external auditory canal or commensal flora of nasopharynx . **(Bluestone and KLien, 2001)**

(Karma *et al*., 1978) have used Gram stain not only to confirm the presence of cultured bacteria but to detected and identify them as well,Gram stain smear were obtain from 108 ear swab ;in 98(91%) of them bacteria were found Seven of the 108 ear swab (6%) were devoid of bacteria both in culture and in the Gram stain.

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(Papastavros *et al*., 1986) indicated that this practice considerable error,because non viable bacteria can be as equally incriminated as the main pathogens present . further more,if the patients is under antimicrobial treatment .

4.7.1 **Normal flora.**

In our study ,we found different type of bacterial normal flora in the external canal were founded ,*Staphylococcus epidermidis* is the most common (20 isolates, 40%), followed by *Corynebacterium* species about(15 isolates, 30%), while other type have various percentages of isolation. Our result is agreed with **(Pelton *et al*.,1980** and **Brook *et al*.1996)** while it is against the result is of **(Saunders *et al*., 2009)**

(Pelton *et al*.,1980& Brook *et al*., 1981),showed that the predominant microflora *were Staph epidermidis,diphtheroid ,and Staph aureus.*

4.7.2 **Pathogenic Bacteria:**

1. ***Pseudomonas aeruginosa* .**

In our study the number of *Ps.aeruginosa* isolates was (40 isolates, 19.41%). our result agreement with studies done by **(Aslam *et al*., 2004)** and **(Verhoeff, 2006)** that *Pseudomonas* most common agents in patients with CSOM,and not approved with **(Saunders *et al*.,2009)** found *Staph.epidemedis* most common causative agents.

(Aslam *et al*., 2004) showed that *Ps.aeruginosa* is most common isolates from infected mastoid cavity and chronic otitis media and most common aerobic bacteria isolated from chronic suppurative otitis media .

(Verhoeff *et al.*, 2006), stated that *Pseudomonas aeruginosa* was the most prevalent bacteriological agent in chronic otitis media, flowed by *Staphylococcus aureuse* .

(Saunders *et al*., 2009) stated that *Staphyllococcus epidermidis* species was the most prevalence bacteriological agent in chronic otitis media.

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2. ***Staphylococcus*** species .

In this study we found that *Staph.aureus* (35 isolates, 16.99%) followed *Ps.aeruginosa* in their incidence,our result agree with study done by **(Aslam et al.,2004)** ,while against the study done by **(Saunders *et al*.,2009)**

(Saunders *et al*., 2009) found that *Staph .epidemidis* (6%) was the most common bacteria isolated from patients with suppurative otitis

media ,followed by methicillin resistant *Staphylococcus aureus* (3%) and *Pseudomonas aeruginosa* (1%).

3. ***Klebsiella*** spp.

In our study, found that *Klebsiella* species isolated from patients suffering from chronic suppurative otitis media was (30 isolates 14.56%) . our patients infected by Enterobacteriaecea such as *Klebsiella* species , most of them among children and infants group, because the Eustachian tube in children shorter and wider than adult .

(Bluestone *et al*.,1974) showed that young children have shorter ,straighter and more compliant Eustachian tube than adult .this permits a reflex from nasopharynx to the middle ear with the consequence of bacterial contamination.

(Brook and Yocum, 1989) found that Klebsiella species (6.2%) isolate from patients with CSOM.while **(Ostfeld and Rubinstein ,1980)** stated (20%) of Klebsiella species presented in young infant with acute otitis media ,but rarely appear in the middle ear effusion of older children with otitis media.

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4. ***Branhamella catarrhalis.***

In our work, we found that Branhamella catarrhalis was (20 isolates, 9.7%).

(Faden, 1994) found that , *Moraxella or Branhamella catarrhalis* were common organisms , *diplococcus* and considered part of the normal flora of human upper respiratory tract ,classified as causative agents to middle ear infection ,it had constituted approximately 10% of all isolates.

(Hanan,2000) showed that *M. catarrhalis* secreted lactamases (cephalosporinases) may protect these bacteria and other type from

antimicrobial agents to which the second target pathogen ordinarily might be suscebtible,can be differentiated from the other *Neisseriae* spp by its lack of carbohydrate fermentation and by its of DNase production**.**

5. ***Proteus*** spp.

In our study we found *proteus* species isolated (20 isolates 9.7%).

Iseh and Adegbite ,(2004)found that proteus species (12.8) isolated from 41 patients with acute suppurative otitis media.

(Vaishnav and changani, 1981) , found that *Proteus* species with highest incidence (44%) of isolates from 100 cases with CSOM.

6. ***Haemophilus and Streptococcus*** species .

In our result,we found that *Haemophilus influenza*e was (20 isolates 9.7%),while *Streptococcus pneumonia* (15 isolates 7.28%).

(Yamanaka *et al*.,2008) showed that *Hemophilus influenzae* and *Streptococcus pneumoniae* are the most prevalent organisms responsible for acute otitis media. However, most studies from different parts of Africa suggest:various bacterial pathogens as acaustive agents . Hence, *Staphyloccocus aureus* and *Streptococcus pyogenes* appear to be most dominant causative organisms among Africans**. (Hussain *et al* ., 1991).**

(Bluestone and Klein, 2001) found that *Streptococcus pneumoniae* and *H.influenzae* are most common bacteria species causing middle ear infection in acute otitis media .

Some European studies found *Haemophilus influenzae* to be the most common organsm followed by *streptococcus pneumoniae* and *Branhamella catarrhalis*.**(Gray, and Canter, 1997).**

7. ***E. coli*.**

In our result, we found that the frequency of *E.coli* was(10 isolates 4.85%) isolated from patients with chronic suppurative otitis media . *E.*

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coli belong to enterobacteriaceae, pathogenic causative agent in acute suppurative otitis media in children and infant. **(Bluestone, 1990)**

(Iseh, 2004) found *E.coli* in patient s with acute suppurative otitis media second causative agent , Ear swab was cultured in only 41 patients (36%). *Staphylococcus aureus* (46.2%) was the commonest bacteria cultured followed by *Escherichia coli* (23.1%).

8. *Corynebacterium* and *Bacillus* species.

In our result ,we found that *Corynebacterium* and *Bacillus* species were (8 isolates 3.88%). for each presnts in externa canal and middle ear cleft as apportuenstic normal flora in individual without otological problems.

(Brook and Schwartz,1981) showed that *Corynebacterium* species was predominant in external canal and middle ear cleft,while **(Kurono *et al*.,1988)** isolated 12 different bacterial species ,in which *Bacillus subtilis* from middle ear cleft and external canal.

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4.8 Antibiotic susceptibility patterns

The organisms that cause otitis media are become more resistant to antibiotic .for example ,according to recent studies,between (30 -60)%of *Streptococcus pneumoniae* bacteria are now partially resistant to the antibiotic such as penicillin and amoxicillin . Antibiotic lose their effectiveness in children who have been continuous treated with them in a short period of time .Ciprofloxacin and Augmenten (amoxicillin-clavulante) is more abundant bactericidal agent for many Gram positive and Gram negative bacteria in AOM,CSOM **.(Gehaanno, 1997)** and (**Winter, 1994)** (90-95)% of cases of Acute otitis media (AOM) with otorrhoea occure in children aged (1-12)years,and typically (2-6)

episodes of AOM. Ciprofloxacin is an effective and safe therapy for AOM and chronic suppurative otitis media (CSOM). **(Force *et al.,*1995)**

The efficacy and safety of a combination of topical dexamethasone 0.1% and ciprofloxacin 0.3% in children with (AOM),otorrhoea resolved more rapidly with combination preparation than with ciprofloxacin alon and produce significantly greater clinical responses early after completion of seven days course of treatment . (**Zipfel, 1999)**

In our study we noted Ciprofloxacin ,(Amoxicillin+clavulanic acid)Augmentin ,Gentamicin were abroad spectrum antibiotic (70-80)% sensitive to different species of Gram negative and Gram positive bacteria in CSOM.

Topical treatment is better than systemic therapy,this probably because a higher local concentration of antibiotic is achieved. **(Macfadyen *et al.*,2006)** the antibiotic should have activity against Gram negative bacteria ,especially *Pseudomonas*, and gram positive bacteria ,especially *Staphylococcus aureus*.The amino glycosides and the fluoroquinolones both meet these criteria but the former may be ototoxic, failures of the antibiotic are usually due to failure to penetration the debris rather than bacterial resistance. **(Marais *et al.*,1998)**

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Aminoglycosides are contraindicated there is evidence that they may cause hearing loss. **(Bance *et al.*,2005)**.

4.9 Breast and Bottle feeding.

our study showed that (25%) of chronic suppurative otitis media among infant used breast feeding, while infant used bottle feeding (16.7%),there was no significant different between them.

In general breast feeding was protected infants by natural immunity receiving from mother during lactation period from various disease ,one of them otitis media.

Children are more prone to develop CSOM than other s age groups but researchers have identified one possible cause which is bottle feeding, because bottle feeding usually associated with excessive sucking .which creat negative pressure in oral cavity and pharynx, lead to decrease the pressure in the middle ear ,this scenario can be avoided by decrease stiffiness of the bottle **(Hauser *et al*.,1989)**

Sucking on pacifiers ,toys, thumbs and similar objects can cause the negative pressure inside nasopharynx and these lead to negative pressure in the middle ear cause serious ear disease including (adhesive otitis media, and cholesteatoma). **(Sakakihara *et al*.,1993)**, While breast feeding reduce the risk vacuum and air bubble formation, produce positive pressure can be equilibrium the negative pressure in order to removed ear infection ,parents should try. (**Brown, 2000)**

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Pacifier use increarsed incidence of otitis media ,while breast feeding for a last three months was protective ;this effect may be associated with position maintained during breastfeeding ,suckling movement ,and protective factors in breast milk.(**Arrieta , 2004)** .

4.10 Geographical distribution of CSOM.

In the present study ,we found that significant variation in the distribution of patients with chronic suppurative otitis media in urban &rural area 83%,16% respectively these attributed to overcrowding ,poor sanitation ,low medical education ,(pollution of air,food and water) in urban area , our result goes with the study done by (**Bowd, 2005)** and **(Couzos *et al*.,2001)**

In communities where children grow up in overcrowded housing ,have poor nutrition and limited access to health care, middle ear disease is more prevalent and more sever ,most children from these communities often experience mild to moderate fluctuating conductive hearing loss during their school years.(**Couzos *et al*.,2001)**

Arnold, (1996) found that otitis media more common in whites, in lower socioeconomic groups, in native americans (particularly in Alaska), and children born with a cleft palate and other structural problems of the face or skull

4.11 Smoking and passive Smoking

In our study, we found 30% of patients with CSOM were smokers, while 70% were non smoker. on the other hand passive smoking about 63% of patients with CSOM.

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Environmental tobacco smoke(ETS) refers to a combantion of exhaled mainstream smoke and sidestream smoke ,passive smoking is the breathing of ETS. Babies lungs and bodies are smaller than adult and so the poisons in smoke are more harmful, over 1.7 million Australian children are at risk of developing a range of illnesses due to exposure to ETS. There are around 4000 chemical compounds in cigarette smoke ,researcher show that many of these chemicals compound cause upper respiratory tract infection ,cancer , .(**Smoke freezone and Arrieta, 2004)**

Exposure to cigarette smoke increase incidence of otitis media and air pollution ,especially if parents smoke Passive smoking caused by parents increased morbidity in children ,with arisk ratio of 1.19, AOM episoid reached to 340 000-2 million and 5200 to 165 000 tympanostomies were attributable to enveromental smoke.

(**American, 2004)**

Conclusions

1-Infan and children were affected more than adult age group.There was no significantly difference between male and female.In general ,the male :female ratio of patients with CSOM was (1.2:1).

2-Conductive hearing loss was most commonest type of hearing loose between patients ,frequently found in age group (6-25)years ,while senserineural hearing loss frequently found in age group (30-60)years .

3- Bacteria play a role in the pathogenesis of chronic suppurative otitis media ,which is evident by the clinical poreferated tympanic membrane and hearing loss.

4-*Psedomonas aeruginosa* and *staphylococcus aureus* species are the most frequent isolates bacteria in CSOM.

5-Ciprofloxacin and Amoxicillin +cluvulanic acid (Augmentin) were the effective antibiotics against bacteria caused CSOM

6-*Staphylococcus epidermidis* and *Corynebacterium* species were the most common normal flora in the external canal

-Breast feeding may play role in pathogensis of CSOM.

7- *Psedomonas aeruginosa* and *staphylococcus aureus* species are the most common resistant antibiotics (more than three drugs).

8-Mixed infection significantly higher than single.

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Recommendations

1- Determine the role of chronic suppurative otitis media as a risk factor for dangerous complications (hearing loss).

2-Study of recurrent bacterial infection .

3-A study the relationship between various bacterial types present in CSOM discharge ,and determine the type of hearing loss.

4- Determine severity of infection by used clinical investigation ,clinical feature and laboratory identification.

5-Study of virulence factors of bacterial pathogens associated with CSOM.

6-Study the relationships between CSOM and other infectious diseases.

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