

Classification and management challenges of otitis media in a resource-poor country

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Abstract

The clinical features and sequelae of otitis media (OM) vary depending on the duration, severity and progression of the disease. As a result, opinions on the modes of classification and management protocols have differed over the years. The need to critically appraise these opinions according to the peculiarities of each region is imperative. This work was aimed at reviewing the world literature on the subject and also highlights the limitations in management in our region. A wide literature search was conducted using the following search engines: PubMed, AJOL and University of Toronto Library. Also incorporated were essential materials obtained from the authors' clinical practices. The search engines returned 22,903 related articles on OM. Further filtration yielded 88 articles on "classification and management" and these were obtained in full and thoroughly read. Extracted materials for review spanned between 1980 and 2008. OM is prevalent the world over with potentially severe complications if inadequately managed, especially in the developing countries. It is of note that in the developing countries, poverty, ignorance, dearth of specialists and limited access to medical care amongst others conspire to worsen the course and complications of OM.

Key words: Classifications, complications, management, otitis media

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Introduction

Otitis media (OM) is a global middle ear disease with health-economic burden, especially in Africa and other developing nations where the disease prevalence could be as high as 11% with severe economic implications.^[1-4] It presents with varied clinical features depending on the duration, severity and progression of the disease. As a result of the complex contiguous relationship of the middle ear and essential intracranial structures, severe complications (intracranial and extracranial) can result from untreated or poorly treated OM. A sound knowledge of the anatomy and physiology of the ear is necessary for the management of this disease. Therefore, it is imperative that a brief description of these features be given prior to further discussions on this disease entity (further readings on from standard texts, e.g. Scott brown series).^[5]

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Classification of Otitis Media

OM is a broad subject which could be classified according to: (1) duration – acute OM and chronic OM; (2) nature of fluid/discharge – suppurative and non-suppurative OM; (3) OM with effusion and aero-OM; (4) causative organism – bacterial OM (common) and specific OM, for example, tubercular and syphilitic OM (less common).

The dynamism in the field of otology has brought about a current classification of choice by Browning *et al.*,^[6] which de-emphasizes the words "suppurative" and "non-suppurative" OM, since it is a progression of same pathologic process. Furthermore, one could lead to the other depending on the prevailing factors. Thus, OM could be further

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described as “active”, “inactive”, “healed”, but the standard classification remains acute OM, chronic OM, OM with effusion and specific OM.^[6]

A brief description of the uncommon types of OM shall be given, whereas the detailed discussion shall focus on the three commonly encountered types of OM, namely, acute otitis media (AOM), chronic otitis media (COM) and otitis media with effusion (OME). The details of the surgical procedures, which can be found in standard ENT and Otolaryngology textbooks, are not included in the description.

Aero-OM is caused by disequilibrium in pressure equalization between the atmosphere and the middle ear cavity via the Eustachian tube. Ascending /fast descent from height or deep sea diving are common sources. Experimental models have shown that the resultant inflammation can extend beyond the middle ear cavity and the Eustachian tube to the nasopharynx and sometimes nasal cavities and paranasal sinuses.^[7]

Acute Otitis Media

This is an acute infection/inflammation of the middle ear mucosa which also involves the mastoid air cells most often. AOM is predominantly a childhood infection without sex preponderance^[8-10] and 75% of the few adults who have this condition are young adults below 44 years of age.^[11]

Etiology

Global reports show that *Hemophilus influenzae* and *Streptococcus pneumoniae* are the most prevalent organisms responsible for AOM.^[12-14] However, most studies from different parts of Africa suggest a different bacteriology spectrum. Hence, *Staphylococcus aureus* and *Streptococcus pyogenes* appear to be the most dominant causative organisms among Africans, usually sensitive to penicillin-based antibiotics, Cephalosporins, and Quinolones.^[15-17] Ruohola *et al.* demonstrated that most cases of AOM consist of mixed infection of bacteria and virus; with about 60% of the viruses belonging to the picornavirus spp.^[18]

Clinical features

The clinical mode of presentation is usually systemic (generalized) and local (ear) symptoms and signs. The typical picture is a child with high-grade fever (40–41°C), refusal of feeds, incessant cries and irritability.^[19,20] There is associated ear pain (otalgia) and sometimes noise in the ear (tinnitus) with difficulty in hearing (conductive hearing loss). Ear discharge is seen in well above 90% of cases^[17] in some parts of the developing world, whereas it is only about 10%^[11] among the developed nations. Delay of presentation to the clinician most probably accounts for this disparity. Other clinical features which are detected via otoscopy [Figure 1] include hyperemic tympanic membrane



Figure 1: An actively discharging ear (hyperemic TM highlighted in yellow) with a central perforation copiously discharging pus. (A video-otoscopy picture of a child taken at ENT clinic of UCH Ibadan, Nigeria, in 2006 by the authors)

(generalized or localized) and sometimes bulging (if there are exudates within the middle ear). Furthermore, the discharge may be mucopurulent or purulent and appears to be pulsating in synchrony with the patient's pulse during active discharge from the point of tympanic membrane perforation on otoscopy.

Sequelae

The inflammation may resolve following appropriate measures such as administration of suitable antibiotics or evacuation of the exudates within the middle ear through spontaneous or guided rupture of the tympanic membrane.^[9] On the other hand, the disease process may progress to complications in the presence of poorly treated or untreated virulent organisms.^[21] Spread of the disease beyond the confines of the middle ear can result in extracranial or intracranial complications. Acute mastoiditis (inflammation of the mastoid air cells)/coalescent mastoiditis,^[22] subperiosteal abscess,^[23] facial nerve paralysis,^[24] labyrinthitis and petrositis^[25,26] are examples of such extracranial complications, whereas the intracranial ones include extradural and subdural abscesses,^[27] meningitis,^[28] otitic brain abscesses,^[29] otitic hydrocephalus and lateral sinus thrombosis.^[30,31]

Management

Ear swabs for discharging ears are taken for microscopy, culture and sensitivity test prior to commencement of broad-spectrum oral and or topical antibiotics. This is usually guided by the knowledge and behavior of predominant causative agents within a given environment. Although a school of thought argues against the use of antibiotic therapy for AOM based on the notion that most are associated with viral infections,^[32-34] however, the experience within the developing world has suggested that most of the cases are either mixed (bacterial +

viral) or bacterial infections.^[18,35] Some native cultural practices and the humid environment of the tropics encourage secondary bacterial infections.^[36] A daily aural toileting of the ear is necessary for the discharging ear. Myringotomy to evacuate the exudates in bulging tympanic membrane (TM) is encouraged prior to management with antibiotics. Adequate analgesia to reduce otalgia is valuable in the management of AOM. Imaging may be necessary, especially in suspected cases of complications. In addition, relevant ancillary investigations like complete blood count, urea and electrolytes should be carried out for optimization of the patients who may require surgery under anesthesia. Complications are managed accordingly. Appropriate exploratory and decompression surgeries such as mastoidectomies, facial nerve decompressions, craniotomies for intracranial abscesses evacuations, etc. under adequate antibiotic cover are usually indicated. The details of the procedures could be referred to in standard otology textbooks like the Scott Brown series and Ballenger.

Otitis Media with Effusion

This is a pathological condition characterized by the accumulation of fluid (non-purulent) within the middle ear cleft with an intact tympanic membrane. Most frequently, the fluid is mucus and thick, but sometimes serous and thin in consistency. OME has also been referred to as “glue ear”,^[37] “mucoid otitis media”,^[38,39] “secretory otitis media”^[40] and “serous otitis media”.^[41]

The pathogenesis of this condition is thought to be double pronged: First, from Eustachian tube disorder, which results in poor aeration of the middle ear and poor drainage of secreted fluids.^[9,42] Secondly, it could result from hyperactivity of the middle ear gland which leads to excessive accumulation of mucus secretions. Histological evidence has shown an increase in the number of the mucus or serous-secreting cells in such situations.^[8]

Just like AOM, OME is predominantly a childhood problem. About 85% of cases of OME occur in children, making it a rare disorder in adults.^[43] In contrast, however, OME has been reported to show some racial bias. It is commoner among the Caucasians, especially Canadian, Australian Aboriginals and native American children compared to Africans.^[44,45]

Etiology

There is a variation in the etiology in children and adults. Eustachian tube dysfunctions from adenoid enlargements, upper respiratory tract infections, congenital defects (e.g. cleft lips and palates) are common etiological factors in children,^[46-48] whereas allergy, barotraumas, nasopharyngeal tumors and rhinosinusitis are associated

with OME in adults.^[49-51] Other factors that could be predisposing to OME include prolonged nasotracheal intubation, head and neck surgeries like maxillectomy, radiotherapy of the head and neck and immunodeficiency disorders like multiple myeloma, cystic fibrosis and HIV/AIDS.

Clinical features

OME usually presents with conductive hearing loss. The magnitude of hearing loss is normally mild to moderate (≤ 40 dB). It is usually most prevalent in children below 5 years of age.^[45] Sometimes the hearing loss may be detected incidentally during routine audiometric evaluations (e.g. preschool age screening tests). Pressure effects of the effusion could give rise to otalgia (ear pains). Furthermore, very early onset of OME can give rise to speech difficulties since the child requires proper hearing for speech acquisition. The otoscopic findings include intact but dull tympanic membrane lacking in the light reflex, with obvious restrictions in mobility. The appearance may range from brown to yellow. The tympanic membrane may show fluid level and/or air bubbles if the effusion is serous and the TM translucent. It might appear bulgy. On the contrary, the TM may exhibit certain degrees of retractions when there is a scanty viscous fluid within the middle ear.

Sequelae

Acute OME resulting from recurrent upper respiratory infections and allergy usually resolves spontaneously after few days of onset without complications. However, if OME fails to resolve within 6 weeks, it becomes chronic with possible serious consequences.^[52] As a result, atrophic tympanic membrane can result from thinning and degeneration of the fibrous layer of the TM, giving rise to atelectasis of the middle ear and retraction of the tympanic membrane. Involvement of the pars tensa of the TM would result in retraction pocket formation near the attic and this predisposes to cholesteatoma formation with its subsequent dangers. Other possible complications of chronic OME include ossicular bone necrosis, especially the long process of incus, tympanosclerosis (calcium laden deposits on the tympanic membrane and the ossicle with its joints) leading to fixation in movement. Stasis in the secretions in the middle ear and mastoid can also result in the formation of cholesterol granuloma.^[53]

Management

After clinical assessment of the patient, specific audiometric investigations and imaging are necessary to confirm diagnosis. Pure Tone Audiogram (subjective test) establishes conductive hearing loss of usually ≤ 40 dB, whereas Impedance Audiometry (tympanometry), which is an objective test also useful in children, establishes a flat type B tympanometry curve for confirmation. X-ray paranasal sinuses (occipitomeatal, occipitofrontal and lateral views)

establish possible focus of infections from paranasal sinusitis. Mastoid views (Towne's and Owen's) may be necessary to rule out mastoiditis and computerized tomographic scan, where available, offers better resolution and is very important in suspected cases of OME from nasopharyngeal tumors, although experience has shown that most patients in the developing world cannot afford this, and therefore, X-rays are still relevant in this part of the world.

The principle of treatment is basically identification of the cause of the OME, evacuation of the fluid, treatment of causes and prevention of recurrence. These could be achieved via medical, surgical management or a combination of the two.

Medical treatment

The use of topical vasoconstrictors/nasal decongestants to reduce the edema 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. The use of anti-allergic drugs for cases of identified allergies/atopy and relevant physical exercises [e.g. jaw exercises (through gum chewing)] and valsalva maneuvers to improve aeration to the middle ear are thought to be useful. However noteworthy is that current evidence-based researches are questioning the usefulness of vasoconstrictors and nasal decongestants in the treatment of OME.^[54,55]

Surgical treatment

When conservative management is not sufficient for the resolution of the problem, surgery is indicated.^[56] First, the causative factors amenable to surgery might require specific surgical repairs or maneuvers to resolve the OME and prevent future occurrences. Examples are cleft lip/palate repairs, antrostomy and wash outs (where appropriate in rhinosinusitis), adenoidectomy/tonsillectomies.^[57-60] Secondly, in situations where the effusion is viscid or massive, myringotomy is indicated for such evacuations. Vertical incision is usually placed either on the antero-inferior or on the antero-superior segment of the tympanic membrane for this procedure, while micro-suctioning is applied for the evacuation of the fluid and tympanostomy tube insertion for aeration. Evidence has shown a better outcome and lesser

complications in OME managed with tympanostomy tube as compared to those managed conservatively.^[61]

Chronic Otitis Media

Essentially, Chronic otitis media (COM) is a "permanent" abnormality on the tympanic membrane following a long-standing middle ear infection emanating from previous AOM, OME or negative pressure to the middle ear.^[62] Several time divides between acute and chronic OME have been suggested, ranging from 2 weeks to 3 months.^[6,9,63] It may be actively discharging or not, and therefore in most texts, COM is referred to as "chronic suppurative otitis media" or "chronic non-suppurative otitis media". However, for reasons earlier cited, current suggestions are that "active", "inactive" or "healed" COM depending on the clinical disease condition are the preferred terms. Pathologically, this disease entity is further subdivided into "mucosal" and "squamous" COM^[62] based on the histologic features of the middle ear mucosa and the edges of the perforated TM. Furthermore, COM was previously classified as tubotympanic – "safe" and atticofacial – "unsafe" based on the anatomical location of the TM perforation.^[9] Whereas marginally located TM perforations, especially at the pars flaccida, were initially regarded as "unsafe" and central perforations as "safe" based on their propensity toward formation of cholesteatoma, evidence-based information has shown that cholesteatoma could form in either condition, thereby nullifying the previous classification.^[64] Table 1 summarizes the current clinical and histological classifications of COM according to Browning.^[65]

Note: The healed COM is the desired end result following the treatment of active COM or the end result of a natural healing from AOM or COM.

COM is commoner in the developing countries, where prevalence is as high as 6–11.1% across the various age groups,^[1,66-68] than in the developed countries. Several researchers have shown that people of lower socioeconomic class are affected more than the upper divide.^[2,69] It affects all age groups, showing preponderance in adults, but no sex discrimination. Seasonal variations with the disease

Table 1: Clinico-pathological classification of chronic otitis media

COM classification	Synonyms	Otoscopic findings
Active (squamous)	Cholesteatoma	Retraction of pars tensa/flaccid, retained squamous epithelium, debris and pus
Active (mucosal)	---	Permanent defect of TM on pars tensa, inflamed middle ear mucosa, mucus discharge
Inactive (squamous)	Retraction	Retracted pars tensa/flaccid usually at postero-superior segment, with potential of becoming active with retained debris
Inactive (mucosal)	Perforation	Permanent perforation on pars tensa, inactive middle ear mucosa
Healed	Healed perforation, tympanosclerosis	Thinning and/or local or generalized opacification of the pars tensa without perforation or retraction

Adapted from Browning^[65], chronic otitis media

progression have been recorded in both the tropical and temperate regions. In the West African sub-region, it has been noted that some inactive COM becomes active during the rainy seasons.^[36] Similar observations were also made in America and Europe during the winter seasons.^[70]

Etiology

COM could be a sequela to AOM and OME following poorly^[71] managed cases. Environmental factor and state of hygiene has been identified as a predisposing factor toward developing COM as seen in AOM. It has also been found that the prevalence of COM tends to be significantly more in patients suffering from gastroesophageal diseases (GERD). In addition, more patients with craniofacial abnormalities (e.g. cleft lips/palates, velopalatine muscle incompetence) tend to be more predisposed.^[58] Genetic/racial link is suggested to explain the difference in preponderance of this disease among certain groups of people with fairly similar living conditions.^[72,73] Furthermore, strong, autoimmune and immunodeficiency states are also suggested to be predisposing factors toward the development of COM.

The bacteriological causative agents most commonly associated with COM include *Pseudomonas aeruginosa*, *proteus* spp., *Escherichia coli* and *St. aureus*.^[15,16] Common anaerobes include *Peptostreptococcus* spp., *Prevotella melaninogenica* and *Bacteroides fragilis*.^[74]

Clinical features

The clinical features of typical uncomplicated COM are not as common as in the case of AOM. The patient is usually afebrile, with a history of prolonged or recurrent ear discharge in active COM with varying characteristic features of the otorrhea. Copious mucopurulent otorrhea is usually a feature of active mucosal COM, whereas scanty,

foul-smelling, and sometimes, sanguineous varieties are seen in active squamous COM (cholesteatoma).^[75] This may be unilateral or bilateral. Otagia is not a common feature except in complicated conditions. Hearing loss, of conductive type and sometimes mixed (conductive and sensorineural), is also a common feature of COM.^[76,77] Otoloscopic examinations [Figure 2a and b] usually show TM perforations of varying features – locations, sizes, shapes, dryness or wetness.^[36] Aural polyp, a coalition of exuberant granulation tissues formed following chronic irritation of the middle ear mucosa, could be present. It sometimes occludes the entire perforation on the TM and prolapses into the external auditory canal. Again, retraction pockets and neo-membranes (dimeric tympanic membrane lacking in the middle fibrous layer) can be prominent features of the healed COM. Clinical features of complications may also be seen as enumerated below.

Sequelae

Active COM can become inactive or healed COM and vice versa depending on the prevailing circumstances and level of interventions. However, it could progress to severe and sometimes life-threatening complications which are broadly classified as “extracranial” and “intracranial”, respectively.^[78]

The extracranial complications are further subdivided into extratemporal and intratemporal. The extratemporal complications include Luc’s (temporalis region), Citelli (sub-periosteal), Bezold’s (sternocleidomastoid) abscesses. These are abscesses formed through the tracking of pus via the middle ear through natural anatomical channels of the head and neck to accumulate at the above designated sites. The intratemporal components of the extracranial complications include: mastoiditis, petrositis, facial paralysis and labyrinthitis. The intracranial complications

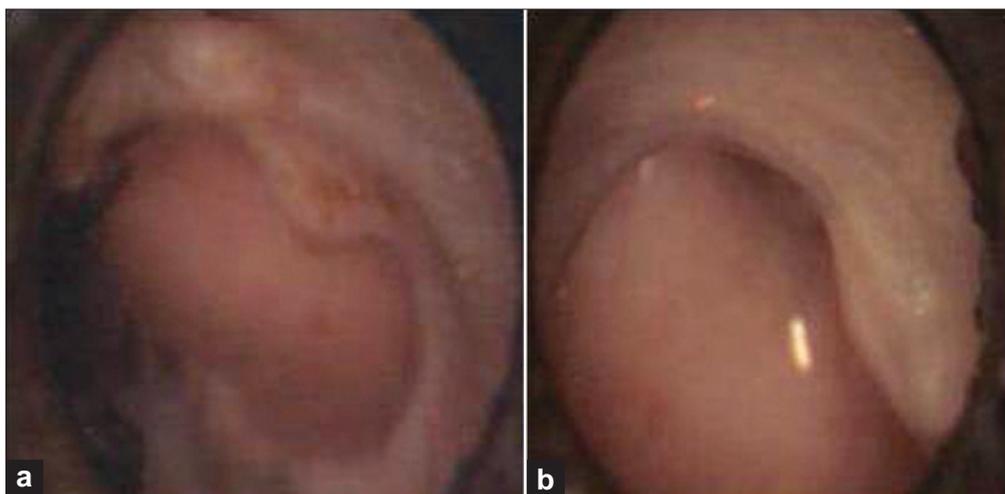


Figure 2: (a) Inactive mucosal COM with kidney shaped dry perforation. (b) Active mucosal COM with a large perforation exposing the middle ear mucosa and its mucopurulent discharge. (Video-otoscopic pictures taken in the ISTH ENT Clinic, Irrua, Nigeria, in 2008 by author 1)

are similar to those of the AOM as earlier enumerated, with otitic hydrocephalus as the least common intracranial complication.^[79]

Management

Akin to the diagnosis of AOM, appropriate ear swab should be taken for microscopy, culture and sensitivity tests. Micropipette technique of deeply seated otorrhea within the middle ear cavity *en route* the perforation on the TM in active COM is the method of choice to avoid contaminants. This is a useful guide for the identification of causative agent and appropriate choice of antibiotics. Appropriate views of radiological imaging (X-rays, CT scan)^[80,81] as in AOM are essential for the grading of the degree of disease and to provide guidance during surgeries, where indicated. Audiological investigations (Pure Tone Audiogram, Speech Audiometry) to assess the types and levels of hearing losses are imperative as these serve as a predictor to the mode of rehabilitation and management of choice.

The principles of management entail eradication of disease and restoration of function to as near normal as possible. Here, treatment could be medical, surgical or both. Active mucosal COM could be managed via aural toileting and appropriate antibiotics (topical and systemic), nasal decongestants and vitamin supplements (vitamins C and A) to enhance healing.^[82-84] Once total disease eradication and dryness is achieved, the level of function loss is assessed to decide whether there is a need for further surgical intervention (e.g. myringoplasty and tympanoplasties) or not.

An argument among otologists on whether or not total dryness should be achieved before reconstructive surgeries are embarked upon is prominent in the literatures.^[85-89] However, experience has shown that dry ears prior to reconstruction may be better favored in our environment (developing countries) considering limiting environmental factors (like limited manpower and resources) and severe cost of failures in such reconstructions.

Healed COM (i.e. dry ear with a permanent central perforation) needs no further active intervention, other than a regular review of the patient.

However, medical intervention alone is insufficient in active squamous COM (chronic ear discharge, usually foul-smelling and scanty, with marginal TM perforation and other otoscopic findings as in Table 1). An aggressive management and total excision of the squamous tissues within the middle ear is mandatory in order to prevent severe sequelae of cholesteatoma formation.^[89] Such situations require a surgical intervention based on the extent of disease and the state of the patient. Thus, canal wall-down and canal wall-up procedures, in addition to mastoidectomies of various classes, necessary to ensure total exenteration of infected mastoid cells and eradication of the diseased

tissues have been described in the past. Over time, further modifications ranging from radical, through modified to selective interventions have been developed in order to conserve functions of hearing while at the same time ensuring a total eradication of disease. The details of these procedures could be read in standard operating otology textbooks.

Specific Otitis Media

Tuberculous OM is uncommon. However, following global re-emergence of tuberculosis, the trend may be on the rise again.^[90] 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.^[91,92] 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).^[93,94] Diagnosis is confirmed by isolation of the organism from the otorrhea and treatment is with anti-tuberculous therapy.

Syphilitic OM is caused by a spirochete which affects both the cochlear and semicircular canal. Therefore, it is usually characterized by severe sensorineural hearing loss and vertigo.^[95] Diagnosis is made through a dark-field microscopic examination of the ear discharge. The treatment is with anti-syphilitic drugs (Penicillin G, Aminoglycosides or Azithromycin).^[96]

Conclusions

OM is prevalent the world over, with potentially severe complications if inadequately managed, especially in the developing countries. It is of note that in the developing countries, poverty, ignorance, dearth of specialists and limited access to medical care amongst others conspire to worsen the course of OM. Thus, hearing loss with risks of developing behavioral, speech, language and cognitive problems are not uncommon complications of OM in the developing countries.

Recommendations

OM (various classes) is a common disease that affects all age groups and races. There is preponderance of the acute variety among children and the younger age group and this responds well to early conservative management. However, COM is more recalcitrant to management and prone to severe and often life-threatening complications. Therefore, we recommend that all cases of COM, persistent/recurrent AOM, OME and specific OM be referred to the otorhinolaryngologist for prompt management.

References

1. Akinpelu OV, Amusa YB, Komolafe EO, Adeolu AA, Oladele AO, Ameye SA.

- Challenges in management of chronic suppurative otitis media in a developing country. *J Laryngol Otol* 2008;122:16-20.
2. Lasisi AO, Sulaiman OA, Afolabi OA. Socio-economic status and hearing loss in chronic suppurative otitis media in Nigeria. *Ann Trop Paediatr* 2007;27:291-6.
 3. Roy E, Hasan KhZ, Haque F, Siddique AK, Sack RB. Acute Otitis media during the first two years of life in a rural community in Bangladesh: A prospective cohort study. *J Health Popul Nutr* 2007;25:424.
 4. Klein JO. The burden of otitis media. *Vaccine* 2000;19(Suppl 1):S2-8.
 5. Anderson HR, Bagger-Sjoberg. Chapter 225 Anatomy and Embryology of external and middle ear. In: Kerr AG, editor. *Scott-Brown's otolaryngology* 7th ed. Vol 3. London: Arnold; 2008.
 6. Browning GG. Chapter 237 Condition of middle ear-classification. In Kerr AG, editor. *Scott-Brown's otolaryngology* 7th ed. Vol 3. London: Arnold; 2008.
 7. Xu X, Zhang Y, Jin Z, Chen J, Zhang S. Experimental study of aero-otitis media. *Lin Chuang Er Bi Yan Ke Za Zhi* 2006;20:1032-4.
 8. Berman S. Otitis media in developing countries. *Pediatrics* 1995;96:126-31.
 9. Dhiangra PL. Disorders of middle ear (Chapter 1. In Elsevier (Ed), *Diseases of Ear, Nose and Throat* 3rd edn. New Delhi: Gopsons; 2004. p. 80-6.
 10. O'Neill P, Roberts T, Bradley Stevenson C. Otitis media in children (acute). *Clin Evid (Online)* 2007;2007:0301..
 11. Culpepper L, Froom J, Bartelds AI, Bowers P, Bridges-Webb C, Grob P, et al. Acute otitis media in adults: A report from the International Primary Care Network. *J Am Board Fam Pract* 1993;6:333-8.
 12. Yamanaka N, Hotomi M, Billal DS. Clinical bacteriology and immunology in acute otitis media in children. *J Infect Chemother* 2008;14:180-7.
 13. Segal N, Givon-Lavi N, Leibovitz E, Yagupsky P, Leiberman A, Dagan R. Acute otitis media caused by streptococcus pyogenes in children. *Clin Infect Dis* 2005;41:35-41.
 14. Commisso R, Romero-Orellano F, Montanaro PB, Romero-Moroni F, Romero-Diaz R. Acute otitis media: Bacteriology and bacterial resistance in 205 pediatric patients. *Int J Pediatr Otorhinolaryngol* 2000;56:23-31.
 15. Oni AA, Bakare RA, Nwaorgu OG, Ogunkunle MO, Toki RA. Bacterial agents of discharging ears and antimicrobial sensitivity patterns in children in Ibadan, Nigeria. *West Afr J Med* 2001;20:131-5.
 16. Brobby GV. The discharging ear in the tropics; A guide to diagnosis and management in the district hospital. *Trop Doct* 1992;22:10-3.
 17. Hussain MA, Ali EM, Ahmed HS. Otitis media in Sudanese children: Presentation and bacteriology. *East Afr Med J* 1991;68:679-85.
 18. Nwawolo CC, Odusanya OO, Ezeanolue BC, Lilly-Tariah BD. Clinical profile of acute otitis media among Nigerian children. *West Afr J Med* 2001;20:187-90.
 19. Ibekwe TS, Nwaorgu OG, Onakoya PA, Ibekwe PU. Paediatric Otorhinolaryngology emergencies: A tropical country's experience. *Emerg Med Australas* 2007;19:76-7.
 20. Ruohola A, Meurman O, Nikkari S, Skottman T, Salmi A, Waris M, et al. Microbiology of acute otitis media in children with tympanostomy tubes: prevalences of bacteria and viruses. *Clin Infect Dis* 2006;43:1417-22.
 21. Zapalac JS, Billings KR, Schwade ND, Roland PS. Suppurative complications of acute otitis media in the era of antibiotic resistance. *Arch Otolaryngol Head Neck Surg* 2002;128:660-3.
 22. Benito MB, Gorricho BP. Acute mastoiditis: Increase in the incidence and complications. *Int J Pediatr Otorhinolaryngol* 2007;71:1007-11.
 23. Migiroy L, Yakirevitch A, Kronenberg J. Mastoid subperiosteal abscess: A review of 51 cases. *Int J Pediatr Otorhinolaryngol* 2005;69:1529-33.
 24. Hyden D, Akerlind B, Peebo M. Inner ear and facial nerve complications of acute otitis media with focus on bacteriology and virology. *Acta Otolaryngol* 2006;48:245-9.
 25. Cavel O, Fliss DM, Segev Y, Zik D, Khafif A, Landsberg R. The role of Otorhinolaryngologist in the management of central skull base osteomyelitis. *Am J Rhinol* 2007;21:281-5.
 26. Zengel P, Wiekstrom M, Jager L, Matthias C. Isolated apical petrositis: An atypical case of Gradenigo's syndrome. *HNO* 2007;55:206-10.
 27. Charlett SD, Moor JW, Jenkins CN, Coatesworth AP. A quartet of lateral sinus thrombosis, extradural abscess, subdural abscess and occipital abscess: Complications of acute mastoiditis in a pre-adolescent child. *J Laryngol Otol* 2006;120:781-3.
 28. Ciorba A, Berto A, Borgonzoni M, Grasso DL, Martini A. Pneumocephalus and meningitis as a complication of acute otitis media: case report. *Acta Otorhinolaryngol Ital* 2007;27:87-9.
 29. Kotulska-Klis M, Jaskiewicz-Burnejko E, Wilcznski K. Otogenic brain abscess. *Otolaryngol Pol* 2007;61:905-8.
 30. Kuczkowski J. Thrombophlebitis of venous sinuses in otitis media. *Otolaryngol Pol* 2007;61:769-73.
 31. Bianchini C, Aimoni C, Ceruti S, Grasso DL, Martini A. Lateral sinus thrombosis as a complication of acute mastoiditis. *Acta Otorhinolaryngol Ital* 2008;28:30-3.
 32. Chonmaitree T, Revai K, Grady JJ, Clos A, Patel JA, Nair S, et al. Viral upper respiratory tract infection and Otitis media complication in young children. *Clin Infect Dis* 2008;46:815-23.
 33. Thompson PL, Gilbert RE, Long PF, Saxena S, Sharland M, Wang IC. Has UK Guidance affected general practitioner antibiotic prescribing for Otitis media in children? *J Public Health (Oxf)* 2008;30:479-86.
 34. Hassmann-Poznanska E. Acute Otitis media-rational antibiotic treatment. *Otolaryngol Pol* 2007;61:774-8.
 35. Tanoh-Anoh MJ, Kacou-Ndouba A, Yoda M, Ette-Akre E, Sanogo D, Kouassi B. Particularities of bacterial ecology of acute Otitis media in an African subtropical country (cote d'Ivoire). *Int J Pediatr Otorhinolaryngol* 2006;70:817-22.
 36. Ibekwe TS, Ijaduola GT, Nwaorgu OG. Tympanic membrane perforations among adults in the tropics. *Otol Neurotol* 2007;28:348-52.
 37. Shokrollahi K, Tanner B. "Glue Ear": beginning of the end for head bandages after prominent ear correction? *J Plast Reconstr Aesthet Surg* 2008;61:1077.
 38. Lin J, Tsuboi Y, Rimell F, Liu G, Toyama K, Kawano H, et al. Expression of mucins in Mucoïd otitis media. *J Assoc Res Otolaryngol* 2003;4:384-93.
 39. Chung MH, Choi JY, Lee WS, Kim HN, Yoon JH. Compositional difference in middle ear effusion: Mucous versus serous. *Laryngoscope* 2002;112:152-5.
 40. Caye-thomassen P, Stangerup SE, Jorgensen G, Drozdziwicz D, Bonding P, Tos M. Myringotomy versus ventilation tubes in secretory otitis media: Eardrum pathology, hearing and Eustachian tube function 25 years of treatment. *Otol Neurotol* 2008;29:649-57.
 41. De Miguel Martinez I, Macias AR. Serous otitis media in children: implication of *Alloioococcus otitidis*. *Otol Neurotol* 2008;29:526-30.
 42. Fireman P. Otitis media and Eustachian tube dysfunction: connection to allergic rhinitis. *J Allergy Clin Immunol* 1997;99:5787-97.
 43. Sade J, Fuchs C. Secretory otitis media and its sequelae. Monographs in Clinical Otolaryngology. New York: Churchill Livingstone; 1979.
 44. Cantekin EI, Bluestone CD, Fria TJ, Stool SE, Beery QC, Sabo DL. Identification of Otitis media with effusion in children. *Ann Otol Rhinol Laryngol* 1980;89:190-5.
 45. Akinlade O, Nwawolo CC, Okeowo PA. Tympanometric screening for otitis media with effusion (OME) in Nigerian Children aged 2-7 years. *Nigerian Quart J Hosp Med* 1998;8:44-6.
 46. Marseglia GL, Pagella F, Caimmi D, Caimmis S, Castellazzi AM, Poddighe D, et al. Increased risk of otitis media with effusion in allergic children presenting with adenoiditis. *Otolaryngol Head Neck Surg* 2008;138:572-5.
 47. Tong MC, Yue Y, Ku PK, Lo PS, Wong EM, van Hasselt CA. Risk factors for otitis media with effusion in Chinese school children: A nested case-control study and review of the literature. *Int J Pediatr Otorhinolaryngol* 2006;70:213-9.
 48. Timmermans K, Vanderpoorten V, Desloovere C, Debruyne F. The middle ear of cleft palate patients in their early teens: A literature study and preliminary file study. *B-ENT* 2006;2(Suppl 4):95-101.
 49. Somefun AO, Adefuye SA, Danfulani MA, Afolabi S, Okeowo PA. Adult onset otitis media with effusion in Lagos. *Niger Postgrad Med J* 2005;12:73-6.
 50. Wang SZ, Wang WF, Zhang HY, Guo M, Hoffman MR, Jiang JJ. Analysis of anatomical factors controlling the morbidity of radiation-induced otitis media with effusion. *Radiother Oncol* 2007;85:463-8.
 51. Sheu SH, Ho KY, Kuo WR, Juan KH. The probability of diagnosis of nasopharyngeal carcinoma in patients with only adult-onset otitis media with effusion. *Kaohsiung J Med Sci* 1998;14:706-9.
 52. Tong MC, Van Hasselt CA. Chapter 237b Otitis media with effusion in adults. In Kerr AG, editor. *Scott-Brown's otolaryngology* 7th ed. Vol 3. London: Arnold; 2008. p. 3388-94.
 53. Daly KA, Hunter LL, Lindgren BR, Margolis R, Giebink GS. Chronic otitis media with effusion sequelae in children treated with tubes. *Arch Otolaryngol Head Neck Surg* 2003;129:517-22.
 54. Dhooge I, Desloovere C, Boudewyns A, Van Kempen M, Dachy JP. Management of otitis media with effusion in children. *B-ENT* 2005;Suppl 1:3-13.
 55. Jezewska E, Kukwa A, Jablonska J, Wozniak M. The efficacy of tympanopuncture in children with OMS. *Otolaryngol Pol* 2008;62:288-90.
 56. Martin J, Burton MJ, Rosenfeld RM. Extracts from The Cochrane Library: Antihistamines and/or decongestants for otitis media with effusion (OME) in children. *Otolaryngol Head Neck Surg* 2007;136:11-3.

57. Ramana YV, Nanda V, Biswas G, Chittoria R, Ghosh S, Sharma RK. Audiological profile in older children and adolescents with unrepaired cleft palate. *Cleft Palate Craniofac J* 2005;42:570-3.
58. Di Francesco R, Paulucci B, Nery C, Bento RF. Craniofacial morphology and otitis media with effusion in children. *Int J Pediatr Otorhinolaryngol* 2008;72:1151-8.
59. Ratomski K, Skotnicka B, Kasprzycka E, Zelazowska-Rutkowska B, Wysocka J, Anisimowicz S. Evaluation of percentage of the CD19+CD5+ lymphocytes in hypertrophied adenoids at children with otitis media with effusion. *Otolaryngol Pol* 2007;61:962-6.
60. Hong CK, Park DC, Kim SW, Cha CI, Cha SH, Yeo SG. Effect of paranasal sinusitis on the development of otitis media with effusion: Influence of Eustachian tube function and adenoid immunity. *Int J Pediatr Otorhinolaryngol* 2008;72:1609-18.
61. Diacova S, McDonald TJ. A comparison of outcomes following tympanostomy tube placement or conservative measures for management of otitis media with effusion. *Ear Nose Throat J* 2007;86:552-4.
62. Browning GG, Merchant SN, Kelly G, Swan IR, Canter R, McKerrow WS. Chronic otitis media chapter 237c. In Kerr AG, editor. *Scott-Brown's otolaryngology* 7th ed. Vol 3. London: Arnold; 2008. p. 3395-445.
63. Gerald BH, Kristina WR. Otitis media and middle ear effusions. In: Ballenger's *Otorhinolaryngology Head and Neck Surgery* 16th edn, Spain: Williams and Wilkins; 1996. p. 249-60.
64. Browning GG. The unsafeness of 'safe' ears. *J Laryngol Otol* 1984;98:23-6.
65. Browning GG. Aetiopathology of inflammatory conditions of external and middle ear. In: Kerr AG, editor. *Scott-Brown Otolaryngology*, 6th edn. Vol 3. London: Arnold; 1977.
66. Ologe FE, Nwawolo CC. Chronic Suppurative otitis media in school pupils in Nigeria. *East Afr Med J* 2003;80:130-4.
67. Rupa V, Jacob A, Joseph A. Chronic suppurative otitis media: Prevalence and practices among rural south Indian children. *Int J Pediatr Otolaryngol* 1999;48:217-21.
68. Berman S. Otitis media in developing countries. *Pediatrics* 1995;96:126-31.
69. Olubango OO, Amusa YB, Oyelami OA, Adejuyigbe E. Epidemiology of Chronic suppurative otitis media in Nigerian children. *Int J Otorhinolaryngol* 2008;5.
70. Bobby R. Update on otitis media. *BCM* 2005.
71. Poelmans J, Tack J. Extraoesophageal manifestations of gastro-oesophageal reflux. *Gut* 2005;54:1492-9.
72. Smith -Vaughan HC, Sriprakash KS, Mathews JD, Kemp DJ. Nonencapsulated *Haemophilus influenzae* in Aboriginal infants with otitis media: Prolonged carriage of P2 porin variants and evidence for horizontal P2 gene transfer. *Infect Immun* 1997;65:1468-74.
73. Gibney KB, Morris PS, Carapetis JR, Skull SA, Smith-Vaughan HC, Stubbs E, et al. The clinical course of acute otitis media in high risk Australian Aboriginal children: A longitudinal study. *BMC Pediatr* 2005;5:16.
74. Saini S, Gupta N, Aparana S, Sachdeva OP. Bacteriological study of paediatric and adult chronic suppurative otitis media. *Indian J Pathol Microbiol* 2005;48:413-6.
75. Vikram BK, Khaja N, Udayashankar SG, Venkatesha BK, Manjunath D. Clinico-epidemiological study of complicated and uncomplicated chronic suppurative otitis media. *J Laryngol Otol* 2008;122:442-6.
76. De Azevedo AF, Pinto DC, de Souza NJ, Greco DB, Goncalves DU. Sensorineural hearing loss in chronic suppurative otitis media with and without cholesteatoma. *Rev Bras Otorhinolaryngol (Engl Ed)* 2007;73:671-4.
77. Olusesi AD. Otitis media as a cause of significant hearing loss among Nigerians. *Int J Pediatr Otorhinolaryngol* 2008;72:787-92.
78. Dankuc D, Milosevic D, Savic L. Simultaneous extracranial and intracranial otogenic complications. *Med Pregl* 2000;53:409-12.
79. Matin MA, Khan AH, Khan FA, Haroon AA. A profile of 100 complicated cases of chronic suppurative otitis media. *J R Soc Health* 1997;117:157-9.
80. Deric D, Arsovic N, Dordevic V. Pathogenesis and methods of treatment of otogenic abscess. *Med Pregl* 1998;51:51-5.
81. Trojanowska A, Trojanowski P, Olszanski W, Klatka J, Drop A. How to reliably evaluate middle ear diseases? Comparison of different methods of post-processing based on multislice computed tomography examination. *Acta Otolaryngol* 2007;127:258-64.
82. Cameron C, Dallaire F, Vezina C, Muckle G, Bruneau S, Ayotte P, et al. Neonatal Vitamin A deficiency and its impact on acute respiratory infections among preschool Inuit children. *Can J Public Health* 2008;99:102-6.
83. Jones R, Smith F. Fighting disease with fruit. *Aust Fam Physcians* 2007;36:863-4.
84. Jones R, Smith F. Are there health benefits from improving basic nutrition in a remote Aboriginal community? *Aust Fam Physician* 2006;35:453-4.
85. Skotnicka B, Hassmann-poznanska E. Myringoplasty in children-success factors. *Otolaryngol Pol* 2008;62:65-70.
86. Uyar Y, Keles B, Koc S, Ozturk K, Arbaq H. Tympanoplasty in pediatric patients. *Int J Pediatr Otorhinolaryngol* 2006;70:1805-9.
87. Onal K, Uguz MZ, Kazikdas KC, Gursoy ST, Gokce H. A multivariate analysis of otological, surgical and patient-related factors in determining success in myringoplasty. *Clin Otolaryngol* 2005;30:115-20.
88. Albera R, Ferrero V, Lacilla M, Canale A. Tympanic reperforation in myringoplasty: Evaluation of prognostic factors. *Ann Otol Rhinol Laryngol* 2006;115:875-9.
89. Mustafa A, Hysenaj Q, Latifi X, Ukimeraj L, Thaci H, Heta A, et al. Managing chronic otitis media with cholesteatoma report of 223 patients seen in a 5 year period. *Niger J Med* 2008;17:20-4.
90. Matsumoto T. Molecular biology of tuberculosis. *Kekkaku* 2007;82:933-40.
91. Emmett JR, Fischer ND, Biggers WP. Tuberculosis mastoiditis. *Laryngoscope* 1977;87:115-63.
92. Awan MS, Salahuddin I. Tuberculous otitis media: two case reports and literature review. *Ear Nose Throat J* 2002;81:792-4.
93. Sethi A, Sethi D, Passey JC. Coexistent acute pyogenic and tubercular petrous apicitis: A diagnostic dilemma. *J Laryngol Otol* 2006;120:875-8.
94. Prasad KC, Sreedharan S, Chakravarthy Y, Prasad SC. Tuberculosis in the head and neck: Experience in India. *J Laryngol Otol* 2007;121:979-85.
95. Ahmad I, Lee WC. Otonerosyphilis masquerading as neurofibromatosis type II. *Oral J Otolaryngol* 1999;61:33-40.
96. Lukehart SA. Syphilis. Chapter 153. In Harrison's Principles Of Internal Medicine 6th edn. Vol 1. USA. McGraw-Hill Companies; 2005. p. 977-85.

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