Review Article

Otitis Media and Middle Ear Effusion - An Overview

Deepti Pandey*

*Consultant, Otorhinolaryngologist, Shri Laxminarayan Marwari Multispeciality Charity Hospital, Varanasi, Uttar Pradesh.

Dr Deepti Pandey is currently providing services as otolaryngology consultant at Sriram Laxminarayan Marwadi Hindu Charitable Hospital at Varanasi, Uttar Pradesh. Her MBBS and DLO studies were at SNHL Medical College and BJ Medical College Ahmedabad, Gujrat respectively. She completed furthur studies at the Vivekanand Institute of Medical Sciences, Kolkata and was awarded the Diplomate of National Board of Examinations (DNB) in otorhinolaryngology. She has since worked at Krishna super-speciality hospital, Nainital, Uttarakhand before the current engagement. Although, worked under experts engaged in cochlear implant, her research interest lies in common problems. She has published 16 research articles in the field, based on work in very diverse regions of the country.

Corresponding author - Deepti Pandey (drdpbent@gmail.com)

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Abstract

Otitis media and middle ear effusion are major health care burden across the globe. The prevalence in developing countries mostly exceeds the WHO stipulated emergency level of 4 %. Failure of Eustachian tube function is key pathogenic element of effusion in middle ear cavity involving complex pathophysiology. Acute otitis media accompanies upper respiratory infection or allergy. Its acute symptoms almost always attract treatment yet, on resolution of acute stage effusion over brief period, occurs in middle ear cavity. Recurrences are driven by risk factors. Chronic effusion is not symptomatic but impairs hearing function that adversely affects intellectual and behavioural development of children. Untreated infection coupled to risk factors would cause suppurative otitis media and perforation of ear drum. The later accompanies purulent ear discharge, and threatening complication s for middle ear integrity and intracranial abscess and thrombosis. The narrative attempts to overview approaches to timely diagnosis and medical and surgical management in current practice for spectrum of the disease.

Key Words: Otitis media, Middle ear effusion, Chronic suppurative otitis media, Ear infection

Introduction

Otitis media (OM) simply means inflammation of middle ear. Hearing loss due to OM is worldwide public health issue. Acute otitis media complications occur in children and old while those due to chronic disease occur in young adults, particularly as cholesteatoma. Patients with intracranial complications remain hospitalized for long periods of time with significant disability and mortality. High incidence of OM in face of inaccurate diagnosis and possible serious sequel make it important health challenge. The disease spectrum may vary from self-limiting to prolonged and complicated disease.

Acute otitis media (AOM) is mostly due to infection and is among commonest maladies in paediatric outdoors. Spontaneous healing takes place in many during adolescence years, however intervening years cast significant impact on academic performance. Virtually all children by age of 6 may get at least one episode of otitis media. Most regions of developing countries have prevalence of otitis media above 4% constituting emergency as per WHO stratification[1].

Otitis media and Middle ear effusion

AOM involves rapid onset of symptoms and signs of up to three weeks duration. After an episode of AOM there is high incidence of persisting middle ear effusion of mean duration of 40 days[2]. It is classified as recurrent acute otitis media (RAOM) if three episodes occur in six months or four in twelve months and there is otoscopic normalization during the inter-crisis period[3]. Chronic otitis media with effusion (CSOM) indicates middle ear inflammation with liquid collection in middle ear space (ME). Chronic disease is variously stated as serous/secretory OM or glue ear. It implies presence of fluid in middle ear for three months or longer. Tympanic membrane is intact and there may not be signs and symptoms of infection. Middle ear effusion may be serous thin watery liquid, mucoid-thick, viscid, mucus-like liquid or purulent pus like liquid. Discharge from ear is termed otorrhea.

Ventilation is essential for regulation of pressure within ME for optimal hearing function. Normal Eustachian tube (ET) structure and function protects ME from abnormal nasopharyngeal sound pressures and secretions. Pumping action of ET to drain ME fluid involves beginning of passive closure at ME end which progresses toward nasopharyngeal end. Eustachian tube (ET) has ability to open up during swallowing which equilibrates pressure of ME and nasopharynx. Posture also affects ET function. Elevation from horizontal position causes venous engorgement of the ET reducing air passage. Functionally and structurally immature ET as well as the immune system is determinents of susceptibility of babies to otitis media. Role of genetic predispositions have been suggested as well[4]. Children with their short, floppy ETs can reflux nasopharyngeal secretion in to middle ear during an acute respiratory viral infection. Nasopharyngeal secretion cannot enter ME with normal ET function, as gas cushion entrapped in intact ME and mastoid air cell system opposes that.

Pathogenesis

Pathogens must enter middle ear cavity (ME), through ET from bacteria and virus reservoir in rhinopharynx.

The normal tubo-tympanum is immunologically protected not only by the adaptive immune system, but also by the mucociliary system and the selected molecules of innate immunity[5]. Viruses in nasopharynx damage mucociliary function and facilitate nasopharyngeal bacterial adherence and colonization and subsequent entry in to ET[6,7]. An obstructed ET leads to negative pressure in ME following reduced ventilation. Such state when prolonged causes overgrowth of mucus producing cells and fluid transudation in ME.

Bacterial and viral infection as well as sterile effusion stimulates release of proinflammatory cytokines from mucosal immune cells. Adhesion receptors in submucosa are up-regulated mediating lymphocyte invasion and accumulation, which secrete cytokines and other inflammatory mediators. The later affect leakage of fluid from mucosa[8]. Nitric oxide and free radicals are implicated in persistent ME effusion[9,10] at stage when there is vascular engorgement and angiogenesis increasing mucosal blood flow. Increase of nitrogen in microcirculation adds creation of negative ME pressure[11].

When clearing function and immune system fails to eliminate them otitis media results. Mucus accumulation allows proliferation of infecting Streptococcus pneumonia and non-typable Haemophilus influenzae. Bacterial endotoxin may impair mucociliary activity, induce effusion, enhance mucus production and cause mucosal metaplasia. Such changes help transformation of planktonik pathogens in to biofilm. Biofilm bacteria are difficult to culture and resistant to antibiotics, hence long persisters. Emerging evidence indicates that, following upper respiratory tract infection, biofilm activity in the adenoid produces a cascade of immune mediators, causing inflammation and up-regulation of mucin genes in the middle ear mucosa, with associated reduction of ciliary function and clearance. It is likely that middle ear ventilation helps disrupt the biofilm infection by increasing and maintaining high middle ear oxygen tension.

Otitis media with effusion, OME is said to result from "hydrops-ex-vacus" mechanism. It is postulated that failure of ET opening causes gas exchange from ME to microcirculation of mucus membrane. As per Doyles postulate, possible increase in circulating inflammatory mediators from local allergic reaction in nose or stomach mucosa inflict alteration of ME-mucosal permeability and thus altered gas exchange[11]. This results in under-pressure within ME that enforces transudation and effusion. Upper respiratory infection may cause ET block inducing effusion as above. Allergic reaction also may adversely affect structure of ET mucus blanket[12].

Chronic suppurative otitis media is the chronic stage following AOM, when there is TM perforation and continuous discharge. CSOM pathogenesis involves loss of ME gas cushion due to TM perforation. In such state, allergic or inflammatory states of nasopharynx promote reflux of infected fluid through ET to ME. When tympanic membrane is perforated or following radical mastoidectomy, the gas pocket is lost. Nasopharyngeal fluid can now reflux through ET in to ME [13]. Similar mechanism is basis of otorrhea after tympanostomy tube insertion or mastoidectomy. Initially it is organisms as H. influenzi and Streptococcus pneumonae. With setting of otorrhea, organisms from external auditory canal such as staphylococci and pseudomonas invade ME causing chronic infection.

Risk Factors

Adenoids serve as bacterial reservoirs[14]. Frequent upper respiratory infections predisposes to RAOM and COME by way of inflammation and harm to mucociliary movement of ET epithelium. This may lead to atelectasis of tympanic membrane-middle ear (the high negative middle ear pressure), sterile OME or acute bacterial otitis media. Passage of nasopharyngeal fluid through ET to effusion containing ME may create mixed state seen as recurrent acute bacterial otitis media. Bacterial biofilm formation on ME mucosa fosters chronic effusion state[15]. Significant mucus content in ME effusion indicates antecedent acute infective otitis media.

Allergy is one of etiological factors of otitis media, as evident from strong association. The OME children have 4 fold higher incidence of atopic symptoms[16]. In a study increased IgE in middle ear effusion was found in 14 of 32 children with allergic rhinitis in contrast to only 2 of 45 non allergic cases[17], IgE does affect ME effusion.

Involvement of gastro-esophageal reflux is also believed to contribute to pathogenesis[18]. Exposure to pH under 4 of the ET results in ciliostasis, inflammation, edema and epithelial damage.

Epidemiology

Otitis media with effusion (OME) is most prevalent in children of 2 to 5 year age. The natural course shows constant improvement in half the sufferers over next three months. Recurrence is high in almost half the cases over a year period. The factors that cause persistence and recurrence in the victims need characterization for primary and secondary prevention.

Otitis media is universally present in infants with untreated cleft palate. Primary cause is failure of opening mechanism of ET. The risk factors influence one or more causal mechanisms viz. race, sex, age which influence structure and function of ET. Age affects immune competence also.

Age is important and several predisposing anatomical, physiological and immunological determinents make children vulnerable. Early first episode itself increases risk for recurrence and chronic otitis media. Male sex appears to have marginally higher susceptibility. Socioeconomic strata are important determinents as well. ET obstruction may be acute or chronic. Periodic ET opening helps prevent accumulation of ME effusion. ET dysfunction is seen prominently during pregnancy and during puberty in female suggesting hormonal influence also[19]. Significant hereditary determinents are understood from high familial aggregation of the disease[20].

Children with anatomical defects (cleft palate, submucous cleft), altered physiological defenses (ET dysfunction, barotraumas), congenital or acquired

immunologic deficiencies (immunoglobulin deficits, chronic grannulomatous disease, AIDS, immunosuppressive drugs) are at vulnerable to severe and recurrent or persistent disease[21]. Infants exclusively breast fed in first 3 to 6 months are somewhat less vulnerable[22]. Crowded living conditions, poor sanitation and inadequate medical care have been associated with otitis media[23]. Increased incidence associated with exposure to passive smoke is reported[22]. Sulphur dioxide pollution of environment increases both pneumococcal diseases and otitis media. Seasonal preponderance with upper respiratory infections in winter is also seen.

Diagnosis

Redness of eardrum is relied as diagnostic sign of AOM. Most important distinction between OME and acute otitis media (acute suppurative otitis media) is that the signs and symptoms of acute infection (eg. otalgia, fever) are lacking. Diagnosis cannot be based on symptoms alone because they are too vague. OME diagnosis, estimate of accompanying hearing loss and risk of complications critically determine development of quality screening, treatment and intervention programme. Specific subjective and objective measures help this[24].

Four characteristics of tympanic membrane, position, mobility, colour and degree of translucency are evaluated. Microscopic otoscopy is of utmost importance for examination of the ear. It also offers the possibility of documentation of the pathology. Pneumatic otoscopy is also a sensitive and specific method for the detection of otitis media. Opacification of the tympanic membrane can be frequently detected. Pneumatic otoscopy reveals either a retracted or convex tympanic membrane with decreased mobility. Tympanometry is useful for assessing tympanic membrane mobility and middle ear function. Pure-tone audiometry or visual reinforcement audiometry (VRA) can be used to diagnose mild hearing loss associated with OME in infants. Adjunct procedures in evaluating recurrent disease include X-ray of nasopharynx and nasopharyngeal fibroscopy to assess adenoid size, nasal examination (septum deviation, enlarged turbinates), immunologic evaluation, allergy testing, etc. Tympanocentesis is the gold standard to detect fluid in ME and sampling the same for culture or PCR typing is when there are craniofacial malformations, or the degree of hearing loss is disproportionate to clinical findings or when sensorineural hearing loss is detected, high resolution CT and MRI scans are helpful to identify the middle ear abnormalities.

OME patients do not present primarily for hearing loss, instead poor speech and language development, inattentiveness in class, behavioural problems and reduced or poor social interaction with other children are the reported concerns. In most cases hearing loss is major sign or symptom. Some children also suffer balance problems and become clumpsy[25]. History of recurrent episodes of OM or developmental concerns and audiometric hearing loss detection facilitate diagnosis. Three month monitoring of hearing status helps decision for surgical intervention. Hearing in better ear at 25-30 dB or worse constitutes indication for surgery.

It is important to identify children at risk for possible developmental sequel of OME, including speech language delay, and behavioural problems. Children who develop OME and have additional disabilities including sensorineural hearing loss, autism, syndromes (eg. Down syndrome), learning disabilities as well as other conditions, are at greater risk of persistent OME and conductive hearing loss.

Management perspectives

Organisms causing AOM are Streptococcus pneumonia or Haemophilus influenza in over third of cases each. A quarter of cases are due to Morexela catarrhalis. Rhinovirus infection contributes to prolong ME effusion[26]. In half the instances S pneumonia is penicillin resistant.

50% of cases of OME, effusion is not sterile and PCR is positive in up to 75% for microbial DNA. Initial benefits of antibiotic therapy are only transient and hence not recommended as routine. There is limited evidence for short term improvement of OME with intranasal steroids[27]. Currently only limited evidence exists on benefit of pneumococcal vaccine in OME prevention. It may be advised in cases with underlying predisposing factors[28].

Newly diagnosed OME in children is found to resolve in half the cases within 1 month, in three fourth cases by 6 month to around in 90 percent in one year. The recurrence rate is high and over some years 30 to 40 percent children suffer repeated episode. Most children with persistent OME require insertion of grommet tube. In older children nasal auto-inflammation is helpful before surgical help. Following grommet surgery, otorrhea can occur either following an upper respiratory infection or water contamination through the grommet. The tube may get blocked or extruded. Otorrhea responds to short course of antibiotic ear drops.

Chronic suppurative otitis media (CSOM), is chronic inflammation of middle ear and mastoid cavity with perforation of tympanic membrane and purulent discharge through same. The disease has usual beginning in childhood. CSOM is heavy disease burden in developing countries with reported 72 cases per 1000 population prevalence[29]. Untreated CSOM has broad range of complications related to spread of infection in adjacent structures and damage of middle ear itself. The complication range from persistent otorhea, mastoiditis, labyrinthitis, facial nerve palsy and intracranial abscess and thromboses[30]. The aim of management is to achieve safe dry ear, eradication of disease and hearing improvement.

Early effective treatment helps avoid complications. Treatment can be medical therapy directed at eradicating pathogenic aerobic and anaerobic organisms[31]. Cases resistant to medical therapy need surgical intervention. Uncomplicated CSOM treatment involves meticulous aural toilet (with sucsion /mopping up of ear debris and discharges followed by instillation of topical and systemic antimicrobial agent. In CSOM Pseudomonas aeruginosa, Staphylococcus aureus, Corynebacterium, Kleibsiella species cause infection. Anaerobes superve in in cholesteatoma.

Amoxycillin-Clavulinic acid is generally safe and effective antibiotic. Second generation cephalosporins eg, cefprozil, cefpodxime, are effective on resistant S pneumonae and group A Strepto cocci. Cefuroxime is also satisfactory for controlling H influenza. The antibiotics need be administered for 10 to 15 days. In half the cases effusion would persist.

Amoxicillin/ampicillin was more frequently used earlier but micro organisms display changing pattern of sensitivity to quinolones, cephalosporins and gentamycin. Ciprofloxacin drops are non ototoxic. Gentamycin drops though believed to possess ototoxicity are still useful as effective agent, since CSOM itself also causes sensorineural hearing loss[32]. Keeping in view the high prevalence of S.aureus and P.aeruginosa and their susceptibility to quinolones (ciprofloxacin) and cephalosporin (ceftazidime), ciprofloxacin ear drops or systemic therapy of ciprofloxacin, pipercilline or ceftazidime can be used safely in all age groups.

The study of microbial patterns and their antibiotic sensitivity determines the organisms prevalent in locations and can guide empirical treatment of the disease and its complications and also prevention of emergence of resistant strains. Control of environmental risk factors (breast feeding, passive smoke etc) makes sense in OME prevention. A causal relationship between allergy and OME has been suggested but not quantified.Management of inhalant and food allergy appears prudent. Inflammatory or infectious processes in nose, nasopharynx or paranasal sinuses also need control to avoid compromise of ET function by secondary mucosal edema. Opening of ET by autoinflation of nose has not proved beneficial[33].

Surgical treatment

Management decisions in children with OME depend on duration of effusion, laterality, and presence and severity of associated symptoms. Signs and symptoms that support clinical assessments may include: Mild intermittent ear pain and sense of fullness; Pain related rubbing of ear, irritability and sleep disturbance; inadequate response to sound in direction and extent; inattentiveness; need for loud tone communi cation suggest hearing loss.

There may be balance problems, delayed motor milestones, and speech language development, problems in school. The episodes of acute otitis media may recur with persisting OME in between. Tympanic membrane alteration eg posterior retraction pocket. The laterality (unilateral versus bilateral), duration of effusion and presence and severity of associated symptoms need documentation in medical record at each instance of OME.

OME and recurrent OM may be treated with myringotomy and insertion of tympanostomy tubes. The principle is to ventilate ME when ET function has failed. In most cases, adenoiditis causes ascending infection up the ET, its malfunction and obstruction[34]. By ventilating the middle ear with grommet tube, effusion is cleared, helping mucosal inflammation to subside and better elimination of established pathogen biofilm. Adenoidectomy reduces burden of nasopharyngeal infection.

Persistent and recurrent otitis media is often addressed by adenoidectomy as initial surgical procedure. Such cases have bacterial colonization of postnasal space early in childhood[35]. There is evidence that myringotomy and adenoidectomy can be effective treatment for OME.

Tympanostomy tube may cause early or late postoperative otorrhea. Adenoidectomy is thus preferred despite some risk of early post-op bleed and rare complications of velopharyngeal insufficiency or even nasopharyngeal stenosis. The procedure does more benefit than harm[36]. Adenoidectomy is recommended for OME (unless child has an overt or submucous cleft palate), and reduces need for future operations by 50 percent[37]. Benefit of adenoidectomy is apparent at age of 2 years and best in 3year and older, irrespective of adenoid size. There is no role for tonsillectomy[38].

Tympanic membrane perforation

Major consequence of tympanic membrane perforation is conductive hearing loss. Normal hearing is defined as an air-bone hearing gap of less than 25 dB. Hearing aids may be an alternative to cope with hearing disability but definitive therapy is surgery. Surgical closure is recommended in children with established chronic otitis media with perforation. Successful myringoplasty closes perforation and improves hearing. Ossicular damage requires specific correction with tympano plasty.

Myringoplasty and tympanoplasty are valid for application at any age. Prior adenoidectomy predicts greater success of the functional outcomes[39]. Myringoplasty is closure of the perforation of parstensa of tympanic membrane, undertaken to restore hearing capability, mixed infections and persistent ear discharge. The graft material commonly used is temporalis fascia. Cartilage or perichondrium from tragus may also be used. Graft takes up and hearing improvement depends on size and site of perforation and surgical technique and skill. Age and gender of patient, past perforation history, duration of dry ear prior to surgery and presence or absence of infection during surgery are important determinents of success. Underlay technique of myringoplasty is simpler and widely used. Overlay technique suits repair of anterior quadrant perforations of tympanic membrane[39]. Patients with small perforation display most hearing gain soon after healing. Patients with large perforation exhibit progressive gain in hearing function as closure of air-bone gap over more than year after surgery.

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References

1) WHO Ciba Found. Workshop. Prevention of hearing impairement from otitis media. Ciba Foundation London. 1996.

2) Shurin, P.A, Pelton, S.I, Donner, A, Klein, J.O. Persistence of middle ear effusion after acute otitis media in children. N Engl J Med. 1979; 300:1121-3.

3) Rovers MM, Schilder AG, Zielhuis GA, Rosenfeld RM. Otitis media. Lancet. 2004; 363:465-73.

4) Casselbrant ML, Mandel EM, Fall PA, Rockette HE, Kurs-Lasky M, Bluestone CD, Ferrell RE. The heritability of otitis media: a twin and triplet study. JAMA. 1999; 282: 2125-30.

5) Lee HY, Andalibi A, Webster P, Moon SK, Teufert K, Kang SH, Li JD, Nagura M, Ganz T, Lim DJ. Antimicrobial activity of innate immune molecules against Streptococcus pneumoniae, Moraxella catarrhalis and nontypeable Haemophilus influenzae. BMC Infect Dis. 2004; 4:12.

6) Giebink GS, Ripley ML, Wright PF. Eustachian tube histopathology during experimental influenza-A virus infection in the chinchilla. Ann Otol Rhinol Laryngol. 1987; 96: 199-206.

7) Bernstein JM, Hard R, Cui ZD, So N, Fisher J, Ogra PL. Human adenoidal organ culture: a model to study nontypable Haemophilus influenzae (NTHI) and other bacterial interactions with nasopharyngeal mucosa implications in otitis media. Otolaryngol Head Neck Surg. 1990; 103: 784-91.

8) Nonomura N, Giebink GS, Zelterman D, Harada T, Juhn SK. Early biochemical events in pneumococcal otitis media: arachidonic acid metabolites in middle ear fluid. Ann Otol Rhinol Laryngol. 1991; 100: 385-8.

9) John EO, Russell PT, Nam BH, Jinn TH, Jung TT. Concentration of nitric oxide metabolites in middle ear effusion. Int J Pediatr Otorhinolaryngol. 2001; 60: 55-8.

10) Shigemi H, Egashira T, Kurono Y, Mogi G. Role of superoxide dismutase in otitis media with effusion. Ann Otol Rhinol Laryngol. 1998; 107: 327-31.

11) Doyle WJ, Seroky JT. Middle ear gas exchange in rhesus monkeys. Ann Otol Rhinol Laryngol. 1994; 103: 636-45.

12) Doyle WJ, Skoner DP, Seroky JT, Fireman P, Gwaltney JM. Effect of experimental rhinovirus 39 infection on the nasal response to histamine and cold air challenges in allergic and nonallergic subjects. J Allergy Clin Immunol. 1994; 93: 534-42.

13) Ohashi Y, Nakai Y, Okamoto H, Ohno Y, Sakamoto H, Sugiura Y, Kakinoki Y, Tanaka A, Kishimoto K, Washio Y, Hayashi M. Serum level of interleukin-4 in patients with perennial allergic rhinitis during allergen-specific immunotherapy. Scand J Immunol. 1996; 43: 680-6.

14) Bluestone CD, Wittel RA, Paradise JL, Felder H. Eustachian tube functions as related to adenoidectomy for otitis media. Trans Am Acad Ophthalmol Otolaryngol. 1972; 76: 1325-39.

15) Ehrlich GD, Veeh R, Wang X, Costerton JW, Hayes JD, Hu FZ, Daigle BJ, Ehrlich MD, Post JC. Mucosal biofilm formation on middle-ear mucosa in the chinchilla model of otitis media. JAMA. 2002; 287: 1710-5.

16) Kraemer MJ, Richardson MA, Weiss NS, Furukawa CT, Shapiro GG, Pierson WE, Bierman CW Risk factors for persistent middle-ear effusions. Otitis media, catarrh, cigarette smoke exposure, and atopy. JAMA. 1983; 249: 1022-5.

17) Bernstein JM, Lee J, Conboy K, Ellis E, Li P. The role of IgE mediated hypersensitivity in recurrent otitis media with effusion. Am J Otol. 1983; 5: 66-9.

18) Tasker A, Dettmar PW, Panetti M, Koufman JA, P Birchall J, Pearson JP. Is gastric reflux a cause of otitis media with effusion in children? Laryngoscope. 2002; 112: 1930-4.

19) Derkay CS. Eustachian tube and nasal function during pregnancy: a prospective study. Otolaryngol Head Neck Surg. 1988; 99: 558-66. .

20) Rovers M, Haggard M, Gannon M, Koeppen-Schomerus G, Plomin R. Heritability of symptom domains in otitis media: a longitudinal study of 1,373 twin pairs. Am J Epidemiol. 2002; 155: 958-64.

21) 21.The link between allergic rhinitis and otitis media. Curr Opin Allergy Clin Immunol. 2002; 2: 21-5.

22) Uhari M, Mäntysaari K, Niemelä M. A meta-analytic review of the risk factors for acute otitis media. Clin Infect Dis. 1996; 22: 1079-83

23) Kero P, Piekkala P Factors affecting the occurrence of acute otitis media during the first year of life. Acta Paediatr Scand. 1987; 76: 618-23.

24) Gravel JS, Karma P, Casselbrant ML, Marchisio P, Andalibi A, Passàli D, Bellussi L, Post CJ, Dhooge I, Vernon-Feagans L, Hunter LL. Recent advances in otitis media. 7. Diagnosis and screening. Ann Otol Rhinol Laryngol Suppl. 2005; 194: 104-13.

25) Cohen H, Friedman EM, Lai D, Pellicer M, Duncan N, Sulek M. Balance in children with otitis media with effusion. Int J Pediatr Otorhinolaryngol. 1997; 42: 107-15.

26) McCracken GH Jr. Treatment of acute otitis media in an era of increasing microbial resistance. Pediatr Infect Dis J. 1998; 17: 576-9.

27) Straetemans M, Sanders EA, Veenhoven RH, Schilder AG, Damoiseaux RA, Zielhuis GA. Pneumococcal vaccines for preventing otitis media. Cochrane Database Syst Rev. 2004; 1: CD001480.

28) Ologe FE, Nwawolo CC. Prevalence of chronic suppurative otitis media (CSOM) among school children in a rural community in Nigeria. Niger Postgrad Med J. 2002; 9: 63-6.

29) Healy GB Resbe KW. Otitis media and middle ear effusion, In Ballenger JJ ed. Ballengers otorhinolaryngology and head nech surgery BC Decker Inc 2003.

30) Brook I. The role of anaerobic bacteria in chronic suppurative otitis media in children: implications for medical therapy. Anaerobe. 2008; 14: 297-300.

31) English MG, Northern JL, Fria T. Chronic otitis media as a cause of sensorineural hearing loss. Arch Otolaryngol.1973; 98: 18–22.

32) Stangerup SE, Sederberg-Olsen J, Balle V. Autoinflation as a treatment of secretory otitis media. A randomized controlled study. Arch Otolaryngol Head Neck Surg. 1992; 118: 149-52.

33) Gates GA. Otitis media the pharyngeal connection. JAMA. 1999; 282: 987-9.

34) Smith-Vaughan H, Byun R, Nadkarni M, Jacques NA, Hunter N, Halpin S, Morris PS, Leach AJ. Measuring nasal bacterial load and its association with otitis media. BMC Ear Nose Throat Disord. 2006; 6:10.

35) American Academy of Family Physicians; American Academy of Otolaryngology-Head and Neck Surgery; American Academy of Pediatrics Subcommittee on Otitis Media With Effusion. Otitis media with effusion. Pediatrics. 2004; 113: 1412-29.

36) Gates GA, Avery CA, Prihoda TJ, Cooper JC Jr. Effectiveness of adenoidectomy and tympanostomy tubes in the treatment of chronic otitis media with effusion. N Engl J Med. 1987; 317: 1444-51.

37) Maw R, Bawden R. Spontaneous resolution of severe chronic glue ear in children and the effect of adenoidectomy, tonsillectomy, and insertion of ventilation tubes (grommets). BMJ. 1993; 306: 756–760.

38) Ribeiro JC, Rui C, Natercia S, Jose R, Antonio P. Tympanoplasty in children: A review of 91 cases. Auris Nasus Larynx. 2011; 38 :21-5.

39) Shah H and Bhalodia N Overlay versus underlay type I tympanoplasty with various graft material: A prospective study International Journal of Scientific Research 2004; 3: 1-6.

Nicotine redemption

Nicotine has a bad reputation as it is the chief ingredient of tobacco products. It is addictive and is responsible for the physical dependence associated with cigarette smoking. But it is a powerful nAChR agonist. This action may be beneficial in neurodegenerative disorders. In a new study done in mice, the effects of low, medium and high doses of nicotine on appetite, body weight, anxiety and nAChR levels were investigated. It was found that nicotine in high doses induced reduction in food intake and weight gain but caused elevated levels of nAChR without inducing anxiety. These results suggest a possible role for nicotine in the treatment of neurodegenerative disease and in prevention of aging of brain. However, nicotine use is forbidden in children and adolescents. The authors are careful to point out that this is definitely not an advertisement for tobacco smoking.

(Texas A&M University news release, accessed 23 September 2016.
http://www.newswise.com/articles/can-nicotine-protect-the-aging-brain)

- Dr. K. Ramesh Rao