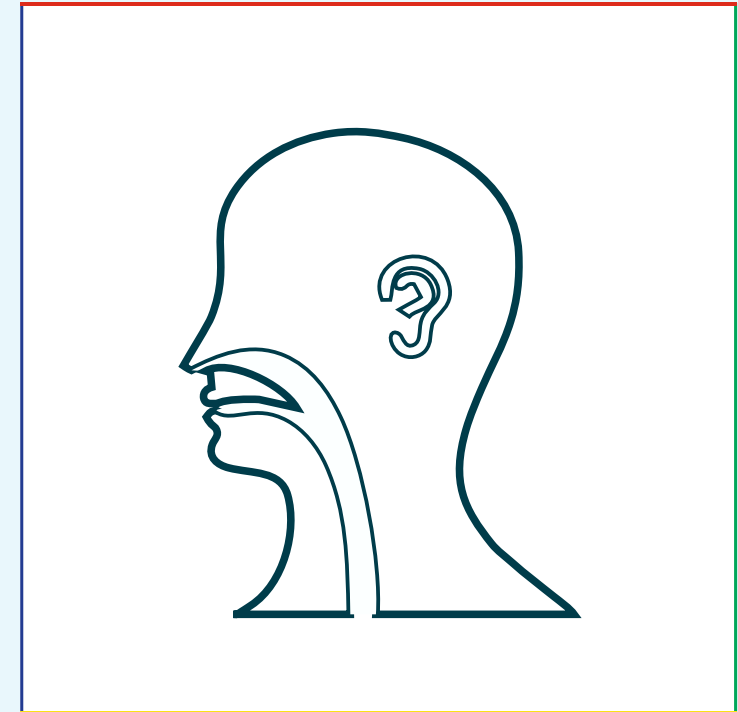




STANDARD TREATMENT GUIDELINES
OTORHINOLARYNGOLOGY



DEPARTMENT OF HEALTH AND FAMILY WELFARE
GOVERNMENT OF KERALA

KERALA.HEALTH



Directorate of Medical Education,
Medical College P.O,
Thiruvananthapuram- 695 011,
Kerala
Ph: 0471-2528575, 2528582
www.health.kerala.gov.in

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STANDARD TREATMENT GUIDELINES
for
OTORHINOLARYNGOLOGY



Foreword

At the outset, I appreciate the work done by the respective thematic teams and coordination done by the DME. The Standard Treatment Guidelines (STG) were prepared and published in 2021 in the thick of the Covid pandemic. On the last page of these volumes the road map was mentioned. The few points are mentioned here for the recall.

“The Department of Health has been taking a systematic approach of creating and enabling multiple initiatives with a focus on prevention along with improving health care services. Health care service delivery is one of the most important services and is always seen as a barometer to assess the Governance. While it is important to develop infrastructure, an essential prerequisite is to develop systems and processes to bring in standardization in management of patient care. The foundation is laid and we take up the challenge to work on the unfinished agenda.”

It was mentioned in the road map to have institutional mechanism to ensure updation of Standard Treatment Guidelines. The next step that was suggested was to do analysis of Karunya Arogya Suraksha Padhati (KASP) and standard treatment guidelines to work on developing a Balance Score Card to give information regarding compliance from the Hospitals and to build a “feedback loop” to improve. These initiatives remained at concept level on the last page! But following detailed discussions with Dr Vishwanathan, Director Medical Education, some of the foundational things were prioritized and given an impetus to take it to finality. In this journey, many committed doctors from various Medical Colleges of respective specialties participated. The previous coordination team members and experts were also consulted and they also participated in discussions and these Standard Treatment Guidelines are prepared.

The standard treatment guidelines will be made available in the Kerala Health portal (health.kerala.gov.in). This will enable the resource book availability not only to people within the state but to all in the country and outside our borders as well. I am confident that it will be used by students and practicing doctors. We request inputs based on the research from the Specialists and Experts. The teams shall continue to update and make any required changes in the STG by doing periodic updates.

The most important thing we all need to internalize is to have a shared vision and

work as a team to reach to a state of 'excellence'. If we take a look at the preparation of the Directorate Medical Education Management Information System, documents of each Medical Colleges, it provides information regarding 'what we are, what we do and what we aspire to do', pandemic preparedness, AMR accreditation and many more such initiatives taken on scale, which are all outcomes of collective TEAM work. This has laid a foundation for involving all the stakeholders including undergraduate and postgraduate students. This should encourage the teams in Medical Colleges to believe in themselves and build future initiatives on such a sound platform.

I express my sincere thanks to Dr Vishwanathan for his patience and bearing with relentless follow ups! I also take this opportunity to thank each and every team and their members and everyone from Directorate Medical Education and Medical Colleges who supported these initiatives.

I would like to express my sincere gratitude to all those who have contributed to publish these Standard Treatment Guidelines.

I wish all the success to DME team to make Kerala MCH as a premier knowledge hub in Medical Science.

Dr Rajan Khobragade IAS

Additional Chief Secretary
Health & Family Welfare and
AYUSH Department
Govt of Kerala.



Message

Patient care today demands evidence-based, standardized, and contextually relevant clinical practice. In this regard, the publication of the **Second Edition of the Standard Treatment Guidelines** marks an important step forward in strengthening the quality, consistency, and accountability of healthcare delivery in Kerala.

The first edition laid a strong foundation for uniform clinical practice across specialties and super specialties. Since then, advances in medical knowledge, evolving treatment modalities, and the growing need for periodic updating have made it essential to revisit and refine these guidelines. The present edition reflects this commitment to continuous improvement and clinical excellence.

I am pleased to note that subject experts from various disciplines of Government Medical Colleges, private institutions and professional bodies have contributed as resource persons in the preparation of these guidelines. Their academic expertise, practical insight, and dedicated involvement have greatly enriched this edition. I deeply appreciate the sincere efforts of all the conveners, contributors, and coordinators whose collective commitment and teamwork made this publication possible.

These guidelines will serve as a valuable reference for clinicians, teachers, trainees, and healthcare institutions, helping to promote evidence-based decision-making and improve patient outcomes. I am confident that this edition will further support standardization of care and contribute to the advancement of medical education and clinical practice in the State.

I congratulate everyone involved in this commendable effort and commend this publication to all healthcare professionals.

Dr. K. V. Viswanathan

Director of Medical Education
Government of Kerala

Committee for Revision of Standard Treatment Guidelines

Otorhinolaryngology

Convenor

Dr. Suchit Roy B R, Professor of ENT, GMC Thiruvananthapuram

Contributors

Dr. Divya GM, Associate Professor (CAP) of ENT, GMC Kannur

Dr. Sankar Mahadevan, State President, AOI Kerala, Senior consultant ENT, Dr. Sankar's ENT Hospital

Dr. Sindhu V Nath, Professor (CAP) of ENT, GMC Konni

Dr. Salima Rema Windsor, Professor (CAP) of ENT, GMC Thiruvananthapuram

Dr. Susan James, Professor of ENT (CAP), GMC Thiruvananthapuram

Dr. Muhammed Noushad, Senior Consultant, Dr. Noushad's ENT Hospital & RC, Kochi

Dr. Manoj M P, Senior Consultant ENT, MESIARC, Kozhikode

Dr. Jayakumar R Menon, Sr. Consultant Laryngologist, Dr. Jayakumar's group, Thiruvananthapuram

Dr. Rupa Vedantam, Former Professor & HOD, CMC Vellore

Dr. Bini Faizal, Professor & HOD of ENT, Amrita Institute of Medical Sciences, Kochi

Dr. Suma R, Professor (CAP) of ENT, GMC Kozhikode

Dr. Shibu George, Professor of ENT, GMC Kottayam

Dr. Venugopal M, Professor of ENT, GMC Kollam

Dr. Sunil Kumar K P, Professor of & HOD of ENT, GMC Kozhikode

Dr. Satheesh S, Professor & HOD of ENT, GMC Thiruvananthapuram

Standard Treatment Guidelines in Otorhinolaryngology

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Section I

Diseases of ear

ACUTE OTITIS EXTERNA

INTRODUCTION

It is defined as diffuse inflammation of the external ear canal, which may also involve the pinna or tympanic membrane. Mostly bacterial infection (*Pseudomonas aeruginosa* & *Staphylococcus Aureus*), but fungi and viruses may also be involved. Non-infectious causes include skin allergies, eczema, and psoriasis. Risk factors in development include humidity, local trauma, narrow ear canal, impacted cerumen, soapy deposits within canal, skin diseases, immunocompromise, radiotherapy or chemotherapy. There is often history of preceding canal trauma (e.g. aggressive ear cleaning, use of ear buds) OR exposure to water (e.g. swimming)

KEY POINTS IN DIAGNOSIS

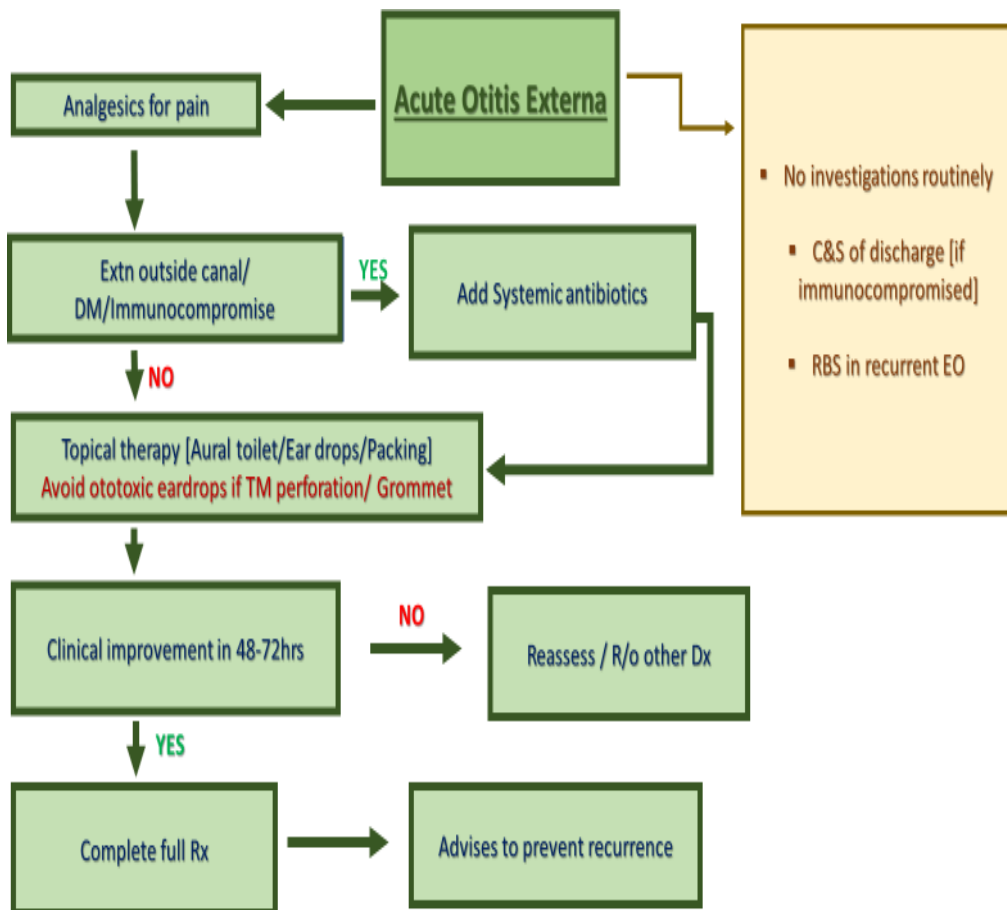
- Rapid onset; usually within 48 hours
- Symptoms: Earache (often severe) &/or jaw pain, itching, hearing loss or fullness, discharge. Pain may be aggravated by jaw movements).
- Signs: Tenderness of the tragus, pinna, or both. Diffuse oedema of ear canal oedema, erythema, or both. Serous or purulent otorrhea, pre/ post aural lymphadenitis, congested tympanic membrane, obliteration of post aural groove
- Sometimes presents as localized inflammation of hair follicle of cartilaginous canal (furunculosis); can lead to abscess formation
- If otomycosis greyish discharge with black (*Aspergillus*) or white (*Candida*) fungal colonies visualized on otoscopy
- Routinely, no investigations necessary. Culture & Sensitivity of the discharge recommended if immunocompromised. In recurrent external otitis investigate for causes of immunocompromise like Diabetes.

MANAGEMENT GUIDELINES

- Treatment depends on the severity and associated co-morbidities on presentation
- Analgesics are recommended based on the severity of pain.
- Topical antibiotic (*Ciprofloxacin/ Ofloxacin + steroid*) drops may be prescribed for the initial therapy of uncomplicated external otitis.
- Non antibiotic topical preparations may be used as an adjunct (e.g. 1% Acetic acid solution)
- Avoid ototoxic ear drops in presence of perforated tympanic membrane or grommet. Ask for history of allergy to topical drugs.

- Aural toilet or ear packing (antibiotic steroid cream) or both should be performed when the ear canal is obstructed or oedematous to facilitate topical drug delivery
- Systemic antibiotics should not be used for initial treatment of uncomplicated external otitis; they are indicated only if there is extension outside ear canal (e.g. peri-aural cellulitis) or associated co-morbidities like Diabetes (*Oral Ciprofloxacin*)
- Specific advice should be given to patients regarding the avoidance of self-cleaning or inserting anything into the ear canal and observing appropriate water precautions for the ear (eg: avoiding swimming in polluted waters).

Treatment algorithm for Otitis Externa



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MALIGNANT (NECROTIZING) OTITIS EXTERNA

INTRODUCTION

Malignant (Necrotizing) Otitis Externa is an aggressive invasive infection of the external auditory canal and temporal bone, usually seen in elderly immunocompromised individuals. The infection begins as an external otitis, but spreading cellulitis along the facial planes of the bony canal can lead to osteomyelitis of the temporal bone and skull base. Unchecked patients may develop multiple cranial nerve palsies and death. The most common organism implicated is *Pseudomonas aeruginosa*, though other microbes like *Staphylococcus aureus*, *Proteus mirabilis*, *Klebsiella* species and *Aspergillus* have also been described.

CRITERIA FOR DIAGNOSIS

1. Refractory otitis externa
2. Severe nocturnal otalgia
3. Purulent otorrhea
4. Granulation tissue deep canal
5. Growth of *Pseudomonas aeruginosa* in culture
6. Presence of diabetes or any other immunocompromised state

NOTE

- Other immunocompromised states include renal failure, transplant recipients, disseminated malignancies, anti-malignancy chemotherapy, lymphoproliferative disorders and AIDS.
- Immunosenescence (immune dysfunction with advanced age), frailty and malnutrition also has been recognised to predispose, due to impaired immune response.
- Apart from the diagnostic criteria, points of importance to be noted in the diagnosis of malignant otitis externa are as follows
 - Unremitting symptoms despite treatment for at least 2 weeks favour diagnosis
 - Inflammatory changes may spread to peri-aural tissue; deep tenderness may be elicited at the soft tissue between the ramus of the mandible and mastoid tip.
 - Tympanic membrane usually appears normal (if visualised).
 - Persistent temporal headaches, altered mentation, diplopia, hoarseness, dysphagia and facial asymmetry may signify the onset of complications.

MANAGEMENT

Investigations

- Do preliminary ESR / CRP; useful to monitor disease progression and to differentiate from malignancy/ Granulomatous disease.
- Take an ear swab for culture right at the first visit (before initiating antibiotic therapy) to direct future change in treatment if needed. Re-culture may be necessary if the initial culture is negative or with poor response to antibiotic. Both liquid (Pus) & solid (Canal granulations) specimens to be sent for culture. Bacterial, Fungal and Mycobacterial culture (+ PCR) desirable.
- Biopsy of granulations required; this is to rule out differential diagnoses like malignancy or chronic granulomatous disease. Repeated negative biopsy for these conditions can be a pointer towards diagnosis of malignant otitis externa (Send biopsy for microbiological analysis also).
- HRCT temporal bone & skull should be the primary radiological
- investigation to assess the extent of disease, bone demineralization and erosions; however, CT may fail to diagnose early skull base osteomyelitis.
- MRI can assess early bone marrow involvement and extraosseous soft tissue spread. It is ideal for complications like venous thrombosis and early intracranial spread; diagnostic mimics like skull base malignancies are better differentiated.
- In most patients a combination of both HRCT and MRI would provide better assessment

Supportive investigations (Not mandatory)

- Nuclear medicine imaging preferably [18F] FDG- PET/CT may be used to assess disease resolution and decide on treatment endpoint. Use of Technetium (Tc 99) & Gallium (Ga 67) scans have declined over time.
- In refractory, culture-negative malignant external otitis, a fungal cause should be suspected; polymerase chain reaction (PCR) assay or biopsy may be useful. Deep tissue samples needed for positivity in fungal disease.

TREATMENT - MEDICAL CARE

Antibiotics

- In patients who do not require hospital admission for diabetes or pain management initial outpatient therapy may be started with oral Fluoroquinolone, Ciprofloxacin 750mg BID (The alternate drug is Levofloxacin). Should not be prescribed for

patients with fluoroquinolone allergy, history of seizure disorder, cranial neuropathy, myasthenia gravis or intracranial complications.

- Hospitalised patients may be started on 3rd generation cephalosporin, intravenous Ceftazidime, as monotherapy; Beta-lactam inhibitor, Piperacillin Tazobactam is an alternative.
- Antibiotic therapy may also be changed as directed by the sensitivity pattern of the initial culture swab obtained
- The duration of antibiotic therapy should be prolonged (at least 6-8 weeks). Early discontinuation can lead to relapse
- Reduction in pain within 48 hours of starting therapy may be taken as an indication for therapeutic response.
- ESR/ CRP estimation may be used to monitor the disease progression. Nuclear imaging may be used to decide the duration of antibiotic therapy.
- Evaluate need for antifungal treatment in non-responders; preferred drug is Voriconazole

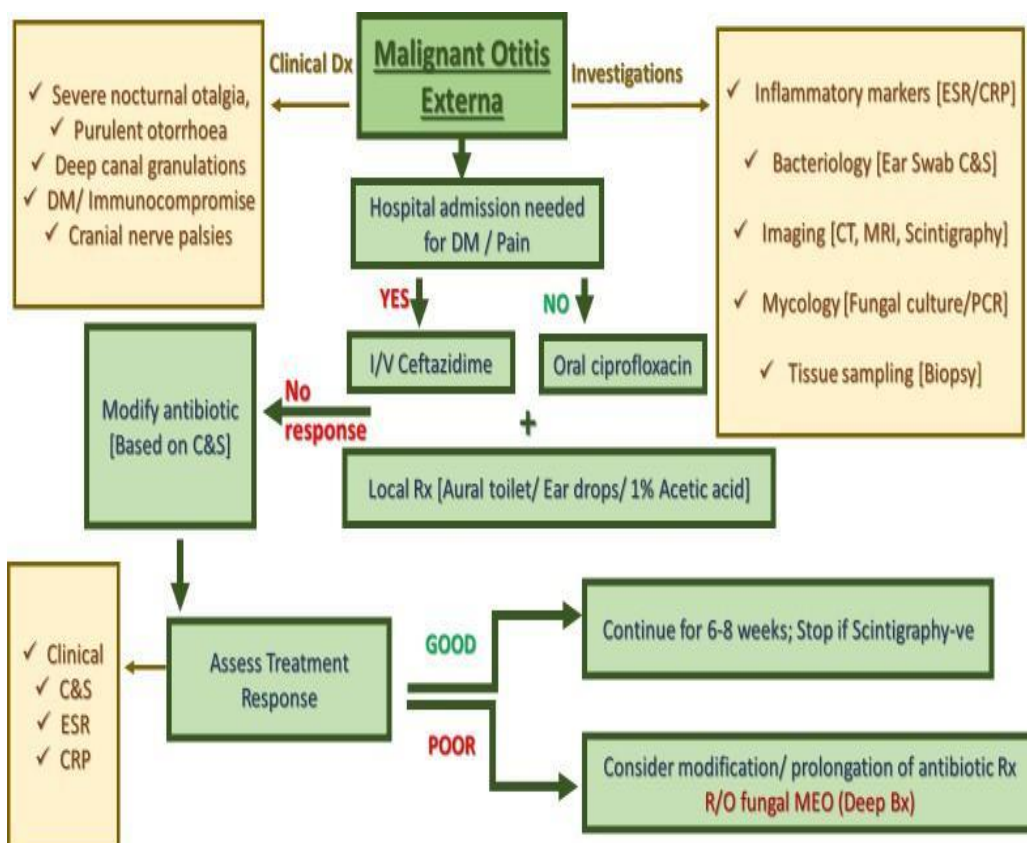
Adjuvant therapy

- Topical antimicrobial therapy may be initiated, preferably ciprofloxacin/ Ofloxacin + Dexamethasone.
- Irrigation with 1% acetic acid solution should supplement topical antimicrobials
- Aural toilet (suction clearance of discharge, removal of granulations and packing the canal with antibiotic+ steroid cream) brings down local inflammation
- Meticulous glycaemic control should be maintained; change over to insulin under the care of a diabetologist. Promote mobilization & activity.
- Evaluate and treat predisposing causes other than diabetes
- Pain management may require appropriate consultations; possibility of diabetic nephropathy to be kept in mind if prescribing NSAIDs.
- Hyperbaric oxygen therapy, if available is a useful adjunct in patients with poor treatment response, Complications or Recurrence.
- Surgery is generally reserved for obtaining deeper biopsy samples, local debridement, removal of bony sequestrum, and drainage of abscess if any.

Prognosis

Poor prognostic factors in malignant external otitis include the long duration of diabetes mellitus, advanced age, elevated inflammatory markers, fungal disease, extensive granulations, cranial nerve involvement and other neurological complications.

Treatment algorithm for Malignant external otitis



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ACUTE OTITIS MEDIA (AOM) IN CHILDREN

INTRODUCTION

AOM is the rapid onset of signs and symptoms of inflammation in the middle ear cleft. It is predominantly a disease of childhood.

History

The history of AOM varies with age. Constant features in different age groups are as follows

- Neonates: Irritability, excessive crying, ear tugging or feeding difficulties
- Older children: Fever and otalgia; there may be associated vomiting in some.
- Older children and adults: Hearing loss, earache; ear stuffiness is also noted

Prior upper respiratory catarrh, the nocturnal peak of otalgia followed by otorrhea may be common to all. Otorrhea is usually mucopurulent and may herald the onset of pain relief.

DIAGNOSIS

Pneumatic otoscopy is the gold standard. Look for

- ✓ Congestion and signs of inflammation on the tympanic membrane
- ✓ Bulging of the tympanic membrane, especially posterior quadrant
- ✓ Small perforation on TM
- ✓ Presence of an exudate oozing through TM with/without pulsation
- ✓ In difficult-to-examine children, tympanometry may show middle ear fluid and increased middle ear pressure, which contributes to the diagnosis

INVESTIGATIONS

Routinely not needed.

MANAGEMENT

1. AOM management should include **pain evaluation** and appropriate treatment
2. **Indications for antibiotics**
 - In children < 6 months even a presumptive diagnosis of AOM should receive antibiotics due to the higher prevalence of suppurative complications
 - Bilateral or unilateral AOM in children aged 6 months or above with severe signs or symptoms (moderate - severe otalgia / otalgia > 48 hours / temperature > 39°C)
 - Non-severe, bilateral AOM in children aged 6 months – 2 years

- Non-severe, unilateral AOM in children aged 6 months – 2 years or non-severe AOM in older children may be managed either with antibiotics or watchful waiting with close follow-up. This decision to withhold initial antibiotic treatment should be taken after due appraisal of parents
- Children whose symptoms have worsened or not responded to the initial antibiotic treatment within 48-72 hours should be reevaluated and treatment changed if indicated (Follow up mandatory)

3. Antibiotic policy in AOM

- Amoxicillin (30 -50 mg/kg/day) is the antibiotic of choice.
- If the child had received Amoxicillin within last 30 days or has concurrent purulent conjunctivitis, or no response even after 48-72 hrs of oral Amoxicillin, additional beta-lactamase coverage needed – Amoxicillin 45 mg/kg/day + Clavulanate 3.2mg/kg/day
- Second line antibiotics recommended are as follows
 - a. High-dose oral amoxicillin-clavulanate (80-90 mg/kg/day of amoxicillin + 6.4 mg/kg/day of clavulanate)
 - b. Oral Cefuroxime axetil (30 mg/kg/day)
 - c. Oral Cefpodoxime (10 mg/kg/day)
 - d. Oral Cefdinir (14mg/kg/day)
 - e. Ceftriaxone IM or IV (50 mg/kg/day)
- For children allergic to penicillin and beta-lactam, Macrolides (Azithromycin/ Clarithromycin) or Clindamycin may be used (If only allergic to penicillin added option of cephalosporins)
- Duration of antibiotic treatment
- For Amoxicillin, amoxicillin-clavulanate, oral cephalosporins & Clarithromycin: In children < 2 years - 10 days; in children > 2years – 5 to 7days
- For Azithromycin – 5days
- For Ceftriaxone – 1-3 doses depending on persistence of symptoms

4. Indications for Myringotomy

- Failed antimicrobial treatment with signs of local or systemic sepsis
- AOM with facial palsy (possible dehiscence of facial canal)
- Other complications requiring culture for adequate therapy

- Children who are immuno-suppressed or immunocompromised
- Neonates with AOM (more likely to have an unusual / invasive pathogen)

5. Recurrent AOM

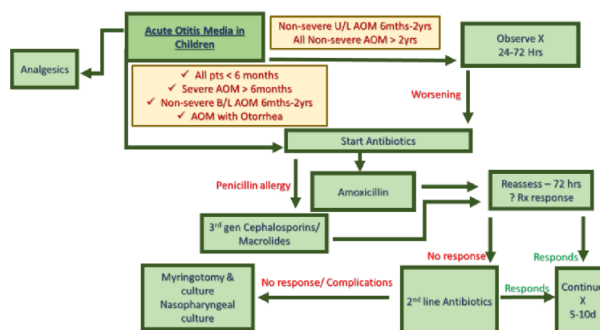
More than three episodes in six months or four or more episodes in one year

In children with recurrent AOM associated with middle ear effusion ventilation tube insertion is recommended to reduce the frequency of AOM episodes. Adenoidectomy may be considered as an option along with ventilation tube insertion in recurrent AOM.

6. Other recommendations

- Pneumococcal conjugate vaccination is recommended in all children
- Exclusive breastfeeding is recommended for six months or longer

Treatment algorithm for Acute Otitis Media in Children



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OTITIS MEDIA WITH EFFUSION (OME)

INTRODUCTION

Synonyms: Serous Otitis Media, Secretory Otitis Media, Non-suppurative Otitis Media, Middle ear effusion, Glue ear

DEFINITION

OME is defined as the presence of fluid in the middle ear without signs or symptoms of acute ear infection. The non-purulent effusion may either be mucoid or serous. It is commoner in pediatric age group.

Chronic OME is defined as presence of middle ear fluid for more than 3 months from the date of onset (if known) or date of diagnosis (if onset unknown).

OME may occur during an upper respiratory infection, spontaneously because of poor eustachian tube function, or as an inflammatory response following AOM, most often between the ages of 6 months - 4 years

DIAGNOSIS

Clinical pointers to possibility of OME in children in history

- Hearing difficulty (hard of hearing, difficulty to perceive in a group, asking for repetition)
- Recurrent earache
- Repeated ear infections
- History of recurrent cold or persistent nasal obstruction
- Delayed speech and language development
- Behavioral problems, inattention, problem child
- Fall of grades at school

Note

At-risk children: Clinicians should determine if a child with OME is at increased risk for speech, language, or learning problems from middle ear effusion because of baseline sensory, physical, cognitive, or behavioral factors. These include the following

- Permanent hearing loss (independent of OME)
- Other speech and language disorders
- Autism spectrum disorder / other developmental disorders
- Syndromes (e.g., Down) with cognitive, speech, or language delay

- Cleft palate & other cranio-facial disorders
- Blindness or uncorrectable visual impairment

Assessment

- Pneumatic otoscopy with visualized & documented presence of middle ear fluid should be the corner stone of diagnosing OME in children. Otoscopy should be performed in children with earache, deafness or both to rule out OME.
- If the diagnosis is uncertain after otoscopy, Tympanometry should be obtained in children with suspected OME. Ensure ear canal is free of wax or foreign body before sending for tympanometry
- Age-appropriate hearing tests should be done persistent OME for ≥ 3 months OR for OME of any duration in 'at-risk children'.
- Imaging modalities like CT or MRI are not routinely indicated in children with OME. [However, it may be of value in adults presenting with unilateral OME when a nasopharyngeal lesion or mass compressing eustachian tube must be ruled out.]

TREATMENT

- Clinicians may manage children with OME who is not at-risk with watchful waiting for three months since most are self-limiting.
- Antibiotics, systemic steroids, decongestants or antihistamines are not recommended for treatment of OME (These may be used only in management of coexisting conditions in patients with OME).
- Auto inflation has been found beneficial in older children with OME and may be advised when being kept under watchful waiting.
- Offer advises to avoid passive smoking, allergens, feeding infants supine and promote breast feeding
- For surgical management children with OME may require grommet insertion +/- Adenoidectomy

Guidelines for grommet insertion for OME in children

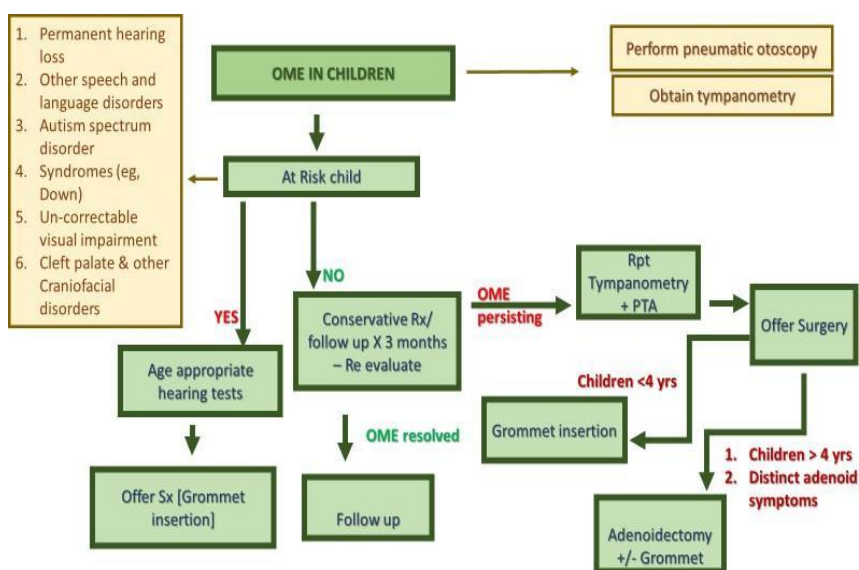
- Bilateral OME in children for > 3 months despite conservative management, + documented hearing loss [B type tympanogram and / or PTA with > 40 dB CHL].
- Unilateral or bilateral OME in children for > 3 months AND symptoms attributable to OME (poor school performance, behavioral problems, ear discomfort, or reduced quality of life).

- Children with history of recurrent AOM who have middle ear effusion during assessment
- OME in at-risk children [since at increased risk for speech, language, or learning problems from middle ear effusion]
- Grommet insertion NOT indicated in children with a single episode of OME < 3 months duration.

Guidelines for associated adenoidectomy with grommet insertion

- Adenoidectomy may be combined with grommet insertion for persistent OME in a children four years or older.
- Grommet insertion alone needed for OME in children < 4 years; adenoidectomy indicated only if there is a distinct indication (e.g., severe nasal obstruction, OSAS) other than OME.

Treatment algorithm for Otitis Media with Effusion (OME)



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CHRONIC OTITIS MEDIA

INTRODUCTION

Chronic otitis media is long standing inflammation of the middle ear cleft

CLASSIFICATION

- **Healed COM** – Healed perforation (Thin replacement membrane, usually circular in outline; use pneumatic otoscopy to confirm) OR Tympanosclerosis (chalk patches on TM).
- **Active mucosal** – Central perforation with middle ear inflammation (mucosal congestion, exudate, polyps or granulations in middle ear)
- **Active squamous** – Cholesteatoma
- **Inactive squamous** – Retraction pockets (either in pars tensa or flaccida)
- **Inactive mucosal** – Central perforation with normal middle ear

DIAGNOSIS

Diagnosis mainstay is history & pneumatic otoscopy to sub-classify into different types as mentioned above

Investigations

- **Pure tone audiometry**–Essential investigation; Look for degree of hearing loss, magnitude of A-B gap, associated sensorineural loss if any, and hearing status of the opposite ear
- **Culture& sensitivity** – Is only of limited value; may be done in active ears especially if suppurative complications are anticipated. Both bacteriological and fungal culture desirable.
- **Radiology** – Preferred modality is CT scan; high resolution scans of the temporal bone (axial and coronal sections) may be obtained. A radiation risk vs benefit assessment should be made in individual patients. In general indications for HRCT imaging in COM are as follows
 - Suspected complications
 - Inability to assess the tympanic membrane (e.g. Narrowed canal)
 - Disproportionate hearing loss
 - Associated congenital anomalies
 - Pediatric cholesteatoma (due to extensive nature of the disease in this age group)

- May also be considered an option prior to tympanomastoid surgery especially revision procedures.
- MRI scan (diffusion weighted non-echo planar image) is recommended in patients to rule out recurrence of cholesteatoma after canal wall up procedures; It is also of use to rule out intracranial complications like lateral sinus thrombophlebitis.
- **Outpatient microscopy / Otoendoscopy** – Desirable as it complements clinical assessment, helps to plan surgery and aid in preliminary management (aural toilet).

MANAGEMENT

● Healed otitis media

- Needs no further treatment if hearing is normal.
- If there is conductive hearing loss of >30 dB the patient may be offered option of tympanoplasty
- Informed consent for guarded success in the presence of tympanosclerosis or middle ear adhesions.
- Hearing aid is an alternative for surgery

● Mucosal disease

- Aural toilet and topical antibiotic (fluoroquinolones) + topical steroids should be the preliminary management in active ears which aids in better clinical evaluation as well.
- Appropriate management of comorbidities like respiratory allergies, tonsillopharyngitis, nasal and sinus disease as indicated.
- Definitive management is tympanoplasty with appropriate ossicular reconstruction.
- Option of combining cortical mastoidectomy in persistent mastoid reservoir despite appropriate initial management.

● Inactive squamous

- Adults - If the retraction pocket is stable with no hearing loss, regular follow up can be the option with suction clearance under microscope as and when necessary. If there is associated hearing loss of >30db &/ or persistent discharge tympanoplasty need be done.
- Children - There is a greater likelihood of progression of the pocket. If the hearing is normal frequent follow up needed with a low threshold for surgery; offer cartilage tympanoplasty in the event of noticing progression of the

retraction. If there is significant hearing loss tympanoplasty with appropriate reconstruction should be offered.

- **Active squamous**

- Patients with confirmed cholesteatoma need surgery to eradicate the disease, improve the hearing and have a self-cleaning epithelialized ear.
- Mastoid exploration with appropriate tympanoplasty procedure should be contemplated
- Type of mastoidectomy (Canal wall up vs Canal wall down) may be decided based on the extent of disease, mastoid cellularity, reliability of the patient, hearing status of the opposite ear, surgeon's experience and surgeon preference.

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CHILDHOOD HEARING LOSS

INTRODUCTION

Hearing loss is a well-known prominent risk for speech and language developmental delay. Without appropriate intervention, children with hearing loss are at risk of delays in multiple cognitive functions, such as working memory and executive functions. These problems have long-term educational and occupational consequences. Hearing loss has also been found to affect a child's quality of life, particularly in the school and social domains, as well as behaviour and behavioural disorders. This guideline is intended to correct or reduce the effects of delay in identifying and managing paediatric hearing loss.

AIMS OF MANAGEMENT

- All infants should undergo hearing screening before discharge from the birth hospital and no later than one month of age.
- All infants whose initial birth screen and subsequent rescreening warrant additional testing should have an appropriate audiological evaluation to confirm their hearing status no later than three months.
- A concurrent or immediate comprehensive otologic evaluation should occur for infants confirmed to be deaf or hard of hearing.
- All infants who are deaf or hard of hearing in one or both ears should be referred immediately to early intervention to receive targeted and appropriate services.
- A simplified, coordinated entry point into an intervention system appropriate for identified children is optimal.
- Early intervention services should be offered through an approach that reflects the family's preferences and goals for their child. They should begin immediately after diagnosis but no later than six months of age.
- The child and family should have immediate access, through their audiologist, to high-quality, well-fitted, and optimised hearing aid technology.
- Access to cochlear implants (CI), hearing assistive technologies, and visual alerting and informational devices should also be assured, depending on the child's needs.

At-risk neonates to be screened at birth

- H/O Maternal infections (TORCH)
- Prematurity
- Family history of hearing loss

- Neonatal hyperbilirubinemia with Serum bilirubin >20gm/dl Low birth weight <2000gm
- Birth injuries/ asphyxia with NICU care >48 hours
- Maternal drug & alcohol abuse
- Maternal diabetes
- Toxemia of pregnancy
- Anatomical abnormalities of the head & neck
- Genetic syndromes (Treacher Collins, Goldenhar, Alport's, Usher, CHARGE, Waardenburg)

Conditions causing permanent hearing loss after birth

- Ear infections
- Ototoxic medications
- Meningitis/Encephalitis Measles
- Chickenpox Mumps Head injury
- Noise exposure

Screening new-born infants for hearing loss

- Otoacoustic emissions (OAE) and Automated Auditory brainstem response (AABR) to screen newborn infants with no risk factors for developing disabling hearing loss.
- Testing is to be done one month after birth. Factors affecting results, like ear debris or ear infections, are to be ruled out as far as possible before testing.
- If the initial screening test fails, the test is repeated after a month, and another failure mandates a detailed hearing evaluation.
- It is to be emphasised that trained personnel should do testing in a quiet room. A trained audiologist should monitor the testing and audit the results regularly. Audiologists do not need to perform all tests but should be responsible for the results.
- Children at risk for hearing loss should proceed directly to detailed hearing evaluation without screening.
- Parents or caregivers of children who pass neonatal hearing screening should be counselled to periodically evaluate milestones of hearing, speech, and language abilities and report for further testing if there is any concern.

Detailed hearing evaluation for children with hearing loss

- This includes Tympanometry, OAE; Click evoked brainstem-evoked audiometry (BERA), and Auditory steady-state response (ASSR).
- Including Tone-burst or Chirp BERA to detect frequency-specific thresholds in addition to ASSR is desirable.
- Magnetic Resonance imaging (MRI) should be offered to any child with severe to profound hearing loss after detailed testing to identify cochleovestibular anomalies early so that appropriate interventions can begin early.

Test battery at 0- 6 months

Otoacoustic emissions, Tympanometry, acoustic reflexes, BERA, ASSR

Test battery at 6- 36 months

Tone-burst or Chirp BERA, ASSR, Otoacoustic emissions, tympanometry with reflexes, Behavioural observation audiometry (BOA), conditioned play audiometry (CPA) & visual reinforcement audiometry (VRA), Speech audiometry. Bone conduction BERA

Test battery at 3 – 5 years

Pure tone audiometry (PTA), speech audiometry, Tympanometry with reflex testing, and Otoacoustic emissions. BERA and ASSR could be used to correlate the findings of PTA.

NOTE

It must be emphasised that standard Click-evoked BERA alone should not be used to quantify hearing loss in young children or infants. In addition, for suspected cases of Auditory Neuropathy Spectrum Disorder (ANSD), cochlear microphonics must be looked for in BERA.

Radiological evaluation

All children with severe to profound hearing loss must be imaged for abnormalities of the peripheral and central auditory pathway

- MRI can be used to rule out cochleovestibular anomalies and the integrity of the nerves. It will also assess the brain for central nervous system abnormalities that may preclude the child's neurological development.
- Computerized Tomography (CT) scanning is not recommended for children under 18 months old, as no additional information can be deduced from it because of the risk of radiation exposure. However, if a cochlear implant is planned, the team may decide to perform a CT scan.

Electrocardiogram

An electrocardiogram is mandatory for all children with hearing loss as they may be associated with Jervell Lange Nielsen (JLN) syndrome or long QT syndrome. These children are prone to syncope and sudden death without treatment and may pose significant risks for surgery under general anaesthesia if not adequately treated.

Genetic Testing

Genetic testing will yield no useful information that may modify treatment but will indeed have implications in the future. Parents should be offered genetic testing, but with a warning that all data currently has a minimal management role.

MANAGEMENT

Hearing Aids

- Children with middle ear effusion must not be treated before ascertaining the degree of hearing loss, as this can affect the diagnosis.
- Sensorineural Hearing loss of any degree more significant than 45dB must be immediately corrected with binaural hearing aids.
- An experienced audiologist must do Hearing aid fitting, and ear moulds are recommended along with Behind-the-ear (BTE) hearing aids.
- Auditory habilitation in the form of therapy must begin immediately following hearing aid fitting. An experienced pediatric habilitationist is best suited for handling such children.
- Asymmetric hearing loss is to be treated on merit. If one ear is normal, it is acceptable not to aid the poor-hearing ear, but the poor-hearing ear must be monitored to look for worsening hearing loss.
- Children who cannot be fitted with hearing aids (e.g., those with an absent pinna, draining ears, or an absent cochlea) should be identified early, and alternate intervention modalities should be planned.
- During therapy following the hearing aid fitting, auditory milestones must be constantly monitored, any delay must be flagged, and a hearing re-evaluation must be advised.

Bone Conduction Aids

- If there is conductive loss, immediate fitting of bone conduction hearing aids is planned. When it is difficult to identify exact bone conduction thresholds, parents must be motivated to speak to the toddler or infant loudly so that auditory input is maximised.

- Bone conduction implants (BCI) are recommended for children above three years of age, and they must be considered for uncorrectable conductive hearing loss.

Cochlear Implants

- Cochlear implants are now recommended for children with Bilateral severe to profound hearing loss who do not benefit from amplification and therapy.
- The pediatric cochlear implant criteria are expanding and should include all children who do not progress adequately with therapy, whose aided audiogram is below the speech banana and whose quality of life is impaired because of hearing loss.
- Asymmetric hearing is also an indication of cochlear implantation. If there is aidable hearing in one ear and no aidable hearing in the other, a combination of a hearing aid in the better ear and a cochlear implant in the worse ear has been shown to improve speech understanding in noise and quiet.
- The FDA has also approved cochlear implantation for unilateral profound hearing loss. Still, parents must be aware of the marginal improvement in quality of life this intervention will provide and weigh that against the cost and maintenance of the device.
- Post-meningitis profound hearing loss and impending ossification of the cochlea are emergency indications for cochlear implantation.
- Before establishing cochlear implant candidacy, ANSD patients must be carefully assessed using behavioural thresholds. It must be emphasised that outcomes following cochlear implantation in ANSD will be significantly variable.
- Finally, before advising cochlear implantation, the parental commitment to providing appropriate therapy and training for the child must be ascertained.

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BELL'S PALSY

INTRODUCTION

Bell's palsy is defined as rapid onset (within 72 hours) of unilateral LMN facial palsy without any identifiable cause (Bilateral Bell's palsy is rare). It is the most common cause of acute LMN Facial palsy

DIAGNOSIS

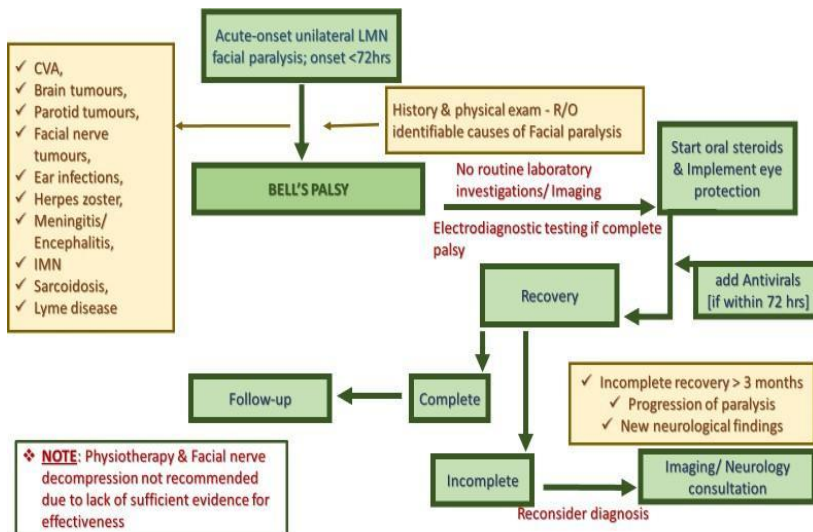
- Bell's palsy is diagnosed when no other aetiology is identified as a cause of the facial weakness. It is a diagnosis of exclusion and hence require careful elimination of other causes of facial paresis or paralysis.
- Other conditions causing facial paralysis, include stroke, brain tumours, parotid tumours, facial nerve malignancies, ear infections and systemic diseases including Herpes zoster, sarcoidosis, and Lyme disease (see treatment algorithm)
- Bell's palsy has been observed to be commoner in 15-45 age group, those with diabetes, immunocompromise or during pregnancy.
- Preceding upper respiratory catarrh has been reported.
- Bell's palsy is typically self-limiting but can cause significant temporary morbidity.

MANAGEMENT

- Detailed evaluation with history and physical examination to exclude identifiable causes of facial paralysis is recommended in all patients presenting with acute onset unilateral facial paralysis.
- Laboratory testing or diagnostic imaging is not routinely indicated when history and physical examination do not suggest an alternative cause.
- Imaging is recommended in patients with new or worsening neurologic findings, progression of paralysis or incomplete recovery even after 3 months following onset of facial paralysis.
- Electrodiagnostic testing is not recommended in incomplete facial paralysis; however, may be offered to patients with complete facial paralysis since it can provide meaningful prognostic information.
- Oral steroids are indicated within 72 hours of onset of symptoms in patients with Bell's palsy. 10-day course of Prednisone recommended (1mg/kg – total 60 mg for 5 days followed by 5-day taper).
- Oral antiviral therapy may be offered in addition to oral steroids within 72 hours of onset

- of facial paralysis in patients with Bell's palsy (Acyclovir 400 mg 5 times daily for 10 days OR Valacyclovir 500mg twice daily for 5 days)
- Eye care precautions should be carried out in patients with Bell's palsy presenting with impaired eye closure.
- Surgical decompression is not routinely indicated. However, the need for the same must be individualized based on the level of recovery of facial nerve function after onset of palsy.
- Physiotherapy is not routinely recommended in patients with Bell's palsy due to lack of evidence favoring its benefit or harm.

Treatment algorithm for Bell's Palsy



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BENIGN PAROXYSMAL POSITIONAL VERTIGO

INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is probably the most common cause of vertigo; it has been estimated that at least 20% of patients who present with vertigo have BPPV. BPPV is characterized by recurrent episodes of rotational vertigo, often severe in intensity, precipitated by change in position of the head and neck in relation to the gravity vector, associated with severe nausea and vomiting, in the absence of auditory symptoms

DIAGNOSIS

- **Other causes of vertigo** (see treatment algorithm) should be ruled out by meticulous history and clinical evaluation
- **POSITIONAL TESTS**
 - The **Dix-Hallpike positional test** is the standard positional test for BPPV. The finding of classic rotatory nystagmus with latency and limited duration is considered pathognomonic of *posterior canal BPPV*. The nystagmus induced by Dix-Hallpike test is typically torsional and up beating clockwise in left ear and anticlockwise in the right.
 - *Lateral canal BPPV* is suspected if the patient has a history compatible with BPPV and the Dix-Hallpike test exhibits horizontal or no nystagmus. **Supine head-roll test** is used to confirm diagnosis of lateral canal variant of BPPV. When the patient is rolled to the affected side, there is intense horizontal nystagmus beating to the under most (affected) ear [geotropic nystagmus]. When the patient is rolled to the healthy (nonaffected) side, there is a less intense horizontal nystagmus again beating toward the under most ear.
 - Positional tests also help to differentiate BPPV from central lesions like cerebellar infarction & brainstem tumors.
- Vestibular testing is not routinely indicated in a patient who meets diagnostic criteria for BPPV in the absence of additional vestibular signs and/or symptoms inconsistent with BPPV.
- Laboratory studies are not recommended in routine diagnosis.
- MR imaging may be used to rule out positional vertigo of central origin; however, patients who meet diagnostic criteria for BPPV with no signs and/or symptoms inconsistent with BPPV do not warrant imaging.

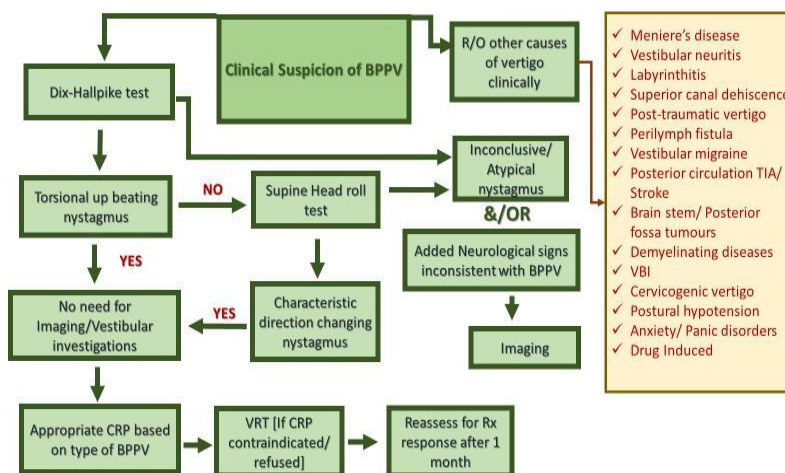
Note**Indications for Radiographic imaging of the CNS in positional vertigo**

- Patients with suspected BPPV but inconclusive or atypical findings on positional testing
- Patients with clinical history compatible with BPPV but also demonstrate added neurologic symptoms atypical for BPPV like headache and visual disturbances.
- Patients with other neurologic signs on physical examination that are not typically associated with BPPV like cranial nerve palsies or meningeal irritation.
- Positional vertigo not resolving with repeated therapeutic maneuvers.

TREATMENT

- **Canalith Repositioning Procedures (CRP)** are the mainstay of treatment in most of the cases of BPPV; since the benefit-to-risk ratio is very high, it is the obvious first choice among treatment modalities.
- The type of CRP chosen depends upon the specific semicircular canal primarily involved in the causation (**Epley** or **Semont** manoeuvres for posterior canal BPPV and *Barbecue roll maneuver* or **Gufoni manoeuvre** for lateral canal BPPV).
- Clinicians should not routinely treat BPPV with vestibular suppressant medications. However, these drugs may be used in short-term management of autonomic symptoms such as nausea / vomiting, in patients who are severely symptomatic yet refuse therapy or in patients who become severely symptomatic after CRP.
- Vestibular rehabilitation exercises, (either self-administered or by the clinician), may be offered as an option in the treatment of BPPV. It may be considered, in patients with persistent disability following CRP, refuse CRP, or are not candidates for CRP. Surgery has only a very limited role in management of BPPV and should not be routinely advocated.

Treatment algorithm for BPPV



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MENIERE'S DISEASE

DEFINITION

Meniere's disease is characterised by recurrent, spontaneous episodic vertigo, hearing loss, aural fullness and tinnitus caused by endolymphatic hydrops

Diagnostic Criteria for definitive diagnosis of Meniere's Disease

- Two or more spontaneous episodes of vertigo each lasting 20 mins-2 hrs
- Audiometrically documented low- to mid-frequency sensorineural hearing loss in one ear, on at least one occasion before, during, or after the episode of vertigo
- Fluctuating aural symptoms (Hearing loss, Tinnitus, Fullness) in affected ear
- Other causes excluded by tests

Investigations

- Pure tone audiogram – Predominantly low-frequency SNHL during early stages; involvement of other frequencies as the disease progresses. Evidence of recruitment.
- Electrocochleography (if available) – Shows SP/AP ratio > 35
- BERA can rule out retro cochlear pathology if present
- Vestibular testing has only limited role; may help to rule out other vestibular disorders
- Investigations to rule out other causes of endolymphatic hydrops – include serum TSH, ESR, CRP, VDRL, RFT, Lipid profile
- MRI brain - to rule out retro-cochlear lesions

MANAGEMENT

Advice

Low salt diet, avoidance of alcohol, smoking, caffeine, chocolates, cola, junk food and water restriction during acute attack

Therapy during acute episode

Rest, anxiolytics (Diazepam), Labyrinthine sedatives (Promethazine, Meclizine, Cinnarizine), Anti-emetics (Scopolamine)

Drug treatment in established cases

- Drugs to lower endolymph pressure - Hydrochlorothiazide, Acetazolamide

- Anti-inflammatory (if autoimmune aetiology suspected) – Corticosteroids
(May also be delivered as intra-tympanic injections)
- Intra-tympanic Gentamycin – Effective in the majority of cases; ideal in unilateral disease in older individuals (Warn patients about the potential danger of cochleotoxicity)

Surgical options

- Reserved for patients unresponsive to drug treatment or Intra-tympanic therapy
- Vestibular neurectomy, Endolymphatic shunt surgery – Conserves hearing and better suited for bilateral disease
- Labyrinthectomy achieves better vertigo control, but destroys residual hearing; ideal for unilateral disease

Vestibular rehabilitation therapy

This will improve the threshold of dizzy spells, increase confidence and well-being of the patient

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Section II

Diseases of nose

RHINOSINUSITIS

SCOPE

This protocol is designed to focus all specialists involved in management of acute/chronic rhinosinusitis (bacterial/fungal) in the secondary or tertiary care level to ensure standard scientific management. This includes the guidelines for accurate diagnosis, prompt identification of complications and scientific management. The guideline is intended for all clinicians who are likely to diagnose and manage adults with rhinosinusitis and applies to secondary or tertiary care setting in which an adult with rhinosinusitis would be identified, monitored, or managed. This guideline, however, does not apply to patients of any age with complicated rhinosinusitis and does not include guidelines for surgical management

KEY WORDS

Adult sinusitis, rhinosinusitis

INTRODUCTION

Rhinosinusitis in adults is defined (EPOS 2020) as symptomatic inflammation of the nose and the paranasal sinuses characterized by the presence of 2 or more of the following symptoms, one of which should be nose block (congestion/ obstruction) or nasal discharge (anterior or postnasal drip) +/- Facial pain/ pressure and +/- Reduction/loss of smell

Rhinosinusitis may be classified by duration as

- Acute rhinosinusitis (ARS), if less than 12 weeks' duration
- Chronic rhinosinusitis (CRS), if lasting more than 12 weeks, with or without acute exacerbations.
- Rhinosinusitis lasting 4-12 weeks being rare, decisions about whether such patients are more like ARS or CRS must be individualized.

ARS may be classified further by presumed aetiology, based on symptoms and time course as

- Acute bacterial rhinosinusitis (ABRS) or
- Viral rhinosinusitis (VRS)

ACUTE RHINOSINUSITIS

Diagnostic recommendations

- Sudden onset of two or more symptoms, one of which should be either nasal blockage/nasal obstruction/congestion /or anterior or postnasal discharge.
- $\pm$ facial pain /pressure

- $\pm$ reduction or loss of smell
- Nasal obstruction or facial pain without purulent nasal drainage is not consistent with ARS
- Acute bacterial rhinosinusitis is diagnosed when
- Symptoms or signs fail to improve within 10 days or more beyond the onset or worsen after initial improvement (double sickening) within 10 days
- Fever, cough, fatigue, hyposmia or anosmia, maxillary dental pain or ear fullness may be present
- Elevated ESR, CRP
- Anterior rhinoscopy may reveal nasal inflammation, purulent discharge, mucosal oedema and structural abnormalities if present

Investigations

- Imaging, hematological and microbiological investigations and endoscopy are not routinely required in the diagnosis of ARS, but may be done in high-risk patients
- Elevated ESR and CRP in Acute bacterial rhinosinusitis

Imaging may be done (Contrast enhanced CT)

- When a complication of ABRS (orbital, intra-cranial or soft tissue involvement) or
- CECT and MRI (T1 with Contrast, T2WI fat suppressed sequence and DWI) when
 - An alternative diagnosis (non-infectious cause of facial pain or malignancy) is suspected or
 - When the patient has modifying factors or co-morbidities that predispose to complications, including diabetes, immune-compromised state, history of facial trauma or surgery

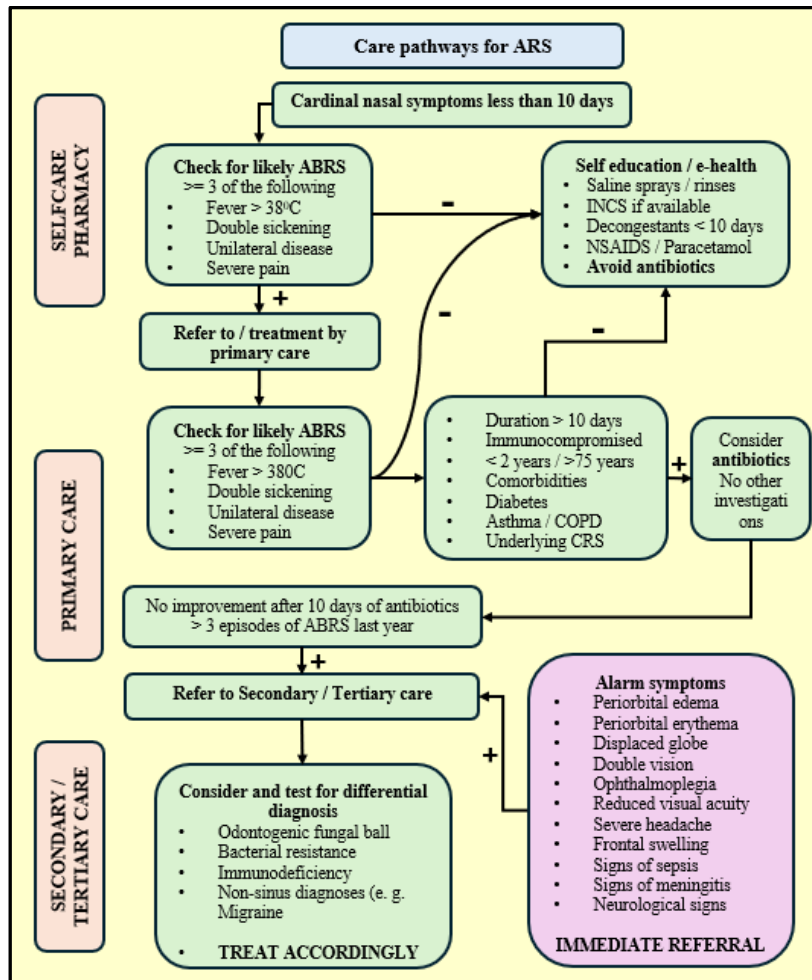
Treatment

Acute rhinosinusitis

- Analgesics may be tried for seven days before starting antibiotic therapy in mild-moderate ABRS only if follow-up is assured
- Antibiotic therapy in ABRS
- High dose Amoxycillin (90mg/kg/day in 2 divided doses) is the antibiotic of choice for 5-10 days with follow-up
- Amoxycillin – clavulanate may be given as first line to those at a high risk of being infected by an organism resistant to Amoxycillin, in moderate-severe infection, with

other co-morbidities

- In case of penicillin allergy, Doxycycline, Levofloxacin or Azithromycin for 5-10 days
- If patient fails to improve with antibiotic therapy by seven days after diagnosis or worsen during initial management, endoscopy directed pus culture & sensitivity from middle meatus and fungal smear may be sent, particularly in diabetics or immunocompromised patients. Complications of acute bacterial sinusitis will require intravenous antibiotics and further evaluation, including imaging. For Penicillin allergic patients, either doxycycline or fluoroquinolone (Levofloxacin or Moxifloxacin) is recommended as an alternative agent for empiric antimicrobial therapy.
- Reassess to exclude other causes or complications



CHRONIC RHINOSINUSITIS (with or without nasal polyps) in adults:

Chronic rhinosinusitis (CRS) is a complex disease, with a pathophysiology that is likely to be affected by multiple factors including genetic and environmental.

Diagnostic recommendations

- Presence of two or more symptoms one of which should be nasal obstruction or nasal discharge (anterior or post-nasal drip) with or without
- Facial pain/pressure
- Hyposmia/anosmia
- For >12wks
- With demonstrable disease - Either endoscopic signs of nasal polyps, and / or oedema / mucopurulent discharge primarily from middle meatus and / or CT changes; mucosal changes in the OMC or sinuses
- Normal nasal endoscopy makes it very unlikely that a patient's facial pain is due to rhinosinusitis

Investigations

- Nasal endoscopic evaluation
- CT scan nose & PNS in severe disease, to exclude malignancy, or before planning for surgery
- In the absence of symptoms, diagnosis of CRS based on radiology alone is inappropriate.
- Tissue biopsy is required to confirm diagnosis in polyp and to exclude from other pathologies especially in unilateral polyp
- Assess the patient with chronic rhinosinusitis for multiple chronic conditions that would modify management such as asthma, cystic fibrosis, immunocompromised state and ciliary dyskinesia.

Treatment protocol***Chronic Rhinosinusitis CRS without polyp:***

- Mild rhinosinusitis (based on VAS scale): intranasal corticosteroid with saline nasal irrigation is sufficient. Evaluation of nasal allergy, non-allergic rhinitis, gastroesophageal reflux, immunodeficiency, cystic fibrosis and systemic disorders causing CRS should be performed (based on history) in all cases and appropriate treatment instituted.
 - If there is improvement, continue intranasal corticosteroid, reduce the dose

to minimum possible and follow up

- If there is no improvement after three months or there is moderate-severe rhinosinusitis with mucosal oedema and purulent discharge on endoscopy – intranasal corticosteroids with nasal saline irrigation and culture & sensitivity of nasal discharge should be done with culture directed antibiotics or consider long- term antibiotics (Azithromycin/ Clarithromycin for 6-12weeks) (Low quality evidence EPOS 2020)
- When considering long-term macrolide treatment, a serum IgE is helpful in trying to identify likely responders (Patients with normal IgE respond to long term antibiotic therapy)
- Do CT scan PNS and consider surgery based on findings
- After surgery, follow up with saline irrigations and intranasal corticosteroids

Chronic rhinosinusitis with polyp

It is becoming increasingly prevalent and is particularly difficult to treat using conventional therapies.

A. Mild/ Grade 1 polyp

- Intranasal saline irrigation with corticosteroid nasal spray
- Review after three months
- If there is improvement, continue the same treatment and review every month.

B. Moderate / Grade 2 polyp

- Intranasal saline irrigation with corticosteroid nasal spray- Consider increasing the dose of spray
- Oral steroid (prednisolone at 0.5mg/kg body weight) for 2 weeks and review.
- If there's an inadequate response, evaluate aspirin sensitivity. If positive add Montelukast tablets.
- Consider long term antibiotic (Doxycycline 100mg per day for 3 weeks) (low quality evidence (EPOS 2020)
- If improvement, follow-up with intranasal corticosteroid
- If no improvement, do radiological evaluation and proceed with surgery.

C. Severe/ Grade 3 Polyps

- Apart from intranasal corticosteroids with saline spray, start a short course

of oral corticosteroid.

- If no improvement after one month, do radiological evaluation, surgery & follow up.
- Consider urgent investigation and intervention if there is evidence of complications.

Patients with classical unilateral or bilateral antrochoanal polyp arising from the maxillary sinus form a unique type of CRSwNP who will not respond to medication alone and will need endoscopic excision of the polyp with wide middle meatal antrostomy and complete removal of the base of the polyp pedicle to prevent recurrence.

CHRONIC RHINOSINUSITIS IN CHILDREN

The four most common symptoms are cough, rhinorrhoea, nasal congestion and postnasal drip with a slightly higher predominance of chronic cough. Adenoids are a predominant contributor to CRS in young children. Management is similar.

- Nasal corticosteroid treatment is a first line treatment in CRS with and without nasal polyps in children.
- In moderate-severe cases or in mild cases without improvement after maximal medical therapy, consider adenoidectomy alone. Maxillary sinus wash or balloon dilatation is recommended if feasible.
- Radiological evaluation & FESS is indicated in further persistence of symptoms (Lund- Mackay score >5)
- FESS is the initial surgical option in patients with cystic fibrosis, nasal polyposis, antrochoanal polyposis, or AFS

ALLERGY AND RHINOSINUSITIS

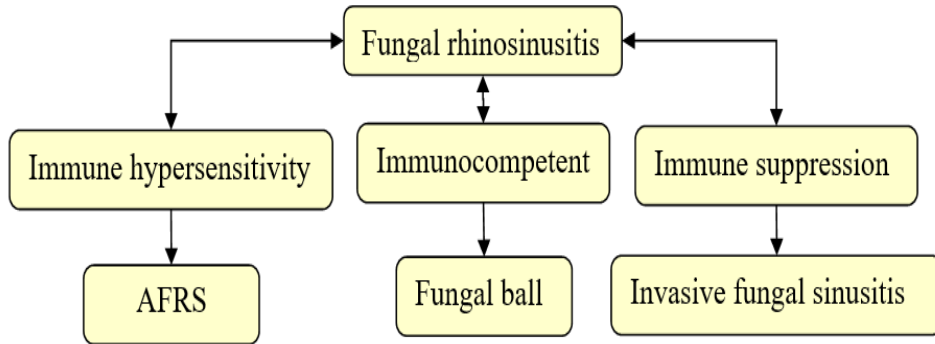
If allergy testing is positive and appears clinically relevant based on individual assessment management

- Environmental control measures, pharmacotherapy or immunotherapy as an immunomodulating approach.
- The clinician may obtain testing for allergy or immune function in evaluating a patient with CRS or acute / recurrent rhinosinusitis when aggressive management has failed or when sinusitis is associated with otitis media, bronchiectasis or pneumonia
- Treatment with biologicals and immunotherapy

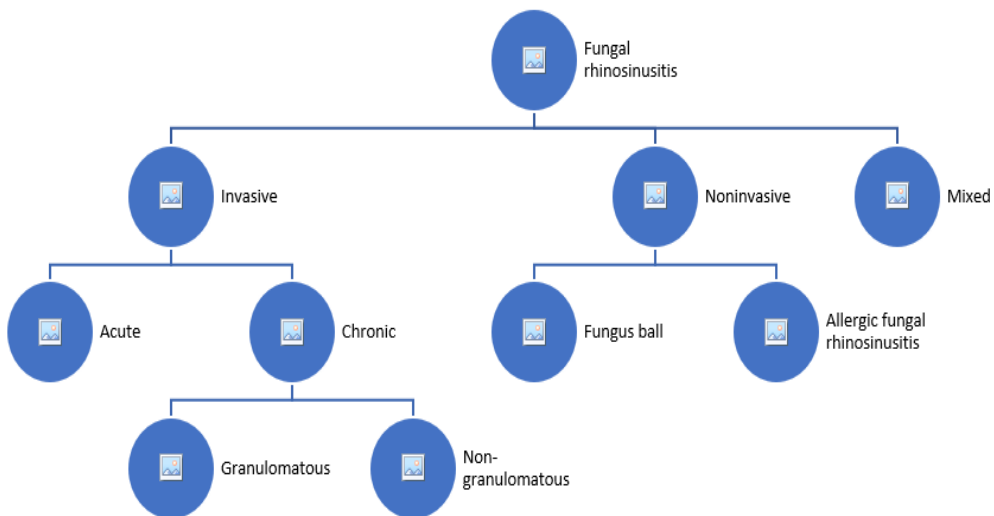
FUNGAL RHINOSINUSITIS

Incidence of fungal rhinosinusitis is on the rise in the last few years with a sharp increase

in cases of invasive fungal rhinosinusitis during the COVID pandemic. The diagnosis and management of different types of invasive and non-invasive fungal sinusitis as well as mixed fungal rhinosinusitis is specific to that type of fungal sinusitis alone.



- The definition and categorization of fungal rhinosinusitis is still controversial, but the most accepted system divides the patients with normal immunity and immunocompromised patients
- *Diagnosis – classified into invasive and non-invasive based on presence or absence of tissue invasion by fungal hyphae*
- *Invasive: Acute invasive, chronic invasive, chronic granulomatous invasive*
- *Non-invasive: Saprophytic fungal infestation, fungal ball, and fungus related eosinophilic rhinosinusitis including AFRS*



Classification of Fungal rhinosinusitis (Modified from deShazo et al, 1997)

Clinical signs of suspicion:

- Unresolved rhinosinusitis even after adequate antibiotic therapy
- Extensive intranasal sloughing
- Numbness of cheek, headache, facial pain
- Diminution or loss of vision on side of involvement
- Ptosis, External ophthalmoplegia on side of involvement
- Black eschar in nasal mucosa especially the middle turbinate
- Palatal blanching especially unilateral
- Uncontrolled Diabetes mellitus with ketoacidosis, signs of renal impairment, immunocompromised patient

Invasive fungal sinusitis:

Diagnostic criteria:

- Rhinosinusitis confirmed by radiological imaging (although this can be minimal) or show tissue destruction
- Histopathological demonstration of fungal hyphae in mucosa, sub-mucosa, bone or blood vessel, positive fungal smear on fungal staining (GMS staining is the most sensitive in identifying fungi)
- CECT scan may be negative in early cases. MRI with contrast is complementary
- Fungal culture remains the gold standard, which may not be rewarding always

Acute invasive:

Acute invasive fungal rhinosinusitis is the terminology preferred to fulminant, or necrotizing fungal rhinosinusitis

- Disease of < 4 weeks duration in immunocompromised patients
- High index of suspicion is needed for early diagnosis
- This is the most dangerous and life-threatening form of fungal sinusitis
- Usually caused by saprophytic fungi of the order Mucorales, including Rhizopus, Rhizomucor, Absidia, Mucor, Cunninghamella, Mortierella and Apophysomyces species and by Aspergillus.
- Seen in immuno-compromised patients and very rarely in immunocompetent individuals. Apophysomyces elegans causes acute invasive rhinosinusitis in immunocompetent individuals.

- The disease process is characterized by an angioinvasion of fungal hyphae producing thrombosis and mucosal infarction, and subsequent tissue necrosis
- Early physical findings on endoscopy include pallor of mucosa, decreased bleeding on abrasion of mucosa.
- Later black eschar with necrosis develops which can extent to palate.
- Full blood count, renal function, blood glucose, capillary ketones, inflammatory markers like ESR, CRP, should be evaluated
- Specific biomarkers like Serum galactomannan, for invasive aspergillosis can be useful.
- Galactomannan and (1,3) beta- D-glucan each have a sensitivity and specificity of around 80% in detection of invasive aspergillosis
- Diagnostic nasal endoscopy and biopsy from necrotic and pale mucosa and KOH staining, fungal culture and histopathological evaluation as early as possible

Treatment

Must be started as soon as provisional diagnosis is made which includes

- Surgical debridement to remove all necrotic tissues which may be repeated
- Appropriate anti-fungal therapy (Amphotericin (Preferably liposomal amphotericin B) if
- the species identification is not available and in mucormycosis/ Voriconazole in
- invasive aspergillosis and candidiasis)
- Reversal of immunocompromised state wherever possible
- Long term follow-up
- Once the patient is clinically stable, switch over to oral Posaconazole or Isavuconazole

Chronic invasive

- Seen in subtle immuno-compromised state like elderly diabetic.
- Patients present with symptoms of long-standing sinusitis over 4 weeks duration.
- Symptoms are usually not acute; fever and mental status changes are absent.
- Commonly caused by *Aspergillus fumigatus*, occasionally *mucor*.

Treatment

Surgical debridement, antifungal therapy, control of co-morbidities like diabetes and long-

term follow-up

Chronic Granulomatous Invasive Rhinosinusitis

- Relatively rare.
- Usually seen in immunocompetent individuals.
- Usually caused by *Aspergillus flavus*.
- Disease has a relatively slow time course over three months
- Patients present with an enlarging mass in the cheek, orbit, nose and sinuses.

Treatment

Surgical debridement, anti-fungal therapy and long-term follow-up.

ANTIFUNGAL THERAPY

Invasive aspergillosis / invasive candidiasis

Primary therapy

Voriconazole (6mg/kg IV BD for 2 doses, followed by 4mg/kg IV BD; oral dosage is 200mg BD)

Alternate therapy

- Conventional Amphotericin B (if tolerated) (0.5-1.5mg/Kg/day), preferably Liposomal amphotericin B (3-5mg/kg/day IV in 500ml 5% dextrose as a single dose over 3hrs)
- ABLC (5mg/kg/day IV), Posaconazole (300 mg bd for one day and then 300mg od orally to be continued for a minimum of 3months), Itraconazole (100-200mg bd)
- Caspofungin (70mg day1 IV and 50mg/day thereafter) in salvage cases

Invasive Mucor Rhinosinusitis

- Liposomal amphotericin B (LAMB) or conventional amphotericin (if tolerated) remains to be the drug of choice. Recommended concentration of liposomal amphotericin B is daily dose of 3-5mg/kg/day for a minimum of 14 days or till the patient is clinically stabilized. Duration of therapy should be determined on an individual basis followed by oral Posaconazole 300mg BD for a day followed by 300mg OD for a minimum of three months OR Isavuconazole 200mg TID for 48hrs followed by 200mg OD.
- In patient with brain involvement liposomal Amphotericin B should be given at a dose at 10 mg/kg administered daily
- Inj Liposomal Amphotericin B is given after test dose (0.25mg(1ml) to be mixed in

100ml 5% Dextrose & infused over 30 minutes

- Pre and post hydrate patient with 500 ml NS over 30 minutes
- Laboratory evaluation of RFT, LFT and CBC should be performed regularly at least once weekly. Serum electrolytes potassium and magnesium should be regularly monitored. appropriate potassium supplementation may be required
- Prompt control of diabetes/diabetic ketoacidosis also equally important
- Common side-effects of liposomal amphotericin B include infusion reactions, phlebitis, acute kidney injury, hypokalemia, hypomagnesemia and anemia whereas side effects of Posaconazole include nausea, vomiting, diarrhea, headache, QTc prolongation and hepatotoxicity and that of Isavuconazole include nausea, vomiting, diarrhea, headache, rash, oedema, hypokalemia and hepatotoxicity

Non- invasive Fungal Rhinosinusitis Fungal ball

- Extra mucosal masses of fungal hyphae without any evidence of invasion.
- Commonly caused by *Aspergillus Fumigatus*
- Usually single sinus is involved.
- Most commonly involved sinuses are maxillary and sphenoid sinuses
- No invasive component to the fungal colonization.

Treatment:

- Removal of fungal ball through endoscopic sinus surgery.
- Antifungal treatment is not recommended.

Allergic Fungal Rhinosinusitis (AFRS)

- Allergic reaction to aerosolized environmental fungi
- A subset of polypoid chronic rhinosinusitis that is characterized by the presence of eosinophilic mucin with non-invasive fungal hyphae within the sinuses and type 1 hypersensitivity to fungi
- Usually of the *Aspergillus* species or (rarely) *Dematiaceous* species
- In an immunocompetent host.

Diagnostic criteria

- Five major and three minor criteria
- Patients with allergic fungal rhinosinusitis (AFRS) normally presents with
- Signs and symptoms of nasal airway obstruction caused by unilateral or bilateral sinonasal polyposis, loss of smell, proptosis or facial swelling. Some patients may have allergic

symptoms like sneezing, watery rhinorrhoea and epiphora too.

➤ **Major criteria** include

- Type 1 (IgE mediated) hypersensitivity to fungi
- Nasal polyposis
- Characteristic hetero-dense radiographic findings (double density sign)
- Eosinophilic mucin without fungal invasion into sinus tissue
- Positive fungal stain of sinus contents removed at the time of surgery for aspergillus

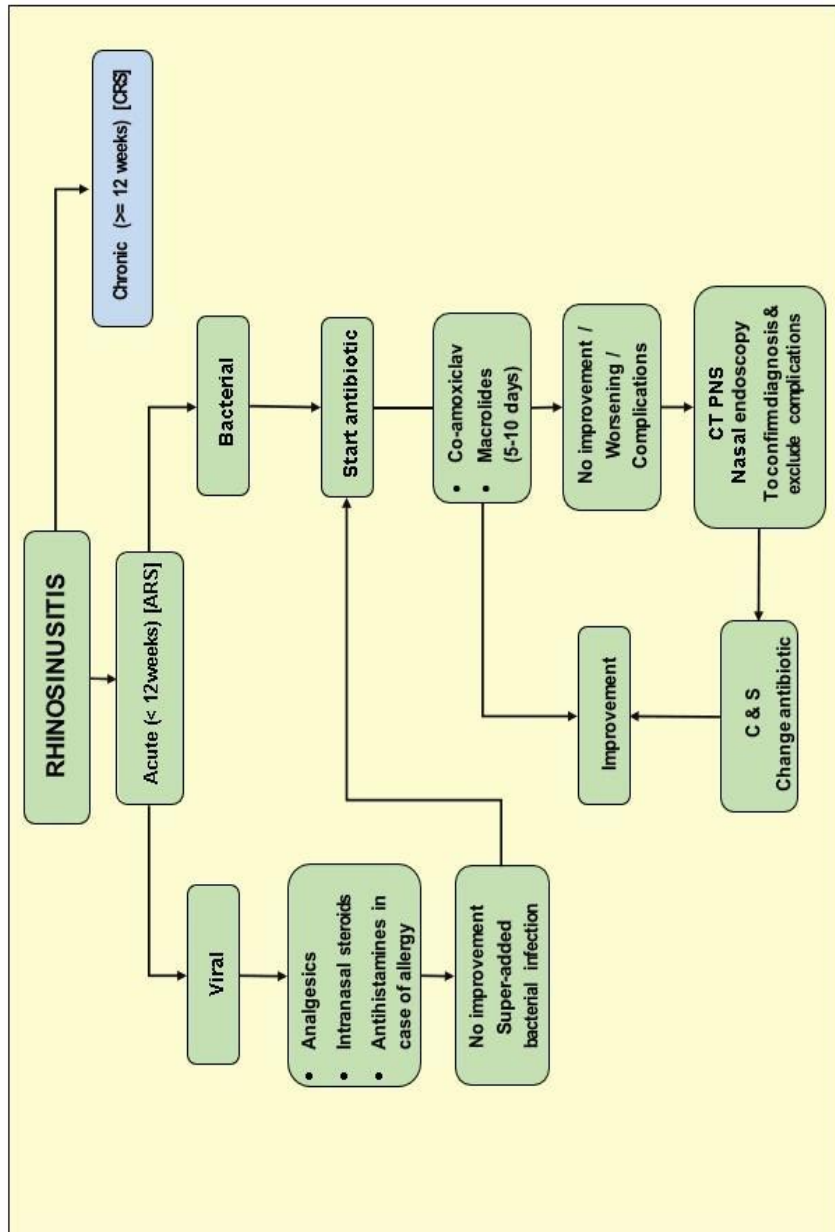
➤ **Minor criteria**

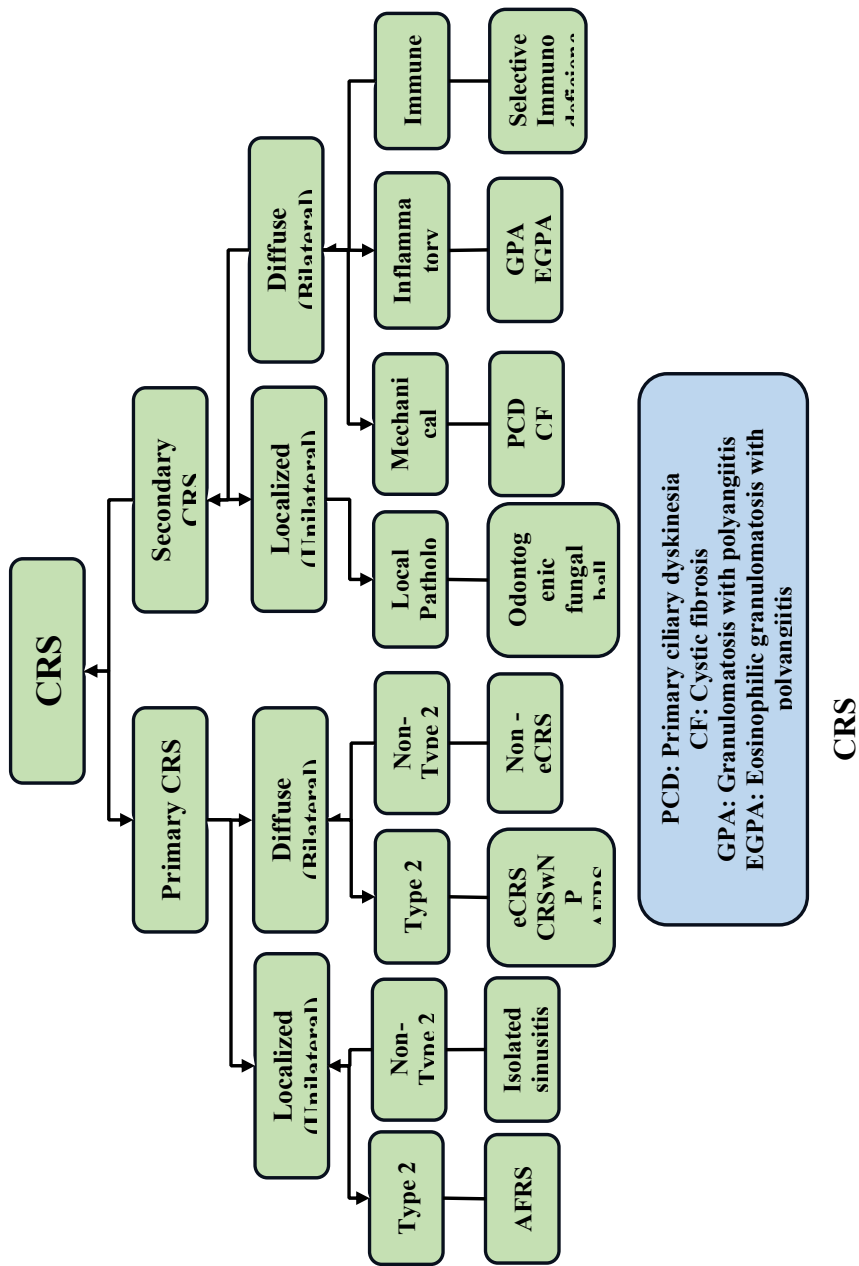
- Bone erosion
- Charcot Leyden crystals
- Unilateral disease
- Peripheral eosinophilia
- Positive fungal culture

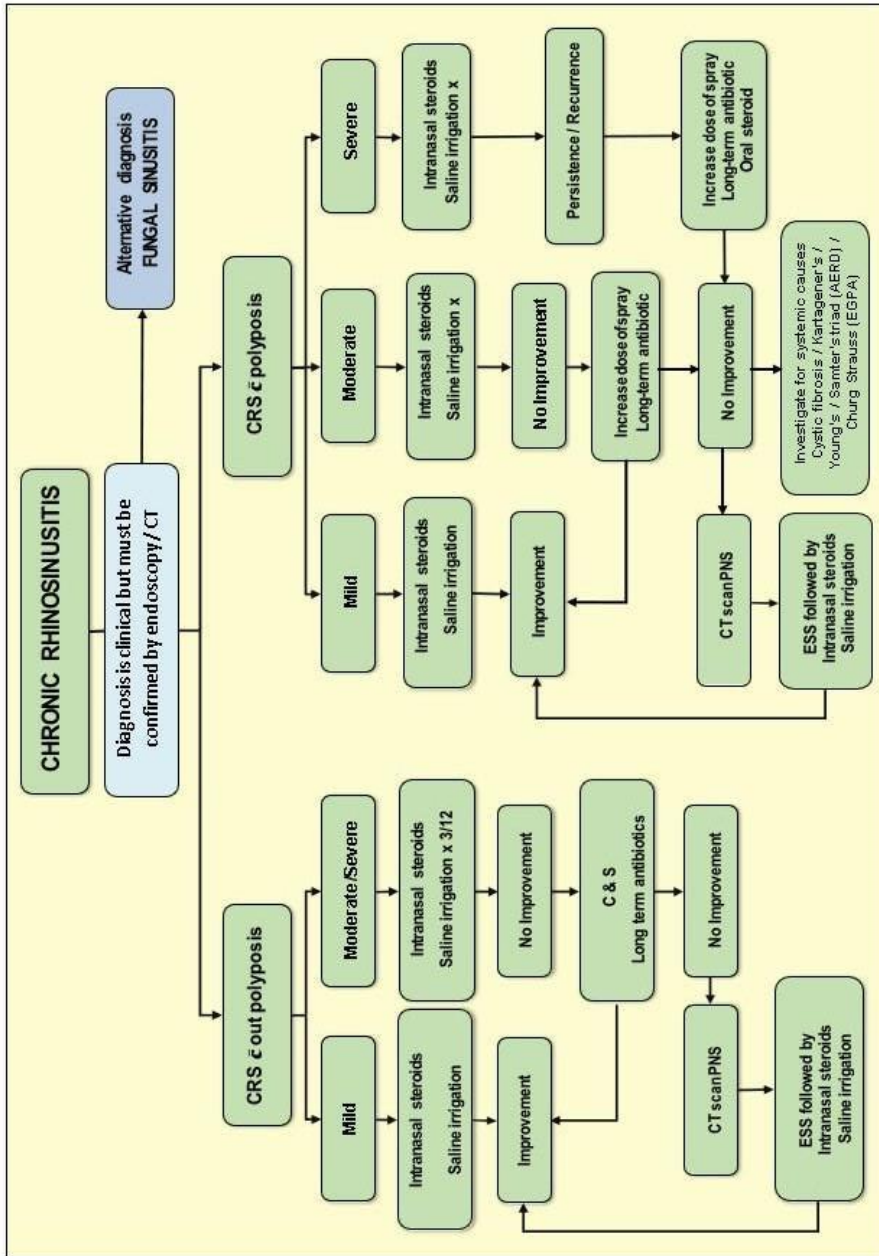
Treatment:

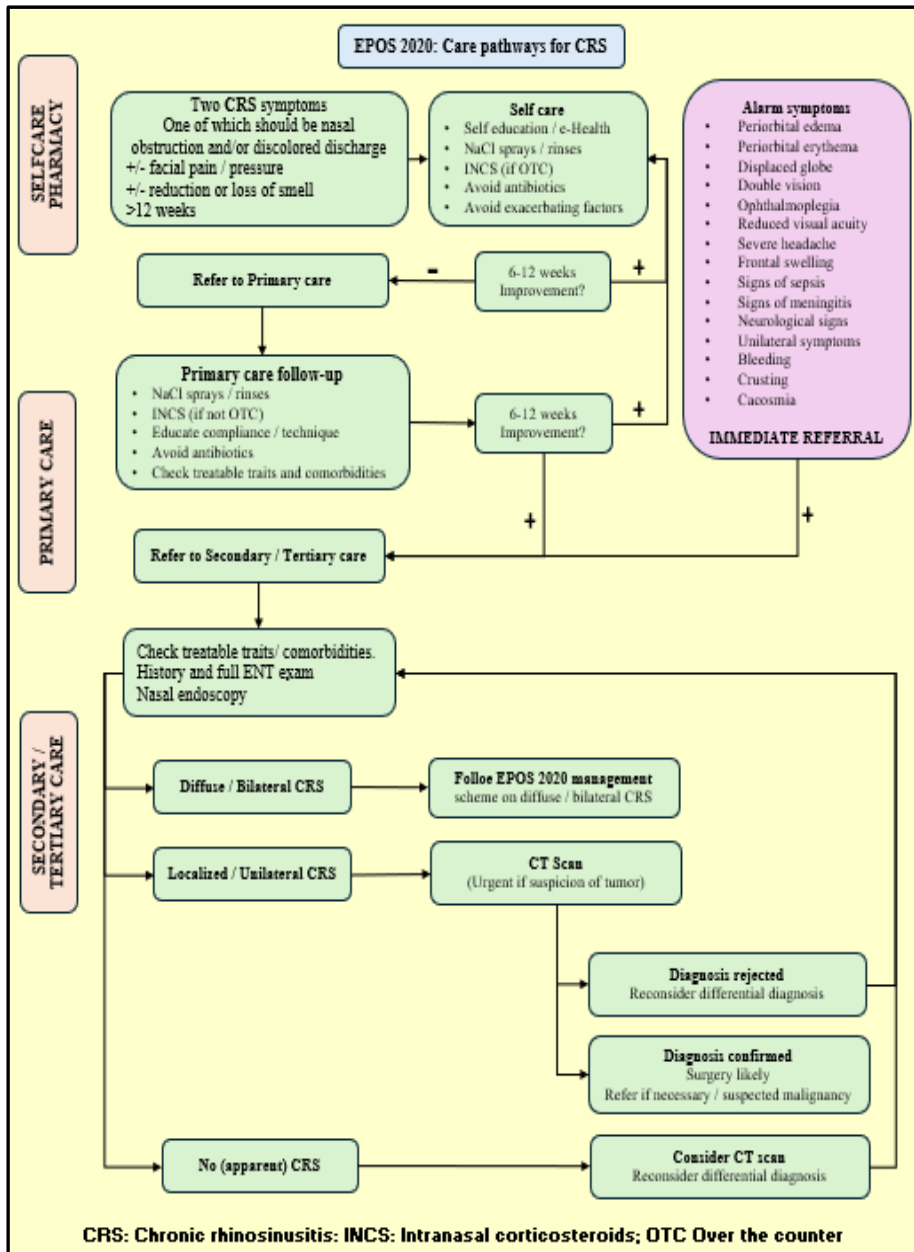
- Endoscopic excision of polypi and allergic mucin with preservation of normal mucosa to restore the ventilation and drainage of sinuses.
- This has to be combined with oral corticosteroids therapy which may be commenced a few days prior to surgery and continued for 2 weeks after. Intranasal corticosteroids (INCS) should be commenced post-operatively and continued long term.
- Patients may also benefit from immunotherapy and antihistamines.
- Antifungal therapy is usually not required as it is the reaction to the fungus that needs to be treated.
- However, in severe recalcitrant disease, some patients have benefited from a short course of itraconazole.

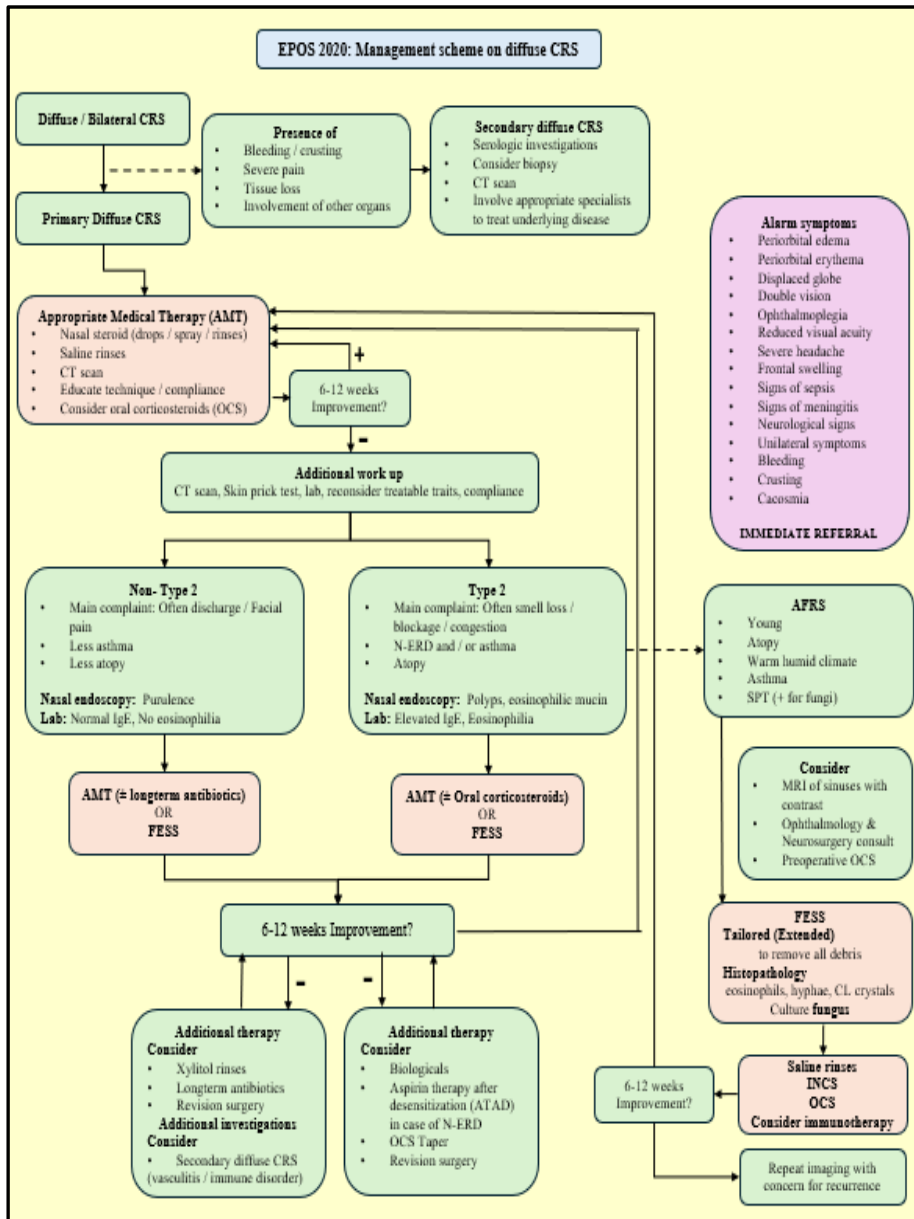
A Flowchart for management of sinusitis is given below









Care pathway for CRS⁹

Management scheme on diffuse CRS⁹

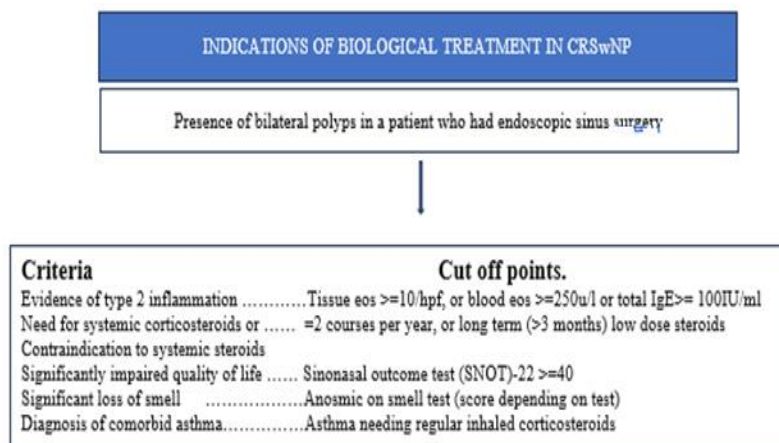


FIGURE -Indications for biological treatment in CRS

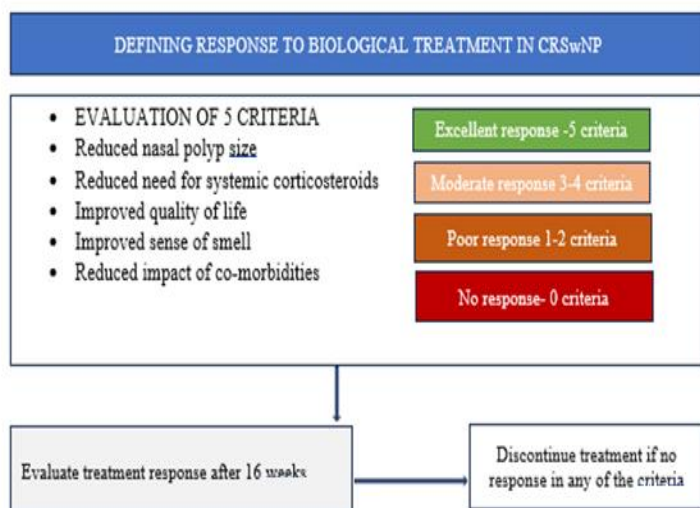


FIGURE: (Response criteria for biologicals in CRS)

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CSF RHINORRHEA

INTRODUCTION

Cerebrospinal fluid (CSF) rhinorrhoea results from a direct communication between the CSF-containing subarachnoid space and the mucosalized space of the paranasal sinuses. Because it may serve as a path for the spread of bacterial pathogens and other microorganisms, CSF rhinorrhoea may lead to meningitis and intra cranial infections, which carry significant morbidity even today.

Although CSF rhinorrhoea is a simple concept, its diagnosis and localization may be problematic; fortunately, contemporary strategies now provide a more direct means of diagnosis and localization.

KEY POINTS

- Cerebrospinal fluid rhinorrhoea may be classified as traumatic (>90%) or nontraumatic (<10%).
- Approximately 80% of all traumatic leaks occur in the setting of accidental trauma, and the remaining traumatic leaks occur after neurosurgical and rhinologic procedures.
- Nontraumatic aetiologies include neoplasms and hydrocephalus.
- Idiopathic nontraumatic cerebrospinal fluid rhinorrhoea has been linked with elevated intracranial pressure.
- Clinical presentation includes unilateral watery drainage with a characteristic metallic or salty taste, often in the clinical setting of possible etiologic factors.
- Differential diagnosis for watery rhinorrhoea includes allergic rhinitis, vasomotor rhinitis, and retained nasal saline irrigation fluid as well as cerebrospinal fluid otorrhea that is draining through the eustachian tube.

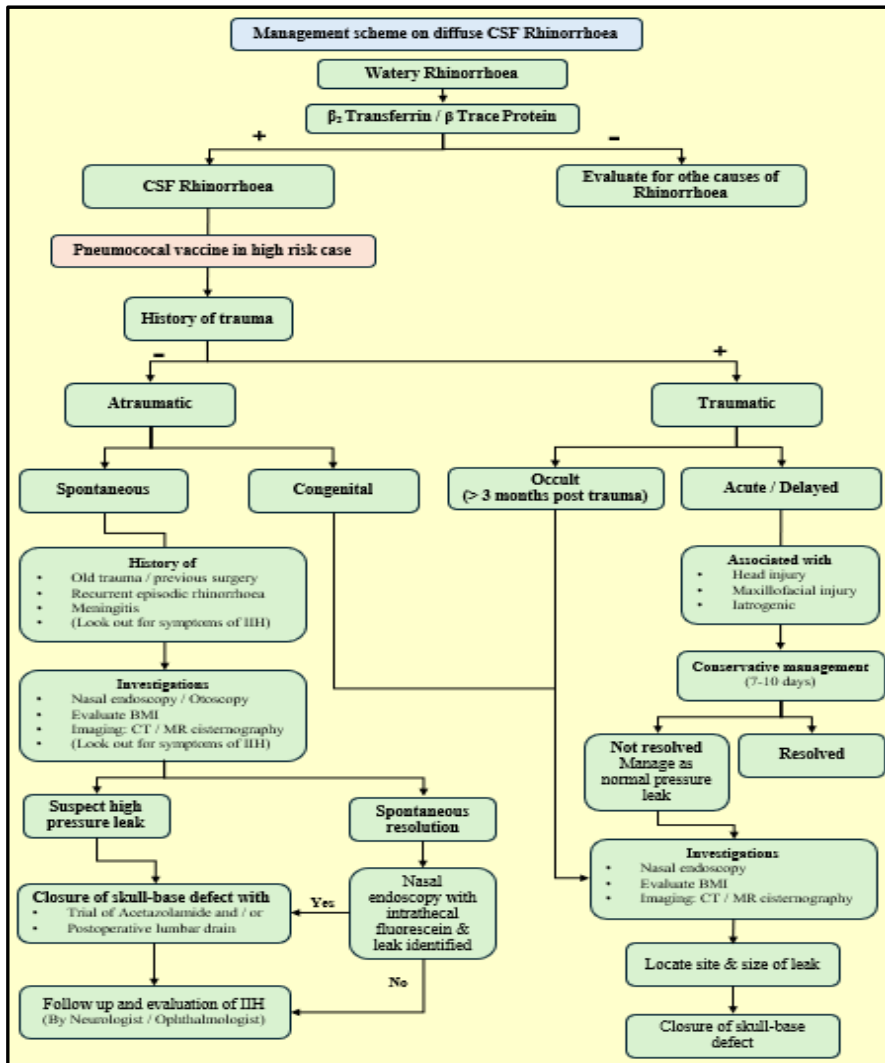
DIAGNOSIS

- Confirmation of a cerebrospinal fluid leak can be achieved through detection of β 2-transferrin or β -trace protein in nasal secretions which is not routinely done.
- Usually rely on clinical examination and localisation of the defect
- High-resolution CT (either 1-mm direct coronal images or coronal images reformatted from 1-mm axial data through a robust software algorithm) of the skull base may provide adequate anatomic information about the integrity of the skull base.
- MRI with contrast is also taken to rule out ICSOL and to look for features of idiopathic intracranial hypertension.

- Cisternogram studies provide diagnosis confirmation and localization information.
- Both CT cisternography and radionuclide cisternography require lumbar puncture for the administration of tracer agent, whereas MRI cisternography can be achieved solely through specific imaging protocols (heavily T2 weighted images)

MANAGEMENT

- Endoscopic repair has emerged as the preferred modality for most cases of cerebrospinal fluid rhinorrhoea requiring operative repair.
- During endoscopic repair, the leak site is identified and then closed with autogenous graft materials (fascia, fat), free mucosal graft, vascularized flaps more frequently nasoseptal flap based on sphenopalatine artery, allograft (acellular dermal allograft), and xenogeneic collagen dural substitutes, or a combination.
- The reconstruction is secured with surgical sealant and resorbable and non-resorbable packing material.
- The role of prophylactic antibiotics remains controversial. Certainly, in the presence of active infection adjacent to the leak site, antibiotics are prudent.
- Traumatic cerebrospinal fluid leaks are likely to resolve with conservative measures (lumbar drainage and bed rest); operative repair is reserved for those cases in which these measures fail or in which massive injury requires urgent formal operative exploration and repair.
- Cerebrospinal fluid leak that is recognized at the time of surgery should be repaired during that procedure.
- Cerebrospinal fluid rhinorrhoea that develops after surgery may be managed conservatively at first, but most cases require operative repair.
- Nontraumatic cerebrospinal fluid rhinorrhoea is unlikely to resolve spontaneously. After potential etiologic factors (brain tumour) have been excluded, operative repair is warranted.
- In case of recurrence, benign intracranial hypertension (BIH) is to be ruled out. Repair to be attempted only after addressing BIH.



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ALLERGIC RHINITIS

SCOPE

This treatment guideline provides clinicians with a basis for rational decisions in the management of allergic rhinitis. This guideline is applicable in any setting where patients with allergic rhinitis would be identified, monitored, or managed. It provides guidance for management of typical patients but does not consider all the unique individual circumstances.

KEY WORDS

Allergic rhinitis, treatment protocol

INTRODUCTION

Definition

Allergic rhinitis is defined clinically by the symptoms caused by immunologically mediated (most often IgE - dependent) inflammation after the exposure of the nasal mucous membranes to offending allergens.

Symptoms

- Bilateral watery rhinorrhoea, nasal obstruction or blockage, nasal itching, sneezing, and post-nasal drip that reverse spontaneously or after treatment. Ocular symptoms are also frequent.
- Allergic rhinoconjunctivitis is associated with itching and redness of eyes and tearing.
- Other symptoms include itching of palate and ears, post-nasal drip and cough
- Allergic rhinitis is a risk factor for asthma

CLASSIFICATION

Based on frequency of symptoms:

Intermittent – symptoms are present less than 4 days a week or for less than 4 consecutive weeks per episode

Persistent – symptoms are present ≥ 4 days a week and ≥ 4 weeks per episode

Which are again classified based on Severity (Sleep disturbance, impairment of daily activities, leisure and / or sport, impairment of school or work, troublesome symptom)

Mild – Normal sleep

Normal work and school

Normal daily activity

No disturbing symptoms

Moderate-severe – one or more of the above

Based on temporal pattern of exposure to a triggering allergen

Seasonal Perennial

Episodic (environmentally not normally encountered in patient's environment e.g. visiting a home with pets)

Based on these definitions, it is possible that a patient may have intermittent symptoms in perennial allergic rhinitis or persistent symptoms in seasonal allergic rhinitis

DIAGNOSIS:

- Characteristic symptoms: two or more of the following symptoms
 - o Anterior or posterior rhinorrhea
 - o Sneezing
 - o Nasal block and / or itching

Other symptoms include itchy eyes, pharyngeal itch, reduced smell, cough and sore throat. Clinical signs include, but not limited to clear rhinorrhoea, pale mucosa, pale to bluish turbinates, red and watery eyes
- External features like allergic shiners, allergic salute, dorsal nasal crease may be present
- Assess for the presence of associated conditions-asthma, atopic dermatitis, sleep disordered breathing, allergic conjunctivitis, sinusitis, OME
- Imaging paranasal sinuses is not required in patients presenting with symptoms consistent with allergic rhinitis.
- Specific IgE (skin and blood) tests may be done in patients who do not respond to empiric treatment, when the diagnosis is uncertain, or when specific causative allergen is to be identified to target therapy

TREATMENT:

Preventive measures

- Exclusive breastfeeding for at least three months may prevent atopic dermatitis, allergic rhinitis, and asthma
- Avoidance of allergen- The best and the most desirable when possible.
- It includes environmental control- removal of pets from indoors, use of air filtration

systems (HEPA-High Efficiency Particulate Arresting), impermeable bed covers, use of face masks, Acaricides (Chemical agents that kill dust mites)

- Avoid exposure to other irritants such as cigarette smoke, perfumes, chemicals and exhaust fumes

Mild intermittent

- With nasal congestion - intranasal corticosteroid (ICS) alone or oral antihistamine with decongestant (phenylephrine or pseudo-ephedrine) or local decongestant for not more than three days
- Relief with pharmacotherapy- maintain with minimum required dose of ICS
- If no relief after 2-4weeks - ICS + intranasal antihistamine (IAH) or ICS + 2nd generation oral antihistamine (OAH).
- Consider diagnostic nasal endoscopy/ radiologic evaluation - Turbinate reduction may be done if required
- With predominant sneezing, itching- II generation OAH / Cromolyn sodium spray (5.2mg/spray 1spray/nose/4-6 times/day or Hydroxy propyl methyl cellulose – 1spray per nostril 2-3 times/day
- Review after 4weeks - if relief, maintain with minimum dose ICS
- No relief in 2-4weeks - consider investigations – Skin prick tests/ID tests and serum levels of specific Ig by the fluorescence enzyme immunoassay
- Consider immunotherapy (Sub cutaneous or sublingual)

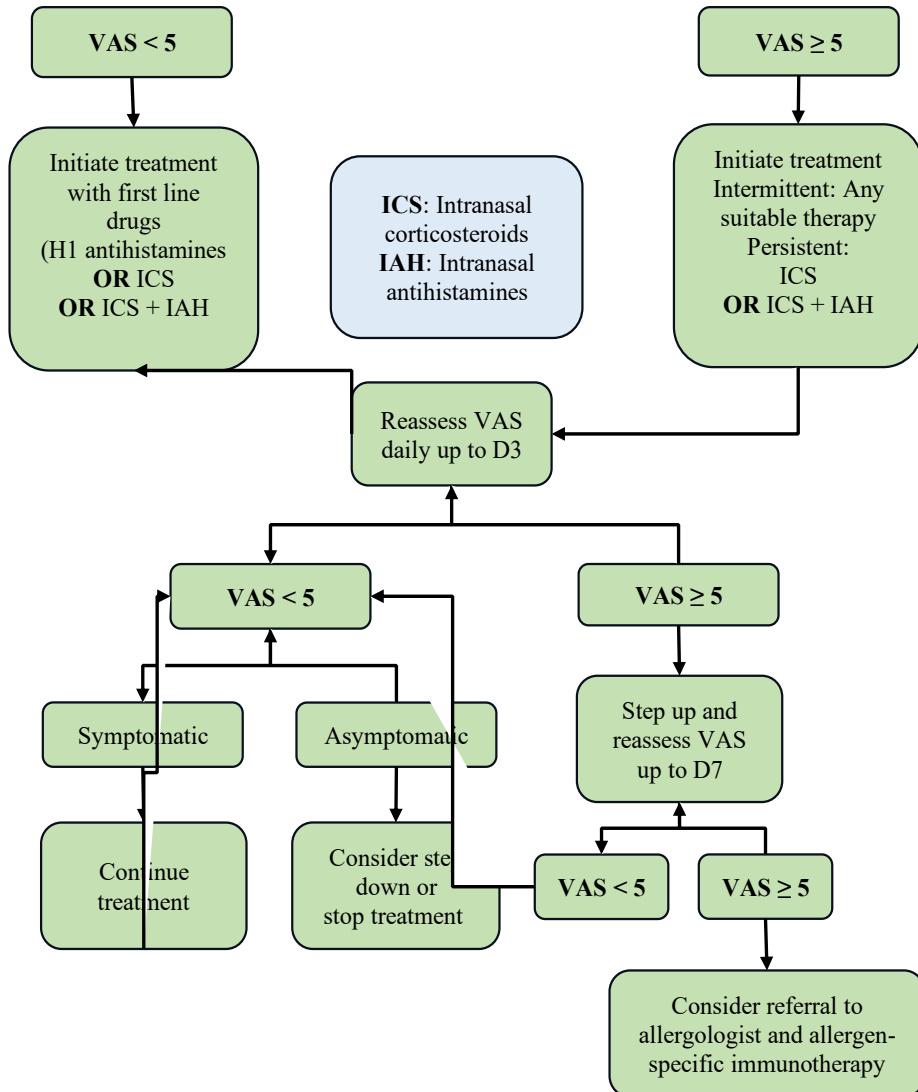
Moderate/severe/persistent symptoms

- ICS / IAH. If there is no relief, ICS may be combined with IAH
- Review after 4weeks- If relief, maintain with minimum dose ICS
- No relief in 2-4 weeks – A short course of oral steroid may be tried. Step up the dose of INC plus Leukotriene antagonists especially if associated with asthma
- Consider investigations – Skin prick tests/ID tests and Serum levels of specific Ig by the fluorescence enzyme immunoassay
- Consider immunotherapy (Sub-cutaneous or sub-lingual)
- Indications for allergen specific immunotherapy include poor symptom reduction despite adequate pharmacotherapy according to guidelines during the allergy season and /or change in natural allergic history
- If conjunctivitis is present, add oral antihistamine, intra-ocular H1 antihistamine,

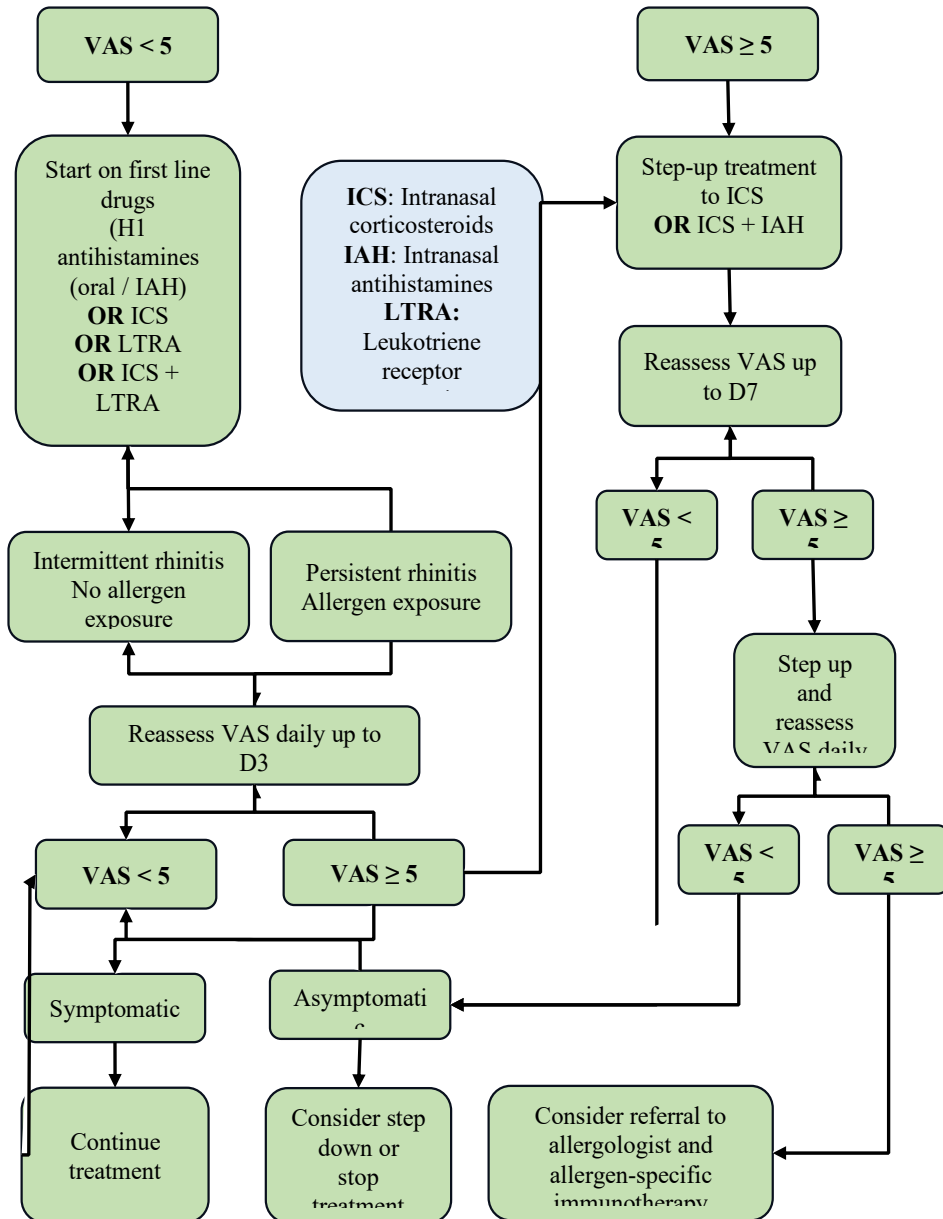
intra- ocular cromone

- If rhinorrhoea is present, add ipratropium nasal spray
- IgE inhibitor (omalizumab) may be used when standard therapies including immunotherapy fail.

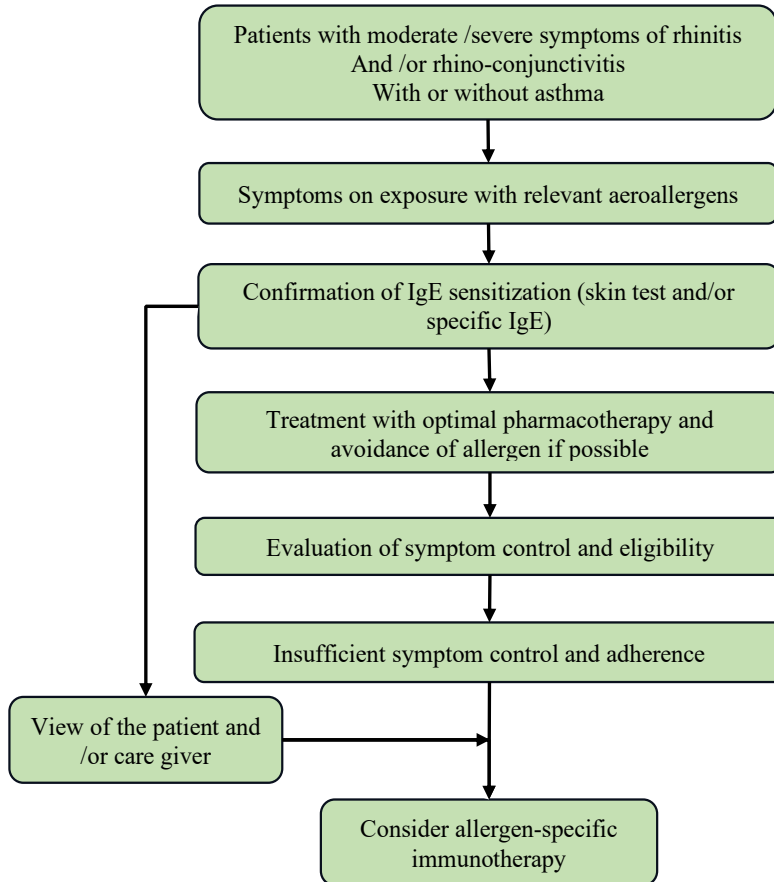
Step-up algorithm in untreated patients above 12 years with allergic rhinitis, based on Visual Analogue Scale



Step-up algorithm in treated patients above 12 years with allergic rhinitis, based on Visual Analogue Scale



Step by step approach to the indication for allergen-specific immunotherapy



Drug therapy of allergic rhinitis

Prophylactics

Cromolyn spray 5.2mg/spray
Dose:1 spray/nose/4-6 times
a day
HPMC powder 4mg/spray (Hydroxy
propyl methyl cellulose +
peppermint -

Oral decongestants

Pseudoephedrine ER 120mg BD

Children

Pseudoephedrine ER > 12 years
Pseudoephedrine > 4 years

Topical Nasal decongestants

Oxymetazoline not > 12 years

Oral antihistamines (II)

Cetirizine	10mg OD
Levocetirizine	5mg OD
Fexofenadine	120mg OD
Loratadine	10mg OD
Desloratadine	5mg OD

Children

Cetirizine	6 months
Desloratadine	6 months
Fexofenadine	6 months
Loratadine	2 years

Intra nasal

Azelastine	137 µg/spray	6
Azelastine	206 µg/spray	years
Olopatadine	665 µg/spray	6
Azelastine + Fluticasone	137 µg/spray + 50 µg/spray	years > 12 years

Dose 1-2 spray/nose/once or twice a day (on age)

Leukotriene

Montelukast 10mg

Children

Montelukast 6

Anti-cholinergic spray

Ipratropium bromide 42 µg/spray > 6 years

Dose: 2 sprays/nose three or four times a day

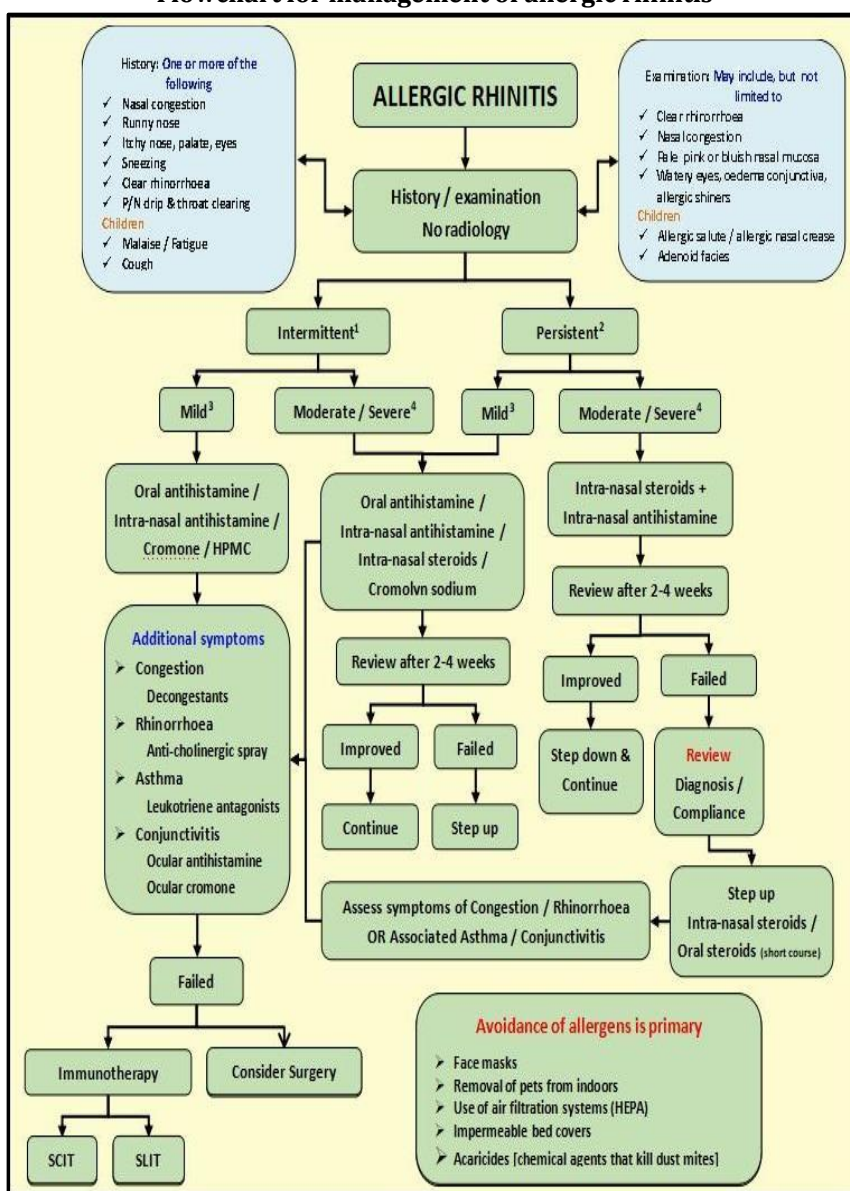
Budesonide (aqueous), Mometasone furoate & Fluticasone propionate

Dose 1-2 spray/nose/once or twice a

Intra nasal steroids		
✓ Triamcinolone acetone	55 µg/spray	> 2 years
✓ Mometasone furoate	50 µg/spray	> 2 years
✓ Fluticasone furoate	28 µg/spray	> 2 years
✓ Fluticasone propionate	50 µg/spray	> 4 years
✓ Budesonide (propellant)	32 µg/spray	> 6 years
✓ Flunisolide (aqueous)	25 µg/spray	> 6 years
✓ Ciclesonide (aqueous)	50	> 6

	µg/spray	years
✓ Ciclesonide (propellant)	37 µg/spray	> 12 years
Pregnancy		

Flowchart for management of allergic rhinitis



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INVERTED PAPILLOMA

INTRODUCTION

Sinonasal inverted papilloma (SNIP) is a benign epithelial tumor characterized by a locally aggressive growth pattern, the propensity to recur, and the potential for malignant transformation [1]. Krouse staging system is used to define the extent of SNIP at diagnosis. The estimated rate of malignant transformation in SNIP is 10% [1]. Squamous cell carcinoma (SCC) is the most common type, and it may present simultaneously with the primary SNIP tumor (synchronous), or after previous treatments for benign SNIP (metachronous). According to a review by Mirza et al. the rate of synchronous malignant transformation is 7.1% and metachronous 3.6% [1].

DIAGNOSIS

- Men are affected more than women with a 3–5:1 predominance, and often presents in the 5th–7th decades.
- The presentation can be similar to other sinonasal masses, with nasal obstruction, sinus pain, and epistaxis [2]. Endoscopy reveals an irregular multinodular pale, fleshy polypoid mass of variable consistency and with a tendency to bleed on touch.
- The definitive diagnosis is determined by biopsy, although in some cases false negatives can appear because these lesions may coincide with benign polyps [3].

MANAGEMENT

- CECT features are mostly non-specific, demonstrating a soft tissue density mass with some enhancement.
- Areas of focal hyperostosis on CT images correspond to the origin of tumour. This is useful not only in suggesting the diagnosis, but also to aid the surgical planning, as the location of tumour origin determines the extent of surgery required.
- MRI often demonstrates a distinctive appearance, referred to as convoluted cerebriform pattern, seen on both T2 and contrast-enhanced T1 weighted images [2] and the absence of this pattern and the presence of necrotic components are indicative of squamous cell carcinoma.

TREATMENT

The preferred method for treatment of SNIP is endoscopic surgery. The aim of the surgery should always be the maximum/complete eradication of the disease in the first attempt itself. The entire mass removed should be sent for Histopathological examination. Identifying and removing the tumor attachment site along with periosteum, followed by drilling or removing the underlying bone, may reduce the risk for recurrence.

Endoscopic sinus surgery, in particular Endoscopic Medial Maxillectomy, is currently the gold standard for treatment of maxillary sinus papilloma [5]. Follow up is critical to assess recurrence.

Post operatively patients should be followed up by endoscopy every three months for the first two years. After this period, patients can be followed up biannually for up to five years. MRI and/or biopsies should be performed when a recurrence is suspected [6]. The findings of a meta-analysis suggest that HPV positivity may contribute to SNIP recurrence. However, the variability in correlation among recently published studies highlights the need for more prospective research studies to establish a definitive association between HPV subtypes and SNIP recurrence[7]

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Section III

Diseases of throat

DYSPHAGIA

SCOPE

Dysphagia is an important problem faced in daily practice. The problems behind this can range from simple throat infection to conditions that may prove to be fatal. The guidelines outlined here will serve as a blueprint in evaluating cases of dysphagia with special emphasis on management of emergency situations.

DEFINITION

Dysphagia is defined as having difficulty in swallowing which may affect any part of the swallowing pathway from the mouth to stomach¹. The approach to which may be medical, surgical, prosthetic or a combination of these depending on the phase of swallowing and age group involved².

INTRODUCTION

Dysphagia is a common condition that can have a significant impact on the patient quality of life and can be associated with significant complications such as aspiration pneumonia, weight loss and malnutrition.

There are two types of dysphagia: oropharyngeal and oesophageal dysphagia, which are usually the result of structural or motility disorders. It can be caused by a number of disorders both benign and malignant and can be associated with multiple systemic disorders. It affects 1.6 -15% of the middle aged and 13 -35% of the elderly population³.

Though more than half of patients with dysphagia are seen in ENT clinics, it is an important problem faced by otorhinolaryngologists, general physicians and general surgeons alike. Our main aim lies in identifying the causes of dysphagia and stratifying those requiring emergency management.

CAUSES

OROPHARYNGEAL DYSPHAGIA⁴

Neurologic diseases

- Cerebrovascular accident
- Parkinson disease
- Multiple sclerosis
- Brain neoplasm
- Polio and post-polio syndrome
- Alzheimer disease
- Huntington disease

Myopathic diseases

- Myositis
- Dermatomyositis
- Myasthenia gravis
- Muscular dystrophies

Metabolic diseases

- Hyperthyroidism

Inflammatory/autoimmune diseases

- Amyloidosis
- Sarcoidosis
- Systemic lupus erythematosus

Infectious diseases

- Meningitis
- Diphtheria
- Botulism
- Lyme disease
- Syphilis
- Viral (coxsackie, herpes, cytomegalovirus)

Structural diseases

- Inflammatory (pharyngitis, abscess, tuberculosis)
- Congenital webs
- Plummer-Vinson syndrome
- Neoplasm
- Cricopharyngeal bar
- Zenker diverticulum
- Extrinsic compression (osteophytes, goiter, lymphadenopathy)
- Bullous skin diseases
- Poor dentition

Iatrogenic diseases

- Medication side effects (neuroleptics)
- Surgical resection
- Radiation-induced
- Corrosive (pill-injury, intentional)

OESOPHAGEAL DYSPHAGIA⁴

Neuromuscular disorders

- Achalasia
- Diffuse esophageal spasm
- Scleroderma
- Gastroesophageal reflux disease

Structural lesions (intrinsic)

- Benign peptic stricture
- Esophageal rings and webs
- Esophageal diverticula
- Foreign bodies
- Esophageal carcinoma
- Medication-induced strictures
- Eosinophilic esophagitis

Structural lesions (extrinsic)

- Vascular compression
- Mediastinal lesions
- Cervical osteoarthritis

CARE PATHWAY

Assessment of dysphagia

Primary care centres:

Primary care doctors face the challenge of dealing with a heterogeneous group of patients with dysphagia, without immediate access to the diagnostic facilities available in secondary care. As majority of patients with dysphagia will be in fluid deprived state, access to IV line and other supportive measures can be initiated here along with base line investigations as per the need.

Referral criteria: Dysphagia due to corrosive poisoning, dysphagia associated with stridor (foreign body obstruction/malignancy) or aspiration should be referred to higher centre immediately

Dysphagia cause not diagnosed with the investigation facilities available should also be referred after initiating the necessary primary measures.

Secondary care centres:

As the easily treatable dysphagia will be eliminated at the primary level, secondary care centres face the difficulty of categorizing those dysphagias that require emergency surgical intervention (e.g.: dysphagia with airway compromise) or those requiring advanced investigation facilities for identifying the cause. Another category of patients referred here is, cause identified dysphagia for rehabilitation options.

As the treatment of dysphagia may be spread over various specialties the referral and admission criteria here will depend on the specialties and the modalities available.

Referral criteria: Dysphagia requiring emergency surgical intervention where facilities are not available should be referred immediately

Dysphagia where rehabilitation facilities are not available should be referred after required investigations.

Tertiary care centres:

Tertiary care centres come face to face with a myriad of patients who are either under or over diagnosed in primary or secondary care centres. They also face the challenge of prioritizing the cases and triaging to required facilities.

HISTORY

- Duration and mode of onset
- Difficulty to swallow solids / liquids
- Cough on food intake (aspiration) - immediate / delayed
- Any change in voice
- Breathlessness / stridor
- Loss of weight. malnutrition, dehydration
- Preceding factors (corrosive ingestion, foreign body, trauma)
- Pertaining to age group (congenital, malignancy)
- Medication history

CLINICAL EVALUATION AT BEDSIDE

- ENT and systemic examination
- Watch the act of swallowing
 - Cough – Immediate / delayed
 - Time taken for swallowing
 - Position/Posture assumed by the patient
- Pulse oximetry for drop in oxygen saturation
- Auscultation of neck
 - High pitched swallowing sounds
 - Multiple swallow sounds

- Stridor – inspiratory / expiratory / biphasic
- Auscultation of chest
 - Crepitations / rhonchi

INSTRUMENTAL EVALUATION

- FEES (Flexible Endoscopic Evaluation of Swallowing)
 - Bolus of different consistency
 - Different positions
 - Therapy techniques
 - Laryngeal sensation
 - Cough efficiency
 - PAS Scale (Penetration Aspiration Score)
- VFS (Videofluoroscopy) or MBS (Modified Barium Swallow)
 - Barium of different consistency
 - AP and lateral views
 - Oral / pharyngeal / oesophageal stages
- TNE (Trans Nasal Esophagoscopy)
 - For assessment of oesophageal stage of swallowing
- Pharyngeal / oesophageal manometry
 - When motility disorders of pharynx & oesophagus is suspected
- Technetium scintigraphy
 - For detecting micro aspiration and planning the bolus speed in swallowing therapy

OTHER INVESTIGATIONS

- Routine investigations – Complete blood count, ESR, Coagulation profile, Blood sugar, Renal and liver function tests, Serum electrolytes, screening
- X-ray – soft tissue neck lateral view: obstruction, abscess, foreign body
- X-ray chest: Aspiration pneumonia
- Computed tomography

- High resolution intraluminal ultrasonography.

Criteria for admission:

All cases of absolute dysphagia

- Dysphagia with malnutrition
- Dysphagia with airway compromise
- Dysphagia due to infective causes (e.g. Diphtheria)
- Corrosive ingestion
- Dysphagia with foreign body
- Dysphagia with deep neck space infection

MANAGEMENT

The management includes

- **Prevention of aspiration pneumonia**
- **Maintenance of nutrition**
 - Enteral feeding
 - i. Nasogastric tube
 - ii. Percutaneous Endoscopic Gastrostomy (PEG)
 - iii. Feeding jejunostomy
 - Parenteral Feeding
 - iv. Total Parenteral Nutrition (TPN)
 - v. Partial Parenteral Nutrition (PPN)
- **Indirect swallowing therapy**
 - Exercises designed to strengthen the weakened muscles
 - o Lip incompetence – lip strengthening exercises like button & thread exercise & holding air in mouth
 - o Buccinator weakness – buccal puckering
 - Tongue dysfunction
 - o Tongue range of movements
 - Palatal dysfunction
 - o Balloon blowing / playing wind instruments

- Defective hyolaryngeal excursion
 - o Shaker exercise
 - o Chin Tuck Against resistance (CTAR)
 - o Neuro Muscular Electrical Stimulation (NMES)
- Laryngeal incompetence – Sensory / Motor
 - o Push pull technique
 - o Half swallow boom
- Thyropharyngeal paralysis / paresis
 - o Forcible ‘ee’
- Cricopharyngeal dysfunction
 - o Hot fomentation
- **Direct Swallowing Therapy**

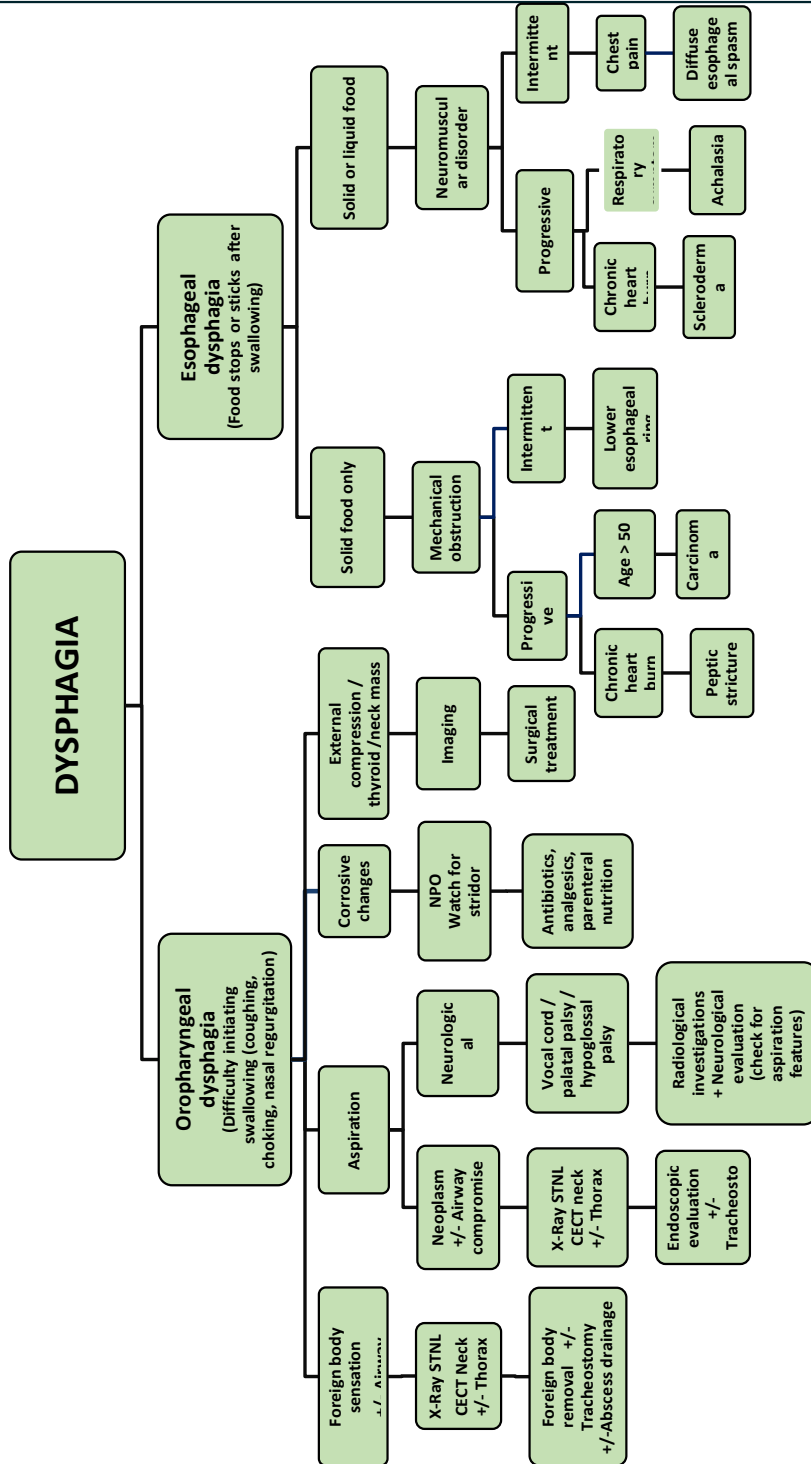
Modifying the consistency, timing of the feeds, temperature and techniques

 - ***Lip Incompetence:***
 - o Semisolid diet, feeding through the normal side
 - ***Buccinator incompetence:***
 - o Feeding through the normal side
 - ***Tongue dysfunction:***
 - o Posterior placement
 - o Jet feeding
 - o Liquid or thin semisolid diet
 - o Masako’s maneuver for Parkinsonism tongue dysfunction
 - ***Palatal dysfunction:***
 - o Nose pinch
 - o Chin tuck
 - o Semisolid and solid diet
 - ***Defective hyolaryngeal elevation:***
 - o Mendelsohn’s manoeuvre
 - o Chin tuck

- ***Laryngeal incompetence:***
 - o Supraglottic swallowing technique
 - o Super supra glottic swallowing technique
 - o Semisolid and solid diet
- ***Thyropharyngeal dysfunction:***
 - o Head turn to the paralyzed side
 - o Head tilt to the normal side
 - o Liquid and thin semi solid diet
- ***Cricopharyngeal dysfunction:***
 - o Large bolus
 - o Warm bolus
 - o Sour bolus
 - o Avoid thin liquids
- **Phagosurgery – Interventional treatments**
 - ***Lip incompetence:***
 - o Filler injections / commissuroplasty
 - o Masseter transfer
 - ***Tongue dysfunction***
 - o Cross nerve grafting
 - ***Palatal dysfunction***
 - o Palatopexy
 - ***Defective hyolaryngeal excursion***
 - o Hyoidopexy
 - ***Unilateral Laryngeal Incompetence***
 - o Injection laryngoplasty
 - o Medialization thyroplasty / arytenoid rotation
 - ***Bilateral Laryngeal Incompetence***
 - o Tracheostomy
 - o Glottic closure

- o Epiglottic over sew
- o Billing's operation
- o Laryngotracheal disjunction
- o Total laryngectomy
- ***Thyropharyngeal dysfunction***
 - o Hypopharyngoplasty (Plication of PFS)
- ***Cricopharyngeal dysfunction***
 - o Botulinum toxin injection to cricopharyngeus
 - o Crico pharyngeal myotomy / myectomy
- ***Oesophageal strictures***
 - o Balloon dilatation

NPO – Nil Per Oral; STNL – Soft Tissue Neck Lateral view; CECT – Contrast Enhanced Computerized Tomography



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DYSPHONIA

SCOPE

Dysphonia is often caused by benign or self-limited conditions, but it may also be the presenting symptom of a more serious or progressive condition requiring prompt diagnosis and management. The protocol, applicable to primary care centres and higher centres, aims to:

- Define actions that clinicians can take, to deliver quality care.
- Addresses the identification, diagnosis, treatment, and prevention of dysphonia
- Update the needs and management options in special populations and among patients with modifying factors.
- To enhance the accurate diagnosis of dysphonia and its underlying causes, promote appropriate therapeutic options and improve counselling and education for prevention and management of dysphonia.

DEFINITION

Altered vocal quality, pitch, loudness, or vocal effort that impairs communication as assessed by a clinician and/or affects quality of life¹.

Hence the management of dysphonia needs to combine both the diagnostic and therapeutic aspects.

INTRODUCTION:

Dysphonia (impaired voice production) is a very common complaint affecting nearly one-third of the population at some point in its life.

The term dysphonia is often used interchangeably with hoarseness; however, this terminology is imprecise, as hoarseness is a symptom of altered voice quality reported by patients, while dysphonia characterizes impaired voice production as recognized by a clinician. Dysphonia is often caused by benign or self-limited conditions, but it may also be the presenting symptom of a more serious or progressive condition requiring prompt diagnosis and management. Voice disorders affect all ages, but some evidence suggests that risks are higher in pediatric and elderly (>65 years of age) populations.

It is important to recognize that patients with head and neck cancer may present with dysphonia. In this group, failure to evaluate the larynx can delay cancer diagnosis. Therefore, the protocol stands to outline certain guidelines to ensure prompt diagnosis with minimal yet necessary investigations and proceed with either referral to a higher centre, if at the primary health care level or to decide on a management option.

CARE PATHWAY

Assessment of dysphonia:

Most patients (90%) with a complaint of hoarseness initially present to their primary care physicians. A major chunk of this can be treated effectively at the primary care center itself, even without immediate access to diagnostic facilities. Clinicians should identify dysphonia in a patient with altered voice quality, pitch, loudness, or vocal effort that impairs communication or reduces QOL.

HISTORY AND EXAMINATION

The history should include, but not be limited to, reviewing

- The duration of the dysphonia
- Type of onset (e.g., sudden, gradual)
- Progression (progressive /non-progressive)
- Diurnal variation
- Voice use details (profession, hours per day, days per week, amplification devices)
- Potential inciting events
- How the condition is affecting the patient
- Associated symptoms (dysphagia, dyspnoea, cough)
- Current medications, Known drug allergy,
- Habits and addictions (smoking, alcohol)
- Concurrent medical conditions (Hypertension, Diabetes, Dyslipidaemia, Thyroid disorders, Bronchial asthma, Nasal allergy, Hearing loss, Acid Peptic Disorder, Covid & Sequelae)
- Prior surgery

Careful clinical evaluation allows the clinician to categorize dysphonia severity, develop a treatment plan, and prioritize patients who may need escalated care

- Subjective evaluation of voice
 - Voice handicap index
 - GRBAS scale
- Objective evaluation of voice
 - Maximum phonation duration (MPD)

- Neck examination
 - Masses, cervical lymphadenopathy or thyroid swelling.
- Neck tenderness
 - Hyoid cornu
 - Thyrohyoid space
 - Cricothyroid space
 - Infrathyroid muscles
 - Mylohyoid
- Breathing type
 - Abdominal / Thoracic / Pectoral / Clavicular / Paradoxical
 - Forcing the voice present / absent
- Resonance
 - Throat resonance Optimal / Sub optimal
 - Oral resonance Optimal / Sub optimal
 - Nasal resonance Optimal / Sub optimal
- Nose
 - Deviated nasal septum / Patency
- Oral Cavity & Oropharynx
 - Tonsillar hypertrophy / Velopharyngeal incompetence

Investigations:

- Flexible laryngoscopy (Chip on tip is preferred) is used to assess for
 - Structural Lesions
 - o Nodule / Polyp / Cyst / Papilloma / Carcinoma / Sulcus / Web (congenital) or Synechia (acquired)
 - Neurological dysfunction
 - o Impaired vocal fold movements
 - o Unilateral / Bilateral
 - o Partial / Total

- o Phonatory Gap: Anterior two third / Vocal process / Body of arytenoid
- o Overriding arytenoid: Right / Left
- o Prolapsing of arytenoid: Right / Left
- o Ventricular band adduction: Ipsilateral / Contralateral / Bilateral
- o Antero posterior compression
- Stroboscopy
 - Mucosal waves - Normal / Diminished / Absent - Right / Left / Both
- Reflux Finding Score
 - Posterior laryngitis
 - Interarytenoid pachydermia
 - Shallow ventricles
 - Pseudosulcus
 - Thick mucus
 - Arytenoid granuloma
- Videolaryngoscopy
- Chest X-ray: to rule out pulmonary Tuberculosis or other granulomatous disease
- Imaging for suspected malignancy cases: CECT neck; MRI, only if needed, to ensure early cartilage involvement in malignancy².

Referral criteria: Dysphonia failing to resolve or improve within 4 weeks or irrespective of duration, or a serious underlying cause is suspected, then refer to secondary care with facilities to perform laryngoscopy and necessary biopsies to decide on further management.

MANAGEMENT GUIDELINES

A. Vocal Nodules

- Voice therapy is advised to
 - Improve vocal ability
 - Reduce vocal demand
 - Impart good vocal hygiene
- If refractory to therapy, endoscopic excision to be considered. Can be done on both sides at the same time, taking care not to create trauma at the anterior commissure.

B. Vocal Cord Polyp

- Endoscopic microlaryngeal excision – Cold knife / CO₂ laser
- Post operative period – Absolute Voice rest for two days, soft voicing for two weeks
- Pain killers and PPI post operatively
- Antibiotics not needed
- Voice therapy six weeks later to prevent recurrence

C. Vocal Cord Cyst

- Endoscopic microlaryngeal excision in toto with cold knife/CO₂ laser
- Lateral Micro flap technique
- Post operative care as described for vocal polyps

D. Recurrent Respiratory Papillomatosis

- Endoscopic excision as complete as possible
- Cold knife / Micro debrider / CO₂ laser
- In case of anterior commissure involvement, either a staged procedure (six weeks apart) or bilateral excision with keel at the anterior commissure. Keel to be removed after two weeks
- Adjuvant therapy needed when two or more surgeries are needed per year
- Cidofovir - Intralesional at 1mg/kg, 3 to 6 injections at four to six weeks interval
- Bevacizumab – Intralesional at 2 mg/kg or Intravenous at 10 mg/kg, 3 to 6 injections at four to six weeks interval

E. Sulcus Vocalis

- Voice therapy aimed at improving breath power and resonance
- Surgery – Endoscopic excision of the scar to correct the adynamic segment - cold knife / CO₂ Laser
- Paraglottic fat injection or medialisation thyroplasty (bilateral) to close the phonatory gap
- Post operative voice therapy

F. Unilateral Vocal Cord Palsy

- If asymptomatic – No treatment
- Symptomatic, when recovery is expected (within 6 – 8 months)

- Small phonatory gap – Voice therapy (Half swallow boom technique)
- Larger phonatory gap – Injection laryngoplasty (Hyaluronic Acid / Hydroxyl Apatite)
- Symptomatic when recovery is not expected (after 6 – 8 months), when the nerve is cut
 - Static surgical procedures
 - Anterior two third gap – Medialisation thyroplasty / Fat injection
 - Vocal process level gap – Arytenoid rotation
 - Body of arytenoid level gap – Adduction arytenoidopexy
 - Dynamic surgical procedures (especially in children and professional voice users)
 - o All adductors paralysis – Non-selective reinnervation
 - o Isolated muscle (adductors) paralysis – Neurotization of the muscle

G. Laryngeal Dystonia

• Adductor Spasmodic dysphonia

- Voice therapy (Falsetto phonation, Auditory feedback therapy)
- Botulinum toxin to both Thyroarytenoid muscles (2.5 to 3.75 units each) under laryngeal EMG / USS / Flexible Laryngoscopy / Direct laryngoscopy
- Surgical Procedures
 - o Type II Thyroplasty (reversible)
 - o Endoscopic Thyroarytenoid myoneurectomy (Endo TAM)
 - o Selective Laryngeal Adductor Denervation – Reinnervation (SLAD-R)

There are no clear-cut indications for these procedures at present, except for those cases not suitable for treatment with Botulinum toxin.

• Abductor Spasmodic Dysphonia

- Voice Therapy (Auditory Feedback Therapy)
- Botulinum toxin injection to PCA (one side at a time)
- Radio frequency assisted partial ablation of one PCA
- Selective denervation of one PCA

Indications for the latter two procedures are not well defined.

H. Functional voice disorders

• Muscle Tension Dysphonia (MTD)

- Type I MTD (Vocal nodules) - Voice therapy / Endoscopic excision in refractory cases
- Type II MTD (Ventricular band phonation) - Voice therapy / Botulinum toxin / Partial excision of ventricular bands
- Type III MTD (Antero-posterior compression) - Voice therapy

• Puberphonia

- Voice therapy
- Type III thyroplasty (Relaxing thyroplasty)

• Androphonia

- Voice therapy
- Wendler's glottoplasty (Anterior half glottic webbing) - length reduction
- Laser Assisted Vocal adjustment (LAVA) - Mass reduction
- Type IV thyroplasty (Cricothyroid approximation) - tensioning thyroplasty

Criteria for admission:

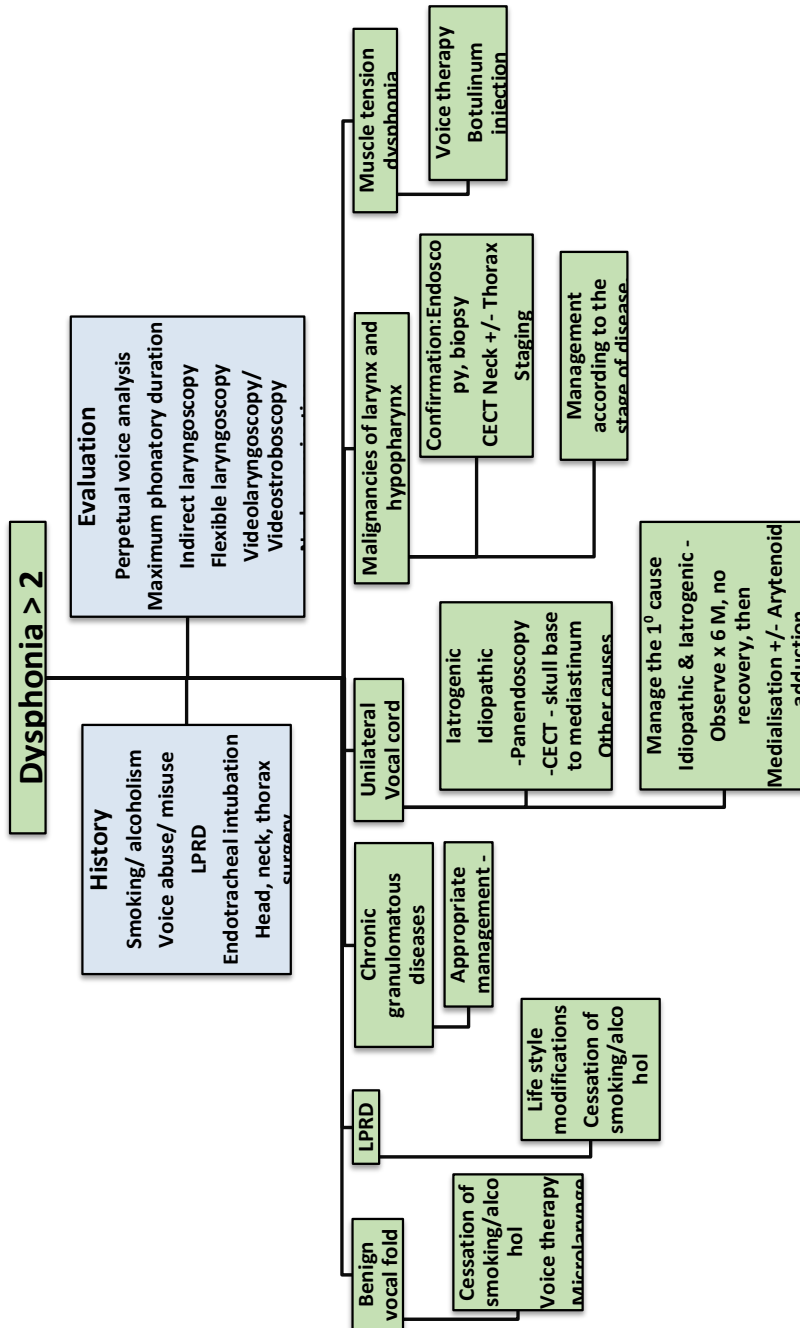
Most cases do not require admission and can be treated under OP care unless for the following:

- Cases requiring emergency airway management warranting a tracheostomy
- For therapeutic surgical intervention, e.g. MLS
- For diagnostic purpose: biopsy of suspected malignancy.

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ACUTE THROAT PAIN & ODYNOPHAGIA

SCOPE

Throat pain & odynophagia is a common problem encountered in the otorhinolaryngological practice. This symptom can be presented as an acute or persistent problem. It encompasses several key areas aimed at ensuring effective diagnosis and treatment and include causes, evaluation methods, and management strategies to ensure optimal patient care.

DEFINITION

Odynophagia is defined as pain during swallowing that can occur in the mouth, throat, or oesophagus. It is distinct from dysphagia, which refers to difficulty swallowing. It is almost always associated with throat pain and can vary in intensity and may be exacerbated by certain foods or liquids.

INTRODUCTION

Odynophagia, characterized by painful swallowing, can significantly impact a patient's quality of life. It may arise from various underlying conditions ranging from infections to structural abnormalities. An accurate diagnosis is crucial for effective treatment.

CAUSES

The causes of acute throat pain and odynophagia are diverse and can be categorized as follows:

Infectious Causes	Non-Infectious Causes	Other Causes
✓ Bacterial/Viral/Fungal - Tonsillitis - Pharyngitis - Epiglottitis - Supraglottitis - Esophagitis ✓ Peritonsillar abscess ✓ Deep neck space infection	✓ Laryngopharyngeal reflux disease (LPRD) ✓ Foreign body impaction +/- deep neck space infection ✓ Trauma or injury to the throat ✓ Neoplastic conditions ✓ Inflammatory conditions ✓ (e.g., esophagitis, laryngitis) ✓ Autoimmune conditions ✓ (Scleroderma, Polymyositis)	✓ Post-operative complications (e.g., following intubation) ✓ Systemic diseases affecting immunity (e.g., HIV/AIDS, Haematological malignancies)

CARE PATHWAY

Assessment of throat pain and odynophagia

Primary care centres: Primary care doctors need to encounter patients with throat pain and odynophagia in their day today practice. Majority of them specifically the infective pathology in tonsil, pharynx can be treated effectively at the primary care centre itself, even without immediate access to diagnostic facilities.

Referral criteria: Acute throat pain and odynophagia failing to resolve or improve within two weeks despite treatment or associated with breathing difficulty and dysphagia needs to be approached seriously and should be referred to a secondary care with facilities to perform endoscopic evaluation to find out the exact pathology and further management.

Secondary care centre:

History taking

- Onset and duration of symptoms
- Associated symptoms (fever, cough, difficulty breathing, change in voice, trismus, otalgia, drooling of saliva, swellings in neck)
- Recent medical history (infections, surgeries, trauma)

Physical examination

- Complete ENT examination focusing on oral cavity, oropharynx, larynx, hypopharynx and neck
- Assessment for signs of dehydration or nutritional compromise

Diagnostic tests

- Routine blood examination - Complete hemogram, Blood sugar, RFT, LFT, Serology
- Throat swab for culture and sensitivity if bacterial infection is suspected.
- Endoscopy for evaluating nasopharynx, oropharynx, larynx, hypopharynx and oesophagus and for impacted foreign bodies.
- Imaging studies if structural abnormalities are suspected.

Referral criteria: Acute throat pain and odynophagia requiring emergency surgical intervention where facilities are not available should be referred immediately.

Tertiary care centre

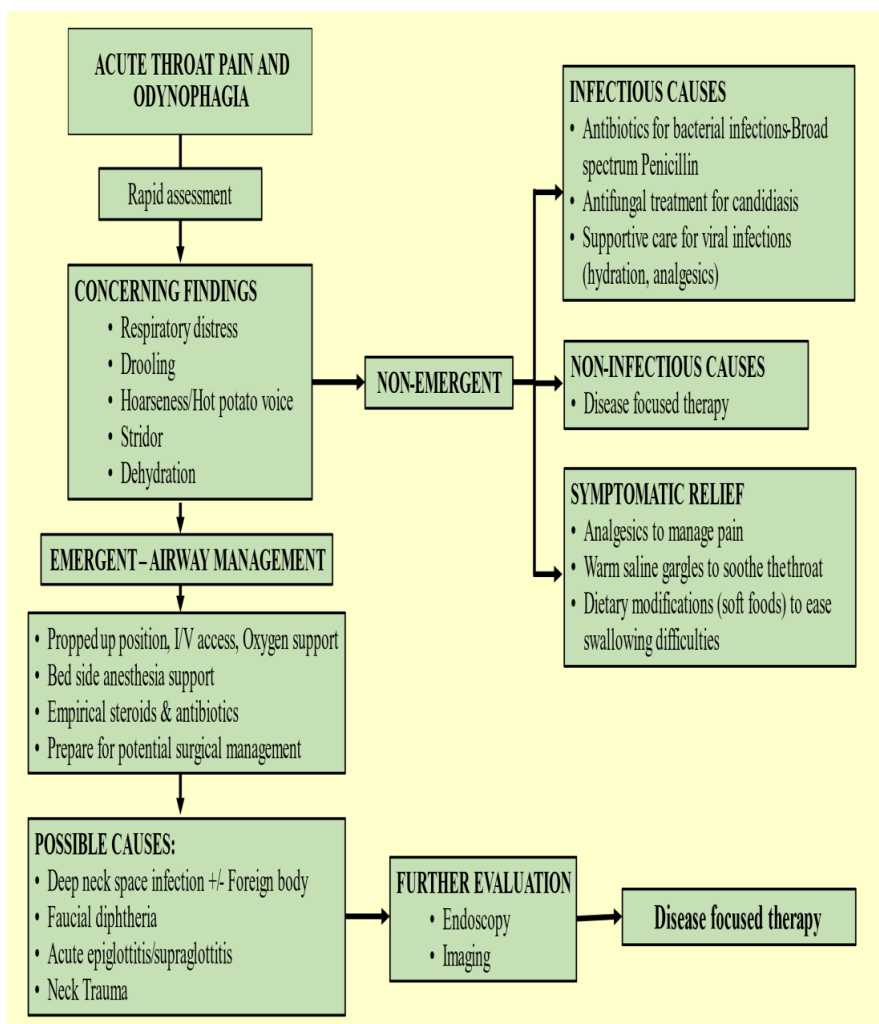
Foreign body impaction or peritonsillar abscess resulting deep neck infection with airway compromise needs timely airway management and addressal of the problem. In suspected

cases of faucial diphtheria also, a tertiary care referral may be needed in fear of airway problem and anticipated systemic complications.

Criteria for admission

Cases of acute throat pain and odynophagia with airway compromise, deep neck space infection, electrolyte imbalance needs inpatient admission for optimal management. Suspected cases of faucial diphtheria and infective throat diseases in the setting of immunocompromised conditions as haematological malignancies are to be managed with special consideration.

Flowchart on management of acute throat pain



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PERSISTENT THROAT PAIN

SCOPE

Persistent throat pain is a common scenario in the otorhinolaryngology practice creating a lot of diagnostic dilemmas. It encompasses a wide variety of complaints as scratchy or hot, burning sensation, lancinating pain, sort of 'raw' feeling, typical deep boring pain that worsens with mastication, swallowing and talking which often radiates to the ears and temporomandibular joints. It affects the quality of life and should not be dismissed as 'fictitious' and 'psychogenic'.

DEFINITION

There is lack of proper consensus regarding the optimum duration. According to literature – majority define it as a throat pain for more than three months despite medication.

INTRODUCTION

Persistent throat pain often seemingly a trivial symptom in real is a big hurdle to an otolaryngologist to deal with, necessitating a heavy demand for bringing relief. There is a serious dearth of an accepted, practical guideline, or a standardized and generalized algorithm on how to approach a patient presenting with persistent throat pain.

CAUSES

The multiplicity and heterogeneity of the spectrum of etiologies of persistent throat pain make the task of enumerating them far from exhaustive. Approximately 30% of patients with persistent throat pain have no identifiable cause. On review of literature and based on clinical experience, the most common causes include chronic tonsillopharyngitis, gastroesophageal reflux disorder (GERD), submandibular sialadenitis, laryngopharyngeal reflux disorder (LPRD), allergy / postnasal drip/chronic rhinitis, neuralgia / stylalgia and psychogenic causes. Miscellaneous includes oral submucous fibrosis, systemic illnesses (like diabetes mellitus, hypothyroidism, uremia, arthritis etc.), Koch's pneumonitis, recurrent aphthous ulcers, and dietary deficiencies (including anemia), temporomandibular joint disorders, oral / oropharyngeal candidiasis, concealed neoplasm / malignancy etc.

CARE PATHWAY:

Assessment of persistent throat pain.

Primary care centres

Primary care doctors need to encounter patients with persistent throat pain and many of the time, for the proper diagnosis and further evaluation, it often needs referral to a secondary care with facilities to perform endoscopic evaluation and imaging to find out the exact pathology and further management.

Secondary care centres

- Proper clinical history
 - Regarding the nature of pain throat, its duration and progression, presence of diurnal variation, exacerbating and relieving factors, localization and radiation, relation with mastication, deglutition, phonation, and respiration.
 - Information about food habits, addiction (including smoking, alcohol, betel nut chewing), voice abuse, heartburn etc.
 - Any systemic illness history including chronic low-grade fever, cough, haemoptysis, lymphadenopathy, recurrent exacerbation of chronic sore throat, local inflammatory disorders or ulcers (like recurrent aphthous), and associated significant ophthalmic, cutaneous, musculoskeletal and genitourinary symptoms are also to be enquired upon.
 - Personal and family history of metabolic and other systemic disorders like diabetes, hypothyroidism, anaemia of chronic disease, collagen vascular disorder etc. should be asked. Other important history to be elicited is regarding the drug history like psychiatric medications, anti-tubercular medications, antiretroviral therapy, DMARD etc.

One of the most important causes for persistent throat pain is laryngopharyngeal reflux disorder which can be diagnosed by the Reflux Symptom Index (RSI), which is given below. RSI more than 13 is considered abnormal.

<i>The Reflux Symptom Index (RSI)</i>						
Within the last month, how did the following problems affect you? <i>Circle the appropriate response.</i>	0 = No Problem 5 = Severe Problem					
1. Hoarseness or a problem with your voice	0	1	2	3	4	5
2. Clearing your throat	0	1	2	3	4	5
3. Excess throat mucus or postnasal drip	0	1	2	3	4	5
4. Difficulty swallowing food, liquids, or pills	0	1	2	3	4	5
5. Coughing after you ate or after lying down	0	1	2	3	4	5
6. Breathing difficulties or choking episodes	0	1	2	3	4	5
7. Troublesome or annoying cough	0	1	2	3	4	5
8. Sensations of something sticking in your throat or a lump in your throat	0	1	2	3	4	5
9. Heartburn, chest pain, indigestion, or stomach acid coming up	0	1	2	3	4	5
TOTAL						

- Complete ENT & Head and Neck Examination.
- Relevant evaluation tools

- Routine blood examination - complete hemogram, blood sugar, RFT, LFT, Serology
- Biochemical, microbiological, cytological tests
- Endoscopy – Flexible nasopharyngolaryngoscopy will help to find out the reflux finding score (RFS) which is given below. RFS can range from 0 to 26, a score of 6 is considered normal but when it is more than 7, diagnostic of LPRD.

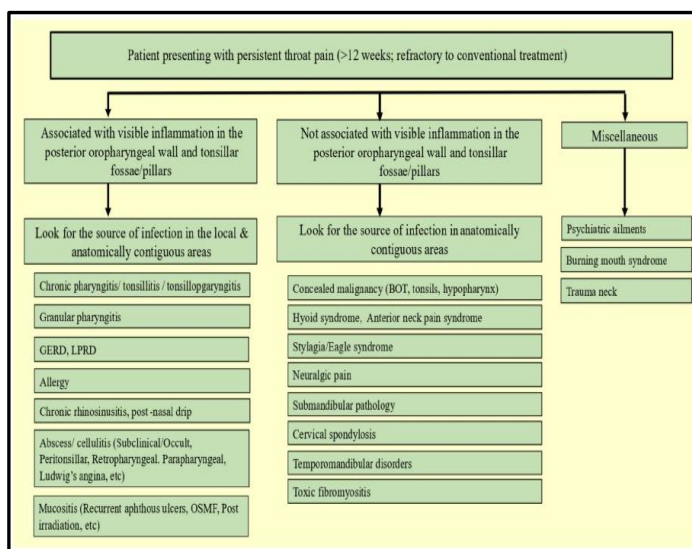
Reflux Finding Score (RFS).	
Subglottic edema	0 = absent 2 = present
Ventricular	2 = partial 4 = complete
Erythema/hyperemia	2 = arytenoids only 4 = diffuse
Vocal fold edema	1 = mild 2 = moderate 3 = severe 4 = polypoid
Diffuse laryngeal edema	1 = mild 2 = moderate 3 = severe 4 = obstructing
Posterior commissure hypertrophy	1 = mild 2 = moderate 3 = severe 4 = obstructing
Granuloma/granulation tissue	0 = absent 2 = present
Thick endolaryngeal mucus	0 = absent 2 = present

- Imaging: Proper imaging like intraoral occlusal view, reverse Towne's view, skiagram of soft tissue neck and cervical spine, barium swallow, computed tomography (CT) scan and/or magnetic resonance imaging (MRI) may be undertaken as required.

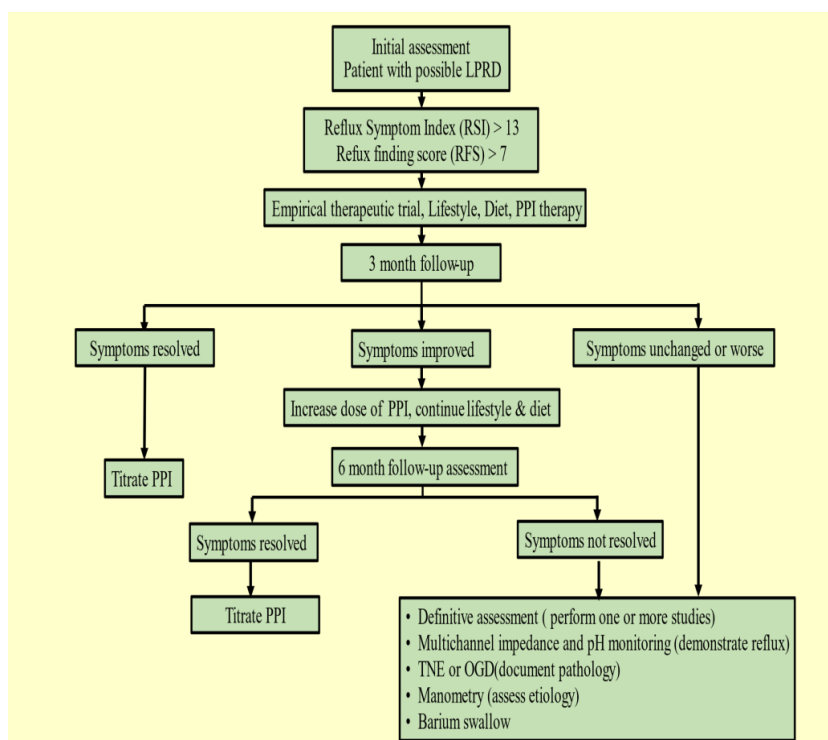
Tertiary care centres

After exact identification of the cause, like as in concealed malignancy, definitive treatment may be needed from a tertiary care centre. Likewise, etiologies necessitating definitive surgical intervention are to be referred to a centre with tertiary care facilities.

Management flow chart of persistent throat pain is given as follows:



Management flow chart of LPRD



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AIRWAY TRACT STENOSIS

SCOPE:

Airway tract stenosis is not uncommon these days. With the increase in road traffic accidents and long term ventilatory support for various causes, the incidence is ever increasing. Added to these are the congenital varieties. This guideline aims to discuss the management options of various common airway stenotic problems.

GLOTTIC WEB

- **If there is no airway obstruction, defer the surgery till the age of three**
- **Thin webs – Endoscopic excision plus keel**
- **Thick webs – Laryngofissure approach**
- **If associated** with subglottic stenosis, anterior cricoid split and costal cartilage grafting to be combined with the above procedure

LARYNGOMALACIA (CONGENITAL LARYNGEAL STRIDOR)

- **Indications for surgery**
 - Failure to thrive
 - Recurring cyanotic episodes
- **Surgical procedure**
 - Type I (Redundant mucosa) – Aryepiglottoplasty
 - Type II (Short aryepiglottic folds) – Aryepiglottoplasty
 - Type III (Floppy epiglottis) – Epiglottopexy

LARYNGEAL CLEFTS

- Type I & II clefts – Endoscopic two-layer repair
- Type III & IV clefts – Open repair (two / three-layer tibial periosteum)

POSTERIOR GLOTTIC STENOSIS

- Type I – Endoscopic division
- Types II – IV – Posterior cricoid split and costal cartilage grafting

BILATERAL VOCAL CORD PALSY

- When recovery expected (6 – 8 months)
 - Tracheostomy / suture cordopexy

When recovery not expected (more than 6 – 8 months / when both RLN are cut) –

- Kashima's posterior cordotomy (unilateral / bilateral)
- Endoscopic arytenoidectomy
- Woodman's operation
- Total selective reinnervation (dynamic procedure)
- PCA pacemaker (dynamic procedure – under evaluation)

SUBGLOTTIC STENOSIS

- **Soft stenosis**
 - Laser / cobaltor cut + balloon dilatation
- **Cartilaginous stenosis**
 - Grade I & II: Expansion laryngoplasty (anterior cricoid split + costal cartilage graft with or without posterior cricoid split + costal cartilage graft)
 - Grade III & IV – Resection and anastomosis (partial cricotracheal resection + thyrotracheal anastomosis)

GLOTTO-SUBGLOTTIC STENOSIS

- Extended partial cricotracheal resection (PCTR + Posterior cricoid split and costal cartilage graft)

TRACHEAL STENOSIS

- **Soft stenosis**
 - Laser / Coblator cuts + balloon dilatation
- **Cartilaginous stenosis**
 - Tracheal resection + anastomosis
- **Congenital complete tracheal rings**
 - Slide tracheoplasty

Section IV

Diseases of salivary glands

DISEASES OF SALIVARY GLANDS

SCOPE:

To provide evidence-based recommendations for practicing physicians and health care providers for the management of common salivary gland disorders. It aims to provide appropriate treatment, prevent complications and prevention of spread of infection among population. The guideline is applicable to any setting where a sudden salivary gland swelling presents, however, it does not extend to treat individual causes of the same. The guidelines do not include chronic conditions of salivary glands and neoplasms.

INTRODUCTION:

The parotid, submandibular, and sublingual glands are the three major pairs of salivary glands in the human body. In addition to this, numerous minor salivary glands are present in the mucosa of the oral cavity and pharynx. Over a twenty-four-hour period, 1 to 1.5 litres of saliva is secreted. The saliva is essential for taste, swallowing, speech, and maintenance of oral immunity through lysozymes and immunoglobulins. The inflammatory conditions of the salivary glands could be due to infective or inflammatory causes. The most common infective conditions are acute and chronic sialadenitis. These occur due to obstruction or other granulomatous conditions (Figure- 1).

Aetiology:

- Viral
- Bacterial (*Staphylococcus aureus*, streptococci, coliforms)
- Juvenile recurrent parotitis
- Radiation/radio iodine therapy

Predisposing factors:

- Patients in their fifties and sixties
- Chronically ill patients with xerostomia
- Patients with Sjögren syndrome and other autoimmune conditions
- Adolescents and young adults with anorexia
- Immunocompromised

INFLAMMATORY CONDITIONS OF THE SALIVARY GLANDS

Acute Viral Parotitis (Mumps)

Viral parotitis or mumps is an acute, self-limiting infection of the salivary glands, most commonly of the parotid. It is the commonest cause of salivary gland infection in children and is usually caused by paramyxovirus. There is an incubation period of two to three

weeks followed by a period of viremia when it localizes in the parotid gland, germinal tissues, and central nervous system. It spreads by droplet infection.

Conditions	Aetiology	Clinical features
Viral sialadenitis	Mumps virus	Bilateral swelling, tenderness
Suppurative sialadenitis	Bacterial	Single gland swelling and tenderness
Chronic sialadenitis	Obstruction – calculus, stenosis	Recurrent episodes
Neoplasm	Benign, malignant	Painless mass

Table 1: showing the common salivary gland disorders

Clinical Features

The child will have fever, malaise, headache, trismus, and dysphagia. It may start from a single parotid gland and later may involve the other major salivary glands. The patient is infective three days before and four days after the onset of parotitis. After the prodromal period, one or both parotid glands will enlarge. In 80% of children, there is bilateral parotid swelling. It resolves spontaneously in about a week. The swelling is associated with pain over the parotid due to stretching of the parotid fascia. There may be referred pain to the ear. The opening of the duct is inflamed, but there may be no pus emanating from it. The blood picture may show leukocytopenia, and the serum amylase may be raised.

Complications

Orchitis can develop in 20 to 30% of males with mumps. This is the most common complication in adult males. Usually, it appears during the first week of parotitis and typically affects one testicle. Oophoritis can occur in 5% of females. It can cause sterility. Aseptic meningitis or meningoencephalitis may occur in 10% of cases. Indications of the central nervous system (CNS) involvement include headache, fever, nausea, and vomiting. Pancreatitis can occur in 5% of cases. Sensorineural hearing loss has been reported in 0.5 to 4% of cases. The sensorineural hearing loss is of rapid onset, unilateral, profound, and usually permanent. Tinnitus, ataxia, and vomiting precede hearing loss. Deep neck space infections with difficulty in breathing and swallowing may occur.

Treatment

Supportive measures with analgesics like acetaminophen or ibuprofen may be done. Adequate oral hydration, sialagogues and topical application of ichthammol glycerine are

advised. Antibiotics and antivirals are not required generally. Oral hygiene should be maintained.

Prevention

- Immunisation: Routine administration of MMR/MR vaccine is advisable in view of the recent surge of mumps in Kerala. The Indian Academy of Paediatrics (IAP) recommends first dose of MMR at 9 months, second dose at 15 months, and a third dose at 4 to 6 years.
- Oral hygiene
- Hydration
- Avoid smoking /tobacco

Acute bacterial sialadenitis

Acute bacterial infections have a similar clinical presentation to viral sialadenitis. The parotid gland is commonly involved. Multiple glands are rarely involved. Staphylococcus aureus is the most common organism. The Haemophilus influenzae, Pneumococcus, and gram-negative organisms can also cause parotitis. Infection may reach the gland due to retrograde transmission of disease from oral cavity or due to stasis of salivary flow due to obstruction. Elderly patients

on medications like diuretics, antidepressants, beta-blockers, anticholinergics, and antihistamines, which reduce the salivary flow, are also at high risk. Calculus(sialolithiasis) may also present as acute sialadenitis.

Clinical features

The clinical presentation is with pain and swelling of the gland. Chewing becomes painful. On gentle pressure over the gland, pus may be seen extruding from the duct opening, which is usually inflamed. In parotitis, pus may be seen in the ipsilateral external auditory canal due to the communication via the fissures of Santorini.

Treatment

Antibiotics are started by the intravenous or oral route, depending on the severity. Depending on the virulence of the organism and the immune status of the patient, sialadenitis may progress to abscess formation. This may lead to sepsis and multi-organ dysfunction in debilitated patients.

Symptoms and signs of sialadenitis

Fever, chills, and unilateral pain and swelling develop in patients with sialadenitis. The salivary gland is firm and diffusely tender, with erythema and edema of the overlying skin. Pus can often be expressed from the duct by compressing the affected gland and should be cultured. Focal enlargement may indicate an abscess.

Diagnosis of sialadenitis

Ultrasonography usually suffices. Contrast enhanced CT or MRI can detect deep seated abscess that is not obvious clinically, although MRI may miss an obstructing stone. If pus can be expressed from the duct of the affected gland, it is sent for Gram stain and culture.

Treatment of sialadenitis

- Antistaphylococcal antibiotics
- Local measures (e.g., hydration, sialagogues, warm compresses, gland massage)
- Initial treatment for sialadenitis is with antibiotics active against *S. aureus* (e.g., dicloxacillin, a first-generation cephalosporin, or clindamycin), modified according to culture results. With the increasing prevalence of Methicillin-Resistant *Staphylococcus aureus* (MRSA), Linezolid or Vancomycin may be required in debilitated patients. Chlorhexidine 0.12% mouth rinses three times a day will reduce bacterial burden in the oral cavity and will promote oral hygiene.
- Abscesses require incision and drainage. If an obstructive pathology like sialolithiasis is identified, sialodochotomy or sialoendoscopy and calculus removal are advisable, once the inflammation settles. Salivary glands should be preserved though, a sialadenectomy may be needed for patients with chronic or relapsing sialadenitis.

When to refer? / RED FLAGS

- High grade fever not responding to supportive care
- Abscess formation/ Neck space infection
- Septic thrombophlebitis
- Respiratory obstruction
- Complications like orchitis, hearing loss

Juvenile recurrent parotitis

It is the second most common cause of parotid swelling in children after mumps. It is characterized by recurrent painful unilateral or bilateral parotid swelling usually associated with non-obstructive sialectasis of the parotid gland. The cause of recurrent parotitis of childhood is unknown although affected individuals are otherwise well. The usual age of onset is 3–6 years. The number of attacks varies from 1 to 5 per year. The frequency of recurrence peaks between 5 and 7 years, and up to 90% of patients have resolution of disease by puberty. Analgesics suffice and antibiotics are not useful. Lavage of the affected gland with saline and steroids through sialoendoscopy have shown promising results.

Recurrent parotitis

Further workup for autoimmune (Sjogren's, IgG4 glandular disease) and granulomatous diseases (Tuberculosis, sarcoidosis) may be needed if the problem persists or recurs (Fig 2).

Bilateral parotitis

Mumps, HIV, Hepatitis (HCV), Sjogren's, sarcoidosis, sialosis, pneumoparotitis, radioiodine parotitis and some drugs may cause bilateral glandular enlargement.

Sialolithiasis

Calculi may present as acute sialadenitis. It is more common in submandibular glands (83%), followed by parotid (20%) and minor salivary glands (7%). Postprandial swelling of the affected salivary gland may be present. Calculi may be palpable.

- **Investigations**

- Ultrasound scanning, CT scan or Cone beam CT scan. Sialoendoscopy where available may also be useful.

- **Treatment:** When small and accessible, manual expression of a stone from the submandibular duct is often feasible, and many can also be removed surgically (sialodochotomy) from distal submandibular duct, but this carries a risk of stricture formation. Minimally invasive procedures such as sialoendoscopy with or without transoral or trans facial approaches have been reported to be highly effective for the removal of sialoliths. Surgical removal of an affected gland may, however, be the only effective treatment if above measures fail.

Salivary gland swelling:

- Treatment of the painless swelling of the salivary gland is beyond the scope of this guideline. Evaluation includes Fine Needle Aspiration Cytology (FNAC), Imaging (USG, CT, MRI) and appropriate treatment given (Fig 2).

Prognosis:

- Guarded in recurrent parotitis and autoimmune parotitis.

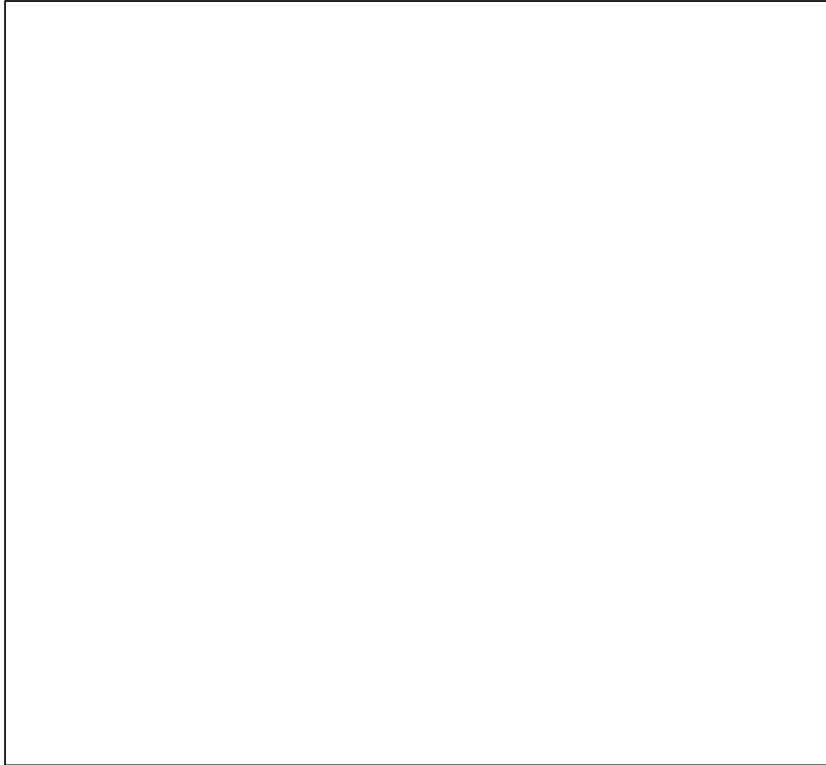


Figure-2- A general workflow of various clinical presentations.

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Section V

Emergencies in ENT

FACIAL TRAUMA

SCOPE

Includes general and detailed strategy for management of facial trauma.

AIM

To develop guidelines for the management of patients with facial trauma.

INTRODUCTION

Faciomaxillary injuries are very common in Road Traffic Accidents. It can cause bony as well as soft tissue injuries and have the potential to interfere with airway, cause disfigurement and loss of function. Hence proper evaluation and early intervention is a must.

CARE PATHWAY

Primary care center

The airway should be assessed and secured if possible before referring the patient. Visual deficits should be ruled out. Minor injuries including contusions, abrasions, lacerations, incised wounds may be managed at PHC.

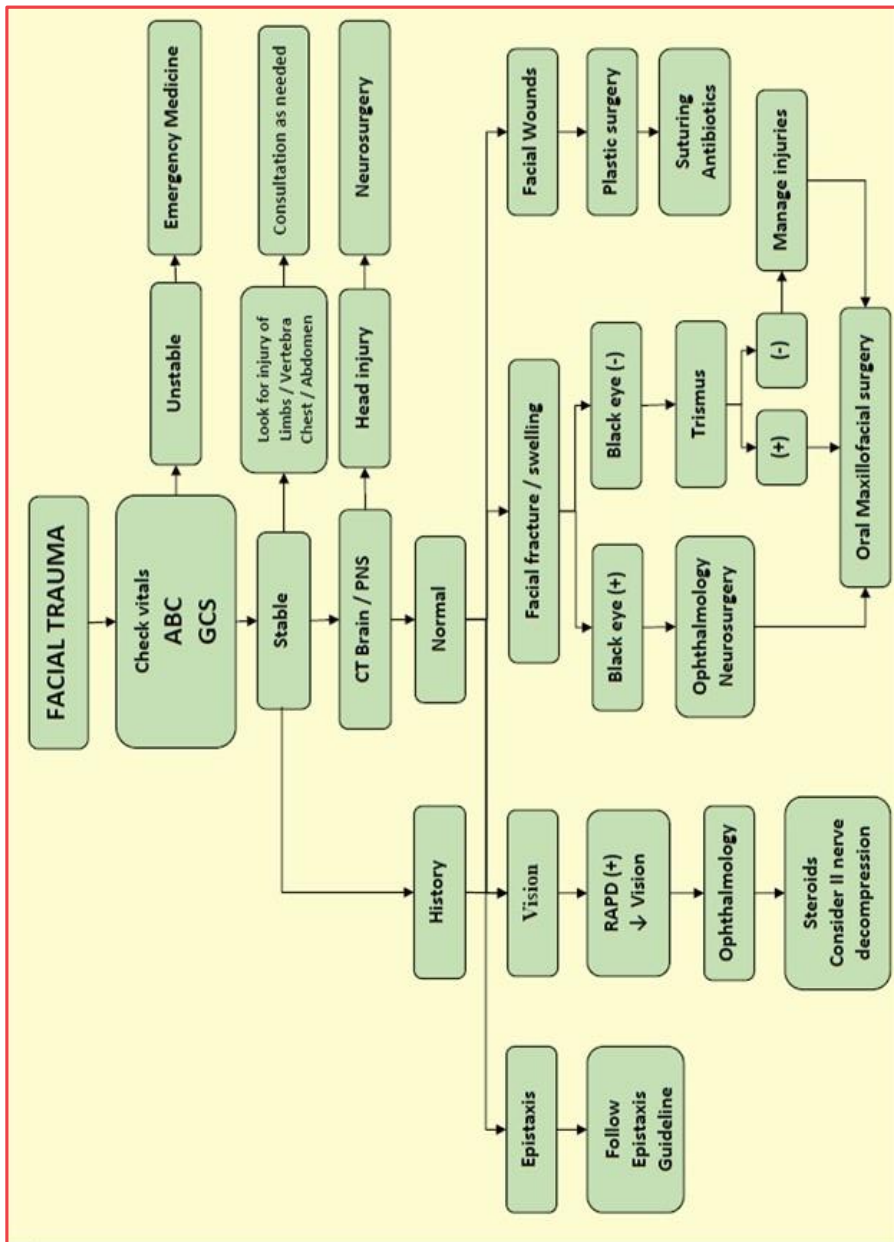
Referral criteria

- Compromised airway
- Visual compromise
- Suspected fracture of facial bones
- Uncontrolled epistaxis
- Extensive soft tissue injuries

Investigations

- CBC, Coagulation profile
- CT Brain / PNS

Flowchart for management of facial trauma is given below



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EAR TRAUMA

SCOPE

Includes general and detailed management strategy for patients with ear trauma, complaining of ear bleed, facial palsy, pinna injury, hard of hearing or vertigo.

AIM

To develop criteria for the management of patients attending emergency medicine department with ear trauma regarding admission / conservative / surgical management

INTRODUCTION

Ear trauma can be blunt or penetrating, the impact of which can vary from pinna laceration, traumatic tympanic membrane perforation, facial nerve palsy, fracture temporal bone, labyrinthine concussion, hearing loss and ear bleed.

Prognosis largely depends on the accurate diagnosis and timely intervention. Ear trauma is usually associated with other injuries including traumatic brain injury, abdominal trauma and other orthopedic injuries. In this scenario, there is a high chance of ear trauma going unnoticed, resulting in permanent handicap.

CARE PATHWAY

Primary Health center

Isolated ear trauma including lacerated pinna injury/traumatic perforation can be managed at PHC

Referral criteria

- Mutilated pinna
- Amputated pinna
- Facial palsy
- Vertigo and ear bleed, if HRCT facility is not available
- Lack of availability of ENT specialist

MANAGEMENT

Assess the general condition

- ABC to be established
- Detailed history and proper ENT examination
- Investigations
- CBC, URE, Coagulation profile

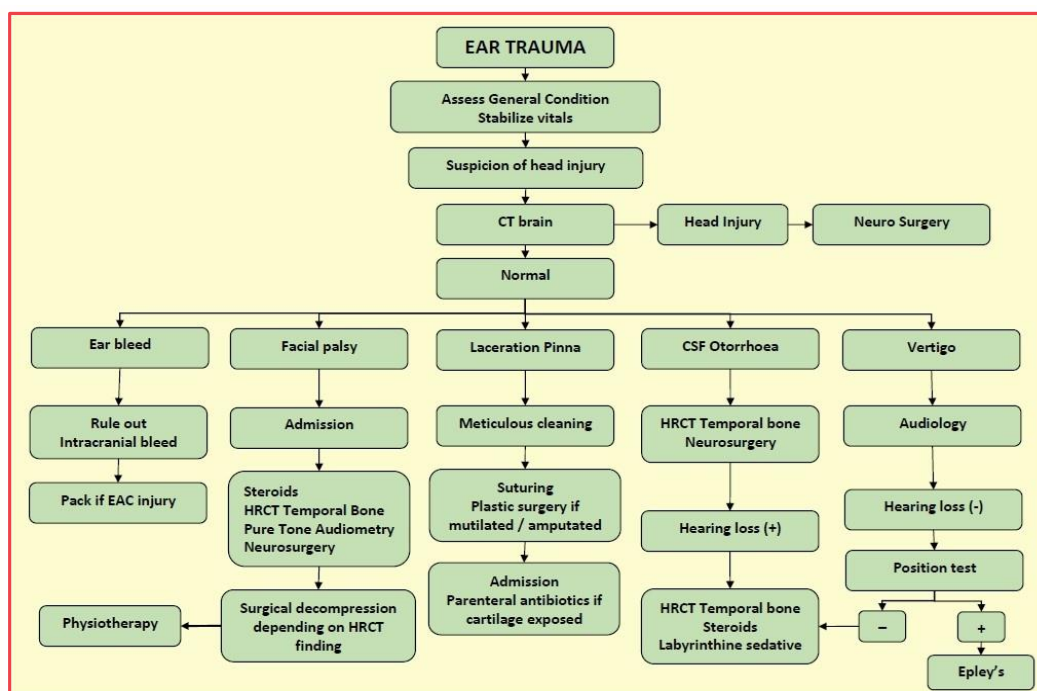
- CT brain
- Special investigations include HRCT Temporal bone and PTA

Criteria for admission

All trauma cases with

- Vertigo
- Facial Paralysis
- Mutilated / amputated / hematoma pinna
- SNHL

Flowchart for management of ear trauma is given below



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FACIAL PARALYSIS

SCOPE

Includes general and detailed management strategy for facial paralysis

AIM

To develop criteria for management of patients attending emergency medicine department with facial paralysis

INTRODUCTION

Facial paralysis can occur isolated or along with CVA. Isolated facial paralysis will be managed in ENT. Post traumatic palsy needs detailed evaluation to decide regarding surgical intervention.

CARE PATHWAY

Primary care center

Detailed history and examination to be done.

If idiopathic, it can be managed at primary care center conservatively.

Referral criteria

- Post traumatic
- Associated hearing loss
- Vesicles around the ear
- Other neurological deficits
- If associated with COM/MOE/AOM

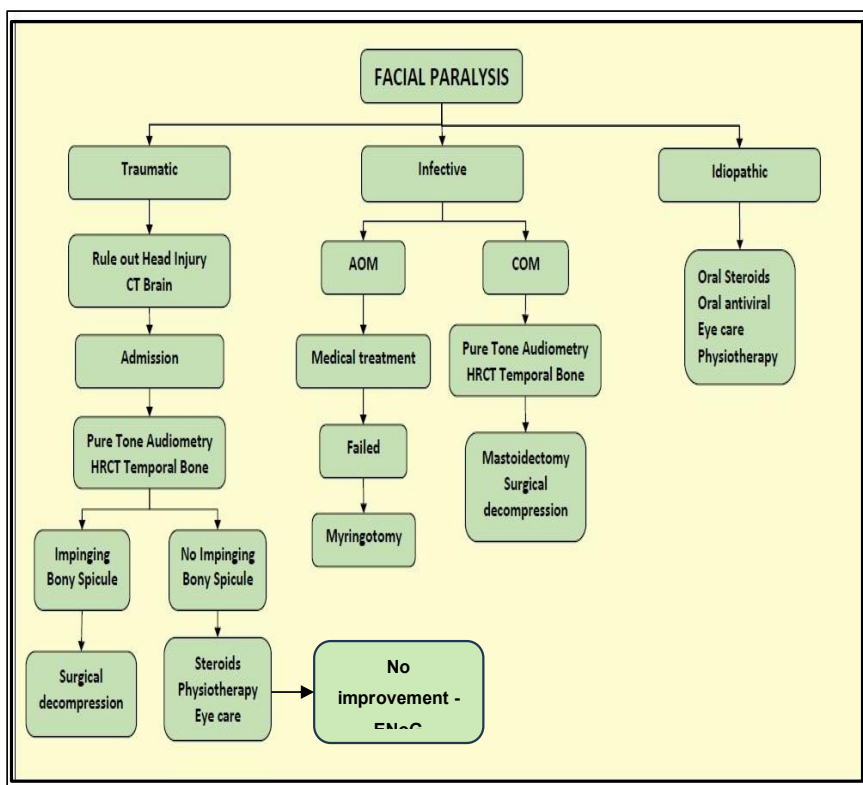
Investigations

- RBE, URE
- CT Brain HRCT Temporal bone
- Pure tone Audiogram

Criteria for Admission

- Associated with complications (AOM & COM)
- MOE
- Post traumatic

Flowchart on management of facial paralysis is given below



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EARACHE

SCOPE

Includes general management strategy and detailed guidelines for earache, which is associated with ear discharge, hard of hearing, itching ear, and referred otalgia.

AIM

To develop guidelines for management of patients attending emergency department with earache regarding diagnosis and management.

INTRODUCTION

Otalgia can be primary or referred. Typical causes of primary otalgia include otitis externa, otitis media, mastoiditis and auricular infections. Many remote anatomic sites share common innervation of ear and pathologies in these areas may be perceived as otalgia.

DEFINITION

Otalgia or earache has two distinct types, pain that originates within the ear is primary otalgia & pain that originates outside the ear is referred otalgia.

CARE PATHWAY

Primary health center

- Otitis externa and media without complications can be managed at PHC.
- Other causes should be treated accordingly.

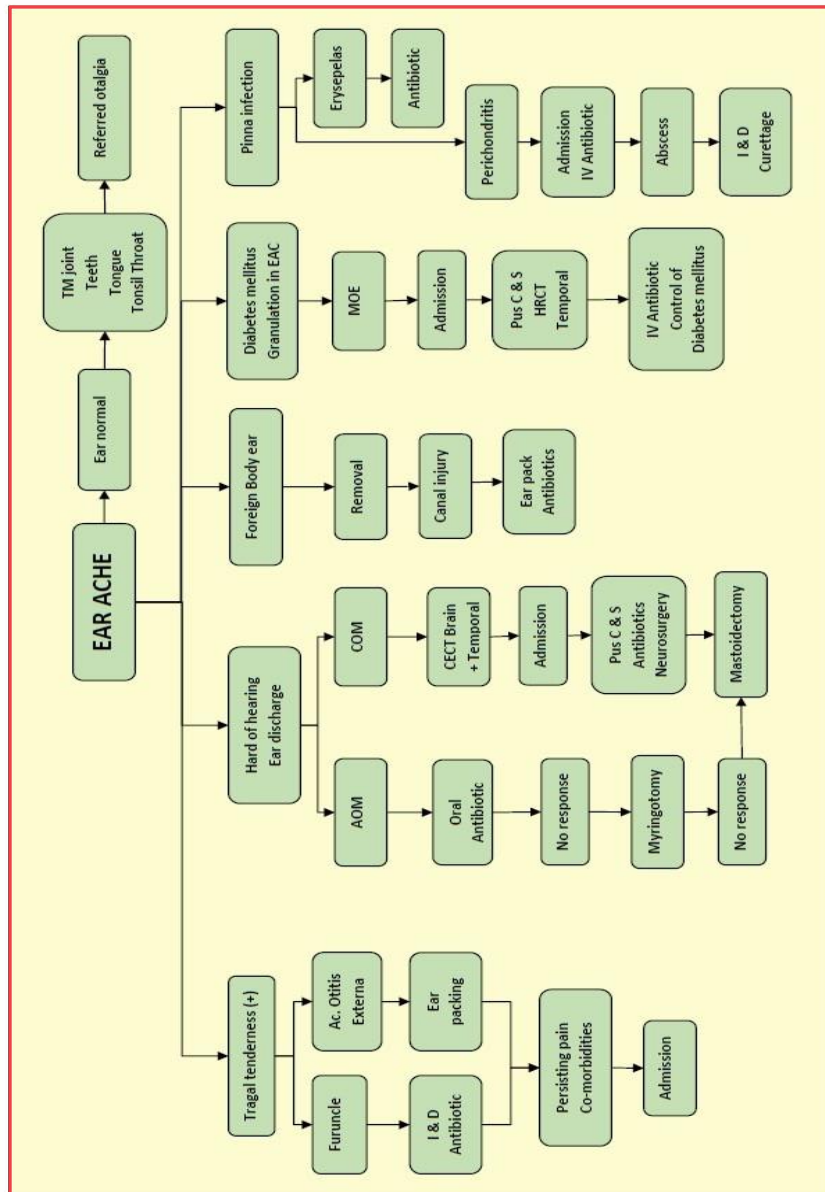
Referral criteria

- Suspicion of complications in AOM / COM / Malignant otitis externa
- Suspicion of malignancy of upper aerodigestive tract

Investigations

- CBC, RFT, LFT, RBS, HRCT Temporal bone, PTA

Flowchart on management of earache is given below



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IDIOPATHIC SUDDEN SNHL

INTRODUCTION

Sudden SNHL is defined as a rapid-onset sensorineural hearing impairment, occurring over a 72-hour period, in one or both ears, with decrease in hearing thresholds of greater than or equal to 30 decibels, affecting at least 3 consecutive frequencies.

Idiopathic sudden sensorineural hearing loss (ISSNHL) is defined as SSNHL with no identifiable cause despite adequate investigations

EVALUATION

- If audiometry confirms a 30dB hearing loss at 3 consecutive frequencies AND an underlying condition cannot be identified by history and physical examination a presumptive diagnosis of idiopathic Sudden SNHL may be entertained.
- Differentiate conductive from sensorineural hearing loss in all patients presenting with sudden hearing loss.
- Differentiate bilateral sudden hearing loss, recurrent sudden hearing loss and hearing loss as a part of other neurological deficits (from Idiopathic Sudden SNHL) in all patients since the approach should be different.
- Look for clinical features that may be associated with a definable underlying disease at the time of presentation. Among such causes are systemic disorders, autoimmune disorders, metabolic disorders, bilateral Meniere's disease, and certain neurological disorders. [see treatment algorithm]
- Retro-cochlear pathology like acoustic neuroma needs to be ruled out by obtaining a MRI brain or Auditory Brainstem Response (ABR). [CT scan of brain is NOT indicated in the initial evaluation of a patient with presumptive SSNHL].
- Routine laboratory tests are NOT immediately necessary in patients with such presumed ISSNHL to avoid false positives, delay in diagnosis and treatment.

MANAGEMENT

- **Systemic corticosteroids** should be offered as the initial therapy to all patients with ISSNHL.

[Suggested treatment regimen would be oral Prednisolone given at 1mg/kg/d in a single dose, (maximum dose of 60 mg daily), and treatment duration of 10 to 14 days; full dose may be prescribed for initial 7 days, followed by taper. Alternatively, equivalent doses of Methylprednisolone (48mg daily) may also be used]

- Antivirals, thrombolytics, vasodilators, vasoactive substances, or antioxidants are **NOT** 'routinely' indicated in patients with ISSNHL. [However, there may be patient

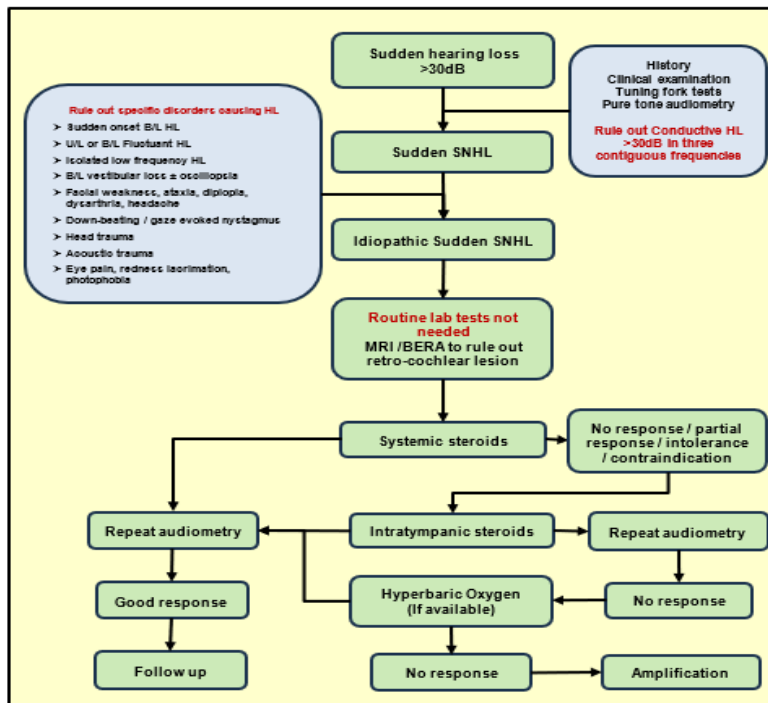
specific indications for each of these options that may be reasonable to try on an individualized basis].

- **Intra tympanic steroid perfusion** may be offered to patients who do not respond to systemic steroids or show only incomplete response. It is also an option in patients exhibiting intolerance to oral steroids or when systemic steroids are contraindicated.
- If facilities are available, **Hyperbaric oxygen therapy** may be suggested to patients with incomplete recovery from ISSNHL after failure of initial management, within three months.

FOLLOW UP

- Follow up audiometric evaluation is recommended within 3 - 6 months to rule out progressive hearing loss.
- The clinician should counsel patients with incomplete recovery of hearing about the possible benefits of amplification and other Hearing-assistive technologies for improving communication in specific listening conditions

Treatment algorithm for Idiopathic Sudden Sensorineural Hearing Loss (ISSNHL)



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VERTIGO

SCOPE

Includes general and detailed management strategy for vertigo

AIM

To develop criteria for the management of patients with vertigo attending emergency medicine department

INTRODUCTION

The symptoms related to balance disorders are described in different ways and the commonest term used is dizziness which can range from true vertigo to presyncope. True vertigo is described as rotational or spinning sensation implying a dysfunction of either central or peripheral vestibular system. Thorough history and clinical examination can help in arriving at a diagnosis which is very important for proper management. Peripheral vertigo is managed in ENT. Common causes of peripheral vertigo include BPPV, Meniere's disease, vestibular neuronitis, labyrinthitis.

CARE PATHWAY

Primary health center

Detailed history and evaluation should be done to differentiate central and peripheral types. Central vertigo should be referred to a higher center for detailed evaluation. History should be elicited regarding the onset, duration, triggering events as well as the associated symptoms.

Ask for dangerous Ds in vertigo

- Diplopia
- Dysarthria
- Dysphagia
- Dysmetria

Clinical examination is aimed at evaluating the cause of vertigo as peripheral or central so as to arrive at a diagnosis and to outline a concrete treatment plan.

HINTS plus test which include Head impulse test, Nystagmus, Test for Skew, Tuning fork tests gives a clue as to whether it is of peripheral or central. A normal head impulse test on both sides with direction-changing nystagmus or skew deviation is concerning for a central lesion. An abnormal head impulse test with unidirectional nystagmus and absent skew strongly suggests a peripheral lesion

If clinically suspicious of BPPV, do positional tests and do corrective maneuvers. For other causes vestibular sedatives may be administered before referring to higher centers.

Referral criteria

- Vertigo associated with other neurological deficits
- Suspected complication of COM
- Intractable vertigo
- Associated with hearing loss or tinnitus

Secondary care:

This involves referral to a centre where the facility for the patient being examined by a specialist like ENT, neurologist, Psychiatrist and physiotherapist are available. Here the patient will undergo more advanced tests like VNG, Puretone audiometry, Computerized Tomography scan etc. Performance of various positional tests. More comprehensive treatment plans including vestibular rehabilitation therapy, exercises and medications will be offered. Patient education and counselling will be done

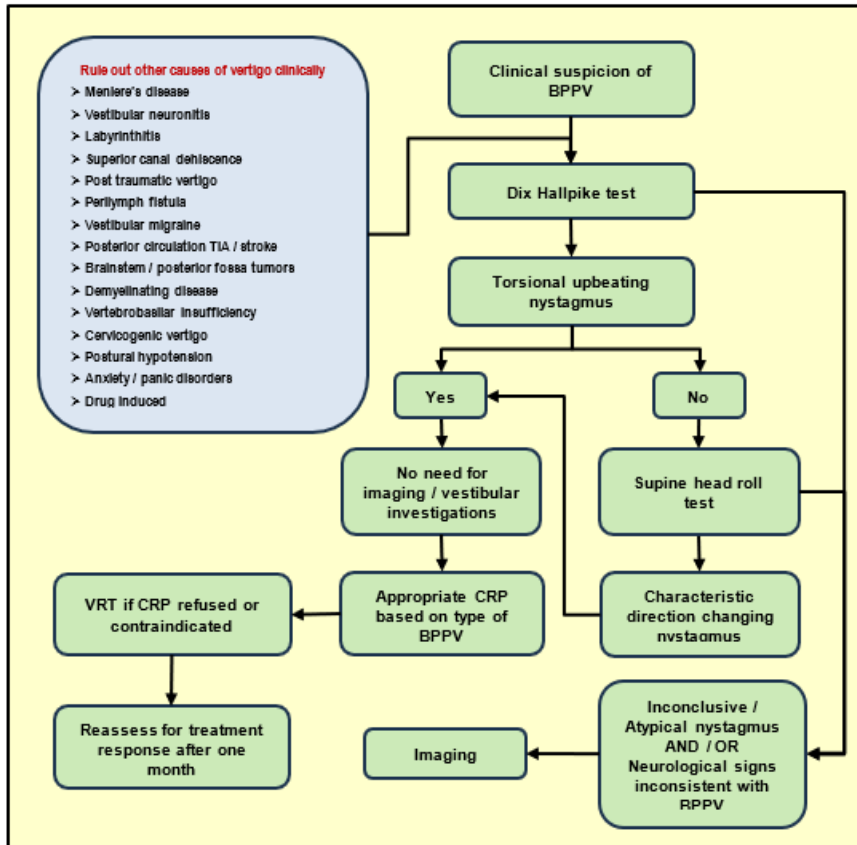
Tertiary care:

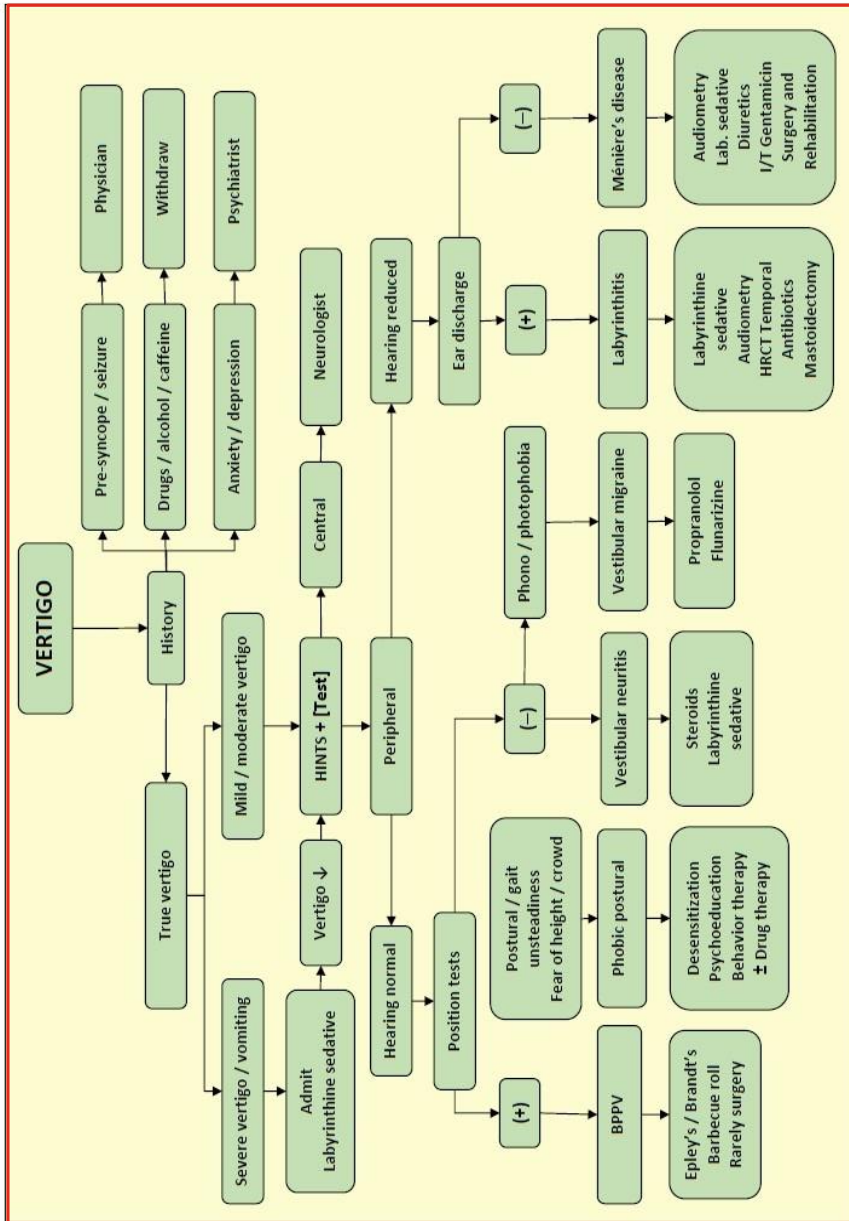
Tertiary care management of vertigo involves referral to a highly specialized centre like a vestibular disorder clinic or balance clinic where patients receive advanced and multidisciplinary care. Here the patient will be evaluated by a highly specialized neurotologist and physiotherapist specialized in balance disorders will evaluate and develop an individualized treatment plan.

Investigations in Vertigo

- CBC, RFT, LFT, RBS, TFT, VDRL, ANA Profile, Vit D3 level.
- X-ray Cervical spine
- Pure tone Audiogram
- Caloric test if indicated
- VNG if available
- MRI with MRV to rule out AICA/Labyrinthine artery occlusion
- HRCT Temporal bone / CT brain in selected cases

Flow chart on suspicion of BPPV





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FOREIGN BODY EAR

SCOPE

Includes general and specific management of foreign bodies in the ear, which is common in the pediatric population

INTRODUCTION

Foreign body ear is common in children and adults. It may even be an incidental finding. They can be live or inanimate. Proper examination should be done to identify the foreign body and removal done with least trauma possible. Corrosive foreign bodies (e.g. Button batteries) are best removed as early as possible. Long forgotten corrosive foreign bodies may cause extensive erosion and granulation; their removal is fraught with danger and done better by experts.

CARE PATHWAY

Primary health center

Removal may be attempted at primary care centers if the following criteria is met

- FB is visible, non-impacted
- No history of ear discharge/ tympanic membrane is intact
- Can be attempted by instrumentation / Syringing (Syringing to be avoided in vegetable/ hygroscopic foreign bodies)

Referral criteria

- Impacted FB
- Suspected FB in middle ear
- Facial Nerve Palsy
- Previously attempted and failed removal

Investigations

- Examination under microscope
- HRCT temporal bone if complications like Facial palsy/ ossicular disruption

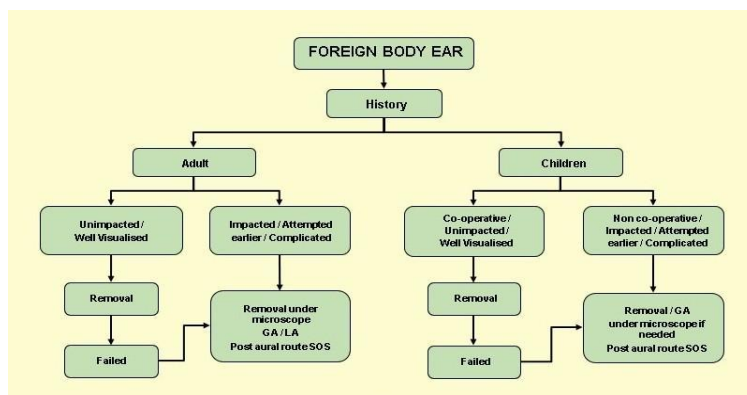
TREATMENT

1. If live foreign body like insect, kill it with liquid paraffin or sterile water before removal
2. If visible, and not impacted, removal in emergency room with grasping forceps, wax hook or micro suction. If tympanic membrane is intact and foreign bodies are non-vegetable, syringing can be tried. Do not syringe if the tympanic membrane

is perforated.

3. If not visible, removal under GA in children and non-cooperative adults.
4. Use surgical microscope in impacted / complicated FB.
5. Post aural approach if needed for impacted FB.

Flowchart on management of foreign body ear is given below



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FOREIGN BODY NOSE

SCOPE

Includes general and specific management of foreign bodies in the nose, which is common in the pediatric population

INTRODUCTION

Foreign body nose is common in children and mentally retarded adults. It should be suspected in any child with unilateral nasal discharge. Proper examination should be done to identify the foreign body and for timely removal.

CARE PATHWAY

Primary health center

Removal may be attempted at primary care centers if FB is visible.

Referral criteria

- Posteriorly located FB
- Suspected FB, if not visualized
- Rhinolith
- Previously attempted and failed

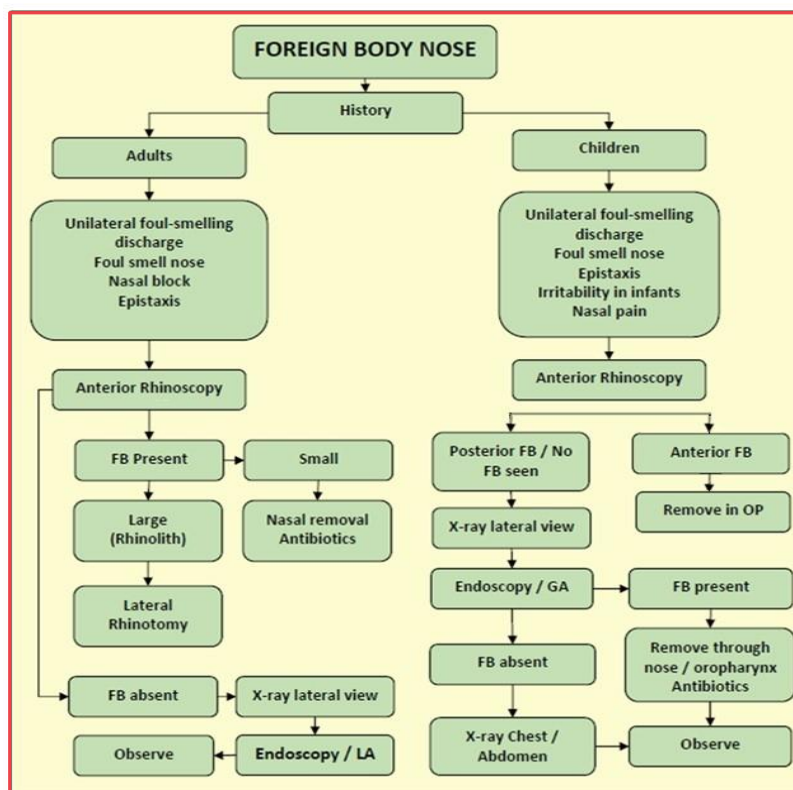
Investigations

- X-ray PNS & skull lateral view if radio-opaque FB is suspected and not visualized on examination
- Diagnostic nasal endoscopy
- CT PNS

TREATMENT

- If visible, removal in the emergency room followed by decongestants and antibiotics.
- If not visible, endoscopic removal under GA in children and mentally retarded adults.
- Endoscopic / external approach in case of rhinolith

Flowchart on management of foreign body nose



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EPISTAXIS

SCOPE

This guideline is applicable to all medical personnel involved in the management of epistaxis. This guideline will act as a reference in secondary and tertiary care hospitals and primary care centers, available to all clinicians involved in the patient management. This guideline includes general management strategy and detailed diagnostic and therapeutic guidelines for conditions causing epistaxis.

AIM

To develop criteria for management of patients attending emergency medicine department with epistaxis

DEFINITION

The management of patients with epistaxis comprises of two concurrent processes- diagnostic and therapeutic culminating in decision to either conservatively manage or to interfere operatively.

INTRODUCTION

Epistaxis is just a symptom, and the majority causes are idiopathic. It can be torrential requiring emergency management or can be mild. The causes of epistaxis are many, but common causes are

- Inflammatory diseases- viral upper respiratory infection, bacterial sinusitis, allergic rhinitis, environmental irritants
- Traumatic
- Neoplasm - malignancy-suspected especially in elderly
- Benign- Vascular malformation, hemangioma, juvenile nasopharyngeal angiofibroma
- Structural- Nasal septal deformity with spurs, septal perforation
- Systemic-Coagulation deficits like thrombocytopenia, Coagulopathies, Liver disease, renal failure, chronic alcohol abuse etc.
- Vascular diseases- arteriosclerotic, Hereditary hemorrhagic telangiectasia
- Cardiovascular conditions - Hypertension
- The most common site of hemorrhage is Little's area in the septum in young adults. In elderly the causes are usually arteriosclerosis in association with hypertension and malignancy.

Initial evaluation

Assessment of haemo-dynamic stability and potential airway compromise of the patient including a quick visual assessment, review of vital signs and laboratory studies that may have been obtained

Management aims at

- Resuscitation
- Establish the site and diagnosis
- Management of bleeding & the cause

Primary care centres

The aim of primary care physicians should be to stop bleeding and refer the patient to higher centers for further management. If the bleeding stops by itself, they can manage the cause as established. The bleeding can be stopped by

- Pinching the nose (Trotter's position)
- Cold compresses
- Controlling blood pressure if high
- Propped up position
- Stop antiplatelet drugs, anticoagulants, NSAIDs (if patient is on these)
- IV access. Patients should be reassured & explained about the planned evaluation and possible treatment
- Topical nasal decongestants. If bleeding does not stop, hemostatic injections like Inj. Tranexamic acid 1amp in 100 ml NS slow IV or Inj. Ethamsylate 1amp iv can be tried.
- If bleeding persists, nasal packs are needed. Anterior nasal packing can be given, and the patient referred to higher centers

Referral criteria

- If bleeding is severe enough which is not controlled by external digital pressure
- Uncontrolled blood pressure
- Associated faciomaxillary injury
- If bleeding is associated with nasal mass (rhinosporidiosis / malignancy)
- If it is unprovoked bleeding especially in adolescent males (JNA to be ruled out)
- If associated with unilateral foul-smelling nasal discharge in a child (suspected

foreign body)

- Recurrent nasal bleeding in elderly
- Coagulopathies
- Post-surgery bleed

Secondary care:

History and examination

The experienced ENT surgeon or senior or junior resident see the case in ENT casualty and provide early assessment and investigations to reduce unnecessary admissions. Based on detailed history and examination, ENT consultants can arrive at various possibilities and order necessary investigations.

Investigations

Hb, CBC, RBS, BT, CT, PT-INR, APTT, RFT, LFT, Peripheral smear, Blood grouping and cross matching

Depending on nasal examination - CT scan Nose & PNS + diagnostic nasal endoscopy

TREATMENT

It depends on clinical diagnosis.

- May be viewed as a continuum ranging from no intervention with only conservative treatment to invasive surgical and angiography procedures
- Admit those with not much control of bleeding. In most patients, bleeding responds to nasal packing or cauterization.

Medical approaches are

- Adequate pain control and antibiotic coverage for patients with pack.
- Avoid Aspirin / NSAIDs
- Control underlying medical illness
- If direct pressure is not enough to control bleeding epinephrine-soaked gauze may be placed to bring about vasoconstriction.
- Cauterization of bleeding point endoscopically
- Warm saline irrigation may be tried as a non-invasive measure to control posterior epistaxis
- Nasal packing – anterior nasal packing, various packing materials are available. (Medicated ribbon gauze or petroleum jelly gauze (0.5 in × 72 in) smeared with an antibiotic ointment is traditionally used. Commercially available Merocel sponge or

absorbable materials (e.g. Gelfoam, Surgicel, Avitene) may be used to prevent trauma during packing removal

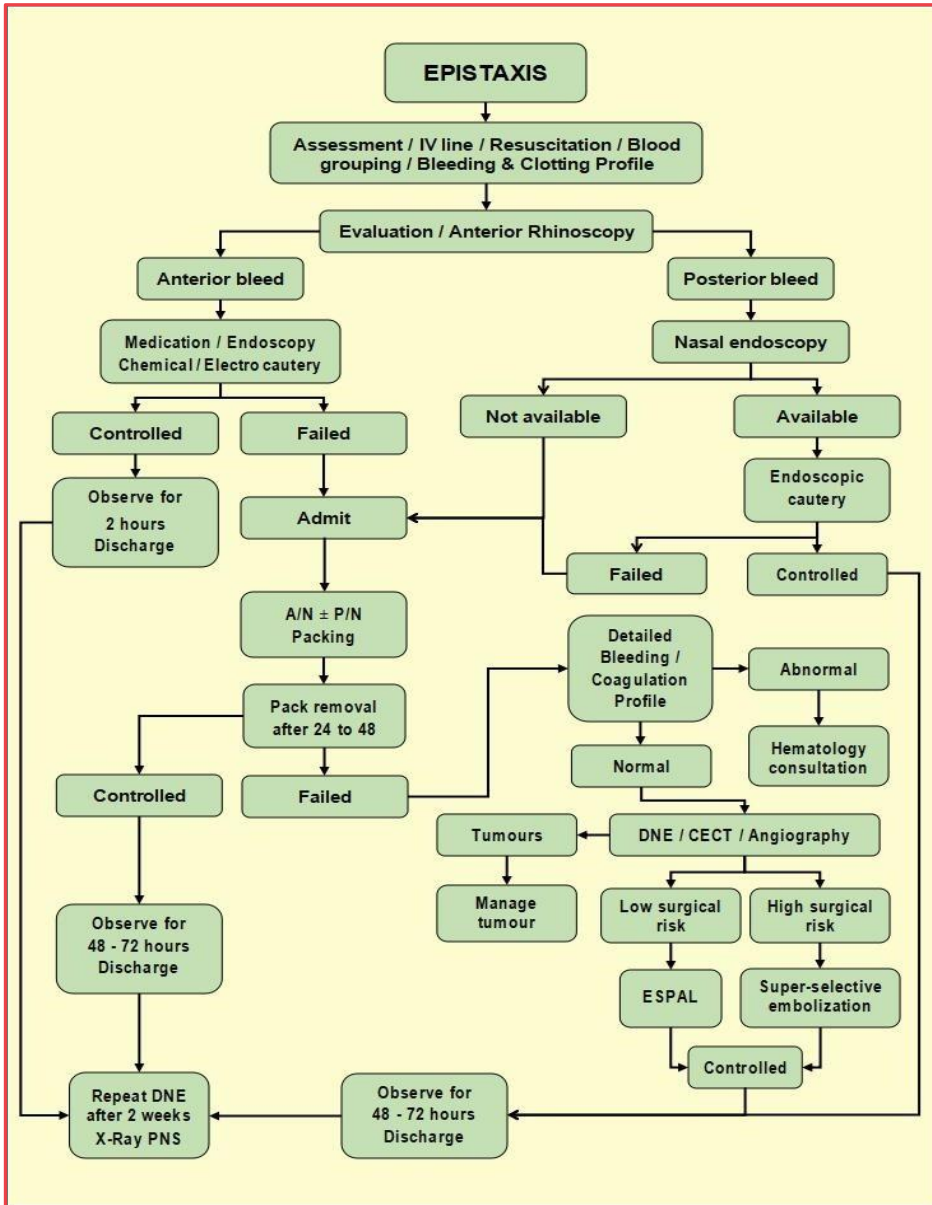
- If not controlled posterior nasal packing with inflatable balloon devices
- A careful endoscopic examination with the patient under general anesthesia may be considered. Bleeding sites may be cauterized, spur or DNS may be corrected, and meticulous packing can be done
- If all the steps fail to control bleeding arterial ligation may be performed - sphenopalatine artery, internal maxillary artery or external carotid artery depending on the case
- Embolization may be tried in expert centres with facilities. Pre- embolization angiography is performed before procedure
- Management of hereditary hemorrhagic telangiectasia (HHT) – Options include coagulation with Potassium – Titanyl – Phosphate (KTP) or Neodymium: Yttrium – Aluminum – Garnet (Nd: YAG) lasers, septodermoplasty, embolization and estrogen therapy.

Prevention of Epistaxis

To the extent possible, patients should avoid the following:

- Strenuous activities - Protection from direct trauma from contact sports activities is afforded using helmets or face pieces.
- Hot and dry environments – The effects of such environments can be mitigated by using humidifiers, better thermostatic control, saline spray, and antibiotic ointment on the Kiesselbach's area.
- Digital trauma – In children, nose picking is difficult to deter. Keeping the child's nails well-trimmed may be helpful.
- Nose blowing and excessive sneezing - Instruct patients to sneeze gently with their mouths open. Inappropriate or careless use of drugs - Consider drug education relating to use or accidental ingestion of aspirin, warfarin (e.g. rat poison in toddlers), or drug abuse in adolescents

Flowchart for management of epistaxis is given below



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HEADACHE

SCOPE

This guideline is to define the management criteria for all patients with headache coming to ENT department.

AIM

To develop guidelines for medical & surgical management of headaches and to define referral criteria.

INTRODUCTION

Headache is one of the most common symptoms presenting in ENT casualty. It can occur due to a variety of causes. Depending upon the cause, it can be managed at a primary care center or referred to a higher center.

Primary care center

Any uncomplicated sinusitis, headache due to viral illness or headache with typical auras of migraine can be managed at primary care center.

Referral criteria

Any clinical suspicion of complication in sinusitis or headache not responding to conventional treatment can be referred to secondary or tertiary care center where expert ENT surgeon, neurosurgeon and investigation facilities are available.

AETIOLOGY

Sinusitis

- If patients had viral rhinitis, there is a high chance for developing secondary sinusitis
- Recent facial trauma
- Caries teeth
- Gross deviation of nasal septum which is obstructing the ostiomeatal complex.

Complications of sinusitis

- Associated fever and constitutional symptoms – possibility of viral illness.
- Purulent nasal or postnasal discharge – sinusitis. Acute sinusitis – pain & tenderness over the sinus. Chronic sinusitis– only heaviness of head. Non-invasive fungal sinusitis also can present with headache.
- Orbital complications presenting with proptosis, chemosis, diminished

- vision in one eye – orbital cellulitis
- If both eyes involved – cavernous sinus thrombosis
- Associated nerve involvement–superior orbital fissure syndrome or orbital apex syndrome.
- Osteomyelitis of sinuses
- Neck rigidity – Meningitis
- Co-existent diabetes or other immune-compromised states –invasive fungal sinusitis like mucormycosis and aspergillosis.

Space occupying lesions of brain

- Headache + localizing signs + projectile vomiting – intracranial space occupying lesions.

Neurovascular headache

- Migraine: Typical auras with unilateral headache, diplopia, intolerance to sound and light, nausea, vomiting followed by remission. Family history/similar episodes earlier may be there.
- Trigeminal neuralgia
- Cluster headache
- Herpes zoster

Investigations

- Digital X-ray PNS-sinusitis
- CT scan head - if suspicion of intracranial lesions
- CT scan orbit nose and PNS – if suspicion of orbital cellulitis
- MRI brain – cavernous sinus thrombosis, meningitis
- Diagnosis of vascular headache is purely based on clinical examination and by excluding other causes.

TREATMENT

- **Acute bacterial sinusitis**

Broad spectrum antibiotics like Co- amoxiclav 625 mg TDS for 10 - 14 days. In penicillin sensitive patients, Erythromycin 500mg QID can be tried. Analgesics like Paracetamol and antihistamines like Chlorpheniramine maleate are given if associated with nasal allergy. Surgery – FESS in non- responding cases.

- **Fungal sinusitis**

Non-invasive cases: FESS + steroid sprays

Invasive cases: ESS debridement + antifungal treatment. IV Amphotericin for mucormycosis.

- **Complications of sinusitis**

- **Orbital cellulitis:** Parenteral antibiotics + immediate orbital decompression after having ophthalmology consultation.
- **Cavernous sinus thrombosis:** Neurology reference. FESS for primary pathology.
- **Meningitis:** Neurology *reference*
- **Brain abscess:** Neurosurgery *reference*

- **Migraine**

- **Acute attack**

Analgesics: Paracetamol 1000mg

Naproxen 500mg

- **Prophylactic:**

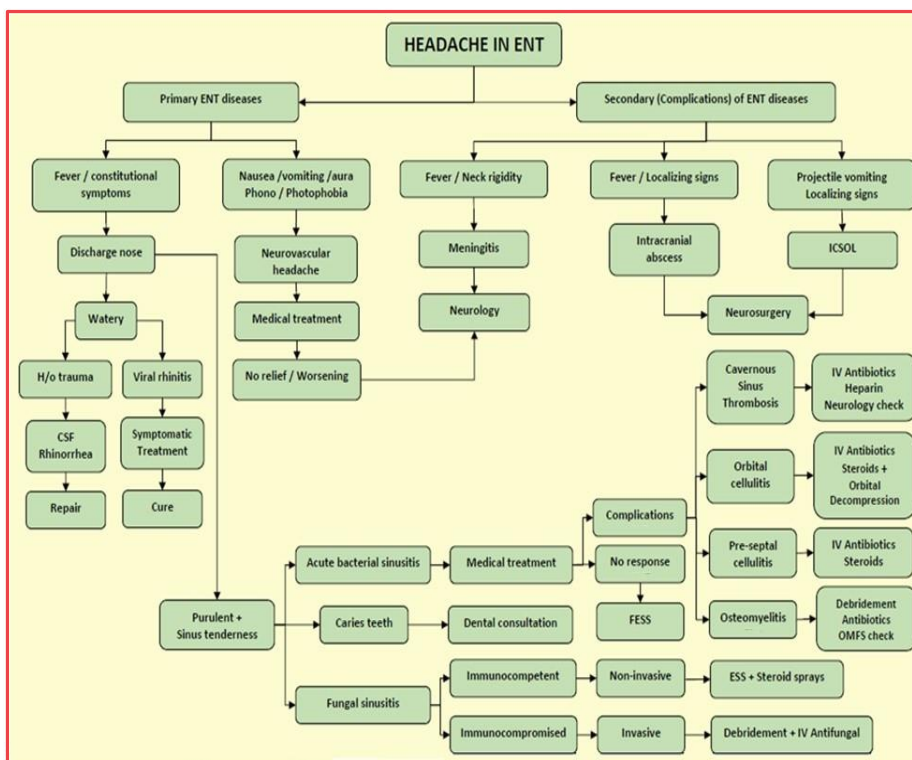
Propranolol 20 - 40 mg BD

Amitriptyline 10 mg HS

Flunarizine 10 mg HS can be tried for long periods.

Neurology reference for resistant cases and atypical headaches

Flowchart on management of headache in ENT



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FOREIGN BODY THROAT

SCOPE

Foreign body throat is a common emergency. This can happen in all age groups and timely and judicious intervention is very important.

INTRODUCTION

Ingested foreign bodies are one of the most frequently encountered emergencies in otolaryngology practice. Many of these foreign bodies get lodged in the upper digestive tract i.e. oropharynx, hypopharynx or oesophagus. Identification and removal of it is very important as they can cause life-threatening suppurative or vascular complications. The most common foreign bodies in children are coins, but marbles, button batteries, safety pins and bottle tops are also reported. In adults, common foreign bodies are bones, meat bolus, dentures, safety pins and metallic wires.

AETIOLOGY¹

- Loss of protective mechanism
- Carelessness: Hasty eating, influence of alcohol
- Narrowed oesophageal lumen
- Psychotics

CARE PATHWAY

Primary care centres

Primary care doctors face the challenge of dealing with ingested foreign bodies. Since the identification and removal needs proper evaluation and instrumentation, all cases need to be referred appropriately.

Secondary care:

History & Examination:

Proper history is very important in these cases. The nature of foreign bodies, day of impaction, symptomatology, associated co-morbidities are key points in identification and further management¹.

A proper examination of the oral cavity and oropharynx is to be done. If the foreign body is visible and proper instruments are available, it can be removed.

If the impaction is lower down, an X-ray can be advised, and the patient can be referred for appropriate removal. The absence of radio-opaque shadow does not exclude foreign body cent percent and hence the associated clinical symptoms should be acknowledged and should be referred for further management.

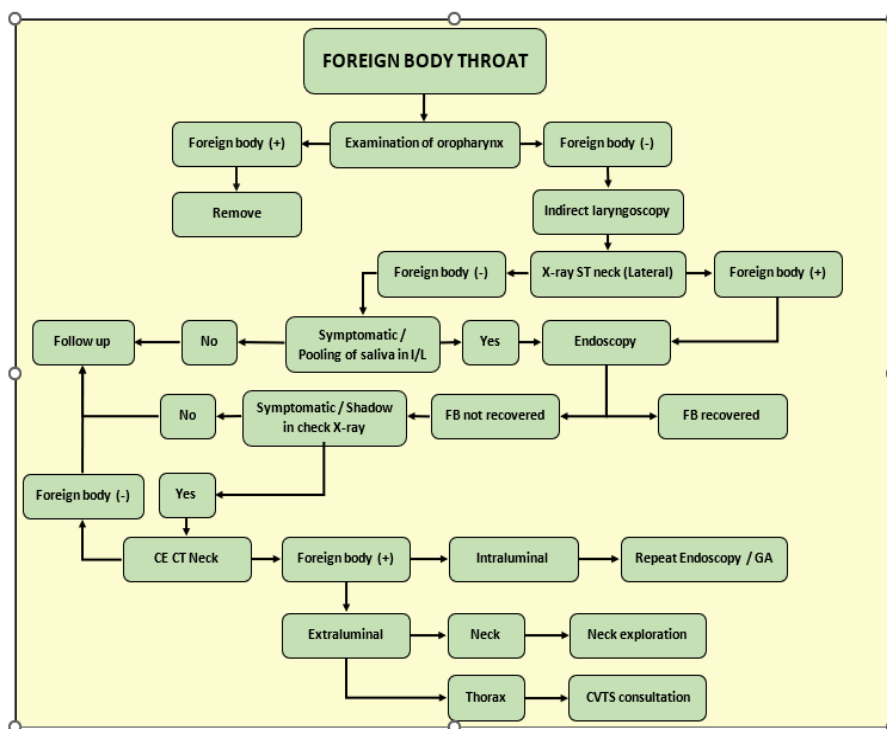
Referral criteria:

- No facility for endoscopy and impaction of foreign body at hypopharynx or esophagus.
- Duration of foreign body impaction > 1 day.
- The nature of foreign body as sharp bones, dentures, safety pins, batteries etc.
- Site of impaction as cervical and thoracic esophagus.
- X-ray negative, but presence of symptoms.
- Associated with complications such as neck space infection, respiratory problems, mediastinitis, perforation etc.
- Associated with severe co-morbidities.

Tertiary care:

- Confirmation of foreign body
- Endoscopic removal
 - Most of the foreign bodies in the esophagus can be removed by esophagoscopy under general/ local anesthesia.
 - Both rigid and flexible scopes have been used to remove foreign bodies from the esophagus.
 - A rigid oesophagoscope, of appropriate for the size of patient with proper forceps is preferred.
 - Soft (meat pieces without bone, vegetable matter) and blunt objects can be removed with flexible scopes.
 - Special care should be taken in case of a denture, especially one with a metal hook pointing upwards. Prompt removal of disc battery is also important as it can lead to complications like stricture, perforation, esophageal fistula, mediastinitis and death.
- **CECT Scan** Neck+/- Thorax - when associated with complications and suspecting extraluminal migration – manage accordingly.
- **CVTS consultation:** Site of impaction in thoracic esophagus and with complications such as perforation or mediastinitis.

Flow chart on management of foreign body throat



REFERENCES

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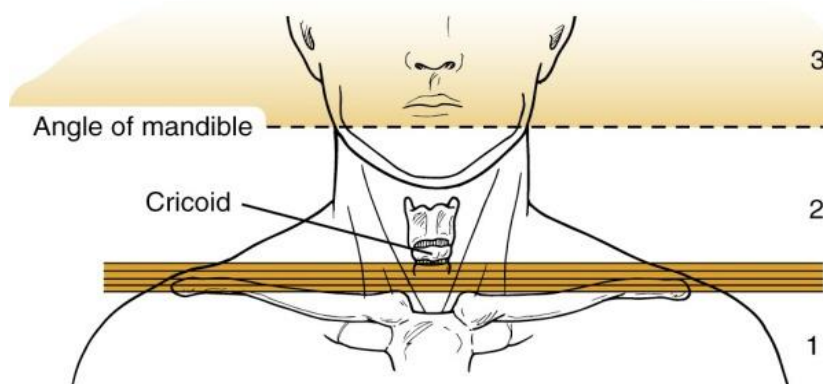
CUT-THROAT INJURY

SCOPE:

Incidence of cut-throat injuries and associated morbidities & mortalities are not uncommon in present day life. These patients need emergency and multispecialty care. Early management of patients can save the life of the patient most of the time.

INTRODUCTION:

Cutthroat injuries, one of the emergency conditions managed by ENT specialists, pose a great therapeutic challenge because multiple vital organs for phonation, deglutition, vascular and neurological structures vulnerable to injuries are present in a small confined unprotected area. Globally, cutthroat injuries account for approximately 5% to 10% of all traumatic injuries with multiple structures being injured in 30% of patients². According to World Health Organization (WHO), every year over 5 million people around the world die because of cut-throat injury¹. In developing countries, the incidence is increasing at a fast rate because of increasing conflict over limited resources, poor socio-economic condition, unemployment, easy access to alcohol and increased crime rate. This may result from accident, homicide, or suicide. These patients need emergency and multispecialty care. The attending physician must have an exceptional knowledge about the anatomy since it has a high mortality rate of 11%.



According to Roon and Christensen's classification, neck injuries are divided into three anatomical zones. Zone I is defined as the area from the clavicles to the inferior margin of cricoid cartilage. Structures within this zone include the vertebral and proximal carotid arteries, major thoracic vessels, superior mediastinum, lungs, oesophagus, trachea, thoracic duct, and spinal cord. Zone II extends from the inferior margin of the cricoid cartilage to the angle of the mandible. The carotid and vertebral arteries, jugular veins, oesophagus, trachea, larynx, and spinal cord are found in this zone. Zone III is located between the angle of the mandible and the base of the skull. It includes the carotid and vertebral arteries, pharynx and

spinal cord².

INITIAL MANAGEMENT:

The initial care of patients with penetrating neck injuries should follow the basic tenants of trauma care. The emergency management of all penetrating neck trauma requires (1) airway establishment, (2) blood perfusion maintenance, and (3) clarification and classification of the severity of the wound. In the emergency department, satisfactory control of the airway is established by intubation, cricothyroidotomy, or tracheostomy. Large-bore intravenous lines are placed, even when the patient is not hypotensive, so that fluids can be rapidly introduced if necessary. Particularly in zone II, hemorrhage or an expanding hematoma will respond to direct pressure and should not be managed by indiscriminate clamping through the wound. Under no circumstances should a penetrating neck wound be probed because clot dislodgement and uncontrollable bleeding can occur. Arrange blood and inform the theatre.

CLINICAL FEATURES:

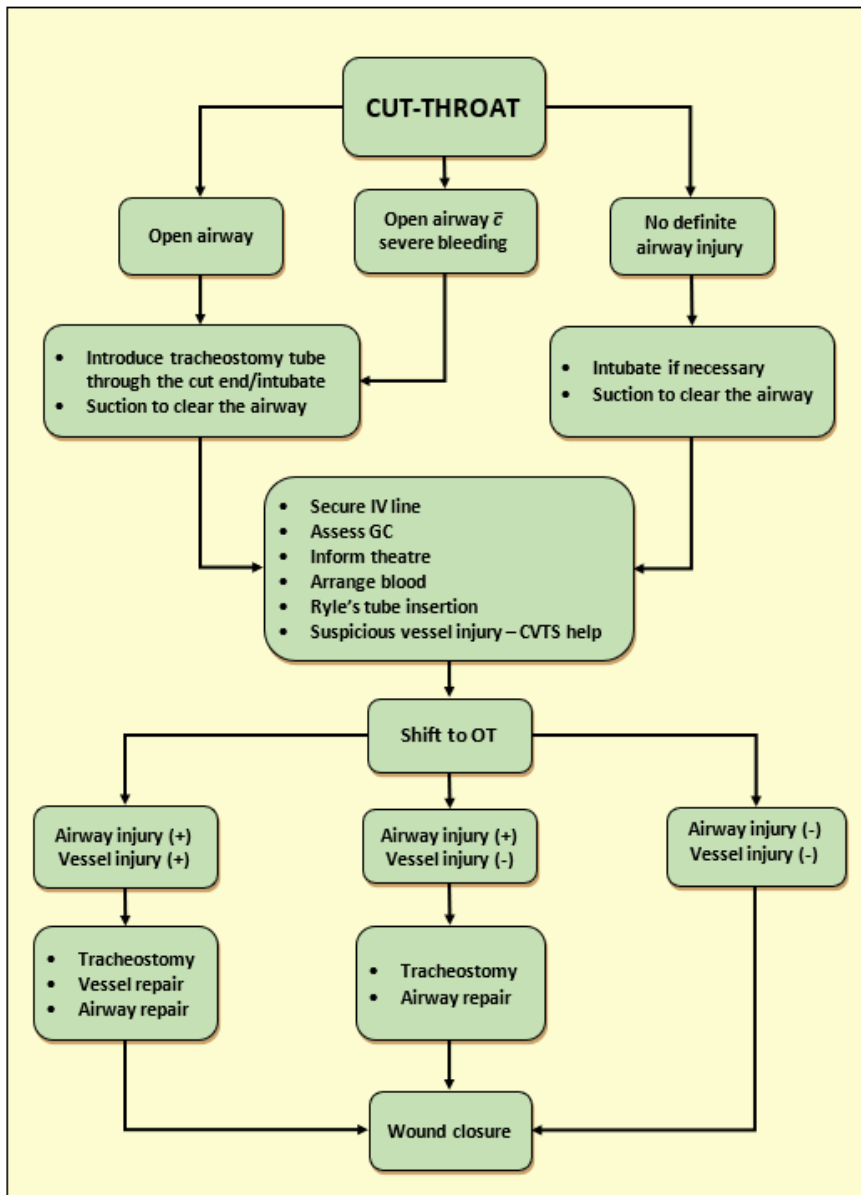
Diagnosis	Signs and symptoms
Airway injury	Respiratory distress, Stridor, Haemoptysis, Hoarseness, Tracheal deviation, Subcutaneous emphysema, Sucking wound
Vascular injury	Hematoma, Persistent bleeding, Neurologic deficit, Absent pulse, Hypovolemic shock, Bruit, Thrill, Change of sensorium
Nerve injury	Hemiplegia, Quadriplegia, Coma, Cranial nerve deficit, Change of sensorium, Hoarseness
Pharynx/oesophagus injury	Subcutaneous emphysema, Dysphagia, Odynophagia, Hematemesis, Haemoptysis, Tachycardia, Fever

DEFINITIVE MANAGEMENT

Obtain brief history regarding the mechanism of injury. Physical examination should include palpation for any thrills or bruit, neurologic examination, examination of chest, abdomen and extremities to rule out associated injuries. The signs of immediate life-threatening injuries include massive bleeding, expanding hematoma, nonexpanding hematoma in the presence of hemodynamic instability, hemomediastinum, hemothorax, and hypovolemic shock which require immediate mandatory exploration. If there is an airway injury, tracheostomy and repair. Seek CVTS assistance if there is a vessel wall injury.

If not treated in time, they may lead to death of the patient due to asphyxia and hemorrhage. Prevention of these complications depends on immediate resuscitation by securing the airway by tracheostomy or intubation, prompt control of haemorrhage and blood replacement⁵.

Flow chart on management of cut-throat



NECK SPACE INFECTIONS

SCOPE

This guideline is applicable to all medical personnel involved in the management of deep neck space infections. It includes general management strategy and detailed management protocols for the following conditions of peritonsillar abscess, Ludwig's angina, retropharyngeal and parapharyngeal abscess.

DEFINITION

Deep neck space infection refers to infection in the potential spaces and fascial planes of neck. The management of patients with deep neck space infections comprises early diagnosis and timely intervention to prevent life threatening complications.

INTRODUCTION

Deep neck space infection remains a common but challenging condition for otorhinolaryngologists and should be treated on an emergency basis. The complex anatomy makes diagnosis and precise localization of deep neck infections difficult. Common infections that we encounter include peritonsillar abscess, Ludwig's angina, retropharyngeal and parapharyngeal abscess. Most common primary sources are dental caries, retained foreign body, tonsillitis, infections of salivary glands etc. Usually, these infections are polymicrobial in nature. Potentially life-threatening complications have been reported to occur at a rate of 10-20%. Many vital organs are at risk of being involved and the resultant respiratory, vascular, neurologic and systemic complications endanger the life of the patient. Treatment includes broad spectrum antibiotics, airway securing, surgical drainage and eradication of sources of infection. Proper diagnosis and prompt management can effectively overcome the disease and provide a cure without complications.

CARE PATHWAY

Assessment of patient

Primary care centre:

Primary care doctors should be vigilant enough to suspect deep neck space infections in patients presenting with symptoms such as fever, throat pain, dysphagia, dyspnea with or without neck swelling. Baseline investigations like complete blood count and x-ray can be done here depending on availability. Empiric antibiotic therapy and supportive measures can be initiated here.

Referral criteria

Patients with breathing difficulty and deteriorating general condition should be referred to a higher centre immediately after initiating necessary primary measures.

All patients should be referred to a higher centre at the earliest if the diagnostic and

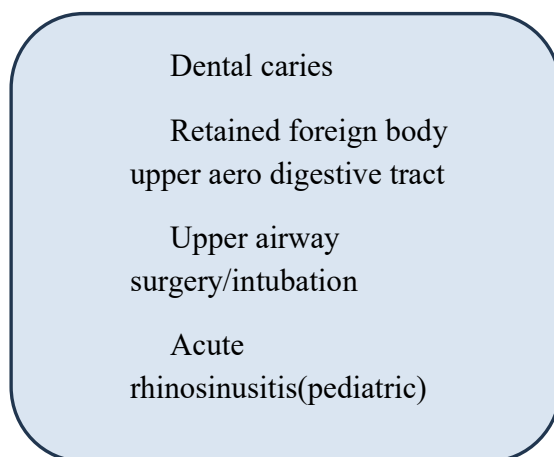
treatment facilities are not available in the primary care centre.

Secondary and tertiary care centre:

- Proper history and clinical examination give clues about location of inflammatory process as well as its potential severity.
- Clinical presentation

Fever, odynophagia, dysphagia, airway compromise	Retropharyngeal abscess
Fever, drooling, odynophagia, hot potato voice	Peritonsillar abscess
Fever, swelling below angle of jaw, dysphagia ± dyspnoea	Parapharyngeal abscess
Fever, pain and swelling submandibular area, odynophagia ± dyspnoea	Ludwig's angina

- Etiological factors

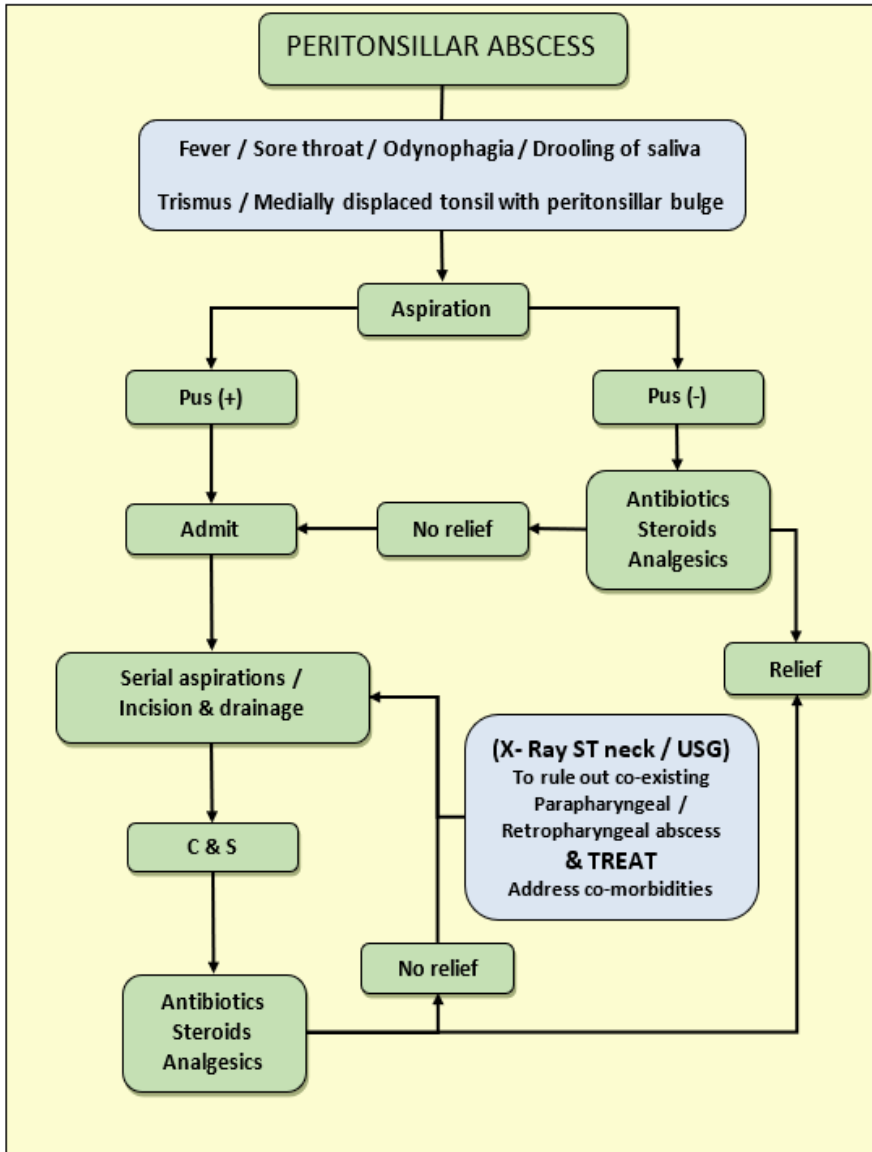


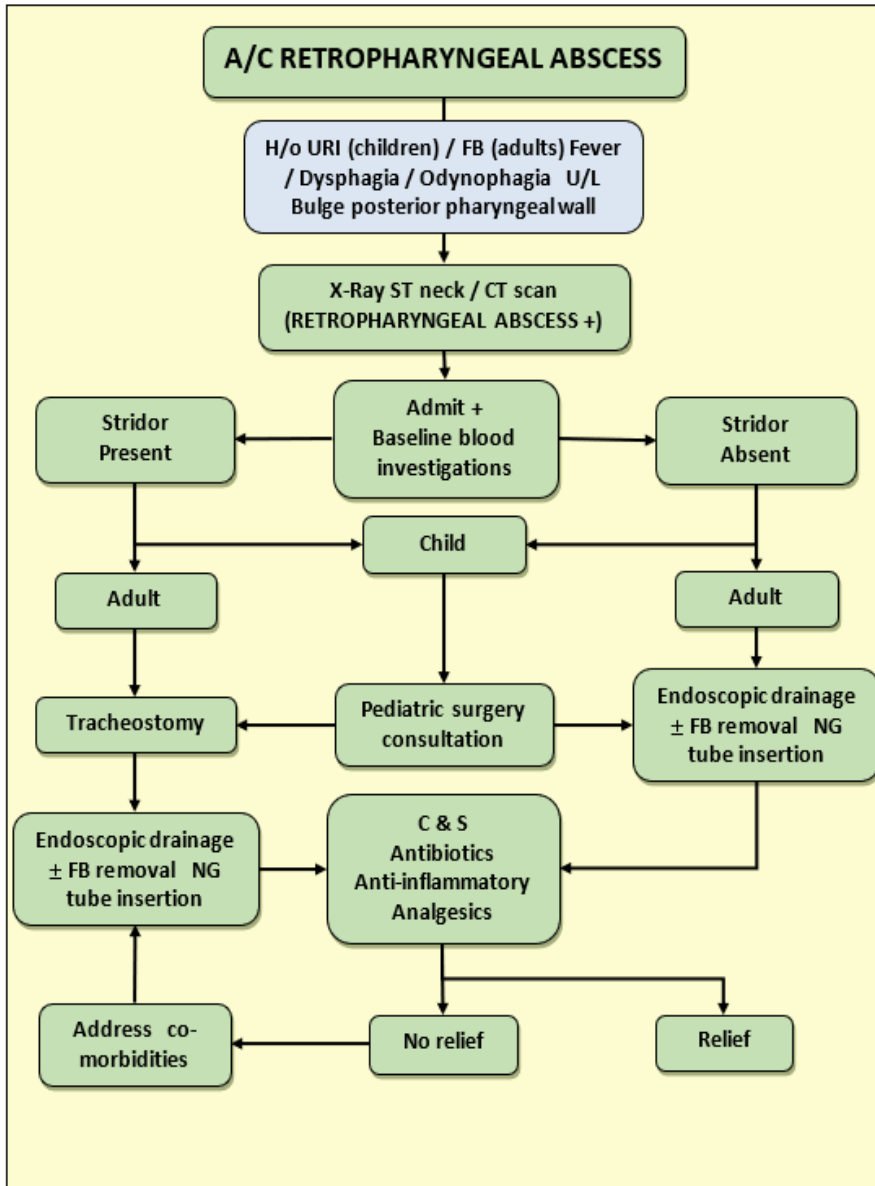
- Medical history: Diabetes mellitus/ other immune deficiency conditions / drug allergy.
- General examination, head, and neck & systemic examination
- **Investigations:**
 - Base line blood investigations including Complete blood count, ESR, Coagulation profile, Blood sugar level, Serum electrolytes, Renal function tests and Screening.

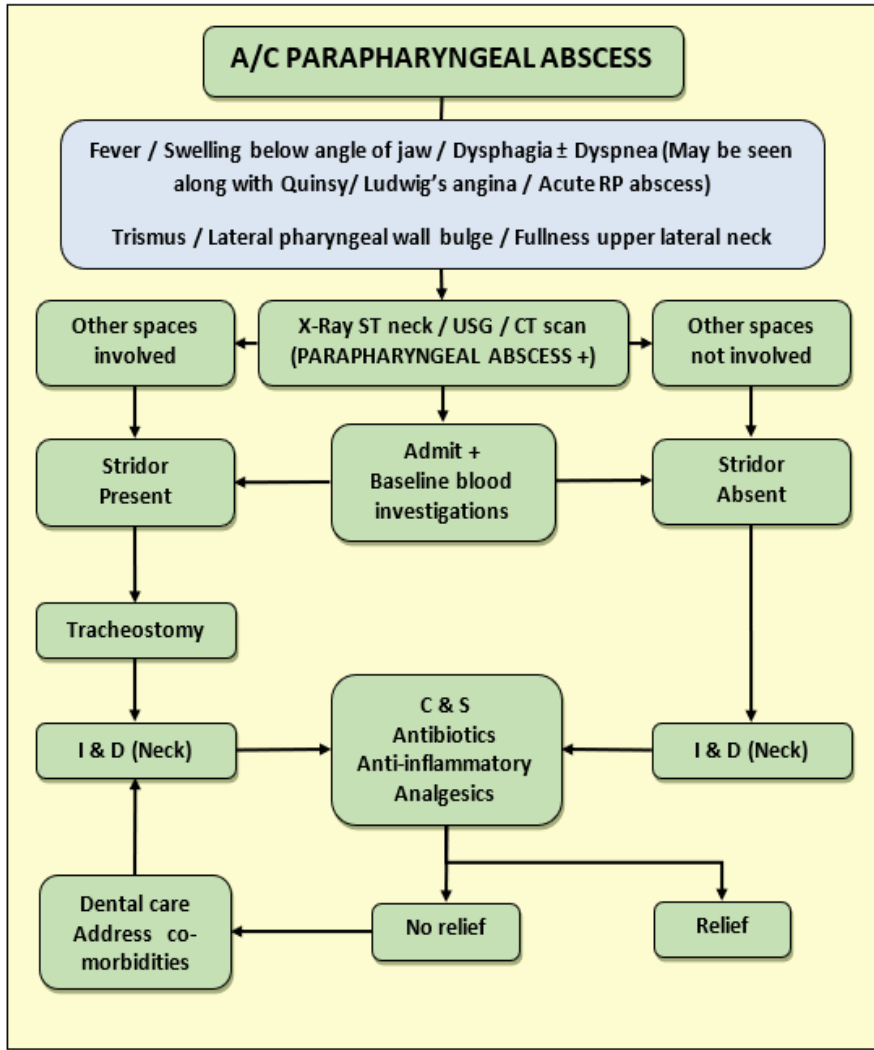
- X ray soft tissue neck lateral view for evaluation of upper aero- digestive tract and airway compromise. The presence of air fluid level and prevertebral widening indicates retropharyngeal abscess.
- X ray chest PA view to rule out any signs of aspiration or mediastinitis
- Ultrasonography of the neck is helpful in cases of Ludwig's angina and parapharyngeal space involvement, but it has a limited role in deep spaces like retropharyngeal space involvement.
- A CECT provides information about the neck spaces involved, their extent and complications.
- Pus culture and sensitivity if possible.
- Dental (caries teeth), paediatric surgery (acute retropharyngeal abscess in children) and CVTS (mediastinal involvement) consultations if needed.

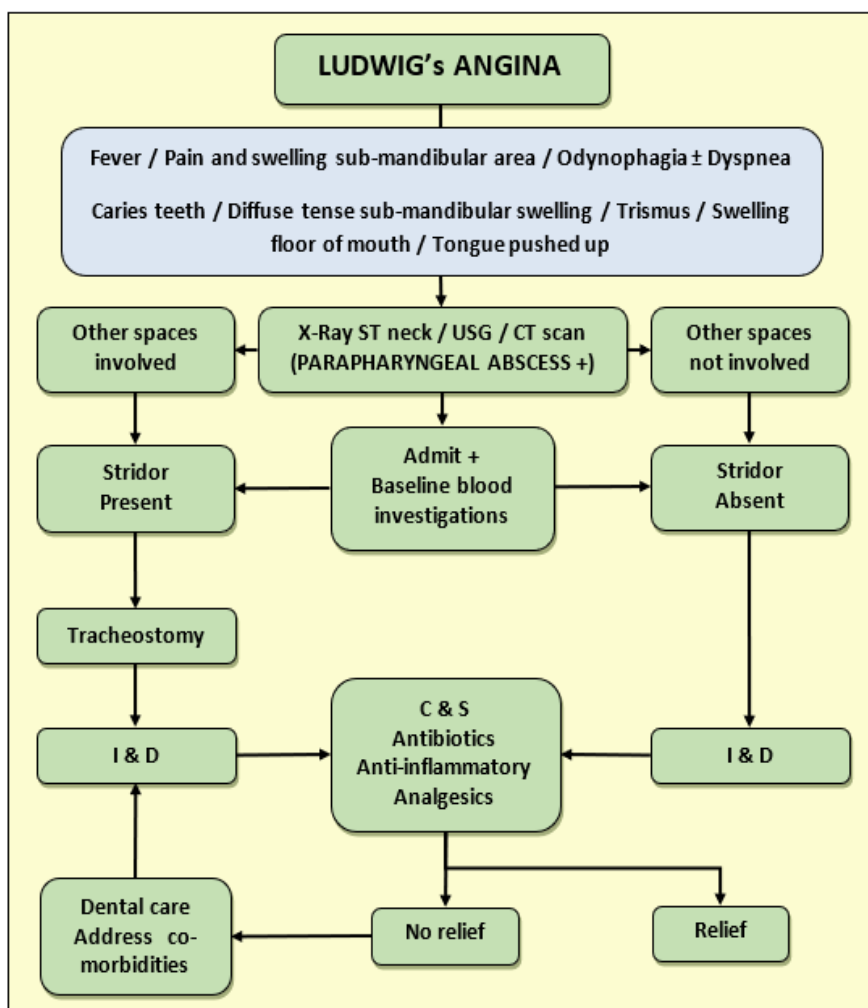
TREATMENT

- All patients need admission.
- Airway complication should be anticipated in all cases.
- Initial management of any patient with known or suspected deep neck space infection is securing the airway. Secure an IV access and give supportive care, broad spectrum antibiotics, steroid and analgesics.
- Then proceed with surgical drainage of the abscess depending on the spaces involved and do tracheostomy if needed.
- Primary source of infection should be eradicated.
- "If patient is clinically stable and otherwise healthy with abscess cavities less than 2.5cm in diameter and involving a single neck space, a 48 - 72-hour trial of empiric IV antibiotic therapy is appropriate" (Cumming's 6th edition)
- Approaches to different neck space infections are given below.









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STRIDOR

SCOPE

This guideline is applicable to all medical staff involved in management of stridor. This guideline will act as a reference in the hospitals and primary care centres, and it includes the general management strategy, detailed management protocol for stridor and the general guidelines on the primary care or ambulatory care of stridor.

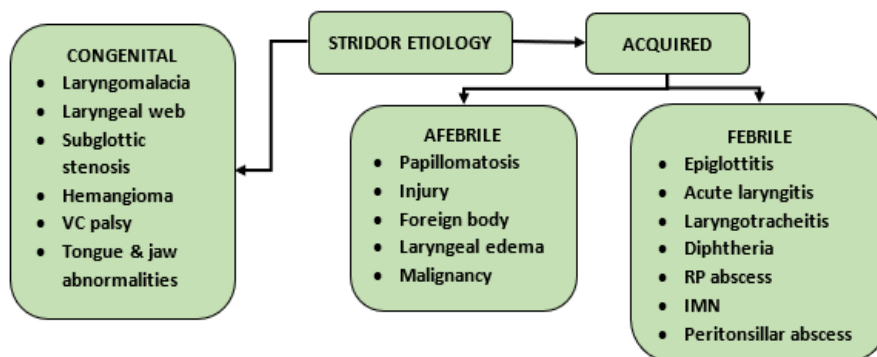
INTRODUCTION

Stridor is noisy respiration produced by turbulent airflow through narrowed air passages. It may be heard during inspiration, expiration or both.

Inspiratory stridor: obstructive lesions of supra-glottis or pharynx, e.g. laryngomalacia, retropharyngeal abscess.

Expiratory stridor: lesions of thoracic trachea, primary or secondary bronchi, e.g. bronchial foreign bodies, tracheal stenosis.

Biphasic stridor: lesions of glottis, sub-glottis, cervical trachea, e.g. laryngeal papillomas, VC palsy, subglottic stenosis



CARE PATHWAY

Primary care centres:

Primary care doctors face the challenge of dealing with stridor from any causes listed above due to unavailability of facilities to secure airway. Hence all cases are to be referred to higher centres.

Secondary care:

History & Examination:

First, confirm the noisy breathing is stridor and record vitals including SpO₂. Secure an IV line and start O₂ inhalation. Intravenous steroids should be given along with nebulization

with steroids.

Meanwhile a proper brief history should be taken to identify the cause of stridor and should include the following:

- Onset & duration
- Similar episodes in the past
- Foreign body ingestion / Anaphylaxis
- Under influence of alcohol/cannabis/sedatives
- Associated symptoms – hoarseness, dysphagia.
- History of COPD
- History of surgery/intubation/trauma
- Treatment history in the form of radiation /chemotherapy.

A quick examination is to be done to assess the neck, chest and airway. Look for swellings, dilated veins & scars over the neck. Assess the laryngeal framework, look for widening & CVC. If stable, perform an indirect laryngoscopy examination to assess the glottic space. Auscultate chest to look for any adventitious sounds.

If the patient improves, continue close monitoring of vitals & supportive management. If it is not improving, plan to secure airway according to level of obstruction.

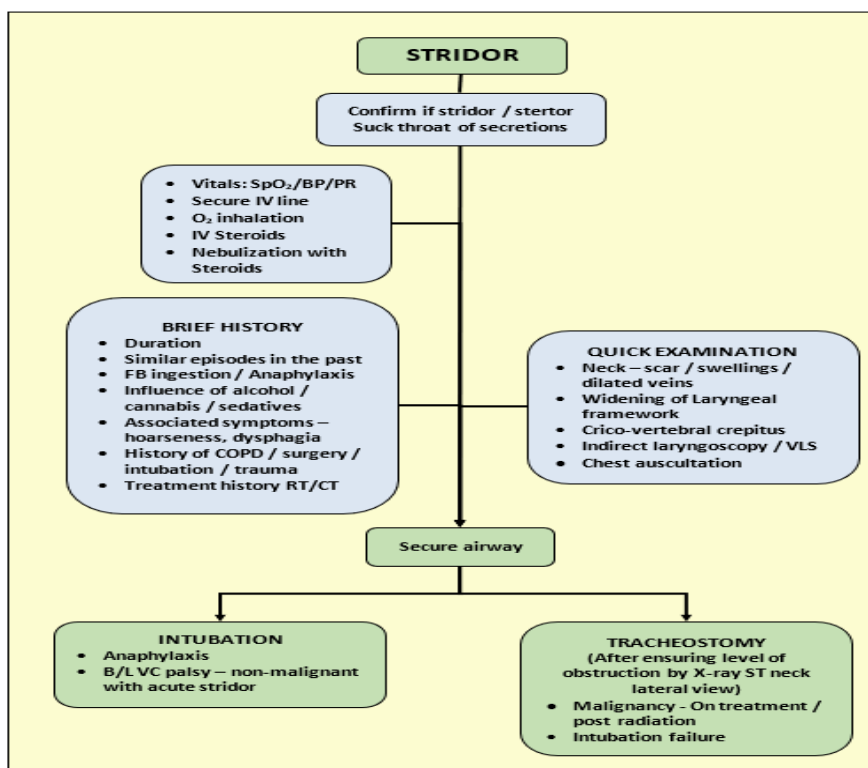
Referral criteria

If facilities for intubation or tracheostomy are not available, patients should be referred immediately.

Tertiary care:

In addition to the initial measures listed above, X-ray Soft tissue neck lateral view may be taken. The cause of stridor should be identified and managed by airway intubation or tracheostomy.

Flow chart of management of stridor



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