

**MANAGEMENT OF
RESPIRATORY TRACT INFECTIONS
IN CHILDREN**

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Introduction

Respiratory tract infections are the main cause of morbidity and mortality in children, especially in the developing world. According to the World Health Organization reports they are responsible for 1.6 – 2.2 million deaths globally in children under 5 years old. Pneumonia is one of the main causes responsible for the above deaths. They occupy most of the consultation time in primary care as well as in hospital care settings. The management of these illnesses and their complications demands more human hours and improved facilities. The cost of them represent a bigger proportion of the total money spent in the health budget.

There are many controversial issues related to the management of respiratory tract infections in children. The criteria to admit to a hospital, investigations to be done, need of antibiotics and steroids are some of areas which need clarification. At the same time, the use of antihistamines and nasal decongestants are done without much insight.

The epidemiology of some of the respiratory tract infections varies according to the regions. The use of antibiotics policies and their resistance patterns varies among institutions.

Facilities available become limited at district and base hospitals, so too the trained staff. As a developing country, the health sector in Sri Lanka should possess farsighted health policies to overcome the above obstacles.

Considering the above factors, preparation of guidelines for the management of respiratory tract infections is a timely intervention to improve patient care. It will facilitate medical officers to make clinical decisions which are supported by evidence based medicine. Clinical audits can be done to standardise the treatment provided in health institutions.

It is our sincere hope our efforts would be contributory, in achieving quality paediatric care for all children in Sri Lanka in future.

CHAPTER 1

PREPARATION OF GUIDELINES AND BEST EVIDENCE

We were given the daunting task of preparing these respiratory guidelines for the paediatric age group. We reviewed some of the published guidelines in reputed journals and web sites. After discussion, questions were formulated relevant to the grey areas and we searched for the best quality evidence although they were not exhaustive systematic reviews. We did our best to provide practical, evidence based, guidelines which can be applied in most of the clinical settings in Sri Lanka without much difficulty.

Following boxes contains the grades of recommendations and level of evidence published by the Scottish Intercollegiate Guidelines Network (SIGN); 2000 . We hope it will help you to assess the strength of our recommendations.

Levels of Evidence

- 1++ : High quality meta-analyses, systematic reviews of randomised controlled trials (RCTs), or RCTs with a very low risk of bias
- 1+ : Well-conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias
- 1- : Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias
- 2++ : High quality systematic reviews of case control or cohort studies. High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal
- 2+ : Well-conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal
- 2- : Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal
- 3 : Non-analytic studies, e.g. case reports, case series
- 4 : Expert opinion

Grades of Recommendations

- A** - At least one meta-analysis, systematic review of randomised controlled trials (RCTs), or randomised controlled trial rated as 1++ and directly applicable to the target population; or A body of evidence consisting principally of studies rated as 1+, directly applicable to the target population, and demonstrating overall consistency of results

- B** - A body of evidence including studies rated as 2++, directly applicable to the target population, and demonstrating overall consistency of results; or

Extrapolated evidence from studies rated as 1++ or 1+

- C** - A body of evidence including studies rated as 2+, directly applicable to the target population and demonstrating overall consistency of results; or

Extrapolated evidence from studies rate as 2++

- D** - Evidence level 3 or 4; or

Extrapolated evidence from studies rated 2+

CHAPTER 2

COMMON COLD

Common cold is a *viral illness* in which the symptoms of rhinorrhoea and nasal obstruction are prominent and **systemic symptoms and signs such as myalgia and fever are absent or mild.**

Rhinoviruses are the most common pathogens. Others are adenovirus, parainfluenza virus and respiratory syncytial virus.

Spread is by physical contact and air borne droplets. Common cold is **self limiting**, and usually lasts for 7-8 days, but may persist up to two weeks in 10% of patients.

Clinical Manifestations

There are *four* distinct clinical stages

1. **Prodromal stage:** Lasts for a few hours. Experiences feeling of tickling sensation of the throat.
2. **Stage of irritation:** Mucous membrane will become red. A watery discharge is present (rhinorrhoea) and it will last for 1-4 days with nasal obstruction and sneezing. Cough is associated in only 30% of cases.
3. **Stage of secondary infection:** after two to three days the mucosa becomes oedematous and bluish with more nasal obstruction and thick mucopurulent secretions.
4. **Stage of resolution:** Complete recovery occurs.

Management

Management of common cold is primarily symptomatic.

1. Nasal obstruction

- Topical vasoconstrictor agents like oxymetazoline and xylometzoline are **not** recommended below two years of age.
- Even if indicated in those over 2 years of age, prolonged use should be avoided (preferably, less than *seven days* to avoid rebound phenomena).

2. Rhinorrhoea

- The first generation antihistamines (sedating antihistamine) reduce rhinorrhoea by 25 - 30%.

e.g. Chlorpheniramine maleate: oral
Child 1 month- 2years 1mg every 12 hours
Child 2-6 years 1mg every 6 hours
Child 6-12 years 2mg every 6 hours

3. Cough

- Cough suppressives are generally not necessary
- Antibiotics are not indicated.

Use of paracetamol is ***not routinely*** recommended in an ***uncomplicated common cold***.

Vitamin C, inhalation of warm humidified air (steam inhalation), guaifenesin have no proven benefit.

Complications

Usually complete resolution without complications occurs. However, if the body resistance is low, secondary invasion by other organisms may occur.

1. Otitis media (5 -30%)
2. Acute sinusitis (5 -13%)
3. Pneumonia
4. Exacerbation of asthma

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CHAPTER 3

BACTERIAL SINUSITIS

Definition

Sinusitis is infectious or non infectious inflammation of one or more sinuses. The inflammation can be caused by infectious (bacterial, viral, fungal) or non infectious (allergic) triggers.

Classification of bacterial sinusitis

Acute bacterial sinusitis;

Infection lasts *less than four weeks* and symptoms resolve *completely* with treatment (duration range 10-30 days)¹

Subacute bacterial sinusitis

Infection lasting *four to twelve weeks* yet resolves *completely* with treatment.

Recurrent acute bacterial sinusitis

Episodes last *less than four weeks* and are separated by intervals of at least ten days during which the patient is totally free of symptoms.

Chronic bacterial sinusitis

Symptoms last more than *twelve weeks* with or without treatment.

Differentiation between acute and chronic sinusitis has clinical significance.

Rhinitis

It is the inflammation of the nasal mucosa. The most common causes are viral or allergy.

Rhinosinusitis

Inflammation of the nasal mucosa and the lining of the sinuses.

Rhinitis and Rhinosinusitis are often misdiagnosed as sinusitis.

An understanding of the development of the paranasal sinuses is important

1. Maxillary and ethmoidal sinuses are present at birth.
2. Sphenoidal sinuses develop by the age of five to six years.
3. Frontal sinus is the last to develop at eight to ten years of age.

Goals of these guidelines

- To increase the accuracy in the diagnosis of bacterial sinusitis.
- To optimize the management of sinusitis and decrease the duration for recovery.
- To reduce antibiotic usage in ill-defined upper respiratory tract infections.
- To optimize the appropriate use of laboratory investigations and timely referral.

Aetiology and pathogenesis

The majority of cases follow a viral upper respiratory tract infection which involves whole upper respiratory epithelium including the paranasal sinuses. Such infections cause hyperaemia and oedema of the mucosa, which blocks the ostia. There will be a cellular infiltration and an increase in mucous production. The infection will also paralyze the cilia, leading to stasis of secretions predisposing to secondary bacterial infection.

The usual bacteria

Streptococcus pneumoniae 30%
Haemophilus influenzae 20%
Moraxella catarrhalis 20%,
Streptococcus pyogenes (occasionally)
Staphylococcus aureus (occasionally)

Predisposing factors

Local

- Pre-existing rhinitis
(viral respiratory infections, allergic, intrinsic factors)
- Nasal foreign body
- Upper respiratory tract infections
(tonsillitis, adenoiditis)
- Nasal anatomical variations
(septal deviations, abnormal turbinates)
- Gastro-oesophageal reflux
Exposure to cigarette smoking (air pollution)

General

- Immunocompromised host
- Mucocilliary disorders
(Kartagener syndrome)

Clinical Manifestations

Symptoms

- Common symptoms : Nasal congestion and discharge
Fever
Cough
- Less common symptoms : Bad breath (halitosis)
Decreased sense of smell
Periorbital oedema.
- Rare symptoms : Headache
Facial pain

Sinusitis may present with nonspecific complaints.

Signs

- Mild erythema in the nasal mucosa
- Swelling of the nasal mucosa with nasal discharge
- Sinus tenderness may be detected in adolescents
- Anatomical anomalies (deviated nasal septum, polyp, large turbinate)

Diagnosis

Diagnosis is mainly dependant on the clinical picture.¹

Differentiation of bacterial sinusitis from viral upper respiratory tract infection is largely determined by duration and severity of symptoms.² Persistent symptoms of upper respiratory tract infections without improvement after 10-14 days is more suggestive of bacterial sinusitis.²

Symptoms of acute sinusitis usually disappear within two to four weeks.

In case of persisting symptoms, one should suspect:

- Complications of acute sinusitis,
- Subacute sinusitis
- Chronic sinusitis

Facial pain alone is not diagnostic of bacterial sinusitis in children^{2,3}

Investigations

An elevated white cell count and ESR are indicative of an acute infection.³

Although the gold standard is aspiration of sinus contents, this procedure is not routinely recommended because of its invasive nature.

Where possible pus from the nose should be cultured and blood cultures should be taken if there are *systemic features with a toxic appearance*.

Nasopharyngeal cultures are NOT recommended due to poor correlation with sinus pathogens.

Plain sinus X-rays and CT scans are *not routinely recommended* in the diagnosis of sinusitis.¹

CT scan is recommended for¹

- Complications of acute sinusitis
- Chronic sinusitis not responding to treatment,
- Severely ill patients where the diagnosis is suspected but not clear.

Consider assessment for allergy in patients with chronic sinusitis.²

Management

The aims of treatment are

- to resolve and limit the course of the acute infection
- to prevent complications
- to correct any precipitating factors.

Studies indicate that up to 60% of cases of acute sinusitis will resolve spontaneously without antibiotics.³

Acute sinusitis

Pain /Fever may be controlled with oral analgesics/antipyretics. (z)

Paracetamol 10-15mg/kg/dose 6H
Ibuprofen 5-10 mg/kg/dose 8H

Following adjunctive therapy are not supported by evidence in the treatment of *acute* bacterial sinusitis and are therefore not recommended.^{1,3}

- Instillation of nasal cavities with normal saline
- Inhalation of steam
- Topical or systemic decongestants

Antihistamines have no proven benefit in acute bacterial sinusitis, but might have a role in chronic bacterial sinusitis where a clear allergic component is demonstrated.

Antimicrobial therapy ^{1,2,3}		
First line agent in the treatment of acute bacterial sinusitis in children		
Recommended therapy and dose	Duration	Comments
<i>Standard dose</i> Amoxicillin 40mg/ kg/day PO div 8H(45mg/kg/day)Or	10days	<ul style="list-style-type: none"> Amoxicillin retains best coverage of oral B-lactam agents against <i>Streptococcus pneumoniae</i> (including intermediate strains)
<i>High Dose</i> Amoxicillin 90mg/ kg/day PO div 8H	10days	<ul style="list-style-type: none"> Higher dose (90mg/kg/day) might be considered in high risk children, recent (<03months) antibiotic exposure and day care centre attendance)
Clarithromycin 15mg/kg/day bid	10 days	<ul style="list-style-type: none"> Can be used in β-lactam allergy
Azithromycin 10mg/kg/day	1day	
5mg/kg/day	4days	

Agents not routinely recommended in acute bacterial sinusitis

- Cephalexin**
 - Poor activity against penicillin intermediate/resistant *Streptococcus pneumoniae*
 - No activity against *Haemophilus/ Moraxella*
- Cefaclor**
 - Poor activity against penicillin intermediate/resistant *Streptococcus pneumoniae*
 - Marginal activity against *Haemophilus*
- Cefixime**
 - Poor activity against penicillin intermediate/resistant streptococcus pneumoniae
 - Excellent activity against *Haemophilus*
- Ceftriaxone**
 - Routine use of this agent is not recommended in sinusitis due to potential for increased resistance to third generation cephalosporins
 - May be an option in severe cases who have failed therapy.
 - Three days of IM/IV therapy are recommended. (Single dose not as effective in eradicating penicillin resistant *Streptococcus pneumoniae*)
- Erythromycin**
 - Poor activity against *Haemophilus/Moraxella*
- Clindamycin**
 - Not routinely recommended for acute bacterial sinusitis
 - No activity against *Haemophilus / Moraxella*
- Ciprofloxacin**
 - Sub optimal coverage of *Streptococcus pneumoniae*

Second line agents in the treatment of acute bacterial sinusitis in children

Recommended therapy	Duration	Comments
<p>Amoxicillin-clavulanate 40mg/kg/day per orally, divided doses, 8 hourly (based on Amoxicillin)</p> <p><i>plus</i></p> <p>Amoxicillin 40mg/kg/day, per orally, divided doses, 8 hourly</p>	10 days	<p>Adding amoxicillin-clavulanate (Co amoxiclav) to Amoxicillin is needed to,</p> <ul style="list-style-type: none"> • provide coverage for penicillin resistant intermediate <i>S.pneumoniae</i> and β-lactamase producing organisms. • allow an increased dose of amoxicillin without an increased dose of clavulanate, thus avoiding side effects (especially diarrhoea) <p>Note:</p> <ul style="list-style-type: none"> • If patient did not respond to high dose amoxicillin treatment, amoxicillin-clavulanate alone is adequate to cover β-lactamase producing organisms. • ratio of amoxicillin to clavulanate should be 7:1 • due to time dependent killing of amoxicillin,tid dosing is recommended.
<p><i>or</i></p> <p>Cefuroxime axetil 40mg/kg/day, per orally, divided doses, 12 hourly</p> <p><i>or</i></p> <p>Cefpodoxime 10mg/kg/day, per orally, divided doses, 12 hourly</p>	10 days	<ul style="list-style-type: none"> • provides best coverage of all oral cephalosporins against penicillin resistant intermediate strains of <i>S.pneumoniae</i> and provides good coverage of Haemophilus <i>Moraxella / S.aureus</i>.
<p><i>or</i></p> <p>Azithromycin 10mg/kg, per orally, daily 5mg/kg, per orally, daily</p> <p><i>or</i></p> <p>Clarithromycin 15mg/kg/day, per orally divided doses, 12 hourly</p>	1 day 4 days 10 days	<ul style="list-style-type: none"> • Macrolides use should be restricted because they are less efficacious against <i>H.influenzae</i> and <i>S.pneumoniae</i> than amoxicillin clavulanate.

Recurrent sinusitis

Management is similar to acute bacterial sinusitis.

If the duration between two episodes is less than 6 weeks, consider second line antimicrobial agents and if it is 6 or more weeks first line antibiotics are indicated.

Chronic sinusitis

Adjunctive therapy is as important as antibiotic therapy.

Topical nasal steroids may be of benefit.

Oral and topical decongestants are of limited benefit.

Instillation of nasal cavities with normal saline might be of benefit.

Antihistamines have a role where a clear allergic component is demonstrated.

A prolonged course of antibiotics has some value in the treatment of chronic sinusitis.

Prolonged use of topical decongestants for more than five days may result in local complications and is not recommended.

Complications of sinusitis

- Periorbital /orbital cellulitis
- Meningitis
- Intracranial abscess
- Intracranial venous thrombosis
- Sepsis

Follow up

A follow up examination at the completion of treatment is not routinely recommended.

If patient shows no improvement after 72 hours following treatment with adjunctive therapy and the first line antibiotics, consider second line agents in the treatment.

If there is deterioration at any time patient should be reassessed for: ²

- Acute complication of sinusitis
- Other diagnoses
- Adherence to treatment

Indications for referral to ENT Specialist

- Sinusitis unresponsive to medical therapy after a 3 week trial of a second line agent and a full course of nasal steroid therapy with evidence of disease on the sinus CT scan.
- Recurrent sinusitis - 3 or more episodes in a 6 month period despite adequate medical treatment as outlined above and evidence of disease on the sinus CT scan.
- Patient with known immune compromise or ciliary motility problem.
- Orbital or cranial complications of sinus infections.
- Recurrent nasal polyps unresponsive to medical therapy and evidence of disease on the sinus CT scan.
- Any evidence of tumour noted on examination or CT.

Background Notes

- It may be difficult to distinguish children with uncomplicated viral upper respiratory infections or adenoiditis from those with an episode of acute bacterial sinusitis.² Most viral infections of the upper respiratory tract involve the nose and the paranasal sinuses (viral rhinosinusitis).^{3,4}
- Bacterial sinusitis does occur rarely in children less than 1 year of age, their exclusion reflects, in part, the difficulty in conducting clinical investigation in this age group. This is a consequence of the small size of the paranasal sinuses and the difficulty in safely performing sinus aspiration.⁴
- Accordingly, the gold standard for the diagnosis of acute bacterial sinusitis is the recovery of bacteria in high density ($>10^4$ colony-forming units/ml) from the cavity of a paranasal sinus. Although sinus aspiration is the gold standard for the diagnosis of acute bacterial sinusitis, it is an invasive, time-consuming, and potentially painful procedure that should only be performed by a specialist (otolaryngologist). It is not a feasible method of diagnosis for many.⁴

- It is recommended that the diagnosis of acute bacterial sinusitis be based on clinical criteria in children less than 6 years of age who present with upper respiratory symptoms that are either persistent or severe.
- The ethmoid and maxillary sinuses form in the third to fourth gestational month and, accordingly, are present at birth. The sphenoid sinuses are generally pneumatized by 5 years of age; the frontal sinuses appear at age 7 to 8 years but are not completely developed until late adolescence.
- Bacterial infections of the paranasal sinuses do not usually involve the nose.
- Although bacterial sinusitis does occur rarely in children less than 1 year of age, their exclusion reflects, in part, the difficulty in conducting clinical investigation in this age group.

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CHAPTER 4

ACUTE PHARYNGITIS

Introduction

Upper respiratory infection is a common problem. One third of such illnesses feature sore throat (pharyngitis) as the primary illness.

Aetiology

There are *infectious* and *non infectious* causes

Infectious causes

1. Viruses
2. Group A beta haemolytic *Streptococcus* (GABHS) accounts for 15-30% of isolates in children ^{1,3,4}
3. Others (uncommon)
 - a. Group C *Streptococcus*
 - b. *Francisella tularensis*
 - c. *Mycoplasma pneumoniae*
 - d. *Neisseria gonorrhoea*
 - e. *Corynebacterium diphtheriae*

Non infectious causes:

- Kawasaki disease (look for the characteristic features like periungual desquamation, bulbar conjunctivitis, pleomorphic rash etc.)
- Gastro esophageal reflux
- Postnasal drip secondary to rhinitis
- Persistent cough
- Thyroiditis, allergies
- A foreign body
- Exposure to smoke ^{1,2,5}

Transmission of typical viral and Group A beta haemolytic *Streptococcus* (GABHS) pharyngitis occurs mostly by **hand contact with nasal discharge**, rather than by oral contact.^{1,2}

The guidelines for the management of pharyngitis will mainly focus on infectious rather than the non infectious causes.

Streptococcal pharyngitis is uncommon before 2-3 years of age.^{6,7}

Colonization with GABHS could be asymptomatic or can cause an acute illness.

M protein is the major virulent factor of GABHS; thus, type specific immunity will develop later.

Clinical Manifestations

Rapid onsets of symptoms are suggestive of bacterial infection.

Symptoms

- Fever
- sore throat
- headache
- Gastrointestinal symptoms (seen commonly)

Signs

- Pharynx is red
- Enlarged tonsils may be covered with yellow blood tinged exudates
- Petechiae are seen over the soft palate and pharynx
- Uvula may be red and swollen
- Anterior cervical lymph nodes (mainly jugulodigastric nodes) are tender and swollen

Viral pharyngitis onset may be gradual.

Pharyngoconjunctival fever is due to an ***adenovirus***. There is associated conjunctivitis.

Coxsackie pharyngitis (herpangina) may produce small 1-2 mm greyish vesicles and punched out ulcers in the posterior pharynx.

Epstein - Barr virus (EBV) pharyngitis is associated with, *tonsillar enlargement and exudates, cervical lymph nodes enlargement, hepatosplenomegaly, rash* and fatigue.

Clinical Diagnosis

The clinical presentations of streptococcal and viral pharyngitis show considerable overlap.

Pharyngitis is more likely to have a bacterial aetiology in the absence of rhinorrhoea, cough and conjunctivitis. ^{7,8}

Throat culture remains the gold standard for diagnosis of streptococcal pharyngitis.⁶

False positives are due to misidentification of other organisms.⁶

False negatives are due to either *inadequate throat swab specimen* or *use of antibiotics prior to culture*.⁶

EBV infection may show lymphocytosis in the full blood count and atypical lymphocytosis in the blood picture. Though it is costly, EBV IgM antibodies are useful in diagnosing acute infection.

Treatment

Primary benefit of treating streptococcal pharyngitis is the prevention of acute rheumatic fever.⁶ Treating with appropriate antibiotics resolves symptoms within 24 hours.

The criteria to start early antimicrobial treatment ⁶

- a) A clinical diagnosis of scarlet fever
- b) Contact history of streptococcal sore throat
- c) Past history of acute rheumatic fever
- d) Recent history of acute rheumatic fever in the family

Antimicrobials 6, 8,9,10,11,12

1. Oral penicillin 250mg 8 hourly (x)
250mg – 500mg (adolescent) for 10 days
2. Oral amoxicillin (preferred in children due to good compliance)
750mg daily for 10 days OR
50mg /kg/day 12 hourly for 6 days
3. Clarithromycin 7.5mg/ kg / dose 12 hourly for 10 days
4. Azithromycin 10mg / kg / daily for 5 days
5. Benzathine penicillin IM single dose
600,000 units < 27 kg
1200,000 units > 27 kg

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CHAPTER 5

ACUTE TONSILLITIS

Definition

Inflammation of tonsil(s).

Causative organisms

Viruses are responsible for tonsillitis in over 40- 50% of cases.

Viruses

- *Influenza*
- *Parainfluenza*
- *Adenoviruses*
- *Enteroviruses*
- *Rhinoviruses*

Features that suggest viral origin^{1, 2, 3}

- Cough
- Conjunctivitis
- Coryza
- Diarrhoea

In many cases it is felt that an initial viral tonsillitis may predispose to secondary infection by bacteria.⁴

Bacteria

- Group A beta –haemolytic *Streptococcus* (15- 30%)³
- *Streptococcus pneumoniae*
- *Haemophilus influenzae*
- Anaerobic organisms

Clinical Manifestations

- There may be a prodromal illness with **pyrexia, malaise, and headache** which precedes the *predominant symptom* of sore throat by one day.

- Pain may radiate to ears or may occur in the neck due to **cervical lymphadenopathy**.
- Swallowing may be painful and the patient's voice may sound muffled.
- There may be **trismus** and **drooling of saliva**.
- Some children may have abdominal pain and, occasional vomiting.
- The tonsils are hyperaemic on examination with pus and debris in the crypts.
- Tender cervical lymphadenopathy involves particularly jugulodigastric group.

Clinical examination should not be relied upon to differentiate between viral and bacterial causes.

- Adenoviruses and other viruses can cause exudative tonsillitis.^{2,3}
- In two thirds of school-aged children with streptococcal tonsillitis there is no exudate.^{2,3}

Investigations

Investigations are not generally indicated.

However to exclude some differential diagnoses **full blood count, blood for Monospot and throat swab** may be helpful.

Indications for throat swab

1. Poor response to standard antimicrobial therapy within 48-72 hours
2. Presence of complications (eg. Peritonsillar abscess)

Management

Most of the cases diagnosed as tonsillitis do not need antibiotics since only 30% are bacterial in origin [A].⁵ But when the child presents with a severe clinical picture antibiotics may be prescribed empirically.

The mainstay of treatment is supportive.

Supportive therapy

- Antipyretics and pain relievers have proven effect [A]⁶
Paracetamol is the drug of choice [C]. Ibuprofen is an alternative [D].

Paracetamol 10- 15mg/Kg/dose 6 hourly
or
Ibuprofen 5-10mg/Kg/dose 6 – 8 hourly
- Gargling with warm salt water (in older children). Salt concentration should be as in normal saline.
- Local relief with lozenges which contain menthol. ⁴

Antimicrobial therapy ^{1,2}

Drug of choice is **oral penicillin. (x)**
250mg 6- 8h for **ten days** for children
250-500mg 6- 8h for **ten days** for adolescents.

A full 10 day course of penicillin orally is recommended even if symptomatic improvement is seen within 3-4 days.⁷

Oral amoxicillin is preferred for children because of taste and availability of chewable tablets.

It is advised to avoid ampicillin or amoxicillin to treat acute tonsillitis in case the patient has infectious mononucleosis (glandular fever), where a generalized maculopapular rash may develop in 80%.^{1,2}

Amoxicillin 750mg daily x 10 days OR
Amoxicillin 50mg /Kg/day divided tid x 10 days.

Alternative treatment in case of penicillin allergy, macrolides are preferred.

Erythromycin - 40mg /Kg/day divided, tds x10 days
Azithromycin - 10mg /Kg/day x 5 days, good compliance when a single dose is used
Clarithromycin 7.5 mg/kg/dose 12 hourly for 10 days
Cephalexin 50mg/kg/day 12 hourly for 10 days

Because of infectiousness the child should be isolated from day care or school for one day after the onset of antibiotic treatment. ⁸

Complications

Local

- Severe swelling causing respiratory obstruction (stridor)
- Abscess formation; Peritonsillar(quinsy)
Parapharyngeal
Retropharyngeal
- Acute otitis media
- Recurrent acute tonsillitis (chronic tonsillitis)

General

- Septicaemia
- Meningitis
- Acute rheumatic fever
- Acute glomerulonephritis.

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CHAPTER 6

CHRONIC TONSILLITIS

Chronic inflammation of the tonsil(s) usually starts as an acute inflammation.

Clinical features

Symptoms

- Repeated attacks of sore throat with imperfect remission indicates chronic inflammation
- Pain around the neck
- Odynophagia (painful swallowing)
- Fever
- Cough and irritation of the throat.
- In hypertrophic tonsillitis breathing problems and snoring are present.
- Halitosis (bad breath.)
- Foreign body sensation in the throat

Signs

1. Tonsils of any size.
2. Debris in the tonsillar crypts.

Investigations

Usually throat swabs for cultures becomes negative for Group A Beta Haemolytic *Streptococci*

Management

Management is mainly surgical

Indications for tonsillectomy

- a) Recurrent episodes of acute tonsillitis.
 - Seven or more attacks during the preceding one year
 - Five or more attacks during each year for the last two years
 - Three or more attacks per year during the last three years
- b) Previous two episodes of peritonsillitis or complications of acute tonsillitis.
- c) Suspected neoplasm (unilateral enlargement or ulceration).
- d) Gross enlargement causing airway obstruction (Obstructive Sleep Apnoea).

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CHAPTER 7

ACUTE EPIGLOTTITIS

It is an airway emergency seen mainly in the age group of 2-6 years. This grave illness is most commonly caused by H.influenzae and carries a high risk of complete obstruction of the upper airway. Incidence can be reduced by over 97% with proper immunization of infants with Haemophilus influenzae type b vaccination.

Symptoms

Illness starts as a sore throat and rapidly progresses *within hours* to a very toxic level evidenced by high fever, irritability and dyspnoea. Dysphagia and drooling of saliva may be observed by the parents. The child prefers to sit forward with open mouth and extended neck.

Clinical features

Usually the child is ill and has a characteristic posture, muffled voice, intercostal and subcostal recessions. Cyanosis and deteriorating level of consciousness are precursors of impending respiratory arrest.

Treatment

The diagnosis is mainly clinical.

Get help immediately, if available, call the consultant anaesthetist, ENT surgeon and the consultant paediatrician. (x)

Any disturbing procedure can be disastrous as it may precipitate a laryngeal spasm and respiratory arrest. Do not attempt to place the child supine on the bed. Keep the child on the mother's lap. Refrain from cannulation at this point.

Give high flow humidified oxygen by a face mask. (x)

Intubate the child under anaesthesia by the most competent person. Failing this, do a tracheostomy. Indirect laryngoscopy during the procedure may show a large, cherry red, swollen epiglottis. (x)

In the absence of a skilled hand cricothyrodostomy with a wide bore needle (blood needle 16G) may be life saving, in a sudden event of a respiratory arrest.

Check the adequacy of ventilation and gain intravenous access.

White blood cell count shows increased count with a left shift. Blood cultures are positive for H. influenzae in 50% of patients. The radiological confirmation by x- ray lateral neck for soft tissues should not be done since it may precipitate a sudden obstruction of the upper airway.

Third generation cephalosporins, ceftriaxone or cefotaxime are preferred over ampicillin and chloramphenicol since 30% of H.influenzae strains are resistant to the latter drugs. Usually there is a rapid response to antibiotics and early extubation is possible after 24 hours.

Ceftriaxone – Initial dose of 100mg / kg (max 2g) followed by 50mg/kg for 7–10 days (x)

Cefotaxime – 50mg /kg /dose 8H for 7- 10 days (x)

*If a third generation cephalosporin is not available **Chloramphenicol** (100 mg / kg / day in divided doses would be an alternative.*

If there is a child under 4 years at home who has not completed Hib vaccination, Rifampicin 20mg / kg daily for 4 days should be given for all household contacts including the index case, to eradicate carrier state. ¹

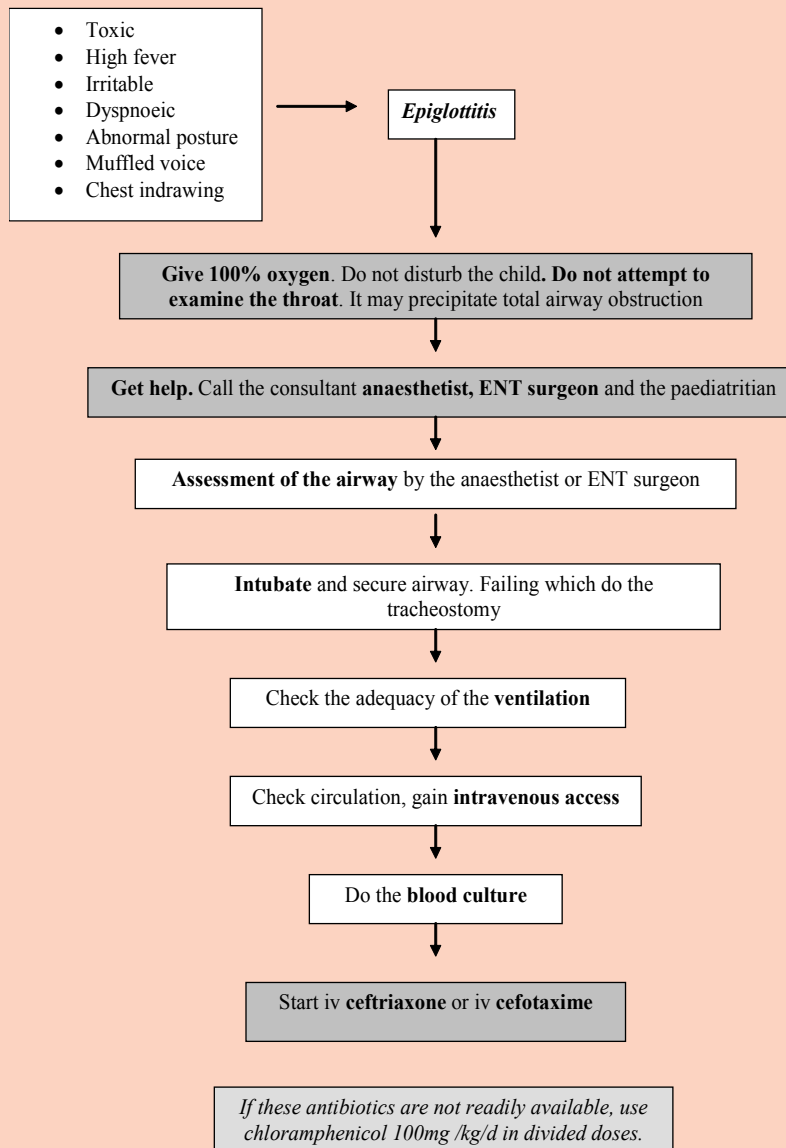
Immunization

Haemophilus influenzae type b vaccination (Hib) is administered at 2nd, 4th and 6th months of life and a booster at 18 months. If immunization is started between 6 months and 1 year of age the child should receive two doses, 1-2 months apart and a booster at 18 months. If immunization is started after 1 year of age only one dose is enough.

References

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The scheme for management of acute epiglottitis



CHAPTER 8

BACTERIAL TRACHEITIS

Definition

Bacterial tracheitis is an uncommon infectious cause of acute upper airway obstruction. It is characterized by a diffuse inflammatory process of the larynx, trachea, and bronchi with adherent or semi adherent mucopurulent membranes within the trachea. Signs and symptoms usually are intermediate, between epiglottitis and croup.

The morbidity and mortality is related to the potential for acute upper airway obstruction. Morbidity is related predominantly due to induced hypoxic insults. Mortality can rise up to 20% in the acute phase. The patients do generally well if the airway is managed adequately and antibiotic therapy initiated very early.

Aetiology

- *Staphylococcus aureus* (most common)
- *Haemophilus influenzae* type B (HiB)
- *Moraxella catarrhalis*
- Streptococcal species, especially *Streptococcus pyogenes*
- *Klebsiella* species
- *Pseudomonas* species
- Anaerobes (*Peptostreptococcus*, *Bacteroides*, *Prevotella*)

Clinical manifestations

Occur in children from 3 weeks to 16 years, with a mean age of 4 years.

Tracheitis is a potentially life-threatening infraglottic infection where most children will need eventual intubation (57-100%), and is similar in many ways to epiglottitis.

- Ill child
- Increasing deep or barking croup-like cough following a previous upper respiratory tract infection
- Worsening or abruptly occurring inspiratory stridor (with or without expiratory stridor)
- High fever
- Variable degrees of respiratory distress increasing in severity over time
 - Chest wall indrawing
 - Dyspnoea
 - Nasal flaring
 - Cyanosis
- No drooling
- No specific position of comfort (The patient may lie supine.)

Differential Diagnosis

- Angioedema
- Candidiasis
- Croup
- Epiglottitis
- Peritonsillar abscess
- Retropharyngeal abscess

Diagnosis

Mainly a clinical diagnosis which is made based on evidence of bacterial upper airway disease and an absence of the classic findings of epiglottitis.

Obtain bacterial culture and Gram stain of tracheal secretions. (can be obtained during intubation).

Obtain blood cultures, though they often do not yield a microbiologic diagnosis.

Imaging Studies:

- Radiographs of the lateral neck
 - In ward, and only in a stable patient
 - Not definitive, not essential

- May reveal subglottic narrowing, clouding of tracheal air column, or irregular tracheal margin
- Concretions of epithelium and inflammatory cells possibly appearing as a foreign body

Management

Once bacterial tracheitis is made as the provisional diagnosis, the child may need PICU management.

Airway

- Maintenance of an adequate airway is of primary importance.
- Check oxygen saturation and give oxygen if saturation is less than 92%.
- Avoid agitating the child. If the patient's respiratory status deteriorates, usually due to movement of the membrane, bag-valve-mask ventilation should be effective.

The child should be in an **intensive care unit** at this stage and need **ENT/ Anaesthetic** referral. **Most patients require eventual intubation (57-100%)** (Annexure 1)

Intravenous Access

Once the airway is stabilized, obtain intravenous access for antibiotics administration.

Antibiotics

Cloxacillin and a third-generation cephalosporin, (x) or chloramphenicol and clindamycin for patients who are allergic to penicillin.

References

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CHAPTER 9

ACUTE LARYNGOTRACHEOBRONCHITIS (CROUP)

Croup is a viral infection of the upper respiratory tract infection commonly seen in preschool children aged 6 months to three years with a peak incidence at 1 - 2 years.

The condition is usually mild and self limiting, although it may occasionally cause severe respiratory obstruction. Secondary bacterial infections are common after five days of illness.

Clinical manifestations

It starts with rhinorrhoea, mild cough and low grade fever which may last up to three days. Then the child develops characteristic features of barking cough, hoarseness and inspiratory stridor. Crying or agitation aggravates the symptoms. The child may prefer the upright position.

Physical examination reveals hoarse voice, tachypnoea and moderately inflamed pharynx.

Decreased oxygen saturation is a late sign in severity of croup. Since it is an upper respiratory tract infection, gas exchange in the alveoli is usually unaffected.

Investigations

It is mainly a clinical diagnosis. Lateral x- ray of the neck for soft tissues may occasionally be warranted in patients with stridor where the diagnosis is uncertain.

Management

Management of the airway is important. Children with croup should be kept calm and distressing procedures (e.g. intravenous cannulation) should be kept to a minimum. They should be allowed to adopt the position they like most [D].

Oxygen must be given via a face mask in moderate to severe croup [D].

In severe croup nebulize with adrenaline 5ml of 1: 1000 (0.5ml/kg to a maximum dose of 5ml) [A/B] which can be repeated twice with a 30 min gap in between.(y)

Still if there is no improvement, the situation should be reviewed urgently.

Be cautious when nebulizing with adrenaline in a child who has congenital heart disease since it can precipitate tachyarrhythmias.

Dexamethasone and budesonide are effective in relieving the symptoms of croup as early as 30 minutes after treatment [B]. The use of systemic corticosteroids has been associated with reduction in disease severity, number of readmissions, the length of hospital stay and the number of co-interventions [A/B].^{1,2}(y)

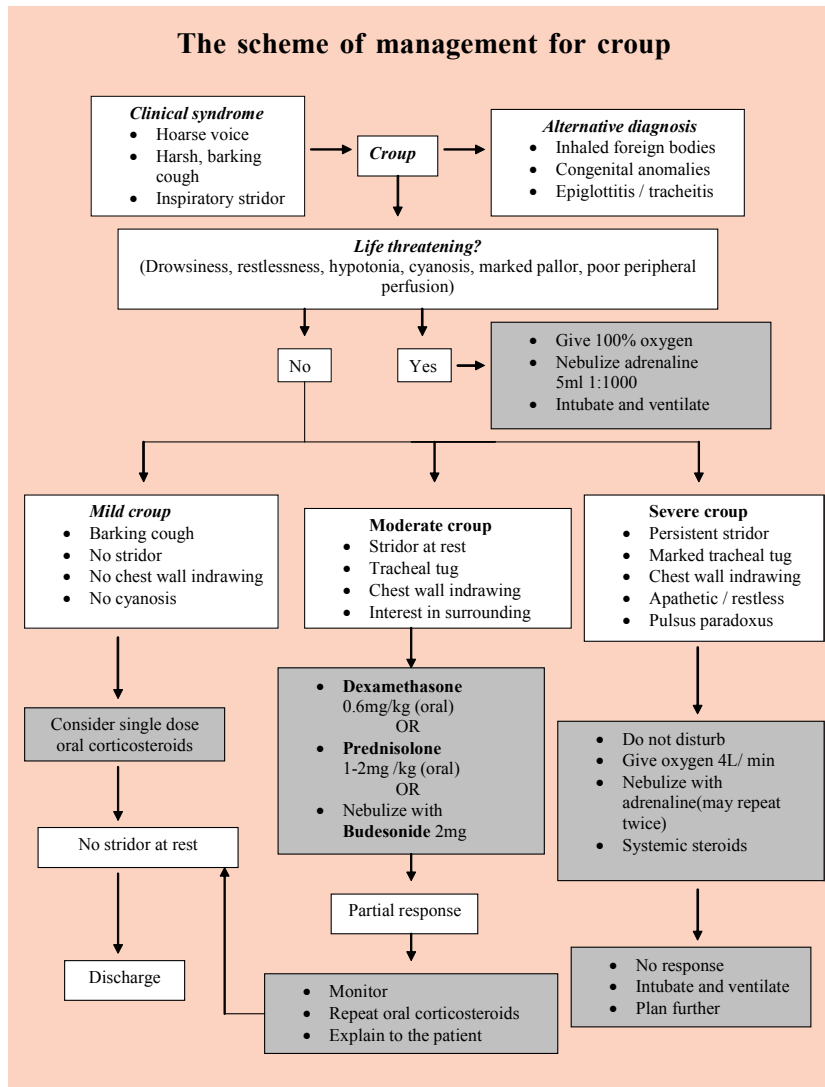
Dexamethasone is given orally 0.6mg /kg as a single dose orally or nebulized with budesonide 2 mg [B].¹

Antibiotics are not indicated unless there is a high degree of suspicion of secondary bacterial infection.

References

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The scheme of management for croup



CHAPTER 10

ACUTE BRONCHIOLITIS

Acute bronchiolitis is the commonest lower respiratory tract infection in infancy.

Respiratory syncytial virus is responsible for most cases and other agents include parainfluenza and adenoviruses.

Acute bronchiolitis is characterized by bronchiolar obstruction with oedema, mucus and cellular debris. Immunological factors also play a significant role in this and impairment of the normal pulmonary gas exchange results in hypoxaemia and hypercapnia.

Clinical features

Bronchiolitis is a clinical diagnosis. It starts as an upper respiratory tract infection with low grade fever and rhinorrhoea. The infant then develops wheezy cough, nasal flaring, tachypnoea and hypoxia. It interferes with the feeding of the child.

Physical examination is characterized by recessions of the suprasternal, subcostal and intercostal regions, and bilateral fine crepitations.

Severe disease is characterized by cyanosis, apnoea and even a silent chest. Apnoea may be the presenting feature especially in very young, premature or low birth weight infants.

Risk factors for severe bronchiolitis

Host factors

Environment

- Pre term
 - Age < 6 weeks
 - Congenital heart disease
 - Immune deficiency
 - Malnutrition
- Parity
Overcrowding
Passive smoking

Investigations

For children with mild bronchiolitis, no investigations are indicated, since they will not influence the management [C].

For moderate to severe infection, investigations such as pulse oximetry, full blood count and chest radiograph may be considered [C/D].

The white blood cell and differential counts are usually normal. The chest radiograph may show hyperinflated lung fields and patchy shadows with pushed down diaphragm and rather horizontal ribs and normal sized heart.

Treatment

Supportive therapy and oxygen supplementation remains the mainstay of treatment [D].

Humidified oxygen is administered via face mask (4L/min) or nasal prongs (2L/min) if saturation is less than 92% to overcome the hypoxia [D].

Although bronchodilators are claimed not to improve oxygen saturation or duration of hospital stay [A], a trial of nebulization with a bronchodilator can be given [D]. If there is no positive response, it should be discontinued.

Multiple studies have failed to demonstrate efficacy of corticosteroids in bronchiolitis [B]. But if there is an apparent improvement after nebulization the infant may have wheezing due to early asthma and may show some response to steroids [D].

The use of nebulized adrenaline has shown some benefits in outpatient settings but it lacks convincing evidence to be used in inpatient settings [B]. Like bronchodilators, a trial of two doses of adrenaline 3ml 1:1000 nebulized 30 minutes apart in moderate to severe bronchiolitis may be appropriate to assess the response [D].

There is no benefit in using antibiotics in hospitalized infants with bronchiolitis [B]. It may be used when secondary bacterial infection such as Staphylococcus or Streptococcus is suspected [D]. This is rare.

Use of ribavirin, a guanosine analogue with a broad spectrum antiviral activity has not been supported by evidence to be beneficial [A].

RSV Prophylaxis

There is no effective vaccine for RSV.

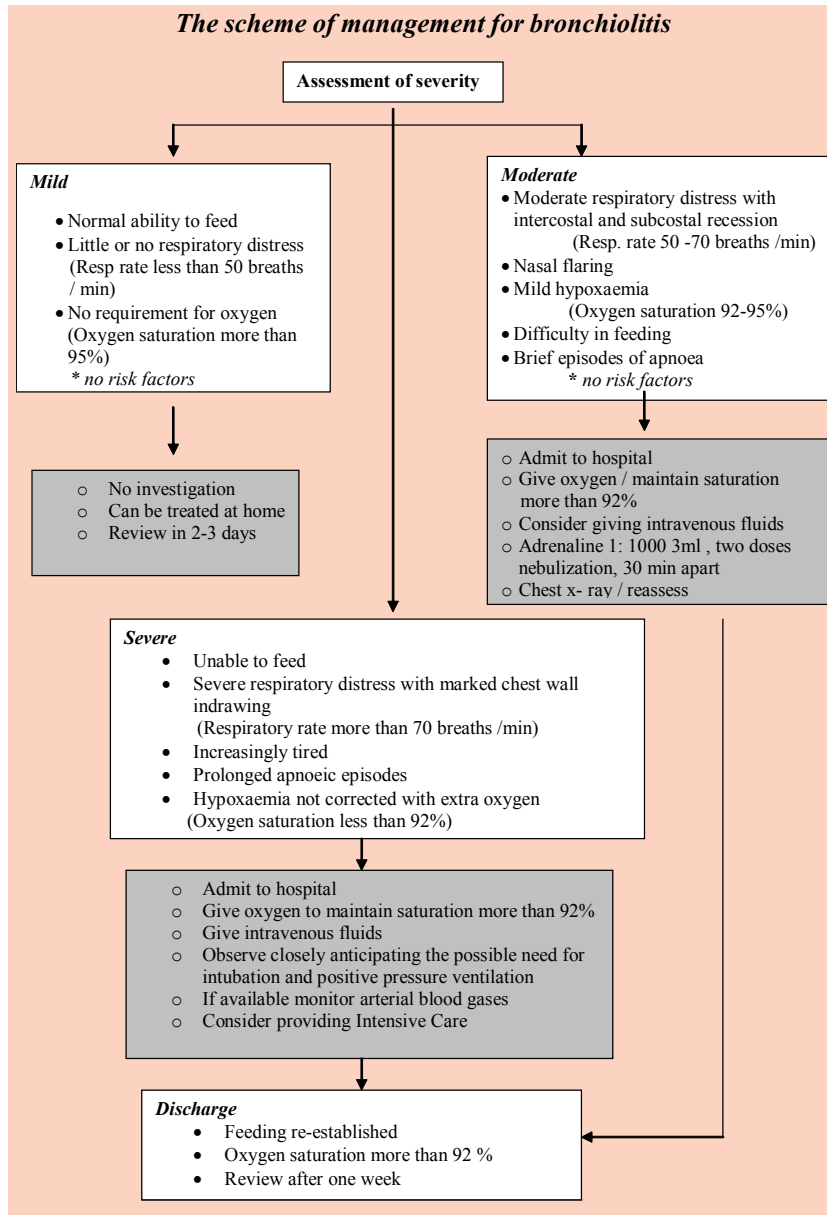
Discharge from the hospital

When the infant is feeding well and oxygen saturation is maintained over 92% without administered oxygen, the baby could be considered for discharge. A review visit may be arranged in one week [D].

References

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The scheme of management for bronchiolitis



CHAPTER 11

PNEUMONIA IN CHILDHOOD

Pneumonia is an inflammation of the parenchyma of the lungs. It is one of the leading causes of mortality in hospital admissions in the paediatric age group.

Common bacteria involved are *S. pneumoniae*, *M. pneumoniae*, Chlamydia, *S. aureus* and *H. influenzae*. *Streptococcus pneumoniae* is the most common bacterial cause of pneumonia in childhood accounting for 25-30% cases [II]. *Mycoplasma* could account for 4-30% cases.

Viruses are most commonly found as a cause in younger children and account for 14-35% cases [II]. Among viruses RSV infection is the commonest. Other viruses responsible are parainfluenzae, influenzae, adeno rhino, VZV, CMV and HSV.

A significant proportion of cases of community acquired pneumonia (up to 40%) represent a mixed infection. In 20–60% of cases a pathogen is not identified [II].

Recurrent pneumonia should raise the suspicion of an underlying disorder such as immunodeficiency, anatomical abnormalities or congenital heart disease.

Clinical definition

There is no single definition for pneumonia. It is defined in terms of symptoms, signs and clinical course. WHO defines pneumonia as a febrile illness with tachypnoea for which there is no other apparent cause.

Community acquired pneumonia (CAP) can be defined clinically as the presence of signs and symptoms of pneumonia in a previously healthy child due to an infection which has been acquired outside hospital.

There are two further clinical definitions for pneumonia. Bronchopneumonia is a febrile illness, with cough, respiratory distress with evidence of localized or

generalized patchy infiltrates on chest x - tray. Lobar pneumonia is similar to bronchopneumonia except that the physical findings and radiographs indicate lobar consolidation.

Pathogens causing pneumonia³

The age is a good predictor of the likely pathogens [11].

Age	Organisms
Neonate	Gp B <i>Streptococcus</i> , <i>E. coli</i> , <i>Klebsiella</i> , <i>Enterobacteriaceae</i> , <i>Listeria monocytogens</i>
1 m to 1 year	<i>RSV</i> , <i>parainfluenza</i> , <i>Chlamydia trachomatis</i> <i>S. Pneumoniae</i> , <i>S. aureus</i> , <i>B. pertussis</i>
1- 5 years	<i>S. pneumoniae</i> , <i>S. aureus</i> , <i>H. influenzae b</i> , <i>Mycoplasma</i> , <i>RSV</i> , <i>parainfluenza</i> , <i>influenza</i> , <i>adeno</i> , <i>M. tuberculosis</i> Less commonly Gp A <i>Streptococcus</i> , <i>Moraxella</i>
>5years	<i>S. pneumoniae</i> , <i>Mycoplasma pneumoniae</i> , <i>Chlamydia pneumoniae</i> , <i>M. tuberculosis</i>

Clinical manifestations

The illness may start with symptoms of upper respiratory tract infection followed by the signs of lower respiratory tract infection.

Tachypnoea, intercostal and subcostal recessions, restlessness and drowsiness may indicate the severity of pneumonia.

Tachypnoea with chest indrawing is the **best predictor** of pneumonia of children in all age groups.

<i>WHO defined tachypnoea</i>	
○ < 2 months of age	over 60 breaths/ min
○ 2-12 months	over 50 breaths/ min
○ >12 months	over 40 breaths/ min

Features of bacterial lower respiratory tract infection (LRTI) ¹

- Fever more than 38.5°C.
- Respiratory rate above the appropriate upper limit for age
- Chest recession.
- Wheezing is not a sign of primary bacterial LRTI (other than Mycoplasma).
- Other viruses may be concurrent.
- Clinical and radiological signs of consolidation rather than collapse.

Features of viral lower respiratory tract infection (LRTI) ¹

- Infants and young children.
- Wheezing.
- Fever lesser than 38.5°C.
- Marked chest wall recessions.
- Hyperinflation.
- Respiratory rate normal or raised.
- Radiograph shows hyperinflation and, in 25%, patchy collapse.
- Lobar collapse when severe.

Indicators for admission to hospital in infants:

- Oxygen saturation (SaO₂) less than 92%, cyanosis
- Respiratory rate over 70 breaths/min
- Difficulty in breathing
- Intermittent apnoea, grunting
- Poor feeding (approximately reduction by half)
- Family not being able to provide appropriate observation or supervision.

Indicators for admission to hospital in older children:

- Oxygen saturation (SaO₂) less than 92%, cyanosis
- Respiratory rate over 50 breaths/min
- Difficulty in breathing
- Grunting
- Signs of dehydration
- Family not being able to provide appropriate observation or supervision

Diagnosis

The diagnosis is primarily made clinically and may be supported radiologically. Radiographic findings are poor indicators of aetiology and severity. Chest x ray may show consolidation and pleural effusion

Chest radiography should not be performed routinely in children with mild uncomplicated acute lower respiratory tract infection.

Follow up chest radiography should only be performed after lobar collapse, an apparent rounded opacity, or for continuing symptoms [C].

Acute phase reactants like CRP do not distinguish between bacterial and viral infections in children and should not be measured routinely [A]. White blood cell count is useful to differentiate bacterial from viral infections.

When facilities are available blood cultures should be performed in children suspected of having bacterial pneumonia [B]. Acute serum samples for antibodies may be saved and a convalescent sample taken in cases where a microbiological diagnosis was not reached during the acute illness [B].

When significant pleural fluid is present, it could be aspirated for diagnostic purposes and sent for microscopic examination and culture. Another specimen could be saved for bacterial antigen detection [B].

Treatment

If the clinical diagnosis is bronchopneumonia or lobar pneumonia empirical treatment is necessary to reduce the morbidity and mortality since the possible organisms are difficult to predict.

Intravenous antibiotics should be used in pneumonia when the child has signs of severe pneumonia and vomiting which disturbs oral intake [D].

Suggested initial empirical treatment

0- 3 months

iv Penicillin or Ampicillin and Gentamicin as first line therapy (x)
Cefotaxime or Co amoxiclav as second line therapy

3 months to 1 year

If the child is not ill – oral Amoxicillin (x)
If ill or lobar infiltrate in chest x-ray – iv Ampicillin (x)
If no response within 48 hours use 2nd or 3rd generation Cephalosporins
(eg. Cefuroxime or Cefotaxime) (x)
If Staphylococcal infection is suspected add iv Cloxacillin
If MRSA is suspected add iv Vancomycin

1-5 years

If the child is not ill - oral Amoxicillin or penicillin (x)
If ill or lobar infiltrate with or without effusion in chest x-ray–iv Penicillin(x)
If no response within 48 hours use 2nd or 3rd generation Cephalosporins (x)
(eg. Cefuroxime or Cefotaxime)
If Staphylococcal infection is suspected add iv Cloxacillin
If MRSA is suspected add iv Vancomycin

>5 years

If the child is not ill - oral Macrolides (x) (Erythromycin, Azithromycin, Clarythromycin)
If toxic or lobar infiltrates with or without effusion – treat as for **1-5 yr** old
No improvement or chest x- ray / clinical findings are ambiguous – add a
Oral macrolide

Duration of the antibiotic therapy should be 5- 7 days and in severe cases, 10 days.

After starting oral therapy if the condition is not improving after 48 hours the child should be admitted to hospital for further management [D].

Intravenous antibiotics could be switched to oral antibiotics, when the temperature falls and breathing difficulty is resolving [D].

Supportive therapy

When a child is in severe respiratory distress oral intake may be impaired. Intravenous fluid can be administered, but should be done cautiously, since syndrome of inappropriate ADH secretion is a possibility.

Oxygen should be administered to children who are restless, tachypnoeic with severe chest indrawing (Oxygen saturation < 92%) [A].

Antipyretics and analgesics can be used to keep the child comfortable and to help coughing. In the ill child minimal handling may reduce metabolic and oxygen requirement.

There is *no proven benefit* of giving anti-tussives in pneumonia.

Routine chest physiotherapy is not recommended [B].

Follow up chest radiography should be performed after lobar collapse, an apparent rounded opacification, or for continuing symptoms.

Management of specific pneumonias

Type	Features	Treatment
Staphylococcal pneumonia Common in infants ¹ 30% younger than 3 months 70% younger than 1 year Common in <ul style="list-style-type: none"> • Complicating influenza • Debilitated children and immunosuppressed infants • Nosocomial pneumonias • Cystic fibrosis 	Clinically suspect if ¹ <ul style="list-style-type: none"> • Onset is abrupt and rapidly progressive pneumonia in an infant • The CXR shows pneumatoceles, pneumothorax or empyema Pneumatoceles commonly lead to pneumothorax*. The long term outcome is good with normal lung functions ^{2,3} .	<ul style="list-style-type: none"> • Cloxacillin IV 100mg – 200mg/kg/day 6 hourly • Cefuroxime IV 100-150mg/kg/day 8 hourly • Vancomycin IV 40mg/kg/day 6 hourly in an infusion if MRSA is suspected Even if there is a small effusion insert a chest drain of largest possible calibre ¹
Haemophilus influenzae⁴ Median age one year	<ul style="list-style-type: none"> • Coryza precedes most cases • Pleural effusions in 50% cases 	<ul style="list-style-type: none"> • Ampicillin IV 100mg/kg/day 6 hourly • About 30% produce beta lactamases against Ampicillin • Ceftriaxone IV 100mg/kg/day
Klebsiella pneumonia (Friedlander's pneumonia) <ul style="list-style-type: none"> • Rare as a community acquired pneumonia in children • Common in nosocomial pneumonias 	<ul style="list-style-type: none"> • Runs a fulminant course • Frequently affects upper lobes • Produce sputum like red currant jelly 	<ul style="list-style-type: none"> • Cefotaxime IV 100mg/kg/day 8 hourly
Chlamydia trachomatis³ Common in infants Upto 4 months of age	<ul style="list-style-type: none"> • Insidious onset • Staccato cough is not specific • Crepitations described more often than wheeze • Hyperinflation in CXR with patchy infiltrates • Conjunctivitis in 50% 	<ul style="list-style-type: none"> • Erythromycin 40mg/kg/day 6 hourly • If conjunctivitis is suspected Erythromycin or Tetracycline eye drops.

Oral treatment in pneumonia¹⁹

Drug	Age	Dose	Frequency	Notes
Amoxicillin	1 m – 2y	125mg/dose or 12.5mg/kg/dose	3	Dose may be doubled in severe infection
	2- 12y	125-250mg or 12.5mg/kg/dose	3	Duration 7—10 days
	12-16y	500mg	3	
Co-amoxiclav Syrup	0 – 1 yr	0.25ml/kg/ dose (125/31)	3	Doses may be doubled in severe infection
	1-6 yr	5ml /dose(125/31)	3	Duration 7-10 days
	7-12 yr	10ml/dose (250/62)	3	
	12-16y	375mg- 625mg/dose	3	
tablets	0 -1 m	10-15mg /kg/dose	3	Doses may be doubled in severe infection
	1m – 2y	125 mg	4	Duration 7 – 10 days
	2-8 yr	250 mg	4	
	9 – 18y	500 mg	4	
Erythromycin	6m-2y	10mg/kg	1	5 days
	3-7y	200mg	1	
	8-11y	300mg	1	
	12-14y	400mg	1	
	>14y	500mg	1	
Azithromycin	birth– 1y	7.5mg/kg/dose	2	7-10 days
	1-6y	125mg/kg/dose	2	
	7-9y	187.5mg/dose	2	
	9-16y	250mg/dose	2	
Clarithromycin	<1m	25mg/kg/dose	2	Duration 7 – 10 days
	1m-1y	62.5mg- 125mg/dose	3	
	1-5y	125mg- 187.5mg/dose	3	
	5-12y	250mg/dose	3	
	12-16y	250mg- 500mg/dose	3	
Cephalexin	<1m	25mg/kg/dose	2	Duration 7 – 10 days
	1m-1y	62.5mg- 125mg/dose	3	
	1-5y	125mg- 187.5mg/dose	3	
	5-12y	250mg/dose	3	
	12-16y	250mg- 500mg/dose	3	

Intravenous treatment in pneumonia ^{1,9}

Drug	Age	Dose	Frequency	Notes
Ampicillin	1 m–16 y	50 mg/kg/dose	4	Max single dose is 1 g
Benzyl penicillin	1 m–12 y	25-50mg/kg/dose	4	In severe infections doses of 50mg/kg may be given 4 hourly (6 times/day)
	12–16 y	300–600 mg (0.5–1 mega units)	4	
Cefotaxime	1m-12y	50 mg/kg/dose	3	Frequency may be increased to four times daily in severe infection
	12-16y	1-2g/dose	3	
Cefuroxime	<7d	25mg /kg/dose	2	Dose may be doubled in severe infection
	>7d	25mg/kg/dose	3	
	1m-16y	10-30mg/kg/dose	3	
Co amoxiclav	1m – 12y	30mg/kg/dose	3	Dosage based on co-amoxiclav content. Over 3 months of age dose frequency can be increased to 4 times daily in severe infections
	12-16y	1.2g/ dose	3	

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CHAPTER 12

ATYPICAL PNEUMONIA

Definition

Atypical pneumonia refers to pneumonia caused by the following organisms^{1,2}.

- *Mycoplasma pneumoniae*
- *Chlamydia pneumoniae*
- *Legionella pneumophila*

Atypical pneumonia due to *Mycoplasma* often causes milder form of pneumonia. They are characterized by a protracted course of illness unlike other forms of pneumonia that can present acutely with more severe early symptoms. However pneumonia due to *Legionella* could be severe.

Symptoms

- Chills
- Fever
- Cough –may be dry or productive
- Headache
- Muscular stiffness and aching
- Loss of appetite
- Malaise

Note: These symptoms could occur in any viral fever.

Mycoplasma pneumoniae

Mycoplasma pneumoniae is a common cause of community acquired pneumonia in the older child [11]. The commonest age group affected is 5- 15 yrs. It is rare under 4 years. The younger child attending daycare is at risk³.

Clinical Presentation^{4,5,6}

Mycoplasma pneumonia is a disease of gradual and insidious onset of several days to weeks.

Clinical findings that raise the suspicion of Mycoplasma pneumonia in community acquired pneumonia (CAP).

- **Dry paroxysmal cough or wheezing in an older child**
- Low grade fever (less than 38.5 °C)
- Prominent headache or myalgia and arthralgia
- Illness lasting more than 1 week
- Extra pulmonary manifestations
- Poor response to routine antibiotics used in CAP

Uncommon features of mycoplasma infection.

- Rhinorrhoea and nasal congestion
- Pleural effusion
- Pulmonary abscess

Fever, arthralgia, headache, cough and crepitations in a schoolchild would suggest mycoplasma infection⁶[IV b]

Extra-pulmonary manifestations⁷

These manifestations are rare. They are caused by an autoimmune response to the organism.

- | | |
|-------------------------|---|
| • Haematological | - haemolytic anaemia , thrombocytopenia |
| • Renal | - nephritic presentation |
| • Skin | - erythema multiforme or Steven Johnson syndrome |
| • Joints | - Arthritis |
| • Nervous system | - peripheral neuritis, central nervous system infections, Guillain Barre syndrome, transverse myelitis, acute psychoses |
| • Cardiovascular | - pericarditis, myocarditis |
| • Ocular | - conjunctivitis, anterior uveitis, optic atrophy |
| • ENT | - bullous myringitis (over 2 years) |

High risk cases

In sickle cell anaemia and immunodeficiencies mycoplasma pneumonia can be severe.

Investigations

Full blood count is not helpful³.

Radiology

There is no radiological feature that is pathognomonic of mycoplasma pneumonia. Interstitial infiltrates, lobar consolidation, hilar adenopathy have all been described. Pleural effusions are rare⁹ [111].

Four distinct patterns are recognized in chest x-ray⁹.

1. Reticulonodular opacification often involving a single lower lobe is the commonest pattern (75%). Focal or bilateral reticulonodular pattern of lower lobes in a clinically suspected case is suggestive of the diagnosis. Lobar consolidation is rare.
2. Hilar adenopathy around 30%
3. Plate like atelectasis noted as thin, flat areas of collapsed lung, often seen in lateral films.
4. Nodular infiltration

Serological testing^{10,11, 12}

Complement fixation test :

A rise in paired titres is the gold standard.⁵ The rise in antibody titre has already occurred by the time the child presents. Therefore a fall in convalescence is also confirmatory. IgM ELISA has been known to reach a diagnostic level during the second week of illness.¹³

Cold agglutinin testing

Used as an acute test in children of 5-14 years. It gives a positive predictive value of 70%.⁵ The diagnosis of mycoplasma pneumonia is supported by the presence of cold agglutinins in a titre of 1:64 or greater.

Cold agglutinin ward test³

This often forgotten bedside test can be very useful in resource poor settings. It is positive in half the cases.

1. Take 0.5 ml of blood to test tube.
2. Add 0.5ml of sodium citrate.
3. Place it in an ice bucket or refrigerator for 20 minutes.
4. Tilt the tube and look for layering of a film of clots from below.
5. The “grains of sand effect” appear on the glass portion of the tube.



Cold agglutinin ward test

Treatment⁵

Macrolides are used if *Mycoplasma* or *Chlamydia pneumoniae* are suspected [D]. Because mycoplasma pneumonia is more prevalent in older children macrolide antibiotics may be used as first line empirical treatment in children aged 5 and above with community acquired pneumonia. [D]

Erythromycin 40mg/kg/ day orally 6 hourly for 7-10 days.

Azithromycin 12mg/kg as a single daily dose for 5 days

Clarithromycin 15 mg/kg day 12 hourly for 10 days.

The newer macrolides are better tolerated and have lesser dosing frequency.

Antibiotic prophylaxis for household contacts is not routinely recommended. However, if there are high risk household contacts, consider prophylaxis.

Chlamydia pneumonia

Chlamydia pneumoniae causes atypical community acquired pneumonia indistinguishable clinically from mycoplasma pneumonia¹⁴. Both can co-exist in CAP in children.

Diagnosis

Chest x ray shows bilateral chest expansion with diffuse infiltrates.¹⁵

Serological testing can distinguish it from the other chlamydial illnesses. (eg. psitacosis, trachoma)

Treatment

Macrolides are recommended as above.

Legionella pneumonia¹⁶

Number of reported cases of legionella in childhood is small. Both community acquired and nosocomial cases of Legionellosis are seen in children. Most children with Legionaire disease are immunosuppressed.

Three epidemiological patterns are recognized.

1. Outbreaks in previously fit individuals staying in hotels, institutions or hospitals where the cooling systems or shower facilities are contaminated with the organism.
2. Sporadic cases are seen in children.
3. Outbreaks occur in immunocompromised patients.

A strong presumptive diagnosis of legionella pneumonia can be made if following clinical features are present.

- A prodromal illness like a viral infection
- A dry cough, confusion or diarrhoea
- Lymphopenia without marked leukocytosis
- Hyponatraemia

Diagnosis

Four fold rise in antibody titre
Urinary antigen test – highly specific

Treatment

Macrolides are the drugs of choice.

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CHAPTER 13

NOSOCOMIAL PNEUMONIA (HOSPITAL ACQUIRED PNEUMONIA)

Definition

Nosocomial pneumonia is pulmonary infiltrates occurring in a patient who has been hospitalized for 1 week or more which is compatible in appearance with a bacterial pneumonia.

Within 1 week of hospitalization, the normal respiratory flora of the patient is displaced by the nosocomial organisms. (Aerobic gram negative bacilli or *Staphylococcus aureus*).

Centres for Disease Control and Prevention(CDC) definition of Nosocomial Pneumonia

(1) Infants of under 12 months of age:

A: Without a chest radiograph must have at least 2 of the following signs or symptoms (apnoea, tachypnoea, bradycardia, wheezing, rhonchi, or cough) AND any of the following (a-f):

- a. Increased production of respiratory secretions.
- b. New onset of purulent sputum or change in character of sputum.
- c. Organism isolated from blood culture.
- d. Isolation of pathogen from specimen obtained by transtracheal aspirate, bronchial brushing, or biopsy.
- e. Isolation of virus or detection of viral antigen in respiratory secretions.
- f. Histopathologic evidence of pneumonia.

B: With a chest radiograph must have a new or progressive infiltrate, cavitation, consolidation, or pleural effusion AND any of the features listed above in A (a-f)

(2) Children of over 12 months of age

A: Without a chest radiograph patient must have crackles or dullness to percussion on physical examination of the chest AND any of the following (a-c):

- a. New onset of purulent sputum or change in character of sputum
- b. Organism isolated from blood culture.
- c. Isolation of pathogen from specimen obtained by transtracheal aspirate, bronchial brushing, or biopsy.

B: With a chest radiograph, the patient must have a new or progressive infiltrate, cavitation, consolidation, or pleural effusion AND any of the following (a-f):

- a. Increased production of respiratory secretions.
- b. New onset of purulent sputum or change in character of sputum.
- c. Organism isolated from blood culture or diagnostic single antibody titre (IgM) or fourfold increase in paired serum samples (IgG) for pathogen.
- d. Isolation of pathogen from specimen obtained by transtracheal aspirate, bronchial brushing, or biopsy.
- e. Isolation of virus or detection of viral antigen in respiratory secretions.
- f. Histopathologic evidence of pneumonia.

If the aerobic gram negative organisms are aspirated into the lungs nosocomial pneumonia may occur. Alternatively, bacteraemia is the other mechanism of acquiring nosocomial pneumonia.

Risk factors of nosocomial pneumonia

- Aspiration
- Intubation
- Bacteraemia

Microbiology of nosocomial pneumonia

Common causes

- *Pseudomonas aeruginosa*
- *Klebsiella pneumoniae*
- *E.coli*

Uncommon pathogens

- *Serratia*
- *Acinobacter*
- *Legionella*

Staphylococcus aureus is a rare cause of infection though it **colonizes** respiratory tract commonly. However in neonatal intensive care units (NICU) methicillin resistant *Staphylococcus aureus* (MRSA) is a common pathogen.

Paediatric intensive care units have higher incidence of *Pseudomonas aeruginosa* infection compared to neonatal intensive care units. *Klebsiella* and *E. coli* are more prevalent in paediatric than in adult intensive care units. Anaerobes and multiple organisms are often found in aspiration pneumonia.

Presentations of nosocomial pneumonia

Necrotizing pneumonia with rapid cavitation within 72 hours is the hallmark of pseudomonas and staphylococcal pneumonias. The cavitation in klebsiella pneumonia occurs 3-5 days after the onset of nosocomial infection.

Treatment of nosocomial pneumonia

Monotherapy is now the preferred choice with broad spectrum drugs that cover klebsiella pneumoniae and pseudomonas. Following table of drugs can be used in the treatment of nosocomial infection considering sensitivity patterns of the isolated micro-organisms.

Antibacterial agent	Comments
Ceftazidime 25mg/kg every 8 hours	<ul style="list-style-type: none"> • <i>Ceftazidime resistant P.aeruginosa</i> has been observed. • <i>Ceftazidime use is associated with increased staphylococcal colonization since it has little anti staphylococcal activity.</i>
Cefepime 50 mg/kg/dose administered IV or IM every 12 hours for 7 to 10 days.	<ul style="list-style-type: none"> • <i>Excellent anti P.aeruginosa activity</i> • <i>Unrestricted Cefepime activity is not associated with resistance problems</i> • <i>Cefepime is effective against most strains of Ceftazidime resistant P.aeruginosa</i> • <i>It has excellent anti staph aureus activity (but not MRSA)</i> • <i>It has few side-effects but uncommonly causes drug fever</i> • <i>Cefepime is less expensive/day to the hospital than Ceftazidime</i>
Meropenem 10 mg/kg/dose 8 hourly Can be given as a bolus or an infusion	<ul style="list-style-type: none"> • <i>Excellent anti P.aeruginosa activity</i> • <i>Unrestricted Meropenem activity is not associated with resistant organisms</i> • <i>Effective against most strains of Ceftazidime resistant P.aeruginosa</i> • <i>Has excellent Staph aureus activity (not MRSA)</i> • <i>Has virtually no side effects</i> • <i>Particularly useful against Extended Spectrum Beta Lactamase (ESBL) producing strains of klebsiella or E.coli</i> • <i>Effective against Acinobacter outbreaks</i> • <i>Useful in patients with anaphylactic reactions to penicillin</i> • <i>Compared with imipenem the incidence of nephrotoxicity is less</i>
Ticarcillin with clavulinate 80mg/kg/dose every 6-8 hours	<ul style="list-style-type: none"> • <i>Active against Pseudomonas, Gram negative bacilli, beta lactamase producing organisms, Proteus sp, Bacteroides</i>

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CHAPTER 14

POORLY RESOLVING PNEUMONIA

Definition

Less than 50% clearing of radiological abnormalities at 2 weeks and less than complete clearing of abnormalities by 4 weeks.

Factors contributing to a poor clinical response to empirical antimicrobial therapy in patients with acute pneumonia

a) Drug factors

1. Inappropriate antimicrobial agents
2. Inappropriate dosing regimes
3. Drug Hypersensitivity or other adverse effects

b) Host factors

1. Poor host defences
 - Immune deficiency
 - Malnutrition
 - Endobronchial obstruction
 - Significant co-morbidity –diabetes, malignancy, gastroesophageal reflux.
2. Age of the child
3. Phlebitis –cannula infection, deep vein thrombosis

c) Complication of pneumonia

1. Infective complications
 - Empyema or abscess
 - Metastatic spread – septic arthritis, endocarditis, pneumonia
 - Superinfections
2. Noninfective complications – hypoxia, dehydration
3. Effusions
4. Atelectasis

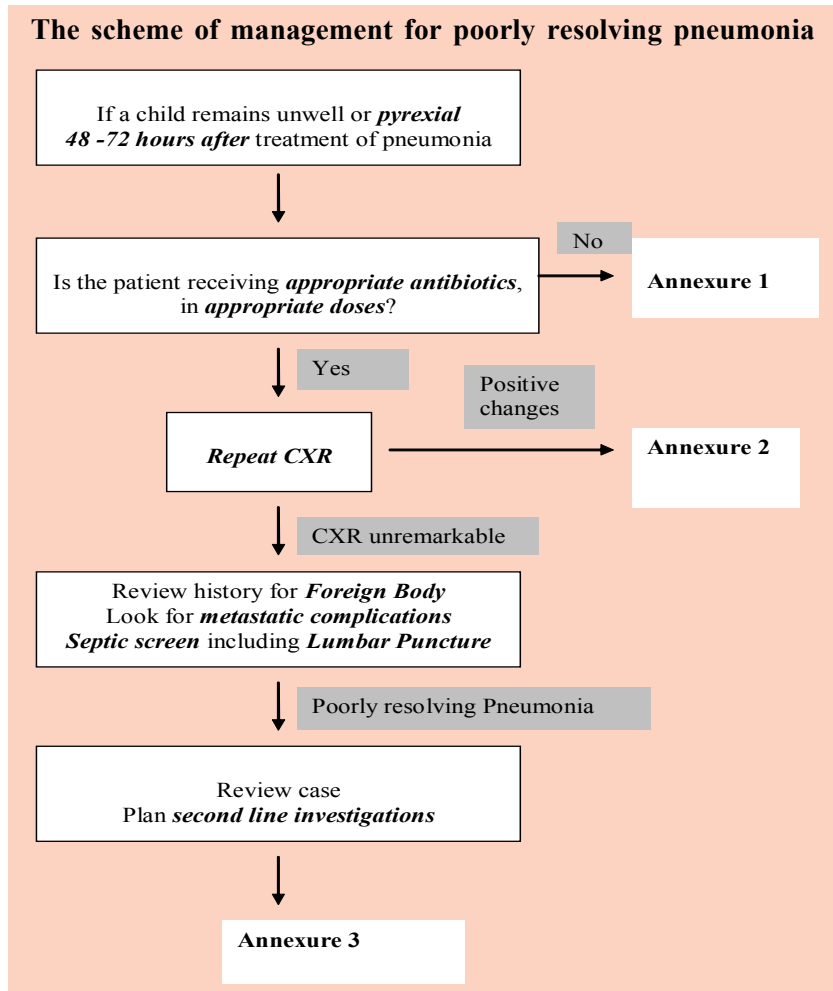
d) Incorrect Diagnosis

Incorrect microbiological diagnosis

1. Mycobacterial infections
2. Atypical pneumonias
3. Opportunistic organisms

Incorrect pathological diagnosis

1. Endobronchial obstruction
2. Bronchiectasis
3. Cystic fibrosis
4. Pulmonary sequestration



Annexure 1

- If antibiotic dose is not correct - adjust dose
- MIC levels
- ABST patterns
- Allergic workup and stop drug if drug reaction

Annexure 2

Repeat CXR and look for the following

Finding	Action to be taken
Is there fluid?	Aspirate, get surgical opinion
Has the pneumonia progressed?	Change to second line antibiotics
Suggestive of FB	ENT opinion
Suggestive of atypical pneumonia	Add macrolides, serology

Pneumatocoeles in CXR may suggest

- **Staphylococcal pneumonia**
- Haemophilus influenzae pneumonia
- Klebsiella pneumonia
- Primary TB Pneumonia with cavitation

N.B. Pneumococcal pneumonia also can rarely cause pneumatocoeles

Unilateral hilar adenopathy may suggest

- Primary TB
- Mycoplasma pneumonia

Foreign body is suggested if there is

- Right middle lobar involvement
- Volume changes

Annexure 3

Review patient's history for

- Contact with TB
- Recent travel
- Contact with birds
- Consanguinity and recurrent pneumonias in the family

Examine patient – Is the diagnosis correct?

- **Clubbing** in
Bronchiectasis
Lung abscess or empyaema

Exclude **co-morbidity**

Review CXR

Second line investigations

- Screen for TB
- Immune screen
- Upper GI studies
- Serological testing
- Sweat test
- Bronchoscopy
- CT scanning
- Lung biopsy

N.B. In malnourished and debilitated children pneumonia may take time to resolve.
Nutritional building takes priority over second line investigations.

CHAPTER 15

PNEUMONIA IN THE IMMUNOCOMPROMISED CHILD

Host defect	Examples of disorders	Likely pathogen
<p><i>Defective polymorpho nuclear leukocytes</i> Neutropenia</p> <p>Chemotaxis</p> <p>Defective intracellular killing</p> <p>Defective alternative path</p>	<p>Acute leukaemia, aplastic anaemia, cancer chemotherapy</p> <p>Chediak Higashi, Hyper IgE</p> <p>chronic granulomatous disease</p> <p>sickle cell disease</p>	<p>Gram negative bacteria <i>Staphylococcus aureus</i> <i>Aspergillus</i>, <i>Candida</i> <i>S.aureus</i>, Gram negative aerobes <i>S.aureus</i></p> <p><i>S.pneumoniae</i>, <i>H.influenzae</i></p>
<p><i>Cell mediated immuno deficiency</i></p>	<p>Corticosteroid therapy, cancer chemotherapy, Hodgkin's</p> <p>AIDS</p>	<p>Mycobacteria, Herpes simplex, Cytomegalo virus, Toxoplasmosis <i>Aspergillus</i> <i>Cryptococcus</i> <i>Strongyloides</i></p> <p><i>Pneumocystis jiroveci</i> <i>Toxoplasmosis</i> <i>Cytomegalovirus</i> Mycobacteria, Opportunistic fungi</p>
<p><i>Humoral Immunity</i></p>	<p>Hypogammaglobulinaemia</p> <p>Selective deficiency IgA, IgG, IgM</p>	<p><i>S.pneumoniae</i>, <i>H.influenzae</i>, <i>P.carinii</i> <i>Cytomegalovirus</i> <i>S. pneumoniae</i> <i>H. influenzae</i></p>

DD of pulmonary infiltrates in the immunocompromised child

Chest radiological course	Likely pathogen
Consolidation	<ul style="list-style-type: none"> • Bacterial pneumonia (gram negative bacilli, <i>S.aureus</i>, anaerobes and <i>Legionella pneumophila</i>) • Fungi • <i>Nocardia asteroides</i> • Mycobacteria
Peribronchial infiltrates	<ul style="list-style-type: none"> • <i>Pneumocystis jiroveci (carinii)</i> • Cytomegalovirus and other viruses
Nodular infiltrates	<ul style="list-style-type: none"> • Bacteria • Fungi • Nocardial infection

	Features	Treatment
Pneumocystis jiroveci (carinii) pneumonia	<i>CXR shows diffuse bilateral perihilar infiltrates. Arterial blood gasses show hypoxaemia.</i>	<i>Trimethoprim-sulfamethaxazole is the drug of choice. Trimethoprim 15 -20 mg/kg/day Sulfamethaxazole 75-100mg/kg/day</i>
Cytomegalovirus pneumonia	<i>Syndrome similar to infectious mononucleosis. CXR – Diffuse interstitial infiltrates. Positive CMV serology.</i>	<i>Ganciclovir</i>
Pulmonary candidiasis	<i>Fever may be the only sign in an immunosuppressed host. Clinical and radiological features may be minimal. Positive blood culture.</i>	<i>Daily IV Amphotericin B 0.5mg/kg Daily oral Flucytocin 150mg/kg</i>
Varicella Zoster virus pneumonitis	<i>Usually pneumonitis occurs while the rash is erupting. Staphylococcus aureus often complicates varicella.</i>	<i>Aciclovir 30mg/kg 8 hrly iv for 7 days or for two days after the last appearance of new skin lesions, whichever is longer.</i>

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CHAPTER 16

WHOOPIING COUGH / PERTUSSIS

Pertussis is the preferred term as whoop may not be a feature.

Pertussis is a prolonged severe respiratory illness in infants and unimmunized children below 5 years.

The challenge in providing effective treatment and chemoprophylaxis of pertussis lies in the **early recognition and reporting** of cases¹.

Aetiology

Bordetella pertussis is the sole cause of epidemic pertussis. *Bordetella parapertussis* is an occasional cause of pertussis. It causes a milder illness.

Whooping cough like illness can be caused by ²

- *Mycoplasma pneumoniae*
- *Chlamydia pneumoniae*
- *Chlamydia trachomatis*
- Adenoviruses

Clinical features

Only 30- 50% children have the characteristic paroxysmal cough and the inspiratory whoop.

Young infants present with apnoea, post tussive vomiting, poor feeding or bradycardia.

Period of communicability is from before the onset of symptoms to three weeks after the onset of cough.

Complications

Common complications seen in infancy are as follows,

- Secondary bacterial pneumonia (commonest cause of mortality)
- Apnoea
- Convulsions
- Encephalopathy
- Pneumothorax
- Otitis media

Recommended case definition WHO

Confirmed case

- A child with a cough who is microbiologically confirmed for *B. pertussis*.

Suspected case

- Any of the following
 - a. Cough of more than 14 days with one of the following criteria
 - i. Inspiratory whoop
 - ii. Post tussive vomiting
 - iii. Apnoea in an infant
 - iv. Subconjunctival hemorrhages
 - v. Absolute lymphocytosis of 15,000 or more
 - b. Cough of any duration in a person with a contact history of a microbiologically confirmed case

Microbiology

If pertussis is suspected per nasal swab for culture should be taken prior to antibiotics. Nasopharyngeal aspiration can be done if the facilities are for available, culture and PCR. PCR will differentiate *B. pertussis* from *parapertussis* infection.

Culture and PCR are most sensitive when cough has been present for less than 2 weeks, but after that time false negative results can occur. PCR may be able to identify the organisms up to 3-4 weeks or longer after the onset of cough.

Technique for obtaining per nasal swab

Swab advanced along the floor of the nose until child gags.
If transport is delayed for more than 24 hours refrigerate the sample.

Do not use cotton swabs since it *inhibits the growth of bacteria*¹¹

Treatment

Erythromycin 40-50mg/kg day 6 hourly for 14 days^{2,5,6} (x)

< 2 Years Erythromycin 125 mg by mouth 6 hourly for 14 days
2 – 8 Years Erythromycin 250 mg by mouth 6 hourly for 14 days
>8 Years Erythromycin 250 -500 mg by mouth 6 hourly for 14 days
Neonates 12.5 mg/kg 6 hourly

Treatment to those who cannot tolerate erythromycin^{1,3,5}

- Clarithromycin 10mg/kg/dose twice a day for 7 days
- Azithromycin 10mg/kg daily for 5 days
- Trimethoprim 8mg/kg-Sulfamethaxazole 40mg/kg /dose twice a day for 14 days.(contraindicated infants aged < 2 months)

- The aim of treatment is limiting the spread.
- There is no benefit, if treatment (or chemoprophylaxis for contacts) is started after 21 days of onset of symptoms.¹
- After 5 days of antibiotics it is considered to be safe to allow the patient to mix with the others.²
- Children should refrain from attending school or nursery until the completion of antibiotics for 5 days.
- It is recommended that infants and children who have recovered from microbiologically confirmed pertussis complete their immunization, since natural immunity doesn't confer life long immunity.¹⁰

Chemoprophylaxis⁷

Erythromycin is given for 14 days. Same dose schedule as treatment ^{5,6,10}
Azithromycin and Clarithromycin can be given alternatively.

A contact

A contact is someone who lives in the same house as the case or who stayed overnight in the same room as the case, since the symptoms started. ³

Vulnerable contacts³

A vulnerable contact is:

- A neonate
- Unimmunized or partially immunized child under five
- Person with a chronic illness eg. asthma ,congenital heart disease

- Immunocompromised person
All vulnerable contacts should receive chemoprophylaxis.

High risk contacts ^{3,10}

- Children under 1 year
- Pregnant women in their 3rd trimester

If there is a high risk contact every one in the family should receive chemoprophylaxis irrespective of their immune status.¹⁰

High risk contacts should receive chemoprophylaxis up to 6 weeks after exposure.¹⁰

Non vulnerable contact ³

A contact who is not vulnerable as defined.

Chemoprophylaxis should be given for non vulnerable contacts who are unimmunized or partially immunized. If fully immunized no chemoprophylaxis is needed.

Complete immunization³

Complete immunization includes 3 primary injections and a booster for those under 5.

Most adults and older children may be partially immunized since vaccination provides protection for about 2-3 years.

Notification

Pertussis is notifiable on clinical suspicion.³

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CHAPTER 17

BRONCHIECTASIS

Definition

Dilatation of bronchi associated with a persistent variable inflammation of the lung.

Aetiology

- Whooping cough or measles
- Inhaled foreign body or tuberculosis (may lead to localized bronchiectasis)
- Cystic fibrosis
- Kartagener syndrome
- Immunodeficiencies
- Allergic bronchopulmonary aspergillosis
- Congenital -Williams Campbell Syndrome, tracheobronchomegaly
- Miscellaneous –alfa 1 antitrypsin deficiency
- Yellow nail syndrome
- Gastro oesophageal reflux

Clinical features

Symptoms

- Recurrent episodes of malaise
- Volume and the purulence increase during exacerbations
- During exacerbations **wheezing** occurs in the majority

Signs

- Halitosis
- Clubbing in more severe cases
- Basal coarse crepitations
- **Productive sputum** – purulent sputum indicate severe disease activity.
- Haemoptysis

Chronic cough and wheezing with clubbing in the absence of cyanotic heart disease needs exclusion of bronchiectasis.

Investigations

CXR will show dilated bronchi or multiple cysts.

Presence of tramline shadows indicate bronchial wall oedema.

37% sensitivity at lung level.

High resolution Computed Tomography (HRCT) **is the investigation of choice** in the diagnosis of bronchiectasis. It should be considered in all patients with a chronic productive cough and recurrent chest infections.

Sputum examination may reveal major pathogens such as *Staphylococcus aureus*, *Pseudomonas*, *Haemophilus influenzae* and anaerobes

Further investigations

Following investigations will be helpful in detecting an aetiology.

- Bronchoscopy if foreign body is suspected
- Immune screen (can be done at MRI Colombo)
- Ciliary studies
- Mantoux testing and Acid Fast Bascilli
- Sweat test
- Alfa 1 antitrypsin
- Aspergillus precipitins

Treatment

Postural drainage.

Antibiotics

Haemophilus influenzae and *Streptococcus pneumoniae* accounts for 70 -80 % of infections. *Moraxella catarrhalis* is also common. Chronic colonization with mucoid *Pseudomonas aeruginosa* is common in severe disease.

- Short courses (10 -14 days) for clearly defined infective episodes. In the absence of pseudomonas amoxicillin is the antibiotic of choice. If treatment fails resistance due to beta lactamase is suspected and alternatives include

Co amoxiclav or quinolones. *Moraxella catarrhalis* is usually resistant to amoxicillin.

- Prophylactic therapy for frequent recurrent exacerbations
- Continuous therapy for persistent infection

Bronchodilators are useful if there is a demonstrable airflow limitation.

Surgery such as segmental or lobar resection should be considered when localized severe disease persists despite adequate medical management.

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CHAPTER 18

LUNG ABSCESS

Definition

Lung abscess is necrosis of the pulmonary tissue and formation of cavities containing necrotic debris or fluid caused by microbial infection.

Primary lung abscess

In a previously healthy child, caused by aspiration or secondary to pneumonia.

Secondary lung abscess

Abscess is caused by a preexisting condition (eg. foreign body obstruction, tracheo-oesophageal fistula, gastro-oesophageal reflux) spread from an extrapulmonary site such as empyema, bronchiectasis, and/or an immunocompromised state, due to post operative complication of tonsillectomy and adenoidectomy, and aspiration following seizures and neurological disease.

Aetiology

Majority have mixed aerobic and anaerobic bacterial pathogens.

Anaerobes consist of 90% of the isolated organisms;

- Peptostreptococcus, bacteroides, fusobacterium species, and microaerophilic streptococcus.

Aerobes up to 50%;

- Staphylococcus aureus, streptococcus pyogenes, streptococcus pneumoniae (rarely), klebsiella pneumoniae, haemophilus influenzae, actinomyces species.

Nonbacterial pathogens;

- These microorganisms may include parasites (eg, *Paragonimus*, *Entamoeba*), fungi (eg, *Aspergillus*, *Cryptococcus*, *Histoplasma*, *Blastomyces*, *Coccidioides*), and *Mycobacteria*

Clinical Manifestations

Onset may be acute or insidious. Following *symptoms* may be elicited in the history.

- Fever - low-grade in anaerobic infections and temperatures $> 38.5^{\circ}\text{C}$ in other infections.
- Cough,
- Shortness of breath
- Chest pain,
- Vomiting
- Sputum production - Unless the abscess is completely walled off, the sputum is purulent and may be blood-streaked.
- Weight loss, night sweats and haemoptysis.

Physical findings may be secondary to associated conditions such as underlying pneumonia or pleural effusion.

- Tachpnoea and dyspnoea
- Chest wall retraction with use of accessory muscles
- Clinical findings of concomitant consolidation +/- effusion are present (eg, decreased breath sounds, dullness to percussion, bronchial breath sounds, coarse inspiratory crackles, shift of mediastinum etc.)
- Digital clubbing may develop rapidly

Diagnosis

Laboratory diagnosis

A white blood cell count with differential may reveal leucocytosis and a left shift.

Obtain sputum for Gram stain, culture, and sensitivity.

If tuberculosis is suspected, acid-fast bacilli stain and mycobacterial culture should be requested.

Blood culture may be helpful in establishing the aetiology.

Imaging

CXR

- Early in the course, chest x-ray may show a segmental or lobar consolidation.
- Parenchymal inflammation with a cavity containing an air-fluid level.
- If a lung abscess fails to communicate with a bronchus, the characteristic cavity with an air fluid level will not be seen radiographically. This often leads to initial misdiagnosis since no clear abscess can be visualized.
- When the radiograph reveals multiple cavitory lesions it usually indicates that a necrotizing pneumonitis is present. This type of presentation is usually acute and fulminant and secondary to virulent aerobic bacteria such as *S. aureus* or *K. pneumoniae*.
- Changes are more common in the posterior segments of the upper lobes and the apical segments of the lower lobes.

CT scan

- Better anatomic definition including location and size as empyema and lung abscess are sometimes difficult to distinguish from one another by CXR

Differential diagnosis of a cavitating lesion on chest radiography

Localized empyema
Infected bulla containing a fluid level
Infected congenital pulmonary lesion, such as bronchogenic cyst or sequestration
Pulmonary haematoma
Hiatus hernia
Lung parasites (eg, hydatid cyst, *Paragonimus* infection)
Actinomycosis
Wegeners granulomatosis and other vasculitides

Management

Conservative management is recommended.

Antibiotic therapy

- Should be given for a total period of 4-6 weeks (2-3 wks of intravenous followed by oral) (x)
- Antibiotics of choice are clindamycin or ticarcillin / clavulanic acid.*^{ne1}
- If gram negative bacteria are suspected or isolated add an aminoglycoside.
- Metronidazole is an effective drug against anaerobic bacteria. The experience with metronidazole in treating lung abscess has been rather disappointing because these infections are generally polymicrobial. A failure rate of 50% has been reported.
- Metronidazole, in combination with penicillin, is considered an appropriate treatment regimen for lung abscess because the penicillin will be active against the aerobic and microaerophilic streptococci that are often resistant to metronidazole.*

Dependent drainage (with appropriate positions based on the pulmonary segment) is commonly advocated using chest physiotherapy and sometimes bronchoscopy.²

Response to treatment

If there is no improvement by 7-10 days of appropriate antibiotic therapy, surgical intervention should be considered. Minimally invasive percutaneous aspiration techniques, bronchoscopic or transtracheal aspirations are useful.

Considerations in patients with poor response to antibiotic therapy include:

- Bronchial obstruction with a foreign body
- Infection with a resistant bacteria
- Mycobacteria
- Fungi.

A nonbacterial cause of cavitary lung disease may be present, such as lung infarction, cavitating neoplasm and vasculitis. The infection of a preexisting sequestration, cyst or bulla may be the cause of delayed response to antibiotics.

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CHAPTER 19

PLEURAL INFECTION AND EMPYEMA

Parapneumonic effusion and empyema have an incidence of 3.3 per 100 000 in children. It is more frequently seen in infants and young children.

Definitions

1. Parapneumonic effusion

Pleural fluid collection in association with underlying pneumonia.

2. Empyema

Collection of pus in the pleural space.

The staging of pleural fluid associated with infection.

1. Exudative (parapneumonic effusion)

Early accumulation of clear fluid with low white cell count.

2. Fibropurulent

Fibrin deposition in the pleural space.

Septation and loculation of the pleural fluid.

White cells are increased.

3. Organization

Thin intrapleural membranes organized into solid pleural peels which prevent lung expansion (*trapped lung*).

Aetiology

In a previously well child pleural effusions are secondary to acute bacterial pneumonia and less often due to chronic infections such as tuberculosis, bronchiectasis and lung abscess. Empyema can be the presentation in a child with underlying malignancy such as a lymphoma.

Common organisms

Pleural cultures are always sterile because of previous antibiotic administration before obtaining pleural fluid sample.

Responsible pathogens

- *S. aureus*,
- *S. pneumoniae*,
- *S. pyogenes*.
- *H. influenzae*
- *Mycoplasma*.
- *M. tuberculosis*

Tuberculous empyema can result from progressive pulmonary tuberculosis.

Clinical Features

There are two common presentations.

1. The child has classic symptoms of pneumonia.
 - Fever
 - Poor appetite
 - Abdominal pain
 - Patient lies on the affected side to splint the involved hemithorax.
2. A diagnosed patient with pneumonia poorly responding to the appropriate antibiotics.

Examination

- Unilateral chest pain
- Poor chest expansion
- Dullness to percussion
- Reduced or absent breath sounds
- Postural scoliosis

Investigations

1. Chest x-ray (postero- anterior) – this may show obliteration of the costophrenic angle and a rim of fluid ascending the lateral chest wall.

2. Ultra sound scan of the chest
Ultra sound scan must be used to confirm the presence of pleural fluid. It is particularly useful when there is “white out” in the chest x-ray [D]. This can also be used to guide the chest drain insertion or thoracocentesis [C].
3. Blood culture (both aerobic and anaerobic)
4. Pleural fluid - gram stain and Acid Fast Bacilli (AFB)
5. Sputum culture – If the child is expectorating sputum (which is rare)
6. Mantoux test
7. C reactive protein – as a marker of progress (if facilities are available)
8. Bronchoscopy is not routinely recommended

Treatment

1. Humidified oxygen is administered to maintain saturation over 92%. Fluid therapy is given if the child is dehydrated or unable or unwilling to drink. All cases should be treated with intravenous antibiotics which will cover the above mentioned organisms [D]. Analgesia and antipyretics are given to reduce pain. Physiotherapy is not indicated.
2. Intravenous antibiotics should be administered. Broad spectrum cover is required in hospital acquired infections, following surgery or aspiration. Whenever possible antibiotic administration should be guided by microbiological report [D]. (x)

Oral antibiotic may be required for 1- 4 week or longer if there is residual infection.

If a child has enlarging effusion or respiratory compromisations , intercostal tube drainage should be instituted; repeated taps are not recommended.

Intrapleural fibrinolytics shorten hospital stay and are recommended for any complicated parapneumonic effusion (thick fluid with loculation) or empyema (overt pus) [B]. (z)

3. Consider referring to a thoracic surgeon when the child has persistent pleural sepsis with pleural fluid collection despite chest tube drainage and appropriate antibiotics.

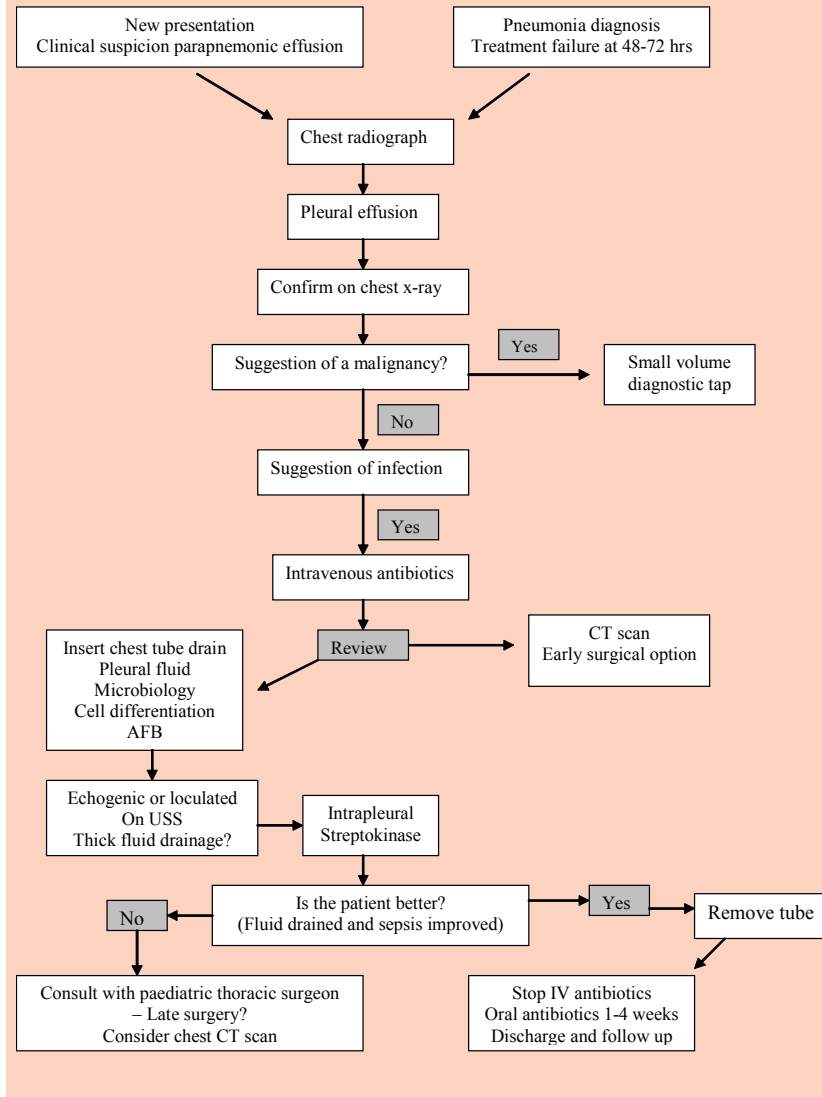
The available surgical options are:

- (i). Video assisted thoacoscopic surgery (VATS) - achieves debridement of fibrinous pyogenic material, breakdown of loculation and drainage of pus under direct vision.
 - (ii). Mini thoracotomy – debridement and evacuation is similar to VATS but it is an open procedure.
4. Children should be followed up after discharge until they have recovered completely and their chest radiograph have returned to near normal [D].

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The scheme for management of pleural infection in children



CHAPTER 20

ACUTE OTITIS MEDIA (AOM)

Definition

Acute otitis media is a disease of infancy and early childhood defined by the presence of inflammation and fluid in the middle ear and accompanied by at least one sign of an acute illness.¹

Aetiology

Pathogenic bacteria are isolated in 65 – 75% of cases.²

- 40% *Streptococcus pneumoniae*
- 25 – 30% *Haemophilus influenzae*
- 10 – 15% *Moraxella catarrhalis*

Respiratory viruses are commonly found in association with pathogenic bacteria.³

- Respiratory syncytial virus

Clinical Manifestations

History

Local symptoms

- Earache (only symptom with positive predictive value)¹
- Discharge from the ear (otorrhoea)

Systemic symptoms

- Fever
- Symptoms of upper respiratory tract infection
- Irritability
- Restless sleep
- Vomiting
- Diarrhoea
- Lethargy
- Anorexia

Child under 2 years

- Systemic symptoms are non specific
- Tugging or rubbing of the ear indicates earache.
- Evidence of conjunctival symptoms and rhinorrhoea are associated with acute otitis media

The pain will be relieved by rupture of the tympanic membrane.

Examination

When AOM is suspected examination of the ear with an auriscope is mandatory. (*Examination with a torch will not visualize the tympanic membrane*).

To visualize the tympanic membrane,

- In a young child the pinna is pulled in a horizontal and backward direction
- In an older child the pinna is pulled upwards and backwards towards the occiput.

Auriscopy

Auriscopic evidence of middle ear effusion: ⁴

- Otorrhoea
- Bulging of the tympanic membrane
- Opaque drum (normally shiny)
- Air – fluid level behind the tympanic membrane
- Impaired drum mobility - *This can be demonstrated by a pneumatic auriscope.*

Auriscopic evidence of middle ear inflammation:

- Distinct erythema of the TM
(*excessive crying can cause mild erythema of the TM*)

Definite diagnosis is defined as a case with the following 3 criteria ⁵

- 1) Rapid onset
- 2) Signs of middle ear effusion
- 3) Signs and symptoms of middle ear inflammation

Severe illness: moderate to severe otalgia with fever higher than 39 ° C

Non severe illness: mild otalgia and fever of 39 ° C or less in the past 24 hours

Differential Diagnosis

(1) *Otitis media with effusion (Glue ear)*

A child with evidence of middle ear effusion with no systemic symptoms of acute illness and no signs of acute inflammation.⁶

(2) *Meningitis*

Meningitis should be excluded in a young child under 2 years with non specific symptoms especially fever, lethargy and irritability associated with poor feeding and drowsiness.

(3) *Otitis externa*

A child with purulent discharge in the absence of fever and systemic symptoms.

(4) *Urinary tract infection (UTI)*

A young child with UTI can present with nonspecific systemic signs.

Common important complications

Intracranial	Extracranial
<ul style="list-style-type: none">• Facial nerve palsy• Acute mastoiditis• Inner ear labyrinthitis• Bezold abscess• Gradenigo syndrome	<ul style="list-style-type: none">• Meningitis• Extradural/ subdural abscess

Management

- 1) Analgesia – Paracetamol 15mg/kg/dose (6 hourly)
Ibuprofen 5- 10 mg/kg /dose (can be given 8 hourly)
- 2) Observation without use of antibacterial agents in a child with uncomplicated AOM is an option. [B]
This is based on:
 - Diagnostic certainty
 - Age
 - Illness severity
 - Assurance of follow-up

Age	Definitive diagnosis	Uncertain diagnosis
Under 6 months	Antibiotics	Refer to a paediatrician
6 m to 2 y	Antibiotics	Refer to a paediatrician in severe illness; Observation in non severe illness
Over 2 y	Antibiotics in severe illness Observation in non severe illness	Observe

- 3) If a decision is made to treat with an antibacterial agent
Amoxycillin 30 mg/kg/dose 8hrly

Other options of oral antibiotics are
Cloxacillin 15 mg/kg/dose 6 hourly
Cephalexin 12.5mg/ kg twice daily

Children under 6 years and children with severe disease a 10 day course is recommended. Children 6years or older with non severe disease 5-7 day course is recommended. ⁵

- 4) If the patient **fails to respond** to the initial management option within 48 to 72 hours, the clinician must reassess the patient to confirm AOM and exclude other causes of illness.⁵

If AOM is confirmed in a patient managed with observation only, the clinician should begin antibacterial therapy. [B]⁷

If the patient was initially managed with an antibacterial agent(s), the clinician should change the antibacterial agent(s).

Can use coamoxiclav, cefuroxime, ceftriaxone, cefixime, cefederm and cefaclor.

5) No definitive place for *topical antibiotics* and *nasal decongestants* [A]⁷

6) **Follow up visit** in:⁷

- A couple of days, in an infant with severe infection or child with persisting pain.
- Two weeks, in a child who has been having frequent recurrences.
- One month, in a child who had a sporadic episode with prompt response to symptomatic treatment

7) **ENT referral** is indicated if acute otitis media is,

- Complicated by mastoiditis (inflammation in the post auricular area with displacement of the pinna inferiorly and anteriorly)
- Associated with facial nerve palsy
- Recurrent acute otitis media (4 episodes over 6 months)

Otitis media with effusion (OME) / Glue ear

Definition

Inflammation of the middle ear accompanied by middle ear effusion without symptoms and signs of acute inflammation.^{6,7}

Aetiology

Middle ear fluid cultures are usually sterile. 30% of the time pathogens found in acute otitis media are recoverable.

Bacteria

- Streptococcus pneumoniae
- Haemophilus influenzae
- Moraxella catarrhalis

Virus

- Respiratory syncytial virus

Children at risk

- Those in day care
- Those with older siblings
- Those with parents who smoke
- Those who present with hearing or behavioural problems

Clinical Manifestations

History

- Mostly asymptomatic.
- Hearing impairment. (this is the main symptom although not identified in infants and young children)
- Language and speech delay.
- Behavioural symptoms such as clumsiness and inattentive behaviour.
- Poor social interaction and education performance.

Auriscopy

Tympanic membrane will appear,

- Retracted/concave with an abnormal colour such as yellow amber or blue opaque
- Air fluid level or air bubbles may be present
- Reduced motility demonstrated by a pneumatic auriscope.

Management

- 1) Antibiotics are not needed in most.⁷
- 2) Antibiotics indicated when there is evidence of bacterial upper respiratory tract infection. Give a 2-4 week course of amoxicillin²
- 3) No place for decongestants, mucolytics and antihistamines [B]⁷
- 4) No place for topical or oral systemic steroids [B]⁷

Follow up

- OME is well recognized to relapse and remit.
- Commonly resolves at 7-8 years.
- Needs 2-3 monthly reviews.

Specialised ENT Opinion sought⁷

- Children under 3 years with persistent bilateral OME should have hearing assessed. If audiometry is not possible ENT referral should be done.
- If hearing loss is less than 25dB with no speech and language, development or behavioural problems child can be safely managed with watchful waiting. Audiometry should be done to exclude more serious hearing loss.

- Children under years with persistent bilateral OME should be referred.
- Children with OME and language and speech, developmental and behavioral problems should be referred.

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Annexure – I

Intubation

1. Ensure that adequate ventilation and oxygenation by face mask before intubation.
2. If endotracheal intubation is not achieved in 30 seconds, discontinue the attempt, and start bag and mask ventilation before the next attempt.
3. Select appropriate laryngoscope and check the brightness of the light.
4. Select an appropriate tube size.

Size of the ETT/ internal diameter (mm)

- $(\text{Age} / 4) + 4$

Length of the tube (cm)

- $(\text{Age} / 2) + 12$ for an oral tube
- $(\text{Age} / 2) + 15$ for a nasal tube

5. Ensure manual immobilization of the neck.
6. Hold the laryngoscope in the left hand, and insert it into the right side of the mouth, displacing the tongue to the left.
7. Visualize the epiglottis, and place the tip of the laryngoscope in the vallecula.
8. Gently but firmly lift the handle, being careful not to lever on the teeth.
9. Insert the endotracheal tube into the trachea, concentrating on how far the tip is placed below the vocal cords. The tip should lie 2- 4cm below the vocal cords depending on the age. If the tube has a “vocal cord level” block marker, place this immediately below the vocal cords. Be aware that flexion or extension of the neck may cause migration downwards or upwards, respectively.
10. Do not use a cuff unless the child is an adolescent.
11. Inspect the chest movement and auscultate the chest for air entry (including the axillae) and the epigastrium.
12. Once the tube is in place obtain a chest radiograph to confirm the correct position.
13. In the case of epiglottitis or croup select an endotracheal tube which is one size smaller for the age (1mm less).

Annexure - II

The Guideline Committee

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Resource

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Dr N Gamathige	Paediatric Registrar

Annexure – III

Abbreviations

AFB	-	Acid Fast Bacilli
AOM	-	Acute Otitis Media
CAP	-	Community Acquired Pneumonia
CDC	-	Centre for Disease Control and Prevention
EBV	-	Epstein Barr Virus
ESBL	-	Extended Spectrum Beta Lactamase
GABHS	-	Group A Beta Haemolytic Streptococci
HiB	-	Haemophilus influenzae type B
HRCT	-	High Resolution Computed Tomography
LRTI	-	Lower Respiratory Tract Infections
MIC	-	Minimum Inhibitory Concentration
MRI	-	Medical Research Institute
MRSA	-	Methicillin Resistant Staphylococcus aureus
NICU	-	Neonatal Intensive Care Unit
OME	-	Otitis Media with Effusion
PCR	-	Polymerase Chain Reaction
RSV	-	Respiratory Syncytial Virus
SaO ₂	-	Saturation of Oxygen
TM	-	Tympanic membrane
UTI	-	Urinary Tract Infection
VATS	-	Video Assisted Thoracoscopic Surgery

