AIR-BORNE DISEASES

AIR-BORNE DISEASES

Air born disease **:-Are diseases transmitted through dissemination of microbial agent by air to a suitable portal of entry, usually the respiratory tract.**

- **Agent enter the body via the respiratory tract.**
- **When a patient or carrier of pathogens talks, coughs, laughs, or sneezes, he/she discharges fluid droplets.**
- **The smallest of these remain up in the air for some time and may be inhaled by a new host.**

MEASLES (GERMAN RUBELLA)

- **An acute highly communicable viral disease especially contagious in catarrhal (secretion) stage before the rash appears.**

- **Common and often fatal in developing areas.**

- **Aetiology: measles virus – paramyxovirus**

Reservoir: **Humans**

EPI:

- **Measles was common in childhood so that more than 90% of people had been infected by age of 20; few went through life without any attack.**

CONT….

- **In contrast, in third world countries, measles infection has its greatest incidence in children under 2 years of age.**
- **The maximum incidence is b/n 6 months and 5 years.**
- **In general measles mortality is highest in children < 2 years and in adults.**
- **The disease is a serious problem with a high mortality (10%) with malnutrition being an important factor in developing countries.**

MOT:



- **Airborne droplets released when an infected person sneezes or coughs.**
- **Contact with nose and throat secretions of infected people.**
- **The infection is acquired through the upper respiratory tract or conjunctiva.**
- **Cases can infect others for several days before and after they develop symptoms.**
- **Spreads easily in overcrowded areas (schools, military barracks (camps), health facilities etc.**
- **Greater than 94% herd immunity (group immunity) may be needed to interrupt community transmission.**

I/P:

- **7-18 days from exposure to onset of fever.**

POC:

- **Slightly before the Prodromal period to 4 days after the appearance of the rash and minimal after the second day of rash.**

Susceptibility and resistance:

- **All those who are *non-vaccinated* or have not had the disease are susceptible.**

- Permanent immunity is acquired after natural infection or immunization.

CLINICAL MANIFESTATIONS

A. Prodromal phase - takes 3~4 days.

- Stepwise increase in fever to 103°F or higher.

- Catarrhal inflammation of URT (Cough, coryza, conjunctivitis, malaise, sneezing, rhinitis, congestion).

- Koplik's spots (are pathognomonic in measles, appear on the buccal and lower labial mucosa opposite the lower molars as White spots inside the mouth).

- **Transient prodromal rashes.**



- ***B. Eruption stage:*** **-**
- **it occurs 4~5 days after fever; but the 4th day is most common.**
- **Shape: maculopapular rash.**

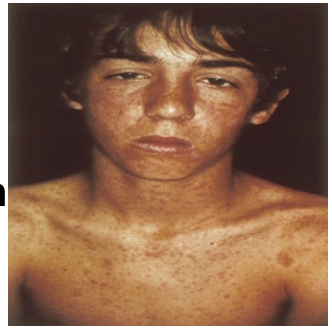
Sequence:



- **Behind the ear → along the hairline → face → neck → chest → back→ abdomen→ limbs→ hand and feet (palm, sole).**
- **Rash persists 5-6 days and fades in order of appearance.**

C. Convalescent stage (Last 6-14days)

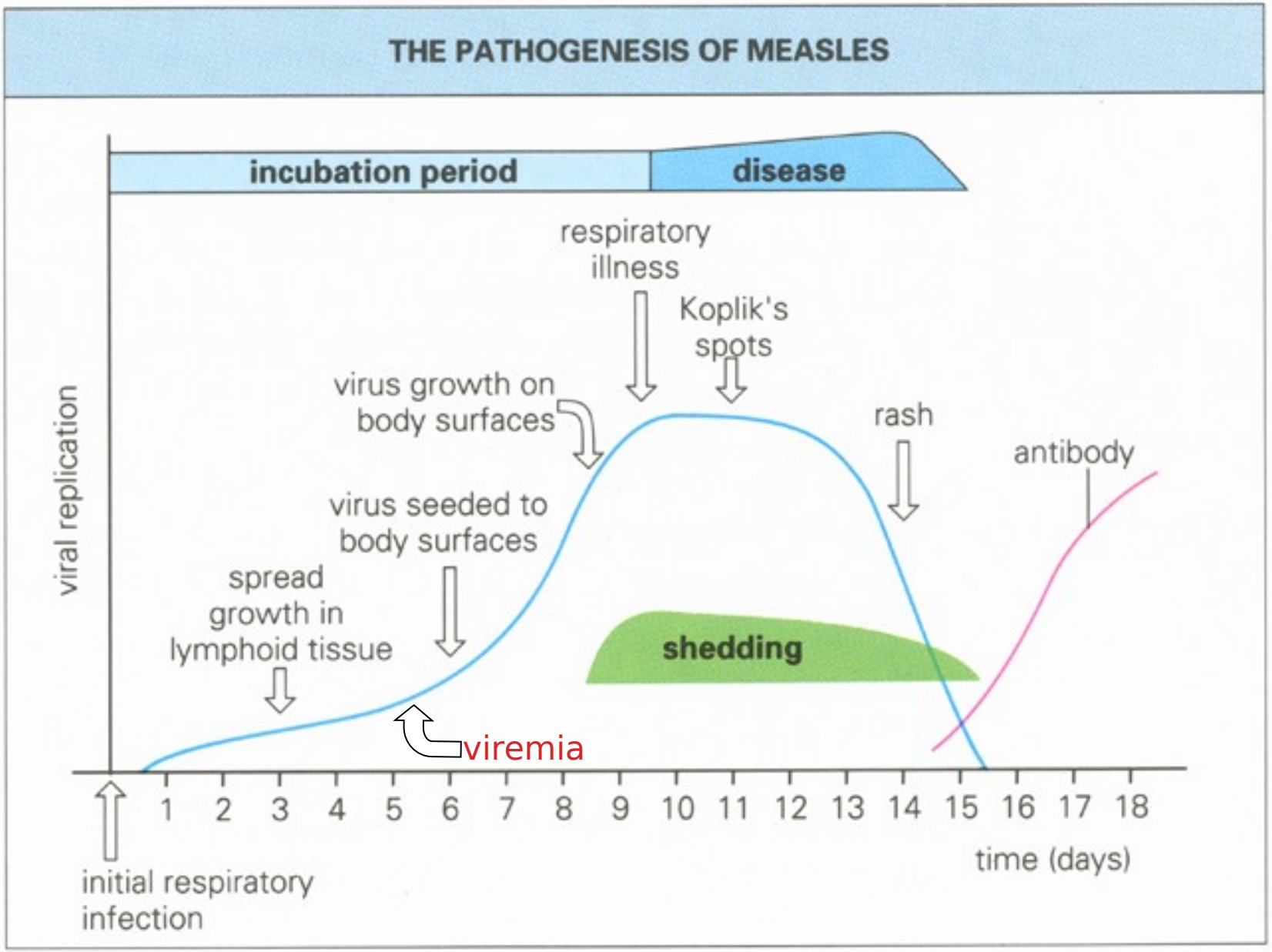
- **Brown staining.**
- **Fine branny desquamation**





MEASLES PATHOGENESIS

- Respiratory transmission of virus
- Replication in nasopharynx and regional lymph nodes
- Primary viremia 2-3 days after exposure
- Secondary viremia 5-7 days after exposure with spread to tissues



n Mims, Playfair, Roitt, Wakelin and Williams (1993) Medical Microbiology

DIAGNOSIS

- **Based on clinical and epidemiological grounds.**
- **Fever and rash plus one of these; cough, coryza or conjunctivitis.**
- **Serological- raised IgM or IgG**

CLINICAL MANIFESTATION

- **Generalized rash lasting >3 days, and**
- **Temperature 101°F (>38.3°C), and**
- **Cough or coryza or conjunctivitis**

COMPLICATIONS

- **Unimmunized children < 5 years and infants are at highest risk of measles and its complications.**

- **Pneumonia : cause death as the virus weakens the immune system.**

- **Complications are due to:**

  1. Bacterial infection

  2. Measles virus which damages respiration and intestinal tracts.

  3. Vitamin A deficiency.

ACUTE COMPLICATIONS:

- **Bronchopneumonia: the most important complication and responsible for most deaths.**
- **Diarrhoea (dysentery and persistent diarrhoea)**
- **Laryngo-tracheo bronchitis.**
- **Conjunctivitis and corneal ulceration.**
- **Otitis media, mastoiditis…….**

LONG TERM COMPLICATION

- Increased susceptibility to other infection (damages immune system)
- Blindness
- Sub acute sclerosing panencephalitis

THE CLINICAL IMPACT OF MEASLES		
site of virus growth	**well nourished child good medical care**	**malnourished child poor medical care**
Lung	temporary respiratory illness	life threatening pneumonia
Ear	otitis media quite common	otitis media commoner more severe
Oral mucosa	Koplik's spots	severe ulcerating lesions
Conjunctiva	conjunctivitis	severe corneal lesions secondary bacterial infection blindness may result
Skin	maculopapular rash	haemorrhagic rashes may occur ('black–measles')
Intestinal tract	no lesions	diarrhoea – exacerbates malnutrition, halts growth, impairs recovery
Urinary tract	virus detectable in urine	no known complications
Overall impact	**serious disease in a small proportion of those infected**	**major cause of death in childhood (estimated 1.5 million deaths/yr worldwide)**

Mims et al., Medical Microbiology 1993

MEASLES COMPLICATIONS

Condition	Percent reported
Diarrhea	8%
Otitis media	7%
Pneumonia	6%
Encephalitis	0.1%
Hospitalization	18%
Death	0.2%

TREATMENT

- **No specific Rx**
- **General nutritional support and Rx of Dehydration**
- **Antibiotics only to ear and severe respiratory infection**
- **Vitamin A two doses in 24 hours and …**
- **Rx of complication**

PREVENTION

- **Educate the public about measles immunization.**
- **Immunization of all children (<5 years of age) who had contact with infected children.**
- **Provision of measles vaccine at nine months of age.**

PERTUSSIS (WHOOPING COUGH)

- An acute disease of the respiratory tract caused by bacteria that live in the mouth, nose and throat.

- The name ‘pertusis’ means “violent cough”, and was first used to describe the disease in 1679.

- In China, the disease is known as “the cough of 100 days”

Agent: Bordetella pertusis

CONT....

EPI:

- It was an endemic disease commonly to children especially young children everywhere in the world.
- A marked decline has occurred in incidence and mortality rates during the past four decades.
- Out breaks occur periodically. Endemic in developing world and 90% of attacks occur in children under 6 years of age.
- Pertussis is highly contagious, with an 80% secondary attack rate among susceptible people.

PERTUSSIS

- **There are 8 species of bordetella - but *B pertussis* and *B parapertussis* cause most human disease (most cause respiratory disease, otitis, sinusitis, but occasional species are associated with wound infections, sepsis, and septic arthritis.**

- **There are no known animal reservoirs for *B pertussis,* and the organism does not survive for prolonged periods in the environment.**

- **No long-term carrier state had been identified, but asymptomatic culture positive persons can be detected during known exposures.**

- **Most dangerous in children of age less than 1 year and the youngest non-immunized children.**
- **Immunity from the vaccine is of limited duration (~10 - 12 years), so fully immunized children were protected, but adults had little or no immunity.**

- **Reservoir**
  - Humans

- **MOT:**

  - Primarily by direct contact with discharges from respiratory mucus membrane of infected persons by air borne route, probably by droplets.

- **Indirectly by handling objects freshly soiled with nasopharyngeal secretions.**

- **Spread through direct contact of respiratory secretions.**

- **Most contagious during first few stages of infection.**
-
- **Resides in upper airway pathways, mostly the trachea and bronchi.**
- **Very contagious**

I/p:

- **1-3 weeks**



- **POC:**

➢ **7 days after a person has been exposed to 3 weeks after the start of the coughing.**

➢ **Highly communicable in early catarrhal stage before the paroxysmal cough stage.**

➢ **Susceptibility and Resistance:**

➢ **Susceptibility to non immunized individuals is universal.**

➢ **One attack usually confers prolonged immunity but may not be lifelong.**

CLINICAL MANIFESTATIONS

1. Catarrhal phase

- **After an incubation period of 1-3 weeks, signs and symptoms of the catarrhal phase begins. Most communicable stage.**

- **Symptoms include Mucoid rhinorrhea, lacrimation, sneezing, conjunctival injection, malaise, low grade fever, and are indistinguishable from those of many other URI's.**

- **After a few days and up to a week of these symptoms, a dry nonproductive cough develops, and this evolves into a characteristic paroxysmal phase.**

- **Cough becomes irritating, hacking (cutting), and nocturnal becoming more severe.**

CONT…

- **Patients are most contagious during the catarrhal phase and during the first two weeks after the onset of coughing.**

- **Prodromal symptoms during this phase can include complaints of pharyngeal discomfort.**

- **During this phase, patients can develop a marked leukocytosis, with WBC counts greater than 50,000, with a relative lymphocytosis (less common in adults).**

2. Paroxysmal phase

- The cough paroxysm consists of a short series of expiratory bursts, followed by an inspiratory gasp, which results in the typical “whoop”.
- The paroxysmal phase usually lasts 1-6 weeks, but can last up to 10 weeks.
- Not all children with pertussis exhibit the characteristic whoop, and it is fairly uncommon in infants, who may have apneic episodes.



CONT….

- **In adults, the disease is generally milder, but the paroxysmal cough may be just as prolonged.**

- **Paroxysms can number more than 30 per 24 hours, and are more frequent at night, and can be stimulated by external stimuli, such as noises or cold air.**

- **Classically they may end with a vomiting episode. They can be associated with sweating, flushing and syncope. Patients may cough up thick yellow plugs. Pertussis is generally more severe in infants**
- **But presentation can be more atypical in infants, as well as partially immunized children and previously immunized adolescents and adults.**

- **In these groups the catarrhal phase can be shortened, and the true whooping phase may be absent.**

3. Convalescent phase

- The convalescent phase begins with a decrease in the intensity of the cough and paroxysms, but can still last for weeks.

- It is not clear if pertussis can cause long term impairment of pulmonary function.

IN GENERAL, PROGRESSION OF WHOOPING COUGH…..

3 Stages

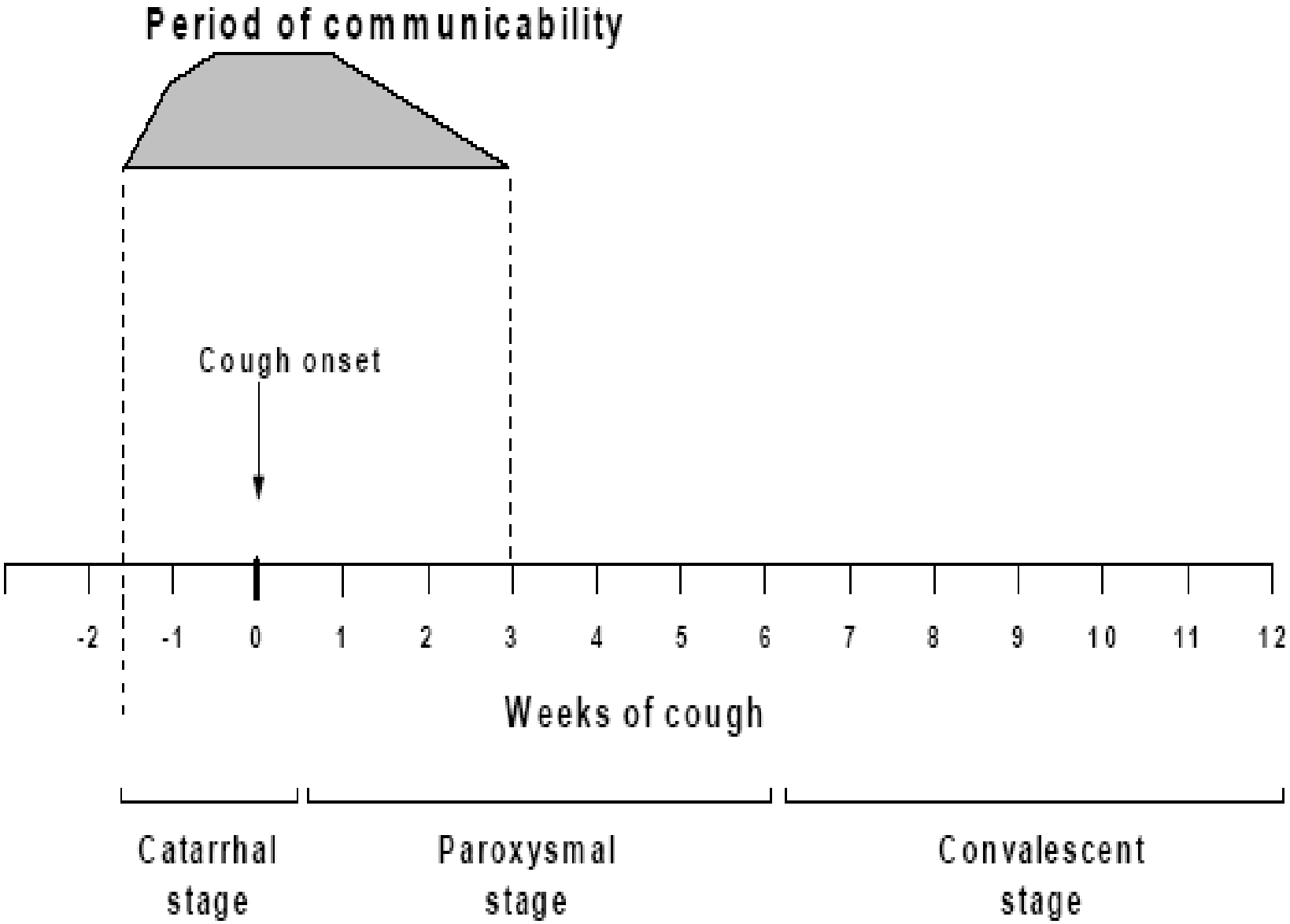
- ***1st Stage- Catarrhal Stage 1-2 weeks.***
  - Runny nose, sneezing, low fever, and a mild cough (common mistaken for cold).

- ***2nd Stage- Paroxysmal Stage 1-6 weeks.***

  - Whooping cough, which consists of bursts or paroxysms of numerous, rapid coughs, severity of the infection is at its greatest stage.

  - These coughing episodes seem to be due to a difficulty in expelling mucus from the tracheobronchial tree.
  - These attacks usually end with a long inspiratory effort which is usually accompanied by the high pitched whoop from which the disease gets its name.
  - The characteristic whoop may not be observed in young infants due to a lack of strength, though these coughing episodes occur.

- ***3rd Stage- Convalescent Stage weeks-months***.
  - Gradual recovery starts


Period of communicability
Cough onset
-2
-1
0
1
2
3
4
5
6
7
8
9
10
11
12
Weeks of cough
Catarrhal stage
Paroxysmal stage
Convalescent stage

Clinical Progression of Pertussis

	Incubation	Catarrhal	Paroxysmal	Convalescent
Duration	7–10 days	1–2 weeks	2–4 weeks	3–4 weeks (or longer)
Symptoms	None	Rhinorrhea, malaise, fever, sneezing, anorexia, Inflammation of respiratory mucosal memb.	Repetitive cough with whoops, vomiting, leukocytosis	Diminished paroxysmal cough, development of secondary complications (pneumonia, seizures, encephalopathy) or death
Bacterial culture		Most infectious, but generally not yet diagnosed		

COMPLICATIONS

- The principal complications of pertussis are secondary infections - otitis media or pneumonia (either secondary to pertussis or other organisms).

- Aspiration can occur secondary to the whooping and associated gasping.

- Patients can develop subconjunctival hemorrhages, facial and truncal petechiae, pneumothoraces, hernias, and rectal prolapse from the severe coughing.

- Rib fractures and herniated discs can also occur.

COMPLICATIONS CONT….

- **CNS abnormalities can occur particularly in children 6 months and younger.**
- These include convulsions in 1.4 %, and encephalopathy in 0.2%.
- Possible etiologies include hypoxia, hypoglycemia, or direct effects of pertussis toxin.



COMPLICATIONS CONT…



Children

Hypoxia

Apnea

Pneumonia

Seizures

Adults

- Pneumonia
- Rib Fracture
- Weight Loss
- Hernias
- UrinaryIncontinence/ females



DIAGNOSIS:

- **Difficult to distinguish it from other URTI**

- **History and physical examination at phase two ensure the diagnosis**

  - Marked lymphocytosis

TREATMENT

- **Erythromycin: 30-40 mg/kg PO QID for 10 days to treat the infection in phase one but can continue to decrease transmission in phase two.**

- **Antibiotics such as**
  - Azithromycin
  - Clarithromycin can also be used.

- **Antibiotics for super infection like pneumonia b/c of bacterial invasion due to damage to cilia.**
- **Fluid and electrolyte needs to be replaced.**
- **Oxygen therapy**

PREVENTION

- **Educate the public about the dangers of whooping cough and the advantages of initiating immunization at 6 weeks of age.**

- **Consider protection of health worker at high risk of exposure by using erythromycin for 14 days.**

- **Vaccination with penta-valent.**

PREVENTION

Children's have to take Pentavalent vaccine.



Good hygiene



Cover mouth/nose when coughing and sneezing.



DIPHTHERIA

- An acute bacterial disease of the mucus membrane of the RT involving primarily tonsils, pharynx, nose, occasionally other mucus membranes or skin and some times the conjunctiva or genitalia.

- Typical sign: specific membrane formation (pseudomembrane, in Greek *diphtheria*, meaning leather, as tough as leather).

- Diphtheria is an acute, toxin-mediated disease caused by toxigenic *Corynebacterium diphtheriae.*

- It’s a very contagious and potentially life-threatening bacterial disease.

CONT...

Aetiology:

- Corynebacterium diphtheriae. The major virulence determinant is an exotoxin, diphtheria toxin.

- After binding to the host cells, the active subunit will interrupt the protein synthesis of the target host cell and results in cell death.

- Toxoid made from diphtheria toxin can be used as vaccine.

CONT…

Epidemiology

- Disease of colder months in temperate zone, involving primarily non-immunized children less than 15 years of age.
- It is often found among adult population groups whose immunization was neglected.
- Unapparent, cutaneous and wound diphtheria cases are much more common in the tropics.

CONT…

- ***Sources of infection:*** Patients and asymptomatic carriers
  - Patients: Transmission time is variable, usually persist 12 days or less, and seldom more than 4 weeks, without antibiotics.
  - Asymptomatic carriers (even important): 1% in population, but may be up to 10-20% during outbreaks in the past.
  - Most of them were transient carriers (less than 2 weeks), but chronic carriers may shed organisms for 6 months or more.

CONT...

Transmission

- Transmission is most often person-to-person spread from the respiratory tract (by small droplet when coughing or sneezing).
- Rarely, transmission may occur from skin lesions or articles soiled with discharges from lesions of infected persons.

- **Reservoir: Humans**

- **I/P: usually 2-5 days**

POC:

- Variable. Until virulent bacilli disappeared from discharges and lesion: usually 2 weeks or less.

- ***Susceptibility and Resistance:***

- Susceptibility is universal.

- **Infants borne to immune mothers are relatively immune, but protection is passive and usually lost before 6 months.**
- **Recovery from clinical dx is not always followed by lasting immunity.**

- **Immunity is often acquired through unapparent infection.**
- **Prolonged active immunity can be induced by diphtheria toxoid.**

PATHOGENESIS AND PATHOLOGY

- **Susceptible persons may acquire toxigenic diphtheria bacilli in the nasopharynx, skin, middle ear or anterior nares.**

- **The organism produces a toxin that inhibits cellular protein synthesis and is responsible for local tissue destruction and pseudomembrane formation.**

- **The pseudomembrane consists of coagulated fibrin, inflammatory cells, destructed mucous tissues and bacteria.**
- **The pseudomembrane in larynx, trachea or bronchi may have the potential for airway obstruction.**

PATHOGENESIS AND PATHOLOGY

- **The toxin produced at the site of the pseudomembrane is absorbed into the bloodstream and then distributed to the tissues of the body.**

- **The toxin is responsible for the major complications of Myocarditis and neuritis, and can also cause low platelet counts (thrombocytopenia) and protein in the urine (proteinuria).**

- **The rapidity of onset, the severity of disease, and the ultimate outcome are determined by:-**

- ***the site of infection***
- ***the virulence of the strain and the status of host immunization***
- ***site and magnitude of the local lesions (pseudomembrane).***

PATHOGENESIS

- **Entry ------ the bacilli multiply locally in the throat and elaborate a powerful exotoxin ----- produce local and systemic symptoms.**

Local lesions:

- **Exotoxin causes necrosis of the epithelial cells and liberates serous and fibrinous material which forms a grayish white pseudomembrane.**

- **The membrane *bleeds on being dislodged.***
- **Surrounding tissue is inflamed and edematous**

Systemic lesions:

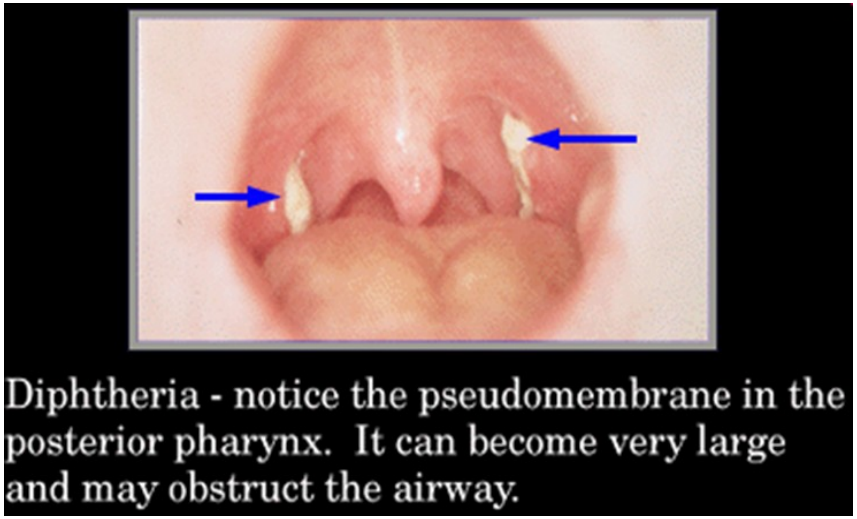
- **Exotoxin affects the heart, kidney and CNS**

CLINICAL MANIFESTATIONS

- **This disease can involve almost any mucous membrane.**

The major sign is pseudomembrane.

- **The typical pseudomembrane is adherent to the tissue, and forcible attempts to remove it cause bleeding.**



Diphtheria - notice the pseudomembrane in the posterior pharynx. It can become very large and may obstruct the airway.



CLINICAL MANIFESTATIONS

- **For clinical purposes, it is convenient to classify diphtheria into four categories depending on the site of disease (or pseudomembrane).**

1. Pharyngeal diphtheria

- **It's the most common type, >80%.**
- **The sites of infection are the tonsils and the pharynx.**

- **Infection at these sites is usually associated with substantial systemic absorption of toxin.**

TYPE OF PHARYNGEAL DIPHTHERIA

Mild type

- Symptoms: malaise, sore throat, anorexia, and low-grade fever.
- Within 2-3 days, **small patches of white pseudomembrane** on the tonsils are found.
- Often occurs in outbreaks.

Ordinary type

- Symptoms: malaise, sore throat, anorexia, vomiting and middle-grade fever.
- Typical adherent, **bluish- or grayish-white pseudomembrane** forms on the congested tonsils.
- With lymph nodes enlargement in the sub mandibular areas of neck.

Grave type

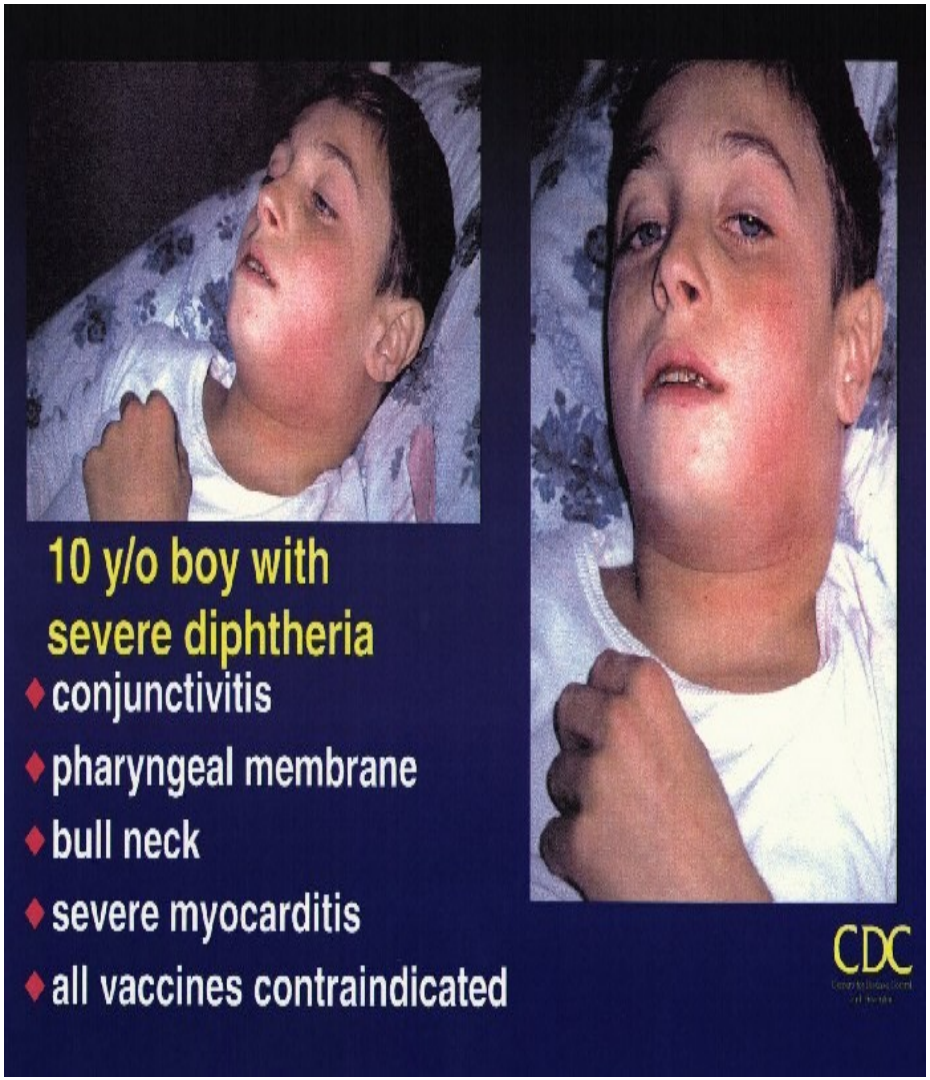
- Serious early symptoms, high-grade fever.
- Skin becomes pale, tachycardia, blood pressure may be normal or slightly depressed (Shock).
- **Large, thick pseudomembrane, and grayish-green or black in color** there has been bleeding, covering the tonsils, uvula, and some soft palate, odoriferous in mouth.
- With enlarged lymph nodes in the submandibular areas of neck.

TYPE CONT.…

Extra-grave type

- **Tachycardia, Tachypnea, depressed blood pressure. Highly congested tonsils and pharynx.**
- **The pseudomembrane is larger than that of grave type, black in color.**
- **Extensive pseudomembrane formation may result in respiratory obstruction.**
- **Patients develop marked edema of the submandibular areas and the anterior neck along with lymphadenopathy, giving a characteristic "bullneck" appearance.**
- **Complications, include myocarditis and thrombocytopenia may occur.**
- **May even die within 6 to 10 days.**

‘BULL NECK’


10 y/o boy with
severe diphtheria
conjunctivitis
pharyngeal membrane
bull neck
severe myocarditis
all vaccines contraindicated
CDC



2. LARYNGEAL DIPHTHERIA

- **Laryngeal diphtheria can be either an extension of the pharyngeal form (often) or the only site involved (rarely).**

- **Symptoms include mild fever (with little absorption of toxin), dyspnea, hoarseness, and a barking cough.**

- **The pseudomembrane can lead to airway obstruction, coma, and death.**

3. ANTERIOR NASAL DIPHTHERIA

- **The onset is indistinguishable from that of the common cold and is usually characterized by a mucopurulent nasal discharge (containing both mucus and pus) which may become blood-tinged.**

- **A white pseudomembrane usually forms on the nasal septum.**

- **The clinical symptoms of this disease is usually fairly mild because of apparent poor systemic absorption of toxin in this location, and can be terminated rapidly by antitoxin and antibiotic therapy.**

4. CUTANEOUS AND OTHER SITE DIPHTHERIA

- **Skin infections are quite common in the tropics and are probably responsible for the high levels of natural immunity found in these populations.**

- **Skin infections may be manifested by a scaling rash or by ulcers with clearly demarcated edges and pseudomembrane.**

- **In general, the severity of the skin disease appears to be less than in other forms of infection.**

- **Other sites of involvement include the mucous membranes of the conjunctiva and vaginal area, as well as the external auditory canal.**

SKIN LESIONS





DIAGNOSIS

- **Bacteriologic exam of discharges from lesions.**
- **Based on clinical and epidemiological grounds.**
- **Consider in patients with sore throat *cervical adenopathy or swelling, and low grade fever accompanied by nasal discharge, systemic toxicity, hoarseness, stridor, palatal,* paralysis with or without pseudo membrane.**

TREATMENT

Principles :

- **Neutralization of free circulating toxin by administration of antitoxin.**
- **Antibiotic to eradicate bacteria**
- **Supportive and symptomatic therapy**
- **Management of complication**

ANTITOXIN

Diphtheria antitoxin :

- **Pharyngeal or laryngeal diphtheria of 48 hours duration : 20,000 to 40,000 units.**
- **Nasopharyngeal lesions : 40,000 – 60,000 units**
- **Extensive disease of 3 or more days duration or patient with swelling of neck : 80,000 – 120,000 units.**
- **Antitoxin may be repeated if the clinical improvement is slower.**

TREATMENT

- **Erythromycin 500mg for 2 weeks but 1 week for cutaneous form or**
- **Procaine penicillin 600,000 IU (50000-75000u/kg/24hrs) IM BID for 14 days or Single dose of B-penicillin IM**
- **Supportive and symptomatic therapy:**
- **Bed rest for 2 – 3 weeks (to reduce cardiac complications)**
- **Antipyretics and sedative (if required)**
- **Monitor rate and rhythm of the heart**

DIPHTHERIA COMPLICATIONS

- **Mostly attributable to toxin**
- **Severity generally related to extent of local disease.**
- **Most common complications are Myocarditis and toxic neuritis with palsy**
- **Death occurs in 5%-10% for respiratory disease**

MENINGITIS

- **An acute bacterial disease that causes inflammation of the meninges (esp. the pia and arachnoid space).**
- **Meningitis: inflammation of the leptomeninges (the tissues surrounding the brain and spinal cord).**

Meningitis

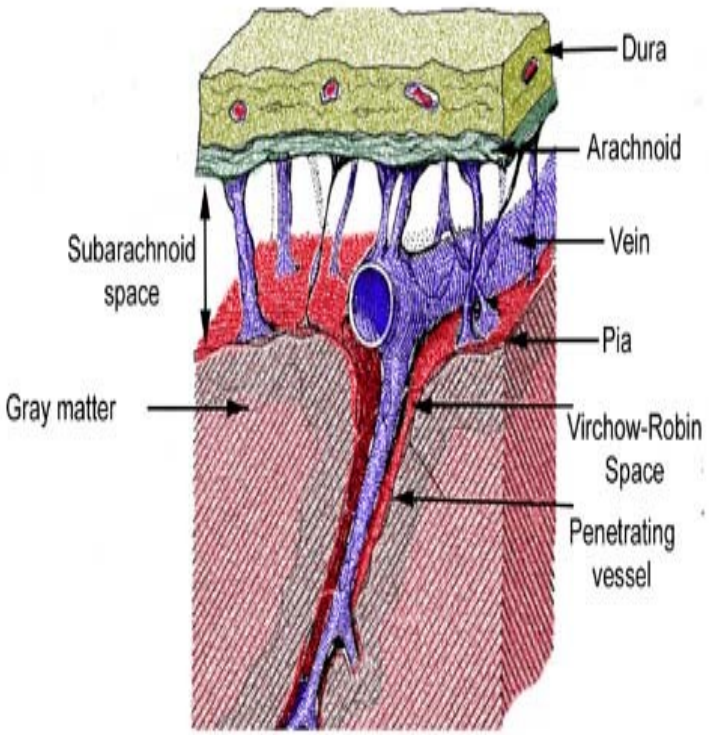
- **Infection of the subarachnoid space with meningeal involvement.**

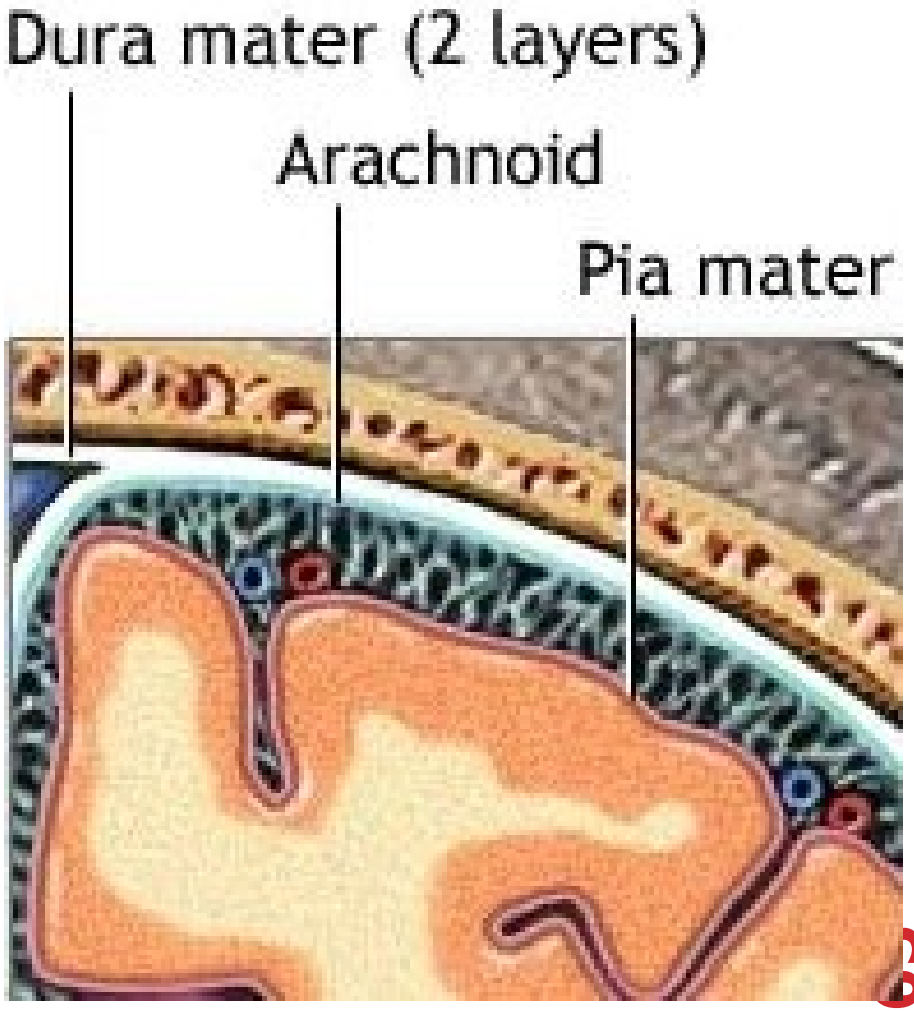
Encephalitis:- Is an Inflammation of brain.

Meningoencephalitis

- **Inflammation of brain with meningeal involvement.**

MENINGES


Dura
Arachnoid
Vein
Subarachnoid space
Pia
Gray matter
Virchow-Robin Space
Penetrating vessel


Dura mater (2 layers)
Arachnoid
Pia mater

INFECTIOUS AGENT

- **Meningitis can be caused by many different organisms including viruses and bacteria.**

- **Meningitis, caused by a bacteria, is life threatening and requires urgent medical attention and treatment with antibiotics. Meningitis caused by a virus is very rarely life threatening but can cause the body to become very weak.**

In neonates **Gram negative bacilli, E.coli, Proteus spp.**

Group B streptococci

Preschool child **Haemophilus influenza B**

Neisseria meningitides

Streptococcus pneumonae

CONT....

Older children → **N. meningitidis**

Adults → **Strep. Pneumonae**

→ **N. meningitidis (A, B, C, X, Y, Z, w135)**

- It is responsible to Meningococcal meningitis
  - Type A is known to cause epidemics

Aseptic Meningitis

- **A syndrome characterized by acute onset of meningeal symptoms, fever, and cerebrospinal fluid pleocytosis, with bacteriologically sterile cultures.**
- **Aseptic meningitis is a syndrome of multiple etiologies, but most cases are caused by a viral agent.**

EPIDEMIOLOGY

- **Greatest incidence occurs during winter and spring**
- **Epidemics occur irregularly**
- **Common in children and young adults**
- **It is a disease of individuals with poor general health and commonly happen in crowded situations.**

Reservoir – Humans

MOT–

- Direct contact with respiratory droplets from nose and throat of infected person.

I/P-

- Varies from 2-10 days, commonly 3-4 days.

POC-

- Until the meningococci are no longer present in discharges from nose and mouth.

Meningococci usually disappear from the nasopharynx with in 24 hrs after institution of Rx with antimicrobial agent to which the organisms are sensitive.

SUSCEPTIBILITY AND RESISTANCE

- **Susceptibility is low and decreases with age**
- **Immunocompromised persons are at high risk**

Pathogenesis:

- **The meninges are congested and infiltrated with inflammatory cells.**
- **A thin layer of pus forms and this may later organize to form adhesions.**
- **This may cause obstructions to the free flow of CSF and impedes absorption of CSF leading to hydrocephalus or they may damage the cranial nerves at the base of the brain.**
- **The CSF pressure raises rapidly, the protein content increases and the glucose content decreases.**

CLINICAL MANIFESTATIONS

Babies and Young Children

- **High temperature, fever, possibly with cold hands and feet**
- **Vomiting or refusing feeds**
- **High pitched moaning, whimpering cry**
- **Blank, staring expression**
- **Pale, blotchy complexion**
- **Stiff neck and Arched back**
- **Baby may be floppy, may dislike being handled, be fretful/ irritable.**
- **Difficult to wake or lethargic**
- **The fontanel (soft spot on babies heads) may be tense or bulged.**

INFANTS



Fever, possibly with cold hands & feet



Refusing feeds or vomiting



High pitched moaning cry or whimpering



Dislike of being handled or fretful



Neck retraction with arching of back



Blank & staring expression



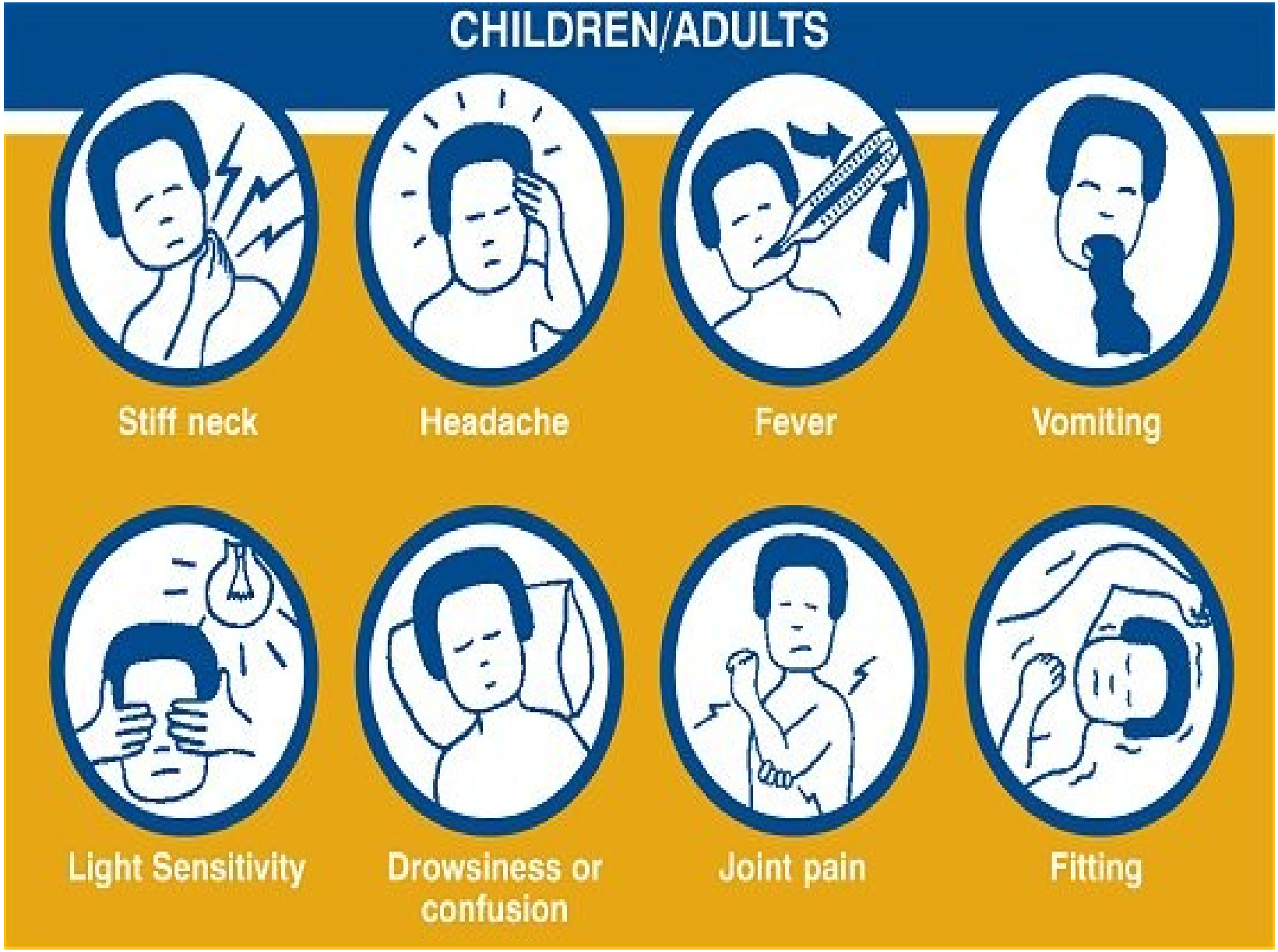
Child is difficult to wake, lethargic



Pale, blotchy complexion

OLDER CHILDREN AND ADULTS

- **High temperature, fever, possibly with cold hands and feet.**
- **Vomiting, sometimes diarrhoea.**
- **Severe headache.**
- **Joint or muscle pains, sometimes stomach cramps.**
- **Neck stiffness (unable to touch the chin to the chest)**
- **Dislike of bright lights.**
- **Drowsiness.**
- **The patient may be confused or disorientated. Fitting/ convulsions may also be seen.**
- **A rash may develop.**


CHILDREN/ADULTS
Stiff neck
Headache
Fever
Vomiting
Light Sensitivity
Drowsiness or confusion
Joint pain
Fitting

One of the physically demonstrable symptoms of meningitis is Kerning's sign.

- **Severe stiffness of the hamstrings causes an inability to straighten the leg when the hip is flexed to 90 degrees.**



- **Another physically demonstrable symptoms of meningitis is *Brudzinski's sign*.**

- **Severe neck stiffness causes a patient's hips and knees to flex when the neck is flexed.**



- **One sign of meningococcal septicemia is a rash that does not fade under pressure (see ‘Glass test’).**

- This rash is caused by blood leaking under the skin. It starts anywhere on the body. It can spread quickly to look like fresh bruises.

-This rash is more difficult to see on darker skin. Look on the paler areas of the skin and under the eyelids.

‘GLASS TEST’

- **A rash that does not fade under pressure will still be visible when the side of a clear drinking glass is pressed firmly against the skin.**
- **If someone is ill or obviously getting worse, do not wait for a rash. It may appear late or not at all.**
- **A fever with a rash that does not fade under pressure is a medical emergency.**



The glass test
Press the side of a glass firmly against the rash so you can see if it fades under pressure. If it doesn’t fade, get medical help immediately. If you are feeling very ill, get help anyway, even if the rash fades or doesn’t appear at all.

RASHES





Diagnostic Test

Bacterial Meningits

- CBC: Increase polymorphonuclear leukocytes
- CSF Analysis
  - Increase CSF pressure
  - Increase Protein
  - Increase Leokocytes
  - Decrease Glucose

Viral Meningits

- CBC: Decrease polymorphonuclear leukocytes
- CSF Analysis
  - Normal or slightly increase CSF pressure
  - Normal or slightly Protein
  - Few Leukocytes
  - Normal Glucose

Medical Treatment

Mannitol

- is given to decrease cerebral edema

Digitalis

- is given to control arrythmias

Antibiotics:

- Ceftriaxone is given to combat causative agent

Anti Convulsant

- is given to control seizure

Prevention

1. Vaccination of HiB for children
2. Vaccination of Neisseria Menigitides Serogroups for older clients.
3. Chemoprophylaxis: Rifampicin


DON'T IGNORE
The signs
Fever? Headache? Vomiting?
Stiff neck?
Rash?
Cold hands & feet?
Sleepy? Confused?
Dislike bright lights?
Pale, blotchy skin?
Limb pain?
Meningitis
Meningitis can kill in hours
It could happen to anyone
It could happen to you!
NHS
© Crown copyright 2007 283253 1p 5k Sep07 (FOR)
These are just some of the warning signs. If you are worried for yourself or someone else get medical help immediately. Contact
For more information visit www.meningitis.org or www.meningitis-trust.org

TUBERCULOSIS

- **Tuberculosis (TB) is an infectious diseases caused by *Mycobacterium tuberculosis, a rod shaped* bacillus called acid-fast due to its staining characteristics in laboratory.**

- **A systemic bacterial disease which primarily affects the lung, but other organs may also be involved depending on the bacteria dissemination and the host resistance.**

CONT....

Infectious agent

- **M. tuberculosis- human tubercle bacilli (commonest cause)**
- **M. bovis-causes cattle and man infection**
- **M. avium-causes infection in birds and man**
- ***Mycobacterium africanum***

EPI

- **It occurs worldwide, however under developed areas or countries are more affected.**

- **Affects all ages and both sexes, but age groups b/n 15-45 years are mainly affected.**

CONT....

Mode of transmission

- **Through aerosolized droplets mainly from persons with active ulcerative lesion of lung expelled during talking, sneezing, singing or coughing directly.**

- **Untreated PTB^{+} cases are the source of infection. They may spread from the initial location in the lungs to other parts of the body via the blood stream, the lymphatic system, via the airways or by direct extension to other organs.**

MOT CONT'D

- **The risk of infection is related to the length of contact an individual shares volume of air with an infectious case.**
  - **i.e. intimate, prolonged or frequent contact is required.**

- **Ingestion of unpasteurized milk transmits bovine TB.**

- **Overcrowding and poor housing conditions favour the disease transmission.**

Incubation period:- **Depends on the site of involvement**

- **4-12 weeks – PTB**
- **3-6 month – Meningeal, miliary and pleural disease**
- **Up to 3 yrs - GI, bone, joint & lymph node disease**
- **8 yrs – Renal tract disease**

PERIOD OF COMMUNICABILITY:

- **As far as the bacilli is present in the sputum.**
- **Some untreated or inadequately treated patients may be sputum positive intermittently for year.**
- **Effective antimicrobial therapy usually eliminates communicability with in 2 weeks.**
- **Extra pulmonary TB and children with primary TB are generally non-infectious.**

SUSCEPTIBILITY AND RESISTANCE

- **Everyone who is non infected or non-vaccinated can be infected.**

Susceptibility to infection is highest among:

- Children under 3 yrs old
- Adolescents
- Young adults
- The very old
- The immuno compromised
- Malnourished individuals

CONT….

- **HIV is an important risk factor for the development of HIV-associated TB by facilitating:**

  - **Reactivation:** reactivation of latent TB infection that was acquired prior to the HIV infection.

  - **Progression of recent infection** - rapid progression of latent TB infection to TB disease following recent TB infection.

  - **Re-infection** - are infection with another strain of M. tuberculosis.

CONT….

Tuberculosis is broadly classified into:

1. **Pulmonary TB (PTB): Accounts for 85 % of all TB cases, and it is further classified in:**

- **A. Smear-positive PTB: Comprises 75-80% of PTB cases.**
- **B. Smear-negative PTB: Comprises 20-25% of PTB cases.**

2. Extra-pulmonary TB (EPTB): It is the result of the spread of tuberculosis to other organs, most commonly pleura, lymph nodes, spine, joints, genito-urinary tract, nervous system or abdomen and

- **It represents 14% of all TB cases in the world and 12% of all TB in HBC.**

NATURAL HISTORY

- **In the great majority (90-95%) of persons infected with *M. Tuberculosis develop immunological* defense either kills the inhaled or ingested bacilli or perhaps more often, keeps them suppressed (silent focus) causing latent *M. tuberculosis infection.***

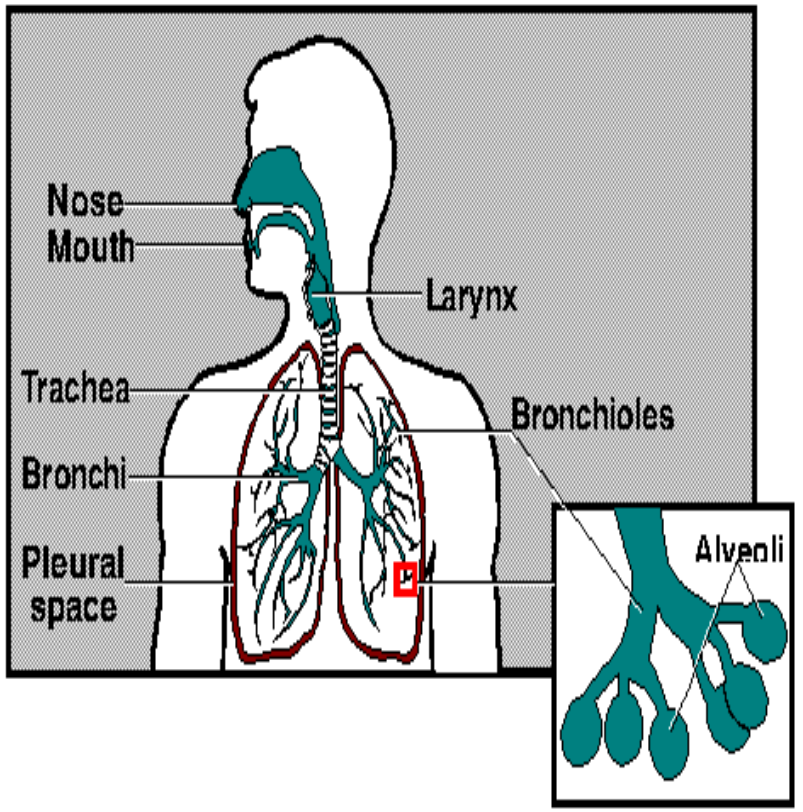
- **Only about 5-10% of such infected persons (primary infection) develop active disease.**

- **Active TB disease arises from progression of the primary lesion as a continuous process within a year or so after infection, or from endogenous reactivation of latent foci, which remained dormant since the initial infection or exogenous re-infection.**

DISEASE PROGRESSION- STAGE 1

Stage 1

- Droplet nuclei are inhaled, and are generated by talking, coughing and sneezing.
- Once nuclei are inhaled, the bacteria are non-specifically taken up by alveolar macrophages.
- The macrophages will not be activated, therefore unable to destroy the intracellular organism.
- The large droplet nuclei reaches upper respiratory tract, and the small droplet nuclei reaches air sacs of the lung (alveoli) where infection begins.
- Disease onset when droplet nuclei reaches the alveoli.



DISEASE PROGRESSION- STAGE 2

- Begins after 7-21 days after initial infection.
- TB multiplies within the inactivated macrophages until macrophages burst.
- Other macrophages diffuse from peripheral blood, phagocytose TB and are inactivated, rendering them unable to destroy TB.

DISEASE PROGRESSION- STAGE 3

- *Lymphocytes, specifically T-cells recognize TB antigen*. This results in T-cell activation and the release of Cytokines, including interferon (IFN).

- The release of IFN causes the activation of macrophages, which can release lytic enzymes and reactive intermediates that facilitates immune pathology.

- Tubercle forms, which contains a semi-solid or “cheesy” consistency. TB cannot multiply within tubercles due to low PH and anoxic environment, but TB can persist within these tubercles for extended periods.

DISEASE PROGRESSION- STAGE 4

- Although many activated macrophages surround the tubercles, many other macrophages are inactivated or poorly activated.

- TB uses these macrophages to replicate causing the tubercle to grow.

- The growing tubercle may invade a bronchus, causing an infection which may spread to other parts of the lungs. Tubercle may also invade artery or other blood supply.

- Spreading of TB may cause milliary tuberculosis, which can cause secondary lesions.

- Secondary lesions occur in bones, joints, lymph nodes, genitourinary system and peritoneum.

STAGE 5

The caseous centers of the tubercles liquefy.

This liquid is very crucial for the growth of TB, and therefore it multiplies rapidly (extracellularly).

This later becomes a large antigen load, causing the walls of nearby bronchi to become necrotic and rupture.

This results in cavity formation and allows TB to spread rapidly into other airways and to other parts of the lung.

CLINICAL MANIFESTATION

Early signs and symptoms

- Productive cough for more than 2 weeks
- Fever
- Unexplained wt loss
- Night sweats

CONT…..

Late signs and symptoms

- Blood stained sputum
- Difficulty in breathing
- Severe wt loss
- Severely diminished loss of appetite
- Weakness
- Symptoms of other organ involvement
  - Enlargement of lymph nodes

DIAGNOSIS

- **By rank of importance the diagnostic methods to confirm/exclude TB are:**
  - **Microscopic examination of sputum smears**
  - **Radiological investigation**
  - **AFB culture**
  - **Histo-pathology**

- ***Remember:* Chest X-ray is supportive to microscopy.**
- ***It is a major error to omit sputum examination and diagnose TB based on X-ray findings alone!***

RADIOLOGY

- Chest radiography is the most important method to detect TB

- TB's characteristics of a chest radiography favor the diagnosis of tuberculosis are:

A. Shadows mainly in the upper zone

B. Patchy or nodular shadows

C. The presence of cavities of course, can also occur in lung abscess, carcinoma, etc

C. The presence of calcification. although a carcinoma or pneumonia may occur in an areas of the lung where there is calcification due to tuberculosis

D. Bilateral shadows, especially if these are in the upper zones

E. The persistence of the abnormal shadow without alteration in an x-ray repeated after several weeks this helps to exclude a diagnosis of pneumonia or other acute infection.

Sputum examination:- There are direct smear and culture.

- Direct smear examination is only positive when large numbers of bacilli begin to be excreted.
- A negative smear by no means excludes tuberculosis.
- A negative smear in the presence of extensive disease and cavitation makes the diagnosis less likely.
- Particularly if the negatives are frequently repeated.
- However one positive result doesn't justify starting anti TB Rx since errors can never be excluded.

EXTRA PULMONARY TUBERCULOSIS (EPTB)

TB lymphadenitis

- Slowly developing and painless enlargement of lymph nodes followed by matting and drainage of pus.

Tuberculosis pleurisy

- Pain while breathing in, dull lower chest pain, slight cough, breathlessness on exertion.

TB of bones and joints

- Localized pain and/or swelling, discharging of pus, muscle weakness, paralysis and stiffness of joint.

DIAGNOSIS OF LYMPHADENITIS

- ***History and P/E***

✓ **Fine needle aspiration (FNA) or surgical biopsy**

- **Differential diagnosis**

✓**Lymphoma,**

✓**Metastatic Cancer.**

Intestinal TB

- ✓ **Loss of wt and appetite**
- ✓ **Abdominal pain, diarrhoea and constipation**
- ✓ **Mass in the abdomen**
- ✓ **Fluid in the abdominal cavity (ascites)**

Tuberculosis meningitis

- **Head ache, fever, vomiting, neck stiffness and mental confusion of insidious onset.**

DIAGNOSIS

- Acid fast stain for AFB can be done for extra Pulmonary TB having pusy discharge.

- Histo pathological examination: Biopsy for EPTB like TB lymphadenitis.

- Tuberculin skin test (TST or Mantoux test) – Helpful in children under 6 yrs of age.

Tuberculin testing

- A positive tuberculin test although it is of great use in children, but it has limited diagnostic significance in older age groups.

- A positive tuberculin skin test indicates tuberculosis infection, with or without disease



VALUES OF A NEGATIVE TUBERCULIN TEST (TST)

- **A tuberculin test is not significant or 'negative' when the diameter of skin indurations *is less than 10mm* (*or less than 5 mm in an HIV infected child).***

- This is regardless of whether or not the child has had BCG.

- **A negative tuberculin skin test doesn't exclude TB. In other words, a negative test is of no help in deciding that someone does not have TB.**

CONDITIONS THAT MAY SUPPRESS THE TST

- HIV infection
- Malnutrition
- Severe bacterial infections, including TB
- Viral infections like measles, chicken pox, glandular fever
- Cancer
- Immunosuppressive drugs like steroids
- Incorrect injection of PPD (purified protein derivative = mantoux test).

VALUES OF A POSITIVE TST

- The criterion for a significant or 'positive' tuberculin test depends on whether a child has previously had BCG vaccination or not.

- This is because a reaction to tuberculin is usual after a previous BCG, for several years. This reaction is usually a weaker reaction (diameter often less than 5mm) than the reaction to natural infection with M. tuberculosis.

CONT'D

- A tuberculin test is considered significant or 'positive' when the diameter of skin indurations is 10mm or more.

- If the child is HIV infected, tuberculin test is considered ''positive'' if the in duration is 5mm or more.

- A positive tuberculin test is only one piece of evidence in favour of the Dx of TB. The younger the child and the greater the diameter of indurations, the stronger is the evidence.

CASE DEFINITIONS

- A case of TB is a patient in whom tuberculosis has been confirmed bacteriologically or diagnosed by a clinician.

- ***New case (N):*** A patient who has never had Rx for TB or has been on anti TB Rx for less than four weeks.

- *Relapse (R):* A patient that has been declared cured or treatment completed of any form of TB in the past, but who reports back to the health service and is found to be AFB smear positive or culture positive.

CONT….

- ***Rx failure (F):*** A patient who, while on Rx remained smear positive or became again smear positive at the end of the five 'months' or later, after commencing Rx.
- A patient who was PTB-negative at the beginning and turned out smear positive at the end of the intensive phase.

Return after default (D): A patient who had previously been recorded as defaulted from Rx and returns to the health facility with smear positive sputum.

CONT….

Defaulter: A patient who has been on Rx for at least 4 weeks and whose treatment was interrupted for 8 or more consecutive weeks.

Transfer out (T): A patient who started treatment in one treatment unit and is transferred to another treatment unit to continue treatment.

Other (O): A patient who does not fit in any of the above mentioned categories (e.g., a PTB smear negative who returns after treatment interruption).

TREATMENT

- Streptomycin (S)
- Ethambutol (E)
- Isoniazid (H)
- Rifampicin (R)
- Pyrazinamide (Z)

❖ **<u>The drugs available in fixed dose combination (FDC) are:</u>**

✓ Rifampicin, Isoniazid, Pyrazinamide and Ethambutol (RHZE 150/75/400/275 mg);

✓ Rifampicin and Isoniazid (RH 150/75 mg);

✓ Ethambutol and Isoniazid (EH 400/150 mg).

✓ All the drugs should be taken together as a single, daily dose, preferably on an empty stomach.

PHASES OF CHEMOTHERAPY:

- ***Intensive phase (initial):*** Three or more drugs for the 1st 8 weeks for new cases and 12 weeks for re-treatment cases.

- It renders the pt non-infectious by rapidly reducing the load of bacilli in the sputum and minimizing the danger of dev't of drug resistance.

- ***Continuation phase:*** Follows the intensive phase and is important to ensure that the pt is permanently cured and does not relapse after completion of Rx. This phase requires at least two drugs to be taken for 4-6 months.

- During the both phases, the drugs must be collected daily by the patient and must be swallowed under the direct observation of a health **worker (DOTS).**

PATIENT CATEGORIES AND TREATMENT REGIMENS

- There are four different treatment categories corresponding Rx regimens.

Category I:

- Short course chemotherapy for smear +ve PTB & seriously ill smear negative PTB and EPTB cases.
-

➢New smear-positive PTB patients

➢New cases with severe extrapulmonary tuberculosis (disseminated, meningeal, pericardial, peritoneal, bilateral pleural, spinal, intestinal and genito-urinary)

Dosage of category I regimens= 2ERHZ/4RH

CATEGORY II (RE TREATMENT REGIMEN)

- Patients previously treated for more than one month and who are still smear positive.
  - Relapses
  - Treatment failures
  - Returns after default
  - PTB patients who become smear positive after 2 months of Rx.
  - Return after default from re-treatment (only once re-treatment again).
  - Relapses after re treatment (only once re treatment again).

= 2 S(ERHZ) / 1(ERHZ) / 5E(RH)3

CATEGORY III

- Short course chemotherapy for smear negative PTB, EPTB and TB in children who are not seriously ill.
- New adult patient with smear negative PTB
- New adult patients with EPTB (milder forms)
  - TB of the lymph nodes
  - TB Osteomylites (Bone TB)
  - TB arthritis
  - Adrenal TB
- TB with unilateral pleural effusion

❖ ***Dosage of category III regimens= 2ERHZ/4RH***

CATEGORY IV – CHRONIC CASES

- **Multi drug resistant cases of TB, defined as smear-positive cases of pulmonary tuberculosis who have previously received a supervised re-treatment regimen.**

- WHAT IS MDR-TB ?

- It is a mutated form of the TB microbe that is extremely resistant to at least the two most powerful anti-TB drugs - ***Isoniazid and Rifampicin.***

- People infected with TB that is resistant to first-line TB drugs will confer this resistant form of TB to people they infect.

- ***MDR-TB is treatable but requires treatment for up to 2 years.***

- ***Extensive-drug resistant TB (XDR):*** is defined as ***resistance to at least Rifampicin and Isoniazid***.
    - In addition to
- ***Fluoroquinolone,*** and to at least one of the following three injectable drugs used in anti-TB treatment:
    - Capreomycin
    - kanamycin and
    - Amikacin

PREVENTION AND CONTROL

- Health education esp. for pts how to dispose sputum and MOT.
- Chemotherapy of cases

- Chemoprophylaxis for contacts

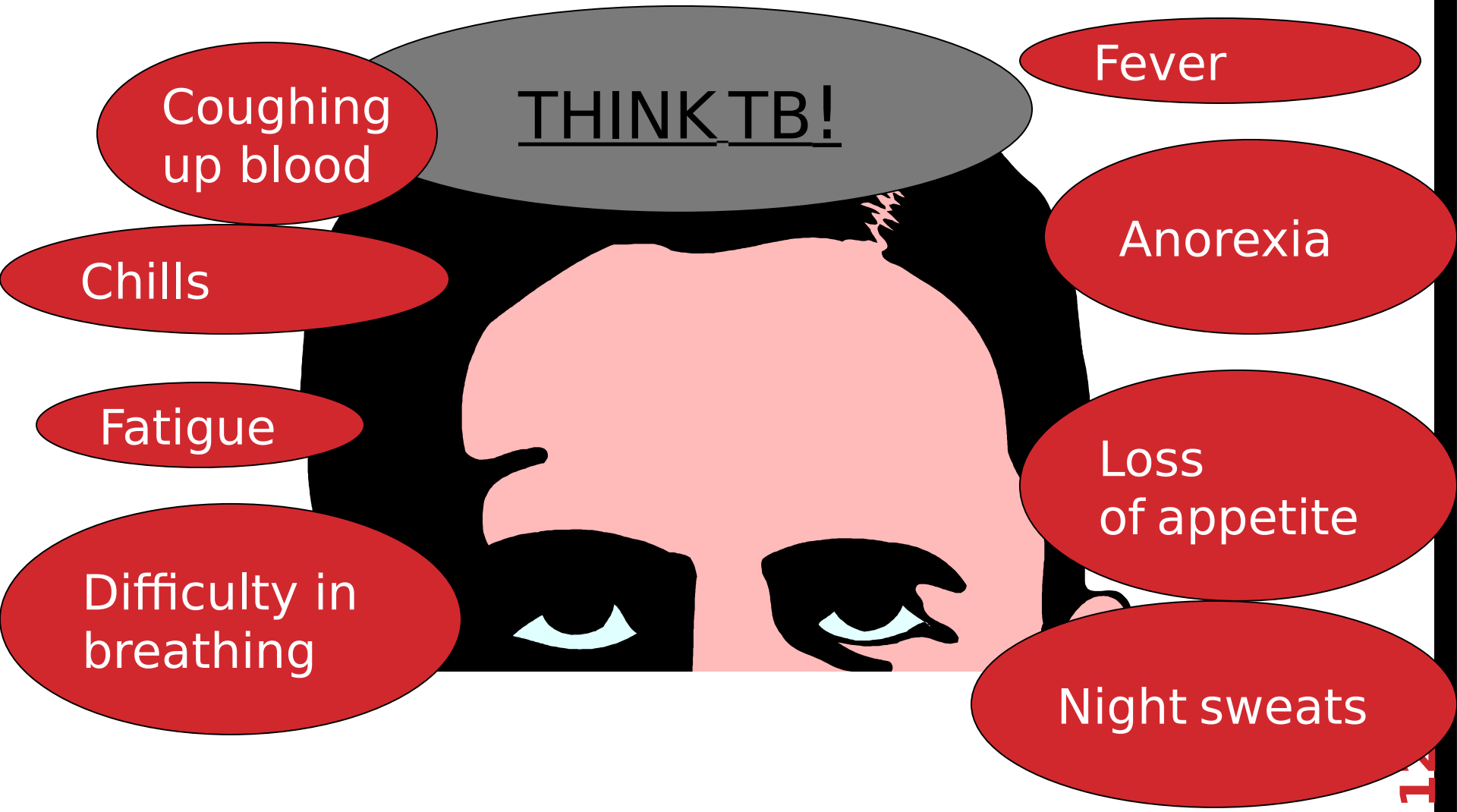
- INH for 06 months blindly (5 mg/kg for 6 months) or INH for 03 months then do PPD test

- PPD test +ve continue for 03 months
- PPD –ve stop INH and **give BCG for the future**

PREVENTION

- Improved standard of living
- Adequate nutrition
- Healthy housing, ventilation and sunlight exposure (windows open and transparent)
- Environmental sanitation
- Personal hygiene & Active case finding and Rx.
- Isolation of PTB +ve case = decreases infectivity
- Immunization of infants with BCG
- Educate the public about the modes of disease transmission and methods of control.

EVEN IF A SKIN TEST IS NEGATIVE…..



LEPROSY(HANSEN'S DISEASE)



LEPROSY

- **Every year, between 200,000 and 400,000 new cases of Leprosy are detected. In 2005, 296,000 new cases of leprosy were detected.**
  - 70% of those were found in India

History of Leprosy:

- **The bacteria that causes Leprosy, (mycrobacterium Leprae) was discovered by Gerhard Hansen in 1873.**

- **Leprosy was known since ancient times, but no one knew what it was or what caused it.**

INTERESTING FACTS

- Since Leprosy attacks the eyes, people with Leprosy eventually lose the blink reflex.

- Because of this, their eyes are scratched by dirt and dust, and they eventually go blind.

- To solve this problem, doctors trained patients to blink on schedule using a timer

.

- In some cases**,** however, the muscle that makes the eye blink is disabled as well

- Doctors solved this problem by connecting a string from the jaw to the eyelid. That way, when you open your mouth, your eyes close.

MORE INTERESTING FACTS:

- In the 1940's having leprosy was considered a criminal offense
  - People with leprosy could be hunted down and shipped off to remote islands where they could spend the rest of their lives in solitude.

- At one point, Patients in Kalawao,Hawaii who were about to be shipped off could request a re-diagnosis
  - It turned out that ten out of eleven people who requested a re-diagnosis did not have leprosy after all

LEPROSY

- It is a chronic infectious disease caused by M. leprae, an acid fast, rod shaped bacillus.

- It mainly affects the skin, peripheral nerves, and mucosa of the respiratory tract etc.

- It has left behind a terrifying image in history and human memory of mutilation, rejection and exclusion from society.

Agent:

- ***M. leprae*, acid fast, rod shaped bacillus**

EPIDEMIOLOGY

- Leprosy affects all ages and both sexes. The age group mainly affected is between 15-45 yrs.

- There are however special groups that are more vulnerable to develop the disease.
- Factors related to poverty increase the risk of developing disease.

- Although common in rural tropics and subtropics, socio-economic condition may be more important than climate itself.

- It is endemic in south and southeast Asia, tropical Africa and Latin America.

CONT'D

- Under normal circumstance only a very small proportion (less than 5%) of all individuals who are infected by the leprosy bacilli will develop the disease during their life.
- In the majority of people, the immunological defence kills all the bacilli.
- The disease has a long incubation period, on average ranging from 3 to 5 years, but it may vary from 6 months to more than 20 yrs.
- Leprosy can cause severe disability mainly as a result of peripheral nerve damage.

Reservoir:

- **Untreated multi-bacillary leprosy patients discharging bacilli are the main reservoir of the disease.**
- ***"In the 19th century leprosy was believed to be a hereditary ailment".***

MOT: - Not clearly established.

- **The transmission of leprosy is thought to occur through the respiratory tract.**
- **Infected individuals discharge bacilli through their nose and a healthy individual breaths them in.**
- **But it is important to note that the extract mechanism is not known.**

- Cutaneous ulcers in lepromatous patients may shed large number of bacilli. Organisms probably gain access (entrance) through the URTI and possibly through broken skin.

- In children less than one year of age, transmission is presumed to be transplacental.
- I/P:- 8 month to 20yrs

POC: Infectiousness is lost in most instances within short period of continuous and regular Rx with dapsone or clofazmin and within 3 days of Rifampicin Rx.

Susceptibility and resistance: the presence and form of leprosy depend on the ability to develop effective cell mediated immunity.

CLINICAL FEATURES

- **Clinical Manifestations vary b/n two polar forms:**
  - **Lepromatous and**
  - **Tuberculoid leprosy.**

1. Lepromatous (multibacillary form) :-
characterized by

- **Nodules, papules, macules and diffused infiltrations are bilaterally symmetrical and usually numerous and extensive (six or more skin lesions).**

- **Involvement of the nasal mucosa may lead to crusting, obstructed breathing and epistaxis. Ocular involvement leads to iritis and keratitis.**



2. Tuberculoid (paucibacillary form)

- **Skin lesions are single or few to five leprosy skin lesions sharply demarcated, anaesthetic or hyperaesthetic and bilaterally symmetrical, peripheral nerve involvement tends to be severe.**



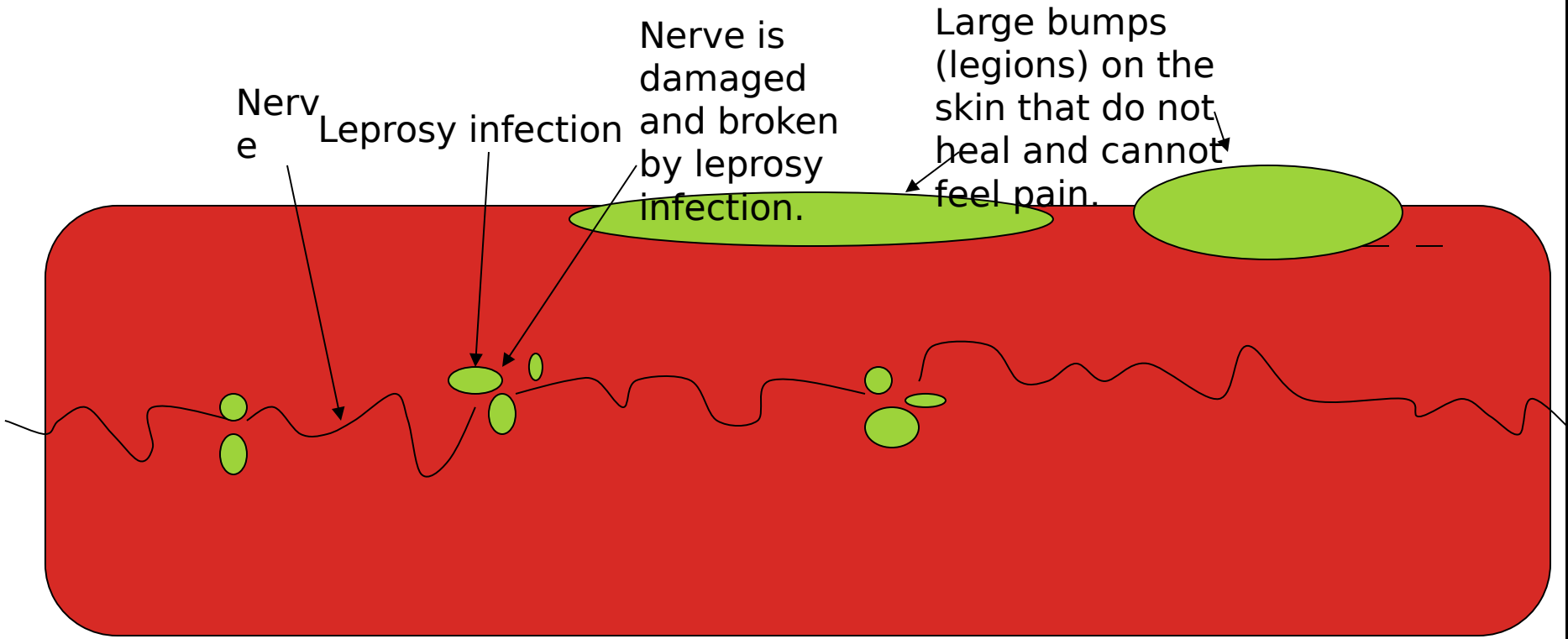
Borderline

- **Has features of both polar form and is more liable to shift towards the lepromatous form in untreated patients and toward the tuberculoid form in treated patients.**





HOW THE HUMAN BODY IS AFFECTED BY LEPROSY



How to examine for leprosy?

- Examine in a well-lit room
- Examine the whole body
- Ask since when the patch was noticed
- Ask what treatments have been tried
- Test for sensation
- Look for any visible deformities

How to diagnose leprosy

- Examine skin
- Check for patches
- Test for sensation
- Count the number of patches
- Look for damage to nerves

DIAGNOSIS

Cardinal signs

- Hypo-pigmented or reddish skin lesion(s) with definite loss of sensation.
- Enlarged peripheral nerve\s with impairment of sensations in the area supplied.
- Acid-fast bacilli in the slit skin smear
- N.B. The finding of one of these three cardinal signs is diagnostic for leprosy.

CONT….

- **A patient is suspect for leprosy when presenting with:**
    - Reddish patches on the skin
    - Loss of sensation on the skin
    - Numbness and tingling of the hands and/or the feet
    - Weakness of eyelids, hands and feet
    - Painful and/or tender nerves
    - Burning sensation in the skin
    - Painless swelling in the face and ear lobes
    - Painless wounds or burns on the hands or feet.

CONT..

- **The main aim of case-finding is to:**
- **Diagnose and treat leprosy cases early, before irreversible damage has occurred.**
- **Interrupt transmission of the disease.**
- **Prevent the occurrence of leprosy related disability.**

CLASSIFICATION

- **Patients should be classified according to:**

❖ **The number of leprosy skin lesions.**

❖ **The result of the skin smear examination**

1. Multibacillary (MB) leprosy:

- Six or more skin lesions.
- Less than six skin lesions, which have a positive slit skin smear result.

2. Paucibacillary (PB) leprosy

- **One to five leprosy skin lesions.**

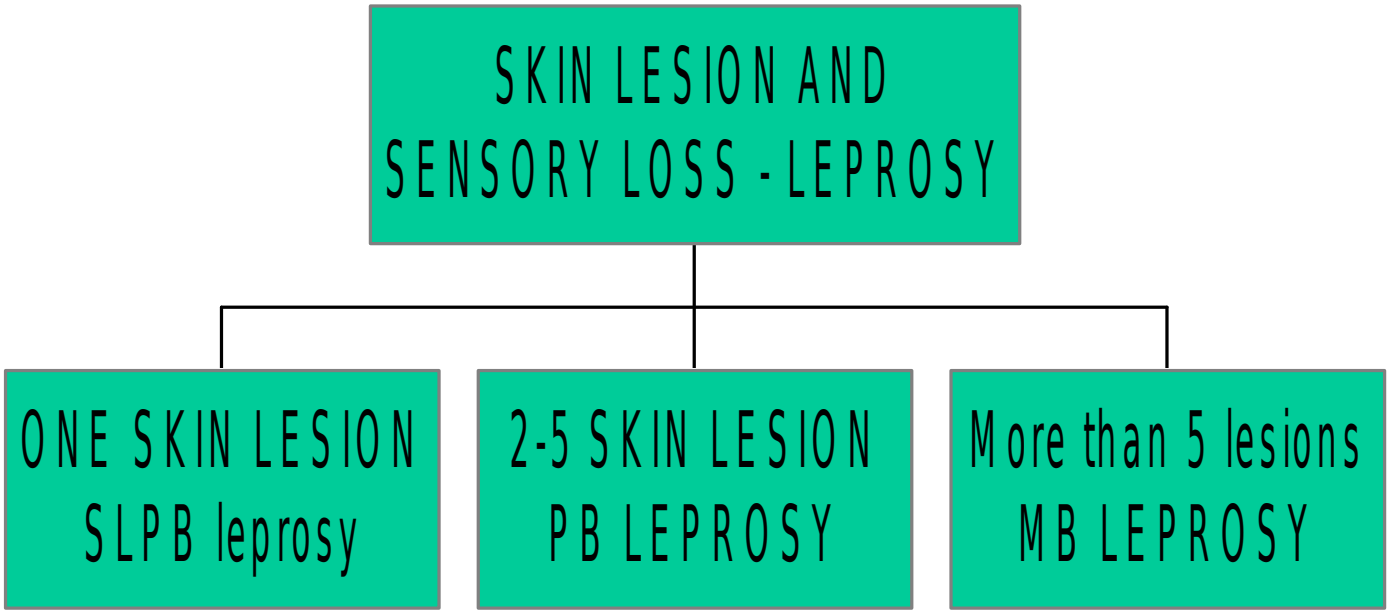
3. Pure neural leprosy

- These are patients, who do not have any skin lesion, but who have clearly thickened nerves with or without signs of nerve damage.

- Patients with pure neural leprosy should be reported and treated as a PB case if only one nerve is affected and the nerve biopsy smear result is negative.

- If two or more nerves are affected, or the nerve biopsy smear is positive, the patient should be reported and treated as an MB case.

FLOW CHART FOR DIAGNOSIS AND CLASSIFICATION



THE CURE

- **The good news is that today leprosy can be cured.**
- **A course of pills, called Multi-Drug Therapy (MDT), cures most patients in 6 months, and the more infectious patients within 1 year.**
- **If treatment is started early deformities and disabilities can be prevented and social stigmatization avoided.**
- **The effects can be incredibly dramatic as the photograph below shows**



TREATMENT

- ***Multi Drug Therapy /MDT regimen***
- Leprosy is treated with Multi Drug Therapy (MDT).

- MDT is the use of a combination of two or three anti-leprosy drugs to treat leprosy.

- Transmission of leprosy is interrupted after the very first dose of MDT. In other words, patients are no longer infectious to others after being administered the first dose of the treatment regimen.

- There are virtually no relapses, i.e. no recurrences of the disease after treatment is completed.

- No resistance of the bacillus to MDT has been detected.

TREATMENT

- PB-MDT regimen- This regimen consists of Rifampicin and Dapsone for a total duration of 6 months. It is to be prescribed to all cases classified as Paucibacillary (PB) leprosy.

Drugs	Child < 10 years	10 - 14 years	≥ 15 years
Rifampicin	300 mg monthly	450 mg monthly	600 mg monthly
Dapsone	25 mg daily	50 mg daily	100 mg daily

CONT…..

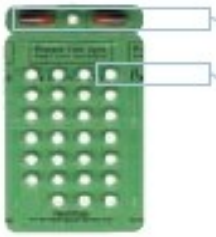
- **MB-MDT regimen -** This regimen consists of Rifampicin, Dapsone and Clofazimine, for 12-month duration. It is to be prescribed to all cases classified as Multibacillary (MB) leprosy.

Drugs	Child < 10 years	10 -14 years	≥ 15 years
Rifampicin	300 mg monthly	450 mg monthly	600 mg monthly
Clofazimine	100 mg monthly	150 mg monthly	300 mg monthly
Clofazimine	50 mg twice a week	50 mg every other day	50 mg daily
Dapsone	25 mg daily	50 mg daily	100 mg daily

MDT Regimens

It is crucial that patients understand which drugs they have to take once a month and which every day.

Each blister pack contains treatment for 4 weeks.



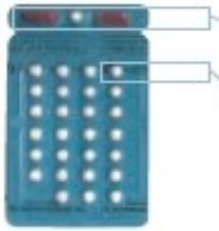
PB adult blister pack

PB adult treatment:

Once a month: Day 1
- 2 capsules of rifampicin (300 mg X 2)
- 1 tablet of dapsone (100 mg)

Once a day: Days 2–28
- 1 tablet of dapsone (100 mg)

Full course: 6 blister packs



PB child blister pack

PB child treatment (10–14 years):

Once a month: Day 1
- 2 capsules of rifampicin (300 mg+150 mg)
- 1 tablet of dapsone (50 mg)

Once a day: Days 2–28
- 1 tablet of dapsone (50 mg)

Full course: 6 blister packs

For children younger than 10, the dose must be adjusted according to body weight.



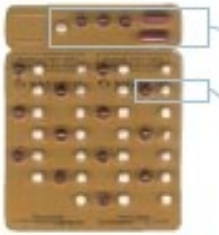
MB adult blister pack

MB adult treatment:

Once a month: Day 1
- 2 capsules of rifampicin (300 mg X 2)
- 3 capsules of clofazimine (100mg X 3)
- 1 tablet of dapsone (100 mg)

Once a day: Days 2–28
- 1 capsule of clofazimine (50 mg)
- 1 tablet of dapsone (100 mg)

Full course: 12 blister packs



MB child blister pack

MB child treatment (10–14 years):

Once a month: Day 1
- 2 capsules of rifampicin (300 mg+150 mg)
- 3 capsules of clofazimine (50 mg X 3)
- 1 tablet of dapsone (50 mg)

Once a day: Days 2–28
- 1 capsule of clofazimine every other day (50 mg)
- 1 tablet of dapsone (50 mg)

Full course: 12 blister packs

For children younger than 10, the dose must be adjusted according to body weight.

Multibacillary (MB or lepromatous) is a 24-month treatment of rifampicin, clofazimine, and dapsone. Paucibacillary (PB or tuberculoid) is a six-month treatment of rifampicin and dapsone.

PREVENTION AND CONTROL

- Health education
- Case detection and early Rx (particularly infectious multibacillary)
- Avoid contact with patients (isolation)
- Disinfection of articles in contact with nasal discharges of infectious patients.

Thank you