Infection: Lodging & multiplication of the organisms in or on the tissues of host.
Primary infection: Initial infection of a host by a parasite.
Reinfection: Subsequent infections by the same parasite in the same host.
Secondary infection: Infection by another organism in a person suffering from an infectious disease.
Nosocomial infection: Cross infections occurring in hospitals.
Superinfections: Infections caused by a commensal bacterium in patients who receive intensive chemotherapy.
Opportunistic infections: Organisms that ordinarily do not cause disease in healthy persons may affect individuals with diminished resistance.
Latent infections: When a pathogen remains in a tissue without producing any disease, but leads to disease when the host resistance is lowered.

Commonest infective disease: common cold.

PYREXIA OF UNKNOWN ORIGION (PUO)
When the temperature is raised above 38.3°C for more than 2 weeks without the cause being detected by physical examination or laboratory tests → PUO (FUO)

Etiology
a) Occult tuberculosis
b) Chronic suppurative lesions of the liver, pelvic organs, urinary tract, peritoneum, gall bladder, brain, lungs, bones & joints & dental sepsis (occasionally).
c) Viral infections:
   - Viral hepatitis
   - Infectious mononucleosis
   - Cytomegalovirus infection
   - Aids
d) Connective tissue disorders:
   - Giant cell arteritis.
   - RA
   - Rheumatic fever
   - SLE
   - PAN (polyarteritis nodosa)
e) Chronic infections:
   - Syphilis
   - Hepatic amoebiasis
   - Cirrhosis liver
   - Malaria
   - Filariasis
   - Leprosy
   - Brucellosis
   - Sarcoidosis
f) Haematological malignancies
   - Leukemia
   - Lymphoma
   - Multiple myeloma
g) Other malignant lesions: Tumours of lungs, kidney etc.
h) Allergic conditions
i) Miscellaneous conditions: Hemolytic anaemia, dehydration in infants etc.


SEPTIC SHOCK

- Endotoxines of gram negative bacilli are responsible for most of the cases.
- More frequent in men.
- Toxic shock syndrome in women: caused by toxigenic strains of staphylococci (gram positive) contaminating vaginal tampons.

Clinical features:
- Features of shock: Hypotension, Weak thready pulse, Cold clammy skin, Tachycardia & Peripheral cyanosis.
- Death is caused by: pulmonary oedema, tissue anoxia, cardiac arrythmias & Disseminated intravascular coagulation

SYSTEMIC DISEASES CAUSED BY COCCI

STREPTOCOCCAL INFECTIONS

Majority of pathogenic strains are: beta haemolytic which possess streptolysin O & S

<table>
<thead>
<tr>
<th>Disease</th>
<th>I. P:</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Streptococcal pharyngitis</td>
<td>2-4 days</td>
<td>I. P: 2-4 days Primary lesion is in throat. Erythematos rash on 2nd day over the neck &amp; trunk sparing palms &amp; soles. Diagnosis: isolation of group A streptococci from exudates.</td>
</tr>
<tr>
<td>Scarlet fever</td>
<td>1-3 days</td>
<td>Features of shock: Hypotension, Weak thready pulse, Cold clammy skin, Tachycardia &amp; Peripheral cyanosis.</td>
</tr>
<tr>
<td>Erysipelas</td>
<td></td>
<td>Acute spreading infection of skin &amp; subcutaneous tissue. Face is commonly affected.</td>
</tr>
<tr>
<td>Pusyderma/streptococcal impetigo</td>
<td></td>
<td>Deeply ulcerated impetigo: ecthyma</td>
</tr>
<tr>
<td>Cellulitis</td>
<td></td>
<td>Spreading inflammation of subcutaneous tissue due to entry of organism through the abrasions of the skin.</td>
</tr>
<tr>
<td>Lymphangitis</td>
<td></td>
<td>Linear red streaks from the site of entry to draining lymph nodes.</td>
</tr>
<tr>
<td>Streptococcal bacteremia</td>
<td></td>
<td>Toxic shock syndrome may be produced by streptococci.</td>
</tr>
<tr>
<td>Necrotizing fasciitis/streptococcal gangrene</td>
<td></td>
<td>Necrosis of fascia &amp; adipose tissue, often sparing the skin.</td>
</tr>
<tr>
<td>Streptococcal myositis</td>
<td></td>
<td>Infection reaches the muscles through the bloodstream.</td>
</tr>
<tr>
<td>Pneumonia &amp; empyema</td>
<td></td>
<td>Streptococcal pneumonia→ bronchopneumonia</td>
</tr>
</tbody>
</table>

ACUTE RHEUMATIC FEVER

Cause:
Hypersensitivity reaction to group A streptococci.
Rheumatic fever follows 2-3 weeks after an attack of streptococcal pharyngitis.

Age group: 5-15 years (mean age- 6 years)
Pathology: 2 stages:
- Exudative stage: acute phase.
- Proliferative stage: prolonged process. Hallmark of proliferative phase is Aschoff bodies.

Heart: Endocarditis, myocarditis & pericarditis (pancarditis). MacCallum’s patch is seen in posterior wall of left atrium due to scarring of mural endocarditis. Pericardium: bread & butter appearance (fibrous inflammation in pericardium).

Joints: Acute synovitis.

Clinical features

JONES CRITERIA (revised)

<table>
<thead>
<tr>
<th>Major manifestations</th>
<th>Minor manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Carditis</td>
<td>i) Previous rheumatic fever or H/o rheumatic heart disease.</td>
</tr>
<tr>
<td>2. Polyarthritis</td>
<td></td>
</tr>
</tbody>
</table>
* [Jaccoud’s arthritis]: affect the hand in 50% cases; non-erosive joint deformities with preservation of hand function.

3. Chorea (Syn: Sydenham’s chorea, St. Vitus dance, minor chorea) → non-repetitive, rapid & jerky involuntary movements of distal joints; mostly upper limbs.

ii) Fever

iii) Acute phase reactants → lab tests helpful in acute phase.

ESR ↑, C-Reactive protein ↑

4. Erythema marginatum → non-pruritic.

5. Subcutaneous nodules

6. Evidence of preceding streptococcal infection (ASO titer)

iv) Prolongation of PR interval in ECG.

---

**Diagnosis**

- Throat swab culture.
- Serodiagnosis
  - Anti-streptolysin O titer (ASO)
  - Anti-streptokinase (ASK)
  - AntiDNAse B
  - Anti-nicotinamide-adenine dinucleotidase (anti-NADase)
  - Anti-hyaluronidase (AH)
  - Anti-streptozyme test (ASTZ)
- Acute phase reactants → lab tests helpful in acute phase.
  - ESR & C-reactive protein - increased.
- ECG:
  - Sinus tachycardia, ectopic beats & 1st & 2nd degree heart blocks.
  - ST elevation → pericarditis.

**POSTSTREPTOCOCCAL GLOMERULONEPHRITIS (GN)**

- This may follow either cutaneous or pharyngeal lesion by group A streptococcus.
- 10-15% of children getting recurrent skin infections may develop glomerulonephritis.
- Serotypes 12, 44, 2, 52, 55, 57 & 4 are more often nephritogenic.
- Latent period for the development of acute GN is 10 days after pharyngitis & 3 weeks after pyoderma.

---

**STAPHYLOCOCCAL INFECTIONS**

**SUPERFICIAL LESIONS**

<table>
<thead>
<tr>
<th>Furuncle</th>
<th>Acute necrotic infection of hair follicle.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbuncle</td>
<td>Large furuncle or an aggregate of interconnected furuncles. Sites: back of neck, hips &amp; thighs. Common in diabetics.</td>
</tr>
<tr>
<td>Impetigo</td>
<td>Bullous impetigo is purely staphylococcal (occurs in newborns &amp; infants)</td>
</tr>
<tr>
<td>Ecthyma</td>
<td>Sites: buttocks, thighs &amp; legs.</td>
</tr>
<tr>
<td>Sycosis barbae</td>
<td>Seen in males after puberty on the beard region.</td>
</tr>
<tr>
<td>Follicular impetigo of Bockhart</td>
<td>Infection of hair follicle in scalp (seen in childhood).</td>
</tr>
<tr>
<td>Staphylococcal pneumonia</td>
<td>Carries a higher mortality especially in old &amp; debilitated patients.</td>
</tr>
<tr>
<td>Osteomyelitis</td>
<td>Primary osteomyelitis</td>
</tr>
<tr>
<td>Staphylococcal bacteremia</td>
<td>Fulminent sepsis may lead to death within 24 hours.</td>
</tr>
<tr>
<td>Staphylococcal food poisoning</td>
<td>Symptoms start within 6-8 hrs. Gastroenteritis or dysenteric symptoms.</td>
</tr>
</tbody>
</table>
Tropical promyositis  Large abscess in the muscles of limbs or trunk in apparently healthy adults.

Toxic shock syndrome  Caused by a diffusible toxin produced by staphylococci (phage Type 1)

PNEUMOCOCCAL INFECTIONS

Pneumococcal Pneumonia  (Syn: Lobar pneumonia)

Most common pneumococcal lesion in adults is pneumonia.

Pathology:  Formation of inflammatory exudate in alveoli.

Stages:  Red hepatisation (alveoli are filled with red cells & fibrin), Grey hepatisation (neutrophil leucocytes predominate) & Resolution.

Clinical features
- Fever, chills & herpes simplex.
- Pleuritic pain & Cough with pinkish or rusty sputum
- Signs of consolidation over the affected lobe.

Laboratory findings
- Rusty sputum
- Pneumococci can be demonstrated by gram staining, Blood culture is positive in 20-25% cases in early stage of the disease. Leucocytosis
- Skiagram of the chest shows a homogenous opacity corresponding to the lobe involved.

Complications
- Local: atelectasis, lung abscess, delayed resolution
- Due to spread of inflammation to adjacent structures: pleural effusion, empyema, pericarditis & peritonitis
- Haematogenous spread: septicaemia, meningitis etc.

MENINGOCOCCAL INFECTIONS

Neisseria Meningitidis  (meningococcus, gram negative) is pathogenic exclusively to man.

Major lesions are CERTERO спинAL MENINGITIS & MENINGOCOCCAL SEPTICAEMIA

<table>
<thead>
<tr>
<th>Meningococcal meningitis  (cerebrospinal fever)</th>
<th>Age; 6 months- adolescence. I.P: 3-5 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical features</td>
<td>Fever &amp; other constitutional features like headache, neck rigidity etc.</td>
</tr>
<tr>
<td>Signs of meningeal irritation</td>
<td>Kernig’s sign: pain over the hamstrings when the knee is extended passively with the hip flexed to 90°, &amp; the extension is restricted.</td>
</tr>
<tr>
<td></td>
<td>Brudzinski’s leg sign: flexion of the opposite knee when kernig’s sign is elicited.</td>
</tr>
<tr>
<td></td>
<td>Brudzinski’s neck sign: flexion of both legs when the neck is passively flexed.</td>
</tr>
<tr>
<td></td>
<td>Rise in intra cranial tension can cause papilloedema, blindness, deafness, hemiplegia &amp; coma.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Meningococcal septicemia  (meningococcemia)</th>
<th>Fulminent or chronic. Schwartsman phenomenon: vascular damage occurs as an allergic phenomenon caused by the endotoxin of meningococci.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Waterhouse- Friderichsen syndrome: haemorrhage into adrenal gland result in acute adrenal failure producing toxaemia &amp; shock.</td>
</tr>
<tr>
<td></td>
<td>Diagnosis: clinical features, lumbar puncture. CSF is under pressure, turbid &amp; shows large number of polymorphs (1000-10000/cmm) containing intracellular gram negative diplococci.</td>
</tr>
</tbody>
</table>
# COMMON BACTERIAL INFECTIONS OF CHILDHOOD

<table>
<thead>
<tr>
<th>Disease</th>
<th>Agent</th>
<th>Clinical features</th>
<th>Complications</th>
<th>Diagnosis &amp; prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diphtheria</strong></td>
<td>Corynebacterium diphtheriae, gram +ve.</td>
<td>I.P: 3-4 days</td>
<td>Mechanical obstruction of the airways.</td>
<td>Demonstration of organism in stained smears made from the membrane &amp; by culture using Loeffler’s medium.</td>
</tr>
<tr>
<td></td>
<td>3 types: Gravis</td>
<td>Pharyngeal diphtheria: Most common.</td>
<td>Toxic complications: most pronounced in the heart &amp; motor nerves.</td>
<td>Fluorescent antitoxin staining- rapid diagnosis.</td>
</tr>
<tr>
<td></td>
<td>Intermedius</td>
<td>Bull neck (gross cervical lymphadenopathy)</td>
<td>3rd, 6th, 7th, 9th &amp; 10th cranial nerves are commonly affected.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mitis</td>
<td>Malignant diphtheria (oedema of submandibular area)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Virulent strains produce exotoxin.</td>
<td>Laryngeal diphtheria: Produces respiratory obstruction early.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nasal diphtheria</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cutaneous diphtheria: punched out ulcer</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Catarrhal stage: most infective stage.</td>
<td>• Bronchitis  • Bronchopneumonia  • Atelectasis  • Emphysema or pneumothorax.  • Bronchiectasis  • Flare-up of tuberculosis.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Paroxysmal stage: cough starts, increase in severity &amp; becomes repetitive &amp; explosive. Each paroxysm is followed by a whoop (inspiratory effort through a narrowed glottis).</td>
<td>B. CNS:  • Convulsions</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Convalescence stage.</td>
<td>C. GIT:  • Severe vomiting with dehydration.  • Tetany  • Prolapse of rectum  • Hernia</td>
<td>D. Haemorrhages  E. Malnutrition</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Haemophilus influenzae infections

| **Haemophilus influenzae (gram –ve)** | 6 types: a - f based on capsular polysaccharide. PRP (polyribosyl ribitol phosphate) determines its virulence. | Frequently cause pharyngitis in children. | Secondary infection by H. influenzae causes bronchopneumonia & exacerbation of chronic bronchitis. | Gram staining & Culture, CSF, sputum etc. (Organism is highly susceptible to low temperatures. So refrigeration during transit should be avoided. **Prevention:** Hib polysaccharide tetanus protein conjugate vaccine/PRP-T) 2nd, 3rd & 4th months; booster dose: after 2 years. |

### OTHER BACTERIAL INFECTIONS.

#### TYPHOID FEVER

Immunity: one attack confers *life long immunity*, but 2nd attack occurs rarely.

<table>
<thead>
<tr>
<th><strong>Epidemiology</strong></th>
<th>Agent: <em>Salmonella typhi</em>; spread: <em>fecal- oral route</em> through contaminated food, water, ice creams, milk etc. <strong>Infective dose:</strong> $10^7$ organisms. Healthy carriers: <em>fecal carriers or urinary carriers</em>. Chronic asymptomatic carrier state may be present with persistent infections in the <em>gall bladder</em>.</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th><strong>Pathogenesis</strong></th>
<th><strong>Factors determining the establishment of infection:</strong> 1. Size of the inoculum; larger the dose, chance of infection ↑ 2. Normal gastric acid kills the bacilli. 3. Virulence of the infecting strain 4. Presence of bacterial flora in the jejunum.</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th><strong>Pathology</strong></th>
<th><strong>Organ involved</strong></th>
<th>Clinical lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1st week:</strong> temperature rises in a <em>step ladder pattern</em>. <em>Rose spots</em> (due to bacterial embolism) over upper abdomen &amp; chest; slow pulse fever; constipation &amp; less often</td>
<td>1. Meninges</td>
<td>Typhoid meningitis</td>
</tr>
<tr>
<td></td>
<td>2. Lungs</td>
<td>Typhoid pneumonia</td>
</tr>
<tr>
<td></td>
<td>3. Gall bladder</td>
<td>Cholecystitis, Cholelithiasis</td>
</tr>
<tr>
<td></td>
<td>4. Bones</td>
<td>Osteomyelitis, bone abscesses (Brodie’s abscess)</td>
</tr>
<tr>
<td></td>
<td>5. Heart valves, Intravascular prosthesis</td>
<td>Salmonella endocarditis</td>
</tr>
</tbody>
</table>
Clinical features
diarrhea, abdominal pain etc.

2nd week: temp. Continuous (40°C); Tongue coated in the centre, margins reddish (enteric fever)

Signs of toxemia: muttering delirium; subsultus tendinum (tremulousness of the hand); carphology (picking movements of the hands); coma vigil (eyes are open); tumidity (abdomen moderately distended) especially over right iliac fossa.

3rd week: fever & toxemia continues. Complications develop during 3rd week. In uncomplicated cases the temperature starts to fall by lysis at the end of the third week & becomes normal within 1 week.

Complications

<table>
<thead>
<tr>
<th>General</th>
<th>Toxemia &amp; typhoid state, DIC, circulatory collapse, dehydration, relapse etc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>GIT</td>
<td>Abdominal distention, diarrhea, perforation of the intestine (site: terminal 50cm ileum), bleeding from the intestine, toxic hepatitis etc.</td>
</tr>
<tr>
<td>CNS</td>
<td>Coma, meningism, meningitis, peripheral neuritis, deafness.</td>
</tr>
<tr>
<td>Skin</td>
<td>Bed sores, alopecia.</td>
</tr>
<tr>
<td>Others</td>
<td>Myocarditis, pyelonephritis, glomerulonephritis, osteomyelitis, arthritis</td>
</tr>
</tbody>
</table>

Diagnosis

1. WBC count: ↓, relative lymphocytosis
2. Isolation of the organism from blood, urine or feces
3. Feces & urine culture become +ve in 2nd & 3rd week of illness.
4. Typhoid meningitis: CSF contains the organism
5. bone marrow culture
6. Widal test: This test detects & measures the H & O agglutinins of typhoid & paratyphoid bacilli in the patient's serum. The antibody titers increase steadily after the first week till the 4th week & decline. A 4 fold rise in the titer of O antibody occurs within a week. The H antibodies also increase but they tend to be less specific than O antibodies. The O agglutinins are of greater value in diagnosis & titer of 1: 200 or more is very suggestive.

PARATYPHOID FEVERS

- Agent: Salmonella paratyphi A, B, C.
- Illness resembles typhoid, though the toxemia & complications are milder.

SHIGELLA INFECTIONS (BACTERIAL DYSENTERY)

Shigellae: 4 species

- Shigella dysentriae type 1 & 2(S. shigae & S. schimitzii)
- S. flexneri.
- S. sonnei.
- S. boydii.

Source: contaminated food or water.

Pathology

The entire colon may be affected.
Toxins are responsible for secretory diarrhea.
Necrosed mucosa— intestinal casts.

Differentiating features between bacillary & amoebic dysentery

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>Bacillary dysentery</th>
<th>Amoebic dysentery</th>
</tr>
</thead>
<tbody>
<tr>
<td>onset</td>
<td>Sudden</td>
<td>Slow</td>
</tr>
<tr>
<td>I. Period</td>
<td>A few days</td>
<td>Weeks to months</td>
</tr>
<tr>
<td>Number of stools in 24 hours</td>
<td>Over 20</td>
<td>Below 15</td>
</tr>
<tr>
<td>Nature of feces</td>
<td>Mucus &amp; blood</td>
<td>Fecal matter mixed with mucus &amp; blood</td>
</tr>
<tr>
<td>Reaction of feces</td>
<td>Alkaline</td>
<td>Acid</td>
</tr>
<tr>
<td>Clinical course</td>
<td>acute</td>
<td>Sub acute &amp; recurrent</td>
</tr>
<tr>
<td>Microscopy of feces</td>
<td>Cellular exudates</td>
<td>Erythrocyte &amp; Entamoeba histolytica.</td>
</tr>
<tr>
<td>Culture</td>
<td>Shigella isolated</td>
<td>Special methods to culture amoeba.</td>
</tr>
</tbody>
</table>

Complications
- Dehydration
- Electrolyte imbalance
- Circulatory failure
- Renal failure
- Intestinal perforation & rectal bleeding
- Paralytic ileus.

**ANTHRAX (Wool Sorter’s disease)**

Agent: *Bacillus anthracis* (gram +ve)

Source: infected animals, contaminated wool, hair etc.

**Clinical features**

1. Cutaneous form (malignant pustule)
2. Pulmonary form: anthrax pneumonia (wool sorter’s disease)
3. Gastrointestinal form
4. Meningitic form

**Diagnosis**

- Blood Culture: polymorph leucocytosis
- CSF: haemorrhagic in anthrax meningitis
- Agar gel precipitation inhibition test: a four fold rise in serum antibody titer confirms the diagnosis.

**Klebsiella pneumoniae**: Freidlander’s bacillus

**Pseudomonas aeruginosa**: produces a greenish pigment→ pycocyanin

**Proteus**: 4 species: *P. mirabilis, P. vulgaris, P. morganii*, and *P. rettgeri*.

They cause super infection in areas of tissue damage (eg. umbilical stump in newborn→ bacteremia & septic meningitis)

**ESCHERICHIA COLI INFECTIONS**

Group: Enterobacteriaceae.

Gram negative

**Antigens:**
- somatic (O)
- Flagellar (H)
- Capsular(K)

**Clinical presentation**

- UTI
- Peritonitis
- Septicemia
- Neonatal infection
- Biliary tract disease
- Gastroenteritis:
  - Enterotoxigenic strains of E. coli (*ETEC*) cause gastroenteritis in children’s nurseries.
  - Traveller’s diarrhea

**HAEMOLYTIC URAEMIC SYNDROME**

- Coli serotypes O15, K7, and H7 cause haemorrhagic colitis.
- Watery diarrhoea→ bloody.
- Verocytotoxines are responsible for haemolytic uraemic syndrome

**PLAGUE**

- Zoonosis.
- Causative agent: *Yersinia pestis* (previously known as Pasteurella pestis); gram negative.
- Vector: *Xenopsylla cheopis*; infected rat flea.
- Reservoir of infection: infected domestic rodents such as *Rattus norwegius, Rattus rattus & Mus musculus*. 

www.similima.com
Pathogenesis: The organisms enter through the skin. The bacilli reach the local lymph nodes which enlarge & suppurate: Bubo. Bacilli proliferate and enter the blood stream to produce metastatic lesions. Spleen may be enlarged twice or thrice its normal size. In primary pneumonic plague, the organisms reach the lungs through the respiratory tract.

Clinical manifestations:
- Bubonic plague:
  a. I.P: 2-6 Days.
  b. Fever & lymphadenopathy
  d. Pestis minor: milder cases seen during epidemics. These present only with buboes.
- Septicemic Plague:
  a. Chills, fever, tachycardia, headache, vomiting and delirium
  b. Death may occur with in a few days before localizing lesions are evident.
  c. Haemorrhagic manifestations may develop.
- Pneumonic Plague: Primary and Secondary
  a. Primary: More fulminant and rapidly fatal. Organisms reach the lungs by inhalation.
  b. Secondary: Organisms reach the lungs through the blood stream

Diagnosis:
- Diagnosis is confirmed by demonstrating the organisms by smear, culture or animal inoculation studies.
- Bubo-fluid can be aspirated and stained for the organisms.
- Sputum can be stained for the organisms and cultured.
- In septicemic plague blood culture yields the organism. There is neutrophil leukocytosis. Specific antibodies develop in patients who recover from the disease.

Prevention:
A heat killed vaccine (Haffkine) is available for immunizing the population at risk.

BRUCELLOSIS (Undulant fever, Malta fever, Abortus fever)
It is an infectious disease of animals which is transmitted to man by handling infective material.

Causative organism: gram negative coccobacilli.
- Brucella abortus: causes abortion in cattle.
- Brucella melitensis: infection spreads through goat’s milk
- Brucella suis: obtained from pigs.

Pathogenesis:
Organisms enter through GIT, RT, and conjunctiva or through the skin. They pass through the local lymph nodes into the blood stream to localize in the reticuloendothelial system. They multiply in RE tissues to produce granulomas.

Clinical features:
- I.P: 2-3 weeks
- Fever, malaise, sweating, chills, arthralgia & backache.
- Spinal tenderness, arthritis & orchitis are common.
- Chronic brucellosis may present as PUO.

Complications:
- Endocarditis
- CNS: meningoencephalitis, myelitis & polyradiculoneuropathy.
• Nephritis
• Hepatic & splenic suppuration
• Calcification in the liver & spleen & cholecystitis
• Uveitis

**Diagnosis:**

• Clinical features: *typhoid like illness* especially if associated with spondylitis or arthritis.
• Diagnosis is confirmed by isolation of the organism from blood, bone marrow etc.
• Special lab techniques are necessary to grow *brucella*.
• Standard tube agglutination tests, Coomb’s antiglobulin tests, complement fixation test & ELISA test are used for detecting agglutinating antibodies. *Coomb’s test & ELISA are more reliable*.
  If the initial test is negative, *test is repeated after 3-4 weeks*.

**DIARRHOEAL DISEASES OF INFECTIVE ORIGIN**

**CHOLERA**

• **Causative organism:** *Vibrio cholerae*; gram negative. *92 serogroups* are identified based on *O* (somatic) *antigen*. The strain producing epidemic cholera possesses *O1 antigen*; hence this vibrio is designated as *vibrio cholerae O1*.
• *Inaba, Ogawa & Hikojima* are the most important pathogenic subtypes.
• Others are collectively designated *non-O1 V. cholerae*.
• *Eltor biotype* is a variant of *V. cholerae O1*; it is characterized by hemolytic activity & resistance to polymyxin. Differentiation between Eltor & *V. cholerae* is by phage typing.
• *Classic disease* is caused by *Vibrio cholerae*, but the majority of outbreaks occurring in India are due to *Eltor biotype*.
• Main vehicles of infection are water, cooked food kept unhygienically exposed to flies, sea foods, fruits & vegetables.

**Pathogenesis:**

*Vibrio cholerae* multiply in the jejunum & small intestine & produce an enterotoxin. By the influence of this toxin the enterocytes (intestinal mucosal cells) secrete large amount of isotonic fluid. The result is *watery diarrhea* which leads to loss of isotonic fluid. Excessive loss of fluid & electrolyte gives rise to hypovolemic shock & metabolic acidosis.

**Cholera enterotoxin:**

- It is a protein of Mol. Wt: 84000.
- 2 immunologically distinct regions: *A* (active) & *B* (binding).
- Binding enables A region to penetrate the mucosal cells
- This toxin leads to formation of *adenylate cyclase* which induces excessive production of cyclic – AMP (*cAMP*), which in turn is responsible for *over secretion of electrolytes & water by the enterocytes*.

**Clinical features:**

- *Eltor cholera*: usually *mild & asymptomatic*.
- Moderate & severe cases: last for 3-5 days.
  - Painless watery diarrhea & effortless vomiting (clear, watery fluid).
  - Excreta: *rice-water appearance* due to the presence of flakes of mucus & large number of vibrios.
  - Dehydration (when severe: sunken eyes, shriveled skin, collapsed neck veins), acidosis & shock. Thrust with dryness of mouth & tongue (earliest indication of fluid deficit); Oliguria & renal shutdown. Mental state is clear.
  - Painful muscular cramps due to hyponatremia.
  - Cold extremities.
  - Abdomen: scaphoid.
- *Cholera sicca*: rarely large amount of fluid may collect in the intestinal lumen & a severe dehydration, shock & death may result even before evacuation occurs.

**Diagnosis:**

- Isolating the organism from stool sample.
- Suitable medium for transporting specimen is: *Venkataraman & Ramakrishnan fluid* or *Carry & Blair medium*.

**Complications:**
Dehydration & severe shock leads to renal cortical necrosis & renal failure.
- Hypokalemia leads to fatal cardiac arrhythmias, abdominal distension & muscle paralysis.
- Injudicious administration of electrolyte solutions intravenously without correcting metabolic acidosis may result in pulmonary oedema.
- Convulsions in children due to cerebral venous thrombosis.
- Severe hypoglycemia.
- Prolapse of rectum in children.
- Florid malnutrition.

**Rehydration therapy:** ORS (oral rehydration salt) solution.

<table>
<thead>
<tr>
<th>Sodium chloride</th>
<th>3.5g</th>
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<tbody>
<tr>
<td>Sodium bicarbonate</td>
<td>2.5g</td>
</tr>
<tr>
<td>Potassium chloride</td>
<td>1.5g</td>
</tr>
<tr>
<td>Glucose</td>
<td>20g</td>
</tr>
<tr>
<td>Water</td>
<td>1 liter</td>
</tr>
</tbody>
</table>

Cholera is a notifiable disease.

**ROTAVIRUS DIARRHEA**
- Rotavirus affects mainly infants & young children aged 6 months to 2 years.
- **I. P:** 1 to 7 days; usually less than 48 hours.
- **Vomiting occurs early & it precedes diarrhea.**
- Diarrhea extends over 5-7 days but virus is shred up for 10 days.
- **Breast milk may have a protective role** due to the presence of maternal Ig A antibodies.
- **Rotavirus group A:** major cause of endemic diarrhea in infants & young children worldwide.

**Norwalk & Norwalk like agents:** Cause mild gastroenteritis in school, community & family settings.

**Calicivirus:** Cause Rotavirus like illness in children & Norwalk like in adults.

**Astrovirus:** Pediatric diarrhea reported in nursing homes.

**Campylobacter jejuni (vibrio fetus):**

**Complications of campylobacter jejuni dysentery:**
- Reactive arthritis
- Guillain-Barre syndrome(GBS)

**Pseudomembranous colitis:** syn: antibiotic associated diarrhea.

**BARTONELLOSIS (Syn: carrion’ s disease, Oroya fever, Verruga Peruana)**

Bartonella consists of 3 main pathogens.

- **Bartonella bacilliformis:** Carrion’s disease. Disease is transmitted by sandfly *phlebotomous verrucarum*.
- **Bartonella henselae:** cat- scratch disease & bacillary angiomatosis in patients with AIDS.
- **Bartonella Quintana:** trench fever.

**Clinical picture**

2 distinct clinical syndromes:

**A) Oroya fever:**
- i. Fever, rigor, headache etc. lasts for 3-4 weeks.
- ii. Organism can be demonstrated in blood smears stained by Giemsa’s or Wright’s strain.
- iii. Death may occur due to severe anaemia or super infection salmonella.

**B) Verruga Peruana:**
- i. Hemangiomatous tumours of skin and mucous membranes
- ii. Mortality is very low

**LEGIONNAIRE’S DISEASE**

- Causative agent: *Legionellaceae*; gram negative; natural habitat: water
- Over 36 species; among them *L. pneumophila* is the most common human pathogen.

**Clinical features:**
Two main clinical syndromes

a) **Legionnaire’s disease**
- Infection is by inhalation of aerosols
- I.P: 2-10 days
- Pneumonia is the commonest presentation followed by GIT symptoms such as watery diarrhoea

b) **Pontiac fever**
- Brief febrile illness resembling influenza caused by other species of legionella

**Diagnosis:**
- Culture of the organism from the sputum & direct fluorescent antibody staining: quick and ready method but less sensitive

**ANAEROBIC INFECTIONS**

**TETANUS (Syn: Lockjaw)**

- Exotoxin produced by Clostridium tetani.
- Exotoxin: neurotoxin; tetanosasmin & hemolysin; tetanolysin.
- I. P: generally **less than 2 weeks;** it may range from **2-60 days.**

**Clinical features:**
- Diagnosis becomes evident when **lockjaw** sets in.
- **Opisthotonous:** hyperextension of the spine & neck due to rigidity and spasm of back muscles.
- **Risus sardonicus:** grinning expression due to sustained contraction of facial muscles.
- Interval between first symptom & the first convulsion is called the **onset period.**
- Deep tendon reflexes are exaggerated but the plantar response is flexor.
- **Local tetanus:** symptoms confined to a part near the site of injury.
- **Cephalic tetanus:** local tetanus involves the facial muscles.
- **Tetanus neonatorum:** tetanus occurs within 10 days of birth; inability to suck the nipple, irritability & excessive crying associated with grimacing movements of the face. Muscles of the back, neck & abdomen become spastic.

**Complications:**
- Respiratory obstruction & aspiration pneumonia.
- Hyperpyrexia
- Myocarditis leads to cardiac failure & hypotension.
- Decubitus ulcers & UTI due to prolonged immobility.

**SIGNS & SYMPTOMS (COMMONEST, FIRST & MOST IMPORTANT)**

<table>
<thead>
<tr>
<th>Tetanus</th>
<th>Trismus</th>
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<tbody>
<tr>
<td>Brucellosis</td>
<td>Sweating</td>
</tr>
<tr>
<td>Acute myocarditis</td>
<td>Tachycardia</td>
</tr>
<tr>
<td>Impetigo &amp; dermatophytoid</td>
<td>Itching is the only symptom.</td>
</tr>
</tbody>
</table>

**GAS GANGRENE (Syn: Clostridial myonecrosis)**

- **Clostridium perfringens** (C. welchi), C. septicum, C. novyi (C. oedematiens), C. histolyticum & C. sordelli.
- They produce local necrosis & distant lethal effects.
- I.P: 1-4 days.

**ANAEROBIC FOOD POISOINING**

- **Clostridium perfringens food poisoning:**
  - Source of contamination: meat
  - Heat resistant spores survive improper cooking. They germinate when the food is cooled.
  - They produce an exotoxin which causes abdominal cramps & diarrhoea.

- **Clostridium botulinum produces botulism.**
  - I. P: 12-36 hours.
  - Fever absent & GIT symptoms are slight.
Prominent symptoms are dysphagia, diplopia, ptosis, dysarthria, muscle weakness & quadriplegia.

**GENITAL SORE**

<table>
<thead>
<tr>
<th>Features</th>
<th>Syphilis (Syn: Hard chancre, Lues venerea)</th>
<th>Chancroid (Syn: Soft chancre, Soft Sore, Ulcus Molle)</th>
<th>Lymphogranuloma Venereum (Syn: Climatic lymphogranuloma Inguinale)</th>
<th>Granuloma Inguinale (Syn: Granuloma venereum, Donovonosis)</th>
<th>Herpes infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organism</td>
<td><em>T</em>-pallidum</td>
<td>Haemophilus Ducreyi</td>
<td>Chlamydia Trachomalis</td>
<td>Calymmatobacterium Granulomatis (Syn: Donovania granulomatis)</td>
<td>HSV (Herpes Simplex Virus) type 2 &amp; less frequently by HSV type 1.</td>
</tr>
<tr>
<td>Incubation period</td>
<td>9-90 days Average 21 days</td>
<td>3-5 days</td>
<td>2 weeks to several weeks</td>
<td>A few days to a few months.</td>
<td>2-7 days</td>
</tr>
</tbody>
</table>
| Clinical features | ● Painless indurated papule that may later turn on to ulcer. 
 ● Painless 'shotty' lymph adenopathy. | ● Painful non indurated ulcer (soft chancre). 
 ● Painful lymphadenopathy. 
 ● Secondary infection with Vincent's spirochetes may develop. | ● Painless papule that often go unnoticed. Sometimes it may ulcerate. 
 ● After the genital lesion has healed painful matted suppurative lymphadenopathy. 
 ● Multiple sinus tracts are formed in lymph nodes. | ● Begins as one or more subcutaneous nodules that erode through skin to produce clean granulomatous sharply defined painless lesions. 
 ● **Groove’s sign;** enlarged nodes are present below and above the inguinal ligament. 
 ● Vaginal and rectal strictures. 
 ● Elephantiasis of vulva. 
 ● **Esthiomene:** pseudo-elephantoid condition of the genitalia caused by lymphatic obstruction. | ● Painful papule that ulcerate in 3-6 days. 
 ● Burning pain S1 to S5 dermatomes. 
 ● Systemic symptoms fever, headache, malaise. 
 ● Inguinal lymphadenopathy occurs. |

**SYPHILIS**

- Caused by *Treponema pallidum.*
- **Incubation period:** about **9-90 days** (average 21 days)
- *T*.pallidum can cross placenta at any stage of pregnancy but lesions of congenital syphilis develop generally after 4 months of gestation with disappearance of langhans layer (cytotrophoblast) in villi.

**Lesions in syphilis**

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Page 13
1. Congenital Syphilis
   - Early congenital syphilis; infantile form (within first 2 years of birth)
   - Late (tardive) congenital syphilis (seen after age of 2 years)

2. Primary Syphilis
3. Secondary Syphilis
4. Tertiary Syphilis

**Early congenital syphilis** (similar to secondary acquired syphilis)

- Syphilitic snuffles (rhinitis) is earliest feature
- Vesicobullous lesion (*syphilitic pemphigus*)
- Snail track ulcers on mucosa
- Rhagades (radiating fissures at angles of mouth)
- Condylomata
- Old man facies
- Alopecia
- Syphilitic wig: growth of black hair
- Hepatosplenomegaly
- Generalised lymphadenopathy
- Coombs negative hemolytic anaemia
- Bleeding diathesis
- Osteochondritis
- *Syphilitic pseudoparalysis*
- Chorioretinitis
- Gumma
- Interstitial keratitis
- IUGR

**Late congenital syphilis**

- Syphilis snuffles (rhinitis)
- Gumma
- Interstitial keratitis

**Stigmata of congenital syphilis**

- **Hutchinson triad**
  - Hutchinson teeth (peg like incisors), Interstitial keratitis & Eighth nerve deafness
  - Olympic brows (frontal bossing), hot cross bun skull & Parrot nodes due to thickening of frontal & parietal bones of the skull.
  - *Saddle nose* due to destruction of nasal bone.
  - Higuemenaki’s sign (U/L or B/L thickening of sternal portion of clavicle) High arched palate & bulldog facies.
  - Saber shins.

**Primary syphilis**

- A small macule develops at the site of inoculation: primary chancre.
- Painless punched out non-bleeding indurated ulcer
- Painless, rubbery, shotty lymphadenopathy

**Secondary syphilis**

- Bilateral symmetrical, asymptomatic rashes on palms and soles
- Generalized non-tender lymphadenopathy
- Condylomata lata
- Moth eaten alopecia
- Arthritis
- Proteinuria

**Tertiary syphilis**

- Gumma
- Neurosyphilis
- General paresis
**Bone lesions**

Granulomatous periostitis & gummatous osteitis.

**Visceral syphilis**

*Hepar lobatum:* surface of the liver becomes lobular.

**CNS:**

GPI, taboparesis, optic atrophy etc.

**Diagnostic test for syphilis**

- *Absolute diagnosis during the first and second stage* can be made by- Direct examination, under dark ground microscopy.
- Serological tests are of two types
  - Nonspecific Treponemal tests
  - Specific Treponemal antibody tests

<table>
<thead>
<tr>
<th>Serological tests for syphilis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonspecific Tests</td>
</tr>
<tr>
<td>---------------------</td>
</tr>
<tr>
<td>a) VDRL (venereal Disease Reference Laboratory test)</td>
</tr>
<tr>
<td>b) Kahn test</td>
</tr>
<tr>
<td>c) Complement fixation test (Wasserman Test)</td>
</tr>
<tr>
<td>Non treponemal antigen tests are used for monitoring patients response to t/t, they become –ve after t/t</td>
</tr>
</tbody>
</table>

- *Most sensitive test in primary Syphilis* - FTA- ABS
- *Most specific test* - TPI (Treponema pallidum immobilization) > FTA- ABS
- *In secondary Syphilis* all tests are 100% sensitive but mostly used is – VDRL
- *Earliest test to become positive* - FTA-ABS
- *VDRL become positive after* - 3-5 weeks of infection or 7-10 days of chancre.

% of patients with positive serological tests for syphilis

<table>
<thead>
<tr>
<th>Test</th>
<th>Primary syphilis</th>
<th>Secondary syphilis</th>
<th>Tertiary syphilis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonspecific</td>
<td>50-70%</td>
<td>99%</td>
<td>70%</td>
</tr>
<tr>
<td>Specific</td>
<td>70-85%</td>
<td>100%</td>
<td>98%</td>
</tr>
</tbody>
</table>

- *Commonest STD: Nongonococcal urethritis*

**RICKETTSIAL DISEASES**

<table>
<thead>
<tr>
<th>Group</th>
<th>Agent</th>
<th>Vector &amp; reservoir</th>
<th>Clinical features</th>
<th>Weil-Felix reaction</th>
</tr>
</thead>
</table>
| A. Typhus | i. Epidemic typhus (syn: louse borne typhus, typhus exanthematicus, Gaol fever) | R. prowazekii | Body louse (man) | • I.P- 7 days  
• Continuous fever (39-40°C)  
• Macular skin rash appears only on the 5th day  
• Starts to fade by about the 10th day  
• I.P- 8-16 days | +++ve for OX-19 |
### ii. Endemic typhus
(syn: Murine typhus, flea typhus)

- **R. typhi**
- **Rat flea; xenopsylla cheopis (rat)**
- **Continuous fever for 12 days and then comes down by lysis**
- **Morbiliform rash develops in the axillae, arms, abdomen, chest, shoulders and thighs by about the 5th day**
- Persons who suffer from typhus acquire lifelong immunity. Relapse occurs after a long latent period in some cases → Brill-Zinsser disease

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<tbody>
<tr>
<td>ii. Endemic typhus</td>
<td>R. typhi</td>
<td>Rat flea; xenopsylla cheopis (rat)</td>
<td>Continuous fever for 12 days and then comes down by lysis</td>
<td>+++ve for OX-19</td>
</tr>
</tbody>
</table>

### iii. Brill-Zinsser disease

- **R. prowazekii**
- **Body louse (man)**

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<tbody>
<tr>
<td>iii. Brill-Zinsser disease</td>
<td>R. prowazekii</td>
<td>Body louse (man)</td>
<td>Usually negative</td>
</tr>
</tbody>
</table>

### B. Spotted fever

#### i. Rocky mountain spotted fever

- **R. rickettsi**
- **Ticks (rabbit, dog & rodents)**

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<tbody>
<tr>
<td>i. Rocky mountain spotted fever</td>
<td>R. rickettsi</td>
<td>Ticks (rabbit, dog &amp; rodents)</td>
</tr>
</tbody>
</table>

#### ii. Mediterranean fever
(fever boutonneuse)

- **R. conori**
- **Ticks (dog & rodents)**

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<tr>
<td>ii. Mediterranean fever</td>
<td>R. conori</td>
<td>Ticks (dog &amp; rodents)</td>
</tr>
</tbody>
</table>

#### iii. Rickettsial pox

- **R. akari**
- **Gamasid mite (mouse)**

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<tbody>
<tr>
<td>iii. Rickettsial pox</td>
<td>R. akari</td>
<td>Gamasid mite (mouse)</td>
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</table>

#### iv. North Asian tick borne typhus

- **R. sibera**
- **Tick (wild animals, cattle, birds)**

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<tbody>
<tr>
<td>iv. North Asian tick borne typhus</td>
<td>R. sibera</td>
<td>Tick (wild animals, cattle, birds)</td>
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</tbody>
</table>

#### v. Queensland tick typhus

- **R. australis**
- **Tick (bush rodents)**

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<tbody>
<tr>
<td>v. Queensland tick typhus</td>
<td>R. australis</td>
<td>Tick (bush rodents)</td>
</tr>
</tbody>
</table>

#### vi. Indian tick typhus

- **R. conori**
- **Tick (rodents)**

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<tbody>
<tr>
<td>vi. Indian tick typhus</td>
<td>R. conori</td>
<td>Tick (rodents)</td>
</tr>
</tbody>
</table>

#### vii. African tick bite fever

- **R. africai**
- **Ticks (cattle & goat)**

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<tbody>
<tr>
<td>vii. African tick bite fever</td>
<td>R. africai</td>
<td>Ticks (cattle &amp; goat)</td>
</tr>
</tbody>
</table>

### C. Scrub typhus

(Syn: Mite typhus, mite fever, Japanese river fever, Tsutsugamushi fever)

- **R. tsutsugamushi**
- **Trombiculid mite (small rodents, birds)**

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<tbody>
<tr>
<td>C. Scrub typhus</td>
<td>R. tsutsugamushi</td>
<td>Trombiculid mite (small rodents, birds)</td>
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</tbody>
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<tr>
<td>C. Scrub typhus</td>
<td>R. tsutsugamushi</td>
<td>Trombiculid mite (small rodents, birds)</td>
</tr>
</tbody>
</table>

### D. Q fever

- **Coxiella burnetii**
- **Ticks transmit disease in animals; human disease is occupational- cattle, sheep, goat**

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<td>D. Q fever</td>
<td>Coxiella burnetii</td>
<td>Ticks transmit disease in animals; human disease is occupational- cattle, sheep, goat</td>
</tr>
</tbody>
</table>

### E. Trench fever

- **R. quintana**
- **Body louse (man)**

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<tbody>
<tr>
<td>E. Trench fever</td>
<td>R. quintana</td>
<td>Body louse (man)</td>
</tr>
</tbody>
</table>

### NON VENEREAL TREPONEMATOSIS

#### YAWS
- Treponema pertenue.
- Primary stage: maculopapular lesions.
- Secondary stage:
  - Crab yaws - over the palms & soles lesions become painful & walking may be restricted due to pain.
  - Gondou - thickening of nasal bones.
- Tertiary stage:

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<tbody>
<tr>
<td>NON VENEREAL TREPONEMATOSIS</td>
<td>YAWS</td>
<td></td>
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</tbody>
</table>

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- **Gangosa**: extensive destruction of facial tissues with gross mutilation of the nose & mouth leads to the formation of a single open cavity.

**PINTA**: Treponema carateum.

## RELAPSING FEVERS

### LOUSE- BORNE RELAPSING FEVER
- Borrelia recurrentis.
- **Vector**: body louse; pediculus humanus corporis.
- Allopathic drug therapy may lead to severe **Jarisch-Herxheimer reaction** with profound fall in temperature, shock & cardiac failure within a few hours.

### TICK- BORNE RELAPSING FEVER
- Borrelia duttoni.
- **Vectors**: soft ticks - ornithodoros tholozoni, O. crossi, O. lahorensis, O. moubata.
- Jarisch-Herxheimer reaction is less pronounced.

### LYME BORRELIOSES
- Borrelia burgdorferi.
- **Vector**: hard ticks; Ixodes ricinus, Ixodes persulcatus, I. dammini, I. pacificus & I. scapularis.
- Stage 1: erythema migrans; rash spreading centrifugally.
- Stage 2: early disseminated infection.
- Neurological features: **Lyme Neuroborreliasis**.
- Meningoradiculitis: **Bannwarth’s syndrome**: CSF pleocytosis & severe radicular pain.
- Jarisch-Herxheimer reaction develops rarely.

### LEPTOSPIROSIS
- Leptospira: 2 species
  - **L. Interrogans**
    - L. icterohemorrhagiae: rat & rodents
    - L. seroje: pig
    - L. canicola: dog
    - L. Pomona: cattle
  - **L. biflexa**
- Leptospirae are eliminated in the urine of their animal hosts.
- **I. P**: 10 days.
- Only 5-10% of persons develop the clinical illness.
- Male, 20-30 age group.
- Nonspecific features of viral fever.
- Conjunctival suffusion, headache, chills & jaundice.
- **4 groups**:
  - common leptospiral syndrome (CLS) 20%
  - CLS+ bleeding tendency 5%
  - CLS+ meningitis 5%
  - CLS+ involvement of abdominal organs 70%
- Hepato-renal lesions predominate in L. icterohemorrhagiae infection (**weil’s disease**)
- Pretibial skin lesions predominate in L. autumnalis (**Fort Bragg fever**)
- Meningeal symptoms prominent in L. canicola infection (canicola fever)
- Complications: Multi organ failure; renal failure, cardiac failure, respiratory failure, thromobocytopenia with bleeding etc.
- Unlike viral hepatitis SGPT are low compared to the degree of jaundice.
- Leptospirae may be demonstrable in urine.
- Serology is positive after 1-2 weeks. Microscopic agglutination test (MAT) & indirect hemagglutination (IHA) are routinely employed.

### MYCOBACTERIAL INFECTIONS

### TUBERCULOSIS
- **Mycobacterium tuberculosis**.
- Lymphocytes play a major role in conferring immunity against Mycobacterium tuberculosis.

**Pathology:** macrophages with engulfed organisms get transformed into epitheloid cells. *Langhan’s giant cells* are formed by the fusion of epitheloid cells. These are surrounded by lymphocytes & fibroblasts. Caseation necrosis occurs at the centre. Pathological hallmark of TB is *tubercle*. Granuloma formation limits the infection. Some bacilli remain in these tissues as persisters.

**PRIMARY PULMONARY TUBERCULOSIS:**
- Initial lesion is a *sub pleural tubercle located in the upper part of lower lobe, lower part of upper lobe or middle lobe* - *Ghon’s lesion.*
- Ghon’s lesion with enlarged lymph nodes & interconnecting lymphangitis: *primary complex or Ghon’s complex.*
- Compression of neighboring bronchi especially of middle lobe leads to collapse-consolidation & bronchiectatic changes; this may later present as *middle lobe syndrome.*
- Fever, loss of appetite, loss of weight & cough: early symptoms.
- Allergic manifestations like phlyctenular conjunctivitis or erythema nodosum may herald primary TB in some cases.
- Presentation in children includes *tuberculous pneumonia, hemoptysis or asthma.*
- **Physical examination:** in some, signs of pulmonary consolidation, collapse or pleural effusion.
- Tuberculin skin test (Mantoux test) is positive.
- Blood: ESR raised & lymphocytosis.
- Chest X-ray: hilar lymphadenopathy & pulmonary lesion may be detected by careful examination.

**POST PULMONARY TB** (*lesion occurring in sensitized individual):
- **Pathology:**
  1. Direct progression of primary lesion
  2. Reactivation of a primary focus
  3. Haematogenous infection of lung from lymph nodes, tonsils etc
  4. Reinfection or super infection.

  - *Upper lobes are commonly affected.*
  - 2 forms: *slowly progressive nodular form & fibrocaseous form with tendency for cavitations (many of the blood vessels in the cavity are occluded by thrombosis, but those that remain patent become aneurysmal)* (*Rassmussen’s aneurysms*).
  - Evening rise of temperature, mild & persistent cough, anorexia, loss of weight & general weakness.
  - Lesion resembles *lobar pneumonia.*

### Complications of pulmonary tuberculosis

<table>
<thead>
<tr>
<th>Early (within months)</th>
<th>Intermediate (within several months)</th>
<th>Late (after several years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild haemoptysis</td>
<td>Massive haemoptysis</td>
<td>Pulmonary fibrosis with compensatory emphysema &amp; cor pulmonale.</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>Secondary infection of cavities</td>
<td>Bronchiectasis</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td>Pneumothorax, pleural effusion, empyema.</td>
<td>Persistence of open cavities without healing</td>
</tr>
<tr>
<td><strong>Poncet’s syndrome (allergy to TB; polyarthritis occurring in TB.)</strong></td>
<td>Progressive fibrosis with dyspnoea.</td>
<td>Aspergilloma; growth of Aspergillus fumigatus in the cavities in the form of a fungus ball.</td>
</tr>
<tr>
<td></td>
<td>Spread to other organs such as larynx.</td>
<td>Apical TB with carcinoma.</td>
</tr>
<tr>
<td></td>
<td>Non-healing of lesion.</td>
<td>Secondary amyloidosis.</td>
</tr>
</tbody>
</table>

**Sputum examination**
- Most important diagnostic investigation.
- At least 3 bacilli should be seen before the smear is declared positive.

**Tuberculin skin test**
- Test is done by injecting PPD in the forearm intradermally. The result is read after 48 hours & the indurations is recorded (+ve: >12mm)
o Diagnostic value is in those individuals who are recent converters. This should suggest the occurrence of infection by mycobacteria.

o Negative results: malnutrition, immunosuppresion therapy & Hodgin’s disease.

o Patients suffering from active TB show an accelerated arthus reaction.

**MILIARY TUBERCULOSIS (Syn: acute hematogenous tuberculosis)**

° Tubercle bacilli entering the blood stream are diffusely disseminated & results in military tuberculosis.

° More common in young children in whom it is seen as a complication of primary TB.

° Areas of caseated vasculitis (Weigert’s foci) occurring in veins or arteries result in the discharge of bacilli into the circulation.

° Majority of cases present varying grades of pyrexia.

° Choroids tubercles may occur rarely but if they occur they are diagnostic.

° Skiagrams of the chest shows numerous, small rounded shadows - the military mottling in upper lobes (in conditions such as pulmonary hemociderosis, eosinophilia, disseminated carcinoma, pneumoconiosis, sarcoidosis & histoplasmosis mottling is more prominent in the lower zones).

**TUBERCULOUS MENINGITIS**

° Develops commonly as a complication of military tuberculosis.

° Small granulomas are formed in the superficial layers of the brain (Rich foci).

° Death occurs in untreated cases in 4-8 weeks.

° Complications:
  
  • Acute: internal hydrocephalus, cerebral infarction, cranial nerve palsies, convulsions, fluid & electrolyte disturbances.
  
  • Chronic: obstructive hydrocephalus, optic atrophy, subdural effusions spinal cord compression etc.

° CSF shows rise in pressure, turbidity & a rise in lymphocytes upto 500-1000 cells/cmm. A fine coagulum (cobweb) forms in the CSF when kept at room temperature for 6-24 hours; this is highly suggestive of tuberculous meningitis. Proteins are moderately increased. Sugar is reduced to less than 50 % of the blood sugar.

**NON-TUBERCULOUS MYCOBACTERIA**

° **GROUP 1**: Photochromogenic: produce yellow or orange pigment on exposure to light, example, M. kansasii, M. marinum.

° **GROUP 2**: Scotochromogenic: produce yellow, orange or reddish pigment in the dark, example, M. scrofulaceum.

° **GROUP 3**: Nonchromogenic: does not produce pigment on exposure to light, example, M. avium intracellulare, M. ulcerans.

° **GROUP 4**: Rapidly growing mycobacteria: organism rapidly grows at 25º C, example, M. fortuitum, M. chilonei.

Lesions caused by non-tuberculous mycobacteria

<table>
<thead>
<tr>
<th>M. marinum (swimming pool bacillus, fish tank bacillus)</th>
<th>Ulceration of the skin</th>
</tr>
</thead>
<tbody>
<tr>
<td>M. ulcerans</td>
<td>Extensive ulceration of the skin &amp; subcutaneous tissue (Buruli ulcer)</td>
</tr>
<tr>
<td>M. kansasii M. scrofulaceum</td>
<td>Pulmonary disease, local abscesses, bone &amp; joint lesion.</td>
</tr>
<tr>
<td>M. chilonei</td>
<td>Pulmonary disease, local abscesses,</td>
</tr>
<tr>
<td>M. avium intracellulare</td>
<td>Lymphadenopathy, pulmonary lesions, AIDS related</td>
</tr>
</tbody>
</table>

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LEPROSY (Syn: Hansen’s disease)
° *Mycobacterium leprae.* Portal of entry: abrasions in the skin & mucous membranes of upper respiratory tract.
° I.P: 2-7 years.
° In lepromatous leprosy males are twice more commonly affected than women.
° Cell mediated immunity (CMI) which develops against the lipid components of *M. leprae* determines the progress of disease.
° Two polar forms are lepromatous & tuberculoid types.
° It affects mainly peripheral nerves. It also affects skin, muscles, eye, bones, testes & internal organs.
° Leprosy is characterized by one of the following cardinal features:
  - Hypopigmented patches.
  - Partial or total loss of cutaneous sensation in the affected areas; the earliest sensation to be affected is temperature, specially the modality of cold sensation.
  - Presence of thickened nerves.
  - Presence of acid fast bacilli in skin & nasal smears.
° Classification
  i. Indeterminate type: 1 or 2 hypopigmented macule. Bacteriologically negative.
  ii. Tuberculoid: one or 2 small well defined lesion. Bacteriologically negative.
  iii. Borderline: Bacteriological positivity of this lesion is variable. Satellite lesions are seen.
  v. Pure neuritic: skin smear is negative.
° In paucibacillary leprosy, the bacterial index is less than 2; in multibacillary leprosy, it is greater than 2.
° Cardinal signs of leprosy includes: loss of eyebrow, hypoanaesthetic patches, ulnar nerve thickening.
° Best diagnostic method for leprosy: split skin smear.
° Lepromin test: reaction is read at 48 hours (Fernandez reaction) & 21 days (Mitsuda reaction). If the diameter is more than 10mm at the end of 48 hours the test is positive. Lepromin test is not a diagnostic test. It is used in estimating the prognosis. The test is strongly positive in tuberculoid case. Lepromatous cases are Lepromin negative.

CHLAMYDIAL INFECTIONS
PSITTACOSIS (Syn: Parrot fever)
° *Chlamydia psittaci;* primary pathogen of bird.
° *Chlamydia psittaci* reaches the pulmonary alveoli & the reticuloendothelial cells of the spleen & the liver.
° I. P: 10 days.
° Fever, sore throat, cough, pulmonary consolidation etc.
° Respiratory failure & toxaemia are the usual causes of death (20%).

VIRAL INFECTIONS
° Only single stranded DNA virus - Parvovirus
° Only double stranded RNA virus - Reovirus
° Naked DNA viruses are - Parvo, Adeno, Papova viruses
° Naked RNA viruses are - Picorna, Calci, Reoviruses
° Segmented RNA viruses are - Bunya, Orthomyxo, Retro, Arenaviruses
° Circular viruses are dsDNA - Papovavirus
Circular viruses are ssRNA - Bunya, Arenavirus

**Viral inclusion bodies**

<table>
<thead>
<tr>
<th>Intracytoplasmic</th>
<th>Intranuclear</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negri body</td>
<td>Councilmann body</td>
</tr>
<tr>
<td>Guarnieri’s body</td>
<td>Small pox</td>
</tr>
<tr>
<td>Henderson Paterson body</td>
<td>Molluscum contagiosum</td>
</tr>
<tr>
<td>Bollinger, Borrel’s body</td>
<td>Fowl pox</td>
</tr>
</tbody>
</table>

- Babes- Ernst, volutin granules: C. diphtheria
- Levinthal Cole Lillie bodies: Psittacosis.
- Miyagawa’s granulocorpuscles: LGV.

**VIRUS**

<table>
<thead>
<tr>
<th>Type of viruses</th>
<th>Names</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DNA Viruses</strong></td>
<td></td>
</tr>
<tr>
<td>Adenovirus</td>
<td>Adenoviruses, Herpes simplex virus types 1&amp;2 (HSV), CMV, EBV, Varicella-zoster virus</td>
</tr>
<tr>
<td>Parvovirus</td>
<td>Parvovirus B19</td>
</tr>
<tr>
<td>Pox virus</td>
<td>Vaccine virus, Viral virus, Polyoma virus</td>
</tr>
<tr>
<td>Papovirus</td>
<td></td>
</tr>
<tr>
<td><strong>RNA viruses</strong></td>
<td></td>
</tr>
<tr>
<td>Picornavirus</td>
<td>Poliovirus, Coxsackie virus, Echovirus, Rhinovirus, Enterovirus</td>
</tr>
<tr>
<td>Togavirus</td>
<td>Rubella virus, Alphavirus, Flavivirus</td>
</tr>
<tr>
<td>Reovirus</td>
<td>Reovirus, Rotavirus, Influenza viruses A,B,C</td>
</tr>
<tr>
<td>Orthomyxovirus</td>
<td>Measles virus, Mumps virus</td>
</tr>
<tr>
<td>Paramyxovirus</td>
<td>Rabies virus, Lassa virus</td>
</tr>
<tr>
<td>Rhabdovirus</td>
<td></td>
</tr>
<tr>
<td>Arenavirus</td>
<td></td>
</tr>
<tr>
<td>Retrovirus</td>
<td></td>
</tr>
<tr>
<td><strong>Viruses associated with gastroenteritis</strong></td>
<td>Rotavirus(groups A-E), Enteric adenovirus (type 40&amp;41), Norwalk and related viruses, Astrovirus, Calicivirus, Parovirus</td>
</tr>
<tr>
<td><strong>Viral infections associated with a maculopapular rash</strong></td>
<td>Arbovirus infection, Adenovirus infection, Cytomegalovirus infection, Enterovirus infection, Measles, Rubella, Rubeola, Infectious mononucleosis, Hepatitis B virus</td>
</tr>
<tr>
<td><strong>Viral infections associated with haemorrhages</strong></td>
<td>Arbovirus, Mosquito borne</td>
</tr>
<tr>
<td></td>
<td>Yellow fever, Dengue haemorrhagic fever, Rift valley fever, Chikungunya.</td>
</tr>
<tr>
<td>TICK BORNE</td>
<td>PARA MVXIVIRUS</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Kyassanur forest disease, Congo haemorrhagic fever.</td>
<td>Lassa fever, Epidemic haemorrhagic fever</td>
</tr>
</tbody>
</table>

### DISEASES CAUSED BY VIRUSES

#### Parvovirus B19
- Erythema infectiosum (*fifth disease*); slapped face appearance.
- Symmetric polyarthritis in adults
- Fetal abnormalities in utero
- Aplastic crisis
- Anaemia in patients with malignancy (eg. ALL)

#### Human papilloma virus (HPV)

<table>
<thead>
<tr>
<th>Type</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Plantar warts</td>
</tr>
<tr>
<td>3</td>
<td>Flat warts</td>
</tr>
<tr>
<td>5/8</td>
<td>Skin squamous cell carcinomas (SCC) in transplant patients.</td>
</tr>
<tr>
<td>7</td>
<td>Warts in food handlers.</td>
</tr>
<tr>
<td>6 &amp; 11</td>
<td>Genital warts (condylomata acuminata); type 6 HPV.</td>
</tr>
<tr>
<td>13</td>
<td>Juvenile laryngeal papillomatosis</td>
</tr>
<tr>
<td>16</td>
<td>Oral leukoplakia</td>
</tr>
<tr>
<td>30</td>
<td>SCC cervix, penis, vulva.</td>
</tr>
</tbody>
</table>

#### Varicella/ Zoster virus
- Chickenpox, Herpes zoster.

#### Herpes virus hominis

**Type 1**
- Herpes labialis (Cold sore)
- Keratoconjunctivitis
- Whitlows
- Primary stomatitis
- Encephalitis
- Genital infection (40%)
- Genital infection (60%)
- Neonatal infections

**Type 2**

#### Enteroviruses (1 & 2)

1. Coxsackie virus.
   - Myocarditis, Pericarditis
   - Pharyngitis, Meningitis
   - Gastroenteritis
   - Bronholm disease
   - Neonatal infection

2. Echovirus
   - Meningitis, Encephalitis, Conjunctivitis
   - Pharyngitis, Gastroenteritis, Neonatal infection

#### Lymphotrophic viruses (1–4)

1. Epstein-Barr virus (EBV)
   - Infectious mononucleosis
   - Burkitt’s lymphoma
   - Nasopharyngeal carcinoma
   - Hairy leukoplakia (AIDS patients)
   - Relative and atypical lymphocytosis

2. Cytomegalovirus (CMV)
   - Infection in immunocompromised patients-pneumonitis, retinitis, generalized infection, Atypical lymphocytosis—less common

3. Human herpes virus 6 (HHV-6) (also called B-cell lymphotropic virus, HBLV)
   - Mononucleosis like syndrome in adults
   - Roseola infantum in children (exanthema subitum)
   - Atypical lymphocytosis
   - Nontender cervical lymphadenopathy

4. Adenovirus
   - Acute pharyngitis in children, conjunctivitis+ Pharyngitis in children
   - Acute follicular conjunctivitis in adults
   - Rarely pneumonia in adults
HIV (Human immuno deficiency virus a retrovirus)  | AIDS, infectious mononeucleosis like illness, Asymptomatic HIV infection, AIDS related complex (ARC also called prodormal AIDS), Persistent generalized lymphadenopathy (PGL) Thrombocytopenic purpura AIDS Dementia complex, Psychosis, Encephalitis
---|---
Influenza virus (A orthomyxo virus)  | Pandemics, epidemic of influenza Milder outbreaks in smaller groups e.g. school camps Rare
| Influenza A virus  |  
| Influenza B virus  |  
| Influenza C virus  |  

- The most common cause of sporadic encephalitis- HSV-1
- The most common cause of epidemic viral encephalitis- Arbovirus
- The most common cause of viral meningitis- Enterovirus
- Mumps virus does not cause pneumonia
- Measles virus does not cause Aseptic meningitis.

**HUMAN HERPES VIRUS GROUP**

<table>
<thead>
<tr>
<th>Human Herpes virus</th>
<th>Common name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>Herpes simplex type-I</td>
</tr>
<tr>
<td>Type II</td>
<td>Herpes simplex type-II</td>
</tr>
<tr>
<td>Type III</td>
<td>Herpes Zoster virus type</td>
</tr>
<tr>
<td>Type IV</td>
<td>Epstein Barr type I</td>
</tr>
<tr>
<td>Type V</td>
<td>Cytomegalovirus</td>
</tr>
<tr>
<td>Type VI</td>
<td>Human B cell Lymphotropic virus</td>
</tr>
<tr>
<td>Type VII</td>
<td>R.K. virus</td>
</tr>
</tbody>
</table>

**HUMAN IMMUNO DEFICIENCY VIRUS (HIV) INFECTION AND ACQUIRED IMMUNO DEFICIENCY SYNDROME (AIDS)**

- **Causative organism:** HIV virus; Lentivirinae sub-family of retroviruses; RNA viruses.
- **Two main types pathogenic to man are**
  - HIV-1:
    - Produce more severe disease.
    - Divided into group M(11 sub-types- A to K), Group O (9 sub types) and group N (new viruses)
    - C is the main sub-type of HIV 1 seen in India
  - HIV-2:
    - Producing milder disease.
    - Divided into 5 sub-types (A-E)
- **Pathogenesis:**
  - With the steady increase in the number of HIV, the number of CD4+ lymphocytes progressively fall. Normal CD4+ T cell count is 800-1200/ cmm.
  - The predominant opportunist infections which affect the immuno-compromised host include:
    - Mycobacterium tuberculosis
    - Pneumocystis carinii
    - Fungi including candida, aspergillus, Cryptococcus, and histoplasma.
    - Protozoa including toxoplasma, Entamoeba histolytica and other amoebae, leishmania donovani, Giardia lamblia.
    - Helminthes such as Strongyloides stercoralis, scabies and possibly several others.
  - Infected monocytes carry the virus to the central nervous system and lead to the development of lesions.
  - The virus can be identified in the plasma by PCR and their number quantitatively determined.
- **Identification of HIV infection:** Preliminary test to detect infection is the presence of antibody to HIV in serum by ELISA. Confirmation by immunoblot test (Western blot test).
ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS)

- Majority of HIV infected persons develop AIDS with in 10-11 years after the infection.
- Rapid progressers develop disease within 2-3 years.
- Above 10% do not develop the disease due to protective genetic and HLA factors: non-progressers.
- I.P: Few months to 14 years (mean of 5.5 years)

Clinical features:

- Initial symptoms include fever, headache, transient arthralgia, maculopapular rash, tender generalized lymphadenopathy, mouth ulcers, diarrhea & rarely signs of encephalopathy.
- An early manifestation is persistent generalized lymphadenopathy (PGL).
- 3 groups:
  - Group A: Acute HIV infection & asymptomatic patients.
  - Group B: symptomatic, but no AIDS specific features.
  - Group C: those who have AIDS.
- CDC & WHO defined the following condition as suggesting AIDS in HIV positive individual:
  
Infections

1. Disseminated cytomegalovirus infection
2. Mucocutaneous disseminated herpes simplex infection- exceeding 1 month duration.
3. Infection by papovirus giving rise to multifocal leucoencephalopathy.
4. Tuberculosis with CD4+ count <200/cmm- pulmonary or extrapulmonary.
5. Other mycobacteria: Kansasi, Avium intracellulare
6. Pneumocystis carinii pneumonia.
7. Candidiasis of oesophagus, bronchi and lung.
8. Cryptosporidiosis more than 1 month duration.
10. Isosporiasis.
11. Disseminated other fungal infections- histoplasmosis, Coccidioidomycosis, Cryptococcosis.
12. Extra intestinal strongyloidosis.

Neoplasms

1. Kaposi’s sarcoma
2. Primary lymphoma- brain
3. Non-Hodgkin’s lymphoma

Many infections which remain localized in immuno-competent subjects tend to become generalized. These include:

1. Bacterial septicemias
2. M.tuberculosis
3. M. avium intracellulare
4. Toxoplasma gondii
5. Cytomegalovirus and other viruses
6. Cryptococcus neoformans
7. Histoplasma capsulatum

COMMON VIRAL INFECTIONS OF THE RESPIRATORY TRACT

<table>
<thead>
<tr>
<th>Disease</th>
<th>Causative agent</th>
<th>Clinical features</th>
<th>Complications</th>
<th>Diagnosis</th>
</tr>
</thead>
</table>

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<table>
<thead>
<tr>
<th>Virus</th>
<th>Clinical Features</th>
</tr>
</thead>
</table>
| Coryza Rhinoviruses | • *I. P:* 3-4 days  
  • Headache, nasal congestion & obstruction.  
  • Mild fever & muscle pain.  
  • Lasts for 2-3 days.  
  • Sinusitis  
  • Lower respiratory tract infection.  
  • Otitis media.  
  | Clinical diagnosis. |
| Influenza | 3 groups of *myxoviruses*  
  Important among them:  
  • Influenza virus A  
  • Influenza virus B  
  • Influenza virus C  
  • *I. P:* few hours to 48 hours.  
  • Sudden onset with fever (38-40°C), severe generalized myalgia, prostration & injected conjunctiva.  
  • Lasts for 3-4 days.  
  • Pulmonary complications:  
  o Primary influenza virus pneumonia.  
  o Infuenzal pneumonia with secondary bacterial infections.  
  o Bacterial pneumonia.  
  • Cardiac complications:  
  o Toxic myocarditis.  
  o Cardiac failure.  
  • Neurological complications:  
  o Febrile convulsion  
  o Meningitis  
  o Meningoencephalitis  
  o Encephalitis  
  o *Reye’s syndrome* (hepatic failure with encephalopathy & rise in intracranial tension).  
  | Clinical diagnosis  
  • Isolation of virus from throat washings or sputum. |
| Parainfluenza | *Parainfluenza viruses; paramyxovirus group.*  
  • *I. P:* 5-6 days  
  • Type 1: laryngotracheobronchitis or croup.  
  • Type 2: tracheobronchitis, bronchiolitis & bronchopneumonia  
  | Secondary bacterial infection  
  o Otitis media  
  o Sinusitis  
  o Bronchiectasis  
  | Sputum  
  • Clinical features. |
| Respiratory Syncytial Virus infection (RSV) | *Paramyxovirus family Pneumovirus genus.*  
  • Lower respiratory tract infection.  
  • *I.P:* 3-5 days.  
  | Severe bronchiolitis leading to respiratory failure.  
  • Death in children below 5 years.  
  | Demonstration of RSV antigens in nasal washings. |
## EXANTHEMES & ENANTHEMES

<table>
<thead>
<tr>
<th>Disease</th>
<th>Causative organism</th>
<th>Pathology</th>
<th>Clinical features</th>
<th>complications</th>
<th>Diagnosis</th>
</tr>
</thead>
</table>
| **Measles** (Rubeola – red spots - Arabic) | Paramyxovirus (RNA virus) | ❖ Droplet infection.  
❖ Portal of entry: respiratory mucous membrane & conjunctiva.  
❖ Koplik’s spots in cheeks consists of vesicles & necrotic epithelium.  
❖ Multinucleated giant cells with **Warthin Finkelday cells** (cytoplasmic inclusion bodies) found in the hyperplastic lymphoid tissues of lymph nodes, tonsils etc. | ❖ *I. P*: 9-11 days.  
❖ High fever, excessive lachrymation, hacking cough & nasal discharge.  
❖ Koplik’s spots: 1-2 days before the rash.  
b. Bronchitis.  
c. Bronchiolitis  
d. Pneumonia  
b. Keratitis.  
c. Blindness.  
❖ Ear: a) Otitis media; most common complication.  
❖ Heart: Myocarditis.  
❖ GIT: Diarrhoea.  
❖ CNS: SSPE (Subacute Sclerosing Panencephalitis: progressive dementia & motor weakness); late complication.  
Secondary bacterial infection. | ❖ Leucopenia in early stages.  
❖ Leucocytosis with secondary bacterial infection.  
❖ CSF: raised protein & lymphocytosis in encephalitis.  
❖ Immunofluorescence: virus antigen can be detected.  
❖ Multinucleated giant cells in Giemsa stained smears of nasal secretions. |
| **Small pox** (Variola) | Variola virus | Cytoplastic inclusion bodies: **Guarnieri bodies**; these are aggregates of the virus particles are called **Paschen bodies**. | ❖ *I. P*: 12 days)  
❖ Rash appears on 3rd or 4th day  
❖ Rash is centrifugal; more on face & extremities.  
❖ Axilla is usually free.  
❖ Rash comes out as a single crop during 1-2 days.  
❖ Rash is in the same stage all over.  
❖ Multilocular & umbilicated vesicles.  
❖ Deep permanent scarring. | ❖ Secondary bacterial infection causes pneumonia, osteomyelitis, septicemia etc.  
❖ CNS: Encephalitis  
❖ Laryngeal oedema. | ❖ Clinical features  
❖ Identification of guarnieri bodies from the vesicular material.  
❖ Isolation of the virus. |
| Chickenpox  
| Varicella zoster virus (V-Z) |
| Vesicles contain serum, polymorphs & multinucleated giant cells. |
| Reye's syndrome may develop during acute phase especially in children and infants. |
| I. P: 14-16 days. |
| Skin rashes (exanthema) come in crops during the first day of fever. |
| Vesicles: unilocular, superficial, elliptical & clear fluid in the beginning (tear drop vesicles). |
| Thrombocytopenia: —hemorrhage. |
| Superadded staphylococcal infection. |
| Bullous lesions in children with impetigo. |
| Varicella gangrenosa: superinfection by hemolytic streptococci. |
| Primary varicella pneumonia. |
| CNS: meningitis, encephalitis. |
| Congenital malformations: if it occurs during 1st & 2nd trimester of pregnancy. |

| Herpes Zoster  
| Varicella zoster virus (V-Z) |
| After chickenpox, V-Z virus becomes latent in ganglia along the entire neuraxis, particularly in trigeminal ganglia & the dorsal root ganglia, remaining mainly in the cytoplasm of the neurons. Attacks are precipitated by immunodeficiency states. |
| Unbearable lancinating, deep-boring or burning pain. |
| Sites of eruption: ophthalmic & maxillary divisions of trigeminal nerve, geniculate ganglion of facial nerve & thoracic & abdominal nerve roots. |
| Rash may involve a whole dermatome or a part of it. |
| The eruptions seldom cross the midline. |
| Pain may persist for months or even years especially in elder patients: post-herpetic neuralgia. |
| Secondary infection. |
| Ophthalmic herpes. |
| Herpes of geniculate ganglion presents as facial nerve palsy & vesicles over ipsilateral auditory meatus (Ramsay-Hunt syndrome). |
| Cervical herpes zoster —weakness of arm. |
| Generalized herpes |
| Congenital malformation of fetus in pregnancy. |
| Post-herpetic neuralgia. |

| Herpes Simplex  
| HSV (Herpes simplex) |
| Clinical features. |
febrillis, fever blister, cold sore)

<table>
<thead>
<tr>
<th>Virus</th>
<th>Description</th>
</tr>
</thead>
</table>
| Herpes Simplex | Recurrent crops of vesicles over the mucocutaneous regions.
| HSV 1 | Sexually transmitted disease. |
| HSV 2 | | |

**Herpes Simplex Virus (HSV)**
- HSV 1
- HSV 2
- Recurrent crops of vesicles over the mucocutaneous regions.
- Sexually transmitted disease.

**Rubella (German measles-three days measles)**

<table>
<thead>
<tr>
<th>Virus</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Togavirus</td>
<td>Lymph nodes are moderately enlarged (occipital, posterior auricular &amp; posterior cervical lymph nodes), showing oedema &amp; hyperplasia.</td>
</tr>
</tbody>
</table>

**Rubella**
- Togavirus; RNA virus.
- Lymph nodes are moderately enlarged (occipital, posterior auricular & posterior cervical lymph nodes), showing oedema & hyperplasia.
- I. P: 2-3 weeks.
- Congenital Rubella: fetal defects include retardation of growth, eye defects like cataract, glaucoma or retinopathy, heart lesions, deafness, mental retardation, hepatospleno megaly & skeletal abnormalities.
- Compared to measles, constitutional symptoms are mild in children.

**Mumps**
- Mumps virus; group: paramyxoviruses.
- Infection spreads through droplets.
- Man is the only known host.
- I. P: 18 days (2-3 week)
- Fever, Pain over the region of parotid gland, especially on opening the jaw, trismus, Sialoadenitis (usually bilateral) etc.
- Complications: in adult males (25%) orchitis, sterility if there is bilateral involvement, pancreatitis, meningitis (heralded by intense headache & meningeal irritation) etc.

**CLINICAL AND EPIDEMIOLOGIC FEATURES OF VIRAL HEPATITIS**

<table>
<thead>
<tr>
<th>Features</th>
<th>HAV</th>
<th>HBV</th>
<th>HCV</th>
<th>HDV</th>
<th>HEV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incubation (days)</td>
<td>15-45 RNA virus</td>
<td>30-180 DNA virus seen as Dane particles under electron microscope.</td>
<td>15-100 RNA virus</td>
<td>30-180 RNA virus; which is incomplete &amp; therefore it requires prior infection by HBV.</td>
<td>14-60 RNA virus transmitted mainly by the enteral route.</td>
</tr>
</tbody>
</table>

Transmission
**Fecal-oral**
- Unusual
- +++
- +++
- +++
- +++

**Percutaneous**
- None
- ---
- ---
- ---
- ---

**Perinatal**
- Occasionally severe
- 0.1-1%
- Occasional (1-10%)
- 0.1%
- Common (50-70%)

**Sexual**
- Common
- 50-70%
- 5-20%
- Occasional (1-10%)
- 1.5-3%
- Moderate

<table>
<thead>
<tr>
<th>Clinical Severity</th>
<th>Fulminent</th>
<th>Progression to Chronicity</th>
<th>Carrier Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>0.1%</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Occasionally severe</td>
<td>0.1-1%</td>
<td>Occasional (1-10%)</td>
<td>1-30%</td>
</tr>
<tr>
<td>Moderate</td>
<td>0.1%</td>
<td>Common</td>
<td>Worse with age</td>
</tr>
<tr>
<td>Occasionally severe</td>
<td>5-20%</td>
<td>Common</td>
<td>1.5-3%</td>
</tr>
<tr>
<td>Common</td>
<td>Variable</td>
<td>Acute good, Chronic poor</td>
<td>None</td>
</tr>
<tr>
<td>Mild</td>
<td>1.2%</td>
<td>Good</td>
<td>Good</td>
</tr>
</tbody>
</table>

**Serology of HBV**
- **Hbs Ag**: *live virus infection*; acute or chronic or carrier.
- **Anti Hbs Ag**: *immune*, cure & no active disease.
- **Anti Hbc Ag**:  
  - Ig M Anti Hbc Ag: *new infection.*
  - Ig G Anti Hbc Ag: *old infection.*
- **Hbe Ag**: *high infectivity*
- **Anti Hbe Ag**: low infectivity.

**ENTEROVIRUSES**

**POLIOMYELITIS** *(syn: Heine Medin Disease, Infantile paralysis)*
- Polio virus: Picornia viruses (group).
  - Three types: type1, 2 & 3; Type 1 is most common & responsible for the majority of paralytic cases.
- Disease is spread by the feco-oral route.
- In paralytic cases, *anterior horn cells* of the spinal cord & the medullary nuclei are swollen & congested. Classic paralytic polio develops only rarely.
- In the early stages, the *CSF shows increase in cells, mainly polymorphs* but this gives way to *lymphocytic increase with the passage of time*. Protein level: moderately increased. Sugar level: normal.
- **I. P:** 7-14 days.
- **Clinical features**: initial symptoms are headache, fever, muscle pain & diarrhea.
  - **Nonparalytic polio**: features of meningitis.
  - **Paralytic polio**: paralysis may involve spinal group of muscles, bulbar muscles or a combination of both.
  - **Spinal form**: predilection for the cervical & lumbar segments of the spinal cord. Paralysis is *asymmetrical*. If there is total destruction of neurons complete paralysis occurs.
  - **Bulbar form**: affection of cranial nerves occurs early in this form *dysphagia, dysarthria & dysphonias* occurs commonly. Facial palsy develops less commonly.
  - **Poliomencephalitis**: alteration in the level of consciousness, convulsions, signs of brainstem involvement & varying combinations of spastic or flaccid paralysis.
- **Post-polio syndrome (PPS)**: Progressive muscular atrophy & muscle weakness occurring several years after apparent recovery from poliomyelitis with residual paralysis. CSF shows elevation of IgM & interleukin-2 which point to an immunological basis for this syndrome.
- **Prevention**:
  - Live vaccine: Sabin (given orally)
  - Killed vaccine: Salk vaccine (subcutaneously)

**Regime for immunization** *(recommended by IMA)*

<table>
<thead>
<tr>
<th>Contact</th>
<th>Age of child</th>
<th>Vaccine</th>
<th>Other care</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>At birth</td>
<td>BCG, OPV</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>6 weeks</td>
<td>DPT, OPV</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>10 weeks</td>
<td>DPT, OPV</td>
<td></td>
</tr>
</tbody>
</table>
**ECHOVIRUSES (Syn: Enteric cytopathogenic human orphan viruses):**

RNA viruses which produce a spectrum of diseases ranging from mild upper respiratory tract infection, fever with rash to Acute Hemorrhagic Conjunctivitis (AHC).

- **Acute Hemorrhagic Conjunctivitis (AHC):**
  - Spinal form
  - Cranial form
  - Combined form

**COXSACKIE VIRUS INFECTION**

- Picorna viruses A & B.
- Syndrome of pleurodynia (devils grip, Borholm’s disease, epidemic myalgia) is caused by coxsackie B group viruses. Severe pain develops in chest & upper abdomen after a prodromal febrile illness.
- **Hand –Foot & Mouth disease:** caused by Coxsackie viruses types A-4, 5, 7, 9, 10 & 16. Febrile illness followed by superficial vesicular eruptions inside the mouth, fingers, palms & soles.

**RABIES (Hydrophobia, Lyssa)**

- **RNA viruses; rhabdoviruses.**
  - **Street viruses:** when isolated first from animals. It has long incubation periods & it can multiply in salivary glands.
  - **Fixed viruses:** it has shorter incubation periods & it can’t multiply in salivary glands.
- **Clinical features:**
  - **I. P: 10 days to more than 2 months.**
  - Neurological involvement leads to spastic & paralytic form.
  - **Spastic form:** Hydrophobia confirms the diagnosis (though not pathognomonic). Death can occur due to respiratory paralysis resulting from bulbar involvement. Whole course extends to less than 5 days.
  - **Paralytic form:** ascending paralysis. This occurs more often after vampire bat bites.
- Neutralizing antibodies against rabies virus can be demonstrated after the initial 10 days of onset.
- Virus can be isolated in saliva or CSF for the initial 2 weeks of illness.
- **Active immunization:**
  - Antirabies vaccination (ARV) introduced by Louis Pasteur. All vaccines are killed vaccines.
  - Avian embryo vaccine: these are produced in chick (Flury) or duck embryo. Neuroparalytic accidents are less common with this vaccine. Used for active immunization of dogs & pets.
- **Cell culture vaccines:**
  - Monkey kidney cell vaccine
  - Purified chick embryo cell vaccine
  - Human diploid cell vaccine (HDCV)
- Passive immunization: Human rabies immunoglobulin HRIG. If the risk is class 3 or even 2, passive immunization should be given.
- Pre-exposure immunization: 3 doses of cell culture vaccines on day 0, 7, 21 or 28 days; for 2 years.

**Classification of risk**

<table>
<thead>
<tr>
<th>Class 1 (light risk)</th>
<th>Class 2 (moderate risk)</th>
<th>Class 3 (great risk)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Licks except on face &amp; fingers</td>
<td>Licks on fresh wounds &amp; cuts &amp; abrasions on fingers</td>
<td>Licks on fresh cuts, scratches &amp; bits on head, neck or face.</td>
</tr>
</tbody>
</table>
Licks on intact mucous membrane
Light bites & scratches over parts of the body except head, neck, face & fingers
Consumption of unboiled milk or handling raw flesh of rabid animal

Scratches on fingers

Bites on fingers & all lacerated wounds

Jackal & wolf bites

Any class 2 patient who has not received RV within 2 weeks

ARBOVIRUSES

1. DENGUE FEVER (BREAK BONE FEVER)
   - Of all the arthropod-borne viral diseases, Dengue fever is the most common.
   - Group B arbovirus.
   - Main vector is Aedes aegypti mosquito.
   - Clinical features:
     - 3 types:
       1. Classical Dengue Fever
       2. Dengue Haemorrhagic fever without shock
       3. Dengue Haemorrhagic fever with shock
     - Incubation period 3 to 10 days.
     - The onset is sudden with chills and high fever, intense headache, muscle and joint or bone pains which prevent all movement.
     - Mottling, or fleeting pin point eruptions on face, neck, chest and limbs sparing palms and soles during the first half of the febrile period and a conspicuous rash that may be maculopapular or scarlatiform on 3rd or 4th day.
     - The fever may rise again producing a saddle-back fever curve.
     - Dengue haemorrhagic fever is confined exclusively to children less than 15 yrs of age. There may be plasma leakage and abnormal haemostasis, as manifested by a rising haematocrit value and moderate to marked thrombocytopenia.
     - In dengue shock syndrome shock is present along with all the above criteria.

SANDFLY FEVER (Syn: phlebotomous fever, Papatasii fever, 3 days fever)

- Transmitted by the sandfly phlebotomous papatasii (female)
- I.P: 2-6 Days
- Absence of rash & lymphadenopathy helps to differentiate it from dengue fever.
- Leucopenia.

YELLOW FEVER

- Group B arbovirus.
- Transmitted by aedes mosquitoes
- I.P: 3-6 Days
- It is a zoonotic disease affecting principally monkeys and other vertebrates.
- It shares clinical features of dengue fever but is characterized by more severe hepatic and renal involvement.
- 3 stages: first stage; most of death occur at this stage; second stage; third stage: intoxication follows within hours to days in severe cases.
- Vaccination: live attenuated vaccine (17D strain)

CHIKUNGUNYA (Chicken Guinea)

- Form of viral fever resembling dengue fever
- Chikungunya virus; Alphavirus (group A arbovirus).
- Transmitted by Aedes aegypti mosquito, though recent research by the Pasteur Institute in Paris claims the virus has suffered a mutation that enables it to be transmitted by Aedes Albopictus (Tiger mosquito).
- The name is derived from the word Makonde meaning "that which bends up" in reference to the stooped posture developed as a result of the arthritic symptoms of the disease
- Clinical features:
Incubation period 3-12 days.

- Sudden onset of flu-like symptoms including a severe headache, chills, fever (>40°C, 104°F), joint pain (especially on the 4th day), backache, nausea, vomiting, petechial or maculopapular rash usually involving the limbs and trunks. Migratory polyarthritis mainly affects the small joints of the hands, wrists, ankles and feet with lesser involvement of the larger joints. Joints of the extremities in particular become swollen and painful to touch.

- The disease has a biphasic course also. Following 1-6 days of fever, the temperature returns to normal for 1-3 days and then there is a second period of fever for a few days.

**JAPANESE ENCEPHALITIS (JE)**
- Group B arbovirus.
- Virus has been isolated from *culex* mosquitoes, *anopheles* mosquitoes and *mansonia*.
- **Clinical features**: The disease has 3 phases:
  - Prodromal
  - Acute encephalitic stage
  - Convalescence (recovery phase)
  - There may be fever, headache, and altered sensorium including coma, convulsion, and neck rigidity.

**KYASANUR FOREST DISEASE (KFD)**
- Group B arbovirus.
- Vectors: *ticks; Hemaphysalis spinigera & Ixodes*.
- Man gets the infection by the bite of the nymph of *H. spinigera*
- I. P: 2-7 days.
- Febrile illness characterized by fever with rigor, meningism, delirium, mental confusion etc.

**SYSTEMIC FUNGAL INFECTIONS**
- **Candidiasis**: *Candida albicans*; in genitalia it usually presents as vulvovaginitis in diabetic women.
- **Aspergillosis**: *A. fumigatus* is most common. Healed tuberculous cavities or other cavities may be the seat of colonization of the fungus.
- **Histoplasmosis** *(Darling’s disease or cave disease)*: *Histoplasma capsulatum*. Pulmonary lesions may resemble different types of tuberculosis.
- **Cryptococcus** *(Syn: Torulosis, European Blastomycosis, Busse- Bushke’s disease)*: *Cryptococcus neoformans*; often the lesions are mistaken for tuberculosis.
- **Rhinosporidiosis**: *Rhinosporidium seeberi*; affects the mucous membrane of the nose, larynx, eyes, ears, mouth, genitalia, rectum & skin.

**ACTINOMYCOSIS & NOCARDIA.**

**ACTINOMYCOSIS** *(Ray-fungus disease)*
- Actinomycetes are bacteria. They form mycelia like fungi.
- **Actinomyces Israeli**.
- Disease present as:
  - Cervicofacial form (extensive destructive lesions in the presence of minimal general symptoms).
  - Thoracic form (lesion resemble TB)
  - Abdominal form(disease may present as acute appendicitis)
  - Bony lesions (osteomyelitis)

**MYCETOMA** *(Madura foot, Maduramycosis)*
- Chronic granulomatous inflammation caused by several species of actinoimycetes & fungi.
- **Nocardia madurae, N. asteroides & N. brasiliensis.**

**PROTOZOAL DISEASES**

**MALARIA**
- Agent: protozoa of the genus plasmodium.
**Airport malaria**: occurrence of malaria in people living near airports or working in airports, caused by bites of infective mosquitoes arriving in aircrafts.

**Vectors**: female anopheline mosquitoes.

**Life cycle**: Sexual cycle (sporogony) in mosquitoes & schizogony (asexual cycle) in man.

---

### Characteristic features of human malarial parasite

**Colour of pigment**

1. *Plasmodium vivax* - yellow brown
2. *P. ovale* - dark brown
3. *P. malaria* - brown black
4. *P. falciparum* - black

**Number of merozoites per schizonts** (*trophozoite divides by binary fission to form schizont, which contains several merozoites*).

<table>
<thead>
<tr>
<th></th>
<th><em>P. vivax</em> (benign tertian malaria)</th>
<th><em>P. malariae</em> (quartan malaria)</th>
<th><em>P. falciparum</em> (malignant tertian)</th>
<th><em>P. ovale</em> (ovaie malaria)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incubation period</td>
<td>14 days</td>
<td>28 days (longest)</td>
<td>12 days (shortest)</td>
<td>17 days</td>
</tr>
<tr>
<td>Duration of erythrocytic cycle (schizogony)</td>
<td><strong>48 hours</strong></td>
<td><strong>72 hours</strong></td>
<td><strong>48 hours</strong></td>
<td><strong>48 hours</strong></td>
</tr>
<tr>
<td>Red cell preference</td>
<td>Reticulocytes</td>
<td>Older cells</td>
<td>Younger cells</td>
<td>Reticulocytes</td>
</tr>
<tr>
<td>Exoerythrocytic cycle of relapse</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Changes seen in infected RBC</td>
<td>Enlarged</td>
<td>Not enlarged</td>
<td>Not enlarged</td>
<td>Slightly enlarged</td>
</tr>
<tr>
<td>Dots</td>
<td><em>Schuffner’s dots</em></td>
<td><em>Ziemann’s dots</em></td>
<td><em>Maurers clefts</em></td>
<td><em>Schuffner’s dots</em></td>
</tr>
<tr>
<td>Gametocytes</td>
<td>Round or oval</td>
<td>Round or oval</td>
<td>Crescentic banana shape</td>
<td>Round or oval</td>
</tr>
<tr>
<td>Trophozoites</td>
<td>Amoeboid</td>
<td>Band shape</td>
<td>Compact</td>
<td>Compact</td>
</tr>
<tr>
<td>Forms in peripheral blood</td>
<td>All stages are present</td>
<td>--</td>
<td>Gametocytes and ring form of trophozoites</td>
<td>--</td>
</tr>
<tr>
<td>Hypnozoites (dormant sporozoite)</td>
<td>present</td>
<td>Absent</td>
<td>Absent</td>
<td>Present</td>
</tr>
</tbody>
</table>

---

### Clinical features of malaria

<table>
<thead>
<tr>
<th>Vivax malaria</th>
<th>Ovale tertian malaria</th>
<th>Falciparum malaria</th>
<th>Malariae malaria (quartan malaria)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paroxysms in 3 stages:</td>
<td>Similar to Vivax malaria.</td>
<td>Prodromal symptoms are severe than vivax malariea.</td>
<td>Fever occurs with quartan (every 72 hours) periodicity.</td>
</tr>
<tr>
<td>Cold stage</td>
<td></td>
<td></td>
<td>It may take several weeks to develop periodicity.</td>
</tr>
<tr>
<td>Hot stage</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sweat stage</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paroxysm repeats at interval of <strong>48 hours</strong>.</td>
<td></td>
<td>Periodicity may be either <strong>quotidian (daily)</strong> or <strong>sub tertian</strong> (less than 48 hours)</td>
<td></td>
</tr>
<tr>
<td>Patient feel well in between the febrile paroxysms</td>
<td></td>
<td>Patient may not feel well in between the febrile paroxysms.</td>
<td></td>
</tr>
</tbody>
</table>
Severe falciparum malaria (any one of the following clinical presentation)

1. cerebral malaria
2. Severe anaemia PCV <15%, Hb <5g/dL, parasitemia>10000/µL.
3. Renal failure urine output<480mL/24 hours in adults or 12mL/kg/24 hours in children &
creatinine >3mg/dL
4. Pulmonary oedema or respiratory distress syndrome
5. Hypoglycemia; whole blood glucose <40 mg/dL
6. Shock
7. Spontaneous haemorrhage
8. Repeated generalized seizures
9. Acidosis

Algid malaria: this occurs in patients with predominantly gastrointestinal manifestations. They become extremely dehydrated & shocked without any warning signal.

Black water fever (BWF): sudden intravascular hemolysis gives rise to fever, rigor, anemia & hemoglobinuria. If severe shock, jaundice & hemoglobinuria supervene.

Diagnosis

- Blood smears are taken at the onset of paroxysm. The general characteristics of plasmodia in Romonowsky- stained films are:
  - Location within erythrocytes
  - Red chromatin
  - Blue cytoplasm.
- Fluorochrome stains & quantitative buff coat (QBC) analysis.
- Fluorescence activated cell sorter (FACS) can detect paralyzed erythrocytes.
- Antigen detection tests like ELISA.
- PCR.

LEISHMANIASIS:

Pathogenic Leishmania are

7 Leishmania donovani causing Kala-azar (visceral leishmaniasis) and Post-Kala-azar dermal Leishmaniasis.
7 L.tropica and L.major causing Oriental sore- cutaneous form.
7 Several sub species of L.braziliensis and L.mexicana causing mucocutaneous Leishmaniasis.

KALA-AZAR: (Visceral Leishmaniasis)

- The name Kala-azar (black fever) is derived from the dark pigmentation that develops in the victims of visceral Leishmaniasis.
- Males are affected more than females.
- Main vector sand fly- Phlebotomus argentipes (females).
- Kupffer cells are distended with LD (Leishman-Donovan) bodies.
- Cell-mediated immunity is impaired.
- I.P: 2-6 months
- Periods of pyrexia and apyrexia alternate for several months.
- Occasional patient may show two peaks of fever in 24 hours. This double rise is very suggestive of Kala-azar.
- Despite the fever patient feels well and has good appetite.
- Progressive emaciation, anaemia and hepatosplenomegaly.
- Napier’s aldehyde test is a simple bedside test helpful in the diagnosis of chronic kala azar.
- Skin test- Leishmanin test of Montenegro has been used to elicit delayed hypersensitivity. Test is negative in active kala azar. The test is positive in cutaneous leishmaniasis.

POST-KALA-AZAR DERMAL LEISHMANIASIS (Syn: PKDL- Dermal Leishmanoid of Brahmachari)

- Chronic granuloma of the skin caused by Leishmania donovani following recovery from kala azar.
- Clinical features: Hypopigmented macules & nodular lesions.
- Aldehyde test is usually negative.
CUTANEOUS LEISHMANIASIS (Syn: Oriental sore, Delhi boil, Baghdad boil, Chiclero’s ulcer)

- Primary infection of the skin caused by direct inoculation of the parasites at the site of lesions by the appropriate vector sandflies.
- Vector: female sandflies; P. papatasii.

AMERICAN CUTANEOUS & MUCOCUTANEOUS LEISHMANIASIS (Syn: Espundiasis)

- Cutaneous lesion resemble s Oriental sore.
- Mucocutaneous form (espundia): the initial ulcer is followed by direct extension or metastatic spread from a distant lesion to the mucosa of nose & pharynx.

TRYPANOSOMIASIS

AFRICAN TRYPANOSOMIASIS (Syn: Sleeping sickness)

- Cause by 2 subspecies of Trypanosoma Brucei, T. gambiense (Gambien sleeping sickness) & T. rhodesiense (Rhodesian sleeping sickness).
- Transmitted by tsetse flies (Glossina).
- I. P: 1-3 weeks.
- The initial lesion is the formation of a nodule at the site of bite: trypanosomal chancre.
- Lymph nodes are more pronounced in the posterior triangle of neck in T. gambiense infection. Enlargement of these groups of lymph nodes are called Winterbottom’s sign.
- Unusual manifestations: circinate erythematous skin rashes, localized oedema over face, eyelids & neck, ulnar hyperaesthesia (kernadel’s sign) neuralgic pains, myocarditis, pericardial effusion, jaundice etc.
- CNS symptoms are more marked in T. b. gambiense.

AMERICAN TRYPANOSOMIASIS (Syn: Chagas Disease)

- Trypanosoma cruzi (schizo-trypanum).
- Disease is transmitted by Reduvid bugs.
- Infected mothers can transmit the disease to the fetus, giving rise to congenital Chagas disease.
- In children disease occurs in an acute form presenting with chagoma, generalized lymphadenopathy, fever, myocarditis etc.
- Initial lesion presenting with unilateral oedema of the conjunctiva & adjacent tissues is called Romana’s sign.

OTHER PROTOZOAL INFECTIONS

AMEBIASIS

- Entameba histolytica; it exists in 2 forms- the cyst & trophozoite.
- Clinical manifestation:
  - Intestinal amoebiasis
    - Acute amoebic dysentry
    - Non dysenteric amoebiasis: an amoebic granuloma (amoeboma) may be felt as a sausage shaped mass in the right iliac fossa.
  - Extra-intestinal amoebiasis:
    - Hepatic amoebiasis: almost all patients give a history of alcoholism.

PRIMARY AMEBIC MENINGOENCEPHALITIS

- Naegleria gruberi; free living soil amebae. Hartmanella (Syn: Acanthamoeba) can also affect man.
- Organisms enter through the roof of nose & spread up the cribriform plate to reach the subarachnoid space.
GIARDIASIS
- *Giardia intestinalis* (G. Lamblia)
- Presenting symptoms range from mild abdominal discomfort to explosive diarrhea.

BALANTIDIASIS
- *Balantidium coli*; large ciliate.
- These resemble mild to moderately severe amoebic dysentery.

TOXOPLASMOSIS
- *Toxoplasma gondii*.
- Full life cycle in cats & other canine hosts.
- Congenital toxoplasmosis: neurological involvement.
- Hepatosplenomegaly, thrombocytopenic purpura, lymphadenopathy & rashes have been described.
- **Toxoplasmin skin test**: intradermal test using toxoplasma antigen.

CRYPTOSPORIDIOSIS
- *Cryptosporidium parvum*.
- Acute watery diarrhea with fever & malaise.
- Cryptosporidiosis is a common opportunistic infection in AIDS.

INTESTINAL NEMATODES

ASCARIASIS
- Ascaris lumbricoides.
- During the stage of larval migration, pulmonary symptoms like cough, wheezing & hemoptysis may occur: **Loeffler’s syndrome**; common cause of respiratory symptoms in children.

HOOKWORM INFECTION (Ancylostomiasis)
- Ancylostoma duodenale & Necator americanus.
- During the stage of larval migration, allergic symptoms like cough, dyspnoea, eosinophilia & hemoptysis may occur: **Loeffler’s syndrome**.

TRICHURIASIS (Whipworm infection)
- Trichuris trichuria.

STRONGYLOIDIASIS
- Strongyloides stercoralis.
- Malabsorption syndrome or chronic diarrhea resembling Crohn’s disease.

ENTEROBIASIS (Oxyuriasis, Thread worm, Pinworm, Seatworm)
- Enterobius vermicularis (Oxyuris vermicularis)

TRICHINOSIS (Trichiniasis, Trichnellosis)
- *Trichinella spiralis*; parasite of several animals like pig, rat, polar bear etc.
- Intercostals muscles, pectoral muscles, diaphragm & shoulder girdle muscles are heavily affected.
- Eosinophilia is the constant finding in early stages.

LARVA MIGRANS
- **Visceral Larva Migrans**: manifestation produced by larvae of non-human ascarides when they migrate through the human tissues. Most often ascarides of dogs & cats- *Toxocara canis* & *Toxocara cats*. It is self limiting, since the encysted larvae die out after varying periods.
- **Cutaneous Larva Migrans**: larvae of *Ancylostoma braziliense* & *A. canicul* (dog’s & cat’s hookworms) & Strongyloides stercoralis cause creeping eruption.
CESTODIASIS (Syn: Tapeworm infection)

TAENIASIS SAGINATA (Syn: Beef Tapeworm, Unarmed Tapeworm)
- Taenia saginata; commonest among large tapeworm found in man.
- Man is the definitive host.
- Cystic stage: Cysticercus bovis.
- T. saginata segment has more than 15 lateral branches.

TAENIASIS SOLIUM (Syn: Pork Tapeworm, Armed Tapeworm)
- Taenia solium; adult inhabits the small intestine of man.
- The name solium is derived from the shape of the rostellum which resembles the conventional figure of the sun.
- Man is the definitive host & pig is the intermediate host.
- Cystic stage: Cysticercus cellulosae (bladder worm).
- Heavily infected pork: measly pork.

ECHINOCOCCOSIS (Syn: Dog Tapeworm, Hydatid Worm)
- Larval form of Echinococcus granulosus; less commonly E. multilocularis.
- Definitive hosts are dogs, wolves, jackals & other canines.
- Hydatid thrill: place 3 fingers of left hand firmly over enlarged liver & percuss on the middle finger with the right hand & feel the sensation over the other 2 fingers, produced by the free floating daughter cysts in the cyst cavity
- Casoni’s reaction: this test is suggestive but not specific.

DIPHYLLOBOTHRIASIS LATUM (Syn: Dibothriocephalus latum)
- Two intermediate hosts are required: fresh water cylops and fresh water fish.

HYMENOLEPIS NANA (Syn: Dwarf Tapeworm)
- Smallest tapeworm found in man

HYMENOLEPIS DIMENUTA
- Parasite primarily affecting rats and mice.

DIPYLIDIUM CANINUM
- Tapeworm seen in cats and dogs

TREMATODE (FLUKE) INFECTIONS

FASCIOLIASIS (Syn: Sheep Liver fluke)
- Fasciola hepatica.
- Snail vector: L.limnaea truncatula. The cercariae encyst on aquatic vegetation
- Man gets the infection by eating contaminated watercress.

HETEROPHYIASIS
- Heterophyes: a small fluke found in small intestine of man, dog, cat & wolf.
- They resemble Clonorchis sinensis.
- Snail vector; Pironella conica.

CLONORCHIASIS (Syn: Chinese liver fluke)
- Infection of the biliary passages by clonorchis sinensis causes clonorchiasis.
- In India it has been reported among the Chinese immigrants.

SCHISTOSOMIASIS (Syn: Bilharziasis)
- Infection by Schistosoma haematobium, S. mansoni (both parasitize only man) or S. japonicum ( affect man & other animals)
- Schistosoma haematobium reaches the bladder & other pelvic organs. The eggs appear in urine.
Schistosoma japonicum affects the small intestine & the ascending colon.
Schistosoma mansoni lodges in the descending colon & rectum.
The eggs of Schistosoma japonicum & Schistosoma mansoni are passed in feces.

**Clinical features:**
- **Swimmer’s itch or cercarial dermatitis:** itching & popular rashes at the site of penetration.
- **Katayama syndrome:** marked eosinophilia develops in about 4-6 weeks (2nd stage).
- **Late complications:**
  - Schistosoma haematobium (Genitourinary schistosomiasis)
  - Schistosoma mansoni (intestinal schistosomiasis)
  - Schistosoma japonicum (Katayama disease, Asiatic schistosomiasis): its pathogenicity is greater since it produce more eggs.

**TISSUE NEMATODES**

**LYMPHATIC FILARIOSES**

- Caused by *Wuchereria bancrofti* & *Brugia malayi*.
- Adult worms living in the lymphatic vessels are responsible for the lesion. Males are smaller than females.
- Microfilaria (embryonated eggs) is numerous in the peripheral blood at night.
- Vectors: many species of Culex & some species of Anopheles mosquitoes.
- Commonest clinical presentation is lymphoedema involving the extremities or genitalia & at the other end of the clinical spectrum tropical pulmonary oesinophilia (TPE) syndrome.
- **Spectrum of disease in lymphatic filariasis:**
  - Endemic normals
  - Asymptomatic microfilaraemia
  - Clinical disease
  - TPE syndrome.

**ICT (Immunochromatographic test):** both card & ELISA based ICT are highly specific & sensitive for the diagnosis.

**Manifestations of lymphatic filariasis**

<table>
<thead>
<tr>
<th>Early phase</th>
<th>silent phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinically asymptomatic. Microfilaria present peripheral blood. Dilatation of lymphatics demonstrated by ultrasonography &amp; lymphoscintigraphy.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Acute manifestations</th>
<th>Chronic manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute adenolymphangitis Acute epididymo-orchitis, funiculitis Acute onset hydrocele due to inflammation Abscess formation Acute abdominal lymphadenitis Haematuria</td>
<td>Lymphoedema/ elephantiasis of extremities, genitalia &amp; breasts Hydrocele Lymph scrotum Chyluria (sometimes pyuria with disintegration of the cells may appear milky: pseudochylous urine), chylecele, chylous ascitis. Lymph node enlargement Lymphadenof-varix.</td>
</tr>
</tbody>
</table>

**LOIASIS**

- *Loa loa*; adult worm migrate in the subcutaneous tissues of various parts including the eyes.
- Infection is transmitted by Chrysops (*C. silacea & C. dimidiata*).
- Disease manifests as Calabar swellings which are recurrent localized allergic inflammatory swellings.
- Diagnosis confirmed by demonstration of microfilaria in peripheral blood during day time.

**ONCHOCERCIASIS (Syn: African river blindness)**

- Cutaneous form of filariasis.
- Onchocerca volvulus.
Vectors: female black flies; genus simulium.
Firm, freely movable subcutaneous nodules commonly seen around head & shoulders or the pelvic girdle.
Intense itchy lesions. Later skin becomes thickened & hyperkeratotic (Crocodile skin).
Ocular lesions ultimately lead to blindness.

OTHER FILARIAL INFECTIONS

- Dipetalonema perstans: adults are seen in subserosal layer of viscera. Vectors: blood sucking insects-culicoides.
- Dipetalonema streptocerca: Vectors: blood sucking insects-culicoides. Adult worms are seen in dermis & subcutaneous tissues.
- Mansonella ozzardi: adult worms are found in the visceral adipose tissue. Vectors include simulium & culicoides.

DRACONTIASIS (Syn: Guinea worm, Serpent worm, Dragon worm)

- Dracunculus medinensis.
- Intermediate host: fresh water Cyclops.
- Urticaria, eosinophilia, nausea, vomiting, diarrhea etc.
- Ulcer may develop & it may act as a portal of entry for clostridium tetani.

RARE HELMINTHIC INFESTATIONS

- Multiceps Multiceps: this is a tapeworm, adult of which live in the intestines of dog. Larvae (bladder worm) are seen in the brain of sheep & other herbivores- Coenurus cerebralis.
- Intestinal capillariasis: the adults (Capillaria philippinensis) parasitize birds.
- Anisakiasis: this is caused by Anisakis species which are intestinal nematodes of marine animals.
- Angiostrongyliasis: infection by Angiostrongylus costaricensis or Angiostrongylus cantonensis. Man gets infected by eating molluscus, prawns or crabs which harbors the larvae.
- Gnathostoma spinigerum: intestinal nematode affecting dogs & cats.
- Sparganosis: Sparganum is the second larval stage of tapeworm of the genera Spirometra & Diphyllobothria. Ova are ingested by Cyclops & the 1st stage larvae (procercoids) develop in them.

CLASSICAL PRESENTATIONS OF PARASITIC DISEASES

<table>
<thead>
<tr>
<th>Presentation</th>
<th>Parasitic Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>“Anchovy sauce” expectoration</td>
<td>Amoebic abscess</td>
</tr>
<tr>
<td>“Grape skin” expectoration</td>
<td>Hydatid disease</td>
</tr>
<tr>
<td>Duodenal ulcer type pain</td>
<td>Hookworm, Strongyloides stercoralis</td>
</tr>
<tr>
<td>Intestinal obstruction</td>
<td>Round worm</td>
</tr>
<tr>
<td>Rectal prolapse</td>
<td>Whip worm, Trichostrongyulus</td>
</tr>
<tr>
<td>Pruritus ani</td>
<td>Pinworm, Thread worm</td>
</tr>
<tr>
<td>Lymphoedema, elephantiasis</td>
<td>Filarisis</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
<td>Schistostoma mansoni</td>
</tr>
<tr>
<td>Portal hypertension</td>
<td>Schistosoma Japonicum</td>
</tr>
<tr>
<td>Terminal haematuria</td>
<td>Schistosoma haematobium</td>
</tr>
<tr>
<td>Haemoptysis</td>
<td>Paragonomiasis</td>
</tr>
<tr>
<td>Cholangitis, pancreatitis</td>
<td>Clonorchis sinensis</td>
</tr>
<tr>
<td>Cholangiocarcinoma</td>
<td>Chagas disease</td>
</tr>
<tr>
<td>Megaoesophagus/-colon</td>
<td>Giardiasis</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>D. latum</td>
</tr>
<tr>
<td>Malabsorption</td>
<td>Hookworm</td>
</tr>
<tr>
<td>Vit. B12 deficiency</td>
<td></td>
</tr>
<tr>
<td>Iron-deficiency anaemia</td>
<td></td>
</tr>
</tbody>
</table>

DISEASES CAUSING AGENTS

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>AGENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rubella</td>
<td>Rubella virus (a togavirus)</td>
</tr>
<tr>
<td>Mumps</td>
<td>Mumps virus (a paramyxovirus)</td>
</tr>
<tr>
<td>Yellow fever</td>
<td>A togavirus</td>
</tr>
<tr>
<td>Dengue fever</td>
<td>A togavirus</td>
</tr>
<tr>
<td>Trachoma</td>
<td>Chlamydia trachomatis</td>
</tr>
<tr>
<td>Disease</td>
<td>Organism/Pathogen</td>
</tr>
<tr>
<td>-------------------------</td>
<td>--------------------------------------------------------</td>
</tr>
<tr>
<td>Q-fever</td>
<td>Coxilla burnetti (Rickettsia like organism)</td>
</tr>
<tr>
<td>Diphtheria</td>
<td>Corynebacterium diphtheriae</td>
</tr>
<tr>
<td>Anthrax</td>
<td>Bacillus anthracis</td>
</tr>
<tr>
<td>Bacillary dysentery</td>
<td>Shigella dysenteriae, flexneri, sonnei</td>
</tr>
<tr>
<td>Tularaemia</td>
<td>Francisella tularensis (transmitted by flies and ticks)</td>
</tr>
<tr>
<td>Oroya fever</td>
<td>Bartonella bacilliformis (transmitted by sand flies)</td>
</tr>
<tr>
<td>Brucellosis (undulant fever)</td>
<td>Brucella melitensis</td>
</tr>
<tr>
<td>Glanders</td>
<td>Pseudomonas mallei</td>
</tr>
<tr>
<td>Syphilis</td>
<td>Treponema pallidum</td>
</tr>
<tr>
<td>Gonorrhoea, ophthalmia neonatorum</td>
<td>Neisseria gonorrhoeae</td>
</tr>
<tr>
<td>Granuloma inguinale (Donovanosis)</td>
<td>Calymmatobacterium granulomatis</td>
</tr>
<tr>
<td>Chancroid</td>
<td>Haemophilus ducreyi</td>
</tr>
<tr>
<td>Lymphogranuloma venerum</td>
<td>Chlamydia trachomatis (L, 1, 2, 3 serotypes)</td>
</tr>
<tr>
<td>Nonspecific urethritis, proctitis, cervicitis</td>
<td>Chlamydia trachomatis</td>
</tr>
<tr>
<td></td>
<td>Mycoplasma hominis</td>
</tr>
<tr>
<td></td>
<td>Ureaplasma Urealyticum</td>
</tr>
<tr>
<td></td>
<td>Trichomonas vaginalis</td>
</tr>
<tr>
<td></td>
<td>HSV</td>
</tr>
<tr>
<td>Acute pelvic inflammatory disease</td>
<td>N.gonorrhea</td>
</tr>
<tr>
<td></td>
<td>Chlamydia trachomatis</td>
</tr>
<tr>
<td>AIDS</td>
<td>HIV 1 &amp; 2</td>
</tr>
<tr>
<td>Molluscum contagiosum</td>
<td>Pox-like virus</td>
</tr>
<tr>
<td>Genital warts</td>
<td>Pailloma virus</td>
</tr>
<tr>
<td>Hepatitis</td>
<td>HBV, HCV, CMV</td>
</tr>
<tr>
<td>Vaginal thrush, balanitis</td>
<td>Candida albicans</td>
</tr>
<tr>
<td>Vaginitis, urethritis, balanoposthitis, (protozoal)</td>
<td>Trichomonas vaginalis</td>
</tr>
<tr>
<td>Genital scabies</td>
<td>Sarcoptes scabei</td>
</tr>
<tr>
<td>Pediculosis pubis</td>
<td>Phthirus pubis</td>
</tr>
<tr>
<td>Syphilis</td>
<td>Treponema pallidum</td>
</tr>
<tr>
<td>Bejel</td>
<td>Treponema pallidum variant</td>
</tr>
<tr>
<td>Pinta</td>
<td>Treponema carateum</td>
</tr>
<tr>
<td>Yaws</td>
<td>Treponema petenue</td>
</tr>
<tr>
<td>Canicola fever</td>
<td>Leptospira canicola</td>
</tr>
<tr>
<td>Weil’s disease</td>
<td>Leptospira icterohaemorrhagica</td>
</tr>
<tr>
<td>Louse-borne relapsing fever</td>
<td>Borrelia recurrentis</td>
</tr>
<tr>
<td>Tick-borne relapsing fever</td>
<td>Borrelia duttoni</td>
</tr>
<tr>
<td>Cancrum oris</td>
<td>Borrelli viventi</td>
</tr>
<tr>
<td>Lyme disease</td>
<td>Borrelia burgdorfer</td>
</tr>
<tr>
<td>Psittacosis (ornithosis, a zoonosis)</td>
<td>C. psittaci</td>
</tr>
<tr>
<td>Kala-azar</td>
<td>L.donovani (transmitted by female sand flies)</td>
</tr>
<tr>
<td>Espundia (mucocutaneous leishmaniasis)</td>
<td>L.braziliensis</td>
</tr>
<tr>
<td>Diffuse cutaneous leishmaniasis</td>
<td>L.amazonensis</td>
</tr>
<tr>
<td>Sleeping sickness</td>
<td>T.brucel (bite of tsetse fly of either sex)</td>
</tr>
<tr>
<td>Chagas disease</td>
<td>T.cruzi (transmitted by faeces of a reduvid bug)</td>
</tr>
<tr>
<td>Toxoplasmosis</td>
<td>Toxoplasma gondii</td>
</tr>
<tr>
<td>Tertian malaria</td>
<td>P.vivax and P.ovale</td>
</tr>
<tr>
<td>Quartan malaria</td>
<td>P.malariae</td>
</tr>
<tr>
<td>Malignant tertian malaria</td>
<td>P.falciparum</td>
</tr>
<tr>
<td>Cerebral malaria</td>
<td></td>
</tr>
<tr>
<td>Algid malaria</td>
<td></td>
</tr>
<tr>
<td>Septicaemic malaria</td>
<td></td>
</tr>
<tr>
<td>Blackwater fever</td>
<td></td>
</tr>
<tr>
<td>Hydatid disease</td>
<td>Echinococcus granulosus and multilocularis</td>
</tr>
<tr>
<td>scabies</td>
<td>Sarcoptes scabei (a mite)</td>
</tr>
<tr>
<td>Tinea versicolor</td>
<td>Malassezia furfur</td>
</tr>
<tr>
<td>Meningitis 2 months to 2 years</td>
<td>H. influenza</td>
</tr>
<tr>
<td>Meningitis after 12 years</td>
<td>Meningococcus</td>
</tr>
<tr>
<td>Acne</td>
<td>Corynebacterium acnes</td>
</tr>
</tbody>
</table>
Sodoku
Favus
Fungus ball in lung cavity (aspergillosis)
Multiple cavities in lung (blastomycosis)
Sinusitis

**ARTHROPOD BORNE DISEASES**

<table>
<thead>
<tr>
<th>Name of arthropod</th>
<th>Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>House fly, Musca</td>
<td>Dysenteries, Salmonelloses, Enteric fevers, Cholera</td>
</tr>
<tr>
<td>Tebanide</td>
<td>Tularaemia, Anthrax</td>
</tr>
<tr>
<td>Tsetse fly, Glossina</td>
<td>African trepanosomiasis</td>
</tr>
<tr>
<td>Black fly, Simulium</td>
<td>Onchocerciasis</td>
</tr>
<tr>
<td>Sand fly, Phlebotomus</td>
<td>Leishmaniasis, Bartonellosis</td>
</tr>
<tr>
<td>Mosquito, Anopheles</td>
<td>Malaria, some arboviruses</td>
</tr>
<tr>
<td>Culex</td>
<td>Bancroftian and Brugian filariasis</td>
</tr>
<tr>
<td>Aedes</td>
<td>Yellow fever, dengue, Chickengunya, other arboviruses</td>
</tr>
<tr>
<td>Soft tick, Ornithodoros</td>
<td>Lyme disease, Tick-borne relapsing fever</td>
</tr>
<tr>
<td>Hard tick, Ixodidae</td>
<td>Some typhus fevers, Kyasanur forest disease, Tularaemia</td>
</tr>
<tr>
<td>Fleas, Xenopsylla</td>
<td>Endemic typhus, Plague</td>
</tr>
<tr>
<td>Mites, Leptotrombidium</td>
<td>Scrub typhus</td>
</tr>
<tr>
<td>Alloptrombicidus</td>
<td>Rickettsial pox</td>
</tr>
<tr>
<td>Lice, Pediculus</td>
<td>Epidemic typhus, louse-borne relapsing fever, trench fever, Diphylidium caninum</td>
</tr>
<tr>
<td>Winged bug, Triatoma</td>
<td>Chagas disease</td>
</tr>
</tbody>
</table>

**Arthropods**

<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>Malaria</td>
<td>Japanese encephalitis</td>
<td>West Nile fever</td>
<td>Bancroftian filariasis</td>
<td>Viral arthritis</td>
</tr>
<tr>
<td>1. Mosquito</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Sand fly</td>
<td>Kala-azar</td>
<td>Oriental sore</td>
<td>Oroya fever</td>
<td>Sandfly fever</td>
</tr>
<tr>
<td>3. Tse-tse fly</td>
<td>Sleeping sickness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Louse</td>
<td>Epidemic typhus, relapsing fever, trench fever, pediculosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Rat flea</td>
<td>Bubonic plague, endemic plague, chiggerosis, hymenolepis diminuta</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Black fly</td>
<td>Onchocerciasis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Reduvid bug</td>
<td>Chaga’s disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Hard tick</td>
<td>Tick typhus, viral encephalitis, viral haem, Fever KED, Turaleium, tick paralysis, human babesiosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Soft tick</td>
<td>Q fever, relapsing fever</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Trombiculid mite</td>
<td>Scrub typhus, Rickettsial pox</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Itch mite</td>
<td>Scabies</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Cyclops</td>
<td>Guinea warm disease, Fish tape worm(D, Latum)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**INCUBATION PERIODS**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Incubation period</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Short-incubation</strong> (&lt; 1 week)</td>
<td>Cholera</td>
</tr>
<tr>
<td></td>
<td>Scarlet fever</td>
</tr>
<tr>
<td></td>
<td>Bacillary dysentery</td>
</tr>
</tbody>
</table>
### Anthrax
2-5 days

### Diphtheria
3-4 days

### Gonorrhoea
3-4 days

### Meningococcaemia
3-4 days

<table>
<thead>
<tr>
<th><strong>Intermediate incubation</strong></th>
<th>(1-3 weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whooping cough</td>
<td>7-10 days</td>
</tr>
<tr>
<td>Poliomyelitis</td>
<td>7-14 days</td>
</tr>
<tr>
<td>Typhus fever</td>
<td>7-21 days</td>
</tr>
<tr>
<td>Lassa fever</td>
<td>14-21 days</td>
</tr>
<tr>
<td>Measles</td>
<td></td>
</tr>
<tr>
<td>Typhoid</td>
<td></td>
</tr>
<tr>
<td>Mumps</td>
<td></td>
</tr>
<tr>
<td>Chickenpox</td>
<td></td>
</tr>
<tr>
<td>Rubella</td>
<td></td>
</tr>
<tr>
<td>Trypanosoma</td>
<td></td>
</tr>
<tr>
<td>Rhodesiense</td>
<td></td>
</tr>
<tr>
<td>Malaria</td>
<td></td>
</tr>
<tr>
<td>Amoebiasis</td>
<td>1 week- months</td>
</tr>
<tr>
<td>Rubella</td>
<td>2 weeks- months</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Long Incubation period</strong></th>
<th>(&gt; 3 weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brucellosis</td>
<td>Days- months</td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>2-6 weeks</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>6 weeks- 6 months</td>
</tr>
<tr>
<td>Leprosy</td>
<td>2-5 years</td>
</tr>
<tr>
<td>Rabies</td>
<td>Variable (2-8 weeks)</td>
</tr>
</tbody>
</table>

### DAY OF APPEARING RASH AFTER FEVER

<table>
<thead>
<tr>
<th>1st</th>
<th>2nd</th>
<th>3rd</th>
<th>4th</th>
<th>5th</th>
<th>6th</th>
<th>7th</th>
</tr>
</thead>
<tbody>
<tr>
<td>Varicella, Rubella</td>
<td>Scarlet fever</td>
<td>Small pox</td>
<td>Measles</td>
<td>Typhus</td>
<td>Dengue</td>
<td>Typhoid</td>
</tr>
<tr>
<td>Very</td>
<td>Sick</td>
<td>Person</td>
<td>Must</td>
<td>Take</td>
<td>Double</td>
<td>Treatment</td>
</tr>
</tbody>
</table>

### PERIOD OF INFECTIVITY

<table>
<thead>
<tr>
<th>Disease</th>
<th>Period of infectivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measles</td>
<td>From prodrome until 4 days after onset of rash</td>
</tr>
<tr>
<td>Mumps</td>
<td>3 days pre-parotitis until 1 week after.</td>
</tr>
<tr>
<td>Chickenpox, rubella</td>
<td>One week before onset of rash &amp; 1 week after last crop of rash.</td>
</tr>
<tr>
<td>Diphtheria, scarlet fever</td>
<td>From onset to 3 weeks after.</td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>Prior to icteric phase.</td>
</tr>
<tr>
<td>Whooping cough</td>
<td>7 days after exposure to weeks after onset of symptoms.</td>
</tr>
</tbody>
</table>

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