

Infectious diseases:

Leptospirosis: Most common zoonotic disease.

Microbiology: ^{Epidemiology} ^{biology}

- caused by the genus Leptospira, Family Leptospiraceae and the order Spirochaetales.
- highly coiled thread-like organism.
- More than 250 serological variants.
- Leptospira interrogans is pathogenic in humans.
- It affects predominantly the wild and domestic animals. Especially rodents (rats).

- It is present in the convoluted tubules of the kidney and passed through urine and the host does not usually get affected.
- It then enters their human host through skin and mucous membrane facilitated by cuts & abrasions.
- Prolonged immersion in contaminated water with fever invasion as the spirochetes live well in water.

Clinical features

- Incubation period - 1-3 weeks.
- Affects mainly kidney, liver, meninges & brain.

Four main clinical syndrome.

i) Bacteraemic leptospirosis.

- Fever, weakness, muscle pain (calf & back).
- Photophobia, headache, Diarrhoea, vomiting.
- Conjunctival congestion is the only sign.
- Persists for week, then other infection.

ii) Aseptic meningitis.

- Difficult to differentiate from viral meningitis.
- Conjunctival congestion is the only DD point.

- ii) Weill disease (Septic leptospirosis),
- It develops during the second (immune phase of leptospirosis).
 - It is a dramatic life threatening event characterized by i) Jaundice ii) Haemorrhage iii) Acute kidney injury iv) Fever.
 - Jaundice due to sepsis on the cholestasis not due to hepatocellular cause.
 - Renal failure - Due to hypotension, oliguria/anuria with presence of albumin, blood and casts.
 - Haemorrhage manifest like petechiae, purpura, on mucosal areas, epistaxis, hemoptysis, haematemesis, malna, then, hemorrhage into cavity like pleural, pericardial, etc. on subarachnoid space.

iv) Pulmonary syndrome

- cough & hemoptysis - symptoms of ARDS.
- X-ray shows multifocal effusion.

Diagnosis:

- CBC $\rightarrow$ Polymorphonuclear leucocytosis, thrombocytopenia.
- Creatine kinase, $\uparrow$ ESR.
- Urine: proteinuria, casts and sediment.
- Blood culture $\rightarrow$ Before 10 days.
- IgM ELISA.

Management

- Blood transfusion, fluid balance, dialysis.
- Antibiotics.

Prevention - Preventing water from contamination.

Plague

- Caused by Yersinia pestis: Gram negative bacillus.
- spread between rats by their fleas, safety pin
- In domestic the fleas bite the human. apparent
- Hurtles and trapping by handling, and by droplets in the later stage of infection (pneumonic plague).
- Epidemics of plague. Black death has occurred.
- Bio measures due to high death rate.

Clinical features

Pathology: through skin by bite of the infected flea and produce papule and infects the lymph nodes $\rightarrow$ Bubo, proliferate and enter the blood stream $\rightarrow$ Septicemic. Droplet infect $\rightarrow$ Pneumonic.

i) Bubonic plague

- Sudden onset. Fever, chills, Aches, swelling of lymph nodes especially of the groin called Bubo.
- causes toemia, RT pale, & BP & delirium.

ii) Septicemic plague

- If bubo is exhibited the patient's death is rapid.
- Disseminated intravascular coagulation occurs and shows petechiae, purpur-like petechiae.
- Metastasis to distant organs.

iii) Pneumonic plague

- Infection in lung $\rightarrow$ cough, dyspnea, hemoptysis.
- highly infectious sputum, ARDS.
- X-ray shows Bilateral infiltrates.

Investigation

- $\rightarrow$ Blood, sputum, Bubo aspirates.
- culture $\rightarrow$ safety pin apparatus. coicobacilli.

Management

- Treatment for DIC, circulatory failure
- Rats and fleas should be controlled, wear masks

Enteric fever

- Typhoid and Paratyphoid fevers.
- Causation $\rightarrow$ *Salmonella typhi* and *Paratyphi* A & B.

Route of Transmission $\rightarrow$ Faeco oral route, through contaminated food & water.

- Epidemiology $\rightarrow$ Indian Subcontinent countries.

Pathogenesis localization in

- After few days, lymphoid tissue of the intestine resulting in lesions in Peyer's patches & follicles.

- They swell and ulcerate and heal.

- After recovery, patient becomes chronic.

carrier, the bacilli live in gall bladder and passed intermittently in stool and urine.

Clinical features

1) Typhoid fever

Incubation - 10-14 days

First week $\rightarrow$ Fever, $\rightarrow$ 1st ep. Leukopenia, malaise.

Relative bradycardia, Constipation, Diarrhea & vomiting in children.

At end of 1st week $\rightarrow$ Rose spots, Splenomegaly.

Cory, Abdominal distension, Diarrhea.

End of 2nd week $\rightarrow$ Delirium, Complications & death.

2) Paratyphoid

- Milder form, abrupt with acute onset.

$\rightarrow$ Rare are abundant with ^{no} internal complications.

Investigations: Complication:
 Bowel → Hemorrhage, Pyrexia.
 Septic foci → cholecystitis, Bone & Joint infect, Meningitis,
 Toxic phenomena → Myocarditis, Nephritis,
 Chronic carrier → Persistent gall bladder carrier.

Investigations:
 - First week → Diagnosis difficult.
 - Second week → stool culture positive.
 - Widal Test → O & H Antigen but is not specific.

Management:

- Improved sanitation, Typhoid vaccine.
- Antibiotic.
- TAB vaccine (Typhoid - Paratyphoid A & B vaccine).

ESCHERICHIA COLI INFECTION

- E. coli belongs to group enterobacteria.
 - It is present as normal intestinal flora.
 - Gram negative motile rod. Three antigen O, H, K.
- Clinical features:
- i) Commensal strain
 - ii) Intestinal pathogenic strain
 - iii) Extraintestinal pathogenic strain

i) Commensal strain:

- Normally present, produce infection in UT, by catheter, obstruction and Immune suppressed.

ii) Intestinal pathogenic strain:

- a) Enterotoxigenic E. coli → contaminated food & water
 Travellers diarrhoea, cholera like illness.
- b) Enteroinvasive E. coli → watery diarrhoea or illness like shigella.
- c) Enteropathogenic E. coli → Mucosal effacement → lack of
 Diarrhoea, vomiting, Absorption

i) Enterocaggregative E. coli → diarrhoea, clarkes.
Attach to mucosa produce
diarrhoea without blood.

ii) Enterohemorrhagic E. coli → Contaminated milk,
uncooked vegetables. Bloody diarrhoea, and
Abdominal pain.
→ It produces Verocytotoxin.

Extraintestinal infection → Usually Hospital acquired

- UTI
- Abdominal & Pelvic infection: pyelitis, epididitis, diverticulitis, cholangitis, phyllophth.
- Pneumonia
- Bacteremia & Sepsis
- Bone and soft tissue infection → In lower
- Disseminated intravascular coagulation

Diagnosis

- Culture of E. coli

Treatment - Prevention

- i) Controlled antibiotic - Normal flora present. Prescribe antibiotic and monitor effectiveness.
- ii) Antibiotic prophylaxis - In patients with urinary tract infection, E. coli is the most common pathogen.
- iii) Antibiotic prophylaxis - In patients with urinary tract infection, E. coli is the most common pathogen.

Brucellonin:

Other names: Undulant fever, Malta fever, Abortus fever

- Epizootic disease: caused by *Critam* ^{ferus}
negative bacilli.

Etiology:

- i) *Brucella melitensis* ^{Goat, sheep, camel} cattle.
ii) *Brucella suis* ^{Pig}
iii) *Brucella canis* ^{Dog}

Mode of transmission:

- Infective animals pass the beast in their milk, human gets infected through contaminated milk. Penetration in the wounds, eating uncooked meat, Respiratory droplet, pathogenesis.

→ They enter the local lymph node and then blood and then the bloodstream and then RES and form granuloma.

Clinical features:

- Undulant fever → wide fluctuation and it persists for long periods, weeks to months.
- Fever, malaise, weakness, anorexia, backache.
- Bone and joint → Migrating polyarthritis, Osteomyelitis, Spondylitis, Psoas abscess.

CVS → Pancarditis, thrombophlebitis.

Lung → Pneumonia, Bronchitis.

^{liver} ~~CVS~~ → Cholecystitis, pancreatitis.

CNS → Meningoencephalitis, Stroke, hemorrhage.

UTI → Urethritis, Orchitis.

Diagnosis: Culture → ELISA → IgG, IgM, IgA.
≥ 1:820 is positive.
PCR.

Cholera

O1 serotype - classical
EL Tor - non-toxic

- O139 serotype has been identified
→ Pandemic
Contamination - Fecal-oral Route, water
clinical picture

- Onset, acute
- Risk: watery stool
- Flank or muscle
- Dehydration, cramp
- Shock & organ
- Death from acute circulatory failure
- Intensely Cholera Sicker with loss of fluid into the bowels. killing before

symptoms appear

- Profuse watery stools

- Vomiting

- Dehydration

- Muscle cramps

- Rapidly progressive

- Shock

- Death

- Recovery

- Prognosis

Biliary dysentery (Sonder, Flexner) dysentery

- 8-10 yellowish to a green negative stool with blood
- 4 main groups by von Drögen
- only infect humans by von Drögen
- contamination after fecal or oral depression
- contamination after fecal or oral depression
- not usually re-infection

CF

- > diarrhea of feces, tenesmus
- > small stools, blood, pus
- > dehydration
- > respiratory distress

Respiratory Infections

Influenza

- An acute respiratory infection caused by Influenza virus $\rightarrow$ Type A, B, C. All known pandemics are caused by Influenza^A strains.

Problem statement

- $\Rightarrow$ It is truly an international disease. occurs as a pandemic 10-40 years due to antigenic changes.

1) Spanish Influenza 1918, 2) Asian Influenza 1957,

3) Hong Kong Influenza 1968.

- $\Rightarrow$ At present three types of Influenza are circulating around the world, (H_1N_1) , (H_3N_2) & B.
 - $\Rightarrow$ Recently Pandemic Influenza from H₁N₁ 2009, originate from swine occurred called as Swine flu.
- ### Epidemiological determinants

① Agent Factors

- 1 $\rightarrow$ Agent $\rightarrow$ Family $\rightarrow$ Orthomyxoviridae, RNA virus, Influenza virus $\rightarrow$ Type A, B, C.

- $\rightarrow$ Influenza A has 2 Antigens

- $\left\{ \begin{array}{l} H \rightarrow \text{Hemagglutinin} \rightarrow \text{Attachment to host} \\ N \rightarrow \text{Neuraminidase} \rightarrow \text{Release of virus} \end{array} \right.$

- $\rightarrow$ It usually changes its antigenic.

Sudden $\rightarrow$ Antigenic shift (Animal to man),

Gradual $\rightarrow$ Antigenic Drift

- b → Reservoir of Infection: Animals and Birds (Swine, horse, dogs, cats).
- c → Source of Infection → Care or subclinical care, from the respiratory tract.
- d → Period of Infectivity → 1 to 2 days before and 1 to 2 days after onset of symptoms.

② Host Factors

- Age & Sex: Olds, age and children under 18 months.
- Human mobility → main cause for infection.
- Immunity → Due to antigenic shift and drift. Immunity of previous infection is not helpful.

③ Environmental Factors

- Season → Winter season.
 - Overcrowding → Schools, Institution, ships etc.
- ④ Incubation period - 18 to 2 hrs.

⑤ Mode of Transmission

- Droplet infection by sneezing, coughing, talking.
- Port of Entry is Respiratory tract.

Pathogenesis

- Hemagglutination → Respiratory epithelium

RNA multiplication

New Virus coming out via

Neuraminidase

- Inflammation, Necrosis of superficial epithelium of the tracheal and bronchial mucosa followed by secondary invasion of Bacteria and No viremia.

Clinical features

- Fever, chills, aches, pains, coughing, weakness
- Fever for 1-5 days,
- Complication $\rightarrow$ Acute sinusitis, otitis media, Purulent bronchitis, pneumonia.
- Reye Syndrome $\rightarrow$ Fatty liver with encephalopathy

Investigation

① Virus Isolation

② Serology $\rightarrow$ ELISA for Hemagglutination inhibition

Prevention

- $\rightarrow$ Good ventilation, crowded places.
- $\rightarrow$ Vaccination is frequently changed according to the strains that are changing frequently.

Influenza Vaccines

① Killed Vaccine:

- $\rightarrow$ One dose ~~to~~ Subcutaneous or Intramuscularly, 0.5ml in adults and 0.25ml in children ~~bel~~ of 6 to 36 months, under-9 require 2 doses.
- $\rightarrow$ It can produce egg allergy, Guillain Barre Syndrome.

② Live attenuated Vaccine.

- $\rightarrow$ Trivalent live, attenuated vaccine.

as a single dose of intranasal spray.

Contraindication

- $\rightarrow$ Chicken egg allergy.
- $\rightarrow$ Anaphylactic allergy
- $\rightarrow$ Guillain Barre syndrome ~~before~~ ^{within} 6 weeks.
- $\rightarrow$ children below 6 months.
- $\rightarrow$ severe illness.

Treatment $\rightarrow$ Antiviral drugs

Avian influenza → Primarily affecting birds with H₅N₁ strain which can infect human at some time.

Pandemic influenza H₅N₁ 2009 (Swine flu)
→ Due to genetic drift and shift and also infects lower respiratory tract, progressive pneumonia.

Symptoms :- cardiopulmonary insufficiency,
 - CNS complication.

a) Complicated ~~to~~ severe influenza

b) Uncomplicated Influenza.

Lab :- RT-PCR chain reaction. from nasopharyngeal swab.

- Viral culture

→ Rapid test kits

→ Four fold increase of Influenza (H₅N₁)

Specific neutralizing antibodies

Infection Control

→ Strictly adherence to hand hygiene.

→ Mask, Ng 5, etc.

Pandemic influenza 2009 Vaccine

a) Inactivated vaccine → Monovalent.

→ 0.5ml multidose vials 10 doses.

→ Single dose intramuscular.

b) Live attenuated vaccine → Nasal spray.

Corynebacterium Diphtheria

↳ Club

↳ Leather

- Infectious disease caused by *Corynebacterium Diphtheria*, which causes leather like membrane in the pharynx.

Problem statement

World → Rare disease,

India → Endemic But retrospective data indicates a declining trend.

Epidemiology

1) Agent Factor

a) Agent → *Corynebacterium diphtheriae*,

Cream positive, Aerobe,

→ No invasive power but produces Exotoxin

4 types = *C. diphtheriae* → Most common
↳ *intermedius*
↳ *Belgiant*
↳ *mitis*

b) Source of infection:

1) Cases → Subclinical to clinical

2) Carriers → 95% are carriers

c) Infective material: Nasopharyngeal secretions,

d) Period of infectivity: 14 to 28 days after onset of symptoms.

2) Host Factors

a) Age & Sex → Mostly children under 5 age.

b) Immunity → In immunocompromised children.

c) Environmental → all season, mainly winter.

d) Mode of transmission → Droplet infection also by contact with lesion, By objects (Toys, pen etc)

5) Portal of entry

- a) Respiratory route.
- b) Non-Respiratory route - Eye, orital, skin lesion
- c) Incubation Period

Clinical Features

① Pharyngotonsillar

- ↳ Pseudomembrane covering the entire tonsil, soft and hard palates.
- thick blue white or grey black, attempts at removing cause bleeding.
- edema over the mandibular area giving a "Bull necked appearance".
- Difficulty in swallowing, low grade fever.

② Laryngeal

- often preceded by pharyngotonsillar disease, associated with fever, hoarseness, croupy cough, and extend to bronchial tree.
- obstruction leading to suffocation.

③ Nasal diphtheria → Milder form, localized to septum or turbinate.

Toxin continues → Toxemia heart, liver, kidney to be produced.

↓
Nerves → symptoms (paralysis,)
Brain → symptoms (vision, speech)

- Non respiratory mucosal, surgical and cutaneous diphtheria also occur.

6) Portal of entry

- a) Respiratory route.
 - b) Non-Respiratory route - Eye, Peritonsillar, skin lesion
- ## a) Incubation Period

Clinical Features

① Pharyngotonsillar

- ↳ Pseudomembrane covering the entire tonsil, soft and hard palate.
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do be produced

↓
Bacteria → Symptoms (vision, speech)

↓
Nerves → Symptoms.

(Paralysis)

- Non respiratory mucosal surface and cutaneous diphtheria also occur.

Control of Diphtheria

① Carer and Carver

- a) Early Detection
- b) Isolation
- c) Treatment

2) Contacts → Immunized and Surveillance -

3) Community → Immunization

Diphtheria Immunization

a) Combined or mixed vaccines

- ① DPT (Diphtheria - Pertussis, Tetanus Vaccine)
- ② DPTw (Diphtheria, Tetanus, whole cell pertussis)
- ③ DTPa (Diphtheria, tetanus, acellular pertussis)
- ④ DT (Diphtheria, Tetanus toxoid)
- ⑤ dt (Diphtheria, Tetanus, adult type)
- ⑥ Pentavalent (Diphtheria, Tetanus, Pertussis, Hepatitis B and Hib) -

b) Single Vaccine

- ① FT (Formal - toxoid)
- ② APT (alum - precipitated toxoid)
- ③ PTAP (Purified toxoid aluminium phosphate)
- ④ PTAH (Purified toxoid aluminium Hydroxide)
- ⑤ TAF (Toxoid, antitoxin flocculus) -

c) Antitoxin

- Diphtheria antitoxin.

A) Combined Vaccines

① DPT Vaccine

- For immunizing infants DPT is the first choice for

① vaccine against three disease

② Pertussis component enhances the potency of the diphtheria toxoid.

Type:- 1) Plain

2) Adjuvanted $\rightarrow$ In aluminum phosphate or hydroxide.

② Storage $\rightarrow$ Should not be frozen.
Kept at 2-8 deg C.

① Age $\rightarrow$ Young infants $\rightarrow$ 6 weeks after birth.

② Number $\rightarrow$ 0.5 ml of 3 doses.
of Dose

③ Interval between dose $\rightarrow$ 4 weeks between 3 doses and a booster at 16-18 months, 5 years.

④ Mode of administration $\rightarrow$ Intramuscularly in anterolateral aspect of the thigh.

⑤ Reactions $\rightarrow$ Fever, and mild local reactions, Swelling, pain and induration.

Severe complication $\rightarrow$ Encephalitis, Convulsion, Infantile spasm / Reye's Syndrome due to pertussis compound.

⑥ Contraindication.

$\rightarrow$ Mild fever, cold, cold are not contraindication.

$\rightarrow$ Only those who are needed to be hospitalized.

$\rightarrow$ Severe reaction for the previous dose.

$\hookrightarrow$ In those cases DT alone is given without pertussis compound.

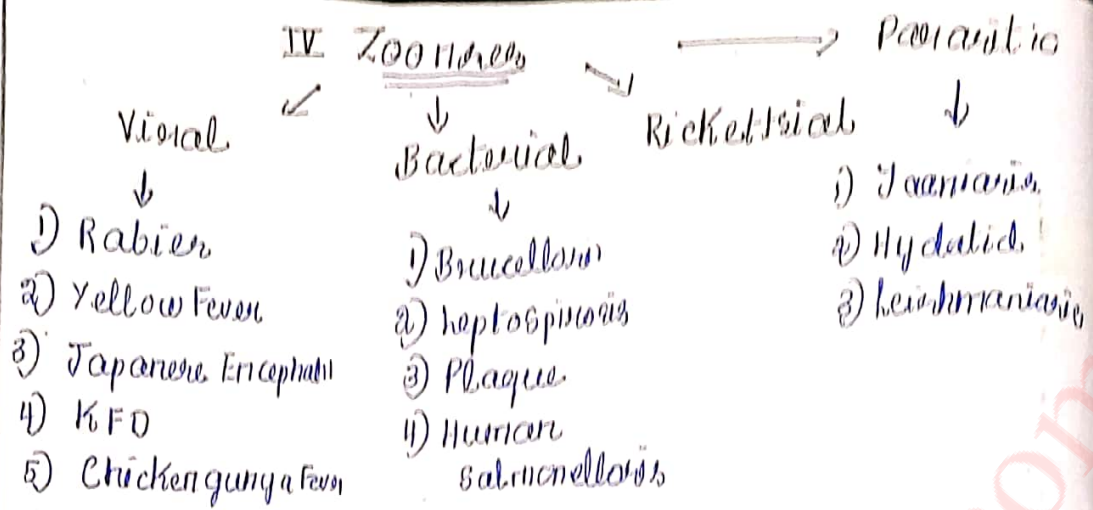
$\rightarrow$ Since pertussis is less after 6 years, it is not recommended for adults $\rightarrow$ 2 doses after 2-6 weeks. Booster at 6 to 12 months.

2) Pentavalent Vaccine

- DPT, Hepatitis, B and Hemophilus influenza B.
- It reduces the prick.
- 0.5 ml. at 6, 10, 14 weeks, and Hepatitis B Bifidone given, in anterolateral thigh intramuscular.

6) Single vaccines → Less used

7) Antiserum → Passive prophylaxis and treatment prepared in horse serum



Rickettsial Infection Zoonoses

Rickettsial zoonoses are a group of specific communicable diseases caused by rickettsial organisms and transmitted to man by the arthropod vectors [except Q fever].

Classification

	Vector	Reservoir
① Typhus Group.		
a) Epidemic Typhus	Louse	Human
b) Endemic Typhus (Murine)	Rat flea	Rodents
c) Scrub Typhus	Thrombiculid Mite	Rodents
② Spotted Fever Group.		
a) Indian tick typhus	Hard tick	} Rodents Dogs
b) Rocky mountain spotted fever	Hard tick	
c) Rickettsial pox	Thrombiculid mite	- Mite. Rodents
③ Others		
a) Q Fever →	Nil	- Cattle, sheep, goats.
b) trench Fever →	Louse	→ Humans.

Causal agents

- Rickettsia are small, bacteria that are obligate intracellular parasite.

→ Shape → Pleomorphic, Rods or cocci and appear in groups or singly.

→ Giemsa stain → Blue and readily visible under microscope.

→ Grow readily in yolk sac of the embryonated egg and enhanced by presence of sulphanilamide.

Clinical Features

→ Except Q.Fever, in which there is no skin lesion, others are characterized by fever, headache, malaise, prostration, skin rash, enlargement of spleen and liver.

Investigation

① Isolation of Rickettsiae

② Serology test like:

↳ Weil Felix reaction.

→ Indirect Fluorescent antibody (IFA)

→ Complement Fixation test.

③ ELISA

④ Fluorescent antibody staining of frozen tissue.

Treatment

Tetracycline.

Q Fever

- Highly infectious zoonotic disease with world wide distribution.
- In persons who are associated with sheep, goats, cattle or other domestic animal

Agent Factors

- ① Agent → Caused by Coxiella Burnetii, which is found in soft tick which is vector as well as reservoir.
- ② Animal host → Cattle, sheep, goats, ticks, (Reservoir) which shed it in feces and ^{in milk} and also placenta which shed during parturition create infectious aerosols.
- Camels, horses, dogs and other domestic animal also found to be linked.

Mode of Transmission

- No arthropod involvement.
- ① Inhalation of infected soil or aerosols
- ② Infection through cuts, abrasions conjunctiva.

Incubation period → Usually 2 to 3 weeks.

Clinical features

- Clinical picture of Influenza or non-bacterial pneumonia rather than a typhus
- No B. mark or lesion in skin
- Acute onset of fever, chills, Malaise, Headache

→ Can cause pneumonia, Hepatitis, encephalitis, endocarditis

Control measures

→ Preventive measures:

- Pasteurization or Boiling of milk.
- Disinfection and disposal of cattle sheds.
- Inactivated Coxiella Vaccine, to the occupationally exposed workers.

Gastrointestinal Bacterial Typhs

- i) Staphylococcus aureus - Gastroenteritis
- ii) Bacillus cereus - Fried rice
- iii) Clostridium perfringens - CNE, Anitoxin, Neurotoxin
- iv) Campylobacter jejuni
- v) Escherichia coli
- vi) Salmonella Typhi and paratyphi
- vii) Clostridium difficile - Antibiotic colitis
- viii) Yersinia Enterocoli - Pseudo-Typhoid
- ix) Vibrio cholera

Shigellosis

1) Staphylococcus aureus

- Fecal-oral route, contaminated food and water
- 1 to 6 hrs onset
- Gastroenteritis

2) Bacillus cereus

- Within few hours onset and self limiting within one day,
- Fried rice.

3) Clostridium perfringens

- Out of large intestine
- Uncooked meat
- 6-12 hrs onset.
- β toxin variety CNE, Clostridium Neurotoxin

iv) Campylobacter jejuni

- Survives well in fresh water
- zoonotic disease
- With washing the meat products

- Complication $\rightarrow$ Ascending Neuroparalysis $\rightarrow$ Guillain Barre Syndrome
- 5 to 7 days self limiting

$\downarrow$
Autoimmune disease

vi) Clostridium difficile

- Normal intestine flora
- After ~~is~~ antibiotics it affects the patient's intestine.
- It can mimic C/F of ulcerative colitis.
- Colon may have pseudomembranous formation.
- Stool examination.
- Sigmoidoscopy.

vii) Yersinia Enterocolic

- Pork contaminated.
- Reactive arthritis (Reiter's Syndrome),
 $\downarrow$
Nongonococcal urethritis,
Iritis,
Arthritis.

ii) Vibrio cholerae

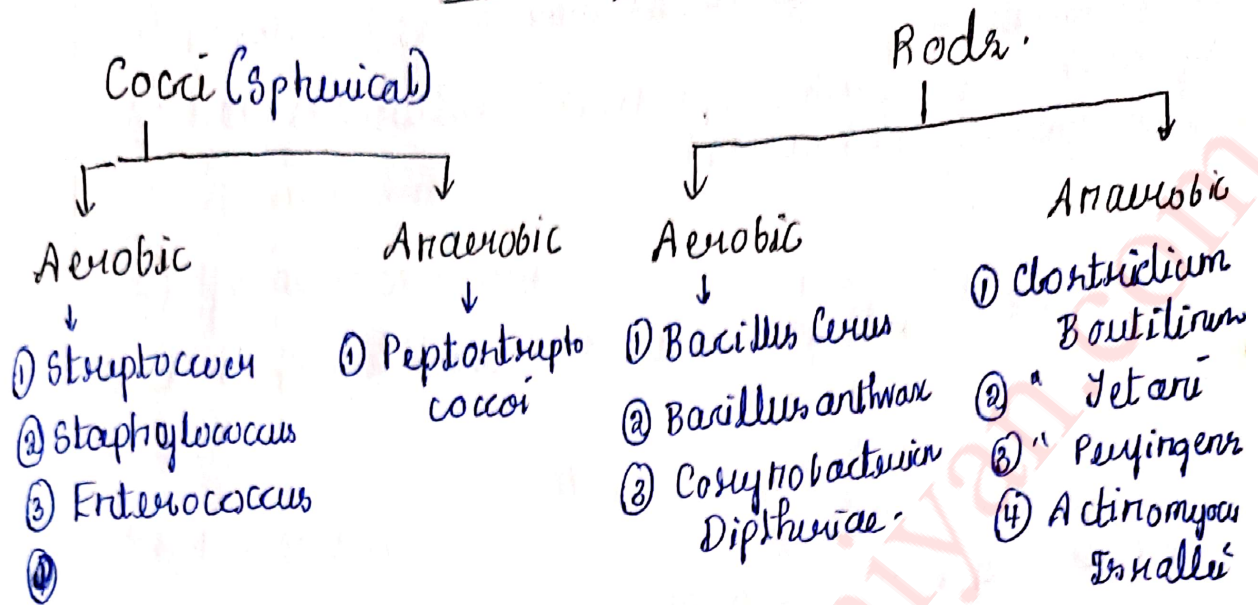
- Fecal-oral route.
- Rice watery stool.
- Choleraemia $\oplus$ From dehydration patient due to stool
- $\oplus$

ii) Shigella

- ~~Bacillus~~ shigella
- ~~Bacillus~~ $\rightarrow$ dysentery $\rightarrow$ $\oplus$
- ~~Bacillus~~ Flexner.

- Produces virulent enterotoxins.
- Presence of tenesmus $\oplus$

Gram Positive Bacteria



Gram negative Bacteria

- ① Rods
 - ① E. coli
 - ② Enterobacter
 - ③ Klebsiella Pneum
 - ④ Proteus
 - ⑤ Pseudomonas
 - ⑥ Yersinia Pestis
 - ⑦ Shigella
- ② Comma shaped
 - ① Helicobacter Pylori
 - ② Vibrio Cholerae
 - ③ Campylobacter Jejuni
- ③ Cocci
 - ① Brucella
 - ② Bordetella pertussis
 - ③ Hemophilus influenza
- ④ Diplococci
 - ① Neisseria Meningitidis
 - ② Neisseria Gonorrhoea
- ⑤ Cocci
 - ① Salmonella

Mycobacteria — Tuberculosis
— Leprosy

Others

- ① Chlamydia
 - ① Chlamydia Trachomatis
 - ② Chlamydia Pneumonia
- ② Mycoplasma
 - ① Mycoplasma Pneumonia
- ③ Spirochetes
 - ① Treponema Pallidum
 - ② Leptospira
 - ③ Borrelia Burgdorferi (Lyme Disease)
 - ④ B. burgdorferi
- ④ Rickettsial related
 - ① Coxiella Burnetii

Viruses

DNA Virus: (CHAPPA)

- 1) Poxviridae → Variola (smallpox) Cow Pox, anthrax, Molluscum contagiosum
- 2) Herpesviridae → Chicken pox, Herpes zoster, Cytomegalovirus, Epstein Barr, Infectious mononucleosis
- 3) Adenoviruses
- 4) Hepadnaviridae
- 5) Papoviridae → Human Papilloma Virus

RNA Virus

- 1) Paramyxoviridae → Measles (Rubella), Mumps, Nipha.
- 2) Togaviridae → Alpha → Chikungunya, Rubivirus → Rubella
- 3) Flaviviridae → Mosquito Borne
 - ↳ 1) Japanese Encephalitis
 - 2) Yellow Fever
 - 3) Dengue Fever, 4) Zika virus
 - Tick Borne → HFD
- 4) Reoviridae → Rotavirus
- 5) Retroviridae → HIV 1-2
- 6) Coronaviridae → SARS, COVID 19, MERS
- 7) Filoviridae → Ebola
- 8) Picornaviruses → Enterovirus
 - 1) Poliovirus
 - 2) Coxsackievirus
 - 3) Echovirus
 - Rhinovirus
- 9) Orthomyxovirus → Influenza

→ Arbovirus

Herpes Viruses:

Type I	Alpha	Herpes Simplex Virus Type 1
Type II	Beta	Herpes Simplex Virus Type 2
Type III	Gamma	Varicella Zoster Virus
Type IV	Beta	Cytomegalovirus
Type V	Gamma	Epicstein Barr Virus
Type VI	Gamma	Human Herpesvirus 6
Type VII	Gamma	Human Herpesvirus 7
Type VIII	Gamma	Human Herpesvirus 8
Type IX	Gamma	Human Herpesvirus 9
Type X	Gamma	Human Herpesvirus 10
Type XI	Gamma	Human Herpesvirus 11
Type XII	Gamma	Human Herpesvirus 12
Type XIII	Gamma	Human Herpesvirus 13
Type XIV	Gamma	Human Herpesvirus 14
Type XV	Gamma	Human Herpesvirus 15
Type XVI	Gamma	Human Herpesvirus 16
Type XVII	Gamma	Human Herpesvirus 17
Type XVIII	Gamma	Human Herpesvirus 18
Type XIX	Gamma	Human Herpesvirus 19
Type XX	Gamma	Human Herpesvirus 20
Type XXI	Gamma	Human Herpesvirus 21
Type XXII	Gamma	Human Herpesvirus 22
Type XXIII	Gamma	Human Herpesvirus 23
Type XXIV	Gamma	Human Herpesvirus 24
Type XXV	Gamma	Human Herpesvirus 25
Type XXVI	Gamma	Human Herpesvirus 26
Type XXVII	Gamma	Human Herpesvirus 27
Type XXVIII	Gamma	Human Herpesvirus 28
Type XXIX	Gamma	Human Herpesvirus 29
Type XXX	Gamma	Human Herpesvirus 30

- Double stranded DNA.
- They have capsid around them.
- Heat labile.
- All cause latent infections.

Herpes Simplex Virus

- HSV Type I $\rightarrow$ Above the waist $\rightarrow$ Oral Herpes
- HSV Type II $\rightarrow$ Below the waist $\rightarrow$ Genital Herpes

Transmission:

HSV Type I $\rightarrow$ Droplet on Direct contact

HSV Type II $\rightarrow$ Venerally.

Pathogenicity:

HSV1 & HSV2 infect the sensory neuron

↓
Travel along the axon

↓
To the cell body & become latent

Face

Genital

asymptomatic

↓
Trigeminal ganglion

↓
Sacral ganglion

↓
Unilateral lesions when the immune system weakens.

Clinical Features

Primary infection is usually asymptomatic.

Oral - Gingivostomatitis, Pharyngitis.

- Clusters of small, fluid-filled blisters, painful white sores & ulcerate and heal without scarring

Genital - Male $\rightarrow$ Shaft of Penis.

Female $\rightarrow$ Labia, Mons Pubis, vaginal Mucosa, Cervix.

Cutaneous:

- In Thumb → Herpes Gladiolus
- In Fingers → Herpes Whitlow → Auto inoculation
- In already infected eczema on buttocks
↳ Eczema Herpeticum

Eyes: Kerato Conjunctivitis

Kernea → Blurry Vision & Branching Dendritic Lesion

Conjunctiva → Pain, Redness, Tearing, Photophobia

CNS: Meningitis & Encephalitis

→ Usually Temporal lobe

→ CSF shows ↑ RBC, ↑ WBC, Protein

PCR shows HSV DNA

Neonatal or Congenital

→ When Baby passes through vaginal mucosa

→ Causes Cutaneous, CNS or Disseminated infect.

Diagnosis

→ PCR, → Viral DNA

→ Specimen Culture

→ Antibodies against the virus

He

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Herpes Zoster (Shingles)

Varicella → Chicken pox. } Caused by
Herpes Zoster → Shingles } ~~Herpes~~ Varicella
Zoster Virus.

Pathophysiology

After the primary infection (Chicken pox)

↓
It goes retrogradely and gets into
Dorsal root ganglion &
Trigeminal ganglion

↓
Remains latent

↓
Triggered when immune system
weakens.

Clinical Features

- Where Varicella is the disease of childhood,
the Shingles is of old age.
- Usually when reactivated by some triggers.
 - Ipsilateral side of Face.
 - Single stripe of vesicles along the course
of the nerve on one side of the Body.
 - Pain, itching, tingling, Paroxysms.
- The sores heal by ~~heal~~ itself within
2 weeks.
- Pain may be present in the affected
dermatome > 90 days
 - ↳ Post herpetic neuralgia.

Ramsay Hunt Syndrome (Herpes Zoster abicus)
→ Facial palsy. → Form of zoster outbreak
affecting facial nerve, causing

Facial
Paralysis

Hearing
loss

Eruption on
Tympanic membrane
and External
auditory canal.

Other symptoms

- Ear Pain, Ringing in ears (tinnitus)
- Difficulty closing one eye.
- Vertigo, change in taste perception.
- Dry mouth on eyes

Diagnosis

→ Basis on skin lesions.

- PCR for DNA.

Tzanck Test

→ viral antibodies

Arbovirus

Flaviviridae → Mosquito Borne

Yellow Fever

- It is a zoonotic disease, which principally affects the monkeys and are transmitted to man by aedes mosquito.

Problem Statement

- It is endemic in Africa and central America.
- No cases have been reported in India.

Epidemiological determinant

① Agent Factors

Agent - Flavivirus arbovirus

Reservoir - In Forest Monkey and forest mosquito

In Urban areas, Human besides aedes.

Period of communicability.

Man - 3-4 days of illness

Mosquitoes - 8-12 days of incubation

② Host Factors

Age → All age groups

Occupation → Wood cutters and Hunters (Forest workers)

Immunity → One attack gives life long immunity.

③ Environmental Factors

Climate - more than 24°C.

Social - Urbanization is making man close to forest and travelling to endemic areas.

Mode of Transmission

Jungle (sylvatic) TF → Monkey → Wild mosquito

Urban TF → Man → Aedes aegypti → Man Humans passing.

Incubation Period - 3 to 6 days.

Clinical Features

- Mild Febrile illness for less than a week, headache, myalgia, conjunctival erythema + bradycardia.
- Usually fever disappears, but reappears in severe cases causing, Back pain, abdominal pain, somnolence, Nausea, Vomiting, Jaundice.
- Liver and Renal damage occur and failure.
- DIC occurs causing petechiae, haemorrhagic bleeding $\rightarrow$ Shock.

Diagnosis

DD $\rightarrow$ Typhoid, malaria, leptospirosis, hepatitis.
Haemorrhagic fever.

- RT-PCR $\rightarrow$ Virus, IgM $\uparrow$, IgG fourfold increase.
- CBC - leucopenia, LFT $\rightarrow$ AST, ALP, GGT.

$\rightarrow$ In Postmortem Finding $\rightarrow$ Councilman Bodies
But not specific.

Control

Surveillance $\rightarrow$ No control, Only Vaccine to Human.
Urban Yellow Fever.

Vaccination $\rightarrow$ 17D Vaccine - live attenuated vaccine
injection at deltoid, subcutaneously, at
0.5ml dose to all age, giving 35 years immunity.
Not recommended in Pregnant, children,
Immunodeficiency, and egg allergy.

1) Vector control $\rightarrow$ Source reduction $\rightarrow$

Anti larval and anti-adult measures -
and protection like repellants, nets.

2) Surveillance: For early detection in endemic.

- Travellers should show Vaccinated

$\rightarrow$ Aircraft and ships from endemic are given
insecticides.

Japanese Encephalitis

- Mosquito borne encephalitis, zoonotic disease, infecting mainly animals and incidentally man causing Acute encephalitic syndrome.

Problem Statement

- The First case was found in Tamil nadu in 1905.⁵
It is endemic in India and reporting outbreaks every year.

Epidemiological Features

Agent Unlike Dengue, JE infects animals and birds.

a) Pig $\rightarrow$ Mosquito $\rightarrow$ Pig.

b) Ardeid bird $\rightarrow$ Mosquito $\rightarrow$ Ardeid bird.

$\rightarrow$ The man is incidentally infected and he is a dead end host. No man to man transmission.

Host $\rightarrow$

Animal host - Pigs mainly, cattle, Buffalo other

Birds $\rightarrow$ Pond herons, ducks, poultry etc.

Mosquito vectors

- Culicine mosquito is mainly found in India which is found in rice fields, shallow ditches, pools.

Incubation Period - Not known, 5-15 days.

Clinical Features $\rightarrow$ 3 stages; Most of them are asymptomatic.

a) Prodromal stage $\rightarrow$ 1-6 day.

- Fever, headache, malaise, lethargy,

C.I. disturbances.

b) Acute Encephalitic stage.

- Fever, Neckal rigidity, Convulsions,

- Signs of Intracranial pressure, Dysarthria,

Dystonia, hemiplegia, quadriplegia, tremors

Coma $\leftarrow$ etc.

c) Late stage and sequelae-

- ↓ ESR and Temperature
- Neurological signs improve, but damage persists if they survive.
- Case Fatality rate in 20-40 percent,
- The period from onset of symptom to death is 9 days.

Diagnosis

- CSF → ↑ Protein × ↑ Cell count, ↑ WBC
- Culture of Blood or CSF → Single stranded RNA, JE.
- IgM Capture ELISA.

Control

① Vaccine:

① Mouse-Brain derived inactivated vaccine by Formalin.

- For children, 1-3 years.
- 2 primary doses 4 weeks apart, and then one year apart, and then 3 years once for adults 10-15 years.

→ It is only shortlived, multiple doses are needed and costly.

② Cell culture derived live attenuated

Vaccine → SA 14-14-2 strain

- 1 dose and 1 booster dose 1 year apart

③ Cell culture derived Inactivated.

- Vaccination of the pigs are given importance.

④ Vector Control

Causers of Acute Encephalitis syndrome

AES

JE

Single stranded
RNA of Flaviviridae
Family.

Non- JE Viral.

Arbovirus

- i) West Nile Enceph J → Not in India
- ii) Tick Borne Enceph J → Endemic
- iii) Dengue
- Enterovirus
- i) Polio
- ii) Non-polio (Cocksackie, Echo)
- Herpes
- i) Herpes
- ii) Varicella zoster
- iii) Molluscipox

Others

- i) Parasite (Malaria)
- ii) Protozoa (Amoebic)
- iii) Spirochete (Syphilis)
- iv) TB meningitis
- v) Fungal (Cryptococcal)
- vi) Toxins
- vii) Hypophysitis
- viii) Chemical

Dengue Fever (DF) (Break Bone, Dandy)
→ It is an Arbovirus of Flaviviridae family.
RNA infecting human through Aedes aegypti
mosquito. Infection can be asymptomatic to
three types of manifestation.

Problem statement

World: Many epidemics have occurred and
increasing over last 3 decades. Many countries
are endemic in many 100+ countries.

India → ↑ in recent years due to urbanization,
life style changes, improper water management
and waste storage increasing the breeding areas.
→ All 4 serotypes are seen in India.

Epidemiological determinants

1) Agent Factors

a) Agent → Genus - Flavivirus,

4 Serotypes → DEN 1, DEN 2, DEN 3, DEN 4.

- One serotype infection causes lifelong immunity
to that serotype.

- Secondary infection with DEN 2, or multiple
infection with different serotype cause DHF/DSS.

b) Vector → Aedes Aegypti and Aedes albopictus.

↓
Tiger
mosquito.

↓
Needs multiple person
to complete blood meal

↓
one person is
enough.

2) Host Factors

a) Age & Sex → All ages, sexes.

b) Immunity → one strain gives one person immunity on
that.

3) Environment Factors

→ Artificial accumulation of water, cold storage,
near to mosquito contact.

④ Transmission of Disease

→ Aedes mosquito becomes infective by feeding on the person from ^{by one} onset to 5th day of illness. After incubation period of 8 to 10 days it becomes infective by the bite, during day and

⑤ Classification Only upto ankle

Early → i) Classic DF ii) Dengue Hemorrhagic Fever ii) Dengue Shock syndrome.

Present i) Dengue with and without signs ii) Severe Dengue.

Pathogenesis

First attack of dengue

Bite → Replicate in lymph node → Replication in RES & skin

Usually self limiting & viraemia ^{after 7} for 3 days.

Subsequent attack of dengue on secondary, multiple long immunity to that strain, no short immunity to other strains.

↓
Extent of viral spread is higher.

↓
Mononuclear cells.

↓
T cells

↓
B cells

↓
To ↓
TNF α ,

↓
Antigen antibody
Complexes

↓
Complement activation.

↓
Too less platelets & too much fibrinolysis

↓
Disseminated Intravascular Coagulation
& Bleeding (Plasma leakage).

→ Clinical Features

Incubation period → 2-7 days.

Prodromal period → 2 days of malaise and headache.

Acute onset → Fever, Backache, arthralgia,

"Break Bone fever", Eye movements, laryngism,

Scleral injection, Nausea, Vomiting, Prostration.

Fever → "Saddle-Back" or continuous with break

on 4th, 5th day and returns.

Rash → Maculopapular starts spreading centrifugally, and sparing palms and soles.

Convalescence → prolonged fatigue syndrome.

arthralgia or depression.

→ Complication

① Dengue Hemorrhagic fever:

→ Increased capillary permeability and hemostatic derangements.

→ Grade I to IV.

→ "Positive tourniquet test" → Petichae appear in the arm after the cuff is inflated for 5 minutes between systolic and diastolic blood pressure which is a non specific test for capillary permeability and thrombocytopenia.

→ Ecchymosis, epistaxis, GI or Vaginal hemorrhages are common.

② Dengue Shock Syndrome (DSS)

→ Serious complication Dengue fever.

→ Shock, with cold, clammy extremities,

Cyanosis, Bradycardia, Hypotension,

Narrowing of Pulse Pressure

Investigation

① Blood $\rightarrow$ $\downarrow$ Pancytopenia, $\downarrow$ Fibrinogen, $\uparrow$ PPT, $\uparrow$ PT, $\uparrow$ D dim
② Virological $\rightarrow$ LFT, RFT

$\rightarrow$ Virus Isolation By Cell Culture.

$\rightarrow$ RT-PCR $\rightarrow$ RNA.

$\rightarrow$ ELISA $\rightarrow$ NS1 Antigen (Non-structural protein 1)

$\rightarrow$ $\hookrightarrow$ 90% sensitive in febrile phase and 1st of fever.

③ Serology

$\rightarrow$ Complement fixation & neutralizing antibody.

$\rightarrow$ IgM ELISA $\rightarrow$ Rise rapidly and ~~down~~ down after months.

Control Measures:

① Mosquito control $\rightarrow$ Antilaval and antiadult measures.

② Vaccines $\rightarrow$ Trivalent vaccine. CYD-TDV
0.5 ml, 3 doses at 6 months gap and also
Contraindication $\rightarrow$ Single dose vial

$\hookrightarrow$ Allergy, Immuno compromised, HIV, Previous injections, Breast feeding, etc.

③ Other measures $\rightarrow$ Repellent, Bed net, clothing, source reduction.

Global strategy for dengue prevention and control 2012-2020.

Treatment $\rightarrow$ IV, Normal saline.

- Blood Components / Transfusion $\rightarrow$ One unit of platelet supplementation will raise them by 5,000 - 10,000 only.

Measles (Rubeola)

Family - Paramyxoviridae

Genus - Morbillivirus.

Species - Measles Virus; RNA Virus

- It is a highly infectious disease which causes fever and 3 C's - Cough, Conjunct, Conjunctiv and exanthemata.

Problem statement:

- Measles is endemic in almost all the country and if introduced to the virgin population it produces 90% of the infection.
- Measles vaccination has reduced it drastically.
- In India the measles is a very severe mortality in children, and WHO says 2% of under 5 mortality is Measles.

Epidemiological determinants -

1) Agent Factors

- 1) Agent $\rightarrow$ RNA virus; 3 hours infectivity in air.
- 2) Source of Infection - Cases, No carriers.
- 3) Infective material $\rightarrow$ Droplets.
- 4) Communicability $\rightarrow$ Prodromal period and the appearance of rash, 4 days before and after 4 days of rash.
- 5) Secondary attack rate $\rightarrow$ Life long immunity as only one antigenic strain is there.

2) Host Factors

- 1) Age $\rightarrow$ Children usually.
- 2) Sex $\rightarrow$ Equal
- 3) Immunity $\rightarrow$ One attack gives life long immunity.

Infants are protected by maternal immunity till 6 to 9 months.

4) Nutrition: Severe malnutrition.

3) Environmental

- Any season, In India Jan to April; Crowded areas.

Transmission

- Directly from person to person, droplets,

- 4 days before and after exanthema.

- I zone.

Incubation period → 10 days for fever and 14 days for rash appearance.

Pathophysiology

Droplets → Trachea or Bronchi → Furo with epithelium
lung

Intertine - Blood ← Lymph nodes ← Multiple.

Brain

Clinical Features

1) Prodromal Period

- From 10 days to 14 days:

2nd day { 3C → Cough, Coryza, Conjunctivitis.

{ High fever, Vomiting or diarrhea.

4th day { Koplik spots.

↳ Like table salt crystal appear on the buccal mucosa opposite the first and second molar & lower molar teeth.

⇒ They are small, bluish-white spots with a red base, smaller than a pin head.

Pathogenic to measles.

2) Eruptive Phase

- Dark red, maculopapular rashes which start from ear (Head) and to the lower part (Cephalocaudal).
- The rash forms as a result of the interaction between the T cells with the virus infected cells in small blood vessel go in Patient with defective cell mediated immunity, no rash appears.

3) Post-Measles

- Lose weight and weak for days.
- Persistent Cough will be present.

Complications

- Lungs $\rightarrow$ Pneumonia, Intestines $\rightarrow$ Diarrhea, Encephalitis.
- Suppresses the immune system upto 6 weeks.
 - $\rightarrow$ Bacterial superinfection
 - $\rightarrow$ Otitis media, Bacterial Pneumonia.

Subacute Sclerosing Panencephalitis

$\rightarrow$ Complication occurring 7-10 years later.

- may be due to abnormal immune response, mutated strain.

$\rightarrow$ Symptoms like Seizures, partial movements, coma.

Diagnosis

- IgM antibodies in Serum.

Treatment

Vitamin A is given to increase immunity and reduce risks and complications.

Measles and chickenpox can occur together -

Prevention

1) Vaccine - One live attenuated vaccine - liquid - freeze dried.

Age $\rightarrow$ 9 months, or even 6 months in outbreak and 4 weeks after.

$\rightarrow$ Route $\rightarrow$ Subcutaneous, or even Intramuscular.

Reaction $\rightarrow$ Fever, CCC after 5 to 12 days.

Immunity $\rightarrow$ 99%., life long immunity.

Contraindications $\rightarrow$ HIV, Allergy, Immunosuppressive.

Complication $\rightarrow$ Toxic shock syndrome.

Combined vaccine $\rightarrow$ MMR, & MMRV (varicella) and MR are effective.

2) Immunoglobulin $\rightarrow$ Passive immunization

$\rightarrow$ Given early in incubation period, within 3-4 days of exposure, and 8-12 weeks

after immunoglobulin should be given.

Eradication - In the process of eradication like small pox.

Right upper arm

Mumps

Family - Paramyxoviridae, Species - Mumps virus.

- Highly infectious disease, which causes glandular enlargement of parotid gland.

Problem Statement

100-1000/1,00,000 people
year

Epidemiology

1) Agent Factors

Agent $\rightarrow$ Myxovirus parotiditis, RNA virus.

Source of Infection $\rightarrow$ Clinical & Subclinical Cases.

Period of Communicability $\rightarrow$ 4-6 days before onset of symptoms to week afterwards.

Secondary attack rate $\rightarrow$ 86%.

2) Host Factors

Age & Sex $\rightarrow$ Childrens especially.

Immunity $\rightarrow$ one attack is life long immunity.

3) Environmental $\rightarrow$ Winter & overcrowding.

Mode of Transmission $\rightarrow$ Droplet ~~infect~~

Incubation period $\rightarrow$ 14 to 16 days.

4) Pathophysiology

Droplet $\rightarrow$ Pharynx $\rightarrow$ Fure & Multiple $\rightarrow$ lymph $\rightarrow$ Blood
(CNS) Viremia

Clinical Features $\rightarrow$ 20-30% are non infective -
Symptomatic

① Swelling of one or Both parotid gland -
but sublingual and submandibular can involve.

② Ear ache.

③ Trismus $\rightarrow$ Spasm of chewing muscles.

CNS - Meningitis $\rightarrow$ Neck stiffness, Fever, Headache.

Encephalitis $\rightarrow$ Difficulty in Balan.

Testicles - $\rightarrow$ Orchitis $\rightarrow$ Testicular atrophy
Epididymis - Epididymitis Decrease in sperm
motility & count

Kidney $\rightarrow$ Glomerulonephritis

Joints $\rightarrow$ Arthritis of hip, knee, shoulder

Heart $\rightarrow$ Myocarditis

- Pregnant women has no risk of congenital defect but induce abortion

Diagnosis $\rightarrow$ Antibody $\rightarrow$ IgM antibody
and 4 fold increase in IgG antibody

Prevention

Vaccine - live attenuated vaccine

- 0.5 ml intramuscular

- After 1 year of age, booster dose later

- Combined with MMR, MMRV

Control = Isolation of the person

AIDS (Slim Disease)

Acquired Immune Deficiency Syndrome is a fatal illness caused by Retrovirus, Human Immunodeficiency Virus (HIV), which makes the immune system ^{weak} and vulnerable to infection.

Problem statement:

- Started in 1980's, and has caused global pandemic too, and in 21st century it has spread very much.
- In India there has been many epidemic.

Epidemiological Features:

1) Agent Factors

Agent $\Rightarrow$ First called as Lymphadenopathy associated virus, then HIV, RNA Retrovirus.

Two strains: HIV 1 $\Rightarrow$ US & worldwide

HIV 2 $\Rightarrow$ Western Africa, Southern Africa

Reservoir of Infection: Both Cerebrum and Cervix.

Source of infection $\Rightarrow$ Blood, Semen, CSF, In some, tears, saliva, milk, Urine, vaginal secretions.

2) Host Factors

Age $\Rightarrow$ Sexual active persons 20-49 years age.

Sex $\Rightarrow$ Man (Bisexual or Homosexual), Prostitutes, women. In some countries equally.

High risk $\Rightarrow$ Prostitutes, Homosexual, multiple partners, Intravenous drug abuse, Blood transfusion.

Mode of Transmission:

1) Sexual Transmission:

- Unprotected sex from infected person.
- Male to Female more common.

- Female in teenage due to thin cervical mucosa and 45 years - due to atrophy.

→ Anal sex is one of the main cause.

→ STD individuals like Syphilis (Chancroid), Herpes are at high risks.

2) Blood contact

- During Blood Transfusions.

- Intravenous Drug abuse like Heroin, Cocaine, etc.

- Needle sharing.

- Skin piercing (Injection, Ear, Tattooing, acupuncture).

3) Vertical transmission

→ From mother to fetus while delivery or peripartum period.

- It can be reduced by antiretroviral therapy.

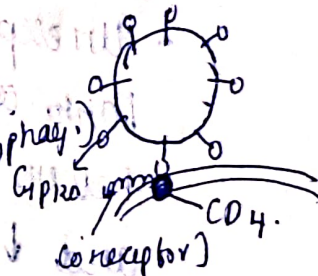
Incubation Period

- Few months to 10 years.

Pathophysiology

HIV virus attaches to the

CD4+ cells (Dendritic cells, T cells, Macrophages)



With help of gp120 in

the envelop and co-receptor (CXCR4 / CCR5)

Single stranded RNA retrovirus

(Reverse transcriptase)

Proviral DNA.

Enter the host DNA and proteins produced.

Virus replications.

So During any infection instead of immune cells, virus are produced.

Clinical manifestation

1) Initial infection

- Mild flu like symptoms early, then goes into a chronic phase, where there is no symptoms.
- HIV antibodies take 2-12 weeks to appear in bloodstream, so the time till the period is called "Window period" and test negative till that.

2) Asymptomatic Carrier state

- No symptoms except generalized lymphadenopathy, But carrier of infection.

3) Aids Related Complex

- It is caused by damage to the immune system but not the opportunistic infection or the cancer. they are.

- Unexplained diarrhea for months, fatigue, loss of 10% of percent of Body weight.

- Fever, sweats, Generalized lymphadenopathy, and $\downarrow$ T_H helper cells.

→ Patient with 2 or more (including lymphadenopathy) is called Aids related Complex.

→ 200 - 500 T cells/mm³.

↳ Mild infections like

Hairy leukoplakia → white patch on tongue.

Caused by Epstein Barr.

Oral candidiasis → Yeast infection.

iv) Aids:

- It is end stage of HIV infection. many number of infection / or cancers occur
Aids Defining Conditions

1) Kaposi Sarcoma

↳ A tumor of reddish brown or purplish plaques or nodules on skin & mucous membrane

2) Oropharyngeal candidiasis → Yeast Fungus

- In tongue or tonsils

3) Pneumocystis pneumonia

4) Herpes zoster or shingles

5) Hairy leukoplakia

6) Cryptococcal meningitis

7) Toxoplasma encephalitis

8) Severe prurigo dermatitis

• Laboratory Diagnosis

Screening Test → Antibody against HIV

ELISA → HIV antibodies → Sensitive

Western Blot → Confirmatory → Specific

→ Virus Isolation

→ CBC → leukopenia, Thrombocytopenia, Anemia

→ $\downarrow$ CD4 cells/ μ L → Normal → 950 cells/ μ L

opportunistic infection
Symptoms come when it becomes less than 200 cells/ μ L

↳ So used to see the prognosis

→ p24 antigen

Control of AIDS - 4 approaches

1) Prevention:

a) Education → Protected sex, Needles, Condoms,
Shared razors, toothbrush,

Women avoiding becoming pregnant.

b) Combination HIV Prevention

→ Structural, Biomedical, Behavioral -

→ DART drugs

2) Male & Female Condoms, 3) Needle & Syringe Program

4) opioid substitute therapy.

5) voluntary medical male circumcision

c) Prevention of Blood Borne Transmission

2) Antiretroviral treatment

① Nucleoside RT inhibitors

② Nucleotide RT inhibitors

③ Non-Nucleoside

④ Protease inhibitors → Ritonavir

3) Specific prophylaxis

→ Prophylaxis for symptoms of AIDS,
like P. carini pneumonia,

Rifampicin to Tuberculosis,
and other infections (Kaposi's, Meningitis).

4) Primary Health Care

→ MHC, Family planning

National AIDS Control Programme → 1987

STD

Syphilis

→ Caused by Treponema pallidum of Gram negative bacteria which belongs to family of Spirochaetes (spiral shape, moves by spinning)

Transmission

① Acquired Syphilis:

→ Direct contact with Body Fluids through

i) Cuts/Breaks → in external genitalia or mouth.

ii) Sexual contact - oral, anal, vaginal.

iii) Contaminated needles.

iv) Direct contact → skin lesions.

② Congenital Syphilis

→ When mother has syphilis, inside the uterus or passing through vagina.

Stages → Acquired Syphilis

① Primary (Early localized)

→ After 1-3 weeks of infection in skin or mucous.

Spirochetes destroy syphilitic tissue

↓
syphilitic chance (Painless with

Hard base and Raised Border) and fluid rich in spirochetes)

↓
Other parts of Body and other

Persons,

Heals on their own in few months.

↓
Lymph node → Enlargement → Blood -
If through Blood transfusion no chance

2) Secondary (Disseminated stage)

- 6 to 12 weeks after infection.
- Enteric Blood and Caustic
- Non-itchy maculopapular rash
- Pustular filled with fluid
- Papulonequamous lesion
- Condylomata (small, white, painless in Genitals, anal, Anus)
- It's the most infectious stage full blood body of spirochetes
- Usually resolves within few weeks to months
- Latent → Becomes dormant and asymptomatic

3) Tertiary Syphilis

Severe Immune response

Type II hypersensitivity reaction

T cells → TNF, IL1, IL6

↓
Severe Inflammation

↓
Gummatous lesion → Granuloma

(Fibroblast, Coagulative necrosis)

↓
Tertiary Organ Damage

CVS

CNS

Liver

Joints

↓
Neurosyphilis

① Cardiovascular system
↳ Inflammation of the supply to aorta
Aorta → Aortic aneurysm.
Aortic Dissection.

② Neurosyphilis (Tabes Dorsalis)
spinochetes in the capillaries which
supply back portion part of the
spinal cord (Dorsal root ganglion).

Damage to the myelin sheath.

↳ Ataxia (loss of coordination)
↳ Ataxia (loss of coordination)
↳ Ataxia (loss of coordination)

↳ Anterior affection causes loss of
sensory, weakness, General paresis
↳ It gets into the brain.
↳ Blurred speech, Altered behaviour,
memory loss, Paralysis, difficult coordination

→ Eyes → Argyll Robertson pupil.
↳ Pupil loses light reflex but
accommodation reflex normal.

Congenital Syphilis

Early disease → First 2 years

↳ Still Born, Dying in womb

→ Maculopapular rash.

→ Snuffles (Full of spirochetes)

→ Damage to liver, spleen, etc.

Late Disease (> 2 years)

- 1) Saddle Nose
- 2) Sabot Rhinos Bent Tibia
- 3) Hutchinsonian teeth Teeth has notch
- 4) Hearing loss

Diagnosis

1) Dark Field Microscopy

2) Serology

1) Non-Treponemal test

1) Rapid Plasma Reagin Test (RPR)

2) Venereal Disease Research Laboratory (VDRL)

3) Anti-Cardiolipin antibody (Reagin)

2) Treponemal test

1) T. Pallidum - Particle agglutination

2) Fluorescent treponemal antibody absorbed (FTA-ABS)

Primary Prevention

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