UNIT 3
MICROBIAL PATHOGENICITY

Infectious Disease

The ability of the organism to cause disease is called microbial pathogenicity. The pathogenic effect of one organism differ from other organism. For example: HIV causes AIDS, *Salmonella typhi* causes typhoid.

Types of pathogen

There are 2 types of bacterial pathogen

- Opportunistic pathogen
- Primary pathogen.

Opportunistic pathogen

When the host immune system is in impaired condition it becomes pathogen and cause disease. Eg. Candidiasis → Candida albicans.

Primary Pathogen

It causes disease in healthy individual who has intact immunological defense.

Eg: Dysentery → *Shigella dystentriae*

Virulence

The organism has the ability to cause disease in relation to resistance to host. It has been classified into 2 strain,

- A virulent strain
- Virulent strain

Avirulent Strain

It does not cause disease because it lacks some antigenic property.

Virulent strain

Which induces disease to human. Eg: Smooth capsule of *Streptococcus pneumonia* induces infection to human whereas rough strain *Streptococcus pneumonia* does not cause any infection.

Invasiveness

Organism invades the host cell, tissue and spreads throughout the body. Eg: Pyogenic infection caused by *Streptococcus pyogenes*.

Toxigenicity

The ability of the micro organism to produce toxin that contributes the development of disease. Eg: Exotoxin produced by *Conjnebacterium diptheriae*, Enterotoxin produced by *Vibrio cholera*, Endotoxin produced by: *Neisseriae* species

Types of infection

Establishment of Infections

Most cell factor: Most possess are receptor and organism efficiently bind with help of Flagella and pili.

Microbial factor: Organism posses pili and fibrae with that it binds to the host cell and facilitate the infection.

Penetration

Organism attaches to the epithelial cell of mucosal coated
↓
Enter submucosal region
↓
And spreads the infection to other parts of body

Multriplication or Colonization

Organism multiplies in the host cell by absorbing the host nutrition.

↓

Resisting phagocytosis

↓

By releasing enzymes like coagulases, nucleases, proteases and capsule.

Toxigenicity

The ability of the pathogen to produce a compound called toxin which are either protein or LPS that produce specific harmful effects to the host. It has been classified into 2 types.

Endotoxin, Exotoxin

Exotoxin: These are the toxin which are secreted outside the bacterial cell. It has been classified into 3 sub types.

i) Cytotoxin
ii) Neurotoxin
iii) Enterotoxin

Cytotoxin is produced by *Corynebaterium diptheriae* Neurotoxin is produced by *Clostridium botubinium*. Enterotoxin by *Virbio cholera*

Endotoxin

These are lipopolysaccharide present in the outer membrane of gram negative bacterial cell wall. The organism lysis its cell and releases the toxin inside the medium. Eg. Endotoxin produced by *Neisseriae* species.

Bacteria, virus and fungi as potential pathogens

Potential pathogens are one that has the ability to cause disease to the human beings and animals by producing various toxins, enzymes and other antigen property. All bacteria, viruses, fungi and parasites are potential pathogens which causes infection in gastro intestinal, respiratory tract and urogenital tract.

Virus

Viruses are obligate intracellular parasites which has the property of multiplying inside the host cell.

Mechanism of viral injury

Viruses can directly damage host cell by entering and replicating at the host expenses.

Mechanism of bacterial injury

The bacterial damage to host tissue depends on the ability of the bacteria to adhere to the host cell, invade cells and tissue to deliver toxin. Pathogenic bacteria have virulence gene that encodes protein and facilitates the infection.

Mechanism of fungal injury

Candida albicans is normal flora of skin, and cavity, gastro intestinal tract etc.

Entry of organism to the host

↓

As a normal flora (opportunistic pathogen) aerosols

↓

It enters upper respiratory tract

↓

It attaches to the epithelial ells of upper respiratory tract (adherence)

↓

It multiplies or colonies rapidly

↓

By absorbing the nutrition from the host cell

↓

It releases enzyme and potent toxin

↓

Which facilitates the invasive nature of infection and resist phagocytosis

↓

It reaches lower respiratory tract (lungs) causing gramiloma infection.

↓

Via lymphnode it enters the blood circulation

↓

From circulation, it reaches heart causing endocarditis

↓

Via blood brain barrier reaches meninges causing meningitis

Clinical symptoms

- Oral thrush
- Candida oerophagitis
- Cutaneous candidiasis
- Lymphadenopathy
- Endocarditis (inf of heart)
- Meningitis

Mechanism of development of resistance

The virulent of the particular strain is not constant, it may undergo spontaneous or induced chain. The virulent strain may spontaneously change into divirulent strain, virulent strain may spontaneously change into virulent strain. This occurs due to mutation, transformation and conjugation. This mechanism is called development of resistance by microorganism. Eg: Organism develops resistance by various factor.

Antiphagocytosis

Organism resist phagocytosis by producing capsule, pili, fimbrae and flagella.

Inhibitors

Some intracellular parasites secrete inhibitors of lysosomal enzyme or even prevent the fusion of lysosome with phagosome. Eg: Mycobacterium tuberculosis**.**

Salmonella typhii fails to stimulate the O_2 dependent killing during phagocytosis.

Staphylococcus aureus inhibits phagocytosis by producing catalase enzyme which blocks oxygenic radial formation.

Antigenic heterogenicity

Variation in antigenic structure.

- Satmonella species have more than 2000 serotypes.
- E. coli posses somatic ‘o’ antigen and flagella ‘h’ ag. Which is of 100 serotype.
- Streptococcus pyogenes posses ‘M’ protein which shares some common epitope between human myocardial self ag.
- Some viruses like influenza, HIV maintain the virulent state without yielding to the host defense system by constantly changing the surface antigen.

Antigenic shift

Antigenic shift is the small change in the surface ag.

Antigenic drift is the drastic alteration in this surface ag.

The reason for antigenic drift and shift are due to mutation. This strategy helps microbes to escape host defense and remain in virulent state.

Development of resistance towards antibiotics by microbes

Antimicrobial resistance: Organism develop resistance by new environmental condition. Some organism like *Staphylococcus aureus* produce *Pencillinase* enzyme which are resistant to penicillin antibiotic. Organism shows resistance to the penicillin antibiotic by converting penicillin into penicillonic acid. Some organism shows resistance by exposing them to high osmosis pressure so it loses its cell wall and resist antibiotic.

Competitive between organism and antibiotic for a essential metabolic analog eg. Sulphanamide.

Cell membrane is altered in such a way that the drug cannot penetrate the cell membrane due to permeability defect. Eg. Polymixin B.

- Alteration in the target site.
- Modification of essential metabolic pathway.
- Drug inactivation mechanism by the production of microbial enzyme.
- Some organism shows resistant to the antibiotic by lapsing into dormant site.

Some micro organism shows resistance to antibiotic such as tetracycline which is plasmid encoded protein, which pumps out the drug out of the cell. Due to decreased permeability, change in shape of receptor or alter in receptor the drug cannot enter the bacterial cell and micro organisms shows resistance towards the particular antibiotics.

Transmission of Drug resistance

Some organisms the resistance gene is transmitted by conjugation process. The isolates of antibiotic sensitive and antibiotic resistance is isolated from the patient suffering from enteric fever. The resistance gene were transferred from *E. coli* which was found in intestinal tract. This factor might be transferred from *E. coli* to *Sphigella dystenriae* which causes enteric fever infection. The resistance is transferred from the resistance factor (R. plasmid) present in *E. coli*.

E. coli is a donor and the recipient are Salmonella, Shigella, Klebsiella (staining).

Bacterial infection

Staphylococcus aureus

Gram positive cocci in clusters, non motile, non capsulated, non sporing, aerobic in nature.

Cultural characteristics

S. aureus is cultivated by following medium

- Nutrient agar
- Blood agar
- Manitol salt agar

Virulence produced by *Staphylococcus aureus*

Enzymes

- Coagulase
- Lipase

- Stapphylokinase
- DNAse
- Hyalauronidases

Toxins

- Alpha toxin
- Beta toxin
- Gamma toxin
- Delta toxin
- Enterotoxin
- Exoflotative toxin
- Toxic shock syndrome I

Antigenic structure

It posses

- Peptidoglycan layer
- Tericholoric acid
- Protein A

Mode of Transmission

- Direct contact
- Aerosol
- Contaminated food and water

Pathogenicity

Enterotoxin

Enterotoxin is responsible to course food poisoning. It consists of 6 sub units. A, B, C_1, C_2, D, E. Among all these subunit type A is responsible to cause food poisoning in human beings.

Exoflotatie toxin

It consists of 2 sub units ETA and ETB.

ETA: is heat stable and chromosomal mediated.

ETB: is heat labile and plasmid mediated

Toxic shock syndrome I

This toxin stimulates the interleukin 1 and induces fever.

Mechanism of toxin action

Enterotoxin

Entry of organism to host cell
↓
By ingestion of contaminated food and H_2O
↓
Organism attaches to the epithelial cells of gastro intestinal region
↓
It releases enterotoxin
↓
Facilitates the infection
↓
Resulting in intestinal obstruction

Clinical symptoms

- Abdominal pain
- Nausea
- Vomitting
- Diarrhoea

Streptococcus

Streptococcus pyogenes

Morphology

- Gram positive cocci in chains
- Non motile
- Non capculated
- Non sporing
- Aerobic

Cultivation

This organism can be cultivated by using blood agar. It produces β haemoloysis pattern.

Virulence factor produced by Streptococcus pyogenes

- Peptidoglycan layers
- Group specific carbohydrate
- Lipoterichoric acid
- Pili
- M.T.R. → Protein
- 'M' protein → major virulence factors causing infection in humans.

Toxins

- Haemolysin → Streptolysin 'O' → 'O_2' labile immunogenic

  Streptolysin 'S' → 'O_2' stable non immunogenic

- Erythrogenic toxin responsible for causing scarlet fever.
- Streptodornase hydrolysis DNA
- Hyaluronidase Hyaluronic acid connective tissue
- Streptokinase Lysis of fibrin clot by activating plasma precursor

Pathogenicity

i) Respiratory infection

Sour throat is a common infection of streptococcus which leads to tonsillitis, pharynitis, followed by inflammation in the middle ear called OTITIS MEDIA and SINULITIS.

Clinical symptoms

- Cervical lymphadenopathy
- Red patches in mucous membrane
- High fever.

ii) Skin and sub cutaneous infection

ii) Genital infection: It causes infection in endometritis

Symptoms

- Puerperal fever (fever with chill)

Systemic infection

It causes infective endocarditis

Post streptococcal sequlae

- Rheumatic fever
- Acute glomerular nephritic (ag-ab complex in nephron)

Biochemical test

- Catalase – positive
- Coagulase – negative
- DNase – Negative
- Manitol fermentation – Negative

Treatment

- Penicillin
- Erythromycin
- Oxacillin
- Cephalexin

Plaque

Causitive agent – Yersinia pestis

Morphology

- Stained with giemia staining
- Capsulateed
- Non sporinig, non motile
- Facultative anaerobic

Cultivation

Organism can be cultivated by

- Nutrient agar
- Blood agar

Antigenic structure

- It consists of heat labile protein envelop antigen (fraction I)
- Fraction I inhibits phagocytosis
- Two Ag Vandw → Present together potent protein

Toxins

- **Plaque toxin**: It possess both characteristics of both exo and endotoxin.
- It is thermo labile in nature

Enzymes

- Coagulase
- Bacteriocin

Mode of Transmission

Zoonotic disease transmitted through rodents.

Pathogenicity

It causes three forms of infection in human beings.

- Bubonic plague
- Pneumonic plague
- Septicemic plague

Entry of organism, to host
↓
By a bite of a Fleo *Xenopsylla choopsis*
↓
Organism enters lymph node via lympholic vessel
↓
From lymph node it enters blood circulation
↓
Causing bacterimial condition
↓
Followed by septicemic condition
↓
Sometimes causes haemorrhages in the skin.

Pneumonic plague

If may occur due to droplets every of organism to the host via aerosols.
↓
Organism enters lungs
↓
Via the lymph node, the organism enters blood stream
↓

From blood circulation, it reaches vital organs.

↓

In Lungs it causes thrombopneumonia

Bubonic plague

It involves the infection of cervical, auxillary in inguinal lymph node involvement.

Septicemic plague

This may occur as the primary infection or as a complication of bubonic or pneumonic plague.

Clinical Symptoms

- Lymphadenopathy
- Bacteremia
- Septicemia
- Pneumonia
- Haemorrhage
- Haematopysis – Blood in sputum

Bio Chemical Test

- Glucose
- Maltose } Sugar fermentation test
- Manitol
- Catalase – Positive
- Oxidase – Negative

Molecular study

Polymerase chain reaction – ELISA

Treatment

- Streptomycin
- Tetracyclin
- Gentamycin

Syphilis

Causative agent : Treponenna palladium / Spirochetes (spiral in shape)

Morphology

- Activity motile
- Presence of Endoflagella
- Exhibits gliding motility
- Micro aerophitic (O_2 – survival)
- Delicate spirochete with tapering ends

Cultivation

Organism can be cultivated by animal inoculation method using Rabits.

Antigenic structure

It possess complex antigenic structure. It has antigen known as cardiozipin antigen. This antigen shares common epitope with OX HEART.

Mode and transmission

Sexual intercourse / venereal disease (STD)

Pathogenicity

Entry of organisms to host by sexual contact

↓

Spirehete enter through minute abrasions on the mucosa or skin

↓

It multiples at the site of entry

↓

After incubation period of about 10 – 30 day it results in primary syphitis.

Primary symphitis

Primary lesions are observed as heart cancer

↓

It is painless, superficial ulcerated lesion

↓

Lympn node is infected (discrete swollen rubbery)

↓

Organism further enters the blood stream

↓

Chancre invariably heals in about 10 – 40 days without treatment

↓

Leaving a thick scar

Secondary synthesis

Popular skin rashes, mucous patches (oropharynoc), condylomata. Patients are more infections during secondary syphitis due to abundant lesion. Further secondary lesion disappears.

As period between 2^o and 3^o syphilis it is known as LATENT SYMPHILIS.

Tertiary Syphilis

Cardiovascular lesion, meningo vascular manifestation, neurological manifestation (paralysis).

Lab Diagnosis

Specimen - Exudates

- Serum sample

WET MOUNT EXAMINATION

- Wassermann 'C' fixation test
- Kahn floccutation test
- Veneral disease research laboratory (VDRL)
- Rapid plasma regain test (RPF)
- *Treponema pallidum* immobilization test (Tp I)
- *Treponema pallidum* fluorescent antibody absorbent test (FTA-ABS)
- *Treponema pallidum* haemagglutination test (TPHA)

Treatment

- Penicillin
- Ceflaxone

Clostridial infection

Closatridial infection is caused mainly by 3 organisms.

- Clostridium tetani which causes Tetanus
- Clostridium botuhinum which cause food poisoning (botulism)
- Clostridum perfringes which cause gas gangrene

Morpholog

- Terminal spore produced by Clostridium tetani
- Subterminal spore C. perfinges
- Central spore by C. botulinum

Gram negative bacilli, non capsulated, actively motile, spore bearer, strict anaerobic

Cultivation

The organism can be grown by using a medicine called Robertson cooked meat media.

i) Clostridum botulinum pathogenicity

Entry of organism to host

↓

By ingestion of contaminated food

↓

Organism reaches large intestine

↓

Toxin is released in the intestine which is neurotoxic

↓

Neurotoxin acts on peripheral (Nervous system PNS)

↓

Prevents the release of acetylcholine at neural synapse

↓

Which leads to inhibition of neurotransmitter

↓

Difficulty in breathing, swallowing

↓

Respiratory failure, paralysis of facial muscles

ii) Clostridium tetany Pathogenicity

Entry of spare of organism by cut or trauma

↓

Reduction of O_2 leads to germination of spores

↓

Release of tetanospasmin toxin

↓

Toxin binds to ganglioside receptor in neurons

↓

Toxins passes through CNS

↓

Diffuses spinal cord

↓

Contraction of voluntary muscles

↓

Contraction of pharyngeal muscles

↓

Respiratory arrest.

iii) Clostridium perfringes Pathogenicity

Entry if organism to host
↓
Through cut, trauma
↓
Tissue damage with clostridinal spores
↓
Reduction of O_2 tension in necrotized cell
↓
Germination of spores
↓
Release of alpha toxin (or) phospholipase 'c'
↓
If degrades lecithin major components of cell membrane, RBC, platelets.
↓
Further production of gas in affective tissues
↓
Leads to accumulation of gases in wound
↓
Gas gangrene

Clinical symptoms

- Fever
- Lymphadenopathy
- Cellulitis
- Necrotis
- Ischemia

Treatment

- Penicillin, erythromycin, entamycin, ampicilllin.

Influenza virus belongs to the family ORTHOMYXO VIRIDAE. Influenza is an acute infectious disease of respiratory tract which occurs in epidemic and pandemic

Morphology

- Influenza is single standard RNA genome
- It is a enveloped virus.
- This virus encode protein which include haemagglutine and neuraminidase. The spikes are projected from envelop.
- It possess RNA dependent RNA polymerase enzyme.

Haemagglutimin

- It contains trimer of glycoprotein sub unit.
- Each subunit consists of 2 polypeptide chains HA_1 and HA_2.
- It facilitates the attachment of the virus to respiratory epithelial cell.
- It undergoes antigen changes from antigenic drift and antigenic shift.

Neuraminidase

- It is the 2^{nd} glycoprotein spike like structure on the surface of influenza. It is mushroom shaped, it contains box which destroys receptor on the host cell.
- This enzyme cleaves the bond between the viral gene agglutimin and the cell receptor

Cultivation

- The virus can be grown by using monkey kidney cell line.

Pathogenicity

Entry of virus to host by aerosols
↓
Organisms ruptures the respiratory tract
↓
Haemagglutimin attaches to the host epithelial cell
↓
Neuronidase facilitates infection by reducing the viscosity of the mucosal timing respiratory tract
↓
Cells are damaged and shedded
↓
Infection is seen in trachea, bronchus, bronchioles and alveolar region
↓
It results in thickening of alveolar walls
↓
Interstitial infiltration with leucocytes
↓
Formation of Hyaline membrane in lungs
↓
In later stages, infiltration of macrophages is observed.

Clinical symptoms

- Productive cough
- Nasal discharge

- Fever
- Headache
- Pneumonia

Lab Diagnosis

- Throat swab
- Asopharyngeal swab
- Serological test
- Complement fixation test
- Immunoflourescent test

Treatment

- Rimatadine
- Amantadine

Fungal Infections

i) Determatophytoses

They are the most common type of cutaneous fungal infection seen in man and animals affecting skin, hair and nails, They are caused by a group o fungi which have the capacity to invade the keratinized tissue of skin and its appendages and these are collectively known as dermatophytes.

Causative agent

- Trichophyton species – skin, nail, hsit
- Microsporum species – skin, hair
- Epidermatophyton species – skin, nail.

Clinical features

It produces ring worm like infection or Tinea infection.

i) Tinea capitis (scalp)
ii) Tinea corporis
iii) Junea imbricate
iv) T. gladiatorium
v) T. facial
vi) T. cruris
vii) T. mannum
viii) T. pedis
ix) T. ungulum

x) T. barbae

Tinea capitis: This is the infection of the shaft and scalp region.

Tinea corporis: This infection is seen globrous skin of body and the infection may extend to scalp, groin or beard.

Tinea imbriata: These are concentric ring of scaling which spreads out peripherally as Hanuman (or Indian Ring worm).

Tinea gladiatorium: It is a converging infection in Wrestler. It is found in athletics due to playing jsnoy

Tinea faciei: This is dermatophytic infection, skin or face and the infection extends to beard.

Tinea cruris: It is the dermatophytic infection of groin mostly present in men. It involves in the perinanal area and may spread to thigh. In female infection is seen in obese women.

Tinea mannum: This is the infection of the skin of palm of hand.

Tinea pedis: This is the infection of the foot, toes and interdigital web spaces.

Tinea ungulum: It is the dermatophytic infection of nail plate and mostly seen in adult.

Tinea barbae: This is the ring worm infection of beard and mustache areas of the face, it is called barbers MCH.

Lab diagnosis

Specimen: Skin, nail, hair,

- KOH mount
- Lactophenol cotton blue staining.

Treatment

- Imidazole
- Amphotercin B
- White man ointment

Mycetoma

It is chronic localized granuloatize infection skin and subcutaneous tissue. This infection is caused by *Actinomadhura machurae, Nocardia cavivae.* This disease was 1st reported in Madhurai, so called Madura mycosis or Madura foot.

Made of Transmission

Entry of fungal spores by picking of rose thorn which contains fungal spore

↓

Spore enters the intact skin through deep penetrating wound

↓

Spores stark germinating which leads to the formation of hyphae growth

↓

Adjacent cell damage which induces inflammatory reaction.

↓

Formation of pus and fibrin deposit in the infected area

↓

Swelling occurs which is commonly called tumefaction

↓

Tumefaction is due to abcess formation

↓

Abcess formation produces a tunnel known as sinus tract

↓

Sinus tract reaches the superficial structure where the pus in released.

↓

The pus are commonly called sulphur granules which contains fungal mass.

Treatment

- Keto conazole

Aspergillosis

Systemic fungal infection seen in immunocompromise or immunocompetent species. It causes primary pulmonary infection. It is ubiquitous in nature. Aspergillus produces a toxin – AFLA toxin which is responsible to cause food poisoning.

Mode of transmission

Through aerosols.

Pathogenicity

Entry of spores through aerosols

↓

It enters upper respiratory tract and reaches bronchial and pulmonary alveoli

↓

Since the size of the spore is less, is the spore will easily get phagocyte

↓

In case of immunocompromised patient, the spores resist and starts germinating

↓

Which leads to aspergilloma.

Clinical features

Pulmonary Aspergilloses

It is classified into 3 types

- Allergic Aspergilloses – Asthma, Pulmonary eosinophyllria, increased concentration of IgE. It causes allergic thrombo pulmonary aspergillosis.
- Aspergilloma – compact mass of fungal mycelia are often surrounded by dense fibrous wall called fungal ball or aspergilloma. Aspergilloma is 8 – 10 cm in dia meter.
- Invasive aspergillosis – it causes chronic granulomatus diseases.

Treatment

- Itraconozole

- Amphotericin B

iv) Candidiasis

Candidiasis is an opportunistic infection caused by *Candida albicans*

Candidiasis is the common fungal disease found in human affecting mucosa, skin, nail and other organs of the body.

Morphology

Budding yeast cell

Antigenic and Virulent properties of *Candida albicans*

- It produces cytoplasmic and cell wall antigen
- The virulence factors are
- Toxin – eg. Endotoxin
- Enzyme – Proteases, phospholipases, esterases.
- **Adhesion Property** – They adhere to endothelial and epithelial cells of host cell receptors.
- **Complement receptor** – It inhibits phagocytosis by binding to the complement protein.
- **Phenotypic switching**

Predisposing factor:

- Immune compromise patient
- Immuno suppressed patient
- Diabetic patients.

Pathogenicity

Refer microbial pathogenicity, iin fungal as a potential pathogen.

Clinical symptoms

- Oral candidiasis
- Alimentary candidiasis
- Valvo vaginitis
- Occular candidiasis
- Mucocutaneous candidiasis
- Cutaneous candidiasis
- Pulmonary candidiasis

Lab diagnosis

- Gram staining
- Germ tube test position
- Sugar assimilation test
- Sugar fermentation test
- Serological test – ELISA

Treatment

- Canozole
- Amphotericin B

Protozoan Disease: Helminth

i) Filariasis: Causative agent – Wuchereria bancrofti

Morphology

- Adult worm
- Microfilaria

Adult Worm

- Minute
- Whitish Thread like
- Filiariform in shape
- Both anterior and posterior end are trapping.

Microfilaria

- First stage larva
- Covered of hyaline sheath

Third stage larva

Infection form of filarial worm elongated.

Life Cycle

Mosquito bites the human suffering from filariasis

↓

Microfilaria reaches intestine of mosquito

↓

It undergoes changes

↓

First stage larva

↓

Second stage larva

↓

Third stage larva

↓

Third stage larva is locked in salivary glande

↓

Mosquito bites the individual

↓

Ingest 3rd stage larva

↓

Wondering in skin

↓

Reach lymphatic

↓

Rest in lymphatic and lymph node

↓

Develops into adults

↓

Trapped in different region

↓

Lungs

↓

Microfilaria in blood

↓

Bitten by mosquito

↓

Enters salivary gland

↓

Bites healthy individual

↓

Ingest 3rd stage larva

Clinical Features

i) Lympohatic filariasis

- Dilation of lymphatic vessel
- Infection of lymphatic vessel
- Obstruction of lymph node
- Lymph adenopathy

ii) Filarial funiculitis

- Inflammation in the sperm cord

iii) Orchitis

- Characteristic by oedematous testis

iv) Hydrocele

- Hydrocele fluid typically consist of blood clot, fibrin cholesterol crystals and ca particles

Lab Diagnosis

Specimen – peripheral blood smear

↓

Giemsa or leishman staining

Observation

- Observation of 3rd stage larva or mircrofilaria
- Serological diagnosis – ELISA

Treatment

- Diethyl carbamazin

Amoebiasis

- Causative agent : Entamoeba histolytica
- Habitat – Large Intestine

Morphology

- It occurs in 3 stage

- Tropsite
- Precyst
- Cyst

Tropisite

- Invasive form of the parasite
- Actively motile
- Infects large intestine

Precyst

- Stage between trophosite and cyst
- Oral in shape

Cyst

- Infective stage of parasite
- Mature cyst contains 4 nuclei

Mode of transmission – Contaminated food and H_2O.

Life cycle

Man acquires infection by
↓
Ingestion of H_2O and contaminated food
↓
With mature quadrinucleate
↓
Cyst undergoes excystation
↓
Cyst → troposite stage
↓
Troposite undergoes cytoplasmic and nuclear division
↓
Forming eight small metacystic trophosite
↓
Invades large intesting
↓
Troposoites excreted infaces
↓
Encystation
↓

Trophosoite – cyst stage
↓
Cyst excreted in faces
↓
Ingested by host

Pathogenicity

After adherence to the trophosites in the intestinal mucosa
↓
It targets the cell
↓
Resulting in cytolysis
↓
It leads to intestinal amoebiasis
↓
Amoebic ulcer is seen in intesting
↓
Which is in flask shape
↓
Which may be localized or generated
↓
The localized amoebic ulcer are present in ileo caecal region and sigmoidorectal region

Clinical symptoms

- Acute intestinal amoebiasis
- Chronic intestinal amoebiasis
- Extra intestinal amoebiasis
- Pulmnary amoebiasis
- Cerebral amoebiasis
- Hepatio amoebiasis
-

Lab Diagnosis

- Specimen – stool
- Saline mount
- Iodine mount
- Concentration flotation method
- Concentration segmentation method
- Sero diagnosis – Elisa

Treatment

- Metionidazole