Dysentery

*Inflammatory (structural) disorder of colon
*Blood, pus & mucus present in feces
*Pain, fever, abdominal cramps

Enterocolitis

*Inflammation of mucosa of small & large intestine

5. Causative agents (of GIT infections):

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Animal reservoir</th>
<th>Food-borne</th>
<th>Water-borne</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bacteria</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Escherichia coli</em></td>
<td>+?</td>
<td>+ (EHEC)</td>
<td>+ (ETEC)</td>
</tr>
<tr>
<td><em>Salmonella</em></td>
<td>+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td><em>Campylobacter</em></td>
<td>+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td><em>Vibrio cholera</em></td>
<td>-</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td><em>Shigella</em></td>
<td>-</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td><em>Clostridium perfringens</em></td>
<td>+</td>
<td>+++</td>
<td>-</td>
</tr>
<tr>
<td><em>Bacillus cereus</em></td>
<td>-</td>
<td>++</td>
<td>-</td>
</tr>
<tr>
<td><em>Vibrio para-haemolyticus</em></td>
<td>-</td>
<td>++</td>
<td>-</td>
</tr>
<tr>
<td><em>Yersinia enterocolitica</em></td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td><strong>Viruses</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Rotavirus</em></td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><em>Noroviruses (previously known as SRSV or Norwalk-like viruses)</em></td>
<td>-</td>
<td>++</td>
<td>+</td>
</tr>
</tbody>
</table>
VT (verotoxin) is a shiga-like toxin (SLT)

Diseases caused:
Dysentery (bloody diarrhea; resembles shigella dysentery)

Pathogenesis:
invades epithelial cells (by endocytosis) → spreads laterally to adjacent cells → tissue destruction, necrosis & ulceration

Clinical features:
Passage of blood, mucus & WBCs in stool

Lab diagnosis:
*Sereny test (instillation of suspension of freshly-isolated EIEC in eyes of guinea pig → mucopurulent conjunctivitis & severe keratitis)
*Tissue culture (penetration of HeLa/Hep2 cells)

Diseases caused:
*Outbreaks of food poisonings
*(Serotype O157: H7 is most commonly involved)

Pathogenesis:
Attaches to colonic mucosa → releases VT* → VT targets vascular endothelial cells → inhibits protein synthesis (cytotoxicity)

Clinical features:
*Mild diarrhea (bloody) to fatal complications (esp in extremes of age), e.g.
  a) Hemorrhagic colitis (mucosal destruction followed by hemorrhage)
  b) Hemolytic-uremic syndrome (is a triad of renal failure, hemolytic anemia & thrombocytopenia)

Lab diagnosis:
*Broadly, VT demo in feces/culture
*NO sorbitol fermentation in sorbitol McConkey agar (for O157: H7, unlike other E. coli strains)
*Cytotoxic effects on Vero/HeLa cells
*DNA probes (to detect toxins)
**Culture**

*Enrichment medium: selenite F broth
*BA: non-hemolytic colonies
*Sorbitol MA: LF (pink colonies)

**Biochem**

*Catalase, H₂S, urease, indole +ve
*Ferments glucose, lactose, mannitol (+) but NOT sucrose
*Citrate –ve

*LF = lactose-fermenting

---

**SUB-SUB-PART GD 02 (B 8): TREATMENT**

13. Is based on symptoms:
   a) 1°treatment: fluid replacement
   b) 2°treatment: antibiotics (in severe cases with systemic involvement)

---

**SUB-PART GD 02 (C): SALMONELLA**

**SUB-SUB-PART GD 02 (C 1): GENERAL**

14. Diseases caused:

<table>
<thead>
<tr>
<th>Disease</th>
<th>Causative Agents</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enteric fever</td>
<td><em>S. typhi</em> (typhoid)</td>
<td>Exclusively human parasite</td>
</tr>
<tr>
<td></td>
<td><em>S. paratyphi A/ B /C</em> (paratyphoid)</td>
<td></td>
</tr>
</tbody>
</table>
### Food poisoning

<table>
<thead>
<tr>
<th>S. typhimurium</th>
<th>S. enteritidis</th>
<th>Essentially animal parasite which also infects humans</th>
</tr>
</thead>
</table>

### Septicemia

<table>
<thead>
<tr>
<th>S. choleraesuis</th>
<th></th>
</tr>
</thead>
</table>

## SUB-SUB-PART GD 02 (C.2): MORPHOLOGY & GENERAL PROPERTIES

15. General properties (of *S. typhimurium*):

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>General</td>
<td><em>Gram (-) bacilli</em></td>
</tr>
<tr>
<td></td>
<td><em>Motile</em></td>
</tr>
</tbody>
</table>

### Culture

<table>
<thead>
<tr>
<th>Type</th>
<th>Name</th>
<th>Appearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Differential</td>
<td>MA</td>
<td>Colorless, NLF (non-lactose fermenting) colonies</td>
</tr>
</tbody>
</table>
| Selective | Wilson & Blair Bi sulfite medium | *Jet black colonies (due to H₂S production)*  
*S. typhi gives green colonies (NO H₂S production)* |
## SUB-PART GD 03 (I): DISEASES CAUSED BY OTHER VIBRIOS

15. Examples:

<table>
<thead>
<tr>
<th><em>Vibrio...</em></th>
<th>Diseases Caused</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>mimicus</em></td>
<td>(Sporadic) <strong>diarrhea</strong> (in USA)</td>
</tr>
<tr>
<td><em>parahemolyticus</em></td>
<td><strong>Food poisoning</strong> (due to seafood)</td>
</tr>
<tr>
<td><em>algинolyticus</em></td>
<td><strong>Eye, ear, wound</strong> Infections (on contact with sea water)</td>
</tr>
</tbody>
</table>
| *vulnificus* | *Wound* infections (on contact with sea water)  
|              | *Septicemia* (in the immunocompromised) |
PART GD 05: HELICOBACTER PYLORI

1. General features:
   a) Gram (-) spiral rods (coccoid in old cultures)
   b) Motile (has a unipolar tuft of lophotrichous flagella)
   c) Microaerophilic (grows best at 5% O₂)
   d) Associated with gastritis, duodenal & gastric ulcers
   e) Is a risk factor for gastric Ca & is a linked to mucosal-associated lymphoid tissue (MALT) lymphoma
   f) Produces P₆ase, urease, oxidase, catalase, H₂S
   g) NO metabolize sugars/ reduce NO₃
   h) Resembles Campylobacter (but they differ significantly in certain biochem & flagellar characteristics)

2. Pathogenesis:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Source</td>
<td>Gastric mucosa (of humans)</td>
</tr>
<tr>
<td>Transmission</td>
<td>Feco-oral route (ingestion)</td>
</tr>
</tbody>
</table>
| Virulence factors | *Considerable genetic diversity  
*Flagella (motility) 
*Adhesins  
*Urease (for NH₃ production)  
*Toxins [coded by cag (cytotoxin associated gene) & vac (vacuolating cytotoxin gene)] |
| Infection    | *May be transient/ chronic  
*Gastric antrum is the most common colonization site  
*Infection is confined to gastric mucosa (& areas of gastric metaplasia & heterotopia in duodenum) |
PART GD 36: VIRAL DIARRHEA

SUB-PART GD 36 (A): CAUSATIVE AGENTS

1. Adenoviruses (types 40 & 41)
2. Nipah virus
3. Astroviruses
4. Rotavirus
5. Calicivirus (includes norovirus & sapovirus)
6. Hendra virus
7. Coronavirus

SUB-PART GD 36 (B): ROTAVIRUS

8. General:
   a) Family: Reoviridae (has wheel-like appearance; hence, rota)
   b) Double-layered icosahedral capsid
   c) Segmented dsRNA
   d) Contains RNA dependent RNA Pol (human cells no have RNA Pol which c. synthesize mRNA from dsRNA template)
   e) Has outer surface Ag (viral HA): type-specific Ag which elicits Ig production
   f) Non-enveloped
   g) 6 serotypes
**E. histolytica (left) & E. dispar (trichrome staining)**

*Sometimes, RBCs ingested by the parasite are visible (makes differentiation via microscopy much easier)*

<table>
<thead>
<tr>
<th>Specific Diagnosis of Active Infection (Trophozoite Demo)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Motile trophozoites</strong> throwing pseudopodia &amp; containing RBCs found in large #</td>
</tr>
<tr>
<td><strong>Endoplasm</strong> appears <strong>bluish/glassy</strong> appearance</td>
</tr>
</tbody>
</table>

*RBCs & pus cells* are found in fair #

*Charcot Leyden* crystals (diamond-shaped, clear, chromotropic, retractile structures) present in faeces

*Nucleus* not visible (but faint outline may be observed)

*Glycogen mass* not visible

<table>
<thead>
<tr>
<th>Serology</th>
</tr>
</thead>
<tbody>
<tr>
<td>The serological tests become reactive in invasive amebiasis:</td>
</tr>
<tr>
<td>a) IHA</td>
</tr>
<tr>
<td>b) ELISA</td>
</tr>
<tr>
<td>c) LAT</td>
</tr>
<tr>
<td>d) Gel diffusion</td>
</tr>
<tr>
<td>e) Countercurrent Immunoelctrophoresis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Amebic Liver Abscess</th>
</tr>
</thead>
<tbody>
<tr>
<td>*Pus appears <strong>anchovy sauce-like</strong></td>
</tr>
<tr>
<td>*Aspirated pus likely to contain <strong>trophozoites</strong> (detected by direct microscopy)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Extraintestinal Amebiasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>*Specimens: liver/ lung/ brain biopsy samples</td>
</tr>
<tr>
<td>*Subjected to routine <strong>H&amp;E</strong> sections</td>
</tr>
<tr>
<td>*Giemsa-stained touch preparations reveal <strong>trophozoites</strong></td>
</tr>
</tbody>
</table>
**SUB-PART GD 37 (B): GIARDIA LAMBLIA**

8. General features:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Features</th>
</tr>
</thead>
</table>
| General | *Also known as *Giardia intestinalis/ *duodenalis*  
*Is an intestinal *flagellate*  
*Inhabits *duodenum* & upper *jejunum*  
*Frequent cause of *traveller's diarrhea*  
*Divides by *binary fission* |
| Epidemiology | *Occurs in tropics & subtropics (where sanitation is poor)*  
*Commonly infects infants & children aged < 10 in developing countries*  
*Found in HIV patients*  
*Outbreaks can occur by contamination of public drinking supplies/ drinking from rivers & streams* |
| Transmission | *Ingestion* *(of contaminated water/ food)*  
*Person-to-person*  
*Sexual contact (in homosexual♂)* |
| Morphology | **Trophozoite**  
*Pear-shaped (rounded anterior, pointed posterior ends)*  
*Convex dorsal surface*  
*2 rounded nuclei (with central karyosome)*  
*4 pairs of flagellae*  
*Suction disc on ventral aspect*  

| Cyst | *(Is the infective form)*  
*Oval, thick-walled*  
*4 nuclei with several internal fibres* |
5. Life cycle:

- Enter body of cattle during grazing
- Oncospheres liberated from eggs in alimentary canal, pass thru intestinal wall into bloodstream → muscles
- Eggs passed in feces along with gravid segments
  (Eggs are viable for 8 wks in soil & infective only to cattle)
- Segments formed from neck & develop into adult worm
- Sexually mature ♂ & ♀ cross-fertilise & produce eggs (in uterine canal)
- Scolex attaches to mucosal surface with suckers
- Can remain in muscles of cattle for 8 months (& develop further when ingested by humans)
- Develop into infective larvae (cysticercus bovis @ bladder worm)
- Human eating raw/undercooked beef with larvae (cysticercus bovis)
6. Clinical features:

1. Asymptomatic

2. Adult worms can cause...
   a) nausea
   b) abdominal discomfort
   c) chronic indigestion
   d) diarrhea alternating with constipation

7. Lab diagnosis:
   a) Gravid segments & eggs demo in feces
   b) Serology: IHA, IFA, ELISA

*Research on serology for coproAgs are giving promising results

8. Treatment: praziquantel (single dose)

9. Preventive measures:
   a) Proper disposal of human feces
   b) Proper cooking of beef
   c) Inspection in slaughter house for larvae in meat

---

**SUB-SUB-PART GD 38 (A 3): TENIA SOLIUM**

10. Morphology:

| Eggs | [Refer #3 above] |
Severity is based on size & location of cyst

23. Lab diagnosis:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microscopy</td>
<td>Diagnostic puncture of cyst is NOT recommended</td>
</tr>
<tr>
<td>Serology</td>
<td>ELISA, LAT, IHAT, CFT, BFT (bentonite flocculation test), RIA</td>
</tr>
<tr>
<td>Skin test (Casoni test)</td>
<td>*Is an immediate hypersensitivity test&lt;br&gt;<em>0.2 mL of sterile hydatid fluid is injected IM into 1 arm &amp; 0.2 mL of normal saline into the other&lt;br&gt;</em>+ve test gives large wheal (5 cm) reaction with multiple pseudopodia (in 30 min)&lt;br&gt;*Low sensitivity (gives false +ve results)</td>
</tr>
<tr>
<td>Radiology</td>
<td>CT scan, X-ray, etc</td>
</tr>
<tr>
<td>DLC</td>
<td>Eosinophilia (in 20% cases)</td>
</tr>
</tbody>
</table>

24. Treatment
   a) Surgery
   b) Praziquantel, albendazole
31. Epidemiology:
   a) *Schistosoma sp.* is distributed worldwide
   b) Human blood flukes are non-hermaphrodite

32. Features (of *S. mansoni*):

1. Unisexual
2. ♂ shorter than ♀
3. ♂ has *gynecophoric canal* (for holding on to ♀ during copulation)
4. Suckers armed with delicate spines
5. Eggs are *non-operculated* & *fully embryonated* when laid
6. Cercariae have *bifid tails* which can *penetrate unbroken skin* of definitive hosts
7. Adult worm resides in *venous plexus* (of humans)

(L-R): egg, cercariae, adult worm

33. Life cycle (*not required for exams*):
53. Pathogenicity: (ascariasis is caused by) **adult worm** & **migrating larvae**

54. Clinical features:
   
   a) General:

   (Effects) depend mainly on worm-load:
   
   a) Low load: malnutrition, colicky pain
   b) High load: [see below]

   b) Specific:

<table>
<thead>
<tr>
<th>Due to Migrating Larvae</th>
<th>Due to Adult Worm</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Loeffler’s syndrome:</em></td>
<td><em>(Ranges from asymptomatic to severe/fatal infection)</em></td>
</tr>
</tbody>
</table>
   | a) (Transient) eosinophilic pneumonia | *Malnutrition* (interferes with proper digestion/absorption, leading to...)
   | b) Fever, dry cough | a) protein deficiency |
   | *Hypersensitivity* (urticaria/asthma) | b) night blindness (↓ vit A absorption) |
   | *Visceral larva migrans:* | c) growth retardation |
   | a) (Rare) | d) GIT obstruction |
   | b) (Occurs when larvae reach other organs via systemic circulation) | *Hypersensitivities:* |
   | | a) Due to *ascarion* (in body of patient) |
   | | b) Leads to *fever, urticaria, wheezing, angioneurotic edema, conjunctivitis* |
   | | *Restless wanderers* (worms tend to probe & may crawl out of mouth/nostrils) |

55. Complications:

1. **Biliary ascariasis** (most common)
2. Intestinal **obstruction**/perforation
3. Appendic-/cholecyst-/pancreat-/periton-itis
66. General

1. Found **worldwide** (but more prevalent in tropical regions of Africa, Asia & South America)

2. Is the **smallest** nematode infecting humans (length: 2 – 3 mm)

3. Adult fertilized ♀ lives buried under **small intestine mucosa**

4. Parasitic ♂ are **shorter & broader** (than ♀) & no have **penetrating power**

67. Morphology:

a) *Eggs:*

   *(In gravid ♀, eggs are conspicuous within its body)*

   *Thin shell, **transparent & oval**

   *(As soon as the eggs are laid), larvae are **ready to hatch**

   *Ovoviviparous

b) *Larvae:*


<table>
<thead>
<tr>
<th>Rhabditiform</th>
<th>Filariform</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Commonly seen in stool specimen of patients</em></td>
<td><strong>Highly infectious</strong></td>
</tr>
<tr>
<td><em>Length: 200 – 300 μm</em></td>
<td><em>Longer (630 μm)</em></td>
</tr>
<tr>
<td><em>Short mouth</em></td>
<td><em>Short mouth</em></td>
</tr>
<tr>
<td><strong>Double bulb esophagus</strong> [<em>indicated in photo below</em>]</td>
<td><em>Long, cylindrical esophagus</em></td>
</tr>
<tr>
<td></td>
<td><em>Notched tail</em></td>
</tr>
</tbody>
</table>
71. Treatment & preventive measures:

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Preventive Measures</th>
</tr>
</thead>
</table>
| - Thia\[bendazole (25 mg/kg b.d. for 2 – 3 days)  
- In hyperinfection, treatment shud continue until larvae are absent (in specimens)  
- Ivermectin (in chronic cases) | - Proper disposal of human waste  
- Avoid contact with feces-contaminated soil  
- Treatment of ALL diagnosed cases (to prevent spread of infection to others) |

**SUB-SUB-PART GD 38 (C.4): TRICHURIS TRICHURA**

72. General:

a) Common name: whipworm

b) Habitat: cecum (most common), appendix, colon (less common)

c) More common in warm, moist regions

73. Morphology:

**EGG**

- *Barrel*-shaped (with mucus plug at each pole)
- *Shell*: yellow-brown (bile stained)
- *Plugs*: colourless
- *Contains unsegmented ova* when freshly passed
- *Floats* in saturated NaCl solution
- *Not infective*

**ADULT WORM**

- *Whip*-shaped, white
- *Anterior part*: thin, hair-like, buried in colon mucosa
- *Posterior part*: thick, stout, extends into lumen, contains reproductive tract
- ♂: shorter, coiled posterior end
- ♀: longer, comma/arc-shaped posterior end
PART GD 39: VIRAL HEPATITIS

SUB-PART GD 39 (A): HEPATITIS A

1. General properties of Hep A virus (HAV @ hepatovirus):
   a) Family: picornaviridae
   b) Is a typical enterovirus (ev 72)
   c) Icosahedral nucleocapsid
   d) ssRNA
   e) Non-enveloped
   f) Single serotype

2. Transmission:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reservoirs</td>
<td>Human</td>
</tr>
<tr>
<td>Route</td>
<td>Fecal-oral</td>
</tr>
<tr>
<td>Mode</td>
<td>*Contaminated water/ food (oysters grown in polluted water &amp; eaten raw)</td>
</tr>
<tr>
<td></td>
<td>*Poor hand hygiene</td>
</tr>
<tr>
<td></td>
<td>*Oral sex (♂ homosexuals)</td>
</tr>
<tr>
<td></td>
<td>*Blood (very rare due to low viremia &amp; absence of chronic hep A)</td>
</tr>
<tr>
<td>Others</td>
<td>*Most frequently infects children</td>
</tr>
<tr>
<td></td>
<td>*Quarantine of patients is NOT effective (infection occurs 2 weeks prior to appearance of symptoms)</td>
</tr>
</tbody>
</table>
11. Lab diagnosis (ELISA):

<table>
<thead>
<tr>
<th>Presence of...</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ag</td>
<td>Ig</td>
</tr>
<tr>
<td>HBs</td>
<td>Current infection</td>
</tr>
<tr>
<td>HBe</td>
<td>Active infection</td>
</tr>
<tr>
<td>HBs</td>
<td>Recovery/ immunity</td>
</tr>
<tr>
<td>HBe</td>
<td>Inactive virus (generally)</td>
</tr>
<tr>
<td>HBc</td>
<td>Present/ past infection</td>
</tr>
<tr>
<td>HBV DNA</td>
<td>Degree of viral activity</td>
</tr>
</tbody>
</table>

*Supercarriers can be converted to simple carriers by administering HBe Ig*
12. Prevention:
   a) Vaccines:
      - **Subunit Vaccine**
        - Consists of HBs Ags
        - Produced in yeast by recombinant DNA technology
        - 95% seroconversion rate in healthy adults
        - Indicated for those who are frequently exposed to blood/blood products, & travellers to endemic areas
        - 3-dose regimen within 6 months (no boosters)
      - **Mixed Immunity**
        - HBlg (high titre of HBs Ig) gives passive immunity
        - Combined (passive-active) immunization is given to needlestick injuries (from HBs Ag +ve patient)/ newborns of HBs Ag +ve mother
   b) All blood for transfusion to be screened for HBs Ag
   c) Anyone with history of hepatitis will NOT be allowed to donate blood

**SUB-PART GD 39 (C): HEPATITIS**

13. General:
   1. Family: Flaviviridae
   2. +ve ssRNA
   3. Icosahedral nucleocapsid, enveloped
   4. 6 genotypes (with multiple subgenotypes)
   5. Genetic variation results in hypervariable envelope glycoproteins:
      a) Due to high mutation rate & absence of proofreading
      b) Multiple subspecies (quasispecies) are present in blood at the same time
CHAPTER HE: ANTHRAX

1. Is the most fatal agent used for bioterrorism

2. Causative agent: *Bacillus anthracis*

3. General properties (of *B. anthracis*):

   1. **Gram (+) bacilli**
   2. Arranged in chains, with truncated ends (bamboo stick appearance)
   3. **Spores** survive in soil for a very long time & are heat-resistant
   4. **Aerobes/ facultative anerobes**
   5. Non-motile
   6. Capsule is composed of polypeptides (mainly D-Glu)
   7. **Spore-forming**

4. Transmission:
   a) Routes of infection: **trauma** (via wounds), **inhalation**, **ingestion**
   b) Primarily a serious disease of herbivores (cattle & sheep)
   c) Infection of humans is due to contact with infected animals (or their products)

5. Virulence factors:

<table>
<thead>
<tr>
<th>Factor</th>
<th>Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Capsular polypeptides</td>
<td>Antiphagocytic</td>
</tr>
</tbody>
</table>
Anthrax toxin is a 3-component exotoxin:

- a) PA (protective Ag)
- b) EF (edema factor)
- c) LF (lethal factor)

6. Pathogenesis:

- All 3 factors are required for pathogenesis

7. Types of human anthrax:

<table>
<thead>
<tr>
<th>Type</th>
<th>Pathogenesis</th>
<th>Clinical Features</th>
</tr>
</thead>
</table>
| Cutaneous (hide porter's disease) | *Incubation period: 9 hrs – 2 weeks  
*Pathogenesis:  
Spores in soil  
Entry via skin lesion  
Formation of a ring of vesicles  
Marked edema, painless ulcer  
Black eschar (malignant pustule) | *High fever  
*Extensive edema  
*Systemic anthrax (in untreated cases) → meningitis  
*Bacteremia, death |
**Culture**

*Christensen’s urea slant:*

a) Gives +ve results in 5 mins (in case of *B. suis* / some strains of *B. melitensis*)
b) Others give +ve results within a few – 24 hrs
c) Sent to reference lab for confirmation

**Biochem**

*Catalase, oxidase, urease +ve*

*Non-hemolytic*

**Serology**

*↑ IgM during 1st week, peaks at 3 months, persists during chronic stage*

*↑ IgG during 3rd week, peaks at 6 – 8 weeks, high levels during chronic disease*

1: 160 titre of convalescent phase serum is diagnostic

*This organism must be dealt with in a biological safety cabinet*

6. **Treatment:**

1. Tetracycline + streptomycin (for 3 weeks)
2. Rifampicin + doxycycline (for 6 weeks)

*These drugs provide symptomatic relief for a few days*

*However, due to intracellular nature (of Brucellae), treatment is prolonged*
11. General:
   a) Severe disease
   b) Occurs more frequently in children from endemic areas
   c) Initial symptoms same as classical dengue
   d) Shock & hemorrhage into GIT & skin
   e) Mortality up to 10%

12. Pathogenesis:

13. Lab diagnosis:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
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</tbody>
</table>
7. Life cycle:

8. Pathogenesis:

- Presence of adult worms in lymph nodes/ vessels
- **Enlarged nodes** (Wuchererian/ Bancroftian filariasis)

9. Manifestations:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incubation period</td>
<td>≥ 1 year</td>
</tr>
<tr>
<td>Causes</td>
<td><em>Mechanical irritation</em> (due to movement of adult parasite)</td>
</tr>
<tr>
<td></td>
<td><em>Metabolites liberation</em></td>
</tr>
<tr>
<td></td>
<td><em>Toxic products</em> absorption (from dead worms)</td>
</tr>
<tr>
<td></td>
<td><em>2° bacterial infection</em> (the worms may harbor bacteria)</td>
</tr>
</tbody>
</table>
Microfilariae detection

*QBC (with acridine orange)
*DEC provocation test (DEC is given to provoke microfilariae into showing themselves)

Adult worm detection

Immunodiagnosis

Anti-filarial Igs

Molecular diagnosis

Gene probes

11. Treatment:
   a) DEC (for microfilariae)
   b) Doxycycline (for Wolbachia infections)
7. Pathogenesis & clinical features:

- Arthropod bite
- Spirochete multiples in tissues
- Incubation period: 2 – 10 days

Fever 1.0
* Sudden onset
* Borrelia abundant in blood (bacteremia)
* Subsides after 4 – 5 days

Afebrile period
* Lasts 10 days
* No Borrelia in blood
* Antigenic structure is altered during this time

3 – 10 relapses (Borrelia reappear in blood)

8. Lab diagnosis:

<table>
<thead>
<tr>
<th>Aspect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood exam</td>
<td>* Wet film, under dark ground microscopy (no stain)</td>
</tr>
<tr>
<td></td>
<td>* Smeared with Giemsa/silver</td>
</tr>
<tr>
<td></td>
<td>* Spirochetes seen</td>
</tr>
<tr>
<td>Culture</td>
<td>(Too difficult &amp; unreliable)</td>
</tr>
<tr>
<td>Serology</td>
<td>(False +ve results are common due to presence of other Borrelia species, &amp; also due to antigenic variation)</td>
</tr>
<tr>
<td>Animal inoculation</td>
<td>Mice intraperitoneal inoculation → Borrelia multiply &amp; appear in blood</td>
</tr>
</tbody>
</table>
9. Treatment & preventive measures:

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Preventive Measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tetracycline</td>
<td>Avoid/ eradication of arthropod vectors</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td></td>
</tr>
<tr>
<td>Penicillin</td>
<td></td>
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<tr>
<td>Erythromycin</td>
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</tbody>
</table>

PART IF 03: LYME DISEASE

10. Causative agent: B. burgdorferi

11. General properties:
   a) Motile spirochetes (can be seen via dark field microscopy)
   b) Stains with Giemsa & silver
   c) Routine culture methods are usually –ve

12. Transmission:
   1. Tick (Ixodes spp.) bite
   2. Reservoirs: mammals
   3. Nymphs transmit infection
   4. Tick must feed (on skin) for 24 – 48 hrs for infection to be successful
13. Pathogenesis:

Tick bite

**Spread** of organism from bite site → **surrounding skin**

**Dissemination** to various organs via blood (**bacteremia**)  
Reaches **heart, joints & CNS**

14. Clinical features:

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
</table>
| 1     | *Erythema chronicum migrans*  
*Spreading, non-pruritic, circular red rash (clear centre at bite site)*  
*Painless, flu-like symptoms*  
*2º skin lesions frequently occur*  
*Arthralgia (but NO arthritis)* |
| 2     | *(Occurs weeks – months later)*  
*(Disseminated infection)*  
*Fever, headache, myalgia, arthralgia*  
*Myocarditis → heart block*  
*Acute aseptic meningitis, neuropathies*  
*(Enters another latent phase)* |
| 3     | *(Presistent infection)*  
**Arthritis**  
*Progressive CNS diseases*  
*Death (if untreated)* |

*Is a progressive disease*
1. Fever = abnormal ↑ in body's temperature (38°C)

2. Types of fever:
   a) Above normal temperature over 24 hrs, swings of > 1°C (e.g. pyogenic infections, abscess)
   b) ↑ temperature over 24 hrs, swings of < 1°C (e.g. typhoid, typhus)

3. Sources of infection:
   a) Exogenous pyrogens: LPS (endotoxin)
   b) Endogenous pyrogens: IL-1 (in phagocytes)

4. Mechanism of fever:

   MICROBES/TOXINS/OTHER CYTOKINE INDUCERS (ENDOGENOUS PYROGENS) ↓
   PYOGENIC CYTOKINES (IL-1/6, TNF) FROM MACROPHAGES ↓
   VIA CIRCULATION ↓
   ANTERIOR HYPOTHALAMUS ↓
   
   CHANGE IN TEMPERATURE SET POINT ↓
   ALTERATION IN AUTONOMIC HEAT LOSS/PRESERVATION MECHANISMS ↓
   FEVER

PART ZA 02: PUO PROPER

5. Fever continuing for ≥ 2 – 3 weeks, diagnosis uncertain despite routine investigations