Shigellae

Shigella species are the causative agents of bacillary dysentery (enterocolitis of humans)

General characteristics of Shigellae

- Gram negative rods, non-motile, non-sporing and non-capsulated
- Aerobic and facultative anaerobes and can not grow on simple media
- All are non lactose fermenter except for Sh.sonnei (late lactose fermenter), but ferment other sugars producing acid only.
- They are urease negative, gelatin not liquefied and H₂S not produced.
- IMViC
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- Manitol reactions are important because they distinguish group A strains (do not ferment manitol) from groups (B, C & D) which ferment manitol.
- Ornithin decarboxylase test also important in differentiation between Shigella subtypes (all are negative only for subgroup D).
Subgroups of Shigella

>40 serotypes ← classified according to their biochemical & serological criteria:

1. Group A (Sh.dysenteriae) → 12 serotypes
2. Group B (Sh.flexneri) → 1-6 serotypes
3. Group C (Sh.boydii) → 18 different serotypes
4. Group D (Sh.sonnei) ← antigenically homogeneous but present in 2 forms

Antigenic structure

The somatic O Ag of Shigella (LPS) and their serologic specificity depends on cell wall polysaccharide.

Toxins:

A. Endotoxin: Toxic polysaccharide released during autolysis causing irritation of bowel wall.
B. Exotoxin: An antigenic protein released by Sh.dysenteriae type 1 (Shiga bacillus). Like E.coli verotoxin, act on gut → diarrhea and on CNS (Neurotoxin) → meningism & coma.

Pathogenesis & pathology

Shigellae cause disease exclusively in GIT, it is highly communicable disease, transmitted by feco-oral rout with low dose (100 organisms). Although some strains produce
enterotoxin but invasion is the critical factor in pathogenesis. Blood stream invasion is rare.

**The pathologic process of bloody diarrhea (dysentery)**

Invasion of mucosal epithelial cells of large intestine and terminal ileum by induce phagocytosis $\rightarrow$ micro-organisms multiply within the cytoplasm and pass to adjacent cells $\rightarrow$ microabscesses of the wall $\rightarrow$ necrosis, superficial ulceration, bleeding and pseudomembrane formation (fibrin, leukocytes, cell deprivs, necrotic tissues and bacteria) $\rightarrow$ then subsides by granulation and formation of scar tissues.

**Clinical findings**

Incubation period of 1-4 days $\rightarrow$ symptoms begin with fever and abdominal camps $\rightarrow$ watery diarrhea caused by action of exotoxin on small intestine then diarrhea will be less liquid containing blood and mucus.

Severity of disease depend on 2 factors:

- Species of shigella $\rightarrow$ *Sh.dysenteriae* is the most sever

  $\rightarrow$ *Sh.sonnei* is the milder

- age of patient (children and elderly people being the most severely affected)
The diarrhea resolves within 2-3 days but in sever cases antibiotics can shorten the course of attack.

-Anti bodies appear after recovery but not protective. Some remains as carriers shedding the bacilli with their stool.

**Laboratory Diagnosis**

1-Specimens, stool or rectal swab → culture.

2-Cultured on same media used for isolation of Salmonella → non lactose fermenter (NLF) but no H₂S production and then identified by biochemical tests and agglutination produced by mixing with specific antisera.

3-Serum specimen may be done for detection of agglutinins, but not diagnostic.

**Treatment**

1-Fluid and electrolyte replacement.

2-Antibiotic in sever cases, usually after sensitivity test because resistance may develop by plasmid.

3-Anti-peristaltic drugs are contraindicated in shigellosis ← they prolong excretion of micro-organisms.