Vibrio

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VIBRIO – Overview

Diseases Caused

- Vibrio cholerae → *Cholera* (severe watery diarrhea)
- Vibrio parahaemolyticus → Diarrhea after eating raw/undercooked seafood
- Vibrio vulnificus → Cellulitis and sepsis, especially after wound exposure to seawater

Important Properties of Vibrio

General Characteristics

- Shape: Curved, comma-shaped
- Gram status: Gram-negative rods
- Oxygen requirement: Facultative anaerobes
- Motility: Motile with polar flagella

Cholera-Causing Strains

- Only O1 and O139 serogroups cause epidemic cholera
    - These produce cholera toxin
    - Toxin gene carried by lysogenic bacteriophage

Serogroup Details

- O1 serogroup:
    - Biotypes: *Classic* and *El Tor*
    - Serotypes: *Ogawa, Inaba, Hikojima*
        - Biotypes = biochemical differences
        - Serotypes = antigenic differences
- O139 serogroup:
    - First appeared in 1992, caused epidemics in India & Bangladesh
    - Identified by O139 polysaccharide antigen

Non-O1 Strains

- Do not cause cholera, but may cause milder diarrhea
- Lack cholera toxin gene (not lysogenized)

Marine Vibrios

- V. parahaemolyticus & V. vulnificus
    - Found in warm, salty seawater
    - Halophilic: Require high NaCl concentration

Vibrio cholerae – Pathogenesis & Epidemiology

Transmission

- Fecal-oral route, mainly via contaminated water & food
- Common in areas with:
  - Poor sanitation
  - Overcrowding
  - Malnutrition
  - Inadequate medical care
- Asymptomatic human carriers:
  - In incubation or convalescence
- Animal reservoirs: Marine shellfish (e.g., shrimp, oysters)

Epidemics

- 1960s–70s: Began in Southeast Asia, spread globally
- 1991: Outbreak in Peru, spread to Central & South America
- Most isolates were:

- El Tor biotype, usually Ogawa serotype

High-Yield Table: Pathogenesis Comparison

Organism	Type of Pathogenesis	Typical Disease	Site of Infection	Main Therapy
Vibrio cholerae	Toxigenic	Watery diarrhea	Small intestine	Fluid replacement
Campylobacter jejuni	Inflammatory	Bloody diarrhea	Colon	Antibiotics
Helicobacter pylori	Inflammatory	Gastritis, peptic ulcer	Stomach, duodenum	Antibiotics

Pathogenesis of Cholera

Essential Requirements

- Colonization of small intestine

- Secretion of enterotoxin (choleragen)

Resistance to Infection

- Large bacterial dose required (due to sensitivity to stomach acid)

- Increased susceptibility:

    - Patients on antacids
    - Gastrectomy patients (↓ stomach acid)

Mucosal Adherence

- Bacteria adhere to brush border of intestinal epithelium

- Use of mucinase enzyme:

    - Degrades protective mucosal glycoprotein layer
    - Facilitates colonization

Cholera Toxin (Choleragen)

Structure

- AB exotoxin

- A subunit (active) → Enters cytosol, catalyzes ADP-ribosylation
- B subunit (binding) → Pentamer; binds GM1 ganglioside receptor

Mechanism of Action

1. B subunit binds GM1 ganglioside on enterocyte surface

2. A subunit enters cell → ADP-ribosylates Gs protein

3. Gs protein becomes locked in active state

4. Adenylyl cyclase is persistently activated

5. ↑ cAMP → activates protein kinase A (PKA)

6. PKA phosphorylates ion channels

    - Causes efflux of Na^+, Cl^-, HCO_3^-, K^+
    - Leads to massive water loss into gut lumen

Result

- Profuse watery diarrhea

- No RBCs or WBCs in stool

Virulence Factors

- CTX bacteriophage (ssDNA):
    - Carries genes for cholera toxin
    - Causes lysogenic conversion of non-toxigenic strains
    - Uses toxin-coregulated pili (TCP) as receptor for phage attachment

Non-O1 V. cholerae

- Occasionally causes diarrhea
- Linked to consumption of shellfish
- Does not cause cholera due to lack of cholera toxin

Clinical Findings

Hallmark Symptom

- Watery diarrhea in large volumes
    - Referred to as "rice-water stools"
    - No blood or pus
    - Odorless, cloudy appearance

Other Features

- No abdominal pain

- Symptoms due to fluid and electrolyte loss

    - → Dehydration
    - → Renal failure
    - → Cardiac failure
    - → Acidosis (due to bicarbonate loss)
    - → Hypokalemia (due to K^+ loss)

Mortality

- Up to 40% without treatment

- With fluid/electrolyte replacement, disease is self-limited (resolves in ~7 days)

Laboratory Diagnosis of Cholera

Diagnostic Approach

- During epidemics:

    - Diagnosis is usually clinical
    - Laboratory testing not always needed

- In endemic areas or for detecting carriers:

- Use of selective media (not commonly used in U.S.)

Stool Culture

- Sample: Diarrheal stool
- Media:
  - MacConkey's agar:
    - Colorless colonies (due to slow lactose fermentation)
  - TCBS (Thiosulfate-Citrate-Bile-Sucrose) agar *(high yield)*:
    - Yellow colonies (due to sucrose fermentation)
  - TSI (Triple Sugar Iron) agar:
    - Acid slant / acid butt
    - No gas or H_2S
- Biochemical Tests:
  - Oxidase-positive (differentiates from Enterobacteriaceae)
- Serologic Confirmation:
  - Agglutination test with polyvalent O1 or non-O1

antisera

- Retrospective Diagnosis:
    - Rising antibody titers in acute vs. convalescent-phase sera

Treatment of Cholera

Mainstay of Therapy

- Prompt fluid and electrolyte replacement:
    - Oral Rehydration Therapy (ORT)
    - IV fluids for severe dehydration
- Addition of glucose:
    - Enhances water and Na^+ absorption via sodium-glucose co-transport

Antibiotics (Adjunctive)

- Not essential but beneficial:
    - Shorten duration of diarrhea
    - Reduce shedding of organism
- Commonly used: Tetracycline

◈ Prevention of Cholera

◈ Public Health Measures

- Ensure clean water and safe food
- Sanitation, hygiene, and sewage control

◈ Vaccination

- Vaxchora (U.S.):
  - Live attenuated oral vaccine
  - For travelers to O1 endemic regions
- Killed oral vaccines:
  - Available in cholera-prone countries
  - Provide short-term protection

◈ Chemoprophylaxis

- Tetracycline effective in close contacts
  - But not effective in stopping epidemics

◈ Carrier Control

- Early detection and treatment of carriers helps limit outbreaks

2. Vibrio parahaemolyticus

General Features

- Marine halophilic organism (requires high NaCl)
- Transmission: Ingestion of raw or undercooked seafood (esp. shellfish like oysters)
- Common in: Japan (due to raw fish consumption)
- Outbreaks reported on Caribbean cruise ships

Pathogenesis

- Not fully understood
- Produces an enterotoxin similar to cholera toxin
- May show limited mucosal invasion

Clinical Features

- Watery diarrhea (mild to severe)
- Nausea, vomiting, abdominal cramps, fever
- Self-limiting illness: lasts ~3 days

Laboratory Diagnosis

- Culture media:
    - Thiosulfate-citrate-bile salts-sucrose (TCBS) agar
    - Tellurite-taurocholate-gelatin agar

Differentiating Feature

- Grows in 8% NaCl (halophilic)
    - 💡 *V. cholerae cannot grow in such high salt*

Treatment

- No specific treatment required
    - Illness is mild and self-limiting

Prevention

- Proper refrigeration and cooking of seafood

3. Vibrio vulnificus

General Features

- Marine halophilic organism
    - Found in warm saltwater (e.g., Caribbean Sea)

Mode of Infection

- Wound exposure to seawater or shellfish → Cellulitis
- Ingestion of raw seafood (e.g., oysters) → Sepsis
- High risk in:
    - Immunocompromised individuals
    - Patients with chronic liver disease (e.g., cirrhosis)

Clinical Features

- Severe cellulitis (esp. in shellfish handlers)
    - Painful skin wounds
- Rapidly fatal septicemia
    - Often presents with hemorrhagic bullae

Treatment

- Doxycycline is the drug of choice