HIGH-YIELD Q&A: VIBRIO, AEROMONAS, CAMPYLOBACTER, AND HELICOBACTER

GENERAL CONCEPTS	
1. What are *Vibrio*, *Aeromonas*, *Campylobacter*, and *Helicobacter*?	• They are ***Gram-negative rods*** that are widely distributed in nature. [1]
2. Where are these organisms typically found?	• ***Vibrio***: marine & surface waters; ***Aeromonas***: fresh & brackish waters; ***Campylobacter***: animals (especially domesttcated); Helicobacter: GI & hepatobiliary tracts of humans & other mammals, chickens & wild birds. [1]
3. Which of these organisms causes cholera?	• ***Vibrio cholerae*** via an **enterotoxin** → profuse watery **diarrhea**, rapid dehydration, death. [1]
4. Which organism is a common cause of enteritis in humans?	• ***Campylobacter jejuni***. [1]
5. Which organism is associated with gastritis and duodenal ulcer disease?	• ***Helicobacter pylori***. [1]
THE VIBRIOS (GENERAL)	
6. What are the general characteristics of **vibrios**?	• **Comma-shaped**, **facultatively anaerobic** rods; catalase & oxidase positive; **motile** by polar flagella. [3]
7. What are the growth requirements of **vibrios**?	• Grow at **14–40°C**, pH **8.5–9.5**; require **NaCl** for growth (**halophilic**). [3]
8. Which ***Vibrio* serogroups** cause cholera?	• ***V. cholerae* serogroups O1 and O139**. [3]
9. Which ***Vibrio* species** cause wound infections, sepsis, or **gastroenteritis**?	• ***V. parahaemolyticus*** and ***V. vulnificus***. [3]
10. How is **cholera transmitted**?	• Associated with poor sanitation, direct contact with or consumption of **contaminated water and/or food**. [3]
VIBBIO CHOLERAE	
11. What is the **morphology** of ***V. cholerae***?	• Comma-shaped, curved, motile rod. [4]
12. On which **media does *V. cholerae* grow well**?	• Routine media and selective media such as **TCBS agar**. [4]
13. How **does *V. cholerae* appear on TCBS agar**?	• Yellow colonies (sucrose fermenter). [4]
14. How do *V. parahaemolyticus* and ***V. vulnificus* appear on TCBS agar**?	• **Green colonies** (non–sucrose fermenters). [4]
15. When should stool specimens be collected for suspected cholera?	• **Early** in the course of diarrheal illness. [4]
16. Which **carbohydrates does *V. cholerae* ferment**?	• Ferments **sucrose** and mannose, but not arabinose; **oxidase positive**; grows on most agar media without additional salt. [5]
17. How many **O** serogroups does ***V. cholerae* have**?	• ~200 O serogroups; serogroups **O1** and **O139 cause epidemic/pandemic cholera**; **non-O1/non-O139 strains can cause cholera-like diarrhea**. [5]
18. **What are the serotypes of *V. cholerae* O1**?	• **Ogawa**, **Inaba**, and **Hikojima**. [5]
19. What **biotypes** of **epidemic *V. cholerae* O1 exist**?	• **Classic** and **El Tor**; **El Tor** produces hemolysin and is resistant to **polymyxin B**. [5]
20. Which ***Vibrio* strains produce acidic polysaccharide** capsules?	• **O139**, non-O1 ***V. cholerae*** strains, and ***V. vulnificus***; not ***V. cholerae* O1**. [5]
21. What is the nature of the ***V. cholerae* enterotoxin**?	• **Heat-labile enterotoxin** with **A** and **B** subunits; **A1 increases intracellular cAMP** → prolonged **hypersecretion of water and electrolytes**. [6]
PATHOGENESIS & CLINICAL FEATURES OF CHOLERA	
22. To whom is *V. cholerae* pathogenic?	• Only **humans**. It grows in association with **copepods** and zooplankton. [6]
23. What are the likely sources of infection?	• Contaminated food and water. [6]
24. What affects the infectious dose of *V. cholerae*?	• **Persons with achlorhydria** or on **PPI** require lower infectious dose (10^3 vs 10^{20}). [6]
25. What is the **incubation period of cholera**?	• 12 hours to 3 days (depends on size of inoculum). [7]
26. What are the **characteristic symptoms**?	• Sudden nausea & vomiting → **profuse watery diarrhea** with abdominal cramps; **"rice water stool"**. [7]
27. How severe can **fluid loss** be?	• Up to **1 L/hour (20–30 L/day)**. [6,7]
28. What are the **complications and mortality if untreated**?	• **Dehydration** → **shock**, **acidosis**, **death**; mortality **25–50%** if untreated, reduced to ~1% with **early fluid replacement**. [7]
29. Is there **immunity after an attack of cholera**?	• **Yes**, an attack is followed by **immunity** to reinfection; duration & degree unknown. [7]
30. Are vibriocidal or **antitoxin antibodies protective**?	• **Vibriocidal antibodies** (titer ≥1:20) associated with protection against **colonization** and disease; **antitoxin antibodies** are **not** protective. [7]
LABORATORY DIAGNOSIS OF CHOLERA	
31. What **specimens are used**?	• **Stool**, collected early in illness; inoculate within 2–4 h (use Cary-Blair if delayed). [8]
32. What is seen on **microscopy**?	• **Dark-field** or phase-contrast: **"shooting star" motiltiy** with polyvalent O1 antisera suggests ***V. cholerae* O1**. [8]
33. How is ***V. cholerae* cultured**?	• Grows well on most agar; rapid growth in **alkaline** peptone broth or water with 1% NaCl (pH 8.5) or on **TCBS agar**. [8]
34. How is ***V. cholerae* confirmed in the lab**?	• Slide agglutination with anti-O group 1 or 139 antisera and biochemical reaction patterns. [8]
TREATMENT OF CHOLERA	
35. What is the most important treatment?	• **Water and electrolyte replacement** to correct severe dehydration and salt depletion. [10]
36. **What is the role of antiblotics**?	• Appropriate **antimicrobial therapy** reduces duration and amount of shedding. [10]
OTHER IMPORTANT ORGANISMS	**OTHER IMPORTANT ORGANISMS**
37. What illness is caused by *V. parahaemolyticus* and how is it acquired?	• **Gastroenteritis** (diarrhea, abdominal cramps, nausea, vomiting, fever) after eating raw or underooked seefood (especially shellfish). Self-limited (2-3 days). [11]
38. **What infections are caused by *Aeromonas* species**?	• **Gastroenteritis and wound infections**; may cause bacteremia in immunocompromised patients. Found in fresh/brackish water (exposure via wounds or ingestion). [1]
39. **What disease is caused by *Campylobacter jejuni***?	• **Enteritis** with **fever**, **abdominal** cramps, diarrhea (often **bloody**); acquired from undercooked poultry, unpasteurized milk, or animal contact. [1]
40. What **diseases are associated with *Helicobacter pylori***?	• **Chronic gastritis, duodenal & gastric ulcers**; long-term risk of gastric adenocarcinoma. [1,12,13]
41. How is ***H. pylori* diagnosed**?	• **Gastric biopsy for histology or culture** (histology most sensitive); blood for antibodies; stool for *H. pylori* antigen. Histology shows curved/spiral organisms (Giemsa/silver/IHC stains). [12]
42. What are the **clinical features of *H. pylori* infection**?	• **Upper GI** symptoms (nausea, pain ± vomiting, fever); symptoms last <1–2 weeks; colonization may persist for years or lifelong. [13]

Vibrio spp.

Q1. What is the shape and oxygen requirement of Vibrio species?

A1. Comma-shaped, facultatively anaerobic rods; catalase and oxidase positive; most are motile via polar flagella .

Q2. Which Vibrio serogroups cause epidemic cholera?

A2. V. cholerae O1 and O139 .

Q3. What is the hallmark clinical feature of cholera?

A3. Profuse watery diarrhea (“rice-water stools”), rapid dehydration, and potential shock; incubation 12 hours to 3 days .

Q4. What is the main pathogenesis mechanism of V. cholerae?

A4. Heat-labile cholera enterotoxin (A/B subunits) increases cAMP → massive secretion of water and electrolytes; attaches to microvilli; mucinases and endotoxin contribute .

Q5. How is V. cholerae diagnosed?

A5. Stool culture on TCBS agar (yellow colonies if sucrose-fermenting), enrichment in alkaline peptone broth; slide agglutination with O1 or O139 antisera .

Q6. What is the main treatment for cholera?

A6. Aggressive fluid and electrolyte replacement; antibiotics (tetracycline, doxycycline, TMP-SMX, fluoroquinolones; erythromycin/azithromycin in children/pregnant women) shorten duration of diarrhea and reduce shedding .

Q7. How do V. parahaemolyticus and V. vulnificus typically infect humans?

A7. V. parahaemolyticus: raw/undercooked seafood → acute gastroenteritis; V. vulnificus: raw oysters or seawater wound exposure → septicemia, cellulitis, necrotizing fasciitis, especially in liver disease or immunocompromised patients .

———

Aeromonas spp.

Q8. Where is Aeromonas commonly found and what diseases does it cause?

A8. Fresh and brackish water; causes gastroenteritis (watery to dysentery-like), wound infections, cellulitis, sepsis in immunocompromised patients .

Q9. What are the main human-associated Aeromonas species?

A9. A. hydrophila, A. caviae, and A. veronii biovar sobria .

Q10. What is the preferred treatment for severe Aeromonas infections?

A10. Third-generation cephalosporins, aztreonam, carbapenems, aminoglycosides, fluoroquinolones; gastroenteritis is usually self-limited without antibiotics .

———

Campylobacter spp.

Q11. What are the optimal culture conditions for Campylobacter?

A11. Microaerobic (5–7% O_2, 10% CO_2), 36–42°C (C. jejuni: 42°C), selective media like Skirrow's medium; colonies colorless or gray, non-hemolytic .

Q12. Which tests distinguish C. jejuni from other Campylobacter?

A12. Positive oxidase and catalase; hippurate hydrolysis test distinguishes C. jejuni from C. coli .

Q13. How is Campylobacter infection transmitted?

A13. Zoonotic: contaminated poultry, milk, or direct contact with animals .

———

Helicobacter pylori

Q14. What are the diagnostic methods for H. pylori?

A14. Gastric biopsy for histology/culture; stool antigen test; blood antibodies. Histology is more sensitive than culture. Giemsa, silver, immunohistochemical stains visualize spiral-shaped bacteria .

Q15. What are the main virulence factors of H. pylori?

A15. Flagella (motility), urease, CagA protein, VacA toxin, mucinase, phospholipase, neutrophil-activating protein; tissue damage → gastritis, ulcers, adenocarcinoma, MALT lymphoma .

Q16. How is H. pylori treated?

A16. Combination antimicrobial therapy (triple or quadruple therapy) eradicates bacteria and improves gastritis/duodenal ulcer disease .