56 – Helicobacter and Clostridium Difficile

Speaker: David Aronoff, MD

2020 INFECTIOUS DISEASE BOARD REVIEW

Helicobacter pylori and Clostridioides Difficile

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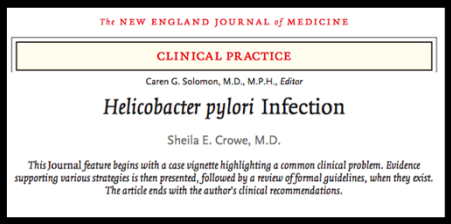
Disclosures of Financial Relationships with Relevant Commercial Interests

- None

HELICOBACTER PYLORI



The NEW ENGLAND JOURNAL of MEDICINE

CLINICAL PRACTICE

Caren G. Solomon, M.D., M.P.H., *Editor*

Helicobacter pylori Infection

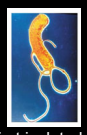
Sheila E. Crowe, M.D.

This Journal feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.

Recent review *N Engl J Med* 2019;380:1158-65.

Microbiology: *Helicobacter pylori*

Gastric Mucosa
- Spiral-shaped
- Flagellated
- Non-invasive



First isolated in 1983

Nobel Prize (Marshall & Warren, 2005)

NEJM 362: 1597, 2010

Agar
- Slow-growing (3-7 days)
- Gram negative rod
- Microaerophilic (5% O_2)
  - Catalase +
  - Oxidase +
  - **Urease +** → **Survival, Colonization, Diagnostic testing**

Urea → CO_2 + NH_3 → ↑pH

Question #1

A young woman undergoes upper endoscopy for unexplained nausea and vomiting. The stomach appears normal. Surveillance biopsies are taken and the gastric biopsy urease test is positive. The biopsies are most likely to show:

A. Hp organisms, but no gastric or esophageal inflammation.
B. Hp organisms plus gastric inflammation (gastritis).
C. Hp organisms plus esophagitis.
D. Neither Hp organisms, nor inflammation because the urease test is often false positive with a normal endoscopy.

Question #2

What is the most likely source for humans to acquire *H. pylori* infection?

A. Perinatally from mother
B. Ingestion of raw vegetables
C. Ingestion of undercooked meat
D. Ingested tap water from a municipal source
E. Contact with infected secretions from another human

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Helicobacter pylori: Key Points

- Humans are the only natural Hp host
- Infects > 50% of the world's population
  - US ~20-40%*
- A leading chronic infection in humans
  - Similar to dental caries
- Majority are asymptomatic but all have chronic active gastritis
- Severity of gastritis varies depending on the Hp strain & the host

*At greater risk, African Americans, Hispanics, Native Americans

NEJM 380:1158-65, 2019
NEJM 362:1597, 2010
Gut 66:6, 2017

Transmission of *H. pylori*

- Exact route of transmission is not known
- Likely **fecal-oral or oral-oral**
- Intrafamilial spread – (person-to-person, esp. mother-to-child)
- Low socioeconomic status, poor sanitation, crowding associated with ↑transmission

JAMA 282:2240, 1999 & Crowe SE, UpToDate (2018)

Disease Paths for *Helicobacter pylori* Infection

Asymptomatic gastritis	85-90%
Peptic ulcer (DU, GU)	1-10%
Gastric cancer	0.1-3%
MALT lymphoma	<0.01%

DU, duodenal ulcer
GU, gastric ulcer
MALT, mucosal-associated lymphoid tissue

NEJM 347: 1175, 2002
Gut 66:6, 2017

H. pylori: Disease Associations

- #1 cause of chronic gastritis
- PUD: 90% DU, 80% GU
- MALT lymphomas (72 – 98%)
- Gastric Cancer (60 – 90%)*

Hp causal

- Iron deficiency anemia, B12 deficiency, ITP
- Eradication Hp neither causes nor exacerbates GERD
- Hp poss. **reduces** risk for Barrett's esophagus/esophageal CA

HP is classified by WHO as a Class 1 carcinogen.
MALT = mucosal-associated lymphoid tissue

Maastricht V. Gut 66:6, 2017
Kasahun GG, Infect Drug Resist 13:1567-1573, 2020

Question #3

A 25-year-old African American woman complains of 6 weeks of symptoms consistent with dyspepsia unrelieved by current use of antacids & an OTC PPI.

The best approach to the diagnosis of *H. pylori* infection in this patient is:

A. Immediate Hp serology
B. Immediate Hp stool antigen EIA
C. Endoscopy with rapid urease test (RUT)
D. Immediate ^{13}C Urea Breath Test
E. D/C PPI for 2 weeks then Hp stool antigen EIA

Diagnosis of *H. pylori* Infection

Noninvasive (global)	Sensitivity	Specificity	
Urea Breath Test (^{13}C)	> 90 – 95%	> 90 – 95%	Live Hp
Stool Antigen (monoclonal)	> 90 – 95%	> 90 – 95%	Live & dead Hp
Serology	85%	79%	Detects exposure

Biopsy-based (sampling error)	Sensitivity	Specificity	
Rapid urease test	90%	95%	2-5 bx recommended
Histology	90 – 95%	95 – 98%	
Culture	73%	100%	Difficult

UBT considered 'best test'. Antigen test is usually less expensive.
Use only monoclonal stool Ag tests.
Histology requires 10^4 organisms to visualize

BMJ 344:44, 2012

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Testing Limitations for Hp

PPI
Antibiotics
Bismuth
Bleeding
} Interfere with all Hp tests

False negatives due to decreased Hp burden.
Recommend delay diagnostic testing until:

- PPI stopped for 2-4 weeks **(OTC antacids & H2RA do not affect UBT/SA testing)**
- Antibiotics, bismuth stopped for 4 weeks
- Bleeding stopped for 4-8 weeks

Crowe SE, UpToDate (2018)
Crowe SE, NEJM 380:1158-65 (2019)

Initial Diagnosis of *H. pylori* with Dyspepsia

- Stool antigen test (SAT)
- Urea Breath Test (UBT)
  - 'Test and Treat' in younger population (< 60 yo)
- Endoscopy mandatory if ≥60 years old or 'alarm symptoms or signs':
  - Unexplained iron-def anemia
  - GI bleeding
  - Unintentional weight Loss
  - Palpable mass
  - Severe abdominal pain
  - Persistent vomiting
  - Progressive dysphagia / odynophagia

Crowe SE, UpToDate (2018)
Crowe SE, NEJM 380:1158-65 (2019)

Question #4

- Which of the following is the most appropriate next step for evaluating a 29 year old previously healthy but overweight male patient with typical retrosternal heartburn symptoms?
  - A. Stool antigen test for *H. pylori*
  - B. Urea breath test for *H. pylori*
  - C. No testing for *H. pylori*
  - D. Serological testing for *H. pylori*
  - E. Empiric therapy for *H. pylori* regardless of testing

Explanation for Q#4

- *H. pylori* is not implicated as an etiological factor in gastroesophageal reflux disease
- Treatment for (eradication of *H. pylori*) can increase the risk for Barrett's esophagus and esophageal adenocarcinoma
- Serology is not a recommended test for *H. pylori* anymore

Reference: Siddique O, *et al. AJM* 2018

Question #5

A 23 yo Eastern European woman presents with persistent epigastric discomfort diagnosed as Hp+ gastritis by endoscopy. Fecal Hp antigen is also positive. As a child, she was treated repeatedly with PCN/amoxicillin for recurrent tonsillitis.

What do you recommend for therapy?

A. Clarithromycin + amoxicillin + PPI
B. Metronidazole + erythromycin + PPI
C. Bismuth subsalicylate + TCN + metronidazole + PPI
D. Metronidazole + amoxicillin + PPI
E. PPI therapy alone given her age

Question #6

After treatment of this patient for Hp gastritis, the *H. pylori* stool antigen test should be repeated:

A. On the final day of *H. pylori* therapy
B. Two weeks after completion of *H. pylori* therapy
C. Eight weeks after completion of *H. pylori* therapy
D. The test should not be repeated to assess cure

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Questions #5 & 6: Explanation

- Clarithromycin resistance in Hp is really common in Eastern Europe
- Repeated exposure to amoxicillin could increase risk for resistant Hp
- Note, tetracycline is better than doxycycline
- Test of cure should be done at least 4 weeks after therapy is completed

Crowe SE, NEJM 380:1158-65 (2019)

Who should be treated for *H. pylori* infection?

Houston Consensus Conference on Testing for *Helicobacter pylori* Infection in the United States

Hashem B. El-Serag,[*,‡] John Y. Kao,[§] Fasiha Kanwal,[*,†,‖] Mark Gilger,[¶,#] Frank LoVecchio,[**] Steven F. Moss,[††] Sheila Crowe,[§§] Adam Elfant,[‖‖] Thomas Haas,[¶¶] Ronald J. Hapke,[##] and David Y. Graham[*,‡]

- "We recommend that all patients with active *H pylori* infection be treated"
- "Infection causes chronic progressive damage to the gastric mucosa that in 20%–25% of individuals will result in life-threatening clinical outcomes such as peptic ulcer or gastric cancer"

El-Sarag HB, *et al. Clin Gastroenterol Hepatol 2018;16:992–1002*

Who should be **tested & treated** for *H. pylori* infection?

Established Indications

- PUD (active/prior hx)
- MALT lymphoma
- Atrophic gastritis
- After gastric CA resection
- 1st degree relative w/ gastric Ca

Consider

- Non-ulcer dyspepsia*
- Use of NSAIDs/ASA
- Long-term PPI use
- Fe deficiency anemia (unexplained)
- ITP (low evidence base)
- Live in high gastric Ca region
- Asymptomatic infection**

*estimate ~10% respond
Goal: eradicate **prior to atrophy or metaplasia. Treatment reverses atrophy but not metaplasia.

Crowe SE, NEJM 380:1158-65 (2019)
Chey W, Am J Gastroenterol;114:1829–1832 (2019)

Principles of Hp Therapy

Goal: To use a regimen with >90% therapeutic success.

1. Ask about abx exposure hx (clarithromycin/metronidazole/fluoroquinolones)
2. Discuss adherence
3. Use high dose PPI (BID dose; increase gastric pH>4-5)
4. Longer (**14 days**) rather than shorter treatment courses
5. Combination drug therapy
6. Consider abx resistance patterns & testing*

Outcome is determined by Hp antibiotic sensitivity, drug dosing, treatment duration & treatment compliance. Smoking inhibits therapeutic responses.

*clarithromycin, metronidazole, levofloxacin

Fallone CA, *et al. Gastroenterology.* 2016 Jul;151(1):51-69.e14

Table 2. Evidence-based Treatment Regimens for *H. pylori* Infection in North America, Listed in Recommended Order.*

Treatment Type	Components	Duration	Comments†
		days	
Clarithromycin-based triple therapy‡	PPI, clarithromycin, and amoxicillin (twice daily for all antibiotics)	14	Recommended unless patient has documented allergy to ampicillin or high level of clarithromycin resistance
Bismuth-based quadruple therapy (Pylera‡)	PPI, bismuth, tetracycline, and nitroimidazole (four times daily for all antibiotics)	10–14	Recommended if patient has high level of clarithromycin resistance or history of macrolide use
Concomitant therapy	PPI, clarithromycin, amoxicillin, and nitroimidazole (twice daily for all antibiotics)	10–14	Not appropriate in patient with high level of clarithromycin resistance or documented allergy to ampicillin
Sequential therapy	PPI and amoxicillin; then PPI, clarithromycin, and nitroimidazole (twice daily for all antibiotics)	7, then 7	Not appropriate in patient with high level of clarithromycin resistance or documented allergy to ampicillin
Hybrid therapy	PPI and amoxicillin; then PPI, amoxicillin, clarithromycin, and nitroimidazole (twice daily for all antibiotics)	7, then 7	Not appropriate in patient with high level of clarithromycin resistance or documented allergy to ampicillin
Levofloxacin-based triple therapy	PPI, levofloxacin (once daily), and amoxicillin (twice daily)	10–14	Not appropriate in patient with documented allergy to ampicillin
Fluoroquinolone-based sequential therapy	PPI and amoxicillin; then PPI, levofloxacin, and nitroimidazole (twice daily for all antibiotics)	5–7, then 5–7	Complicated with regard to treatment adherence; not appropriate in patient with documented allergy to ampicillin

Evidence-based Treatment Regimens for *H. pylori* Infection in North America Listed in Recommended Order

Crowe SE, NEJM 380:1158-65 (2019)
Chey W, Am J Gastroenterol;114:1829–1832 (2019)

H. pylori & Antimicrobial Resistance

Amoxicillin	Low (<5-10%)
Tetracycline	Low (<5-10%)
Clarithromycin	High (10-50%)
Metronidazole	High (10-80%)*
Levofloxacin	High? (5-30%)

Ask about history: clarithromycin, quinolones & metronidazole
Rates of resistance show substantial geographic differences.
Prior, even distant, Abx hx can inform likelihood of Hp Abx resistance.
*Nitroimidazole resistance may be overcome by increased dosing (≥1500 mg/day).
NOTE: Rx with amoxicillin & tetracycline yields low response rates.

Aliment Pharmacol Ther 43:514, 2016
Maastricht V. Gut 66:6, 2017
Kasahun GG, Infect Drug Resist 13:1567-1573, 2020

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Management Issue: **Test of cure** for *H. pylori* Infection

- Stool antigen test Perform ≥ 4 weeks post-rx*
- Urea Breath Test Perform ≥ 4 weeks post-rx.

Some recommend testing 6-8 wks post-rx.

Endoscopy required if gastric ulcer, for example.

*FDA-approved

Maastricht V. Gut 66:6, 2017

KEY TAKE AWAYS

DIAGNOSIS:

- In most: Stool Hp antigen test, UBT
- If ≥60 years old or alarm symptoms / signs then endoscopy is mandatory

KEY TAKE AWAYS

TREATMENT:

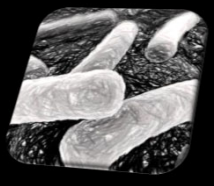
- Triple or Quadruple therapy
- Increasing emphasis on antibiotic resistance testing
  - Fecal or biopsy genotypic testing for clarithromycin
  - MIC testing for clarithromycin, nitroimidazole, FQ resistance

KEY TAKE AWAYS

FOLLOW UP:

- TOC mandatory (stool Hp antigen test, UBT)
- At least 4 weeks after completion of therapy

CLOSTRIDIOIDES DIFFICILE

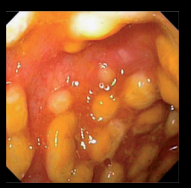


C. DIFFICILE INFECTION (CDI)

Most common health care-associated infection, USA
Leading cause of gastroenteritis death, USA

Ooijevaar RE, *et al. Clin Micro Infect.* 2018; 24(5):452-462

McDonald C, *et al. Clin Infect Dis.* 2018;66(7):987-994



Lancet 371:1486, 2008

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Antibiotic-associated Diarrhea (AAD)

- Common
  - In 5-25% of antibiotic treatment courses especially with > 3 days of Abx but one dose is sufficient
- 10-40% of AAD is associated with *C. difficile* infection (CDI) but nearly all AA **colitis** is CDI
- Disruption of colon microbiome & bile acid physiology are key mechanisms

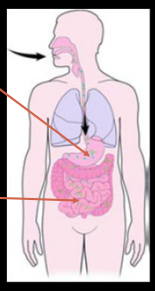
Pathogenesis of CDI

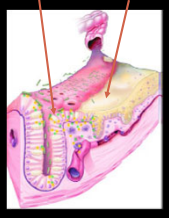
1. **CDI spores** survive in the environment for long periods of time. Following ingestion, they traverse the acidic environment of the stomach.

2. Spores germinate within the intestine.

3. Altered lower intestine flora (due to antimicrobial use) allows proliferation of *C. difficile* in colon.

4. Toxin A & B Production leads to colon damage +/- pseudomembrane.

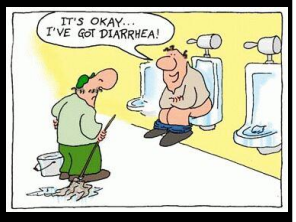




Slide adapted from CDC.gov. Sunenshine & McDonald Cleve Clin J Med 2006; 73(2):187-197.

Common Clinical Manifestations

- Watery & mucusy diarrhea up to 10 - 15 times daily
- Lower abdominal pain & cramping
- Low grade fever (15%+)
- Leukocytosis (around 15,000 cells/μl)
- Nausea
- Anorexia
- Malaise

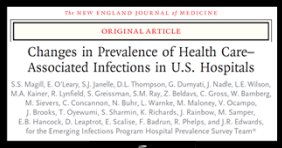


Complications of CDI

- Sepsis ± multiple organ dysfunction
- Megacolon: need for surgical intervention
  - Colectomy
  - Loop ilios tomy
- Bowel Perforation
- Lack of treatment response
- Recurrent infection (20%+)
  - Relapse
  - Reinfection

Epidemiology of CDI

2015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ORIGINAL ARTICLE

Changes in Prevalence of Health Care–Associated Infections in U.S. Hospitals

S.S. Magill, E. O'Leary, S.J. Janelle, D.L. Thompson, G. Dumyati, J. Nadle, L.E. Wilson, M.A. Kainer, R. Lynfield, S. Greissman, S.M. Ray, Z. Beldavs, C. Gross, W. Bamberg, M. Sievers, C. Concannon, N. Buhr, L. Warnke, M. Maloney, V. Ocampo, J. Brooks, T. Oyewumi, S. Sharmin, K. Richards, J. Rainbow, M. Samper, E.B. Hancock, D. Leaptrot, E. Scalise, F. Badrun, R. Phelps, and J.R. Edwards, for the Emerging Infections Program Hospital Prevalence Survey Team*

Magill S, *et al. NEJM* 2018;379:1732-44.
Photo from http://www.mckeenassociates.com/blog/2013/01/healthcare-associated-infections-plague-12-

Top Causative Pathogens	% of HAI	Rank
C. difficile	15	1
S. aureus	11	2
E. coli	10	3
Candida spp.	6	4
Enterococcus spp	5	5
Enterobacter spp.	5	6
P. aeruginosa	5	7
K. pneumo/oxytoca	5	8
Streptococcus	5	9

Major Risk Factors for Acquisition of CDI

1. **Antibiotic use**
   - Disruption of microbiome
2. **Recent hospitalization or LTCF**
   - Increased exposure
   - Co-morbidities reduce immunity or alter microbiome
3. **Age > 65 years**
   - Reduced gastric acidity
   - Impaired immunity
   - Altered microbiome

REMEMBER:
Even healthy people in the community without antibiotic exposure can get CDI

Dubberke E, *et al. Infect Control Hosp Epidemiol* 2011;32(4):360-366
Pacheco & Johnson. *Curr Opin Gastroenterol* 2013, 29:42-48
Loo V, *et al. NEJM*. 365:18

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Minor Risk Factors for Acquisition of CDI

4. Gastric acid suppression (**proton pump inhibitor**)
   - Reduced biochemical defense
   - Altered microbiome
5. Abdominal surgeries
   - Altered microbiome
6. Immunocompromised host
   - Impaired mucosal immunity
   - Altered microbiome

McFarland LV. *Curr Opin Gastroenterol.* 2009 Jan;25(1):24-35
Dubberke E, *et al. Infect Control Hosp Epidemiol* 2011;32(4):360-366
Pacheco & Johnson. *Curr Opin Gastroenterol* 2013, 29:42-48

CDI Severity

- **Leukocytosis**
- **AKI**
- **Sepsis/shock**
- **Megacolon**

Stool frequency is not part of severity assessment

Clinical Definition	Supportive Clinical Data
Nonsevere	Leukocytosis with a WBC count of ≤15,000 cells/mL and a serum creatinine level <1.5 mg/dL
Severe	Leukocytosis with a WBC count of ≥15,000 cells/mL or a serum creatinine level >1.5 mg/dL
Fulminant	Hypotension or shock, ileus, megacolon

Table from Wilcox M. *IDSE* (2018)
McDonald LC, *et al. Clin Infect Dis.* 2018 Mar 19;66(7):987-994

C. difficile Diagnostic Testing

Whom to test?

Appropriate epidemiology/ill with diarrhea/endoscopic findings

No laxatives within last 48 hrs

Test diarrheal stools (unless ileus). *One stool.*

>3 liquid stools over 24h

Only test specimens if patient > 1 year old

McDonald LC, *et al. Clin Infect Dis.* 2018 Mar 19;66(7):987-994

C. difficile Diagnostic Testing

Simplified approach:

Diarrhea* + **Toxigenic *C. difficile* &/or toxin in stool** → **TREAT**

***No laxatives or other obvious causes**

C. difficile Diagnostic Testing

Nucleic acid amplification test (NAAT; PCR):

Detects the gene for toxin B

Advantages

- **High sensitivity**
- **Rapid**
- **Relatively inexpensive**

Disadvantages

- **Does not detect actual toxin**
- **Cannot differentiate colonization from infection**

Patient selection is critical

C. difficile Diagnostic Testing

Glutamate dehydrogenase (GDH) antigen EIA:

Detects *C. difficile* bacteria by secreted antigen

Advantages

- **High sensitivity**
- **Rapid**
- **Relatively inexpensive**

Disadvantages

- **Does not detect toxin**
- **Detects NON-toxigenic strains**
- **Cannot differentiate colonization from infection**

Must be combined to test for toxin (NAAT or EIA)

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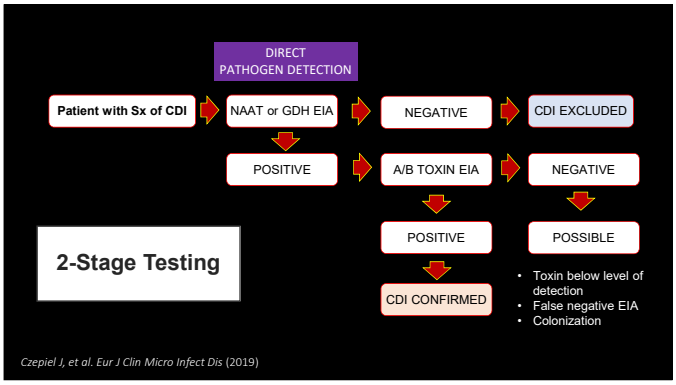
C. difficile Diagnostic Testing

Toxin A/B detection by EIA:

Detects *C. difficile* toxin(s) directly

Advantages	Disadvantages
• Good specificity • Rapid • Relatively inexpensive	• Poor sensitivity • False positives possible

Usually used in a 2-step protocol with NAAT or GDH



Czepiel J, et al. Eur J Clin Micro Infect Dis (2019)

CDI TAKE AWAYS

Careful selection of patients for testing, especially with NAATs, is extremely important

Only patients with diarrhea (≥3 stools in ≤24 hrs)

NO formed or soft stools (unless ileus)

NO 'Test of Cure'

Question #7

- 67 year old woman develops diarrhea while hospitalized for community acquired pneumonia. She is afebrile, her WBC count is 12,000/μl, creatinine is 1.2 mg/dl (baseline 1.0 mg/dl) and she is experiencing 12 small loose stools daily with abdominal cramping. Stool PCR is positive for *C. difficile* toxin B. Which of the following therapies is recommended?
  - Metronidazole 500 mg po TID x 10 days
  - Vancomycin 500 mg PO qid x 10 days
  - Vancomycin 125 mg PO qid x 10 days
  - Bezlotoxumab + vancomycin x 10 days
  - Fidaxomicin 200 mg PO BID + metronidazole 500 mg PO TID x 10 days

Therapy of CDI

- D/C antibiotics/change to 'lower risk abx'
- No antiperistaltics
- This is a time of transition for treatment guidelines
- Recurrent CDI occurs in **≥1 in 5** patients

McDonald LC, *et al. Clin Infect Dis.* 2018 Mar 19;66(7):987-994

Therapy of CDI

Table 1. Recommendations for the Treatment of *Clostridium difficile* Infection in Adults

Clinical Definition	Supportive Clinical Data	Recommended Treatment
Initial episode, non-severe	Leukocytosis with a white blood cell count of ≤15 000 cells/mL and a serum creatinine level <1.5 mg/dL	
Initial episode, severe[b]	Leukocytosis with a white blood cell count of ≥15 000 cells/mL or a serum creatinine level >1.5 mg/dL	
Initial episode, fulminant	Hypotension or shock, ileus, megacolon	• VAN, 500 mg 4 times per day by mouth or by nasogastric tube. If ileus, consider adding rectal instillation of VAN. Intravenously administered metronidazole (500 mg every 8 hours) should be administered together with oral or rectal VAN, particularly if ileus is present.

VANCOMYCIN 125 mg po QID x 10 d
FIDAXOMICIN 200 mg po BID x 10 d

No more metronidazole

McDonald LC, *et al. Clin Infect Dis.* 2018 Mar 19;66(7):987-994

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Recurrent CDI

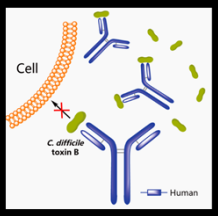
Table 1. Recommendations for the Treatment of *Clostridium difficile* Infection in Adults

Clinical Definition	Supportive Clinical Data	Recommended Treatment[a]
First recurrence	…	• VAN 125 mg given 4 times daily for 10 days if metronidazole was used for the initial episode, OR • Use a prolonged tapered and pulsed VAN regimen if a standard regimen was used for the initial episode (eg, 125 mg 4 times per day for 10–14 days, 2 times per day for a week, once per day for a week, and then every 2 or 3 days for 2–8 weeks), OR • FDX 200 mg given twice daily for 10 days if VAN was used for the initial episode
Second or subsequent recurrence	…	• VAN in a tapered and pulsed regimen, OR • VAN, 125 mg 4 times per day by mouth for 10 days followed by rifaximin 400 mg 3 times daily for 20 days, OR • FDX 200 mg given twice daily for 10 days, OR • Fecal microbiota transplantation[c]

McDonald LC, *et al. Clin Infect Dis.* 2018 Mar 19;66(7):987-994

Recurrent CDI

- Bezlotoxumab, a monoclonal antibody directed against toxin B produced by C. difficile, has been approved as adjunctive therapy for patients who are receiving antibiotic treatment for CDI & who are at high risk for recurrence



McDonald LC, *et al. Clin Infect Dis.* 2018 Mar 19;66(7):987-994
Figure from http://en.pharmacodia.com/web/drug/1_9806.html

Prevention of C. *difficile* Disease (HCW & visitors)

Contact precautions for patient care.
Gloves, gowns while diarrhea persists.
Single rooms
Handwashing with SOAP & WATER
Alcohol gel rubs do not kill *Cd* spores
Sporocidal solutions for hospital cleaning.
(eg. hypochlorite solutions)
Antibiotic restriction policies
(Antimicrobial stewardship programs).

Lancet ID 17:194, 2017 Scotland
Lancet ID 17:411, 2017 England

CDI **TAKE AWAYS**

Epidemiology
- Most CDI is health-care associated

Diagnosis
- Need to demonstrate toxin B in stool with NAATs, EIA
- Send only unformed stools when diarrhea meets CDC definition

Treatment: Primary or Recurrent CDI
- Vancomycin & fidaxomicin > Metronidazole
- Bezlotoxumab & fidaxomicin associated with lower risk for recurrent CDI
- Consider FMT for second or more recurrence

Prevention
- Hand wash as alcohol gels ineffective
- Bleach
- Antimicrobial Stewardship Programs

Thank you

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