Preventing gastric cancer by treating H. pylori

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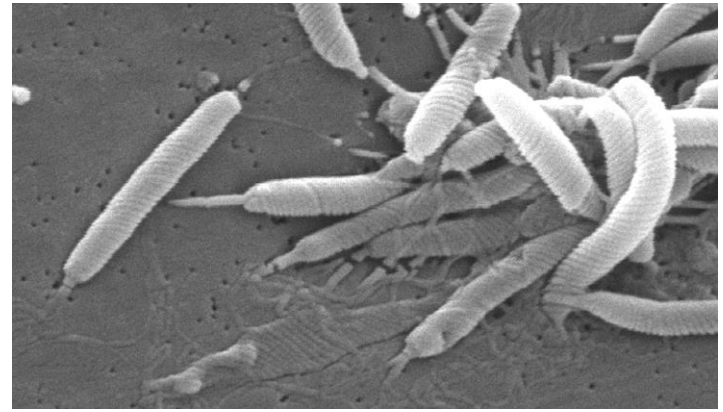
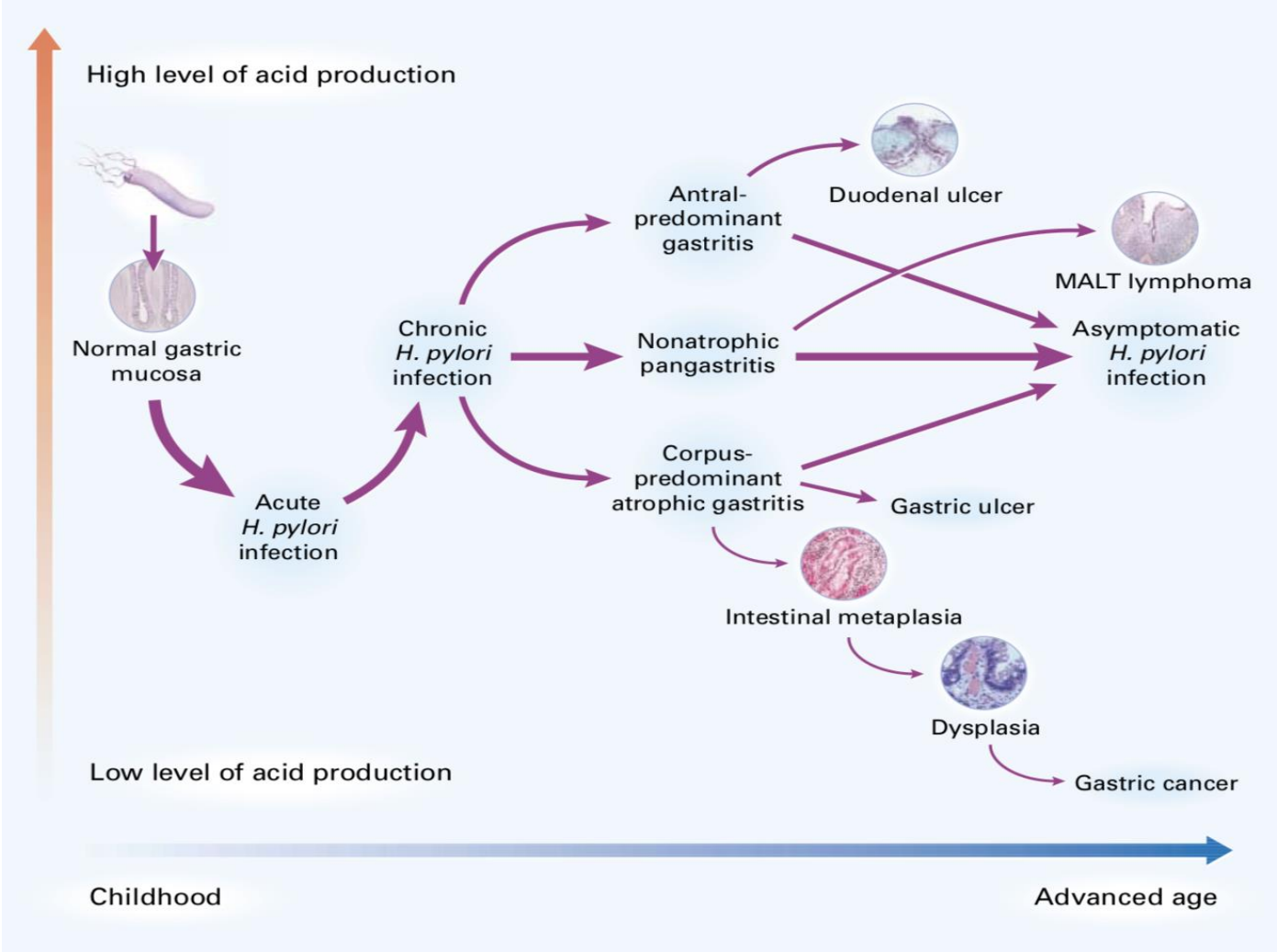
H.Pylori is a Common Gastrointestinal Infection

- 36% of US population infected[1]
- Duration of human infection is usually lifelong
- Individuals are typically infected by 10 years of age[2]
- Gram negative spiral bacterium with unipolar flagella which lives on gastric mucosa
- Can cause infection leading to
  - Atrophic gastritis
  - Peptic ulcer
  - Gastric cancer



1. Hooi JK, et al. Gastroenterology. 2017;153:420-429
2. Pacifico L, et al. World J Gastroenterol. 2010;16(41):5181-5194

H. Pylori Infection Can Follow Several Paths and Lead to Adverse Sequelae



Suerbaum S, Michetti P. Helicobacter pylori infection. N Engl J Med. 2002 Oct 10;347(15):1175-86.

H. Pylori Is the #1 Risk Factor for Gastric Cancer

- Classified as a group 1 carcinogen by The International Agency for Research on Cancer (IARC), indicating that there is definitive evidence to conclude H. pylori can cause cancer in humans[1,2]

- University of Pennsylvania conducted anaylsis of over 370,000 H. pylori patients in the VA health administration database showed that successfully eradication H. pylori led to a 75% reduction in the risk of gastric cancer[3]

- However, in a recent survey of over 275 US prescribers, only 34% believed H. pylori was a major risk factor for gastric cancer[4]

1. National Cancer Institute (2019). Surveillance Epidemiology and End Results. SEER Stat Fact Sheet. Stomach
2. Schistosomes, liver flukes, and Helicobacter pylori. IARC working group on the Evaluation of Carcinogenic Risks to Humans. Lyon, 7-14 June 1994. IARC Monogr Eval Carcinog Risks Hum. 1994;61:1-241
3. Kumar S, et al. Gastroenterol. 2019 Oct 22
4. November 2019 survey of 279 prescribers including gastroenterologists (n=75), PCP (n=154), NP/PA in primary care (n=25), and NP/PA in gastroenterology practices (n=25).

Latest Evidence on Gastric cancer and H. pylori

- Gastric cancer is a common and often lethal cancer and like cervical and liver cancers, can be largely attributed to an infectious cause (H. pylori)[1]

- Germline pathogenic variants in cancer – predisposing genes are also essential in surveillance and prevention of gastric cancer, e.g. CDH1 is a risk gene for hereditary diffuse gastric cancer

- Usui Y et al. evaluated the association between germline pathogenic variants in 27-cancer predisposing genes and the risk of gastric cancer in a sample of 10,426 gastric cancer patients and 38,153 control patients[2]

- Germline pathogenic variants in 9 genes (APC, ATM, BRCA1, BRCA2, CDH1, MLH1, MSH2, MSH6, and PALB2)[2] were associated with the risk of gastric cancer. Specifically an interaction between H. pylori infection and pathogenic variants in homologous-recombination genes with respect to the risk of gastric cancer

1. Muller A, et al. N Engl J Med. 388;13:1225-1229
2. Usui Y, et al. Helicobacter pylori, Homologous-Recombination Genes, and Gastric Cancer. N Engl J Med. 388;13:1181-1190

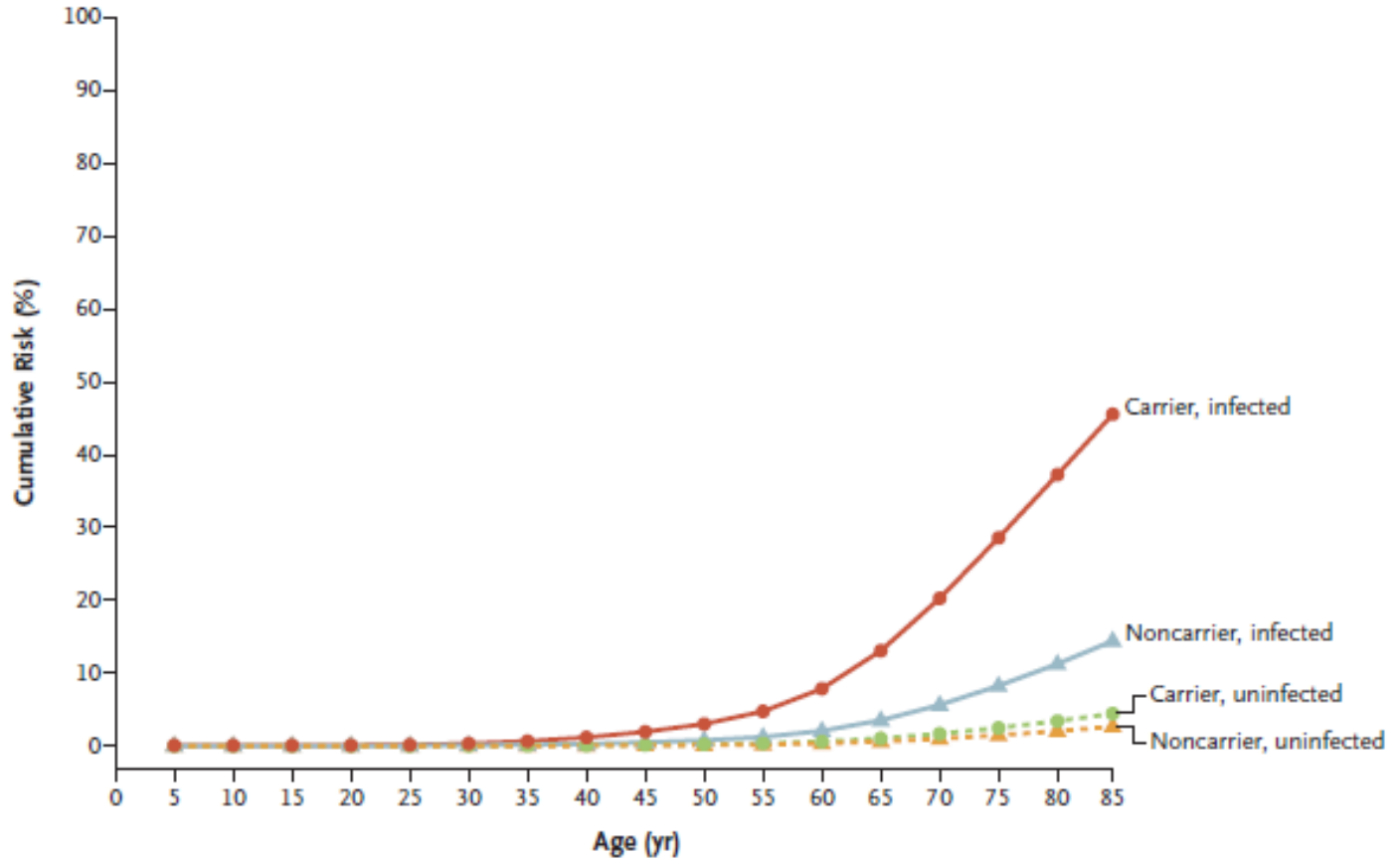


Figure 2. Cumulative Risk of Gastric Cancer through 85 Years of Age According to Germline Pathogenic-Variant Carrier Status and *Helicobacter pylori* Infection Status.

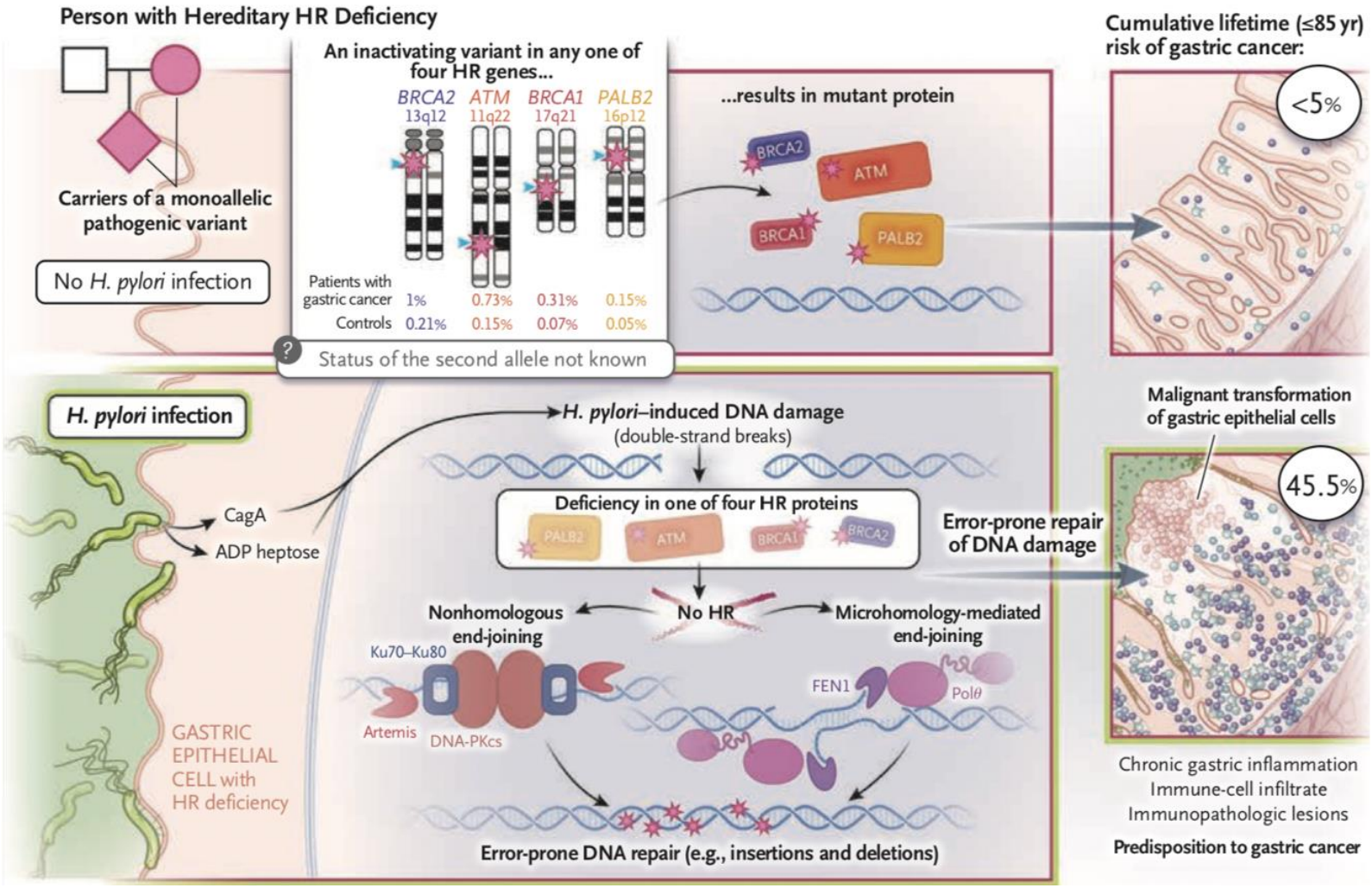
Cumulative risks of gastric cancer were estimated for carriers and noncarriers of germline pathogenic variants and persons who were positive and negative for *H. pylori* infection in the Hospital-based Epidemiologic Research Program at Aichi Cancer Center. Noncarriers were defined as persons without pathogenic variants in gastric cancer risk genes (*APC*, *ATM*, *BRCA1*, *BRCA2*, *CDH1*, *MLH1*, *MSH2*, *MSH6*, and *PALB2*). Only carriers of pathogenic variants in homologous-recombination (HR) genes (*ATM*, *BRCA1*, *BRCA2*, and *PALB2*) are shown in this figure because of the limited number of participants with variants in non-HR genes (*APC*, *CDH1*, *MLH1*, *MSH2*, and *MSH6*).

Latest Evidence on Gastric cancer and H.pylori continued

- H. pylori infection influences the risk of gastric cancer in patients with the aforementioned pathologic variants
- The lifetime risk of gastric cancer was 45.5% among persons with a pathogenic variant of homologous recombination gene and H. pylori infection
- In contrast, the risk was <5% among non-infected carriers and 14.4% among infected non-carriers of the homologous recombination gene
- This suggests that hereditary contribution to the risk of gastric cancer is more important that previously though and implies that DNA damage induced by H. pylori, if repaired incorrectly or not repaired at all, is a major driver of gastric carcinogenesis

Usui Y, et al. Helicobacter pylori, Homologous-Recombination Genes, and Gastric Cancer. N Engl J Med. 388;13:1181-1190

DNA Repair Mutation Presence With And Without H. Pylori



Conclusions

- 1. In persons known to carry a pathogenic variant homologous-recombination gene the evaluation and eradication of H. pylori is a paramount importance

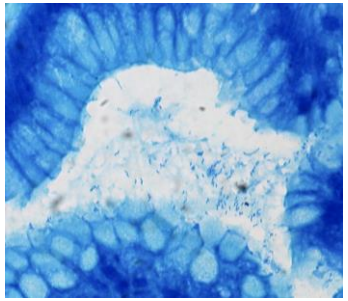
- 2. Since the carrier status of a pathogenic variant of homologous-recombination gene is not available clinically then the recommended best clinical strategy is to evaluate and treat H. pylori infection as well as confirming successful eradication

Several Diagnostic Testing Options Available

- Non-endoscopic
  - Urea breath test (UBT)*
  - Stool antigen test*
  - Serology - inadequate test for active infection
    - May remain positive even after successful eradication
    - ACG guidelines generally recommend against serology for H. pylori

  - *Patients need to be off PPIs for 2 weeks for valid UBT or stool antigen results.

- Endocsopic Biopsy
  - Rapid urease test
  - Histology
  - Culture



Chey WD, et al. Am J Gastroenterol. 2017;112(2):212-239
El-Serag H, et al. Clin Gastroenterol Hepatol. 2018;16:992-1002

2017 ACG Guideline on Testing and Treating

- All patients with a positive test of active infection with H. pylori should be offered treatment

Criteria	Strength of recommendation	Quality of evidence
Active or history of PUD	Strong	High
Low-grade MALT lymphoma	Strong	Low
History of endoscopic resection of early gastric cancer	Strong	Low
Uninvestigated dyspepsia (Age < 60; no alarm features)	Conditional	Moderate
Endoscopy for dyspepsia	Strong	High
Long-term, low-dose aspirin	Conditional	Moderate
Prior to chronic NSAID therapy	Strong	Moderate
Established on NSAID therapy	Conditional	Low
Unexplained iron deficiency	Conditional	Low

Chey WD, et al. Am J Gastroenterol. 2017;112(2):212-239

Treatment of H. Pylori Infection: General Considerations

- Require a positive test
- Offer treatment to all who test positive
- Explain the treatment, possible side effects, and potential complications of treatment non-compliance
- Choice of treatment depends on
  - Availability of local antimicrobial sensitivity and resistance rates
  - History of macrolide or quinolone exposure
  - Presence of *true* penicillin allergy
- Confirm eradication in everyone

Current Most Commonly Prescribed First Line H. Pylori Regimens

- Clarithromycin triple therapy (14 days)
  - PPI (BID)
  - Clarithromycin (500mg BID)
  - Amoxicillin (1gm BID) or metronidazole (500mg TID)

- Bismuth quadruple therapy [10–14 days (14 days associated with higher eradication rates)]
  - PPI (BID)
  - Bismuth subcitrate (part of Pylera, 420mg QID) or Bismuth subsalicylate (300mg or 524mg QID)
  - Tetracycline (500mg QID)
  - Metronidazole (250mg QID or 500mg TID)

Fallone CA et al. The Toronto Consensus for Treatment of Helicobacter pylori infection in Adults. Gastro 2016; 15:51
Chey WD, et al. Am J Gastroenterol. 2017;112(2):212-239

Resistance Compromises Clarithromycin Based Treatment as a Reliable Option

- In the US, >80% of currently prescribed regimens contain clarithromycin
- A recent study at TTUHSC El Paso evaluating H. pylori treatment regimens and subsequent eradication showed that 78.6% of patients received clarithromycin based regimens with an eradication rate of 83.2% in contrast to Bismuth based quadruple regimens' eradication rate of 91.5% ($p<0.05$)[1]
- Eradication rates for current standard of care therapies such as clarithromycin based triple therapy range from 60-75%[2,3,4]
- Treatment failure risk increases 3-7-fold in clarithromycin resistant strains treated with clarithromycin containing regimens[5,6]

1. Guzman J, Kalas MA, et al. The Efficacy of Bismuth Based Quadruple Therapy Compared With Clarithromycin-Based Triple Therapy for *Helicobacter pylori* in a Predominantly Hispanic Population: A Retrospective Cohort Study. Poster presented at ACG 2022
2. Malfertheiner P, et al. Gut. 2012;61:646-664
3. O'Connor A, et al. Helicobacter. 2015;20:54-61
4. Venerito M, et al. Digestion. 2013;88:33-45
5. Park JY, et al. Dig Dis Sci. 2016;61:2373-2380
6. Savoldi A, et al. Gastroenterol. 2018;155:1372-1382

2017 ACG Guideline Advises When to Avoid Clarithromycin-based H. pylori Therapy

Question		ACG Recommendation
Has my patient ever taken a macrolide antibiotic?	YES	Avoid Clarithromycin-based therapy
	NOT SURE	Avoid Clarithromycin-based therapy
Are my local H. pylori resistance rates for clarithromycin > 15%?	YES	Avoid Clarithromycin-based therapy
	NOT SURE	Avoid Clarithromycin-based therapy
Has this patient ever been treated for H. pylori with a clarithromycin-based regimen?	Yes	Avoid Clarithromycin-based retreatment
	NOT SURE	Avoid Clarithromycin-based retreatment

Chey WD, et al. Am J Gastroenterol. 2017;112(2):212-239

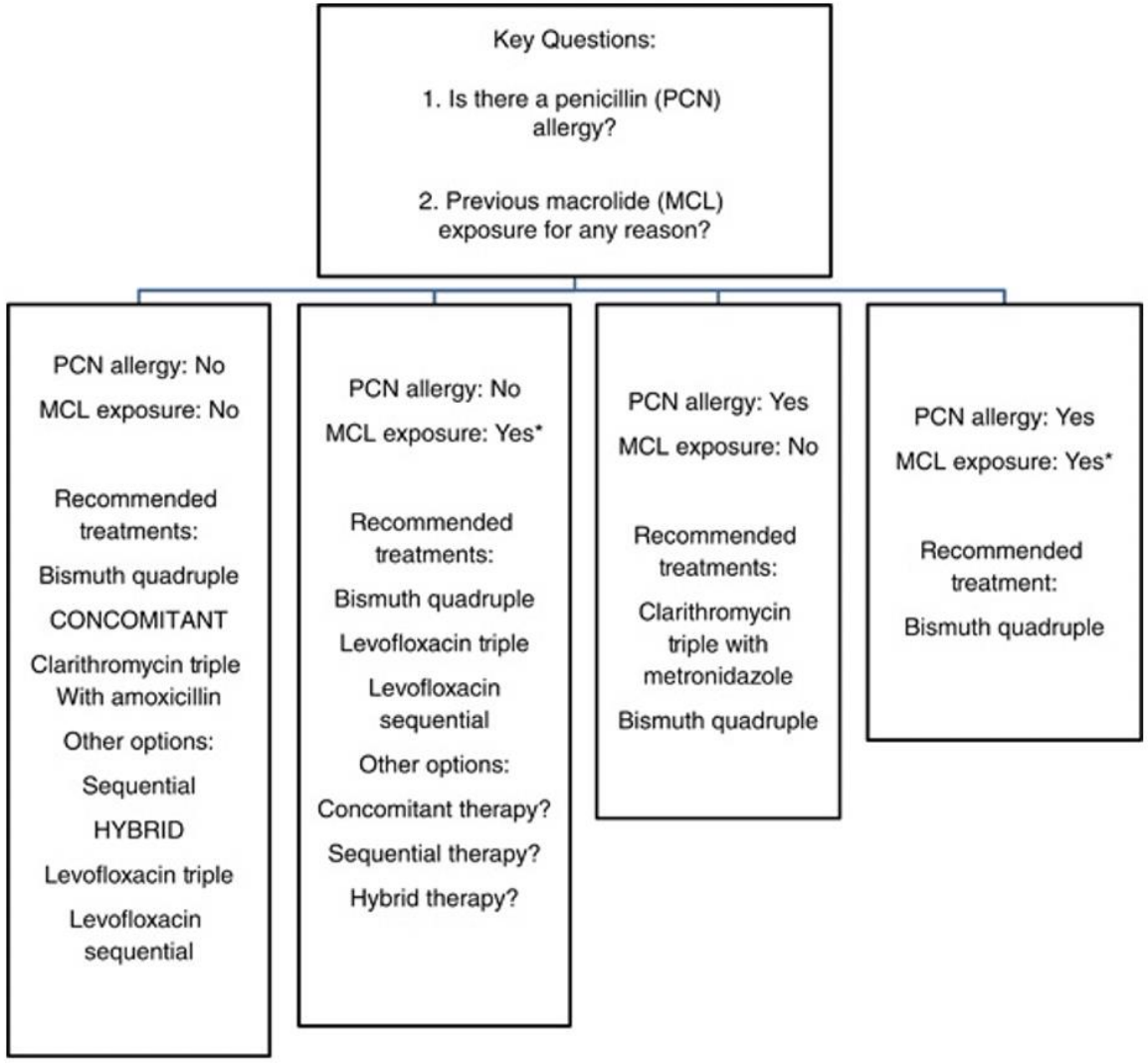
H. Pylori approved salvage regimens

Regimen	Drugs (doses)	Dosing frequency	Duration (Days)	FDA approval
Bismuth quadruple	PPI (standard dose)	BID	14	No[a]
	Bismuth subcitrate (120–300 mg) or subsalicylate (300 mg)	QID		
	Tetracycline (500 mg)	QID		
	Metronidazole (500 mg)	TID or QID		
Levofloxacin triple	PPI (standard dose)	BID	14	No
	Levofloxacin (500 mg)	QD		
	Amox (1 grm)	BID		
Concomitant	PPI (standard dose)	BID	10–14	No
	Clarithromycin (500 mg)	BID		
	Amoxicillin (1 grm)	BID		
	Nitroimidazole (500 mg)	BID or TID		
Rifabutin triple	PPI (standard dose)	BID	10	No
	Rifabutin (300 mg)	QD		
	Amox (1 grm)	BID		
High-dose dual	PPI (standard to double dose)	TID or QID	14	No
	Amox (1 grm TID or 750 mg QID)	TID or QID		

BID, twice daily; FDA, Food and Drug Administration; PPI, proton pump inhibitor; TID, three times daily; QD, once daily; QID, four times daily.

[a]PPI, bismuth, tetracycline, and metronidazole prescribed separately is not an FDA-approved treatment regimen. However, Pylera, a combination product containing bismuth subcitrate, tetracycline, and metronidazole combined with a PPI for 10 days is an FDA-approved treatment regimen.

Clinical pathway to treatment



Sequential therapy consists of a PPI and amoxicillin for 5–7 days followed by a PPI, clarithromycin, and a nitroimidazole for 5–7 days
Hybrid therapy consists of a PPI and amoxicillin for 7 days followed by a PPI, amoxicillin, clarithromycin and a nitroimidazole for 7 days

Confirmation of Eradication is Critical

- Symptom relief does not correlate with eradication
  - 2013 study demonstrated no difference in dyspeptic symptoms at 1 year post-treatment between H. pylori-eradicated and H. pylori-persistent patients[1]
  - ACG guideline recommends routine post-treatment testing[2]
    - UBT or fecal antigen
    - Endoscopy based testing only if endoscopy is clinically indicated
- Post treatment testing should be obtained atleast 4 weeks after treatment completion
- PPI should be held for 1-2 weeks prior to testing

1. Kim SE, et al. J Neurogastroenterol Motil. 2013;19(2):233-243
2. Chey WD, et al. Am J Gastroenterol. 2017;112(2):212-239

Role of probiotics in first-line therapy

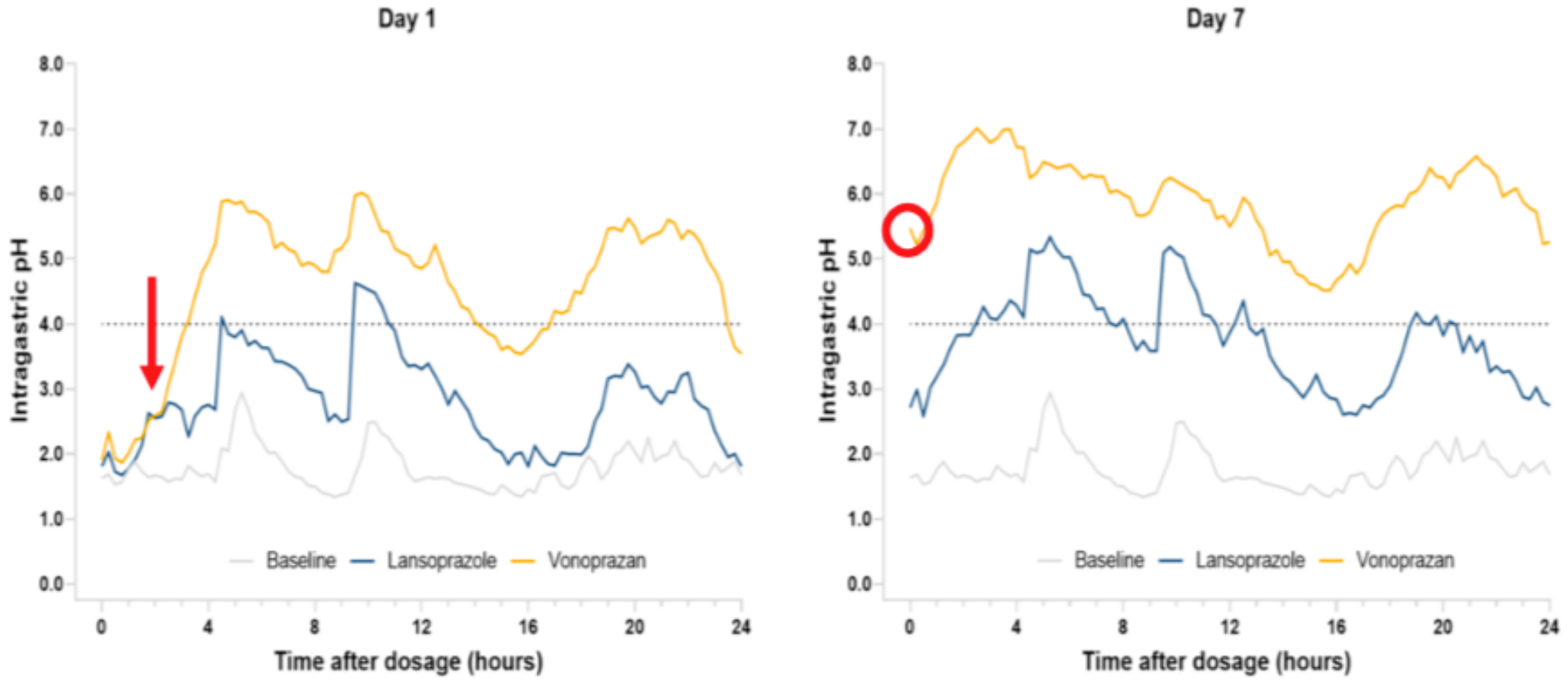
- There is growing interest in the United States of probiotics as adjuvant therapy in the treatment of *H. pylori* infection.
- Emerging evidence suggests an inhibitory effect of *Lactobacillus* and *Bifidobacterium* species on *H. pylori.*
- Furthermore, these probiotic strains may also help to reduce the side effects of eradication therapies and improve compliance with therapy
- A meta-analysis of 10 clinical trials of adjuvant probiotics in patients with *H. pylori* infection demonstrated increased cure rates with probiotic supplementation

Whats New in H. pylori Management?

- Potassium competitive acid blockers (PCA-B):
  - Licensed mainly in Asia and South America
  - Fast onset of action and more profound control of acid secretion compared to PPIs
  - Examples include: revaprazan, vonoprazan, tegoprazan, and fexuprazan
- Phase 3 trials of vonoprazan in US and europe done for H. pylori infection and erosive esophagitis

Abdel-Aziz et al, Aliment Pharmacol Ther 2021;53:794

Comparison of vonoprazan and lansoprazole on 24-hour intragastric pH



Laine et al, Am J Gastroenterol 2022;117:1158

Summary

- Resistance rates are increasing
- Avoid clarithromycin unless known local susceptibiliy
- Never re-treat with clarithromycin
- Resistance to amoxicillin, tetracycline, and rifabutin remains very rare
- Future availability of testing by next gen sequencing on stool or gastric mucosal biopsy likely to change practice
- H. pylori and gastric cancer is related to genomic profile and treatment can prevent significant morbidity and mortality
- Newer medication regimens are being studied (including PCA-B)

Thank You

H. pylori: Diagnosis and Management

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Disclosures: None

Objectives

- Describe *H. pylori* diagnostic tools and their appropriate use
- Discuss treatment regimens
- Describe strategies for documentation of eradication

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Question

A 23 year old female complains of 6 weeks of epigastric burning discomfort, bloating and postprandial nausea unrelieved by current use of antacids & OTC PPI.
The best approach to the diagnosis of *H. pylori* infection in this patient is:

A. Immediate *H. pylori* serology
B. Immediate *H. pylori* stool antigen
C. Endoscopy with rapid urease test (RUT)
D. Immediate 13C Urea breath test
E. D/C PPI for 2 weeks then *H. pylori* stool antigen EIA

Diagnosis of *Helicobacter pylori* Infection

Non Invasive (global)	Sensitivity	Specificity	
Urea Breath Test (^{13}C)	> 90->95%	> 90-95%	Live *H. pylori*
Stool Antigen (monoclonal)	> 90-95%	> 90-95%	Live & dead *H. pylori*
Serology	85%	79%	Detects Exposure
Biopsy-based (sampling error)	**Sensitivity**	**Specificity**	
Urease test	90%	95%	2-5 Bx recommended
Histology	90-95%	95-98%	
Culture	73%	100%	Difficult

2012, BMJ, Braden

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Testing Limitations for *H. pylori*

PPI
Antibiotics
Bismuth
Bleeding

} Interfere with
All *H. pylori* tests except serology

False negatives due to decrease *H. pylori* 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

2019. NEJM. Crowe. H. pylori infection
Up-to-date. 2024

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Initial Diagnosis of *H. pylori* with Dyspepsia

- Urea breath test (UBT)
  - Test and treat in younger population (<60 y/o)
- Stool antigen test (SAT)

- Endoscopy mandatory if >60 y/o or “Alarm symptoms or signs”:
  - Unexplained iron-def anemia
  - GI bleeding
  - Unintended weight loss
  - Palpable mass
  - Severe abdominal pain
  - Persistent vomiting
  - Progressive dysphonia/odynophagia

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Question

- A 35 y/o male presents for evaluation of 2-year history of substernal burning after large meals. He has no other medical problems aside from obesity with BMI 35. Which is the best approach in this patient?
  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*

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Explanation

H. pylori is not implicated as an etiological factor in gastroesophageal reflux disease

Treatment for (eradication of *H. pylori) can have unpredicatable effects on GERD*

Serology is not recommended as first line test for *H. pylori* in most circumstances

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2018, AJM, Siddique O, et al.

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Question

A 44 year old woman presents with chronic dyspepsia. *H. pylori* antigen is positive. As a child, she was treated repeatedly with **Penicillin/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

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Questions: explanation

Clarithromycin resistance is common in many regions of the US

Past exposure to amoxicillin could increase risk for resistance

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2019. NEJM. Crowe. H. pylori infection

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Evidence-based treatment regimens for H. pylori infection in North America
Listed in Recommend order

		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

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2019. NEJM. Crowe. H. pylori infection
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H. pylori treatment regimens

Author/ reference/ year	Regimen										Duration of therapy (days)	Intent to treat Eradication Rate	95% CI
	A	C	T	B	M	E	O	L	P	R			
Laine 15/1998	■	■					■				10	75%*	(70 - 81)
Fennerty 16/1998	■	■						■			10	81%	(74 - 88)
	■	■						■			14	82%	(74 - 88)
Laine 13/2000	■	■				■					10	78%*	(70 - 85)
Laine 14/2003			■	■	■		■				10	88%	(82 - 93)
	■	■					■				10	83%	(77 - 90)
Bochenek 17/2003	■	■							■		7	65%*	(57 - 73)
		■			■				■		7	77%*	(69 - 84)
Vakil 18/2004	■	■								■	7	77%	(71 - 83)
	■	■								■	10	78%	(72 - 84)
	■	■					■				10	73%	(67 - 79)

E= Esomeprazole, A= Amoxicillin, C= Clarithromycin, O= Omeprazole, B= Bismuth, M=metronidazole, T= tetracycline, L=lansoprazole, P=pantoprazole, R=rabeprazole

Usual doses: PPI twice a day, Clarithromycin 500 mg twice a day, Amoxicillin 1 gram twice a day. Esomeprazole was studied in a single daily dose.

*** = mean of 2 studies**

Vakil N and Megraud F. Gastroenterology. 2007;133(3):985-1001.

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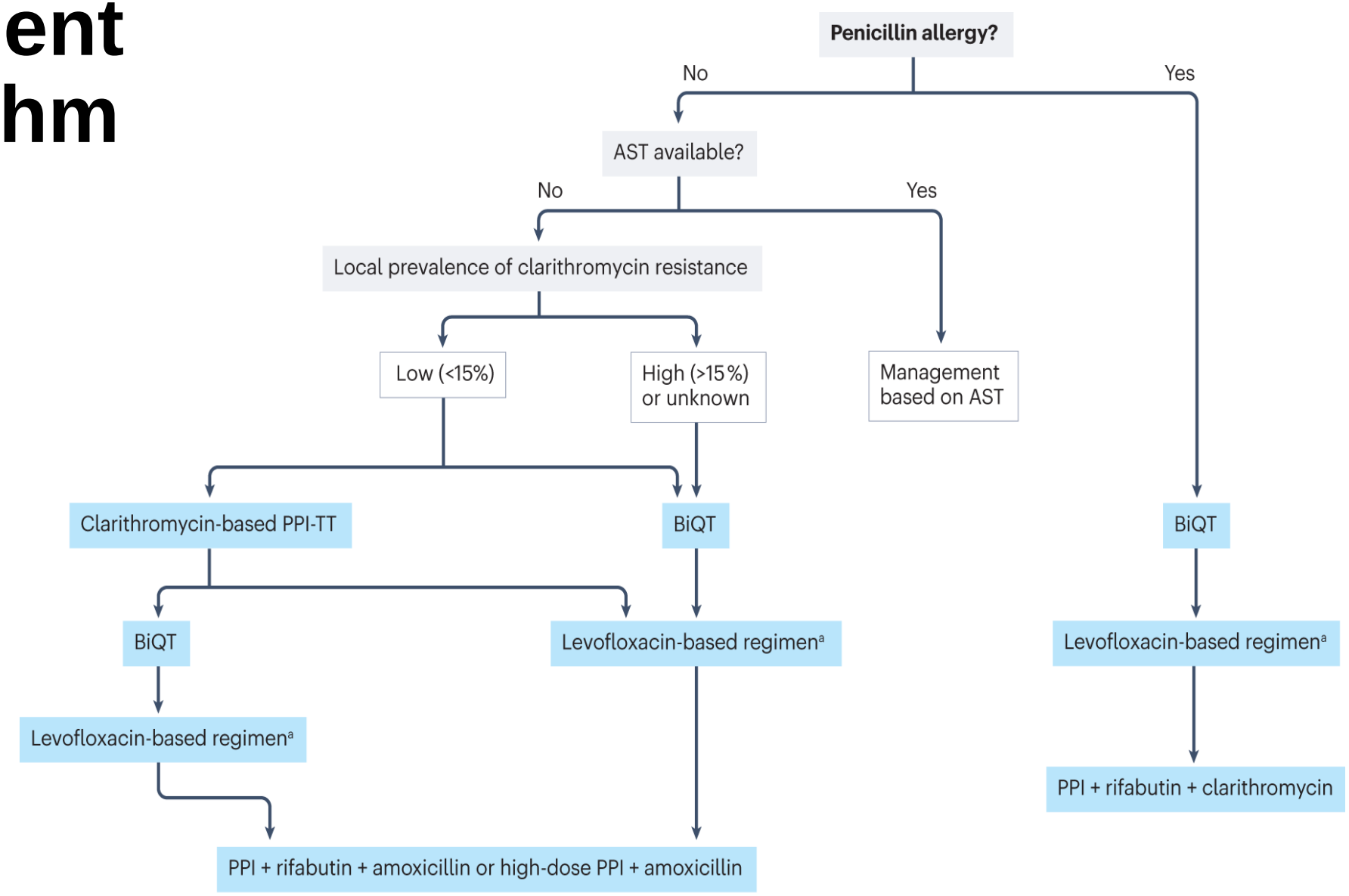
Treatment Algorithm Old



[a]Individual AST should be performed; levofloxacin-based regimen can be used if *H. pylori* is susceptible or community resistance is <15%; otherwise, use rescue therapy.

Malfertheiner et al *Nature Reviews Disease Primers* 2023

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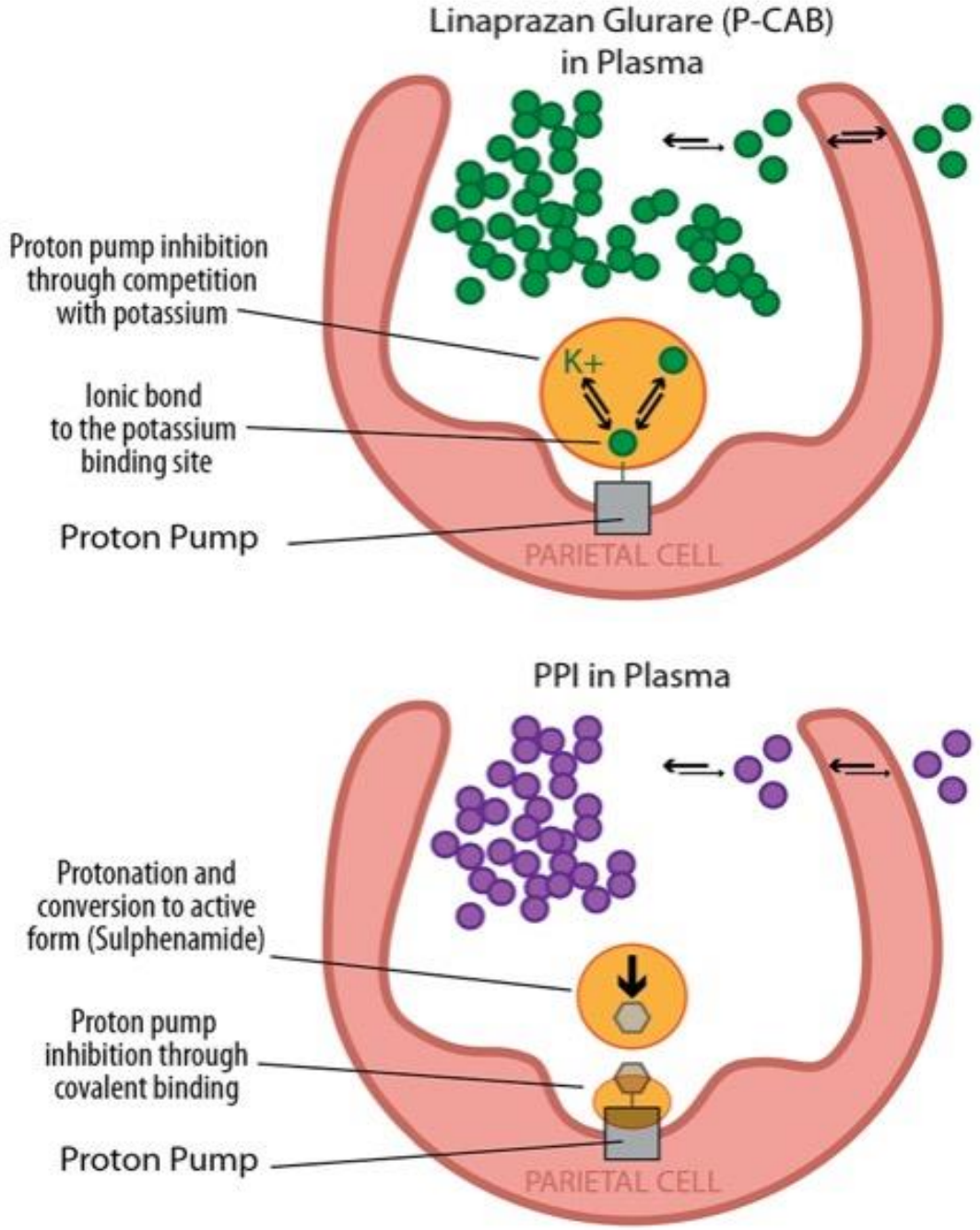
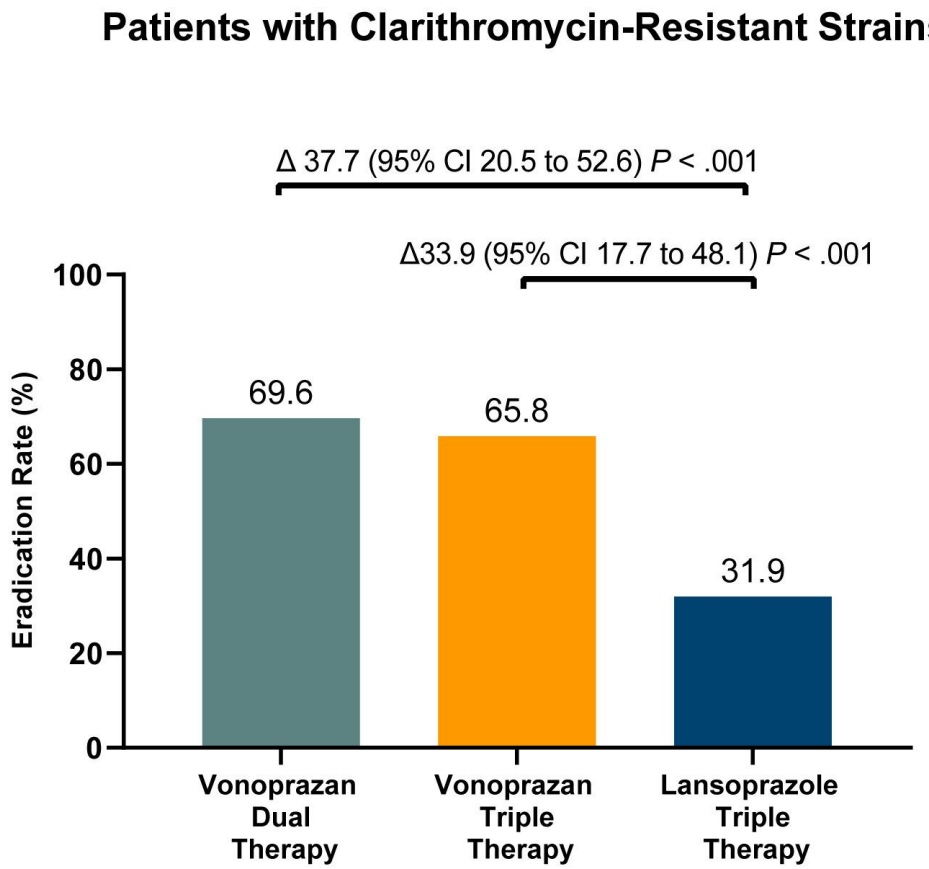
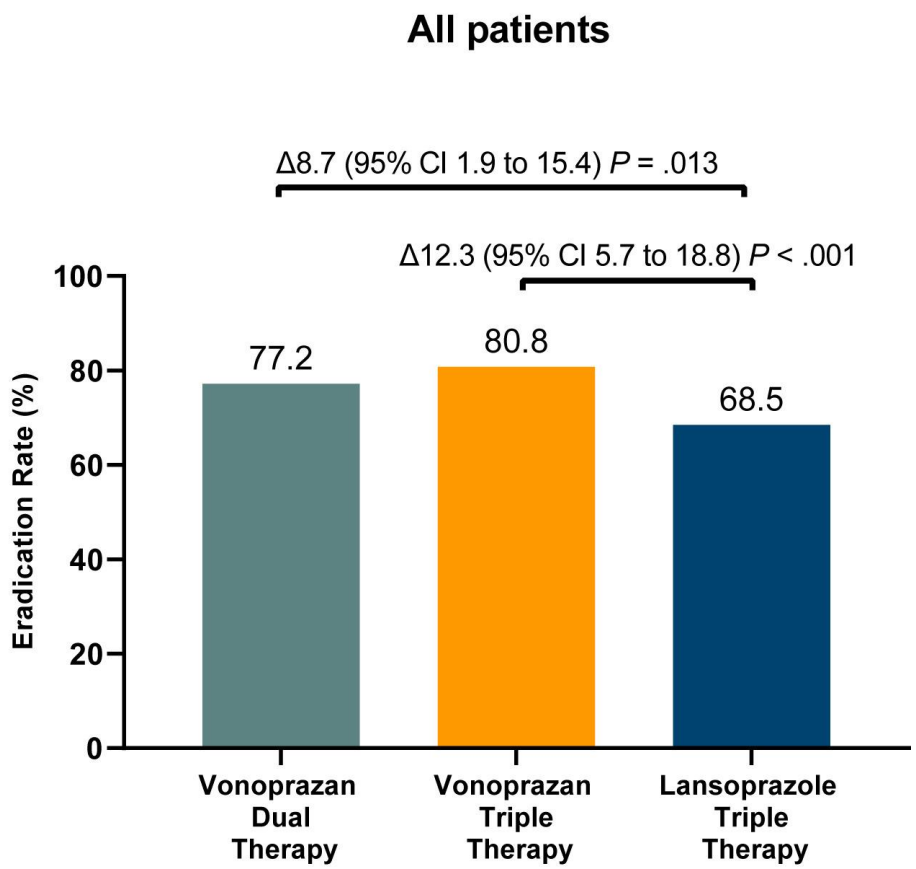


https://www.gastroendonews.com/Esophageal-Disorders/Article/07-23/P-CABs-Rival-PPIs-for-Rapid-Healing-In-Patients-With-Erosive-Esophagitis/70725

Vonaprazan (PCAB) in *H. pylori* treatment





2023. CGH. Chey et al.

2022. CGH. Chey et al.

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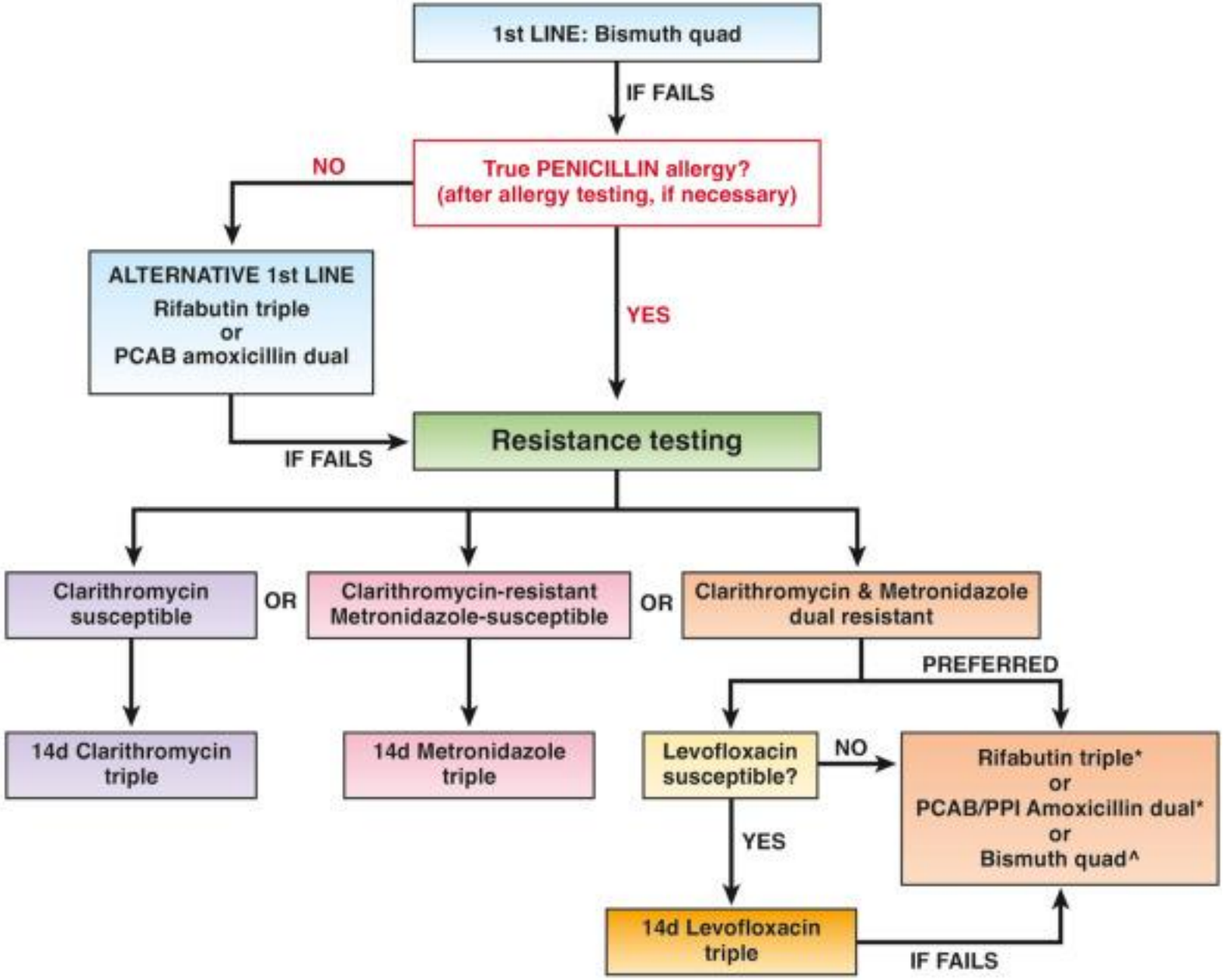
Treatment Algorithm New (2024)



2023. CGH. Moss et al.

Question

After treatment of this patient for *H. pylori*, the stool antigen test should 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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ACG Guidelines for post-treatment testing

- Test of active infection is recommended when endoscopic follow-up is unnecessary
- Urea breath test is the most reliable non-endoscopic test to document eradication
- Testing should be performed at least 4 weeks after treatment completion
- Serologic testing in the posttreatment setting should be avoided
- Results can remain positive for years after successful eradication

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Chey WD, Wong BC; Practice Parameters Committee of the American College of Gastroenterology. *Am J Gastroenterol.* 2007;102(8):1808-1825.[2]

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Summary

- H. pylori testing should be considered in patient with dyspepsia with or without alarm symptoms
  - Alarm symptoms = endoscopy
  - Breath test, stool antigen, and gastric biopsies are options
  - Serology may be falsely positive
  - All tests except serologies are affected by PPI use
- Treatment – first line in most situations now should be BQT
  - Remember PCAB regimens
- Post-treatment testing should be performed in all patients after at least 4 weeks

Thank you!