HELICOBACTER PYLORI

EDITED BY:

<u>DR. RGW. PINTO</u>

PROFESSOR AND HEAD

DEPT OF PATHOLOGY

GOA MEDICAL COLLEGE

BAMBOLIM GOA

EX DEAN GOA UNIVERSITY

PRESIDENT ASIAN SOCIETY OF CYTOPATHOLOGY

H.PYLORI

BY:DR.R.G.W.PINTO

Helicobacter Pylori Camplyobacter pylori

Gram negative Flagellated Helical bacteria Rid shaped curved Resides in Stomach

In 1983 Australian doctors Barry Marshall and Robin Warren

Two thirds of the world's popularition harbour s this org Most are asym

Colonization and virulence

Gastric
Chronic gastritis
Atrophic gastritis
DU
GU
Gastric Carcinoma
MALT Lymphoma. .DLBCL

89 percent of Gastric carcinoma are due to this org

Extra GIT lesions Anemia iron def .it B 12 def Diabetes CVS Neurological

H pylori protect / protective against Asthma Esophageal ca IBD GERD Crohns

Stains Giemsa H and E Warthin Starr silver impregnation Acridine orange Phase contrast Alcian. yellow toluidine blue Genta stain IHC

Org produce biofilms

Oxidase Catalase Urease

Genome of org is sequenced

No symptoms Lasts lifelong

It is a Class I carcinogen Ca Stomach Gastric Lymphoma

Gastric carcinoma cases 50 percent present with LN mets at initial presentation

ROS Reactive oxygen species RNS Reactive Nitrogen species

Cytokines Pro inflammatory DNA damage Oxidative DNA damage Oxidative stress DNA breaks

H pylori also causes Gastric polyps Colorectal polyps

Clinical

No symptoms

St ache .nausea .dyspepsia.indigestion .depression. anxiety

Abd pain .bloating.belching

Hunger

Vomiting. Heartburn

Bad breath weight loss

Complication bleeding

Tarry stools

Evades the immune response

Colonize

Flagella

Urease Adhesions Protease Exotoxins CagA is a oncoprotein assoc with Ca St

Epigenetically Decreased DNA repair Increased DNA damage

Methylation

Free radicals
Mutation
Perigenetic pathway
Inf
TNF IL 6
Mutations in tumor suppressor genes

Flagella 2 to 7 In one pole Burrows in mucus Gastric pits Biofilm

Urease breaks down urea into Ammonia and HCO 3

CagA Cytotoxin assoc antigen.A Oncoprotein

Biofilms Bioploymerase Microcolonies Aggregated bacteria

Diagnosis
When to test
Chronic peptic ulcer disease
Low grade MALT L
After Endoscopic resection of early Gastric ca
First degree relatives with Gastric ca
Cases of indigestion
Long term aspirin
Non steroidal anti inf
Unexplained iron def
ITP

Non invasive Serology Stool for antigen Urea breath test Carbon urea breath test Biopsy multiple samples Rapid Urease Culture

Transmission Contagious Oral Oral Feco Oral

Bacteria is seen in feces . Saliva .dental plaques Contaminated water

It thrives on high salt diet

Recurs Original strain. Recurrence Diff strain Reinfection

Epidemiology Two thirds of the world's population harbours this org Common in South America Sub Sub Saharan Africa Middle East

Early age intense inf Old age mild

In 1982 -1983 Barry Marshall and Robin Warren University of western Australia Perth Australia

Robin Warren was a pathologist
Barry Marshall was a clinician
Barry Marshall drank a beaker of H Pylori culture
After Few days .10 days became ill Nausea .Vomiting
Endoscopy showed Gastritis
Both received the Nobel Prize in Physiology or Medicine in 2005

H pylori related gastritis Lamina propria expanded Lymphocytes .Plasma cells Plasmacytosis Neutrophil mediated ep injury H pylori demonstrated by IHC Sites antrum Cardia H pylori is the first and only bacterium to be labelled as a carcinogen

It causes Gastric Adenocarcinoma and Gastric Lymphoma

H pylori inf leads to increased epithelial cell proliferation of the Gastric ep cells in a background of chronic inflammation

Inflammatory milieu

Geno toxic agent

Reactive oxygen species

Reactive Nitrogen species

Oncogenes

Oncoproteins

Cytotoxin Associated A gene (CagA)

Unregulated cell growth

Only 3 percent of infected pts get cancer or Lymphoma or both

ANTI HELICOBACTER PYLORI DRUGS

By:Dr.Pranali Mopkar

ANTIMICROBIALS

- Antimicrobials that are used clinically against H. pylori are: amoxicillin, clarithromycin, tetracycline and metronidazole/tinidazole.
- However, any single antibiotic is ineffective.
- Resistance develops rapidly.
- Anti H pylori regimen consist of 2 antimicrobials.
- STANDARD TRIPLE THERAPY
- PPIs + 2 antimicrobials
- BISMUTH QUADRUPLE THERAPY
- CBS + PPIs + 2 antimicrobials
- advocated for eradication failure cases.

one week triple drug regimen

- For majority of cases
- H.pylori eradication rate is upto 85% cases

two week triple drug regimens

- higher eradication rate (upto 96%)
- Produces more adverse effects, are more expensive and compliance is poor.
- Reserved for patients not achieving complete eradication with 1 week regimens.

Anti-H. pylori Regimens*

- 1. First-line regimen (for 7 days)
 - PPI
 - Amoxicillin
 - Clarithromycin or Metronidazole
- 2. Second-line regimen (for 7 days)

(for patients still symptomatic or H. pylori positive after 1st line treatment)

- PPI + Amoxicillin
- Clarithromycin or Metronidazole (whichever not used in 1st line treatment)
- 3. Alternative second-line regimen (for 7 days) (for patients who have received both metronidazole and clarithromycin)
 - PPI + Amoxicillin
 - Tetracycline or levofloxacin
- 4. Regimen for patients allergic to penicillin (for 7 days)
 - PPI
 - Clarithromycin
 - Metronidazole
 - 5. Third-line regimen (4 drugs for 10 days)
 - PPI + Amoxicillin
 - Bismuth subcitrate (CBS)
 - Clarithromycin or Metronidazole or Tetracycline

Standard dosing schedule for adults

Amoxicillin 1000 mg BD Omeprazole 20 mg BD Clarithromycin 500 mg BD Esomeprazole 20 mg BD Metronidazole 500 mg BD Lansoprazole 30 mg BD Tetracycline 500 mg QID Pantoprazole 40 mg BD Levofloxacin 500 mg BD Rabeprazole 20 mg BD Colloidal bismuth subcitrate (CBS) 120 mg

Note: Tinidazole can be substituted for metronidazole.

*Adopted from British National Formulary (BNF) 83, pp. 91-92, Sept. 2022

PPI: Proton pump inhibitor.

- All regimens are complex and expensive, side effects are frequent and compliance is poor.
- Higher failure rates (20–40%) have been reported from India.
- Also, 5 year recurrence rate of H. pylori infection is higher.
- Three week treatment is being advocated by some.
- Nevertheless, long-term benefits of anti-H. pylori therapy include lowering of ulcer disease prevalence and prevention of gastric carcinoma/lymphoma.