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HELICOBACTER PYLORI: THE UNDERVALUED BACTERIUM

INTRODUCTION

Helicobacter pylori were first seen in the stomach in 1899, by a Polish doctor, Dr Jaworski. He published his findings in a medical journal; unfortunately, it was in Polish and had little impact on the rest of the world. Almost a century had passed, before the next publication describing this interesting bacterium. Prof Warren and Marshall published their findings in the Lancet, in a paper titled "Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration". Despite initial skepticism by their colleagues, the pair continued their research into this "unidentified bacilli". In 2005, Prof Warren and Marshall were finally recognized for their pioneering work and awarded the Nobel Prize for Medicine.

In 1987, Thomas Brody, another Australian, developed the use of triple therapy for treatment of patients with duodenal ulcer and Campylobacter pylori.

THE BACTERIA

The bacteria was initially thought to be part of the Campylobacter genus and was named Campylobacter pylori (gatekeeper).It wasn't until 1989 that research revealed this was not part of the Campylobacter genus and the bacteria was classified into the Helicobacter genus, hence, Helicobacter pylori.

H pylori are a gram negative microaerophilic bacterium. The CAG A gene codes for one of the major H pylori virulence proteins and is associated with its ability to cause ulcers. In order for H pylori to survive the acidic environment of the stomach, it produces urease, to help neutralize the acid.

Worldwide, the prevalence of H pylori is high. Approximately 80% of peoples in developing countries are colonized, as compared to less than 40% in other countries. In Singapore, I found that 24.6% of patients undergoing gastroscopy had H pylori.

H PYLORI AND DISEASE

H pylori colonize the antrum of the stomach, resulting in inflammation. This in turn induces G cells to produce gastrin, resulting in increased acid production and damage to the lining of the stomach and duodenum. Thus, H pylori can cause gastric and duodenal ulcers, gastritis, and duodenitis and are associated with non ulcer dyspepsia. H pylori has been designated a WHO (World Health Organization) Class 1 carcinogen for its ability to cause stomach cancer. Interestingly, almost all gastric and duodenal ulcers were associated with H pylori in the latter part of the twentieth century. More recently, under half of peptic ulcer patients have H pylori. This is thought to be due increased use of aspirin, antiplatelet agents, non steroidal anti-inflammatory drugs and other associated causes of ulcer disease.

TESTING FOR H PYLORI

To test for H pylori one can do serology, urea breath testing, stool testing, or if undergoing gastroscopy a CLO test or histology. For patients not undergoing gastroscopy, MOH guidelines 2004 recommend urea breath testing (UBT). Tan Tock Seng hospital has recently purchased a new UBT machine. This machine uses the only FDA approved C13 capsule. To undergo a UBT, patients need to cease any proton pump inhibitors or antibiotics for 4 weeks. They fast before the test to increase the accuracy. The patient takes a C13 labeled capsule. Twenty minutes later, the patient breathes into a bag, a sample of the breath is taken and analyzed. If labeled CO_2 is detected, the patient has H pylori present in the stomach.

WHOM TO TEST FOR H PYLORI

Many people have H pylori and are not aware of this. Common symptoms include: epigastric pain, abdominal pain, loose stools, nausea, loss of appetite, vomiting and halitosis. Any patient who has any of the following diagnoses should be tested for H pylori if not previously done so: gastric ulcer, duodenal ulcer, gastritis, duodenitis, gastrointestinal metaplasia, gastric carcinoma and MALToma. Patients about to be commenced on long term warfarin/NSAID therapy should be checked for H pylori. We would recommend post eradication therapy testing to ensure successful eradication.

ERADICATION OF H PYLORI

Standard eradication therapy involves the use of one proton pump inhibitor and two antibiotics, twice daily for a weeks duration. See table 1. Use of amoxicillin, clarithromycin and omeprazole has an 87-93% cure rate with good patient compliance. For penicillin allergic patients, amoxicillin is exchanged with metronidazole. To confirm eradication of H pylori, we would recommend a Urea Breath Test be performed at least one month after therapy is completed. If H pylori remain, a second course of eradication using bismuth is recommended.

TABLE 1.

Type	Standard 7 Days	Penicillin Allergy	Quadruple Therapy 14 days	**Quadruple Therapy 7 days
	Omeprazole 20mg BD	Omeprazole 20mg BD	Omeprazole 20mg BD	Omeprazole 20mg BD
	Amoxycillin 1g BD	Metronidazole 400mg BD	Amoxycillin 1g BD	Metronidazole 400mg BD
	Clarithromycin 500mg BD	Clarithromycin 500mg BD	Clarithromycin 500mg BD	Tetracycline 500mg BD
			*Bismuth 240mg BD	*Bismuth 120mg QDS
Cure Rate	^87-93%	-	^85-95%	70%

*Tripotassium tricitratobismutate
**MOH Practice Guidelines 2004
^SHEN EF, C OH, NT PIN, A WEE. J Gastro and Hepatol 2008