HELICOBACTER PYLORI

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H. pylori is involved in the aetiology of a number of common gastroduodenal diseases, such as chronic gastritis, peptic ulceration and gastric cancer. Although Bizzozero identified the presence of spirochaetal organisms in gastric mucosa, it was not until the early 1980s that Warren and Marshall confirmed Koch’s postulates with respect to H. pylori and gastritis. Both received the Nobel Prize in Medicine or Physiology in 2005. (b)

120 Liquid Solid 100 80 60 40 Proportion remaining 20 0 0 40 60 10 30 50 20 Time (min) Figure 67.8 Dual-phase solid and liquid gastric emptying. The use of two isotopic labels allows the liquid and solid phases of the emptying to be followed separately. (a) Image acquisition. (b) Gastric emptying curves in a normal individual showing a typical lag period in the solid phase before linear emptying (courtesy of Dr V Lewington, Southampton, UK).

lyse urea, resulting in the production of ammonia, a strong alkali. The e ff ect of ammonia on the antral G cells is to cause release of gastrin via a negative feedback that is responsible for the modest, but inappropria te, hypergastrinaemia in patients with peptic ulcer disease, which, in turn, may result in gastric acid hypersecretion. The organism’s obligate urease activity is utilised by various tests used to detect the presence of the organ 13 14 ism, including the C and C breath tests and the CLO test (a commercially available urease test kit), which is performed on gastric biopsies. The organism can also be detected histo logically (Figure 67.9), using the Giemsa or the Ethin–Starry silver stains, and cultured using appropriate media. Previous or current infection with the organism may also be detected ser ologically . Breath tests or faecal antigen tests are recom mended for the pretreatment diagnosis of H. pylori infection in the community . Less accurate, hospital-based serology tests have a place within a non-invasive test-and-treat strategy . Infection with H. pylori leads to disruption of the gastric mucous barrier by the enzymes produced by the organism, and the inflammation induced in the gastric epithelium is the basis of many of the associated disease processes. The association of the organism with chronic (type B) gastritis is not in doubt. Some strains of H . pylori produce cytotoxins, notably the Cag A and Vac A products. Production of cytotoxins seems to be associated with the ability to cause gastritis, peptic ulceration and gastric cancer. The e ff ect of the organism on the gastric epithelium is to incite a classical inflammatory response that involves the migration and degranulation of acute inflamma tory cells, such as neutrophils, and also the accumulation of chronic inflammatory cells, such as macrophages and lympho cytes. It is evident how H. pylori infection results in chronic gastri tis and also how this may progress to gastric ulceration, but for a while it remained unclear how the organism could be involved in duodenal ulceration, as the normal duodenum is not col onised. As mentioned above, the production of ammonia does incr ease the level of circulating gastrin and it has been shown subsequently that eradication of the organism in patients with duodenal ulcer disease will reduce the acid levels to nor subjects and those with duodenal ulcers is considerable and the modestly increased acid levels in patients with Helicobacter - associated antral gastritis are insu ffi cient to

explain the aetiol - ogy of duodenal ulceration. The explanation can probably be found in the phenome - non of duodenal gastric metaplasia. Gastric metaplasia is the - normal response of the duodenal mucosa to excess acidity . It can be thought of in the same way as any other metaplasia in the gastrointestinal tract: an attempt by the mucosa to resist an - injurious stimulus . Although normal duodenal mucosa cannot be infected with H. pylori , gastric metaplasia in the duodenum is commonly infected and this infection results in the same inflammatory process that is observed in the gastric mucosa. - The result is duodenitis, which is almost certainly the precursor of duodenal ulceration. Infection with H. pylori may be the most common human infection. The incidence of infection within a population increases with age, and in many populations infection rates of 80–90% are not unusual. Up to 50% of the world’s popu - lation may be infected with Helicobacter . It appears that most infection is acquired in childhood and the possibility of infec - tion is inversely related to socioeconomic group. The means of spread has not been identified, but the organism can occur in the faeces and faecal–oral spread seems most likely . The organ - ism is not normally found in saliva or dental plaque. There is evidence in di ff er ent environments and in di ff erent population groups that the manifestations of the infection may be di ff er - - ent. Predominantly antral gastritis, which is commonly seen in the W est, results initially in increased levels of acid production - and peptic ulcer disease, whereas gastritis a ff ecting the body , common in the dev eloping world, may lead to hypochlorhydria - and gastric neoplasia. It has been known since 1984 that Helicobacter infection is amenable to treatment with antibiotics. The profound hypo - - chlorhydria produced by PPIs combined with antibiotics is also e ff ective in eradicating the organism. Commonly used eradication regimes include a PPI and two antibiotics, such as metronidazole and amoxicillin. V ery high eradication rates, mal. in the r egion of 90%, can be achieved with combinations that include the antibiotic clarithromycin, although it may be that in the future antibiotic resistance will become a problem. Reinfection following successful eradication appears rare (<0.5%) but incomplete eradication is a more important clinical problem. At present, eradication therapy is recommended for patients with duodenal ulcer disease, but not for patients with non-ulcer dyspepsia or in asymptomatic patients who are infected. How - ever, r ecent data show that a proportion of patients with non-ulcer dyspepsia do respond to treatment. H. pylori is now classified by the World Health Organization as a class 1 carcin - ogen, and it may be that the further epidemiological studies on the risk of gastric cancer change current advice on treatment.

Figure 67.9 Antral mucosa showing colonisation with Helicobacter pylori (modi /f_i ed Giemsa stain).

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