Postgrad Med J (1990) **66**, 801 – 802

Leading Article

Helicobacter pylori – our knowledge is growing

D.G. Colin-Jones

Gastroenterology Unit, Queen Alexandra Hospital, Cosham, Portsmouth, Hants PO6 3LY, UK.

The interest generated by the organism now called *Helicobacter pylori* (formerly *Campylobacter pylori*)[1] has been tremendous, with a huge literature developing in the short time since it was first described in 1983 by Marshall and Warren.[2] It is a highly specialized spiral organism that lives under the mucus layer, predominantly in the gastric antrum. It may also be found in areas of gastric metaplasia in the duodenal cap. *H. pylori* is now accepted as the predominant cause of non-autoimmune chronic active gastritis and plays an important role in peptic ulcer disease.[3] However, its role in upper gastrointestinal disease has not yet been fully elucidated, nor have the therapeutic implications – but these are very exciting.

H. pylori is a commonly found pathogen throughout mankind, with a prevalence in the western world of about 20% under the age of 20, rising to about 50% at the age of 50 years. Its presence in the gastric mucosa is strongly correlated with chronic active gastritis of the non-autoimmune type, being found in about 90% of cases. If the organism can be eradicated there is a striking improvement in the histology of the gastric mucosa.[4] However, eradication of the organism is not easy, because although sensitive to many antibiotics *in vitro*, *in vivo* resistance develops swiftly and eradication is difficult. *H. pylori*, however, is particularly sensitive to bismuth and it seems that a combination of bismuth with an antibiotic will increase the eradication rate from about 20 to 30% with bismuth alone to 70 to 80% with a combination. This is allowing a number of trials to be undertaken, with eradication being defined as absence of *H. pylori* one month after completing a course of treatment.

But does *H. pylori* cause disease? There is still controversy as to whether or not the organism and the associated gastritis is one of the causes of non-ulcer dyspepsia. Although treatment of *H. pylori* results in improvement of gastritis, symptomatic improvement is not always significantly more than with placebo.[4,5] However, two researchers undertook self-inoculation studies and developed dyspeptic symptoms of short duration, so there is no doubt that the organism does cause gastric symptoms with an acute infection – and in one chronic gastritis developed later.[6,7] More work is needed, and indeed is being undertaken.

H. pylori is found in about 80% of gastric ulcers and more than 95% of duodenal ulcers – indeed the dictum, 'no acid, no ulcer' might be complemented by a new dictum, 'no *H. pylori*, no duodenal ulcer'. This does *not* mean that the organism is solely responsible for peptic ulceration because such a high prevalence of *H. pylori* is found in the general population. However, the rarity of a duodenal ulcer not associated with *H. pylori* suggests that it has an important part to play[8] – probably as an enabling infection with other factors triggering the final ulceration, for example smoking, non-steroidal anti-inflammatory drugs, and dietary factors may all contribute to the ulcerative process in a mucosa which has been damaged by this organism. The organism does produce an inflammatory reaction with change in the mucus layer and therefore probably reduces the mucosal defence, but how it leads to the gastric hypersecretion often associated with duodenal ulcer is not known. Recently, it has been suggested that this organism, which has the curious feature of producing ammonia (and does so under the mucus layer adjacent to the cells of the gastric antrum), might raise the pH next to the cells of the antrum and thereby stimulate the gastrin secreting cells.[9] This gastrin hypersecretion has been reported, in preliminary studies, to diminish after eradication of the organism.

One of the major clinical implications of this organism lies in its role in ulcer recurrence. It has now been convincingly demonstrated that eradication of the organism reduces the relapse rate from about 80% per annum for duodenal ulcers to about 20%,[10] or perhaps even to nil.[11] This has great potential therapeutic benefit, but currently the difficulty of safely and easily eradicating the organism is making it difficult to advise patients on the optimum therapy. Nonetheless there is growing evidence that bismuth compounds in association with antibiotics might well be the treatment of the future, not only to heal ulcers but to reduce their recurrence rate, and possibly even to cure.

Correspondence: D.G. Colin-Jones, M.D., F.R.C.P.
Received: 18 June 1990

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