# Gastro-duodenal ulcers. Who ? When to treat ?

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Since the discovery of *Helicobacter pylori* by the Australians Robin Warren and Bary Marshall in 1983, there has been a fundamental change in the approach to patients with peptic ulcer disease (1).

Until then, intraluminal gastric acid was traditionally considered to be the most important pathogenetic factor in recurrent peptic ulcer disease. Their findings were the basis of a concept which was soon developed suggesting that peptic ulcer disease was nothing else than a complication resulting from a chronic infection with *Helicobacter pylori.* It took several years to convince the medical community, but well-controlled trials world-wide showed always the same result : eradication of the organism was not only capable of healing duodenal and gastric ulcers but it could also cure the disease. In 1994, the first official international guidelines for diagnosis and therapy were published by the US National Institute of Health (2). This report ended a period where there were not only "believers" and "non-believers", but also "treaters" and "non-treaters" as well. Since then several European countries have organised their own consensus-meetings thereby adjusting and refining the NIH recommendations and guidelines. In september 1996 the European *Helicobacter pylori* Study Group (EHPSG) organised a special meeting in Maastricht in order to develop a European consensus. Their guidelines, where special attention was paid to the role of the primary care physician, were published last summer (3).

Nowadays, no one will argue anymore against the current recommendation that all patients with *H. pylori*-positive peptic ulcer disease, past or present, including those in remission or receiving anti-secretory maintenance treatment need an appropriate eradication therapy. Several studies have clearly demonstrated that *H. pylori* is present in more than 90% of patients with duodenal ulcer and 60-80% of patients with gastric ulcer and that with current regimens eradication can succesfully be obtained in more than 90% of patients. Taking into account these numbers, one could even question whether in the case of uncomplicated duodenal ulcer disease it is still necessary to test for *H. pylori* before or even after therapy. The approach to gastric ulcer disease should be different and more aggressive since malignancy remains always a possibility and must be ruled out by multiple biopsies in these patients.

One of the most recent studies from the Hong-Kong group provides the strongest evidence up to now that *H. pylori* is etiologically related to duodenal ulceration (4). They showed that duodenal ulcers could be healed by using antibiotics alone without the use of acid-suppressive drugs.

More and more data become available about the long-term outcome of ulcer disease after succesfull eradication of the organism. Van der Hulst *et al.* recently demonstrated in a prospective study that relapses of duodenal or gastric ulcers remained absent after complete eradication of *H. pylori*, even after follow-up to 9.8 years (5) !

Several studies during the past years (6-8) also have reported that there is an important reduction of rebleeding when *H. pylori* was eradicated in patients who present with a bleeding duodenal ulcer. This should not be surprising since eradication of *H. pylori* changes the natural history of peptic ulcer disease. "No *H. pylori*, No ulcer" and no bleeding anymore ! Because of the high risk of rebleeding when *H. pylori* remains present, these patients need tests to confirm eradication. It is still unclear whether these patients should receive maintenance acid-suppressive therapy after *H. pylori* has been cleared.

Perforating duodenal ulceration seems not to be closely associated with *H. pylori* infection. These patients more common have a history of non-steroidal inflammatory drug (NSAID) ingestion (9). In elderly patients taking NSAID's the presence of *H. pylori* seems to be even associated with a lower risk of gastrointestinal bleeding (10). Cullen *et al.* also found that in these elderly patients with peptic ulcer bleeding the relative role of NSAID usage was more important than *H. pylori* infection (11).

The pathogenetic role of *H. pylori* in recurrent ulceration in the post-operative stomach (Billroth I, Billroth II and/or truncal vagotomy) seems to be less convincing and very few data are available about this specific condition. It has been suggested that local factors (ischemia, parietal cell mass, bile reflux) may be more important in these situations.

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