

Treatment of peptic ulceration

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The vast majority of uncomplicated peptic ulcers are treated medically. Surgical treatment of uncomplicated peptic ulceration has decreased markedly and is now seldom performed. Surgical treatment was aimed principally at reducing gastric acid secretion and, in the case of gastric ulceration, removing the diseased mucosa. When originally devised, medical treatment also aimed to reduce gastric acid secretion, initially using the highly successful H₂-receptor antagonist and, subsequently, PPIs. This has now largely given way to H. pylori eradication therapy. Medical treatment It is reasonable to suggest modifications to the patient's life-style, particularly the cessation of cigarette smoking; however, pharmacological measures form the mainstay of treatment. H₂-receptor antagonists and proton pump inhibitors revolutionised the management of peptic ulceration. Most duodenal ulcers and gastric ulcers can be healed by a few weeks of treatment. There remained, however, high doses of H₂-receptor antagonists. This is largely now irrelevant as PPIs can effectively render a patient achlorhydric and all benign ulcers will heal using these drugs, the majority within 2 weeks. Symptom relief is rapid, most patients being asymptomatic within a few days. Like H₂-antagonists, PPIs are safe and relatively devoid of serious side effects. The problem with all gastric antisecretory agents is that, following cessation of therapy, relapse is almost universal. Eradication therapy Eradication therapy is now routinely given to patients with peptic ulceration. If H. pylori is the principal aetiological factor (essentially in patient not taking NSAIDs) then complete eradication of the organism will cure the disease. Reinfection as an adult is uncommon. Eradication therapy is therefore the mainstay of treatment for peptic ulceration. It is extremely economical by comparison with prolonged courses of antisecretory agents or surgery. It is also considerably safer than surgical treatment. There are some patients with peptic ulcers in whom eradication therapy may not be appropriate, and this includes patients with NSAID-associated ulcers. Such patients should avoid these drugs if possible; if not, they should be co-prescribed with a potent antisecretory agent. Similarly, patients with stomal ulceration are not effectively treated with eradication therapy and require prolonged prescription of antisecretory agents. Patients with Zollinger-Ellison syndrome should be treated in the long term with PPIs unless the tumour can be adequately managed by surgery. Ulcers that fail to heal The introduction of antisecretory agents and effective treatments for H. pylori have revolutionised the management of peptic ulcers. Despite these advances, peptic ulceration fails to heal in a small minority of patients. Endoscopic re-evaluation should be regarded as mandatory to confirm healing of all gastric ulcers. Furthermore, endoscopy permits the differentiation between a refractory ulcer and persistent symptoms despite ulcer healing. The most common cause of failed healing is persistent H. pylori infection. Biopsies should be repeated at the time of endoscopy as false-negative results with breath tests may be expected soon after eradication therapy and serum antibody titres may not fall for 6 months after successful eradication. Failure of eradication is

usually due to poor compliance or bacterial resistance and bacteriological culture will guide further attempts at *H. pylori* eradication. The ingestion of NSAIDs should once again be addressed. A diagnosis of Zollinger–Ellison syndrome (described in detail in Zollinger–Ellison syndrome) should be suspected in *H. pylori* -negative, non-NSAID-related peptic ulceration and serum gastrin levels should be measured. Very rarely a recently described auto immune immunoglobulin G4-related (IgG4) phenomenon is the cause of resistant and recurrent gastric ulceration.

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