

Leading articles

Helicobacter pylori and peptic ulcer surgery

A decade ago no one considered that peptic ulceration was an infectious disease. Now it is known that *Helicobacter pylori*, a motile flagellate bacillus which dwells in the mucous layer of the stomach, is the main culprit of peptic ulceration. These bacteria are found in over 90 per cent of patients with duodenal ulcer and about 70 per cent of those with gastric ulcer. If the bacteria are eradicated, ulcer recurrence is dramatically reduced¹. Indeed, healing of both duodenal² and gastric³ ulcers can be achieved with eradication alone, without the addition of acid suppression. For the first time peptic ulcers can be cured medically without the need for long-term maintenance therapy. Such new information has not only revolutionized thinking on the pathogenesis of peptic ulceration, it has also forced a reconsideration of treatment policy. Indications for peptic ulcer surgery and the choice of ulcer operations must be revised in the light of these new discoveries.

Because *H. pylori* is protected by the mucous layer of the stomach, its eradication requires multiple drugs to be administered simultaneously. The original triple therapy regimen uses bismuth salts; side-effects and patient compliance are common problems. Newer regimens use a proton pump inhibitor combined with two antibiotics and may be administered on a twice-daily basis; side-effects are less common and high rates of eradication of over 90 per cent may be achieved with 1 week of treatment⁴.

If long-term cure can be achieved with a 1-week course of tablets, it makes little sense to offer elective ulcer surgery to patients without first attempting a medical solution. Only patients who fail to achieve eradication after multiple attempts and those who are *H. pylori* negative should be considered for surgery. Such patients are rare indeed and elective ulcer operations are now becoming extinct. Surgery for peptic ulcer in the new millennium will be confined to dealing with complications.

Bleeding is the commonest complication of peptic ulceration. In the past decade endoscopic haemostasis has superseded surgery as the first-line method directed at stopping ulcer bleeding. There is now good evidence that recurrent ulceration and bleeding can be prevented by *H. pylori* eradication⁵. Several studies, including two randomized controlled trials, have shown that after eradication the rebleeding rate over a follow-up of up to 1 year is zero, compared with 15–30 per cent in those who have not had eradication. Eradication therapy has also been shown to be superior to long-term maintenance with histamine receptor antagonists⁶. With effective endoscopic methods of stopping bleeding and the knowledge that medical cure of the ulcer diathesis is possible, the temptation is to persist with endoscopic therapy, especially in elderly patients. This may be dangerous as repeated blood loss, hypovolaemia and multiple transfusions lead to poor outcome. A well organized gastrointestinal bleeding team with clearly defined guidelines and endpoints is of vital importance in achieving good results.

The aggressive use of endoscopic haemostasis leads to a smaller proportion of patients requiring emergency surgery. Those who do are likely to be elderly and to have concomitant medical illnesses. They tend to have lost significant amounts of blood as they typically will have had one (or two) unsuccessful attempts at stopping bleeding by endoscopy. Such patients present a difficult challenge for the new generation of surgeons, who have not had the opportunity to experience many elective gastric resections. The aim of emergency surgery should be to control the bleeding securely, rather than to prevent ulcer recurrence. Whether gastrectomy or a more conservative approach (under-running of the ulcer, with or without vagotomy and drainage) is superior has been hotly debated over the years. The conservative option is technically less demanding but carries a higher risk of rebleeding in the immediate postoperative period. In practice the choice of operation is determined by the size of the ulcer; small ulcers (less than 2 cm) may be safely under-run (or excised) with little risk of rebleeding, but giant ulcers that penetrate into the pancreas require a technically difficult gastrectomy.

For patients with perforated peptic ulcer the conventional treatment is urgent laparotomy and an omental patch repair. For those under 40 years of age, conservative non-operative management is effective⁷. However, with the laparoscopic revolution, laparoscopic patch repair has become relatively easy and should be within the competence of most general surgeons⁸. Definitive ulcer operations on the other hand, especially in the emergency setting, are likely to be attempted by only a few enthusiasts. Seventy per cent of perforated duodenal ulcers harbour *Helicobacter* infection. Such infection is associated with a higher preponderance of males, dyspepsia, and a much lower incidence of non-steroidal anti-inflammatory drug usage (5.9 per cent) than that found in non-infected patients (45 per cent)⁹. This suggests that perforations caused by *H. pylori* and non-steroidal anti-inflammatory drugs may be two distinct entities, and supports a policy of patch repair followed by eradication of *H. pylori* in those who are infected.

Mechanical gastric outlet obstruction due to chronic scarring from peptic ulceration classically required operative intervention. Non-operative treatment with balloon dilatation has met with limited success; recurrent ulceration and reobstruction occurred in up to 50 per cent of patients after a 3-year follow-up¹⁰. There is little information on the relationship between *H. pylori* and peptic pyloric stenosis. In the authors' unit the incidence of *H. pylori* infection in patients with pyloric stenosis is around 50 per cent (unpublished data). Resolution of gastric outlet obstruction after eradication of infection has been reported¹¹. Whether balloon dilatation plus eradication is adequate treatment for patients with gastric outlet obstruction associated with *H. pylori* infection awaits further study.

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