4.2 Gastric and duodenal ulceration

Peptic ulceration

Overview

Peptic ulceration commonly involves the stomach, duodenum, and lower oesophagus; after gastric surgery it involves the gastro-enterostomy stoma. Healing can be promoted by general measures, Smoking cessation p. 304 and taking antacids and by antisecretory drug treatment, but relapse is common when treatment ceases. Nearly all duodenal ulcers and most gastric ulcers not associated with NSAIDs are caused by *Helicobacter pylori*.

Helicobacter pylori infection

Eradication of *Helicobacter pylori* reduces the recurrence of gastric and duodenal ulcers and the risk of rebleeding. The presence of *H. pylori* should be confirmed before starting eradication treatment. If possible, the antibacterial sensitivity of the organism should be established at the time of endoscopy and biopsy. Acid inhibition combined with antibacterial treatment is highly effective in the eradication of *H. pylori*; reinfection is rare. Antibiotic-associated colitis is an uncommon risk.

Treatment to eradicate *H. pylori* infection in children should be initiated under specialist supervision. One week triple-therapy regimens that comprise omeprazole p. 60, amoxicillin p. 351, and either clarithromycin p. 340 or metronidazole p. 344 are recommended. Resistance to clarithromycin or to metronidazole is much more common than to amoxicillin and can develop during treatment. A regimen containing amoxicillin and clarithromycin is therefore recommended for initial therapy and one containing amoxicillin and metronidazole is recommended for eradication failure or for a child who has been treated with a macrolide for other infections. There is usually no need to continue antisecretory treatment (with a proton pump inhibitor or H_2-receptor antagonist); however, if the ulcer is large, or complicated by haemorrhage or perforation then antisecretory treatment is continued for a further 3 weeks. Lansoprazole p. 59 may be considered if omeprazole is unsuitable. Treatment failure usually indicates antibacterial resistance or poor compliance.

Two-week triple-therapy regimens offer the possibility of higher eradication rates compared to one-week regimens, but adverse effects are common and poor compliance is likely to offset any possible gain.

Two-week dual-therapy regimens using a proton pump inhibitor and a single antibacterial produce low rates of *H. pylori* eradication and are **not** recommended.

See under *NSAID-associated ulcers* for the role of *H. pylori* eradication therapy in children starting or taking NSAIDs.

Test for *Helicobacter pylori*

^{13}C-Urea breath test kits are available for confirming the presence of gastro-duodenal infection with *Helicobacter pylori*. The test involves collection of breath samples before and after ingestion of an oral solution of ^{13}C-urea; the samples are sent for analysis by an appropriate laboratory. The test should not be performed within 4 weeks of treatment with an antibacterial or within 2 weeks of treatment with an antisecretory drug. A specific ^{13}C-Urea breath test kit for children is available (*Helicobacter Test INFAI for children of the age* 3–11®). However the appropriateness of testing for *H. pylori* infection in children has not been established. Breath, saliva, faecal, and urine tests for *H. pylori* are frequently unreliable in children; the most accurate method of diagnosis is endoscopy with biopsy.

NSAID-associated ulcers

Gastro-intestinal bleeding and ulceration can occur with NSAID use. Whenever possible, NSAIDs should be **withdrawn** if an ulcer occurs.

Children at high risk of developing gastro-intestinal complications with a NSAID include those with a history of peptic ulcer disease or serious upper gastro-intestinal complication, those taking other medicines that increase the risk of upper gastro-intestinal side-effects, or those with serious co-morbidity. In children at risk of ulceration, a proton pump inhibitor can be considered for protection against gastric and duodenal ulcers associated with non-selective NSAIDs; high dose ranitidine p. 56 is an alternative.

NSAID use and *H. pylori* infection are independent risk factors for gastro-intestinal bleeding and ulceration. In children already taking a NSAID, eradication of *H. pylori* is unlikely to reduce the risk of NSAID-induced bleeding or ulceration. However, in children about to start long-term NSAID treatment who are *H. pylori* positive and have dyspepsia or a history of gastric or duodenal ulcer, eradication of *H. pylori* may reduce the overall risk of ulceration.

If the NSAID can be *discontinued* in a child who has developed an ulcer, a proton pump inhibitor usually produces the most rapid healing; alternatively the ulcer can be treated with an H_2-receptor antagonist.

If *NSAID treatment needs to continue*, the ulcer is treated with a proton pump inhibitor.

GASTROPROTECTIVE COMPLEXES AND CHELATORS

Chelates and complexes

Sucralfate

Sucralfate below is a complex of aluminium hydroxide and sulfated sucrose that appears to act by protecting the mucosa from acid-pepsin attack; it has minimal antacid properties.

Sucralfate

- **INDICATIONS AND DOSE**

Benign gastric ulceration | Benign duodenal ulceration

- ▶ BY MOUTH
- Child 1 month–1 year: 250 mg 4–6 times a day
- Child 2–11 years: 500 mg 4–6 times a day
- Child 12–14 years: 1 g 4–6 times a day
- Child 15–17 years: 2 g twice daily, dose to be taken on rising and at bedtime, alternatively 1 g 4 times a day for 4–6 weeks, or in resistant cases up to 12 weeks, dose to be taken 1 hour before meals and at bedtime; maximum 8 g per day

Prophylaxis of stress ulceration in child under intensive care

- ▶ BY MOUTH
- Child 1 month–1 year: 250 mg 4–6 times a day
- Child 2–11 years: 500 mg 4–6 times a day
- Child 12–14 years: 1 g 4–6 times a day
- Child 15–17 years: 1 g 6 times a day; maximum 8 g per day

- **UNLICENSED USE** Not licensed for use in children under 15 years. Tablets not licensed for prophylaxis of stress ulceration.
- **CAUTIONS** Patients under intensive care (**Important:** reports of bezoar formation)