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Letter to the Editor:

Hepatic Penetration of a Single Large Duodenal Ulcer

(Accepted 25th July 2005)

Sir,

We present a rare case of erosion of a duodenal ulcer into the liver in a gentleman with few risk factors for peptic ulcer disease (PUD) and on long term protium pump inhibitor (PPI).

PRESENTATION AND MANAGEMENT

An 85 year old male, who had been on omeprazole for 18 months, was admitted with sub acute small bowel obstruction of 2 weeks duration. His past medical history was Type 2 diabetes mellitus and warfarin for atrial fibrillation. Prior to commencement of omeprazole, he had been investigated for weight loss with positive faecal occult blood test positive, an oesophago-gastro-duodenoscopy (OGD) which revealed a very mild streaky gastritis and negative H. pylori test, and a complete colonoscopy that showed three histologically confirmed metaplastic polyps. He had no history of PUD, was not on NSAID's or steroids, smoked about 15 cigarettes / day and drank alcohol occasionally.

He subsequently developed acute small bowel obstruction necessitating an emergency laparotomy and was found to have an obstructing caecal tumour; a right hemicolectomy and ileocolic anastomosis was performed. Histology confirmed an adenocarcinoma. On post operative day fifteen he started passing moderate amounts of melena; there were no features suggestive of an acute abdomen. He was still haemodynamically stable and liver function tests essentially normal though haemoglobin had dropped from 10g/dl to 8g/dl by the next day when he had an OGD. A deep copiously bleeding ulcer approximately 2cm in diameter was found on the anterior wall of the first part of the duodenum.

A laparotomy was performed after unsuccessful attempts at injection with adrenaline. The ulcer was then found to have deeply eroded into the posterior surface of the medial inferior segment of the liver. This bleeding defect was adequately controlled by figure-of-eight liver stitches but he suddenly became hypotensive from a myocardial infarct, failed to respond to resuscitative measures and died intra-operatively.

DISCUSSION

Most perforations of a duodenal ulcer into the liver are 'silent' with absent or minimal abdominal pain.[1] Diagnosis may be by histological examination of endoscopic biopsies[2] but most only become only obvious at laparotomy or autopsy.[3]

PPIs are the most effective drugs for the suppression of gastric acid production;[4] in duodenal ulcers, omeprazole 20mg daily produced healing rates of 90 – 100% after 4 weeks.[5] PPIs are also efficacious in prevention of bleeding from stress ulcers.[6]

Possibilities in this unusual case are that the ulcer was missed during the initial OGD, that he developed a fresh ulcer despite PPIs, or that this was a case of a single large penetrating stress ulcer. Since in any case PPIs were meant to be effective, this may have been case of resistance to omeprazole. Claessens *et al* identified previous use of PPIs, heavy smoking and age over 60 years as factors consistently associated with non response to PPIs.[7] This patient had all three.

CONCLUSION

Resistance to PPIs is uncommon[7] but may occur in the elderly smoker post major surgery, leading to potentially serious complications.

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