At laparotomy about 3 litres of blood and clot were found in the peritoneal cavity. The sources of bleeding were a macerated spleen and a raw, bleeding edge of the left lobe of liver. There was also a haematoma in the transverse mesocolon and some bruising in the mesentery of the small bowel near the duodenojejunal flexure. There was obvious Crohn's disease of the distal half of the transverse colon, the descending colon, and the sigmoid colon. Vascular omental adhesions were present between the transverse colon and the spleen and liver, and these appeared to have been damaged. After evacuating the blood from the peritoneal cavity the splenic hilum was ligated and the necrotic splenic tissue removed. Haemostasis on the liver was secured with cauterisation and the application of haemostatic sponge. Microscopy of the pulped splenic tissue showed haemorrhage and necrosis but no other specific abnormality. Recovery was satisfactory and the patient's symptoms have now been controlled with a combination of prednisolone and azathioprine.

Comment

Telmos and Mittal¹ reported a case of splenic rupture after colonoscopy that was unrecognised for three days. The examination had been technically difficult and had ended at the transverse colon. In our case the colonoscopy was not difficult but the bowel was affected by Crohn's disease and the injuries to the spleen and liver resulted from rupture of pre-existing inflammatory adhesions between the colon and these organs. Other reports of such a case are hard to find. Smith recorded no splenic injury in a review of nearly 8000 colonoscopies.² Perhaps patients with Crohn's colitis are at greater risk.

We thank Dr R A Parkins for permission to report details of a patient under his care.

¹ Telmos, A J, and Mittal, V K, *Journal of the American Medical Association*, 1977, **237**, 2718.

² Smith, L E, *Diseases of the Colon and Rectum*, 1975, **18**, 214.

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Benign gastric ulceration with pernicious anaemia

Since 1910 Schwartz's dictum "no acid—no ulcer"¹ has only rarely been found to be false and it continues to be of clinical value. Indeed, the absence of peptic ulceration in pernicious anaemia has been regarded as evidence that acid is necessary for the development of gastric ulcers. Rare examples of achlorhydric peptic ulceration have been reported but there has been doubt about the adequacy of the maximal stimulus.² A patient with a peptic ulcer associated with long-standing pernicious anaemia, hypothyroidism, and insulin-dependent diabetes has also been reported.³ We describe a further case in which pernicious anaemia presented with symptoms of anaemia and pyloric obstruction from a benign gastric ulcer.

Case report

A 63-year-old housewife presented with a four-month history of fatigue, breathlessness, anorexia, weight loss of 10 kg, and intermittent vomiting of food taken several hours earlier. Her father had died from an anaemia treated with liver extract. She denied taking alcohol or analgesics. Her general practitioner had found an anaemia with a low serum vitamin B₁₂ concentration and a prepyloric ulcer on barium meal examination. She was referred to us for endoscopy and haematological assessment. On examination she was anaemic. A gastric succussion splash could be elicited. Endoscopy confirmed superficial and chronic ulceration in the prepyloric region with deformity. The instrument could not be passed through the pylorus.

Investigations showed a haemoglobin of 7.2 g/dl with mean corpuscular volume (MCV) 84 fl and mean corpuscular haemoglobin (MCH) 24.7 pg and platelets of $258 \times 10^9/l$ ($258\,000/mm^3$). The blood film was dimorphic and the serum B₁₂ concentration 106 ng/l (normal 170–1000 ng/l). Serum iron concentration was 4 $\mu\text{mol/l}$ (22.3 $\mu\text{g}/100\text{ ml}$) (normal 14–22 $\mu\text{mol/l}$ (78–2123

Gastric secretory studies in a case of gastric ulceration with pernicious anaemia

Investigation	Preoperative	Postoperative
Basal acid output		
Total volume in 1 hour	17 ml	32 ml
Minimum pH	7.4	7.0
Maximum acid output to pentagastrin (0.6 $\mu\text{g/kg}$)		
Total volume in 1 hour	51 ml	38 ml*
Minimum pH	8.4	8.0
Schilling test		
Without intrinsic factor	1.6%	1.2%
With intrinsic factor†		
(1) 30-mg dose		5.3%
(2) 300-mg dose		13.1%

*No intrinsic factor detected on analysis of aspirated secretions.

†Hog intrinsic factor, Armour Pharmaceuticals.

$\mu\text{g}/100\text{ ml}$) with a total iron binding capacity of 76 $\mu\text{mol/l}$ (424 $\mu\text{g}/100\text{ ml}$) (normal 50–64 $\mu\text{mol/l}$ (279–357 $\mu\text{g}/100\text{ ml}$)). The serum folate concentration was twice estimated at 1.4 and 2.2 $\mu\text{g/l}$ (normal $>2.0\text{ }\mu\text{g/l}$) and the red cell folate concentration was only slightly reduced at 141 $\mu\text{g/l}$ (normal 160–640 $\mu\text{g/l}$). Bone marrow examination confirmed megaloblastic erythropoiesis with absence of stainable iron. Stool was positive for occult blood. Serum was positive for parietal cell antibody but negative for intrinsic factor antibody. Blood urea and electrolyte concentration, and the results of routine liver function tests were all normal. The results of gastric secretion studies are shown in the table. Except for the first Schilling test with intrinsic factor, performed 10 days after surgery, the postoperative studies were done after three months of vitamin B₁₂ and iron treatment when the haemoglobin was 13 g/dl. Biopsy specimens of tissue taken during two endoscopies showed only inflammatory changes but it was felt that malignancy could not be excluded. Polya gastrectomy was therefore performed. Macroscopically, the specimen showed a 0.5-cm diameter prepyloric ulcer, but extensive sectioning showed only superficial gastritis and intestinal metaplasia without malignant change. One year later the patient was well on vitamin B₁₂ injections. Endoscopy of the gastric remnant showed pronounced atrophic gastritis, confirmed by histological examination of a biopsy specimen. A further Schilling test produced a urine recovery of 1.1% of the administered vitamin B₁₂.

Comment

Although the anaemia seems to have been partly due to iron deficiency secondary to bleeding from the ulcer, the abnormal Schilling test corrected by intrinsic factor administration, in the presence of achlorhydria to pentagastrin, substantiates the diagnosis of "Addisonian" pernicious anaemia. The postoperative secretory studies confirm that these were not transient abnormalities related to the ulceration and gastritis or to iron deficiency, since a Polya gastrectomy alone would not reduce secretions to such levels. It has been suggested that previous cases of benign ulceration in the absence of demonstrable acid secretion might reflect methods not sufficiently sensitive to stimulate or detect acid secretion.² The presence of pernicious anaemia in our case seems to resolve this issue, since even the strongest of stimuli will fail to produce any acid secretion in pernicious anaemia. In the absence of alcohol or drugs bile reflux was probably the cause of the ulceration. Seemingly, therefore, our case and others weaken Schwartz's dictum. But in practice we agree with Isenberg⁴ that gastric ulceration with achlorhydria should be managed as gastric carcinoma, since a possible intramucosal carcinoma might be detected only on examining the resected specimen.⁵

We thank Drs N C Allan and Anne Ferguson for their advice and permission to report this case.

¹ Schwartz, K, *Beitrage zur klinischen Chirurgie*, 1910, **67**, 96.

² *British Medical Journal*, 1975, **4**, 426.

³ Manier, J W, and Beltaos, E, *Gastroenterology*, 1975, **69**, 744.

⁴ Isenberg, J K, et al, *New England Journal of Medicine*, 1971, **285**, 620.

⁵ Taylor, R H, et al, *Lancet*, 1978, **1**, 686.

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