Barrett's oesophagus and development of dysplasia and adenocarcinoma

EDITOR—Itfikhar et al (Gut 1992; 33: 1155–8) present results from a 15 year prospective study of endoscopic surveillance of 102 patients with columnar lined epithelium (for example, Barrett's oesophagus). The aim of the study was to identify any significant risk factors for the subsequent development of adenocarcinoma. Data are presented suggesting that the length of columnar lined oesophagus was considerably longer in patients with dysplasia. None of the patients with dysplasia had a columnar lined oesophagus of less than 5 cm. The authors conclude that the length of Barrett's oesophagus is a significant risk factor in the development of dysplasia and subsequent carcinoma and recommend intensive follow up of patients with Barrett's oesophagus greater than 8 cm in length.

The results and conclusions of the study are inappropriate given the exclusion of patients with less than 5 cm of circumferential Barrett's oesophagus. Adenocarcinoma has been reported in tongues or short segments of Barrett's oesophagus.1 At least 52 per cent of a series of 28 resected specimens with adenocarcinomas centred in the oesophagus had a length of Barrett's less than 5 cm. Additionally, adenocarcinomas occurring near the gastro-oesophageal junction may arise from small areas of specialised epithelium, which may be obliterated and not discovered.

It would be inappropriate to ignore patients with the potential for dysplastic change when short segment Barrett's oesophagus is found at endoscopy. Systematic biopsies should be taken and subsequent follow up should not differ from those patients with longer segment Barrett's oesophagus unless appropriately conducted studies show a lesser risk of cancer.

Screening and management of familial adenomatous polyposis

EDITOR—Tait et al advocate annual colonscopy as the ideal screening method for first degree relatives who carry the gene for familial adenomatous polyposis (FAP). (Correspondence on Letter to the Editor and reply Gut 1993; 34: 576.) Bradburn and Rhodes on the other hand make the case for the selective use of colonoscopy, recommending it only for those at high risk for FAP but without obvious polyps (or microadenomas) by their late teens, and in those whom prophylactic colectomy has been delayed. This selective approach is suggested to minimise the morbidity and mortality associated with colonoscopy.

We report on two cases of colonoscopic morbidity, which, though anecdotal, add 'meat to the bones' of the present discussion.

Case 1: A 12 year old son of a patient with FAP was found to have polyps at sigmoidoscopy. Histological examination showed these to be adenomas. At colonoscopy at 16 years, the bowel was perforated. A laparotomy was performed for peritonitis and a defunctioning colostomy was created; this was closed six months after laparotomy. He was referred to St Mark's Hospital after his father died from an upper gastrointestinal malignancy, but refused to attend for further hospital appointments or to have surgery, and is now under psychiatric counselling.

Case 2: A 15 year old son of a patient with FAP was found to have rectal adenomas at colonoscopy. Annual colonoscopies were then performed, and at the age of 19 years, 20 polyps (size 2 mm) were removed. After this procedure the patient became unwell. A laparotomy was performed and a defunctioning colostomy was drained. He was referred six months later to St Mark's Hospital for definitive surgery. At laparotomy a large mesenteric desmoid tumour (not apparent at the first operation) was found. Neither the colon nor the desmoid tumour was able to be removed.

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