Increasing incidence of Barrett’s oesophagus: education, enthusiasm, or epidemiology?

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For unknown reasons, the incidence of oesophageal adenocarcinoma is rising rapidly in the UK1 and the USA. In particular, in Scotland, the incidence of oesophageal adenocarcinoma has risen from 3·0 per 100 000 in 1976 (age-standardised rate) to 4·6 per 100 000 in 1989.² Barrett’s oesophagus is a premalignant metaplastic change in the lining of the distal oesophagus and is the only recognised risk factor for the development of oesophageal adenocarcinoma conferring a 30–125 times increased risk. It might be suspected that this increase of oesophageal adenocarcinoma is mirrored by a similar rise in the incidence of Barrett’s oesophagus, however, currently there are no data to substantiate this. We have studied the incidence of endoscopically diagnosed Barrett’s oesophagus in Tayside to determine whether there is an increasing incidence of Barrett’s oesophagus in our population.

The incidence of endoscopically diagnosed Barrett’s oesophagus was determined by searching the endoscopy database between the years 1980 and 1993. This represented 53 433 upper GI endoscopies performed in our population of 384 000 over the 13-year period. Barrett’s oesophagus was defined as either a positive diagnosis of Barrett’s determined by the endoscopist, or if the squamocolumnar junction was equal to, or greater than, 3 cm above the oesophago-gastric junction. With these criteria 961 new cases of Barrett’s oesophagus were identified, representing 18 new cases per 100 000 per year (18 cases per 1000 endoscopies). The incidence was, however, not constant throughout the study period but showed a substantial rise from 1/100 000 per year (1·4/1000 endoscopies) in 1980–1981 to 48/100 000 per year (42·7/1000 endoscopies) in 1992–93 (see figure). When only cases with histological confirmation were considered, the incidence from 1992–93 was 18/100 000 per year (16·5/1000 endoscopies). This latter figure probably underestimates the incidence of Barrett’s oesophagus due to the failure of some endoscopists to confirm the endoscopic finding of Barrett’s oesophagus histologically.

Our data show a substantial rise in the incidence of Barrett’s oesophagus in the Tayside population. The reason for this changing incidence is unknown. The initial low incidence may be partly explained by a failure to recognise Barrett’s oesophagus endoscopically. With subsequent education on the endoscopic features of Barrett’s there may have been a resulting increase in endoscopic diagnosis. Enthusiasm towards making the endoscopic diagnosis of Barrett’s, due to local interest in the condition, may have also contributed to the rising incidence. Changes in the GP endoscopic referral pattern, resulting in increased endoscopy of patients with symptoms of gastro-oesophageal reflux disease, a condition known to be related to Barrett’s oesophagus³ may be another factor. However, we believe that there is a true increase in incidence in this condition, the cause of which is unknown. Irrespective of the cause, this increasing incidence has implications for endoscopic workload, particularly if surveillance for premalignant change in these patients is to be undertaken.³ In addition, it is of concern if it is a harbinger of a further increase in the incidence of oesophageal adenocarcinoma.


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Fixation of urinary sediment

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In patients with haematuria, the presence of dysmorphic erythrocytes and erythrocyte casts in the urine is a sign of a glomerular source of the bleeding.¹ For a reliable interpretation of the urinary sediment, a freshly voided urine sample is required to avoid changes of cellular morphology and disappearance of cells and casts. Several fixation methods have been proposed, but none has gained wide acceptance.²³ We found that using CellFIX, a formaldehyde-based fixative, it was possible to preserve the sediment. We compared sediments from freshly voided urine with sediments fixed from the same sample.

Based on the results of a previous study of fresh urinary sediments from 107 patients with known nephropathological or urological causes of haematuria, we chose a value of 40% dysmorphic erythrocytes as the cut-off point for differentiating between glomerular and non-glomerular haematuria. By including the presence of erythrocyte casts as an additional criterion in that study, the sensitivity for a diagnosis of glomerular haematuria was 88·1% and the specificity 100%.¹ Urinary sediments were prepared by centrifuging 10 mL of urine for 5 min at 1500 rpm. The supernatant was decanted until less than 0·5 mL remained. One sample was examined by an experienced nephrologist within 3 h after voiding. Four drops of a second sample (about 0·2 mL) were put into a 2 mL plastic vial containing 0·2 mL of CellFIX (Becton Dickinson). The
Endotracheal tube caecostomy

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Although the use of a caecostomy seems to be declining, it remains a valuable option. We describe a new approach for the creation and subsequent closure of a temporary caecostomy. A purse-string is placed in the caecum and the endotracheal tube and its balloon port are passed through the anterior abdominal wall, secured to the skin and connected to a urine bag. The caecal wall is then sutured to the parietal peritoneum at a convenient position. On removal of the tube, the external opening of the fistula is plugged with a table-tennis ball. A stoma appliance is comfortably worn over this (figure) until the effluent dries up.

We treated seven patients over three years. All caecostomy fistulae have healed without complications and were dry within 2 weeks of removal of the tube. At a median follow-up of 10 months there has been no recurrent fistulation to the anterior abdominal wall.

The usual approach in the formation of a caecostomy is to use a Foley catheter as the method of decompression. However, even the larger diameter catheters are prone to blockage, may be difficult to irrigate and can be complicated by leakage and/or subcutaneous collections. An endotracheal tube, with its wider and rigid nature, avoids these problems. Our stoma care nurses also suggest that skin irritation is minimised. On removal of the endotracheal tube, the contour of a table-tennis ball fits snugly over the caecostomy fistula and acts as a valve allowing the escape of gas but hindering faecal loss. To date the fistulas have all closed quickly as compared with when Foley catheters are used where closure may take 6 or more weeks. When inoperable large bowel obstruction is encountered and the patient is not expected to survive more than a few days we feel that an endotracheal-tube caecostomy allows a more comfortable and dignified outcome than decompression with a stoma.


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