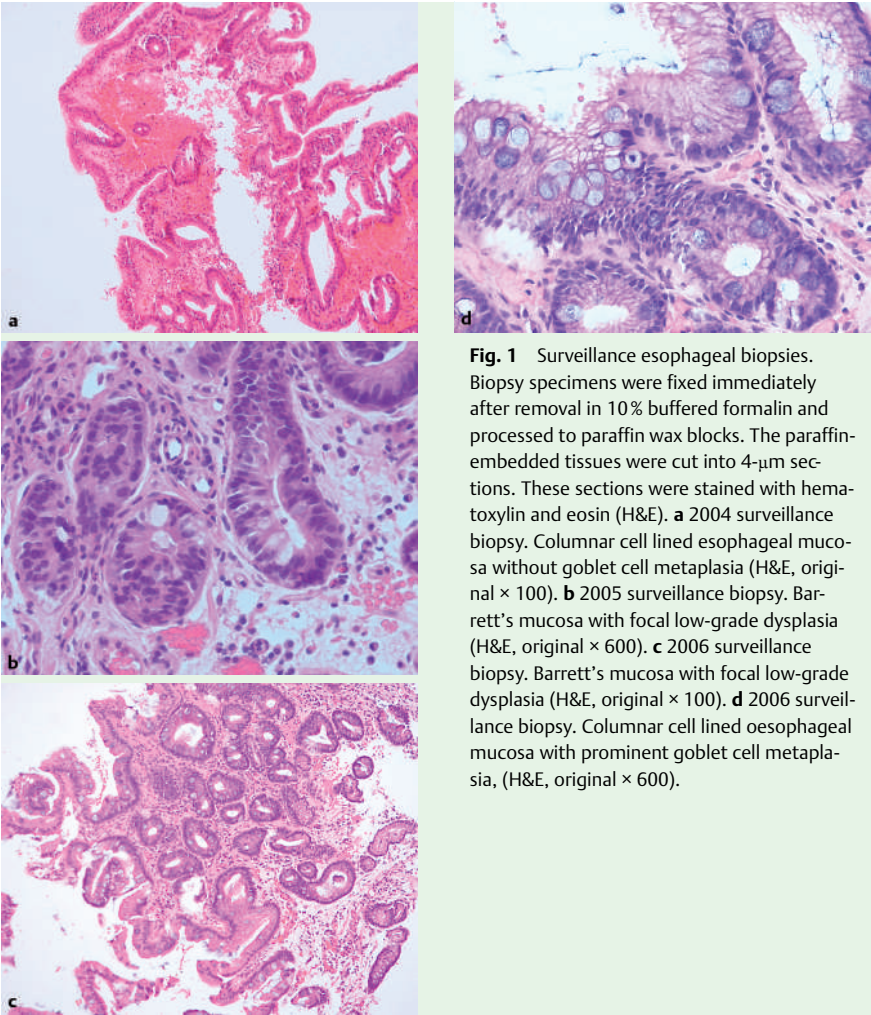


Progression of Barrett’s esophagus to adenocarcinoma despite antireflux surgery

Table 1 Endoscopic surveillance and histologic findings

Year and procedure	Histology
1996 Laparoscopic Nissen fundoplication	
2002 Esophagogastroduodenoscopy	Ulcer base material with gastric type mucosa
2003 Esophagogastroduodenoscopy Redo laparoscopic Nissen fundoplication	Intense esophageal inflammation with ulceration
2004 Esophagogastroduodenoscopy	Barrett’s esophagus
2005 Esophagogastroduodenoscopy	Adenocarcinoma arising in Barrett’s esophagus



In 2004, a 63-year-old patient was diagnosed with Barrett’s esophagitis. A laparoscopic Nissen fundoplication was carried out as previously described, using a five port technique [1]. Following this, annual surveillance endoscopy was performed by an experienced endoscopist, and multiple biopsies (> 8) were taken on each occasion. The patient developed recurrent reflux after 6 years, and underwent successful repeat surgery. Intra-operatively, it was confirmed that the previous wrap had broken down. Fundoplication was repeated and the hiatus reinforced with a polytetrafluoroethylene (PTFE) patch. The proximal stomach was also anchored to the diaphragm. Subsequent annual surveillance endoscopy demonstrated persistent Barrett’s esophagitis with ulceration, but no dysplasia. (Table 1, Fig. 1). On routine surveillance in October 2005 there was evidence of change in macroscopic appearances at the gastro-esophageal junction. This was accompanied by subtle dysphagic symptoms. Biopsies confirmed adenocarcinoma (Fig. 2). A high-resolution CT thorax, abdomen, and pelvis, and whole body positron emission tomography were performed, both of which suggested that the disease was confined to the distal esophagus. The patient underwent a two-stage transhiatal esophagectomy. Histopathological examination confirmed an infiltrating moderately differentiated adenocarcinoma arising in an area of extensive Barrett’s, extending through the full thickness of the wall, with extensive surface ulceration. Metastatic tumor was present in 4 of 23 nodes and lymphovascular invasion was observed (pT3 N1 MX) (Fig. 2). Postoperatively all histology dating from identification of Barrett’s esophagitis was reviewed, and only low-grade dysplasia was identified. The incidence of adenocarcinoma in Barrett’s esophagus is low [2,3], and guidelines recommending frequency of endoscopy do not exist. We believe this case exhibits the need for long-term endoscopic surveillance in patients with a history of Barrett’s esophagitis. This patient developed an interval tumor in a setting of yearly surveillance endoscopy, suggesting that despite annual surveillance in high-risk patients and antireflux surgery, interim progression to adenocarcinoma may occur.

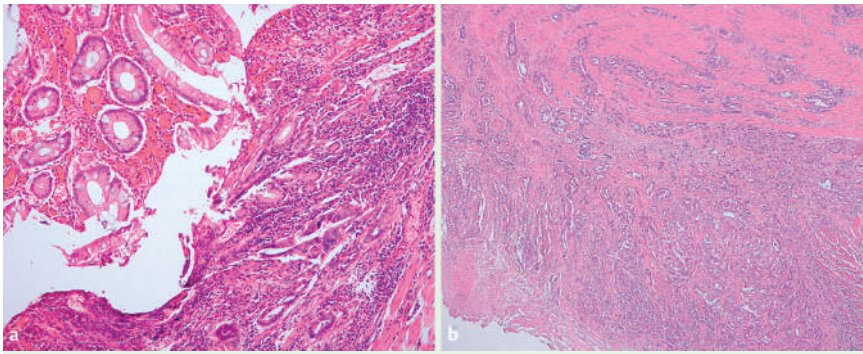


Fig. 2 Adenocarcinoma arising in Barrett's esophagus. **a** Junction between carcinoma and Barrett's mucosa. **b** Invasive adenocarcinoma of the lower esophagus arising from Barrett's mucosa (both H&E, original $\times 40$). Biopsy specimens were fixed immediately after removal in

10% buffered formalin and processed to paraffin wax blocks. The paraffin-embedded tissues were cut into 4- μ m sections. These sections were stained with H&E. Resection specimens were gross and sampled. Tissues sampled were processed in the same way as the biopsies.

Bibliography

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