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 esophagus 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 transthoracic 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.

### Table 1: Endoscopic surveillance and histologic findings

<table>
<thead>
<tr>
<th>Year and procedure</th>
<th>Histology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1996 Laparoscopic Nissen fundoplication</td>
<td></td>
</tr>
<tr>
<td>2002 Esophagogastroduodenoscopy</td>
<td>Ulcer base material with gastric type mucosa</td>
</tr>
<tr>
<td>2003 Esophagogastroduodenoscopy</td>
<td>Intense esophageal inflammation with ulceration</td>
</tr>
<tr>
<td>2004 Esophagogastroduodenoscopy</td>
<td>Barrett’s esophagus</td>
</tr>
<tr>
<td>2005 Esophagogastroduodenoscopy</td>
<td>Adenocarcinoma arising in Barrett’s esophagus</td>
</tr>
</tbody>
</table>

**Fig. 1** Surveillance esophageal biopsies. 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-μm sections. These sections were stained with hematoxylin and eosin (H&E). 

- **a** 2004 surveillance biopsy. Columnar cell lined esophageal mucosa without goblet cell metaplasia (H&E, original × 100).
- **b** 2005 surveillance biopsy. Barrett’s mucosa with focal low-grade dysplasia (H&E, original × 600).
- **c** 2006 surveillance biopsy. Barrett’s mucosa with focal low-grade dysplasia (H&E, original × 100).
- **d** 2006 surveillance biopsy. Columnar cell lined oesophageal mucosa with prominent goblet cell metaplasia (H&E, original × 600).
Department of Surgery, University College Hospital Galway, Galway, Ireland

References


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 × 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-μm 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.