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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 transthiatal 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>
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</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>Redo laparoscopic Nissen fundoplication</td>
<td></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>

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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.

References

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