Surgical therapy for Barrett’s esophagus: prevention, protection and excision*

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Barrett’s esophagus is a complication of gastroesophageal reflux disease and is a manifestation of chronic tissue injury. It is characterized by the presence of intestinal metaplasia in an esophagus lined with cardiac-type mucosa; alternatively described as a columnar-lined esophagus with intestinal metaplasia or Barrett’s mucosal. When present, the abnormal mucosa confers a more than 30 to 125-fold risk of progression to esophageal adenocarcinoma, which emerges at a rate of about 1–2 cancers per 100 patient-years of follow-up.1–3

Current dogma states that Barrett’s esophagus develops quickly to its full extent, with little subsequent increase in length.4 This is based on data from patients with only long segments of Barrett’s mucosa within the tubular esophagus, i.e. > 3 cm. It is now accepted that intestinal metaplasia occurs in segments of cardiac-type mucosa that are less than 3 cm in length, and that intestinal metaplasia can even occur in extremely small segments of cardiac-type mucosa located just below the squamous epithelium of an endoscopically normal appearing gastroesophageal junction.5–7

It is known that cardiac-type mucosal at the gastroesophageal junction is almost always inflamed, as evident by an inflammatory infiltrate on microscopy, and is exposed to increased acid exposure on 24 h esophageal pH monitoring.7 Consequently, this finding is referred to as carditis and is one of the early signs of gastroesophageal reflux disease. The length of the inflamed cardiac-type mucosa has been shown to be related to the degree of esophageal acid exposure, the level of lower esophageal sphincter pressure and the length of the sphincter exposed to the abdominal pressure environment.8 It is now known that the segments of cardiac-type mucosa can show intestinal metaplasia on biopsy and that the prevalence of this finding is directly related to the length of the segment.8 On the bases of these studies, it is hypothesized that Barrett’s esophagus spreads progressively upwards in a stepwise fashion. Initially, short segments of inflamed cardiac-type mucosa develop below an endoscopically normal appearing gastroesophageal junction. The inflammation causes a loss in the pressure and length of the lower esophageal sphincter resulting in greater esophageal acid exposure with extension of inflamed cardiac-type mucosa into the distal 3 cm of the esophagus. With further loss of sphincter pressure and length, the process extends higher into the esophageal body. With time, and under proper conditions, intestinalization of the cardiac mucosa can occur with a prevalence that is related to the duration and functional severity of the disease. It is the occurrence of intestinalization that is associated with a cancer risk and is required for the diagnosis of Barrett’s esophagus. Understanding the disease in this way has strong implications regarding its therapy.

Based on the above, the goals in the management of Barrett’s esophagus are as follows: (i) to prevent the development of the metaplastic epithelium by stopping reflux early in the disease process; (ii) to promote or induce healing or regression of the metaplastic epithelium such that the cancer risk mucosal change (intestinal metaplasia) is eliminated; and (iii) to induce a quiescence of the intestinalized metaplastic epithelium and halt its progression to dysplasia and cancer. The goal of therapy for Barrett’s esophagus according to the American College of Gastroenterology is to ‘control the symptoms of gastroesophageal reflux disease’. They state that ‘symptom relief is an appropriate end-point for the therapy of Barrett’s esophagus’.9 This approach has been shown to be ineffective, in that the eradication of symptoms cannot be equated with elimination of
reflux, nor has it been able to reliably achieve the second and third management goals.\textsuperscript{10–14} Hameeteman and colleagues from the Netherlands reported that out of 50 patients with a columnar-lined esophagus who were treated medically and followed from 1.5 to 14 years (mean 5.2 years), initially only 34 had intestinal metaplasia on biopsy of the columnar mucosa. At the completion of the study, 37 patients had intestinal metaplasia, suggesting that three patients developed the cancer risk epithelium during the 5-year study period. In addition, at the start of the study, six patients had low-grade dysplasia and one patient had high-grade dysplasia. By the end of the 5-year study period, 10 patients had low-grade dysplasia, three had high-grade dysplasia, and five had adenocarcinoma.\textsuperscript{1} In another study, Sharma et al. followed 32 medically treated patients with short-segment Barrett’s (mean length = 1.5 cm) for a mean of 36.9 months and found a 5.7\% annual incidence of progression to dysplasia.\textsuperscript{15} During the 98 patient-years of follow-up in their series, two patients developed high-grade dysplasia, and five had adenocarcinoma.\textsuperscript{1} Another study by Thor and Silander\textsuperscript{25} and Johansson and colleagues\textsuperscript{26} both mention healing of esophagitis, and/or promotility agents. They commented that most patients developed dysplasia while on acid suppression medication, and they concluded that medical treatment does not prevent the development of dysplasia.

Additionally, Lagergren and colleagues reported that the risk of esophageal adenocarcinoma was increased nearly eightfold among persons in whom heartburn, regurgitation or both occurred at least once a week compared with persons without these symptoms. It is important to note that the risk of esophageal adenocarcinoma was three times higher among patients who used medication for symptoms of reflux compared with those who did not use any antireflux medication.\textsuperscript{16}

**EVIDENCE THAT SURGERY PREVENTS THE DEVELOPMENT OF BARRETT’S ESOPHAGUS**

As Barrett’s esophagus is associated with gastroesophageal reflux disease, it would seem logical that stopping reflux by a surgical fundoplication should effectively prevent the development of Barrett’s esophagus in patients who have gastroesophageal reflux without Barrett’s. Antireflux surgery has been shown to restore lower esophageal sphincter (LES) function and abolish reflux of gastric content, acid or bile, into the esophagus.\textsuperscript{17} Consequently, an antireflux operation ends the repetitive injury to the esophageal mucosa. Randomized clinical studies have confirmed superior control of reflux following antireflux surgery compared with medical therapy, and have shown the surgical antireflux procedure to be safe and durable.\textsuperscript{12,18–20} Further, recent advances in minimally invasive surgical technology have shown that the procedure can be performed laparoscopically with the same outcome, less morbidity, a shorter hospital stay and a more rapid, full recovery.\textsuperscript{21–23}

Very few authors have recorded the presence of Barrett’s metaplasia following antireflux surgery when it was absent preoperatively. The long-term study of Luostarinen and colleagues managed to perform endoscopy 20 years after Nissen fundoplication in 21 patients. In 15 patients, the fundoplication appeared to be intact, and in six it appeared to be defective.\textsuperscript{24} Two out of the 15 with intact fundoplications were found to have Barrett’s esophagus on long-term follow-up, but the authors admit that these two patients did not have preoperative biopsies, and hence, it cannot be concluded that Barrett’s esophagus developed (Table 1). In contrast, five out of the six patients with defective fundoplication developed Barrett’s esophagus during the follow-up period. This report emphasizes that the patient with intact fundoplication had the potential to develop Barrett’s and did not, and that the benefits of surgery are dependent upon performing an effective and durable repair.

A review of studies with endoscopic follow-up of patients treated using antireflux surgery showed that most concentrate on the healing of esophagitis. Reports by Thor and Silander\textsuperscript{25} and Johansson and colleagues\textsuperscript{26} both mention healing of esophagitis, and neither report identifies a single case of Barrett’s esophagus developing within 5 years after an antireflux operation, which was not present before the operation (Table 1).

Despite the limitation of these studies, it appears that the *de novo* development of Barrett’s esophagus

<table>
<thead>
<tr>
<th>Author</th>
<th>n</th>
<th>No. with columnar-lined esophagus</th>
<th>Follow-up years</th>
<th>No. developing Barrett’s esophagus</th>
</tr>
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<tbody>
<tr>
<td>Luostarinen et al.\textsuperscript{24}</td>
<td>15*</td>
<td>0</td>
<td>20</td>
<td>2†</td>
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<tr>
<td>Thor and Silander\textsuperscript{25}</td>
<td>31</td>
<td>4</td>
<td>5</td>
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<td>Johansson et al.\textsuperscript{26}</td>
<td>33</td>
<td>4</td>
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<tr>
<td>Total</td>
<td>79</td>
<td>8</td>
<td>5–20</td>
<td>2</td>
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\*All intact fundoplications.

†No preoperative biopsy in these patients to exclude Barrett’s esophagus.
is exceedingly rare in patients who have had effective antireflux surgery. This is in marked contrast to long-term medical treatment. A recent report from Austria found that up to 34% of patients on long-term acid suppression developed Barrett’s esophagus while on therapy.27 Seven out of 12 patients (58%) on continuous omeprazole therapy developed Barrett’s esophagus. Consequently, a policy of correctly performing an effective fundoplication early in the course of the disease would likely reduce the incidence of Barrett’s esophagus in the future. Risk factors for Barrett’s esophagus, such as young age of onset, long duration of symptoms, persistent esophagitis, defective lower esophageal sphincter and mixed duodenogastric reflux should encourage early operation.

EVIDENCE THAT ANTIREFLUX SURGERY PROMOTES OR INDUCES HEALING OR REGRESSION OF THE METAPLASTIC EPITHELIUM SUCH THAT THE CANCER RISK EPITHELIUM (INTESTINAL METAPLASIA) IS ELIMINATED

Does Barrett’s mucosa regress following antireflux surgery? Brand, in 1980, was the first to describe complete regression of all metaplastic epithelium in four out of 10 patients with Barrett’s following an antireflux procedure.28 Subsequently, most reports have demonstrated that although some regression of the length of Barrett’s is common, complete regression after an antireflux procedure occurs only occasionally, but progression in the length of Barrett’s rarely occurs. A review of the English language literature since 1977 documents follow-up on 340 patients after antireflux surgery. Complete regression occurred in only 13 patients, whereas in 256 out of the 340 patients (75%), the Barrett’s epithelium remained unchanged.29 Therefore, regression of traditional Barrett’s cannot be reliably predicted or anticipated following antireflux surgery.

In contrast to the unreliable regression of the traditional >3 cm segments of Barrett’s, we recently showed that 73% of patients with intestinal metaplasia at the gastroesophageal junction had complete regression of the intestinal metaplasia component of the Barrett’s mucosa, i.e. the at-risk mucosa, following antireflux surgery.30 In comparison, only 4% of patients with a long segment of Barrett’s had loss of intestinal metaplasia after an antireflux procedure. Recently, Low and colleagues also reported the loss of intestinal metaplasia in two patients with short segment (< 3 cm) Barrett’s esophagus.31

Theoretically, the ideal treatment for a patient with Barrett’s esophagus is one that will restore the normal squamous mucosa and eliminate the cancer risk associated with the intestinalization of the cardiac-type mucosa. Currently, neither medical nor surgical therapy reliably offers this. However, a number of experimental techniques for mucosal ablation show promise. In animal and limited clinical trials, some of these ablation techniques have been successful in removing the columnar mucosa and allowing subsequent squamous re-epithelialization. The elimination of gastroesophageal reflux is critical to successful squamous re-epithelialization. Consequently, persistent or recurrent areas of intestinal metaplasia plague most series of mucosal ablation in which patients take proton pump inhibitors in an effort to stop reflux. In contrast, Salo and colleagues recently reported successful ablation of Barrett’s using Nd-YAG laser after antireflux surgery.32 They followed 11 patients for a mean of 26 months after the last laser treatment, and noted complete squamous regeneration in all patients. There was no residual metastatic epithelium except in two patients with persistent intestinal metaplasia at the gastroesophageal junction. Ablation, when combined with an antireflux procedure, is likely to be the most reliable method to remove the metaplastic mucosa and allow squamous regeneration.

EVIDENCE THAT ANTIREFLUX SURGERY INDUCES QUIESCENCE OF THE BARRETT’S MUCOSA AND HALTS THE PROGRESS TO DYSPLASIA AND CANCER

Patients with Barrett’s esophagus can progress to low-grade dysplasia, high-grade dysplasia, and eventually adenocarcinoma while under medical therapy, as noted by clinical experience. In contrast, clinical evidence is mounting that in patients with Barrett’s esophagus, surgical therapy is associated with a reversal of the first step in this process. In our series of 60 patients with intestinal metaplasia of the esophagus or esophagogastric junction, we found preoperatively low-grade dysplasia in 10 patients.30 In seven out of the 10 patients, the low-grade dysplasia reverted to Barrett’s without dysplasia following an antireflux procedure. Similarly, Low and colleagues noted that four out of their 14 patients had low-grade dysplasia and, in all four patients, it regressed to Barrett’s without dysplasia after antireflux surgery.31 In our opinion, preventing reflux with a properly constructed antireflux procedure stops the continual irritation of the metaplastic mucosa and allows the cells to become quiescent. If low-grade dysplasia persists after antireflux surgery, consideration should be given to mucosal ablation without acid suppression therapy.

Perhaps more significant is whether Barrett’s esophagus progresses to high-grade dysplasia or cancer after surgical treatment of reflux disease. McCallum and colleagues prospectively followed
181 patients with Barrett’s esophagus. In total, 29 had antireflux surgery whereas the remaining 152 patients were treated medically.\textsuperscript{33} After a mean follow-up of 5 years in the surgical group and 4 years in the medical group, there was a significant difference in the incidence of dysplasia and adenocarcinoma. Dysplasia was found in 3.4% of the surgical group compared with 19.7% in the medically treated group. No patient in the surgically treated group developed adenocarcinoma of the esophagus, whereas this did occur in medically treated patients. They concluded that compared with medical therapy an antireflux operation in patients with Barrett’s esophagus was significantly associated with a reduction in the incidence of dysplasia and cancer. Similarly, Katz and colleagues followed 102 patients with Barrett’s for a mean of 4.8 years.\textsuperscript{34} By 3 years, approximately 8% of the medically treated patients had high-grade dysplasia. In contrast, patients treated using antireflux surgery had a significantly reduced risk of developing dysplasia. The 9-year dysplasia and cancer-free survival was 100% for 15 patients after an antireflux procedure and 50% for 82 patients treated with medical therapy ($P = 0.03$).

A complication of analyses of progression of Barrett’s to high-grade dysplasia or cancer after antireflux surgery is that the cellular and genetic alterations leading to the development of high-grade dysplasia and adenocarcinoma may have already occurred before performance of the antireflux procedure. It has been estimated to take up to 6 years for adenocarcinoma to develop within Barrett’s with low-grade dysplasia, and thus some cancers, particularly those which present during the first few postoperative years, probably do not represent progression of disease after surgery. McDonald and colleagues made this point in a study from the Mayo Clinic.\textsuperscript{35} They found invasive adenocarcinoma in two patients and high-grade dysplasia in one patient during surveillance after antireflux surgery, but they noted that no patient developed carcinoma or high-grade dysplasia after 39 months despite a median follow-up of 6.5 years, and a maximum follow-up of 18.2 years.

Re-examination of the English language literature since 1975 uncovered 11 series and a total of 346 patients with Barrett’s esophagus followed after fundoplication.\textsuperscript{29} Patients were found to have esophageal adenocarcinoma after antireflux surgery in only seven out of the 11 reports. Separately from these series, four isolated reports were found describing adenocarcinoma developing in Barrett’s esophagus after an antireflux operation. Although the length of follow-up was not always available, 11 out of the 19 cancers (58%) developed within 3 years of fundoplication, and 15 (79%) developed within 5 years of fundoplication. The remaining four cancers developed from 5 to 10 years after fundoplication but, in each case, the patients had recurrent reflux on the basis of symptoms or positive 24-h pH monitoring. Thus, a functioning fundoplication seems to provide protection from progression of Barrett’s to adenocarcinoma in patients in whom this process has not started before the surgical procedure.

### HIGH-GRADE DYSPLASIA IN BARRETT’S MUCOSA: RATIONALE FOR ESOPHAGEAL RESECTION

The histologic identification of dysplasia within Barrett’s mucosa is, at present, the most reliable way of identifying patients who are already harboring adenocarcinoma or are at risk of developing one. However, the ability to identify dysplasia may differ between pathologists. Investigators have searched for better indicators of malignancy with techniques such as flow cytometry to identify changes in DNA ploidy and immunohistochemical techniques or polymerase chain reaction to look for specific chromosomal mutations. Most notably has been the use of p53 tumor suppressor gene abnormalities. These techniques provide considerable insight into the neoplastic progression of Barrett’s esophagus but, at present, do not provide a benefit in the identification of patients with or who will develop adenocarcinoma.

Dysplasia is classified histologically according to the guidelines for inflammatory bowel disease described by Riddell et al.\textsuperscript{36} and modified for application to the esophagus.\textsuperscript{37} It is defined as an unequivocal neoplastic transformation that is distinguishable from reactive and regenerative changes. In a comparative study between eight experienced pathologists, there was an 86% interobserver agreement over the diagnoses of high-grade dysplasia and intramucosal adenocarcinoma.\textsuperscript{35} In contrast, the diagnosis of indefinite for dysplasia and low-grade dysplasia was more variable with only 58% and 75% agreement, respectively. The reason for the poor agreement in the lower grades of dysplasia was mainly due to inflammatory atypia, which was less of a problem in the presence of high-grade dysplasia. Even though the diagnosis of high-grade dysplasia can be made with confidence, patients considered to have high-grade dysplasia should have the histologic slides reviewed by two experienced pathologists before making a decision for therapy.

The average age of patients presenting with esophageal adenocarcinoma is about 20 years older than that of patients with Barrett’s esophagus. The long duration between the development of Barrett’s esophagus and its malignant degeneration, the accessibility of the esophagus for inspection and biopsy, and the observation that the results of surgical treatment for esophageal adenocarcinoma are directly linked to the stage of disease at the time of
de novo significantly better survival than those who present on surveillance present at an earlier stage and have patients with esophageal adenocarcinoma detected. This approach has been supported by the reports that surveillance for patients with Barrett’s esophagus. Discovery have led to the introduction of endoscopic surveillance with multiple biopsies every 2 cm along the visible length of the columnar mucosa, with additional biopsies from any abnormal-appearing area.

Patients who are diagnosed with indefinite or low-grade dysplasia should undergo a repeat endoscopic examination with meticulous examination of the Barrett’s epithelium and documentation of the location of any areas of mucosal irregularity. Biopsies are obtained along the entire length of the columnar mucosa, in the standard fashion, and the location of each biopsy is recorded. Patients who have not been on treatment for their reflux disease should receive a 3-month course of intensive acid suppression with high-dose proton pump inhibitors to reduce the active inflammation. After this course of treatment, the patient is re-endoscoped and the esophagus extensively biopsied, paying particular attention to areas previously reported to show dysplastic change. If the low-grade dysplasia persists, the patient should undergo an antireflux procedure followed by endoscopic surveillance with multiple biopsies every 6 months. In about 50% of patients, the dysplastic changes will regress and surveillance can be performed less frequently. Those who do not regress should have ablation of their Barrett’s mucosa. This is particularly successful if the patient has had a previous antireflux procedure.

The optimal treatment of patients with Barrett’s esophagus harboring high-grade dysplasia is controversial. Some investigators advocate continued surveillance in patients who present with, or have progressed to, high-grade dysplasia. Surgical therapy is considered to have high morbidity and, in a few isolated cases, mortality has been reported in patients free of cancer in the resected esophageal specimens. This more conservative approach is based upon the presumption that high-grade dysplasia is a separate entity to invasive adenocarcinoma and that the condition does not always progress to malignancy. It hypothesizes that preoperative biopsies may accurately differentiate between patients with high-grade dysplasia and invasive adenocarcinoma.

Levine et al. reported on this approach in patients with high-grade dysplasia. They used an aggressive biopsy protocol in which up to 12 biopsies per cm of esophageal columnar lining were obtained using large-size biopsy forceps rather than the traditionally accepted four-quadrant biopsies every 2 cm. It has been questioned by these investigators and others whether such a protocol is appropriate for clinical practice. In a similar prospective study by Cameron et al., 23 patients with high-grade dysplasia underwent a rigorous preoperative biopsy protocol using standard-sized biopsy forceps. Only one out of the 15 patients who were resected had adenocarcinoma in the esophageal specimen, whereas one patient died postoperatively. Such an observation would favor a conservative approach to patients with high-grade dysplasia, if the mortality for esophagectomy at the institution is high.

Several retrospective surgical studies have reported on the difficulty in differentiating high-grade dysplasia from adenocarcinoma on the basis of endoscopic biopsy examination and reported that 50% of esophagectomy specimens resected from patients thought only to have high-grade dysplasia harbored invasive cancer. These studies have been criticized because there was no report on the extent of the preoperative biopsies. In our own experience, 23 patients were referred to us from endoscopic surveillance programs with the diagnosis of high-grade dysplasia. Extensive systematic re-biopsy by us before surgery detected nine patients with adenocarcinoma. High-grade dysplasia was found on the second biopsy in the remaining 14 patients. After esophagectomy, adenocarcinoma was detected in six out of these 14 patients. This gave a biopsy error of 43% in detecting adenocarcinoma in these patients.

Recently, the need for esophagectomy in patients with high-grade dysplasia or intramucosal adenocarcinoma has been questioned and endoscopic techniques to ablate the suspected mucosa have been encouraged. Widespread adoption of this alternative therapy would be premature before the extent of neoplastic disease in such patients is known and the two therapies have been compared. We have analyzed the operative specimens in 25 patients whom on preoperative endoscopy had no visible lesion but on
biopsy had intramucosal adenocarcinoma.\textsuperscript{47} The surgical specimen showed that the tumor was limited to the mucosa in 22 patients (88\%) and to the submucosa in three patients (12\%). A total of 10 of these patients, known to have adenocarcinoma before surgery, had an \textit{en bloc} esophageal resection with systematic mediastinal and upper abdominal lymphadenectomy. These specimens were analyzed for the total number of lymph nodes removed, and the number and location of lymph node metastases. Immunohistochemical evaluation was performed on specimens with histologically normal nodes to detect micrometastasis. The median number of nodes removed was 36 (18–53) per patient. A total of 370 lymph nodes were analyzed using routine histology and by immunohistochemistry. In only one node was there evidence of metastatic cancer. In other words only one out of the 10 patients had a single-node metastasis. It was located along the left gastric artery.

We also analyzed 12 patients who on preoperative endoscopy had a visible lesion, i.e. a nodule or ulcer, and a biopsy showing high-grade dysplasia or intramucosal adenocarcinoma. In total, 11 patients had intramucosal carcinoma and one had high-grade dysplasia on biopsy. All had carcinoma in the surgical specimen. The tumor was limited to the mucosa in three patients, submucosa in five patients, muscularis propria in two patients and penetrated through the esophageal wall in two patients. Nine of these patients underwent an \textit{en bloc} esophagectomy with systematic lymph node dissection. A total of 339 lymph nodes were examined using routine histology and immunohistochemistry and seven nodes showed evidence of metastatic cancer. In other words, five out of the nine patients had lymph node metastasis: one node each in three patients and two nodes each in two patients. Consequently, if an endoscopic lesion is seen in a patient with high-grade dysplasia or intramural carcinoma, an \textit{en bloc} surgical resection of the tumor and the regional lymph nodes should be performed.

Table 2 is a comparison between photodynamic therapy (PDT)\textsuperscript{48} and esophagectomy for the diagnosis of high-grade dysplasia in a patient with no endoscopically visible lesion. As stated above, 15 of these patients had early adenocarcinoma in their surgical specimen. It is not reported how thoroughly those who received PDT were investigated before therapy, but we assume that none of them had an endoscopically visible lesion. It is impossible to know how many had an occult tumor as the mucosa was ablated, but it is reasonable to expect a prevalence based on the surgical studies between 43\% to 65\%. After PDT, two patients with cancer have emerged and both had an esophagectomy. Both had lymph node metastasis suggesting that the tumor had advanced between the time of PDT and its discovery. At the present time, follow-up is too short to encourage widespread use of this technology for high-grade dysplasia.

The observation from the study of the surgical specimen that 90\% of patients with no visible endoscopic esophageal lesion and occult carcinoma are free of both histologic and immunohistochemical lymph node metastases encourages a more limited resection, and places a greater emphasis on achieving improved postoperative function to provide a quality of life compatible with the excellent cure rate. A vagal-sparing esophageal stripping with a colon interposition achieves this goal. It can be carried out with much less morbidity than a transhiatal esophagectomy, and provides postoperative alimentation that is exceptionally good and far exceeds that which occurs after the standard forms of esophagectomy.

On the basis of this experience, we have concluded that the lack of an endoscopically visible lesion does not preclude cancer invasion beyond the muscularis mucosa, and that the presence of an endoscopic lesion in a patient with a biopsy showing high-grade dysplasia or intramucosal tumor is a strong indicator for the presence of adenocarcinoma that extends deeper than the muscularis mucosa. Therapy designed with a depth of tissue destruction limited to the mucosa would be inadequate in a minority of patients with the former condition and a majority of those with the latter condition.

Most patients whose tumor penetrates through the muscularis mucosa, but not through the esophageal wall, have < 4 lymph nodes involved. Most of the involved lymph nodes are found along the lesser curvature of the stomach, although lymph node metastasis can occur in more distant areas. To achieve a complete resection with confidence in these patients requires a systematic mediastinal and abdominal lymphadenectomy. Survival after such a procedure in patients with a visible lesion and the diagnosis of high-grade dysplasia or intramucosal carcinoma is similar to those who had the identical diagnosis but no visible lesion and were treated with a vagal sparing esophagectomy without a lymph node dissection.

\begin{table}[h]
\centering
\begin{tabular}{|l|c|c|}
\hline
 & Photodynamic therapy* (%): & Esophagectomy† (%): \\
\hline
No. patients & 73 & 23 \\
Completeness of removal & (75–80) & 100 \\
Follow up PPI Rx & 100 & 0 \\
Persistent dysplasia & 21 & 0 \\
Subsquamous persistent & 3 & 0 \\
Occult cancer & ? & 65 \\
Subsequent cancer & 3 & 0 \\
Mortality (unrelated) & 3 & 0 \\
Stricture & (34) & 4 \\
\hline
\end{tabular}
\caption{Barrett’s esophagus: treatment for high-grade dysplasia}
\end{table}

*Overholt et al.\textsuperscript{48}.
†High-grade dysplasia with no endoscopically visible lesion. Systematic preoperative biopsies detected nine intramucosal carcinomas and six were discovered after surgery.
Consequently, our recommendation would be that for patients with high-grade dysplasia and no endoscopically visible lesion, a vagal sparing esophagectomy should be performed. If a visible lesion is present, a more formal esophageal resection with abdominal and thoracic lymphadenectomy should be performed.

References


