The natural history of Barrett’s esophagus (BE) is a matter of debate in the gastrointestinal literature. We report two unique presentations of BE, which demonstrate highly variable biologic behavior. This poses a management dilemma when faced with options of surveillance, surgery or ablation modalities. These cases suggest, advancement from HGD to BEAC is not always rapid and the cancer can have variable biologic behavior. We report two interesting cases wherein one developed poorly differentiated adenocarcinoma and the other developed changes approaching adenocarcinoma. Both remained asymptomatic and died of non-gastrointestinal causes. These cases demonstrate the fact that, patients often do not die from BE or BEAC but most often from concurrent cardiovascular disease. The surveillance endoscopies appear to have made little difference in the quality or duration of their lives.

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INTRODUCTION

The natural history of Barrett’s esophagus (BE) is a matter of debate in the gastrointestinal literature. We report two unique presentations of BE, which demonstrate highly variable biologic behavior. This poses a management dilemma when faced with options of surveillance, surgery or ablation modalities.

CASE 1

A 79-year-old diabetic, hypertensive male had long-segment BE (LSBE) with three years of high-grade dysplasia (HGD). He then developed a small-elevated plaque at 36–37 centimeters (cm) from the incisors confirmed as Barrett’s adenocarcinoma (BEAC) (Figures 1 and 2). Because of age and comorbidities, endoscopic mucosal resection (EMR) and photodynamic therapy (PDT) were offered. The patient was resolute and refused treatments. Esophagogastroduodenoscopy two and a half years later revealed a 3 cm fungating, exophytic and non-obstructing poorly differentiated BEAC at 35–38 cm from incisors (Figure 3). Computer tomography scans showed a right paratracheal node less than one centimeter in size. He eventually died five years after diagnosis of BEAC from congestive heart failure (CHF) but remained asymptomatic from the cancer.

CASE 2

An 83-year-old male had a significant medical history of coronary artery disease (CAD), hyperlipidemia, CHF and hypertension. He was under surveillance for fourteen years because of LSBE with variable grades of dysplasia. Initial biopsy showed low-grade dysplasia (LGD) (Figure 4). Three years later HGD approaching BEAC...
was noted (Figure 5). Proton pump inhibitors dose was doubled and a repeat biopsy three months later showed no dysplasia. He was lost to follow-up but four years later, was found to have LGD and HGD. Remarkably he demonstrated HGD intermittently over a total of fourteen years and died of pulmonary embolus but never developed BEAC.

DISCUSSION

BE is an acquired condition from chronic gastroesophageal reflux disease (GERD). The normal squamous epithelium of the lower esophagus is replaced with metaplastic intestinal type columnar epithelium (1). The transition involves multistep histological change from columnar metaplasia to intestinal metaplasia to BEAC (1). It has been reported that, about 40% of the patients with HGD harbor occult adenocarcinoma at the time of esophagectomy (2). A change in the genetic structure leading to aneuploidy is significant in the progression from Barrett’s mucosa to dysplasia and subsequent adenocarcinoma (3). There is a 5% lifetime risk to develop adenocarcinoma in patients with BE (4).

Trials with three new modalities of endoscopic treatment for Barrett’s dysplasia, photodynamic therapy (PDT), argon plasma coagulation (APC) and endoscopic mucosal resection (EMR), have shown promise (3,8,9). Even with these recent advances, the 5-year survival of adenocarcinomas is still 5%–13% (5,6,7). Surveillance

Figure 3. EGD two and a half years later revealed a three-centimeter fungating, exophytic and non-obstructing poorly differentiated BEAC at 35–38 centimeters from incisors.

Figure 4. EGD showing Barrett’s esophagus (BE) with focal low-grade dysplasia (LGD) at 25 centimeters from the incisors.
endoscopy versus ablation with PDT, APC or EMR for BE remains a major dilemma. Management often depends on the co-morbidity, biologic tumor behavior, local resources and patient preferences.

These two cases are very interesting. One developed poorly differentiated adenocarcinoma and the other developed changes approaching adenocarcinoma. Both remained asymptomatic for an extended period of time (8 years and 14 years respectively) and died of non-gastro-intestinal causes. These cases suggest, advancement from HGD to BEAC is not always rapid and the cancer can have variable biologic behavior. Surgical interdiction or even PDT may well have caused the patient’s demise years earlier because of associated co-morbidities. These cases also demonstrate that, patients often do not die from BE or BEAC but most often from concurrent cardiovascular disease. The surveillance endoscopies appear to have made little difference in the quality or duration of their lives.

References