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CASE REPORT/OLGU SUNUMU

Barrett's Esophagus; Case Report

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Abstract
Barrett's esophagus is the transformation of the normal squamous epithelium in the distal esophagus into columnar epithelium with intestinal metaplasia. Barrett's esophagus generally develops as a result of chronic mucosal irritation due to gastroesophageal reflux. Although Barrett's esophagus is usually seen over the age of 50, it can also be seen at younger ages. In our case, it was seen in a 34-year-old male patient. The patient applied to the general surgery outpatient clinic with complaints of swelling in the stomach, pain and watering in the mouth. When endoscopy is performed; the cervical and thoracic esophageal mucosa appeared normal, and the Z line was 40 cm distally. The esophageal mucosa had a hyperemic appearance. Multiple biopsies were taken from these areas. In histopathological evaluation, special columnar epithelium, mucin-filled cytoplasm, goblet cells (intestinal metaplasia) were observed. Goblet cells were positively stained by PAS/Alcian Blue and Mucincarmine histochemical staining.
Key Words: Barrett's esophagus, gastroesophageal reflux, intestinal metaplasia

Barrett Özofagus; Olgu Sunumu

Özet
Barrett özofagus, distal özofagustaki normal skuamöz epitelin intestinal metaplazi ile kolumnar epitele dönüşmesidir. Barrett özofagus genellikle gastroözofagial reflüye bağlı kronik mukozal irritasyon sonucu gelişmektedir. Barrett's özofagusu genellikle 50 yaş üzerinde görülmekle birlikte daha genç yaşlarda da görülebilmektedir. Bizim olgumuzda 34 yaşında erkek hastada görülmüştür. Hasta midede şişlik, ağrı ve ağızda sulanma şikayetleri ile genel cerrahi polikliniğine başvurmuştur. Endoskopi yapıldığında; servikal ve torasik özofagus mukozası normal görünümdeydi, Z çizgisi distalde 40 cm idi. Özofagus mukozası hiperemik bir görünüme sahipti. Bu alanlardan birden fazla biyopsi alındı. Histopatolojik değerlendirmede; özel kolumnar epitel, müsin dolu sitoplazma, goblet hücreleri (bağırsak metaplazisi) gözlendi. Goblet hücreleri, PAS/Alcian Blue ve Mucincarmine histokimyasal boyama ile pozitif olarak boyandı.

Anahtar Kelimeler: Barrett özofagus, gastroözofagial reflü, intestinal metaplazi

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INTRODUCTION

Barrett's esophagus is defined as the replacement of normal squamous epithelium in the distal part of the esophagus with columnar epithelium containing intestinal metaplasia (1). In fact, although Tileston was the first to describe the columnar epithelial line in the lower esophagus, the lesion was first described by Norman Barrett in 1950 as a congenitally short esophagus accompanied by an intrathoracic stomach, and it was later reported that the lesion had abnormal columnar epithelial alignment in the distal esophagus (2). Barrett later explained that the disease is acquired (3). Today, for the definition of Barrett's esophagus, it is necessary to both endoscopically visualize the columnar line in the esophagus and detect the presence of intestinal metaplasia by biopsy (3). In the case report, we aimed to present a case diagnosed as Barrett's esophagus on tissue taken by endoscopic biopsy to emphasize the importance of Barrett's esophagus.

CASE REPORTS

In our case, it was seen in a 34-year-old male patient. The patient applied to the general surgery outpatient clinic with complaints of swelling in the stomach, pain and watering in the mouth. When endoscopy is performed; the cervical and thoracic esophageal mucosa appeared normal, and the Z line was 40 cm distally. The esophageal mucosa had a hyperemic appearance. Multiple biopsies were taken from these areas. In histopathological evaluation, special columnar epithelium, mucin-filled cytoplasm, goblet cells (intestinal metaplasia) were observed. Goblet cells were positively stained by PAS/Alcian Blue and Mucincarmine histochemical staining.

DISCUSSION

Barrett's esophagus patients typically present with symptoms of gastroesophageal reflux disease (GERD) (5). Heartburn and regurgitation are major symptoms (4). There may also be dysphagia, odynophagia, chest pain. While some patients have symptoms at first, they may disappear later, because the areas of esophagitis transform into columnar epithelium, which is less sensitive to acid (5). Most of the patients are middle-aged, smokers and alcohol users. Barrett's esophagus patients may also be symptom-free. The frequency and duration of heartburn are associated with increased frequency of Barrett's esophagus, but the severity of reflux symptoms is inversely proportional to Barrett's esophagus (6). Among the symptoms of Barrett's esophagus, extraesophageal findings such as hematemesis, melena, occult gastrointestinal bleeding and hoarseness, asthma and dental disorders can be seen (5,6). Erosion and ulceration, peptic ulcer, fistula formation, stricture, dysplasia, and tumor development are the main complications and symptoms related to these complications may also develop (7). Dysphagia is an important finding that may suggest adenocarcinoma. Similarly,

gastrointestinal bleeding may be related to esophageal ulcer and severe esophagitis (8). If the cause of chronic blood loss cannot be explained by colonoscopy, such patients should undergo Barrett's esophagus and upper gastrointestinal system endoscopy for esophagitis (9).

Esophageal adenocarcinoma (EAC) is a very deadly type of cancer, and it is the fastest growing cancer type with an annual increase rate of 4-10% in the Americas and Europe (8,9). Barrett's esophagus is the only known precursor lesion of EAC and usually occurs due to GERD (14). However, adenocarcinoma develops in only 1.3-5% of patients with Barrett's esophagus, and the time required for this is estimated to be 20-30 years (10). The risk of developing EAC in patients with Barrettt's esophagus is 30-125 times higher than in normal people (11). The aim of Barrett's esophageal screening in patients with gastroesophageal reflux disease is to detect esophageal cancer in an early and treatable period and to reduce EAC-related deaths (11,12). The American Gastroenterology Association reported that upper gastrointestinal system (GIS) endoscopy should be performed in patients with chronic GERD because of the high incidence of Barrett esophagus, but whether routine screening is necessary or not is still controversial (13). The median age at diagnosis of Barrett's esophagus was 40, and the age of development of EAC was 64. Eloubeidi and Provenzale stated that over the age of 40 is an independent predictor for Barrett's esophagus (14). Therefore, it is considered appropriate to start Barrett's esophageal screening at this age. Although there is no definitive evidence, it is thought that Barrett's esophageal screening is cost-effective and reduces mortality due to EAC (13,14).

Goblet cells do not show regular distribution in Barrett's esophagus, and their rates differ significantly between patients and specimens (15). Columnar cells among goblet cells resemble gastric foveolar cells or intestinal absorptive cells. Pseudogoblet cells, similar to goblet cells but containing neutral mucin, may be misleading in diagnosis. In Barrett's esophagus, the predominant form of intestinal metaplasia-incomplete intestinal metaplasia consists of a mixture of goblet cells and foveolar type epithelium containing PAS-positive neutral mucin (14,15). These foveolar epithelial cells may contain Alcian Blue-positive acidic mucin, which shows less intense staining than that observed in goblet cells, but detection of Alcian Blue-positive columnar cells is not sufficient for the definitive diagnosis of Barrett's esophagus in the absence of goblet cells (16). Less frequently, the glandular component contains varying proportions of goblet cells, well-formed intestinal absorptive cells. In some cases, incomplete and complete intestinal metaplasia coexist. Pancreatic acinar metaplasia can also be detected, but its diagnostic significance is limited

(17). Typically, the lamina propria shows mild chronic inflammation and fibrosis, with marked thickening and separation, or double-ordering, of the muscularis mucosa. Intestinal metaplasia is mixed with cardiac-type or fundic-type epithelial foci corresponding to normal gastric mucosa, except for varying degrees of mucosal distortion, glandular atrophy, and mild inflammation (18). In the biopsy specimen taken from the distal esophagus in Barret's esophagus, cardiac or fundic type mucosa without intestinal metaplasia (at least 2 cm columnar esophagus) and absence of goblet cells may be the result of sampling error only (17,18).

The treatment approach in Barrett's esophagus should aim to:

(i) relieve reflux symptoms.

(ii) prevent reflux of acid and duodenal contents into the esophagus.

(iii) prevent complications such as erosion, peptic ulcer and stricture.

(iv) arresting the proximal progression of intestinal metaplasia.

(v) accelerate regression of intestinal metaplasia to normal mucosa.

(vi) halt the progression to dysplasia.

(vii) accelerate regression of dysplasia to nondysplastic cells.

(viii) stop the progression to adenocarcinoma (19).

The treatment of Barrett's esophagus determines whether high-grade dysplasia (HDD) is present. In Barrett's esophagus without high-grade dysplasia, the most preferred option is medical therapy (20). Developing medical drugs are sufficient to prevent symptoms in most of the patients. Medical treatment can be successful not only in preventing typical reflux symptoms but also in treating cases with complications (19,20). It has been reported that proton pump inhibitors (PPI) are more effective in relieving reflux symptoms compared to H2 receptor blockers. With proton inhibitor therapy, improvement and improvement in symptoms was detected in 95% of patients with erosive esophagitis and Barrett's ulcer (21). Proton inhibitor also reduce bile reflux by reducing gastric secretions.It is difficult to determine the duration of medical therapy in patients with Barrett's esophagus, as discontinuation of treatment almost always causes exacerbation of reflux symptoms (22). Despite symptomatic improvement with medical treatment, complete regression of Barrett's epithelium is not achieved with neither PPI nor H2 receptor blockers. Although aggressive antireflux therapies are successful in reflux control, they cause achlorhydria and increase the risk of adenocarcinoma development

CONCLUSIONS

Ochronosis is a disease that shows autosomal Barrett's esophagus is usually seen over the age of 50, but it can also be seen at younger ages. Follow-up should be done more frequently in younger patients. The risk of developing cancer

in the Barrett esophagus floor is high. The risk of developing cancer in the Barrett esophagus floor is high. The presence and degree of dysplasia in Barrett's esophagus determines the type of treatment to be applied. In cases with non-dysplasia and low-grade dysplasia, medical treatment, antireflux surgery or ablation methods should be preferred.

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Author Contributions:

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