SHORT COMMUNICATION

Barrett's esophagus

Kollar T, Majek J, Slezak P, Makovnik P, Mlkvy P

*National Cancer Institute, Bratislava, Slovakia.***kollar@nou.sk**

Abstract

Patients with Barrett's esophagus have 30–40 times higher risk of developing adenocarcinoma of distal esophagus. Treatment of Barrett's esophagus without dysplasia is identical with treatment of gastroesophageal reflux disease. (Tab. 1, Ref. 5.)
Key words: Barrett's esophagus, cancer, risk cancer, carcinoma, dysplasia.

Barrett's esophagus develops in patients with chronic gastroesophageal reflux as a response to chronic irritation of distal esophageal mucosa. Injured mucosa heals by form of incomplete intestinal metaplasia – columnar epithelium replaces the squamous one. Barrett's esophagus is present in 1 % of general population and 6–14 % of patients with reflux disease. The risk of developing carcinoma in distal esophagus is 30–40 times higher in patients with Barrett' s esophagus. High risk patients include white males, age over 50 years and patients with chronic reflux disease (>50 years).

Diagnosis

Recently, there has been a change in the definition of Barrett's esophagus. Barrett's esophagus is defined as the histological presence of intestinal metaplasia in esophagus of any length. Intestinal metaplasia in cardia is not considered to be Barrett's esophagus. The primary diagnostic tool in the detection of Barrett's esophagus is endoscopy with biopsy and histological proof of intestinal metaplasia. A considerable effort has been made to improve the assessment of the best site for biopsy. The length of Barrett's esophagus is defined as the distance from Z-line (squamo-collumnar junction) to gastroesophageal junction (proximal margin of gastric folds); biopsy needs to be taken from this area to confirm the diagnosis. Chromoendoscopy can be used to improve the identification of the site of intestinal metaplasia or dysplasia. The most commonly used dyes are Lugol's solution, indigo carmine, toulidine blue or methylene blue. Erosive esopha-;gitis can interfere with the diagnosis of Barrett's esophagus; endoscopy and biopsy needs to be repeated after the healing of esophagitis. Should a confirmed resection be considered, the finding of high-grade dysplasia needs to be confirmed by another biopsy examined by an experienced pathologist. The standard protocol includes four quadrant biopsy, each of 1–2 cm depending on the presence or absence of dysplasia.

Management

Patients with Barrett's esophagus have to be regularly examined and followed to detect dysplasia or early carcinoma (Tab. 1). Mucosal nodularity in high grade dysplasia can be treated by endoscopic mucosal resection (EMR).

Therapy

The treatment of Barrett's esophagus without dysplasia is identical with that of gastroesophageal reflux disease. However, patients with Barrett's esophagus have higher acid exposure in distal esophagus and thus require higher doses of proton pump inhibitors. Appropriate patients may be treated with antireflux surgery that will also improve the symptoms of reflux. However, none of these approaches leads to the disappearance of intestinal metaplasia.

The main goal of surveillance is to detect high grade dysplasia or early cancer. Esophagectomy has a 90 % chance to cure these cases and remains to represent their standard treatment.

National Cancer Institute, Bratislava, and St. Elizabeth Oncology Institute, Bratislava, Slovakia

Address for correspondence: T. Kollar, MD, National Cancer Institute, Klenova 1, SK-833 10 Bratislava 37, Slovakia.
Phone: +421.2.59378382

Tab. 1. Surveillance of patients with Barrett's esophagus.

Dysplasia	Documentation	Endoscopy
Absent	Two EGD with biopsy	Every 3 years
Low grade	Highest grade on follow-up exam	Every year until disappearance of dysplasia
High grade	Repeat EGD with biopsy to rule out early cancer/confirm high grade dysplasia; confirm by experienced pathologist	Focal - every 3 months Multifocal - treat Mucosal nodularity - EMR

The procedure, however, has high mortality (3–12 %) and morbidity (30–50 %) rates. High grade dysplasia occurs more frequently in older patients, the fact of which further increases the procedure risk. The identification of patients with dysplasia can be difficult. The degree of dysplasia series as the basis for the prediction of risk of carcinoma; however, the interobserver agreement often varies among pathologists (50 % for low grade and 85 % for high grade dysplasia). At the time of surgery carcinoma is present in approximately one third of patients with high grade dysplasia. Low grade dysplasia is not specific for neoplasia; similar changes can be present as a response to chronic irritation. Chromoendoscopy, endoscopic ultrasound, flow cytometry, expression of p53 seem to be promising to detect patients at higher risk of cancer.

In the past few years ablative endoscopic therapy of Barrett's esophagus has been gaining its popularity. The most frequently used procedures include endoscopic mucosal resection, photodynamic therapy, laser ablation and argon plasma coagulation. These procedures can be used in high risk patients for esophagectomy; however long term results are still missing. The expression of cyxlooxygenase-2 has been detected in Barrett's esophagus recently; the addition of COX-2 inhibitors to the treatment regimen may prevent the development of cancer. The studies are ongoing.

In spite of recent advances in the diagnosis and management of Barrett's esophagus many questions remain unanswered. Better identification of patients at high risk of cancer, the optimal level of acid suppression and the best therapy for patients with high grade dysplasia and early carcinoma are among the issues that are to be solved in the future.

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