

International guidelines for diagnosis, surveillance and treatment of Barrett's oesophagus.

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BARRETT'S DEFINITION

No definite consensus on definition. The American Gastroenterological Association workshop in Chicago defined Barrett's oesophagus (BO) as the displacement of the squamocolumnar junction proximal to the gastro-oesophageal junction with the presence of intestinal metaplasia. The diagnosis of BO is established when intestinal metaplasia is found in biopsy specimens obtained from salmon-coloured mucosa in the distal oesophagus proximal to the junction of the oesophagus and stomach. The yield of biopsy specimens of columnar-type mucosa in the distal oesophagus for the detection of intestinal metaplasia varies from 25% to 50% in short-segment BO (<3 cm) and up to 80% in long-segment BO (≥3 cm).

RISK OF CANCER

The risk of oesophageal adenocarcinoma (OA) is approximately 0.4% per person-year of follow-up in patients with BO compared with only 0.07% per year in patients who do not have BO. Risk factors for the development of OA in patients with BO include: white, male, longer duration, severity and frequency of GORD, size of hiatal hernia, obesity, smoking, diet low in fresh fruit, dysplasia and, possibly, the length of oesophagus involved by BO.

SCREENING?

Although screening for Barrett's epithelium is controversial, guidelines from the ACG recommend a 'once in a lifetime' gastroscopy for the detection of Barrett's epithelium in patients with chronic GORD symptoms (10-15% of the chronic GORD patients). It should be noted that this is associated with a high cost and unproven benefit. It is also noted that OA is very uncommon in patients younger than 50 years of age and therefore, the benefit of a screening endoscopy in such patients remains unproven. Furthermore, there is no indication for screening and surveillance in patients who are ineligible, a priori, for oesophagectomy or ablative therapy because of age or significant co-morbidities.

SURVEILLANCE STRATEGY?

Surveillance is recommended in patients with BO and dysplasia, because dysplasia (graded as absent, indefinite, low- or high-grade) is a risk factor for OA. The risk of progression to cancer is greater with high-grade than with low-grade dysplasia. Unfortunately, there is significant interobserver variability in the grading of dysplasia, with less than 50% agreement in the classification of low-grade dysplasia. The risk of progression from low to high-grade dysplasia or cancer ranges from 10% to 28% over five years, and estimates of progression from high-grade dysplasia to cancer range from 16% to 59% over five to seven years.

Endoscopic surveillance should include a standard biopsy protocol and should be performed while the patient continues to take therapy sufficient to optimize symptom relief and to minimize the impact of inflammation and regeneration on the interpretation of biopsy specimens.

Surveillance in patients with BO without dysplasia is more controversial. For those enrolled in a surveillance program, endoscopy should be repeated every two to five years. There is limited evidence that surveillance programs may lead to earlier cancer detection and improved survival, and retrospective analyses suggest that patients undergoing surveillance had better two-year (86% versus 43%) and five-year (62% versus 20%) survival rates for OA than those who did not undergo surveillance. However, there are also strong arguments against surveillance. There is a low absolute incidence of OA and the benefits of surveillance are not clearly defined as they are in colon cancer. Surveillance endoscopy is expensive and time-consuming. About 93% to 98% of OA occur in patients without a prior diagnosis of BO. Finally, it has been suggested that surveillance may be of marginal benefit because most patients with BO do not die from oesophageal cancer. During a nine-year follow-up of 166 patients with BO, only eight patients developed OA, which was the cause of death in only two cases.

TREATMENT: PPI

The goals of therapy in patients with Barrett's epithelium include symptom control, maintenance of mucosal healing, regression of BO, and regression of dysplasia. Management includes acid suppressive medication, ablative therapies or surgery. Four randomized controlled trials have assessed acid suppressive therapy with a PPI, H2RA or anti-reflux surgery in patients with BO. Acid suppression was associated with symptom control but not with disappearance of Barrett's epithelium despite some reports of regression. Anti-reflux surgery did not prevent progression, with dysplasia developing in 10.5% and OA in 2.5% of 161 patients seven to 21 years postoperatively. In two long-term studies, there was no difference in the rate of development of OA between patients receiving medical or surgical anti-reflux therapy. No study has demonstrated a reduction in OA rates with medical or surgical acid suppression. Use of PPI in asymptomatic patients is not recommended.

TREATMENT: DYSPLASIA

The presence of high-grade dysplasia should be confirmed by repeat endoscopy and biopsies within three months of initial detection with a concentrated biopsy protocol and expert pathologist review of all biopsies, after the patient has had continuous treatment with double dose (twice-daily standard dose) PPI to heal associated erosive oesophagitis and minimise reflux related injury, inflammation and proliferative changes. However, for some patients, such as those with multifocal high-grade dysplasia, referral for therapy does not require a repeat confirmatory endoscopy. Expert consultations should be obtained to ascertain the optimal endoscopic or surgical management strategy.

Endoscopic mucosal resection and ablation are available in some centres for the management of localized, high-grade dysplasia but experience to date is limited (275,284-290). In general, surgery is the recommended strategy for patients who are otherwise healthy but it should be done in centres where these procedures are performed regularly (291). Oesophagectomy can be associated with important side effects including dumping syndrome, dysphagia, diarrhoea, early satiety and weight loss, and can reduce quality of life substantially (264,292-295). Vagal-sparing oesophagectomy and other techniques are under investigation and may be promising in the future. Endoscopic treatments require further study before they can be routinely recommended. Optimal management will generally require input from one or more appropriate specialists in the fields of gastroenterology, oncology, gastrointestinal histopathology and thoracic surgery. Ablation therapy should be considered for individuals with high-grade dysplasia or OA who are unfit for or unwilling to undergo surgery.

Personal proposal:

BO with no dysplasia: control endoscopy every 2 years, only over 50 years (use of magnification, chromoendoscopy and other techniques might improve detection rate and decrease number of biopsies)

BO with LGD: control every year until disappearance

BO with HGD: control within 3 months (under double dosage of PPI)

If confirmed: staging EMR or EMR-ablative therapy or oesophagectomy (depending on local staging, patient fitness, and endoscopic or surgical expertise)

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