Barrett’s esophagus (BE) is defined as the extension of salmon-colored mucosa into the tubular esophagus ≥ 1 cm proximal to the gastroesophageal junction with biopsy confirmation of intestinal metaplasia. Patients with BE are at increased risk of esophageal adenocarcinoma (EAC), and undergo endoscopic surveillance biopsies to detect dysplasia or early EAC. Dysplasia in BE is classified as no dysplasia, indefinite for dysplasia (IND), low grade dysplasia (LGD) or high grade dysplasia (HGD). Biopsies are diagnosed as IND when the epithelial abnormalities are not sufficient to diagnose dysplasia or the nature of the epithelial abnormalities is uncertain due to inflammation or technical issues. Specific diagnostic criteria for IND are not well established and its clinical significance and management has not been well studied. Previous studies have focused on HGD in BE and led to changes and improvement in the management of BE with HGD and early EAC. Only recently, IND and LGD in BE have become focus of intense study. This review summarizes the definition, neoplastic risk and clinical management of BE IND.

Key words: Barrett’s esophagus; Dysplasia; Progression; Biomarkers; Esophageal adenocarcinoma; Indefinite for dysplasia

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years, abnormal p53 expression, active inflammation, and abnormal DNA content as detected by flow cytometry may help in risk-stratifying this patient population.

INTRODUCTION

Barrett’s esophagus (BE) is a complication of chronic esophageal injury from gastroesophageal reflux disease (GERD) and develops when reflux damaged esophageal squamous cells are replaced by mucous-secreting columnar cells. A definitive diagnosis of BE is established by the extension of salmon-colored mucosa into the tubular esophagus ≥ 1 cm proximal to the gastroesophageal junction (GEJ) with esophageal biopsy showing intestinal metaplasia, defined by the presence of goblet cells. Intestinal metaplasia in BE is a well-established marker of esophageal adenocarcinoma (EAC), and as such patients diagnosed with BE undergo regular endoscopic surveillance and biopsy to detect dysplasia or curable neoplasia. According to the published criteria by Reid et al, the biopsies are classified based on five-tiered histologic classification of dysplasia as negative for dysplasia, indefinite for dysplasia (IND), low-grade dysplasia (LGD), high-grade dysplasia (HGD) and intramucosal adenocarcinoma (IMAC).

Dysplasia remains the best available clinical marker for cancer risk. Published guidelines have recommended endoscopic surveillance and treatment strategies based on the grade of dysplasia. The management of LGD and HGD in BE has been reviewed extensively and discussed in many published guidelines. Many studies have focused on the high end of neoplasia in BE, HGD and IMAC, leading to a much improved and less invasive endoscopic management and interobserver reproducibility studies and found that its diagnostic reproducibility is poor. Histologic criteria used to diagnose BE IND varied in different studies (Table 1) and even more so by pathologists in routine practice. For instance, the criteria for IND described by Reid et al included moderate architectural distortion, nuclear abnormalities less marked than those seen in dysplasia, frequent dystrophic goblet cells, more extensive nuclear stratification, diminished or absent mucus production, increased cytoplasmic basophilia, and increased mitoses (Figure 1A). The diagnosis of IND should be limited to cases in which the changes are worrisome but not sufficient for the diagnosis of dysplasia (Figure 1B). Using similar criteria, other groups performed intraobserver and interobserver reproducibility studies and found that BE IND has significant interobserver variability. In daily pathology practice, the BE IND category appears to expand, one such example being basal crypt dysplasia-like atypia. The concept of basal crypt dysplasia-like atypia remains controversial and is interpreted by some as IND while others believe that it truly represents dysplasia without surface involvement.

NEOPLASTIC RISK OF BE IND

Regardless of the definition, illustration, and intraobserver /interobserver variability, BE IND category is not uncommonly used in daily pathology practice. Several studies recently investigated the clinical significance of BE IND and the results are reviewed and summarized in Tables 2 and 3.

RISK OF PREVALENT NEOPLASIA IN BE IND

Only few studies investigated the risk of neoplasia in BE IND. Prevalent neoplasia risk, defined as LGD, HGD or EAC detected within 1 year of the diagnosis of BE IND,
was reported in 3 studies and ranged from 12.9% to 25%. Prevalence of advanced neoplasia, i.e., detection of HGD or EAC within 1 year of the diagnosis of BE IND, varied between 1.9% and 15% [9,11,12,14,15]. When a 6-mo interval was used as a cut-off, the prevalence of LGD and advanced neoplasia in BE IND was at least 2.8% [9]. In one case, the mucosal ulceration was associated with EAC [11].

**RISK OF INCIDENT NEOPLASIA IN BE IND**

The incidence of neoplasia in BE IND is summarized in Table 3. The incidence of all neoplasia in BE-IND is reported to be 4.5 cases per 100 person-years at risk. The progression to advanced neoplasia was 0.43 to 1.2 cases per 100 person-years at risk. The progression to EAC varied between 0.18 to 1.10 cases per 100 person-years at risk. In a study of 82 patients with BE IND, the mean length of BE segment was 6 cm in progressors vs 3 cm in non progressors (P = 0.01). The length of BE segment (HR = 1.2, 1.03-1.3) and multi-focality of BE IND (HR = 2.9, 1.09-7.6) were significantly associated with a higher risk of progression [12]. One study examined the progression to advanced neoplasia

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**Table 1** Histopathologic criteria for Barrett’s esophagus with epithelial change indefinite for dysplasia

| Ref.                  | Criteria                                                                                                                                 |
|-----------------------|------------------------------------------------------------------------------------------------------------------------------------------|
| Reid et al [2], 1988  | The architecture may be moderately distorted. Nuclear abnormalities are less marked than those seen in dysplasia. Other features that may lead to a diagnosis of IND include more numerous dystrophic goblet cells, more extensive nuclear stratification, diminished or absent mucus production, increased cytoplasmic basophilia, and increased mitoses. |
| Montgomery et al [7], 2001 | Preserved gland architecture, mild crypt distortion, minimal nuclear stratification and slight nuclear atypia or enlargement |
| Sonwalkar et al [3], 2010 | When a diagnosis of genuine dysplasia cannot be made. This is often due to the co-occurrence of inflammatory changes or when evaluation of surface maturation is not possible |
| Kestens et al [4], 2015 | Cytologic changes similar to those seen in LGD but with surface maturation or presence of inflammation |
| Sinh et al [5], 2015 | Downgraded from BE LGD to BE IND by an expert pathology panel |
| Duits et al [6], 2015 | The presence of architectural and cytologic atypia in small and mal-oriented biopsy specimen or those with inflammation or ulceration exceeding those expected for reactive changes. In some cases, it is due to basal dysplasia with surface maturation |

BE: Barrett’s esophagus; BE IND: Barrett’s esophagus with epithelial change indefinite for dysplasia; LGD: Low-grade dysplasia.

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**Table 2** Risk of Prevalent neoplasia in patients with Barrett’s esophagus with epithelial change indefinite for dysplasia

| Ref.                  | Number of cases | Prevalent LGD, n (%) | Prevalent HGD, n (%) | Prevalent adenocarcinoma, n (%) | Prevalent advanced neoplasia |
|-----------------------|-----------------|----------------------|----------------------|---------------------------------|-------------------------------|
| Montgomery et al [7], 2001 | 7               | 0 (0)                | 0 (0)                | 1 (15)                          | At least 1 (15)               |
| Sonwalkar et al [3], 2010 | 41              | At least 1 (2.4)     | 0 (0)                | At least 1 (2.4)                | At least 1 (2.4)              |
| Choi et al [8], 2015   | 96              | At least 14 (14.5)   | Not known            | Not known                       | At least 10 (10)              |
| Horvath et al [9], 2015 | 107             | 7 (8.2)              | 2 (2.35)             | 2 (2.35)                       | 4 (4.7)                      |
| Kestens et al [4], 2015 | 842             | 101 (12.1)           | Not known            | Not known                       | 16 (1.9)                     |
| Sinh et al [5], 2015   | 83              | Not known            | 0 (0)                | 0 (0)                           | 0 (0)                        |

LGD: Low-grade dysplasia; HGD: High-grade dysplasia.

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Figure 1 Examples of Barrett’s esophagus with epithelial changes, indefinite for dysplasia. A: This esophageal biopsy shows inflamed BE with moderate architectural complexity, more extensive nuclear stratification, diminished or absent mucus production, increased cytoplasmic basophilia, resembling low-grade dysplasia, but there is presence of marked inflammation (HE stain, × 40). This biopsy is best interpreted as indefinite for dysplasia. B: This tangentially sectioned esophageal biopsy shows foci of glands with enlarged and hyperchromatic nuclei (HE stain, × 100). Because of the lack of surface epithelium as a result of tangential section, this biopsy is best interpreted as indefinite for dysplasia. BE: Barrett’s esophagus.
in a cohort of BE IND (n = 36) which was downgraded from an original diagnosis of BE LGD and reported an advanced neoplasia incidence of 0.9 cases per 100 person-years at risk, similar to a rate of 0.6 cases per 100 person-years at risk in patients with BE negative for dysplasia (n = 153)\(^{13}\). In contrast, BE LGD (n = 75) agreed upon by a panel of expert pathologists had an advanced neoplasia incidence of 9.1 cases per 100 person-years at risk\(^{13}\). Using 6-mo follow-up as a cutoff, Sonwalkar et al\(^\text{[9]}\) (2010) reported that 8.1% of BE IND patients progressed to LGD and 8.1% BE IND progressed to EAC during a median follow-up of 38.7 mo (range: 6-122). Interestingly, none of the 6 patients with BE IND progression had a consensus diagnosis of IND by all three reviewing pathologists.

Some studies did not distinguish between incident and prevalent dysplasia in BE IND. In a study by Montgomery et al\(^{14}\) the neoplasia detection rate among patients with BE IND during a median follow-up of 36 mo was 18% where 4 of 22 patients developed carcinoma. In another study, Choi et al\(^{14}\) reported 1-, 2-, and 3-year detection rates of HGD or EAC among patients with BE IND as 10%, 13% and 20%, respectively.

### BIOMARKERS FOR RISK STRATIFICATION OF BE IND

Few studies evaluated the role of biomarkers to aid in predicting the progression of dysplasia and/or cancer. In a study of 96 BE IND patients, Choi et al\(^{14}\) identified active inflammation (by histology) and DNA flow cytometric abnormalities (either aneuploidy and/or increased 4N fractions greater than 6% of the nuclei) as significant risk factors associated with subsequent detection of dysplasia or neoplasia (hazard ratio for the combiner marker was 18.8, \(P < 0.0001\)). Sonwalkar et al\(^{14}\) reported that the expression of alpha-methylacyl-CoA racemase (AMACR) in more than 1% of cells correlated with progression in BE IND. However, this role of AMACR expression in risk stratifying BE IND was not seen in a subsequent study by Horvath et al\(^{17}\) and they instead showed that high expression of p53 (defined as intense staining in > 5% nuclei), later associated with prevalent advanced neoplasia and progression to advanced neoplasia in BE IND.

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**Table 3 Risk of Incident neoplasia in patients with Barrett’s esophagus with epithelial change indefinite for dysplasia**

| Ref. | No. of cases | Follow up in months (range) | Incident LGD n (%) | Incident HGD n (%) | Incident adenocarcinoma n (%) | Incident Advanced neoplasia (per 100 person-years) | Risk factors for advanced neoplasia |
|------|--------------|-----------------------------|--------------------|--------------------|-------------------------------|---------------------------------------------|---------------------------------|
| Duits et al\(^{15}\), 2015 | 40 | Median 31 (16-59) | 0 | 1 (2.5) | 0 (0) | 0.9 | Not done |
| Horvath et al\(^{15}\), 2015 | 82 | Mean 59 (13-182) | 14 (8.3) | 3 (2.3) | 2 (2.3) | 1.2 | p53 expression in >5% nuclei |
| Kestens et al\(^{15}\), 2015 | 631 | Not known | No data | 10 (1.6) | 6 (1.0) | 0.43 | Older age |
| Sinh et al\(^{15}\), 2015 | 83 | Mean 68.4 (SD: 37.2) | No data | 3 (3.6) | 1 (1.2) | 0.86 | Not done |
| Sonwalkar et al\(^{15}\), 2010 | 37 | Median 38.7 (6-122) | 3 (8.1) | 0 (0) | 3 (8.1) | Not done | Expression of AMACR in more than 1% of cells |

BE IND: Barrett’s esophagus with epithelial change indefinite for dysplasia; LGD: Low-grade dysplasia; HGD: High-grade dysplasia; SD: Standard deviation; AMACR: Alpha-methylacyl-CoA racemase.

**Table 4 Guideline recommendations for the management of Barrett’s esophagus with epithelial change indefinite for dysplasia**

| Guidelines | Diagnosis | Treatment and surveillance |
|------------|-----------|-----------------------------|
| ACG guidelines\(^{12}\) | Acid suppressive medications for 3-6 mo | |
| BSG guidelines\(^{12}\) | Review by a second GI pathologist, and the reasons for use of the ‘indefinite for dysplasia’ category should be given in the histology report in order to aid patient management | A repeat endoscopy after optimization of should be performed |
| ASGE\(^{13}\) | Clarify presence and grade of dysplasia with expert GI pathologist | If BE IND, surveillance in 12 mo |
| Australian Guidelines\(^{13}\) | Confirm by a second pathologist, ideally an expert gastrointestinal pathologist. | Optimisation of antireflux medication |

BE IND: Barrett’s esophagus with epithelial change indefinite for dysplasia.
Clinical management of BE IND

The diagnosis of BE IND is challenging due to varying definitions and inter and intraobserver variability. Therefore, all biopsies should be reviewed by a second pathologist preferably a gastrointestinal pathologist. The patients are treated with aggressive acid suppression. Then, a surveillance endoscopy is performed within 6–12 mo. The biopsy protocol consists of four quadrant biopsies every 1 cm interval. If nondysplastic BE is found, then surveillance interval can be lengthened beyond one year. If LGD or HGD are found, then endoscopic eradication therapy should be considered after confirmation of the diagnosis. The guidelines for management of BE IND are presented by major societies and are summarized in Table 4.

CONCLUSION

In summary, the diagnosis of BE IND is difficult. Recent studies reveal that BE IND carries a significant risk of prevalent advanced neoplasia (at least 2.8%), 31 out of 1135 patients, ranging from 0% to 15% (Table 2). In addition, the diagnosis of BE IND is associated with risk of progression to advanced neoplasia (0.43 to 1.2 cases person-years at risk) (Table 3). These figures are similar to the risk of LGD without histology review, but much lower than the progression risk in consensus diagnosis of LGD. It is worth bearing in mind that 73% of cases with a diagnosis of BE LGD originally rendered by practicing pathologists were down-graded to BE IND or BE negative for dysplasia by an expert pathology panel. Therefore, cases with initial impression of BE IND or LGD should be reviewed by additional GI pathologists to confirm the diagnosis. Patients with a confirmed diagnosis of BE IND should be placed on intensive acid suppressive therapy and have a surveillance endoscopy with four quadrant biopsies every 1 cm interval in BE segment within one year. BE IND patients with follow-up biopsies which are negative for dysplasia have low risk of neoplasia progression and may be reverted to routine surveillance. The length of BE, multi-focality of BE IND, older age (> 60 years old), abnormal p53 expression, active inflammation, and abnormal DNA content as detected by flow cytometry are useful to risk-stratify this patient population. The role of these predictors in clinical management of patients with BE IND requires further scrutiny.

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