ISSN 1009-3079 (print)
ISSN 2219-2859 (online)

世界华人消化杂志®

WORLD CHINESE JOURNAL OF DIGESTOLOGY

Shijie Huaren Xiaohua Zazhi

2024 年 4 月 28 日　第 32 卷　第 4 期　(Volume 32 Number 4)

4/2024

《世界华人消化杂志》是一本高质量的同行评议, 开放获取和在线出版的学术刊物. 本刊被国际检索系统《化学文摘(Chemical Abstracts, CA)》、《医学文摘库/医学文摘(EMBASE/Excerpta Medica, EM)》、《文摘杂志(Abstract Journal, AJ)》、Scopus、《中文科技期刊数据库(CSTJ)》和《超星期刊域出版平台(Superstar Journals Database)》数据库收录.

ISSN 1009-3079
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编务 吴云晓健; 送审编辑 张砚梁; 组版编辑 张砚梁; 英文编辑 王天奇;
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世界华人消化杂志

Shijie Huaren Xiaohua Zazhi

吴阶平　题写封面刊名
陈可冀　题写版权刊名

(月刊)

创　刊 1993-01-15
改　刊 1998-01-25
出　版 2024-04-28
原刊名 新消化病学杂志

期刊名称
世界华人消化杂志

国际标准连续出版物号
ISSN 1009-3079 (print) ISSN 2219-2859 (online)

编辑部
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7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: wcjd@wjgnet.com
http://www.wjgnet.com

出版
百世登出版集团有限公司
Baishideng Publishing Group Inc
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: bpgoffice@wjgnet.com
https://www.wjgnet.com

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《世界华人消化杂志》是一本高质量的同行评议, 开放获取和在线出版的学术刊物. 本刊被国际检索系统《化学文摘(Chemical Abstracts, CA)》、《医学文摘库/医学文摘(EMBASE/Excerpta Medica, EM)》、《文摘杂志(Abstract Journal, AJ)》、Scopus、《中文科技期刊数据库(CSTJ)》和《超星期刊域出版平台(Superstar Journals Database)》数据库收录.

《世界华人消化杂志》正式开通了在线办公系统(https://www.baishideng.com), 所有办公流程一律可以在线进行, 包括投稿、审稿、编辑、审读, 以及作者、读者和编者之间的信息反馈交流.

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定价
每期136.00元 全年12期1632.00元

WCJD 世界华人消化杂志®

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Volume 32 Number 4 April 28, 2024

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Indexed/Abstracted by Chemical Abstracts, EMBASE/Excerpta Medica, Abstract Journals, Scopus, CSTJ and Superstar Journals Database.

Shijie Huaren Xiaohua Zazhi

Founded on January 15, 1993
Renamed on January 25, 1998
Publication date April 28, 2024

NAME OF JOURNAL
World Chinese Journal of Digestology

ISSN
ISSN 1009-3079 (print) ISSN 2219-2859 (online)

EDITORIAL OFFICE
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World Chinese Journal of Digestology
Baishideng Publishing Group Inc
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: wcjd@wjgnet.com
https://www.wjgnet.com

PUBLISHER
Baishideng Publishing Group Inc
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: bpgoffice@wjgnet.com
https://www.wjgnet.com

PRODUCTION CENTER
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Chaoyang District, Beijing 100025, China
Telephone: +86-10-85381901

PRINT SUBSCRIPTION
RMB 136 Yuan for each issue
RMB 1632 Yuan for one year

INSTRUCTIONS TO AUTHORS
Full instructions are available online at https://www.wjgnet.com/1009-3079/Nav/36. If you do not have web access, please contact the editorial office.

WCJD 世界华人消化杂志®

在线投稿: https://www.baishideng.com

世界华人消化杂志 2024年4月28日; 32(4): 267-275

DOI: 10.11569/wcjd.v32.i4.267

ISSN 1009-3079 (print) ISSN 2219-2859 (online)

文献综述 REVIEW

巴雷特食管: 当前面临的挑战

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基金项目: 2021年广东省自然科学基金项目, No. 2021A1515012127.

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收稿日期: 2024–01–25
修回日期: 2024–02–10
接受日期: 2024–04–15
在线出版日期: 2024–04–28

Barrett's esophagus: Current challenges in diagnosis and treatment

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Supported by: 2021 Natural Science Foundation Project of Guangdong Province, No. 2021A1515012127.

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Received: 2024-01-25
Revised: 2024-02-10
Accepted: 2024-04-15
Published online: 2024-04-28

Abstract

In recent years, the incidence of Barrett's esophagus (BE) has been increasing in China, the Asia-Pacific region, and Western countries. As the only known precancerous lesion and the strongest risk factor of esophageal adenocarcinoma (EAC), BE has a high rate of missed diagnosis and a low survival rate after malignant transformation into EAC. The diagnosis, treatment, and screening of BE play a very important role in the prevention and treatment of early esophageal cancer. However, due to the different incidence rates of BE in different countries and regions, as well as the different awareness and attention to this disease, there are still many controversies over its diagnosis and treatment. This article reviews the current research status and challenges in the diagnosis, detection, and treatment of BE based on the latest guidelines for Barrett's esophagus diagnosis and treatment in the Asia-Pacific region, Europe, and America.

Key Words: Barrett's esophagus; Esophageal adenocarcinoma; Endoscopy; Intestinal metaplasia; Atypical hyperplasia; Precancerous lesions

Citation: Lao SH, Huang JL, Wu LF. Barrett's esophagus: Current challenges in diagnosis and treatment. Shijie Huaren Xiaohua Zazhi 2024; 32(4): 267-275
URL: https://www.wjgnet.com/1009-3079/full/v32/i4/267.htm
DOI: https://dx.doi.org/10.11569/wcjd.v32.i4.267

摘要

近年来, 无论在中国, 亚太地区, 还是西方国家, 巴雷特食管(Barrett's esophagus, BE)的发病率均呈上升趋势, 作为唯一已知的食管腺癌(esophageal adenocarcinoma, EAC)的癌前病变和最强风险因素, 其漏诊率高, 恶变为EAC后生存率低, 其诊疗、筛查对于防治食管早癌具有非常重要的作用. 但由于不同国家及地区的发病率不同, 以及对此病的认识及重视程度各异, 有关BE的诊断和治疗策略仍存在诸多争议. 本文结合亚太、欧美BE诊治的最新指南, 综述BE在

当前诊断、检测及治疗方面的研究现状和挑战.

关键词: 巴雷特食管; 食管腺癌; 内镜; 肠化生; 不典型增生; 癌前病变

核心提要: 巴雷特食管(Barrett's esophagus, BE)在全球的发病率不断上升, 其作为食管腺癌(esophageal adenocarcinoma, EAC)的危险因素, 漏诊率高, 且进展至EAC的生存率低, 但不同国家和地区对BE的认识存在差异, 因此, 本文就BE在诊治方面的研究现状和挑战作一综述.

文献来源: 劳上洪, 黄纪乐, 吴灵飞. 巴雷特食管: 当前面临的挑战. 世界华人消化杂志 2024; 32(4): 267-275
URL: https://www.wjgnet.com/1009-3079/full/v32/i4/267.htm
DOI: https://dx.doi.org/10.11569/wcjd.v32.i4.267

0 引言

食管癌是消化系统中常见的恶性肿瘤之一. 2020年全球癌症数据显示, 食管癌在所有恶性肿瘤中发病率排行第七, 总体死亡率排名第六[1]. 食管鳞状细胞癌(esophageal squamous cell carcinoma, ESCC)和食管腺癌(esophageal adenocarcinoma, EAC)是食管癌的两种主要亚型. 在我国、亚洲和非洲人群中ESCC占90以上[2,3]. 而在巴雷特食管(Barrett's esophagus, BE)基础上发展的EAC则是西方国家食管癌的主要亚型[4]. 据日本食管癌统计报告, 尽管食管鳞状细胞癌占日本所有食管癌病例的90%, 但在过去二十年, EAC在日本的发病率增长三倍[5-7]. 在我国因BE导致的EAC近年亦呈增加趋势[8]. 因而此病愈来愈受到学者们的关注[9-11]. 本文就当今进展进行系统综述.

1 BE的介绍

BE是胃食管反流病(gastro esophageal reflux disease, GERD)的一种类型[12]. 其病理组织学特征为食管远段的鳞状上皮被柱状上皮所取代, 是EAC发生的危险因素, 一旦出现肠化生(intestal metaplasia, IM), 则属于EAC的癌前病变. 目前普遍认为BE是获得性疾病, 并与反流性食管炎密切相关, 少数与遗传及黏膜先天发育不良有关. 该病1950年由英国心胸外科医生Norman Barrett首次提出, 1957年确认, 并逐渐为人们所关注. BE在亚洲国家患病率为0.06%-1.3%[13,14], 在西方国家大约为0.7%-2%[15,16]. 近半个世纪以来, BE的发病率一直呈逐年上升趋势. 在全球人群中, BE的发病率为1%-1.5%[17,18], 每年大约0.1%-0.6% BE进展为EAC[17,19-21], 因此, BE在食管癌防治中具有重要地位.

2 BE的诊断

2.1 BE的诊断标准及其演变 BE的诊断主要依据胃镜检查和食管黏膜的活检结果, 包括以下三方面有争论的内容.

第一: 病变位置的判定. 正常状态下, 内镜下可见的食管鳞状上皮与胃柱状上皮的交界线(squamous-columnar junction, SCJ), 齿状线, 又称Z线, 与胃食管结合部(gastroesophageal Junction, GEJ)处于同一位置, 即三位一体. 一旦食管下段的灰红色复层鳞状上皮被橘红色柱状上皮所取代, 则出现SCJ与GEJ二者位置分离. 在我国, 内镜下可见SCJ相对于GEJ向上移行≥1 cm, 并经病理组织学证实为柱状上皮化生, 可伴或不伴IM即可诊断为BE[22].

准确描述GEJ的位置对于内镜诊断BE至关重要. 食管远端栅栏血管和胃黏膜皱襞近端均可作为鉴别GEJ的标志. 栅栏血管纵行于食管下括约肌的黏膜层, 进入贲门后沿黏膜下层向下, 在解剖学上栅栏血管远端与GEJ边界重合, 日本学者认为以此判定GEJ比较合适[23]. 然而, 世界内镜组织在里约热内卢最新发布的BE共识, 推荐胃黏膜皱襞近侧缘作为判定GEJ位置的首选[24]. 我国[22]与大多数西方国家[24-28]均采用胃黏膜皱襞近侧缘判定GEJ. 但此法亦有缺陷, 因为胃皱襞近端位置很容易随胃运动、呼吸和内镜下的充气程度而发生改变. 目前, GEJ最佳标志仍未定论.

第二: 病变长度的规定. 2017年日本指南中BE被定义为食管远端出现任意长度的黏膜柱状上皮[29]. 但在美国的一项大规模多中心临床试验中发现, 小于1 cm的BE患者5年内进展至高级别异型增生(high grade dysplasia, HGD)或EAC的风险较低[30], 因此美国及其他国家, 包括我国的指南都把BE病变长度设定为1 cm以上[24-28]. 实际上, 小于1 cm的判定存在较高的主观随意性, 较早的美国胃肠病学协会亦没有规定诊断BE要求提供病变长度[31]. 2014年英国胃肠病学首次要求BE≥1 cm[26], 随后大多数国家均采用这一标准, 仅日本仍按原标准诊断BE[29,32]. 此外, 在内镜下根据病变长度BE可分为长段BE(long segment Barrett's esophagus, LSBE, ≥3 cm)和短段BE(short segment Barrett's esophagus, SSBE, <3 cm). 在日本的标准中, LSBE的诊断是在食管出现环周黏膜病变的基础上长度超过3 cm[29], 强调环周黏膜病变的重要性, 与布拉格分型强调全周型化生黏膜的长度的重要性相吻合[33], 且相比于大多数国家只要求长度超过3 cm即诊断LSBE标准更为严格.

第三: 病理组织学的界定. BE的病理诊断一直只强调食管鳞状上皮被柱状上皮所取代, 是否要求IM世界各

地意见不一. 我国指南[22]特地加上“可伴或不伴肠上皮化生”, 与较早的亚太共识[25]、日本指南[29]和英国胃肠病学协会的指南[26]相一致. 我国虽然不将IM的存在作为BE诊断的必要条件, 但仍强调了在制定食管癌监测策略时IM的重要性. 相对而言, 我国和日本诊断BE的标准仍较宽松, 日本2021年指南仍然沿用原来的标准[32]. 然而, 里约热内卢BE共识[24]、2022年美国胃肠病学会(American College of Gastroenterology, ACG)[27]及2023欧洲胃肠内镜协会(European Society of Gastrointestinal Endoscopy, ESGE)[28]等最新指南都明确将IM作为诊断BE的必要条件, 认为BE是EAC的癌前病变. BE诊断标准的演变过程见表1.

2.2 BE的诊断方法

2.2.1 内镜检查: 内镜检查是BE的主要诊断手段, 适用于高危人群检查, 其中GERD是BE最主要的危险因素[35]. 据报道, GERD患者内镜诊断BE为12.0%, 经组织学证实BE为7.2%[36]. 流行病学调查表明, GERD患者BE的发病率为6%-14%, 其中约0.5%-1%会进展为EAC[37]. 近年, 染色内镜、窄带成像(narrow-band imaging, NBI)、荧光内镜、共聚焦激光内镜(confocal laser endoscopy, CLE)等技术在BE及EAC诊治中取得了长足进步. 其中国际NBI小组开发的NBI分类系统对BE诊断的准确率与专家级内镜医生近85%的一致性[38], 显示此分类系统的实用价值. 我国2022版食管癌筛查与早诊早治指南(简称食管癌防治指南)[39]对已诊断或新发现具有巴雷特食管高危因素的患者, 推荐内镜下每隔2 cm行4点位活检. 各种内镜技术检查的优缺点具体见表2.

2.2.2 其它微创检查: 对于具有高危因素、不适合常规内镜检查的患者, 亦可选择经鼻内镜(transnasal endoscopy, TNE)、食管胶囊内镜(esophageal capsule endoscopy, ECE)、光学相干断层摄影术(optical coherence tomography, OCT)、食管新型细胞收集器(细胞海绵Cystoponge、胶囊海绵EsophaaCp)等方法. 这些检查技术的优缺点具体见表3.

2.2.3 细胞生物学: 对BE患者定期检查, 有助于及早发现异型增生或肿瘤[50]. 肿瘤早期阶段会出现遗传和表观遗传修饰、转录和翻译失调以及代谢变异. 表观遗传修饰包括组蛋白翻译后修饰、DNA甲基化模式和染色质重塑等方面的改变[50-52]. 检测TFF3的表达水平有助于BE诊断[53]. ESGE指南建议生物标志物TFF3与Cytosponge结合可作为内镜检查的替代方法[28], p53不仅有助于发现BE异型增生, 还可协助监测病变的进展[54-58]. 不过, 最新欧洲指南认为上述生物标志物目前处于研究阶段, 尚未进入临床实际应用[28]. 我国2022年食管癌防治指南亦不推荐采用生物标志物用于食管癌筛查[39].

3 BE的分型

3.1 按病变长度分型 BE在内镜下根据病变长度分为LSBE, SSBE和超短段BE(ultrashort segment Barrett's esophagus, USSBE), <1 cm. 后者由于其癌变风险较低且观察者间一致性较差, 目前多数指南已删除该定义. 日本一项关于LSBE的多中心前瞻性队列研究发现, 人群中SSBE发病率明显高于LSBE, LSBE进展为EAC的发生率为每年1.2%[59]. Hamade等[9]在欧美对1883名无异型增生的BE患者进行为期6年的多中心观察, 结果表明SSBE患者进展EAC的年发生率(0.07%)明显低于LSBE(0.25%). 荷兰的一篇报道证实BE的长度、病理分级和年龄均是此病进展为EAC的重要危险因素[60], 表明较长BE节段进展为EAC风险更大[61].

3.2 内镜下标准分型 目前西方内镜分型主要采用布拉格(Prague C&M)标准[33]. 它以全周型化生黏膜的长度C和最大长度M为基础, 对BE进行内镜分级, 且有较高的可信度. 有数据表明, 严格按布拉格分类, 内镜下异型增生的检出率明显增加[62]. 然而, 亚洲巴雷特联盟进行569个国家/或地区的调查中发现, 仅有16.3%的内镜医师在评估BE时使用布拉格标准, 多数医生在内镜操作时仍使用本地区制定的标准, 32.3%的内镜医师从未使用该标准[63].

3.3 BE的组织病理学分型 在美国2022 AGA共识中建议在诊断BE进行取样时应使用西雅图活检方案[64](每1 cm-2 cm进行4象限活检, 针对任何可见病灶进行活检[35]); 而欧洲2023年ESGE指南则要求对可见黏膜异常处每2 cm进行随机四象限活检[28]. 病理诊断除化生外还要明确是否有异型增生及癌变. 我国2022年食管癌防治指南参照美国标准每隔2 cm行4点位活检(至少8块组织)[39].

4 BE的筛查和监测

4.1 BE的筛查 大多数指南不建议一般人群进行内镜筛查. 对于具有高危因素(男性、白人、大于50岁、吸烟史、慢性胃食管反流病史、肥胖、BE和EAC家族史)的患者才进行内镜筛查. 除内镜外, 还可应用Cystoponge等食管上皮细胞采集技术或虚拟染色内镜进行观察, 不推荐对初次内镜筛查结果阴性的患者进行重复筛查[35]. 除了WATS-3D作为辅助手段可提高BE诊断率[28]外, 洛杉矶分类也可用于辅助筛查. 文献报道[65], 按洛杉矶分类标准, 中重度反流性食管炎患者27%合并有BE, 这些患者应及时进行抗返流治疗及监测, 以防病变进展.

4.2 BE的监测 目前, 标准白光内镜和色素内镜均可用于BE监测, 结合多部位活检可最大限度减少BE诊断的偏差[34]. 在2022年ACG[27]和美国胃肠病协会(American Gastroenterological Association, AGA)[35]指南中, 均建议对

表 1 巴雷特食管的诊断标准

指南	GEJ标志	长度标准	组织学	参考文献
欧洲ESGE(2023、2017)	胃黏膜皱襞近侧缘	≥1 cm	肠上皮化生	[28,34]
美国ACG(2022)	胃黏膜皱襞近侧缘	≥1 cm	肠上皮化生	[27]
日本(2021、2016)	食管纵行栅栏样血管末端	任意长度	柱状上皮化生	[29,32]
里约热内卢共识(2020)	胃黏膜皱襞近侧缘	≥1 cm	肠上皮化生	[24]
中国(2017)	胃黏膜皱襞近侧缘	≥1 cm	柱状上皮化生	[22]
亚太共识 (2016)	胃黏膜皱襞近侧缘	≥1 cm	柱状上皮化生	[25]
英国BSG(2014)	胃黏膜皱襞近侧缘	≥1 cm	柱状上皮化生	[26]
美国ACG(2008)	胃黏膜皱襞近侧缘	任意长度	肠上皮化生	[31]

GEJ: 胃食管结合部; ESGE: 欧洲胃肠内镜协会; ACG: 美国胃肠病学会; BSG: 英国胃肠病学会; AGA: 美国胃肠病协会.

表 2 内镜检查

内镜种类	优点	缺点	参考文献
HD-WLE	视野更宽、显相更清晰	成本高, 对肿瘤病变漏诊率高	[40]
染色内镜	宽视野成像, 黏膜增强; 高精度识别IM和异型增生区域, 减少活检次数, 价格便宜	亚甲蓝染色剂可能有致癌性; 耗费时间	[41-43]
NBI	不使用染色剂, 无潜在毒性; 易于操作, 诊断HGD高灵敏度(96%)和特异性(94%)	仍然需要白光内镜检查作为辅助治疗	[41,43]
荧光内镜	敏感性较好	特异性较差, 假阳性率高	[41,43]
CLE	减少活检次数	设备成本高, 医师技术要求高	[43]

HD-WLE: 高清白光内镜; IM: 肠化生; NBI: 窄带成像; CLE: 共聚焦激光内镜.

表 3 其它微创检查

非内镜种类	优点	缺点	参考文献
TNE	安全、耐受性好、成本低; 无需全身镇静	光学能力有限; 对医生技术要求高	[42]
ECE	安全, 患者耐受性好	成本效益尚高	[44,45]
OCT	无需与组织接触, 诊断HGD与EAC高灵敏度(83%)与特异性(75%)	仍需普通内镜引入	[46]
EsophaCap	相比Cystoponge更小更软; 患者耐受性好, 并发症少	发现异常, 仍需内镜检查治疗	[47]
Cystoponge	安全, 耐受性好, 成本低下, 不依赖操作者、速度快, 无需专门的设备以及培训; 诊断BE特异性(92.4%)敏感性(79.9%)	发现异常, 仍需内镜检查治疗	[48,49]

TNE: 经鼻内镜; ECE: 食管胶囊内镜; OCT: 光学相干断层摄影术; EsophaCap:胶囊海绵; Cystoponge: 细胞海绵; BE: 巴雷特食管.

无异型增生的BE患者按照病变长度确定监测时间: BE长度≥3 cm的患者每隔3年进行1次内镜复查; BE<3 cm的患者则每隔5年进行1次内镜复查. 若在监测期间病情进展, 则需要重新评估以选择合适的治疗方案. 2023年ESGE[28]指南与美国指南大致相同, BE长度1-3 cm, 每5年进行一次胃镜检查, 但对于不规则Z线型/柱状食管<1 cm的患者, 不建议进行常规活检或内镜监测; 对于长度≥10 cm的患者建议转诊至Barrett食管中心进一步诊治. 我国则推荐无异型增生的BE患者, 每隔3-5年进行1次内镜检查及病理活检; 如伴有异型增生/低级别上皮内瘤变, 则需行食管内镜下治疗[39].

5 BE的治疗

5.1 非内镜治疗 肥胖和不良饮食习惯会增加胃食管反流的发生, 同时生活中应该避免如甜品、辛辣食物、咖啡、浓茶等食物. 使用阿司匹林和摄入维生素C、叶酸和膳食纤维可显著降低BE的风险[66]. 长期胃酸和胆汁反流是BE的重要危险因素, 有研究发现, 控制反流可明显减缓BE的进展[67]. 质子泵抑制剂不仅可缓解反流症状, 预防巴雷特食管发生, 还能阻止病变的进展[68]. 每日服

用质子泵抑制剂(proton pump inhibitors, PPIs)两次(PPIs-BID)联合冷冻消融, 在BE的任何阶段都是最经济、高效且安全的方法[69]. Thorell等[70]认为, 在接受PPI治疗或接受抗反流手术的患者中会降低BE进展为HGD/EAC的风险, 显示抗反流治疗对BE及EAC有预防作用.

5.2 内镜治疗

5.2.1 非异型增生: 根据BE病变程度进行分层, 再选择相应的治疗方法[35]. 仅有上皮化生而没有异型增生的患者不建议内镜治疗, 因为此类患者并不能从风险成本比中获益, 但应每间隔一段时间进行内镜复查, 已了解病变进展状况[28]. 在我国, 明确有IM的此类患者, 1年后建议内镜活检复查, 以降低组织取样误差的风险.

5.2.2 异型增生(不典型增生): (1)低级别异型增生(Barrett's esophagus with low-grade dysplasia, LGD): 欧洲ESGE建议使用消融术进行治疗[28]. 我国也建议射频消融治疗[39], 对于未行治疗者强调每6 mo-12 mo应随访1次. 美国ACG则建议内镜随访或内镜根除疗法(endoscopic eradication therapy, EET)以降低进展为HGD或食管腺癌的风险[27]. EET主要包括内镜下黏膜切除术(endoscopic mucosal resection, EMR)和射频消融术(radio frequency ablation, RFA)和冷冻疗法(cryotherapy). 在法国一项大型的联合14个中心的前瞻性研究发现RFA可降低了LGD的患病率和3年时的进展风险[71]. Pouw报道[72]一组LGD患者射频消融后随访73 mo, 超过90%的患者获得了持续治愈. RFA在多发、病变较长或累及食管全周的BE及早期食管癌治疗中具有明显优势. 然而, 亦有不同的结果[73]. 在一项长达10年的随访研究中, 430例接受RFA治疗的LGD患者, 78.4%出现了IM[74]; (2)高级别异型增生(Barrett's esophagus with high-grade dysplasia, HGD)及早期癌: 美国ACG对HGD和早癌患者建议予EET治疗, 并强调完整切除病灶及治疗后随访[27]. 使用EET切除BE患者的年复发风险为1.0%[75,76]. 欧洲ESGE则根据BE病灶黏膜浸润深度采取不同的治疗[28]. 对于扁平HGD的BE, 建议内镜下进行RFA治疗. 但也有学者建议3 mo复查后再行治疗更为合理[77]. 对于HGD≤黏膜内20 mm, 推荐使用EMR. 这个阶段无法仅根据活检钳获取的材料来确定病变的深度, 但发生淋巴结转移的风险较较低(<1%)[78]. 对于HGD>黏膜20 mm或存在黏膜下浸润的病变, 建议内镜黏膜下剥离术(endoscopic submucosal dissection, ESD)完整切除. 我国2022年食管癌防治指南中, 对于具有内镜切除适应证的患者, 首选ESD; 若病变长径≤10 mm, 如果能保证整块切除, 亦可考虑EMR[39]. van Munster等[79]对来自荷兰9个BE研究中心130例HGD及早期EAC患者进行ESD切除, 发现总体完全切除率为97%, T1a、T1b的EAC的R0分别为87%、49%. 病变侵入黏膜的位置越深, 其癌变的风险越大. 一旦病变延伸到整个粘膜下层, 则不适合内镜治疗. 对于此类患者, 传统手术切除更为合适[78].

5.3 治疗热点-人工智能AI 尽管内镜技术在不断进步, 但是诊断BE后早期食管癌的高漏诊率仍然是当前面临挑战之一. 目前, 使用人工智能技术协助早癌诊断在此领域初露头角. Ebigbo小组[80]研究发现AI系统对食管早癌诊断准确率达89.9%. van der Sommen等[81]开发可以识别BE癌前病变及癌变的AI系统, 对早癌诊断的敏感性和特异性均超过80%. Knabe等[82]使用AI技术对食管早癌诊断的总准确率达73%. 德国一项研究证实AI系统在识别食管早癌(T1a和T1b期)的敏感性、特异性和准确率方面与病理学专家没有统计学差异[83], 显示AI在BE及早癌筛查领域有广阔的应用前景.

6 结论

以内镜筛查有GERD症状的BE患者并定期随访的预防策略, 有助于EAC的早期发现及早期治疗. 开发Cytosponge等食管上皮细胞采集技术, 尤其是能及时识别有进展风险的高敏感、高特异性生物标志物及AI技术具重要意义. 进一步完善我国BE诊断质量标准, 建立全国性数据管理系统, 开展多中心大样本临床队列研究, 开发多维度、综合性BE风险分层预测模型并实时收集数据, 对于异型增生的病变及早消融或切除, 将有助于提高我国BE的诊疗水平并为食管癌的防治做出贡献.

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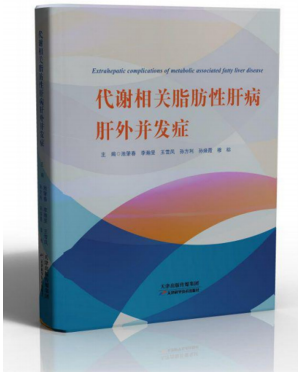
科学编辑: 张砚梁 制作编辑: 张砚梁

ISSN 1009-3079 (print) ISSN 2219-2859 (online) DOI: 10.11569

• 消息 •

书讯



本刊讯 由池肇春教授等主编的《代谢相关脂肪性肝病肝外并发症》已由天津科学技术出版社出版发行.

本书的出版为国内首创, 填补了国内有关这方面的空白, 拓宽了对《代谢相关脂肪性肝病》认识的高度和深度. 《代谢相关脂肪性肝病肝外并发症》分总论和各论两部分. 1-4章为总论, 分别介绍代谢相关脂肪性肝病肝外并发症研究现状与进展, 包括发病风险、发病机制和治疗近展; 脂肪代谢生物化学和分子生物学; 代谢相关脂肪性肝病肝外并发病免疫学; 肠道微生物生态失衡与代谢相关脂肪性肝病肝外并发病. 5-18章为各论, 分别介绍代谢相关脂肪性肝病肝外并发症与机体各系统疾病的相关性. 可为消化科、肝病科、内分泌代谢科、普外科、肿瘤科、影像科、其他相关科临床医师和从事MAFLD研究的人员学习和参考.

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ISSN 1009-3079