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共同主编

党双锁, 博士, 教授, 研究员, 主任医师, 710004, 陕西省西安市, 西安交通大学医学院第二附属医院感染科

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Editor-in-Chief of *World Chinese Journal of Digestology*, Kai-Feng Wang, Chief Physician, M.D., Department of Oncology, Shanghai Jiaotong University School of Medicine Affiliated Renji Hospital, No. 160 of Pujian Road, Pudong New Area, Shanghai 200000, China. wangkf2000@zju.edu.cn

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Barrett食管危险因素的研究进展

马韶泽, 陈鸿鑫, 梁振东, 祁兴顺

马韶泽, 陈鸿鑫, 梁振东, 祁兴顺, 北部战区总医院消化内科 辽宁省沈阳市 110840

马韶泽, 大连医科大学研究生院 辽宁省大连市 116044

陈鸿鑫, 辽宁中医药大学研究生院 辽宁省沈阳市 110031

马韶泽, 硕士, 主要从事消化系统疾病方面的研究.

作者贡献分布: 本文由马韶泽查阅文献及撰写; 陈鸿鑫、梁振东校正文章书写及格式; 祁兴顺审核.

通讯作者: 祁兴顺, 博士, 博士后, 副主任医师, 110840, 辽宁省沈阳市文化路83号, 北部战区总医院消化内科. xingshunqi@126.com

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Risk factors for Barrett's esophagus: Recent advances

Shao-Ze Ma, Hong-Xin Chen, Zhen-Dong Liang, Xing-Shun Qi

Shao-Ze Ma, Hong-Xin Chen, Zhen-Dong Liang, Xing-Shun Qi, Department of Gastroenterology, General Hospital of Northern Theater Command, Shenyang 110840, Liaoning Province, China

Shao-Ze Ma, Graduate School of Dalian Medical University, Dalian 116044, Liaoning Province, China

Hong-Xin Chen, Graduate School of Liaoning University of Traditional Chinese Medicine, Shenyang 110031, Liaoning Province, China

Corresponding author: Xing-Shun Qi, M.D, Vice Chief Physician, Department of Gastroenterology, General Hospital of Northern Theater Command, No. 83 Wenhua Road, Shenyang 110840, Liaoning Province, China. xingshunqi@126.com

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Abstract

Esophageal adenocarcinoma (EAC) is the most common malignant tumor of the esophagus in the West. During the past few decades, its morbidity has been increasing in China. Barrett's esophagus (BE) is defined as the replacement of normal squamous epithelium in the lower esophagus by metaplasia of columnar epithelium. BE is closely related to the occurrence of EAC. Knowledge regarding the risk factors for the occurrence and development of BE is of great significance for early screening and diagnosis of BE and prevention of EAC. In this paper, we review the clinical, demographics-related, lifestyle-related, and medications-related risk factors for BE to provide more valuable scientific evidence for the prevention and treatment of BE.

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Key Words: Barrett's esophagus; Esophageal adenocarcinoma; Gastroesophageal reflux disease; Risk factors

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摘要

食管腺癌(esophageal adenocarcinoma, EAC)是西方国家最常见的食管恶性肿瘤. 近年来, 其发病率在我国也呈上升趋势. Barrett食管(Barrett's esophagus, BE)是指食管下段的鳞状上皮被化生的柱状上皮所取代的病理现象. BE与EAC的发生密切相关. 了解BE发生发展的相关危险因素对早期筛查和诊断BE及预防EAC具有重要意义. 本文主要从临床、人口统计学、生活习惯和药物使用四个方面阐述BE的危险因素, 以期BE的临床防治提供更多有价值的科学依据.

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关键词: Barrett食管; 食管腺癌; 胃食管反流病; 危险因素

核心提要: Barrett食管(Barrett's esophagus, BE)是食管腺癌(esophageal adenocarcinoma, EAC)的癌前病变. 充分了解BE的危险因素是十分必要的. 本文将从临床、人口统计学、生活习惯和药物四个方面阐述BE的危险因素, 以指导临床防治BE.

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0 引言

食管癌是常见的上消化道恶性肿瘤之一, 其发病率及死亡率分别居世界恶性肿瘤的第8位及第6位^[1]. 在西方国家, 食管腺癌(esophageal adenocarcinoma, EAC)已替代食管鳞癌成为最常见的食管癌病理类型^[2], 近年来, 其发病率在中国也呈上升趋势^[3]. Barrett食管(Barrett's esophagus, BE)是指食管与胃黏膜交界处的连接线(齿状线, 又称Z线)1 cm以上的食管下段的正常复层鳞状上皮被化生的单层柱状上皮所取代的一种病理现象^[4]. BE是目前唯一已知的EAC的癌前病变^[5], 有报道显示^[6], 80%的EAC与BE密切相关, BE患者罹患EAC的风险是正常人群的30-40倍^[7]. 因此, 识别和了解BE发生发展的相关危险因素对其早期诊治及降低EAC的发病率具有重要意义. 本文通过全面概述BE发生发展的相关危险因素的临床研究证据, 为BE的临床防治提供更多有价值的科学依据.

1 临床相关危险因素

1.1 胃食管反流病和反流症状 胃食管反流病(gastroesophageal reflux disease, GERD)是指胃十二指肠内容物反流至食管引起的相关症状和(或)并发症的疾病, 是公认发生BE最主要的危险因素^[8]. 一项基于爱尔兰人群的病例对照研究证实, GERD与发生BE显著相关($OR = 12.0$, $95\%CI = 7.64-18.70$)^[9]. 然而, 并非所有的GERD患者均会出现反酸、胃灼热等反流症状^[10,11]. 为此, 多项研究也进一步探讨了反流症状与发生BE之间的关系, 它们发现, 反流症状的发作频率和严重程度与BE的发生风险呈正相关^[11-18]. 一项纳入了26项研究的荟萃分析发现, 反流症状与短段BE的发生风险无关($OR = 1.15$, $95\%CI = 0.76-1.73$), 但与长段BE的发生风险显著相关($OR = 4.92$, $95\%CI = 2.01-12.00$), 反流症状似乎只能作为长段BE的可靠预测指标^[19]. 持续的胃酸反流可诱导食管鳞状上皮

改变发育转录因子的表达模式, 使其逐步转化为柱状上皮和肠上皮, 最终发展为BE^[20]. 除胃酸反流外, 胆汁反流在BE的发生发展中也可能起着重要作用. 由胆汁酸诱导的食管组织氧化应激和DNA损伤可激活BE细胞的抗凋亡通路并增加炎症反应, 从而促进BE的发生发展^[21]. 当前指南认为, 长期GERD病史和(或)>5年的反流症状是BE的危险因素^[4,22-25].

1.2 食管裂孔疝 食管裂孔疝(hiatal hernia, HH)是指除食管以外的任何腹腔组织结构通过扩大的食管裂孔进入胸腔形成的疝^[26]. BE患者中HH的发病率高达96%^[27]. 一项横断面研究发现, BE患者HH的患病率显著高于非BE患者(79.8% vs 52.8%, $P < 0.01$); 并且HH是BE的独立危险因素($OR = 3.04$, $95\%CI = 1.77-5.23$, $P < 0.01$)^[28]. 一项纳入了13项研究的荟萃分析也证实, HH显著增加了BE的发生风险($OR = 2.74$, $95\%CI = 1.58-4.75$)^[29]. 这可能是因为HH可降低食管下括约肌(transient lower esophageal sphincter, LES)压力, 从而加重胃酸和胆汁反流, 最终增加BE的发生风险.

1.3 中心性肥胖 根据脂肪分布部位, 肥胖可分为全身性肥胖和中心性肥胖. 中心性肥胖是指以腹部或内脏脂肪积聚为主引起的肥胖, 比全身性肥胖对健康的危害更大, 是多种慢性病的重要危险因素^[30]. 研究表明^[15,31-33], 中心性肥胖是发生BE的独立预测因素. 两项病例对照研究比较了有无BE患者的脂肪分布、脂肪含量和体脂百分比, 以探讨肥胖类型与BE的关系; 结果显示, 腰围、腰臀比等中心性肥胖指标与BE的患病风险呈正相关, 而体重指数(body mass index, BMI)、脂肪含量、体脂百分比等全身性肥胖指标与BE的患病风险无关^[15,32]. 此外, Nelsen等^[33]发现内脏脂肪($OR = 4.88$, $95\%CI = 1.04-22.85$, $P < 0.01$)和胃食管交界处脂肪($OR = 5.97$, $95\%CI = 1.28-27.74$, $P < 0.01$)堆积也会增加BE的患病风险, 且不受BMI的影响. 多项荟萃分析也证实, 通过腰围、腰臀比和(或)内脏脂肪组织面积鉴定的中心性肥胖可独立预测BE的发生风险^[34-36]. 这是由于中心性肥胖可以导致腹内压力增加并机械地破坏胃食管交界处屏障的完整性, 从而促进胃酸和胆汁反流, 进而加重反流性食管炎并诱导食管鳞状上皮化生, 最终导致BE形成^[37-40]. 另一方面, 中心性肥胖患者内脏脂肪组织代谢活跃, 促进了白细胞介素-6(interleukin-6, IL-6)、白细胞介素-1 β (interleukin-1 β , IL-1 β)、肿瘤坏死因子- α (tumor necrosis factor- α , TNF- α)等炎性细胞因子和脂联素(adiponectin, APN)、游离脂肪酸(free fatty acid, FFA)、瘦素(leptin, LEP)等脂肪因子的释放, 这些生物活性物质通过远距分泌和旁分泌的方式发挥全身慢性炎症作用, 独立或协同地促进BE的发生发展^[40-45].

1.4 阻塞性睡眠呼吸暂停综合征 阻塞性睡眠呼吸暂停综合征(obstructive sleep apnea syndrome, OSAS)是指睡眠期间气道部分或完全阻塞, 导致以间歇性缺氧和低通气为主要临床表现的一种潜在致死性综合征^[46]. OSAS患者睡眠时常因呼吸道阻塞导致吸气时胸腔负压和食管内负压增高, 且呼吸暂停后连续吸气会引起胸腔和LES压力降低, 从而加重胃食管反流并诱发BE^[47,48]. 此外, OSAS患者多肥胖^[49], 相较于健康人群, 肥胖人群的腹内压力显著更高, 因此更容易引起胃食管返流, 最终导致BE的发生^[41]. 最近, Hadi等^[50]的研究结果显示, BE患者OSAS的患病率显著高于非BE患者(90.7% vs 72.4%, $P<0.01$). 当校正GERD和BMI后, OSAS仍显著增加了BE的发生风险($OR = 3.26$, 95%CI = 1.72-6.85, $P<0.01$), 且随着OSAS的程度加重, BE的发生风险随之上升, 这可能与缺氧诱导的全身慢性炎症反应有关, 而不完全依赖于胃食管反流和肥胖^[50].

1.5 糖尿病 仅少数研究探讨了糖尿病(diabetes mellitus, DM)与BE之间的关联, 且研究结果并不一致. 一项大型的病例对照研究发现, BE患者的DM患病率显著高于非BE患者(5.8% vs 5.3%, $P<0.05$); 校正BMI、GERD等危险因素后, DM仍显著增加了BE的发生风险 ($OR = 1.49$, 95%CI = 1.16-1.91, $P<0.01$)^[51]. 然而, 另一项基于英国人群的病例对照研究发现, DM并不会增加BE的发病风险 ($OR = 1.07$, 95%CI = 0.98-1.18)^[52]. 最近一项荟萃分析也发现, DM与BE无关($OR = 0.93$, 95%CI = 0.71-1.20), 但显著增加了EAC的发生风险($OR = 1.63$, 95%CI = 1.19-2.22). 这些研究结果提示DM可能不会影响BE的发病, 但会促进BE进展至EAC^[53].

1.6 代谢综合征 代谢综合征(metabolic syndrome, MS)是以胰岛素抵抗、中心性肥胖、高脂血症及高血压等疾病为主的症候群^[54]. 多项研究显示, MS是发生BE的高危因素^[52,54,55]. 最近, 一项纳入了14项研究的荟萃分析也证实, MS显著增加了BE的发病风险($OR = 1.35$, 95%CI = 1.15-1.60, $P<0.01$)^[56]. 此外, 一项基于白人男性退伍军人的病例对照研究表明, MS的严重程度与BE的发生风险呈剂量-反应关系, 即BE的发病风险随着MS成分数量的增加而呈线性增加, 且高脂血症对BE的发生风险影响最大^[57]. 除了脂肪因子可发挥全身慢性炎症作用外^[45], MS患者血清中的高甘油三酯水平还会促进胆囊收缩素的分泌, 从而延缓胃排空并降低LES压力^[58], 最终加重胃食管反流并诱导BE形成. 此外, 胰岛素样生长因子-1(insulin-like growth factor-I, IGF-1)与靶细胞表面的相应受体结合可调控食管上皮细胞的增殖和分化^[59]. 因此, 继发于MS的血清高IGF-1水平可能会进一步增加MS患者发生BE的风险^[60].

1.7 幽门螺杆菌感染和幽门螺杆菌根除治疗 幽门螺杆菌(*Helicobacter pylori*, *H. pylori*)是一种定植于胃黏膜的微需氧革兰氏阴性菌, 1994年世界卫生组织将其认定为I类致癌原^[61]. 多数研究认为, *H. pylori*感染, 尤其是CagA阳性菌株感染, 与BE的发病风险呈负相关^[62-65]. 最近, 一项荟萃分析也表明, *H. pylori*感染可能是发生BE的保护因素($OR = 0.70$, 95%CI = 0.51-0.96, $P<0.05$), 并且CagA阳性菌株感染对发生BE的保护作用更为明显($OR = 0.28$, 95%CI = 0.15-0.54, $P<0.01$)^[66]. 这可能是因为持续的*H. pylori*感染通过破坏壁细胞的功能、升高LES压力以及减少一过性食管下括约肌松弛(transient lower esophageal sphincter relaxations, TLESR)的频率及持续时间, 减少了胃酸的分泌和反流, 最终降低了BE的发生风险^[37,67,68]. 目前, 仅有一项基于瑞典人群的前瞻性队列研究探讨了*H. pylori*根除治疗与BE发病风险的关系, 结果显示, *H. pylori*根除治疗后, 瑞典人群BE的发病率显著增加($SIR = 3.67$, 95%CI = 3.15-4.25)^[69], 提示*H. pylori*根除治疗可能是BE的危险因素.

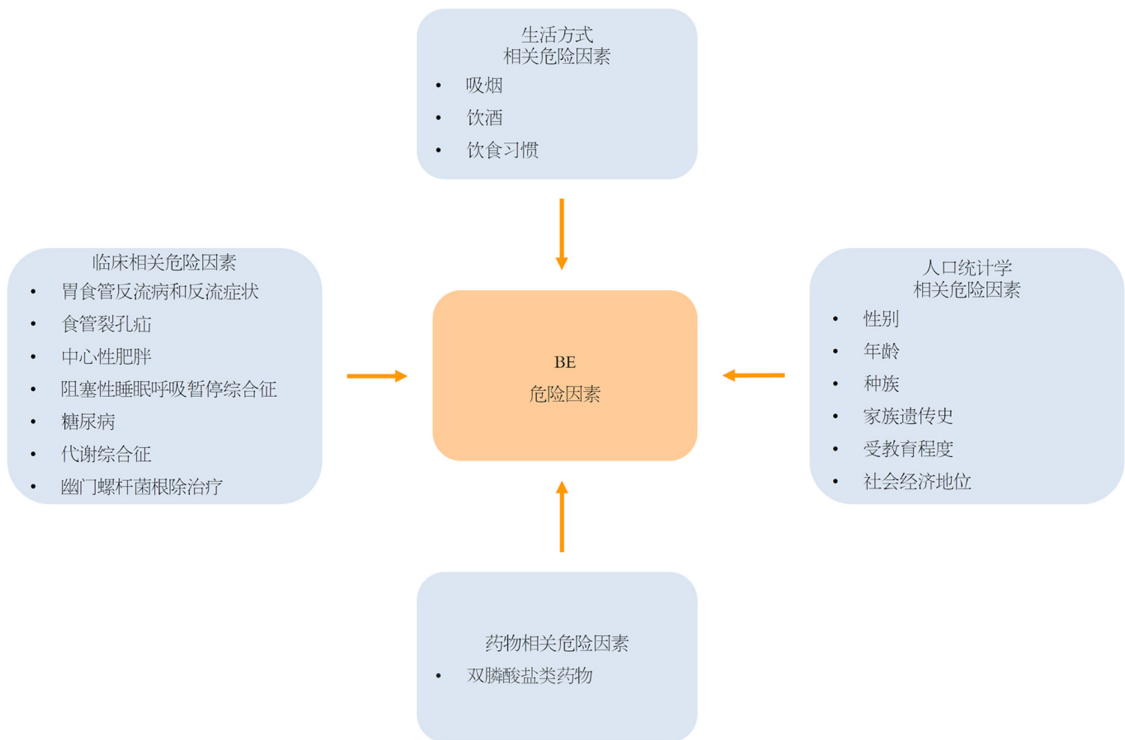
2 人口统计学相关危险因素

2.1 性别 男性BE的发病率是女性的1.5-4.0倍^[70-72]. 性激素的差异可能在一定程度上解释了这一现象. 一项基于男性的病例对照研究发现, 游离睾酮($OR = 5.36$, 95%CI = 2.21-13.03, $P<0.01$)和游离双氢睾酮($OR = 4.25$, 95%CI = 1.87-9.66, $P<0.01$)可显著增加BE的发生风险, 睾酮可降低LES压力并增加TLESR的频率, 从而加重胃食管反流^[73]. 一项动物实验也发现, 雌激素可通过抑制壁细胞的功能并减少壁细胞的数量, 以降低基础胃酸分泌, 从而降低BE的发生风险^[74].

2.2 年龄 年龄是发生BE的独立危险因素. 年龄每增加10岁, 罹患BE的风险将增加1-2倍^[13,75,76]. 一项来自英国和荷兰的多中心、回顾性队列研究比较了不同年龄段人群发生BE的风险; 结果显示, 在40岁-44岁组, 英国和荷兰BE的发病率分别为16/10⁵和24/10⁵; 在70岁-74岁组, 英国和荷兰BE的发病率分别增至85.6/10⁵和87/10⁵, BE的发病率与年龄呈线性相关^[77]. 目前国内外多项指南也推荐, 年龄>50岁是发生BE的独立预测因素^[4,22-25,78].

2.3 种族 全球范围内, 白人种族BE的发病率最高^[79]. 一项纳入20412例患者的观察性研究发现, 白人发生BE的风险是黄种人的6倍($OR = 6.03$, 95%CI = 3.56-10.22)^[72]; 同样, 一项来自美国的大型横断面研究表明, 白人BE的发病率是黑人的5倍^[71]. 目前尚不清楚种族对BE发生发展的作用机制. 白人种族肥胖率更高可能是其BE高发率的原因^[80].

2.4 家族遗传史 BE可能是具有遗传倾向的后天性疾病^[81],



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图 1 BE的危险因素. BE: Barrett食管.

BE及其相关癌症家族史可增加BE的发生风险. Chak等^[82,83]发现, 约7.3%的BE患者有BE或EAC家族史. 目前, 几项全基因组关联研究已确定CRTC1、FOXP1、BARX1、TBX5、GDF7等是BE和EAC的易感基因^[84-86].

2.5 受教育程度 仅少数研究探讨了受教育程度对BE发病风险的影响. Kendall等^[87]发现, 受教育程度与BE的发病风险呈负相关, 随着受教育程度的提高, BE的患病率显著降低. 受教育程度高的患者依从性好, 自身接受能力、自我保健意识、认知疾病的能力强, 有利于控制危险因素^[88]. 因此, 受教育程度高的人群罹患BE的风险较低.

2.6 社会经济地位 一项英国的病例对照研究表明, 较高的社会经济地位可能是发生BE的独立危险因素(OR = 1.58, 95%CI = 1.15-2.15)^[72]. 此外, 一项纳入了20,975例受试者的回顾性队列研究也发现, 与低社会经济地位人群相比, 高社会经济地位人群BE的患病率显著更高($P<0.01$), 且确诊为BE时的年龄更小^[89]. 这似乎与受教育程度高的人群罹患BE的风险更低的结论相矛盾. 然而我们需要澄清的是, 高社会经济地位人群常有着高糖高脂的饮食习惯^[90], 且就医条件更好, 可能会通过更频繁的内镜检查发现BE的患病情况^[89].

3 生活方式相关危险因素

3.1 吸烟 吸烟可能会增加BE的发生风险. BE患者吸烟史

的比例显著高于人群对照组, 且BE的发生风险与吸烟包年数呈正相关^[91]. Balasubramanian等^[92]进一步指出, 吸烟的GERD患者BE的患病率显著高于无吸烟史的GERD患者(5.2% vs 17.0%, $P<0.01$), 吸烟是GERD患者发生BE的独立危险因素; 随着戒烟时间的延长, BE的发生风险逐渐降低(P for trend <0.01). 此外, 一项随访16年的前瞻性队列研究也发现, 吸烟史和吸烟时长均显著增加了BE的发生风险^[93]. 这是由于尼古丁可降低LES压力并导致频繁的TLESR, 从而促进胃食管反流, 最终诱导Barrett上皮生成^[94].

3.2 饮酒 多数研究未发现饮酒与BE之间存在直接关联^[13,89,93,95-98]. 然而, 最近一项纳入了62项观察性研究的荟萃分析却表明, 饮酒可能是发生BE的危险因素(RR = 1.23, 95%CI = 1.13-1.34, $P<0.01$)^[99]. 这可能是由于酒精会导致LES压力降低并加重反流症状, 从而引起食管黏膜损伤并诱导BE形成^[98].

3.3 饮食习惯 近期, 多项研究探讨了饮食习惯与BE的相关性. 随着糖类^[100-102]和脂肪^[102,103]摄入量的增加以及膳食纤维^[104,105]和富含膳食抗氧化剂的蔬菜、水果^[101,102,106-108]摄入量的减少, BE的发生风险将逐渐增加. 高糖高脂和低膳食纤维饮食通过损伤食管黏膜或影响其它危险因素, 如GERD、HH和中心性肥胖, 直接或间接地促进BE的发生发展^[109]. 当蔬菜、水果的摄入量过少时, 人体便会缺乏某些抗氧化活性成分(维生素C、维生素E、硒、

镁和β-胡萝卜素^[101,107,108],从而诱导食管组织氧化应激和DNA损伤,进而促进BE细胞增殖并增加炎症反应,最终增加BE的发生风险^[110-112]。

4 药物相关危险因素

仅少数研究探讨了药物和BE发病风险之间的相关性。一项基于美国退伍军人的病例对照研究比较了有无BE患者双膦酸盐类药物的使用情况,结果发现,BE患者双膦酸盐类药物的使用率显著高于非BE患者(4.6% vs 2.5%),且口服双膦酸盐类药物可能是发生BE的危险因素(OR = 2.33, 95%CI = 1.11-4.88, $P < 0.01$)^[113]。双膦酸盐是破骨细胞介导的骨质吸收的选择性抑制剂,用于治疗 and 预防骨质疏松症^[113]。它可通过抑制甲羟戊酸途径干扰细胞周期进程,从而影响食管上皮干细胞的增殖与分化,最终加重胃食管反流引起的食管损伤并诱导Barrett上皮生成^[114]。此外,有研究也发现,非甾体类抗炎药^[99,115]和他汀类药物^[116]的使用可能会降低BE的发生风险。

5 结论

作为目前唯一已知的EAC的癌前病变,BE在全球的发病率不断升高。因此,掌握BE发生发展的危险因素对于EAC的预防、风险分层、早期识别和诊治至关重要。除性别、种族、遗传等不可变因素外,以GERD、中心性肥胖、吸烟等为主的可变危险因素应引起重视(图1)。未来应正确引导人们认识这些危险因素,提高人群健康意识,保持良好的生活习惯,从而降低BE和EAC的发病率。

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