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肝硬化胃静脉曲张内镜下组织黏合剂注射治疗的并发症及防治策略

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Endoscopic tissue adhesive injection for gastric varices secondary to hepatic cirrhosis: Complications and management strategies

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Abstract

Gastric varices is a common clinical presentation of portal

hypertension induced by hepatic cirrhosis, and gastric variceal bleeding is an emergency with high mortality. Endoscopic tissue adhesive injection plays an important role in the primary and secondary prophylaxis of gastric variceal bleeding, and acute bleeding control. However, this is a technique-demanding operation with nonnegligible complications, of which some may be fatal, such as ectopic embolism. Therefore, the prevention and treatment of such complications are of great importance. Herein, we present a review summarizing the complications and treatment strategies of endoscopic tissue adhesive injection for gastric varices secondary to hepatic cirrhosis.

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Key Words: Gastric varices; Endoscopic tissue adhesive injection; Hepatic cirrhosis; Complication

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摘要

胃静脉曲张是肝硬化门静脉高压常见的临床表现之一, 胃静脉曲张破裂出血是临床常见急症, 病死率高. 内镜下组织黏合剂注射在胃静脉曲张出血的一级预防、控制急性出血和二级预防起着至关重要的作用. 然而该操作有一定难度, 存在一定的并发症风险, 部分并发症比如异位栓塞, 严重时可危及生命, 因此其防治非常重要. 本文就肝硬化胃静脉曲张内镜下组织黏合剂注射治疗的并发症及防治策略作一综述.

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关键词: 胃静脉曲张; 内镜下组织黏合剂注射; 肝硬化; 并发症

核心提要: 内镜下组织黏合剂注射在肝硬化胃静脉曲张出血的一级预防、控制急性出血和二级预防起着至关重要的作用。本综述系统介绍了该技术的并发症, 包括操作器械相关并发症、异位栓塞、排胶出血、感染、其他并发症, 并概述了其防治策略。

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0 引言

肝硬化临床工作中是引起门静脉高压最常见的原因, 胃静脉曲张破裂出血(gastric variceal bleeding, GVB)是肝硬化门静脉高压的常见并发症, 也是消化内科临床常见急症, 病死率高。内镜下组织黏合剂注射在GVB的一级预防、控制急性出血和二级预防起着至关重要的作用。然而该操作有一定难度, 存在一定的并发症风险, 部分并发症比如异位栓塞, 严重时可危及生命, 因此其防治非常重要。

1 胃静脉曲张概述

门静脉高压症(portal hypertension, PH)是指各种原因所致的门静脉系统压力升高所引起的一组临床综合征, 其中90%以上由各种原因所致的肝硬化引起^[1,2]。PH的基本病理生理特征为门静脉系统血流受阻和/或血流量增加, 门静脉及其属支内静力压升高并伴侧支循环形成, 可引起腹水、食管胃静脉曲张及出血、肝性脑病等。胃静脉曲张(gastric varices, GV)见于17%-25%的肝硬化门脉高压患者, 其3年出血率为16%-45%, 一旦出血其死亡率较EVB高^[3]。目前GV常用分型国外为Sarin分型^[4], 我国为LDRf分型^[5]。Sarin将GV分为GOV1[食管静脉曲张(esophageal varices, EV)沿胃小弯侧延伸]、GOV2型(EV沿胃大弯侧延伸)、IGV1(孤立胃静脉曲张位于胃底)和IGV2(孤立胃静脉曲张位于胃体或胃窦)。LDRf是具体描述静脉曲张在消化道内所在位置(location, L)、直径(diameter, D)与危险因素(risk factor, Rf)的分型记录方法, 统一表示方法为: LXx D0.3-5 R0,1,2, 具体描述方法参考我国相应指南^[5]。2021年美国胃肠病学会提出了一种新的GV分类方法, 将GOV1定义为小弯GV, GOV2和IGV1定义为贲门胃底GV, GOV2定义为远端GV, 并根据有无胃肾分流分为

不同亚型^[6]。不同类型GV的发生率和出血概率有所区别(表1)。

2 组织黏合剂在胃静脉曲张治疗中的作用

2.1 组织黏合剂概述 广义的组织黏合剂包括了氰基丙烯酸酯、凝血酶和纤维蛋白胶, 虽然后面两种组织黏合剂在欧洲指南中有提及且有部分应用于GVB治疗的研究结果发表^[7-10], 我国指南对其并未作出推荐, 故本综述中组织黏合剂主要是指氰基丙烯酸酯。氰基丙烯酸酯的作用机理为: 在血液、组织液中阴离子作用下, 快速聚合, 固化成膜, 产生一层具有高抗拉强度的弹性薄膜, 胶膜的网状结构可阻止血细胞、血小板通过, 封闭创面断裂的小血管网从而达到有效封闭止血的目的。目前常用的氰基丙烯酸酯包括正丁酯、正辛酯。目前尚缺乏关于各种类型组织黏合剂疗效对比的高质量研究, 我国目前市场上有的商标类型主要包括康派特(α -氰基丙烯酸正丁酯)、Glubrans胶(α -氰基丙烯酸正丁酯+甲基丙烯酸磺酸钠)、贝朗(曲妥克, α -氰基丙烯酸正丁酯)、福爱乐(α -氰基丙烯酸正丁酯+正辛酯混合物)、白云(α -氰基丙烯酸正丁酯)。

2.2 组织黏合剂在胃静脉曲张中的作用 组织黏合剂最早于20世纪80年代应用于急性GVB的治疗^[11], 我国于90年代最早由解放军总医院报道^[12]。研究显示内镜下组织黏合剂注射在GVB的一级预防、控制急性出血和二级预防方面均发挥重要作用。在GVB的一级预防方面, 一项随机对照临床研究(randomized controlled trials, RCT)结果显示: 与非选择性 β 受体阻滞剂(non-selective receptor blocker, NSBB)相比, 组织黏合剂注射可降低首次出血概率^[13]。组织黏合剂治疗GVB的即时止血率为86.8%-100%, 再出血率为7%-28%^[14-19]。一项纳入3项RCT研究的Meta分析结果显示: 内镜下组织黏合剂治疗GVB的即时止血率高于内镜下套扎术(endoscopic variceal ligation, EVL)(93.9% vs 79.5%), GOV2型再出血率二者相当(35.7% vs 34.8%), 而GOV1型和IGV1型组织黏合剂治疗后再出血率低于EVL(26.1% vs 47.7%, 17.6% vs 85.7%)^[20]。在GVB二级预防方面, 一项RCT结果显示, 与NSBB相比, 组织黏合剂注射可减少再出血率(15% vs 55%)及死亡率(3% vs 25%), 并发现不同预防措施、伴门脉高压性胃病和曲张静脉直径 ≥ 2 cm是再出血的高危因素^[21]。一项纳入9项RCT的Meta分析结果显示: 与NSBB相比, 组织黏合剂注射可减少GVB二级预防患者的再出血率及死亡率^[22]。目前国内外各大指南均对内镜下组织黏合剂注射在GV中的应用做了明确推荐(表2)。

3 组织黏合剂治疗胃静脉曲张的并发症及防治策略

3.1 操作器械相关并发症 操作器械相关并发症在临床

表 1 不同Sarin分型肝硬化门脉高压症GV的比率及出血概率^[4]

Sarin分型	描述	比率	未治疗者出血概率
GOV1	EV沿胃小弯延续	70%	28%
GOV2	EV沿胃大弯延续	21%	55%
IGV1	GV位于胃底	7%	78%
IGV2	GV位于胃体、胃窦	2%	9%

GOV1: 食管胃静脉曲张1型; GOV2: 食管胃静脉曲张2型; IGV1: 孤立性胃静脉曲张1型; IGV2: 孤立性胃静脉曲张2型; GV: 胃静脉曲张; EV: 食管静脉曲张。

表 2 目前国内外代表性指南对GVB一级预防、控制急性出血和二级预防的建议

指南	一级预防	控制急性出血	二级预防
中国指南 ^[5]	Leg型: NSBB/EVL; Lg型: NSBB/组织黏合剂	Lg型: 组织黏合剂、外科手术、TIPS均有效; Leg型: ①食管EIS; ②胃组织黏合剂注射; ③联合序贯治疗(先对胃进行组织黏合剂注射, 择期或同时对食管EVL或EIS)	Leg型: NSBB、EVL、EIS或NSBB与EVL或EIS联合; Lg型: 组织黏合剂、TIPS、外科手术
Baveno VII ^[2]	高风险GV: NSBB; GOV2和IGV1: 可考虑使用组织黏合剂	GOV1: EVL、组织黏合剂; GOV2和IGV: NSBB/卡维地洛和/或EVL; TIPS可作为组织黏合剂; Child-Pugh C级评分<14或Child-Pugh B级评分>7内镜治疗失败或出血时HVPG>20: TIPS; 有分流道	GOV2、IGV1: 可考虑BRTO
AASLD ^[23]	GOV1: EVL; GOV2和IGV1: NSBB	GOV1: EVL或组织胶注射; GOV2和IGV1: TIPS、组织胶	GOV1: NSBB+EVL; GOV2和IGV1: TIPS/BRTO, 亦可考虑组织黏合剂
EASL ^[24]	GOV1: NSBB/EVL; GOV2和IGV1: NSBB	GOV1: EVL; GOV2和IGV1: 组织黏合剂; 伴SPSS的GOV2和IGV1: BRTO/BATO	GOV1: NSBB+EVL; GOV2和IGV1: TIPS
AGA ^[6]	/	组织黏合剂、TIPS; 有胃肾分流: BRTO	/

EIS: 内镜下硬化治疗; EVL: 内镜下套扎术; TIPS: 经颈静脉肝内门体分流术; BRTO: 球囊导管逆行性静脉栓塞术; BATO: 球囊导管顺行性静脉栓塞术; NSBB: 非选择性β受体阻滞剂; GVB: 胃静脉曲张出血; GV: 胃静脉曲张; HVPG: 肝静脉压力梯度; AASLD: 美国肝病学会; EASL: 欧洲肝病学会; AGA: 美国胃肠病学会; GOV: 食管胃静脉曲张; IGV: 孤立性胃静脉曲张。

工作中偶尔可遇见, 其发生与医师的操作水平及是否规范有关, 包括粘滞/堵塞并发症(与组织黏合剂特性有关)及穿刺点出血。一篇纳入628例次内镜下组织黏合剂注射的文献报道显示^[25], 9例(1.43%)发生注射针粘滞在曲张静脉内、17例(2.71%)发生鞘管堵塞、1例(0.159%)发生内镜粘滞致取出困难, 2例(0.318%)在后续套扎治疗时出现套扎器粘滞。其中注射针粘滞及鞘管堵塞多与组织黏合剂用量过大、使用未稀释的组织黏合剂原液、注射过程中注射针或鞘管未及时回退、针鞘刺入曲张静脉内等有关, 因此合理控制组织黏合剂用量、针及针鞘及时回退、使用三明治法注射组织黏合剂等可减少此类并发症发生率。内镜粘滞多见于GOV1或IGV1型曲张静脉治疗时, 与组织黏合剂用量过大、内镜对曲张静脉压迫有关, 因此合理控制组织黏合剂剂量、使用角度好的治疗内镜可减少其发生率。为减少套扎器粘滞的发生率, 我们要合理控制组织黏合剂用量, 在食管下段及贲门附

近行套扎前先观察曲张静脉颜色及硬度, 判断是否已被组织黏合剂充填。

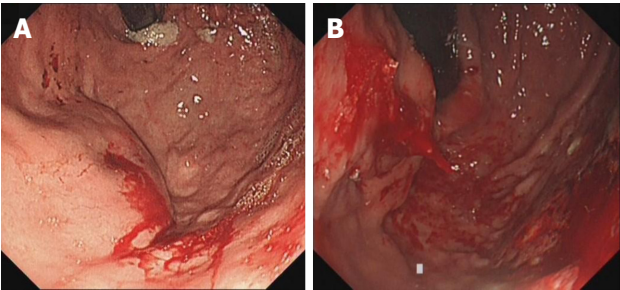
穿刺点出血(图1)主要与反复穿刺、提前穿刺及组织黏合剂用量不足有关, 因此在治疗过程中我们建议穿刺见回血后注射, 但不强求回血, 尤其是对于直径较小的曲张静脉, 因为刺入静脉后有明显落空感, 而且注射过程中曲张静脉隆起形态也可判断是否为曲张静脉内注射。如判断曲张静脉需要治疗, 一定要等到组织黏合剂等准备好再行穿刺。如为用量不足所致, 可先使用针鞘堵住出血点, 待药物准备完毕后补充治疗。

3.2 异位栓塞 异位栓塞是组织黏合剂治疗后最严重的并发症, 常见的为肺栓塞、脑栓塞和脾梗死^[18,26-40]。虽然术后影像学检查发现较大比例患者肺内可出现少量碘化油, 但绝大多数无症状, 真正有症状、需要抗凝治疗的患者极少。目前文献报道异位栓塞发生率为0%-10.71%, 截至目前报道的样本例数在60例以上的存在异

表 3 目前较大样本量文献报道的异位栓塞并发症发生情况

研究者	发表年	病例数	组织胶用法	异位栓塞发生率	异位栓塞分布情况	栓塞结局
Zhou ^[26]	2021	439	硬化剂+组织胶+硬化剂	3.19%(14/439)	1肺栓塞、1脑栓塞、12门静脉栓塞	肺栓塞患者死亡, 余存活
Tseng ^[27]	2018	652	硬化剂+组织胶+硬化剂	0.77%(5/652)	1肺栓塞、1脾栓塞、2脑栓塞、1肠系膜上动脉栓塞	肺栓塞及肠系膜上动脉栓塞患者死亡, 余存活
Al-Bawardy ^[28]	2016	95	盐水+组织胶+盐水	2.1%(2/95)	2肺栓塞	1例死亡, 1例存活
Bhat ^[29]	2016	152	EUS引导弹环圈+组织胶	0.66%(1/152)	1肺栓塞	存活
Jun ^[30]	2014	455	碘油+组织胶	0.22%(1/455)	NA	存活
Prachayakul ^[31]	2013	90	碘油+组织胶	4.44%(4/90)	NA	存活
黄湘俊 ^[32]	2012	150	碘油+组织胶	10.71%	7肺栓塞、5脑栓塞、2门静脉栓塞、1脾栓塞	存活
Mosca ^[33]	2012	65	碘油+组织胶	1.54%(1/65)	1脾栓塞	存活
Kang ^[34]	2011	127	碘油+组织胶	1.57%(2/127)	1肺栓塞、1脾栓塞	存活
Sato ^[35]	2010	129	碘油+组织胶	2.33%(3/129)	1肺栓塞、2脾栓塞	存活
Cheng ^[36]	2010	753	碘油+组织胶	0.66%(5/753)	1肺栓塞、1脑栓塞、3脾栓塞	存活
Paik ^[18]	2008	121	碘油+组织胶	1.65%(2/121)	1脑栓塞、1脾栓塞	存活
Caldwell ^[37]	2007	92	碘油+组织胶	2.17%(2/92)	1脑栓塞、1冠脉栓塞(心脏骤停死亡)	冠脉栓塞患者死亡, 脑栓塞患者存活
Joo ^[38]	2007	85	碘油+组织胶	2.35%(2/85)	1肺栓塞、1脾栓塞	存活
Mostafa ^[39]	1997	80	碘油+组织胶	1.25%(1/80)	1肺栓塞	死亡
D'Imperio ^[40]	1996	80	碘油+组织胶	2.50%(2/80)	2肺栓塞、1脾栓塞	存活

NA: 文中未具体交代异位栓塞类型. EUS: 超声内镜.



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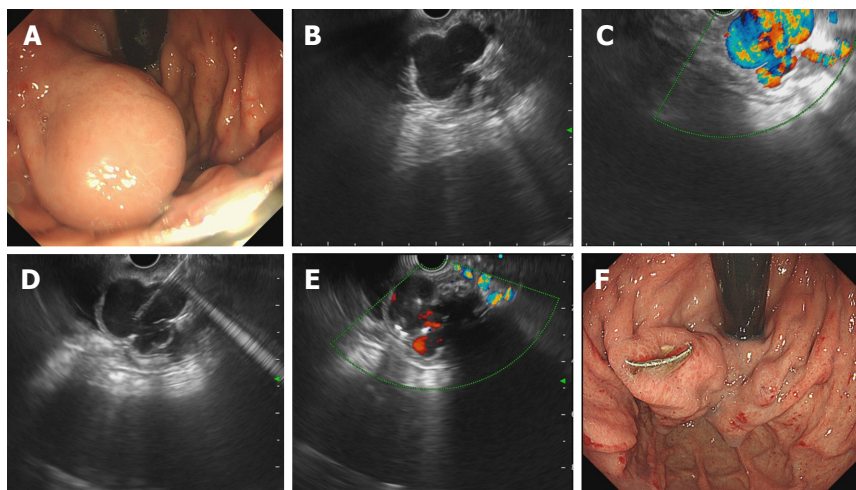
图 1 穿刺点出血. A: 组织黏合剂注射治疗后穿刺点少量渗血(可自行停止, 多无需追加治疗); B: 组织黏合剂注射治疗穿刺点出血量较大(常因组织黏合剂用量不足所致, 需要补充注射).

位栓塞并发症的文献总结见表3. 此外, 其他少见的异位栓塞包括肾静脉^[41]、肾上腺静脉^[42]、冠状静脉^[43,44]、横膈栓塞^[45]等.

发生异位栓塞的危险因素目前尚未完全明确, 目前认为其可能的危险因素包括门体分流道的存在、使用碘油做预充剂、组织黏合剂用量过大、推注速度过快等^[32,36,46,47]. 因此建议内镜下注射组织黏合剂时使用三明治法, 使用盐水、高渗糖或聚桂醇作为预充剂, 同时控制推注速度. 对于非急性出血患者, 建议完善门静脉CT成像明确有无门体分流道的存在^[48,49], 如存在脾肾或胃肾分流, 建议使用球囊闭塞下经静脉逆行栓塞术

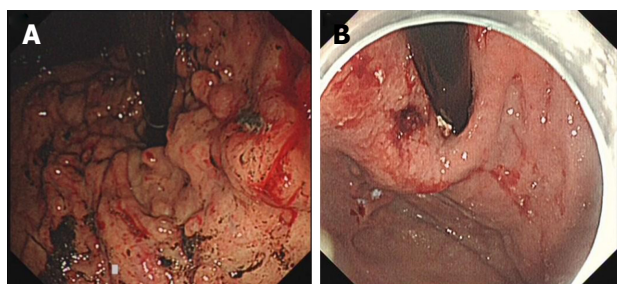
(balloon-occluded retrograde transvenous obliteration, BRTO)或超声内镜(endoscopic ultrasonography, EUS)引导下弹环圈+组织黏合剂注射(图2), 或使用金属夹^[2,6,24,50-52], 目前Baveno VII^[2]、美国胃肠病学会^[6]和E欧洲肝病学会^[24]均推荐BRTO用于伴脾肾或胃肾分流GV患者的治疗. 研究显示BRTO治疗GVB的再出血率低于组织黏合剂治疗^[53,54]. 我国华西医院的一项随机对照临床研究结果显示, 在GOV2和IGV1型的二级预防方面: 与内镜下组织黏合剂注射相比, BRTO可降低再出血率(33.4% vs 15.6%), 二者并发症发生率和死亡率相当^[55].

3.3 排胶出血 内镜下组织黏合剂注射治疗后, 大部分患



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图 2 EUS引导下弹环圈+组织黏合剂治疗. A: 胃底见巨大曲张静脉团; B: EUS下曲张静脉图; C: EUS下曲张静脉团内见丰富血彩; D: EUS引导下弹环圈+组织黏合剂治疗; E: 治疗后曲张静脉内血彩基本消失; F: 5 mo后复查, 见弹环圈排出, 曲张静脉塌陷. EUS: 超声内镜.



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图 3 排胶溃疡出血. A: 胃底静脉曲张内镜下组织黏合剂治疗后, 可见多个排胶面出血; B: 胃底小弯侧近后壁排胶出血.

者在1 mo-3 mo内开始排胶, 平均时间为术后23天, 并在6 mo-12 mo内基本排完, 但亦有少数患者排胶过程长达1-2年. 排胶出血的发生率约为3.1%-14.2%^[36,56-58], 发生排胶出血(图3)危险因素尚未完全明确, 研究显示Child-Pugh评分越高、组织黏合剂剂量越大(GV直径越粗)、组织胶用量不足者发生排胶出血的风险越高^[36,56-58]. 此外, 组织黏合剂误注射入黏膜下层而非曲张静脉内, 术后发生排胶出血的概率也会增加. 因此, 合理选择治疗对象、结合术CT等影像学技术评估曲张静脉容积, 个体化设计组织黏合剂用量, 术中仔细确认曲张静脉是否固化, 可能有助于降低排胶出血的发生率. EUS可显示实时曲张静脉固化情况, 对于判别排胶出血的原因有一定价值^[59]. 如为排胶溃疡出血, 可使用质子泵抑制剂及黏膜保护剂进行治疗; 如为残留静脉曲张出血, 则可追加组织黏合剂注射治疗. 研究显示, 与组织黏合剂相比, TIPS治疗可减低GV治疗后再出血率, 然而TIPS发生肝性脑病的概率增加^[60]. 对于急性GVB患者, 组织黏合剂治疗比TIPS的经济效益比更优^[61].

3.4 感染 虽然高达31.9%患者组织黏合剂治疗后可出现菌

血症, 但多为一过性^[62], 真正引起症状、需要治疗的感染发生率仅为1%左右, 且常见于急性出血期患者^[36,62-64]. 报道的感染并发症包括败血症、纵隔炎症、腹腔炎症甚至脓肿^[65]、肾脓肿^[66]、肾上腺脓肿^[42]、吸入性肺炎等^[36,62-64]. 研究显示, 感染致病菌多为口腔或消化道定植菌, 与注射针或内镜腔道培养的菌种高度同源^[62,63], 现在随着内镜清洗消毒的进一步规范, 此类感染已明显减少. 研究显示注射次数、注射总量、输血量、Child-Pugh评分是发生菌血症的危险因素^[62]. 急性食管胃静脉曲张破裂出血患者各大指南均建议急性出血患者预防性使用抗生素, 疗程为3 d-7 d^[2,5,6,10,24], 如感染仍持续存在, 可适当延长抗生素应用时间. 不过值得一提的是, 组织黏合剂注射治疗后出现的发热并不一定提示感染, 大部分为吸收热, 体温多<38℃, 可自行缓解, 应加以鉴别. 对于使用组织黏合剂行一级、二级预防患者是否预防性使用抗生素, 目前仍有争议^[63,67], 建议结合患者情况综合评估感染风险后酌情应用.

3.5 其他并发症 其他报道的并发症包括腹部不适、胸骨后不适或烧灼感、腹痛、非感染性低热、白细胞升高、

呃逆或呕吐, 最高达60.4%, 这些多为一过性, 无需特殊处理, 可自行恢复正常, 症状严重者可予对症处理^[32,34,47]。其他少见的并发症包括胃黏膜坏死, 该并发症多见于序贯治疗的患者, 可能是由于组织黏合剂误注射入动脉或局部存在动静脉瘘所致^[36]。此外还有2例并发急性心包炎的病例报道, 主要表现为术后胸痛明显, 严重时危及生命, 可能是组织黏合剂从胃静脉曲张通过交通静脉转至膈、纵隔、心包交通静脉^[44,68]。

4 结论

GVB是肝硬化门静脉高压的常见并发症之一, 是消化内科常见急症之一。内镜下组织黏合剂注射在GVB的一级预防、控制急性出血和二级预防中起到重要作用, 疗效确切, 即时止血率高。该治疗的并发症发生率相比较低, 且大部分经内科保守治疗可获得缓解, 然而仍有极少数患者发生严重并发症, 如异位栓塞、急性心包炎等, 因此要引起足够重视。并发症的发生率与患者病情(如GV直径、门体分流、Child-Pugh评分等)及医生的操作水平有关, 因此准确的术前评估及规范化操作时减少并发症的前提。近年来随着一些新型治疗技术的兴起, 如EUS引导下静脉曲张治疗、BRTO等, 这些方法是否有助于减少并发症的发生率仍有待于进一步研究。此外, 三明治法使用不同预充剂的疗效与并发症的对比, 其他类型组织黏合剂如凝血酶、止血粉、纤维蛋白胶与组织黏合剂疗效及安全性的对比等均需要进一步研究证实。

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