·继续教育·

TIPS 再狭窄的研究现状和进展

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经颈静脉肝内门腔分流术（Transjugular Intrahepatic Portosystemic Shunt，TIPS）是治疗门静脉高压症的介入技术。该技术由 Rosch 于 1969 年首先报道[1]，1989 年 Richter 首次用于临床[2]，经过 30 多年的发展，TIPS 技术已成熟，被广泛地应用于食管胃底静脉曲张出血、顽固性腹水、Budd-Chiari 综合征等门静脉高压症的治疗以及肝移植术前等待供体期间防止致命并发症等，并取得了显著的疗效[3-12]。

但 TIPS 术后支架及易发生再狭窄或闭塞，致使门脉高压复发。据统计其术后 1、2 和 5 年支架再狭窄率分别高达 5% ~ 64%、33% ~ 70% 和 60% ~ 85%[13-18]，严重影响 TIPS 的中远期疗效。尽管目前有一系列处置 TIPS 再狭窄的治疗方法，包括溶栓、再次球囊扩张、再放置支架等，有的也有较好的疗效。但显而易见，这不是解决再狭窄问题的根本方法，只有阐明 TIPS 再狭窄的形成机制，才有可能更好地解决这一问题。

与血管支架术比较，TIPS 术的独特性在于在肝实质内门静脉和肝静脉之间开辟了一条非血管的通道，并用支架维持其通畅。因此，研究 TIPS 再狭窄的病理基础和形成机制具有重要的意义。本文将就 TIPS 再狭窄研究的有关现状及进展作一综述。

一、TIPS 再狭窄的病理基础

TIPS 再狭窄的病理基础根据时间分为两类：一是早期支架内狭窄闭塞，主要是血栓性狭窄，为 TIPS 术后 2 周内的主要表现，术后 1 周内主要表现为支架内血栓形成，血小板（白血栓）聚集，纤维素沉积和红细胞吸附。这一现象与血管内置支架后的表现相仿。但是，目前尚无法确认血栓中血小板、纤维素和红细胞究竟哪一种与再狭窄最为相关？二是中远期狭窄，在人体标本研究中主要是假性内膜的增生所致，而假性内膜是由胶原基质表面覆盖一层内皮细胞（EC）所组成，胶原基质内含增殖的平滑肌细胞（SMC）[19-23]，在 TIPS 猪模型的研究中似乎 SMC 增殖的时间更早，增殖的量更大[24-29]。

早期分流道狭窄是指术后 1 个月内发生的狭窄闭塞，在临床上主要与内支架位置不当及术后抗凝不够有关。支架置放时定位不准确，术后移位，支架长度不够而未能深入门静脉及肝静脉而覆盖全部分流道是造成早期狭窄闭塞的主要原因。还有支架张力不够出现塌陷或支架未用球囊导管充分扩张，支架在肝实质内成角过大也易造成狭窄。还有人认为 TIPS 术中肝脏穿刺对肝组织损害严重或球囊扩张压力过大对周围肝组织造成较大损伤，会导致局部急性炎症反应，引起狭窄闭塞的发生[30]。

中远期再狭窄的原因和机制是 TIPS 再狭窄研究的中点。LaBerge 等[19,20] 较早对分流通道内组织进行研究，发现 TIPS 术后分流道内表面凹凸不平，肝实质自支架网格间深入，很快有一些小的血栓及纤维蛋白附着于分流道及支架的金属丝表面，并填充于凹陷部分。术后 4 d 分流道内表面即可有斑片状的内皮细胞出现，约 3 周后形成光滑而基本完整的内皮细胞层覆盖分流道内表面，与其下方的肉芽组织共同形成假性内膜。如果假性内膜厚度不超过 1 mm，这样的分流道能保持长期通畅，而不狭窄或闭塞。各处因素刺激使得假性内膜组织过度增生，均会导致分流通道的狭窄及闭塞。

对于假性内膜的成分，不同的研究者有着不同的描述。LaBerge 等[19,20] 认为表面内皮细胞层下方主要是肉芽组织，含少量炎症细胞及大量基质及胶原束。Ducoin 等[23] 对 17 例术后死亡或肝移植取得的 TIPS 分流通道组织病理分析发现：术后 2 周内仅可见到不规则纤维蛋白、血小板凝块，炎性细胞和红细胞附着在支架网孔间；随着胶原纤维越来越密集，其内所含的细胞成分也越多，主要是成纤维细胞（fibroblast）及成肌纤维细胞（myofibroblast）；支架内假性内膜在术后 2 周后方可确定，假性内膜含大量的胶原纤维及平滑肌细胞，在纤维层表面可见一层连续的成熟内皮细胞。Sanyal 等[22] 综合了多组研究，认为 TIPS 假性内膜是由一层内皮细胞和内皮下胶原基质组成，而胶原基质内含大量间充质细胞（mesenchymal cell），这种间充质细胞就是平滑肌细

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胞的原型。组织胚胎学中,间充质细胞是一种分化程度很低的细胞,在胚胎发育过程中能分化成各种结缔组织细胞、内皮细胞和平滑肌细胞;胚胎时期的中胚层间充质就是由间充质细胞和基质组成。Ducoin 等[23]也提到,TIPS 分流通道与血管成形术后狭窄不同,无中层平滑肌细胞可以迁移到内膜。

随着对 TIPS 假性内膜认识的逐渐加深,人们意识到假性内膜不总是具有负面的影响,其形成有着重要的意义。假性内膜可保护肝实质直接暴露于血液成分,尤其是血小板,避免血栓形成;也保证金属支架不受血流及其他因素的干扰,同时还保护血液红细胞免受各种刺激因素的破坏[22]。要保持 TIPS 术后支架的通畅,首先要避免形成早期血栓性狭窄闭塞的因素,而支架的中远期通畅率有赖于假性内膜的形成。

TIPS 分流道内表面内皮细胞层的形成即支架内腔的内皮化可能阻止平滑肌细胞增生,从而保持支架内的通畅。这与血管内支架植入术后再狭窄的病理特点是类似的。在 PTA 术中,内皮细胞的损伤、修复及功能改变在再狭窄中扮演重要角色。PTA 可造成血管损伤,内皮剥脱,血小板聚集并血栓形成。血小板释放的血小板衍生生长因子(PDGF)可能与中膜平滑肌细胞的激活和迁移有关[31]。内皮细胞可调节平滑肌细胞的增生和基质分泌,势必影响血管重构(remodeling),血液因素通过内皮依赖性血管收缩和舒张也影响血管的重构。从对血管支架再狭窄的认识及 TIPS 相关研究的结果可以引伸:TIPS 术后支架内内皮细胞也可能分泌多种因子调节平滑肌细胞的生长,从而调节细胞外胶原基质的合成,阻止假性内膜的过度增生;完整连续的内皮细胞层可以阻止血栓形成,避免由于血栓因素所造成的一系列后果[31-36]。

但是,最近的一些新发现提出了新的问题和思考,首先是内皮细胞的来源问题。Ducion 等[23]提出,有 3 种可能来源:来源于假性内膜成肌纤维细胞的分化;来源于邻近门静脉或肝静脉血管内皮细胞的迁移;来源于肝窦。Sanyal 等[21,22,37]的实验结果表明,TIPS 通道内的内皮细胞来源于肝窦内皮细胞,其特性与血管内皮细胞不同:虽然都表达Ⅷ因子相关抗原,但血管内皮细胞胞内可见 Weibel-Palade 小体,CD31、CD34 表达呈强阳性,CD14 表达阴性;肝窦及 TIPS 通道内的内皮细胞胞内无 Weibel-Palade 小体,CD31、CD34 表达呈弱阳性或阴性,CD14 表达阳性。根据他们的实验,TIPS 内皮细胞的某些功能特征显然也应与血管内皮细胞不同。如果这个结果和结论是可靠的,那么对于我们进一步研究 TIPS 通道再狭窄的发生机制有着重大的意义。

TIPS 术在肝脏中开通的是无内皮细胞的血管性通道。而肝脏对其反应类似于内皮细胞剥脱后的血管反应,通道内内皮细胞和 SMC 原型间充质细胞发生作用,分泌胶原基质。当然,TIPS 假性内膜中内皮细胞影响着 SMC 的活动。Sanyal 等[37]又进一步显示了其中的关系:通过分离 TIPS 假性内膜内皮细胞及其下方的平滑肌细胞进行离体培养,观察内皮细胞对平滑肌细胞的影响。结果发现:TIPS 假性内膜内皮细胞明显刺激平滑肌细胞的增殖和移动。因而认为内皮细胞促进了 SMC 的迁移和增殖,从而促进假性内膜形成。这一发现与目前普遍认知的内皮细胞抑制平滑肌细胞增殖和迁移的观点完全相反。Sanyal 等[37]还提出,内皮细胞可能有 2 种方式影响 SMC:内皮细胞分泌细胞因子影响 SMC 的增殖和迁移;内皮细胞与平滑肌细胞间的直接作用。目前的研究仅证实了前者,后者则只是假设。他们还发现 TIPS 术后平滑肌细胞的细胞指纹(fingerprint)与来源于其他脏器的对照组平滑肌细胞有不同,并推测这 2 种平滑肌细胞的特征与功能可能也不同。

二、胆管损伤并胆汁漏出对 TIPS 再狭窄的影响

在 TIPS 术中,胆道损伤并胆汁漏出似乎难以避免。LaBerge 等[19]于 1991 年首先报道了 TIPS 术后标本检查上发现了部分闭塞的支架内胆汁漏出染色现象,并推断胆道横断损伤并胆汁漏出是引起支架再狭窄的原因之一,进而分析胆汁可能有刺激平滑肌细胞增殖的作用。Stout 等[38]也曾报告一例患者在 TIPS 术后6.5 个月死亡尸检时见分流通道狭窄处有胆管上皮细胞增生并形成囊肿样改变。其他学者在用猪模型的动物实验中证实这一发现[24-26,39],并认为这一现象与 TIPS 术后再狭窄相关。

支架植入以后的再狭窄并非 TIPS 术所特有,然而,TIPS 术后的再狭窄发生率之高和发展之迅速似乎远在其他部位支架植入如血管支架术之上。支架植入术中胆道损伤并胆汁漏出为 TIPS 术不同于其他支架术最为显著的独特因素。因此,根据上述临床标本和动物实验所见,结合胆汁的某些生理作用如胆汁的促血栓形成作用[40],有理由推测,TIPS 术中胆汁漏出可能促进 TIPS 再狭窄,即胆汁可能刺激 SMC 增生。这一推测似乎解释了为何 TIPS 术后的再狭窄发生率之高和发展之迅速。

为了进一步探讨胆计与 TIPS 支架再狭窄的关

系,我们进行了系列的研究[29,41-45]:胆汁干预平滑肌细胞及内皮细胞离体培养,并通过 100 余只 TIPS 猪模型的组织病理学研究,证实了胆汁漏出并染色现象与支架内有新生胆道增生。SMC 的体外胆汁干预培养结果发现,高浓度的胆汁使 SMC 死亡,1.0%胆汁不仅无刺激 SMC 的增殖的作用,反而是抑制其生长的作用。这一结果恰与 LaBerge 等推测相反。进一步的动物模型实验解释了这一惊奇的发现:胆汁漏出组的 TIPS 再狭窄率明显高于无胆汁漏出组,但前者支架腔内增生组织以血栓为主,而不是 SMC。因此,该系列研究得出了 TIPS 术中胆汁漏出抑制 SMC 增生,促进血栓形成,从而使支架再狭窄的结论。那么,TIPS 术中胆汁漏出是如何促进 TIPS 支架血栓形成?用胆汁干预分离的脐静脉 EC 进行体外培养的结果发现:胆汁对血管内皮细胞具有抑制作用,其抑制作用是与浓度有关:低浓度的胆汁对内皮细胞无明显的抑制作用,但随胆汁浓度的逐渐增大,对内皮细胞的抑制作用也越来越显著,至中高浓度,可完全抑制内皮细胞的生长;同时胆汁抑制血管内皮细胞 NOS 活性,抑制内皮细胞分泌 vWF。因此,最终得出结论:胆汁是引起 TIPS 再狭窄的重要因素,但其作用并非由于胆汁刺激 SMC 增殖所致,而是通过抑制支架内皮化而导致再狭窄。结合这系列的研究结果,提出了以胆汁漏出为中心的 TIPS 支架再狭窄形成机制:大量的胆汁漏出刺激强烈的炎症反应,并抑制内皮细胞生长及功能,促进血栓形成而造成 TIPS 早期狭窄闭塞;少量的胆汁漏出引起较轻的炎症反应,而导致假性内膜过度增生,从而导致 TIPS 的中远期狭窄。

三、阻断胆汁漏入分流道,预防 TIPS 支架再狭窄

多年来,对 TIPS 再狭窄预防并未取得突破性进展。覆膜支架(stent-graft)在 20 世纪 90 年代兴起,其原理是想利用在血管外科被广泛使用的 PTFE 等高分子聚合材料使金属支架与宿主有更好的组织相容性,从而减少再狭窄。覆膜支架也被应用于 TIPS 术中。动物实验及小样本临床应用的结果表明:覆膜支架有降低 TIPS 再狭窄率的作用[24,46-48]。但是,最终未能被广泛接受和应用。

随着胆汁漏出可能导致 TIPS 支架再狭窄理论的确立,终于诞生了以完全阻断胆汁漏入 TIPS 分流道的新型覆膜支架:Viatorr 覆膜支架。Viatorr 支架由经特殊降解处理的 3 层不同孔径 PTFE 膜组成,可以完全阻断胆汁向支架内漏。目前,已在欧洲应用,美国也正在进行前瞻性、多中心研究。包括一组多中心临床研究在内的多组临床研究证明:用 Viatorr 覆膜支架的 TIPS 的 1 年的初次通畅率(primary patency)达 80% ~ 84%,经过介入处理的再次通常率(secondary patency)几乎达 100%[49-55]。Saxon 惊呼[56] 新型的 TIPS 覆膜支架的广泛应用将预示着 TIPS 的新纪元即将来临。

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第三届中国东部介入放射学学术会议

征文通知

由中华医学会实用介入技术培训推广中心(济南)、《介入放射学杂志》编辑部共同主办的第三届中国东部介入放射学学术会议将于 2005 年 6 月在山东省烟台市举行。届时会议将邀请国内著名介入放射学专家进行专题讲座。此次会议内容包括医学继续教育讲座、学术交流、影像设备与器械展示等内容,欢迎广大医务和科研工作者撰写论文并参加会议,与会代表可获得国家级Ⅰ类学分。

会议征文:

1、论文全文包括中文摘要和正文,3000 字以内。摘要应包括目的、材料与方法、结果、结论四部分,600 字以内。

2、论文需标明篇名、作者姓名、工作单位、详细地址、邮编、电话。稿件须附单位介绍信或加盖公章。

3、稿件要求必须电脑打印(WORD 格式),附软盘及打印稿各一份。请自留底稿,恕不退稿。

征文内容:

1、介入治疗的基础和临床研究及经验总结;

2、介入治疗的新技术、新方法及新材料、新器械的应用;

3、介入放射学规范化、标准化;

4、介入放射学病房管理和临床护理;

5、介入放射学技术;

截稿日期:2005 年 3 月 30 日。会议具体时间、地点请见第二轮通知。

来稿请寄 济南市经五路 324 号 山东省医学影像学研究所

介入放射学研究室 孙增涛 陈颉收 邮编:250021

中华医学会实用介入技术培训推广中心(济南)

《介入放射学杂志》编辑部

TIPS再狭窄的研究现状和进展

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