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急性肠系膜缺血诊断及治疗研究进展

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摘 要

急性肠系膜缺血是肠系膜动脉血流中断引起肠坏死的急腹症，可导致多器官衰竭、甚至死亡。发病率逐年升高，死亡率高，早期诊断能最大限度降低死亡率。因此，该文综述了急性肠系膜缺血的类型、病理生理和诊断、治疗方面的最新进展，以期为临床诊疗提供新的思路。

关键词

急性肠系膜缺血，肠坏死，临床表现，生物标志物，血管内治疗

Advances in the Diagnosis and Treatment of Acute Mesenteric Ischemia

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Abstract

Acute mesenteric ischemia is an acute abdominal condition in which interruption of mesenteric artery blood flow causes intestinal necrosis, which can lead to multi-organ failure and even death. The morbidity is increasing year by year and the mortality rate is high, and early diagnosis can minimize the mortality rate. Therefore, this article reviews the types of acute mesenteric ischemia, pathophysiology, and the latest progress in diagnosis and treatment, in order to provide new ideas for clinical diagnosis and treatment.

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Keywords

Acute Mesenteric Ischemia, Intestinal Necrosis, Clinical Manifestations, Biomarkers, Endovascular Therapy

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1. 流行病学

一项纳入 163 项研究的 meta 分析显示：急性肠系膜缺血(Acute mesenteric ischemia, AMI)在全球的平均发病率大约为 6.2 (95% CI 1.9~12.9)/10 万人年。最常见的是动脉闭塞型，约占所有急性肠系膜缺血病例的 68.6% (95% CI 63.7~73.2)，血栓性和栓塞性动脉闭塞大致相同，分别占所有 AMI 病例的 30.0% (95% CI 24.4~36.3)和 33.3% (95% CI 27.3~39.9)。非闭塞性肠系膜缺血占 15.1% (95% CI 11.8~18.7)，肠系膜静脉血栓形成占 11.5% (95% CI 9.1~14.2) [1] [2]。随年龄增长，AMI 发病率呈指数上涨[3]，75 岁以上就诊急腹症患者中，AMI 比急性阑尾炎更加常见[4]。

2. 肠道血供及疾病分类

2.1. 肠道血供

肠道的血流供应主要依靠腹腔动脉、肠系膜上动脉和肠系膜下动脉来提供，彼此之间建立了丰富的吻合支，血管的变异性很大，有着强大的抵抗缺血能力[5]。当肠系膜血液供应减少 50%以上或平均动脉压力 ＜45 mmHg，才发生肠道缺血事件[6]，甚至持续 12 小时血流减少 75%，也不见得能造成严重损伤，但血流完全阻断 6 小时就会引起肠坏死可能[7]。

2.2. 疾病分类

急性肠系膜动脉栓塞多发生于肠系膜上动脉，可能肠系膜上动脉开口和腹腔动脉成锐角便于血栓通过，大多数栓子位于距肠系膜上动脉开口处远端 3~10 cm 处，因此近端空肠和结肠常受累较轻[8]。肠系膜上动脉的侧支循环相对不发达，对缺血事件抵抗能力较弱。由于栓子意外出现造成急性血管内梗阻，短期肠道发生严重缺血[7]，最终导致肠坏死。

肠系膜动脉血栓形成的病理基础是动脉粥样硬化，多位于肠系膜上动脉开口处[9]。长期餐后腹痛、畏惧饮食及体重减轻是临床典型“三联征”[7]。动脉硬化和餐后肠道血供需求增多是引起餐后腹痛的主要原因。急性发作时往往两支或三支血管已经严重受累，肠坏死程度、影响范围均较急性肠系膜动脉栓塞严重[10]，抗动脉粥样硬化治疗是预防本病发生的重要措施之一。

非闭塞性肠系膜缺血通常由于肠系膜上动脉血管强烈收缩与内脏血流减少造成[11]，血流未完全中断，多累及近端结肠。此类患者常患有严重的慢性疾病，如心力衰竭、脓毒症、休克、心脏停搏复苏成功后，应用血管收缩剂也可诱发本病[8]。

肠系膜静脉血栓形成可分为肠系膜上静脉血栓形成和肠系膜下静脉血栓形成，前者最为常见，肠系膜下静脉血栓形成，仅占肠系膜静脉血栓形成病例的不足 11% [12]-[14]。多波及回肠(64%~83%)、空肠(50%~81%)和十二指肠(4%~8%) [15]。

3. 病理生理

AMI 的病理生理进程具有时间依赖性。起初 6 小时内，血流中断伴血管痉挛。此时恢复血流灌注，损伤一般是可逆的。随后的 6~12 小时，缺血由肠道粘膜层蔓延到肠壁全层，并损伤到肠壁内疼痛感受器，所以疼痛感可减缓。发病超过 12 小时，肠道坏死多已不可逆，弥漫性腹膜炎、脓毒症和多器官衰竭等并发症相继出现，此期死亡率极高[7] [16]。

4. 临床表现

AMI 是危及生命的急腹症，延误诊断死亡率极高。最新研究表明，本病多见于 60 岁及以上患者，女性发病率约为男性 3 倍[7]。患者常突发剧烈腹痛，伴有恶心、呕吐和腹泻[17]，腹部柔软无压痛，肠鸣音活跃。当血流阻断 12 小时以上或出现反跳痛(Blumberg 征)、板状腹、肠鸣音减弱或消失、血性腹泻、发热和休克多提示肠梗死已不可逆[7]，此时往往需外科手术干预。

非闭塞性肠系膜缺血的患者常有严重的基础疾病，如：心脏停搏复苏成功后、脓毒症、休克、重大手术、严重创伤等[18]。临床表现缺乏特异性，当重症患者出现右侧腹痛、粪便带血或便出鲜血时需警惕本病。一项回顾性研究表示，肠系膜缺血是脓毒性休克的常见死亡原因之一[19]。实验和观察性研究表明，肾上腺素/去甲肾上腺素、血管加压素等常用抢救药物，可能导致肠道粘膜灌注受损[20]，从而诱发非闭塞性肠系膜缺血。

5. 诊断和辅助检查

5.1. 影像学检查

CT 血管成像对 AMI 灵敏度为 85%~98%，特异性为 91%~100%，是目前 AMI 一线诊断工具[8] [21] [22]。选择性肠系膜血管造影术不仅能明确诊断，还可进行血管内治疗，不过由于技术要求高、侵入性等缺点，很多时候只能作为二线检查[23]。磁共振血管成像在肠系膜血管检查中，具有无创、无造影剂的优势，现已是评估肠系膜动脉和静脉血管的成熟技术，但在早期，可能无特殊性影像学征象[24]。多普勒超声既往被认为价值较低[23]，这是一种误解。对正在接受镇静、镇痛治疗或意识障碍的重症患者，床旁超声检查能快速判断腹腔内是否存在腹腔积液及肠坏死情况，同时可在剖腹手术中协助判断肠道活力。新型双能 CT、人工 AI 技术的应用也能协助 AMI 的早期诊断[25]。

5.2. 生物标志物

I-FABP、D-二聚体、α-GST 是目前研究的热门[26] [27]。I-FABP 在血管性肠缺血诊断中的应用价值很高，被视作最有潜力的生物标志物[28]。当出现急腹症时，检测出高阴离子间隙乳酸酸中毒(88%) [29]、D-二聚体阳性[30]、血凝和白细胞计数升高(90%) [31]，可能高度提示 AMI [29]。一项研究表明，在 AMI 病例中，血清乳酸水平升高 > 2 mmol/l 与肠梗死风险比相关：4.1 (95% CI 1.4~11.5, $p < 0.01$) [32]。研究表示，D-二聚体是肠缺血的独立危险因素[33]，D-二聚体 > 0.9 mg/L 的特异性、敏感性和准确性分别为 82%、60%和 79% [30]。遗憾的是，目前仍没有生物学标志物可以取代影像学检查结果[34]。但开发新的生物标记物可以在血液和尿液样品中测量，减少侵入性检查并简化检查程序[35]，更好帮助判断病情进展。

6. 综合管理

6.1. 药物治疗

药物治疗主要包括：液体复苏、吸氧、抗凝、溶栓、抗生素及对症支持治疗。液体复苏是 AMI 药物治疗的关键，主要目的是使组织、器官恢复足够的灌注和氧供应[36]。在确诊 AMI 后，应尽早给予患者

足量液体复苏和吸氧。由于肠道血管侧支循环丰富，损伤后大量毛细血管渗漏，液体需求极高。疾病早期，补液量可高达 100 mL/kg，接受血运重建的患者，24 h 内即可达到 10~20 L [10]。平衡晶体液、胶体液及血液制品间的搭配使用，大量输注晶体液[18]可能导致组织水肿，反而不利于恢复肠道灌注。吸氧有助于缓解组织细胞能量不足的问题，有报道称，接近生理水平的氧合，维持氧饱和度 94%~96%，有助减轻高氧诱导的肠道损伤[37]。血流动力学障碍患者，因血管加压素可能会加重肠道缺血需慎用。可优先考虑多巴酚丁胺、小剂量多巴胺和米力农等现已被证明对肠系膜血流影响较小的药物[38]。解除血管痉挛能够进一步优化血液灌注[39] [40]，可使用罂粟碱和/或前列腺素 E1 在内的血管扩张剂进行治疗[41]，提高血流灌注。

抗凝能够阻止血栓延长、预防再次血栓栓塞，是潜在的独立保护因素[5]。在灌注再通或肠管切除后，所有患者均应立即进行抗凝治疗[42]，并尽可能维持全程，甚至终身。目前，大量研究表示推荐使用肝素开始抗凝治疗[21]，并监测凝血功能指标变化，维持活化部分凝血活酶时间(APTT)在 40~60 之间[18]。不同于既往反对溶栓，现有研究表明发病 6 小时内，溶栓治疗是安全有效的[21]，及时的溶栓能有效协助恢复灌注[43]，尤其是无腹膜炎体征的患者。另外，血管内介入治疗局部使用溶栓药也有利于血管再通。使用抗生素能减少“感染、肠道细菌过度生长、革兰氏阴性菌易位和内毒素吸收”的风险[21] [44] [45]，并降低重症患者死亡率[46] [47]。一项纳入 18 例患者的前瞻性研究中使用静脉注射哌拉西林钠他唑巴坦钠方案治疗全身严重感染患者，最终有 11 例不需肠切除[48] [49]。具体抗生素使用时机、治疗方案存在争议，亦有反对经验性使用抗生素研究。

6.2. 血管内介入治疗

早期血管重建首选血管内介入治疗，死亡率低、手术后并发症少[50]。无腹膜炎、休克的患者接受血管内介入治疗更优于传统外科手术[51]。一项包括 4665 例样本数据的研究表明，血管内介入与剖腹手术相比死亡率较低(24.9%与 39.3%) [43] [52]。介入失败可采取剖腹手术补救治疗，这并不意味介入治疗可能浪费时间，介入治疗失败没有增加患者死亡率，可一旦成功，患者死亡率将从 50%降低至 36% [43]。有病例报道称，一位肠系膜上动脉支架置入术的患者发生支架内再狭窄，最终进行了动脉旁路术，成功缓解了危机[53]。接受血管内治疗的患者中有 31%避免了剖腹手术[54]。血管内介入治疗的患者出现腹膜炎表现时，应及时开腹探查[21]。肠切除术和短肠综合征在接受剖腹手术组中发生频率更高。剖腹手术和血管内手术的 30 天死亡率(42%与 28%, $P = 0.03$)和 1 年死亡率(58%与 39%, $P = 0.02$) [54]，血管内治疗后的长期生存率和生活质量也是优于开放手术后。

6.3. 剖腹手术

手术干预包括：血运重建、切除坏死肠段，在此基础上尽可能保留肠段。可用血管造影判断血管情况，条件不允许时可使用多普勒超声替代。一般采用栓子切除术，不成功可以进行动脉旁路术。后者又多用于急性肠系膜血栓形成患者。同时，可以进行局部溶栓治疗[21]。一项回顾性研究表明晚期患者直接剖腹手术进行一站式治疗，可能更有助于提高生存率[55]。有 57%的患者需要进行肠切除[21] [52]，53%的患者要接受二次手术[29] [38]。切除的难点在于判断肠段活力，肠道内层坏死不容易被识别，有时还需二次手术[21]。肠切除和死亡率相关，短肠综合征也会严重降低患者生存质量、增加护理费用[29]。总体剖腹手术患者死亡率较高，大约在 26%~65%之间[21]。统计信息来源存在选择偏倚，因为选择剖腹手术时多数病例已经非常严重。

6.4. 综合管理

早期识别、多学科合作和综合管理对于降低死亡率及改善预后提高生活质量有着重要意义[56]。采用

多学科综合管理的“肠卒中中心”可提高 AMI 患者的生存率并预防肠衰竭[51]。一项前瞻性研究表示在由胃肠病学家、血管和腹部外科医生、放射科医生和重症监护专家组成的肠卒中中心中，患者 30 天存活 95% [48]。

7. 讨论和展望

延误诊断仍然是导致急性肠系膜缺血高死亡率的原因之一，提升早期识别能力有助于改善患者临床结局。目前，尚无统一的实验室检查评估工具，未来开发出新的生物标志物评价量表、改进影像学检查手段有助于疾病早期诊断能力的提升。调整早期再灌注治疗方案、联合多学科共同管理能进一步改善患者临床结局。

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