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·文献综述·

回肠造口性腹泻的早期识别和处理的研究进展

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摘　要　回肠造口术作为外科治疗的常见术式，术后患者常因失去结肠对消化液的吸收引起水、电解质丢失增加，处于相对液体耗尽的状态。回肠造口术后几周内机体存在液体吸收增加的适应过程，但常有患者无法充分适应，造口液大量丢失，也会有其他原因导致回肠造口性腹泻。许多回肠造口性腹泻患者有脱水和电解质丢失的风险，需再入院治疗，消耗大量的医疗资源。外科医生除掌握肠造口手术技术外，应深入对其病理生理的认识，以便实施针对性治疗。笔者对回肠造口性腹泻发病机制及治疗作一综述，以期为此类患者的治疗提供参考。

关键词　回肠造口术；腹泻；手术后并发症；适应，生理学；综述

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Research progress of early recognition and treatment of ileostomy diarrhea

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Abstract　Ileostomy is a common procedure in surgical practice. After this procedure, the patients may be in a state of relative fluid depletion due to the increased fluid and electrolyte losses resulting from the loss of colonic absorption. Within weeks after ileostomy creation, there is a body adaptation process of increased fluid absorption. However, some patients will fail to adapt adequately and have a large volume of ileostomy output, or will develop ileostomy diarrhea due to other causes. Many patients with ileostomy diarrhea are at risk of dehydration and electrolyte loss, and often require hospital readmission, which consumes a lot of medical resources. Except for the mastery of surgical skills of ileostomy, surgeons should fully understand its pathophysiological processes, so as to allow the implementation of targeted treatment. Here, the authors address the pathogenesis and treatment of ileostomy diarrhea, hoping to provide a reference for the treatment of patients with this condition.

Key words　Ileostomy; Diarrhea; Postoperative Complications; Adaptation, Physiological; Review

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结肠手术、严重腹部外伤以及小肠切除等患者，因治疗疾病的需要，需永久性或临时性粪便改道行回肠造口术[1-2]。回肠造口性腹泻作为肠造口术后常见的并发症，指正常情况下每日造口液排出量>1 L/d或>6次/d的造口袋排空，是一种常见且具有潜在危险的疾病，术后早期大约16%~50%的患者出现造口高流出量，存在脱水、电解质紊乱和急性肾脏损伤的风险[3]。术后液体的丢失通常发生在术后几周内，机体通过剩余小肠的适应性改变而缓解。然而部分患者无法充分适应，或因其他致泻原因引起回肠造口高流出量，其中高达20%的患者需再入院治疗，而在造口性腹泻患者中，高达51%的患者需要持续药物治疗[3-4]。有研究[5-6]指出糖尿病和全直肠结肠切除是造口高流出量的术前预测因素，术后腹腔脓肿引起肠道水肿进而导致肠道吸收功能减弱是造口高流出量重要原因。外科医护人员应深入对回肠造口性腹泻病理生理的认识，以便早期识别并实施针对性治疗，但目前尚无针对造口性腹泻的识别和处理方案。本文结合文献最新进展和临床体会，对回肠造口术后造口性腹泻、肠适应的生理学、发病机制及治疗进行阐述。

1 肠造口术后消化液运输的变化

正常情况下9~10 L/d的液体通过屈氏韧带，经空、回肠吸收后，大约有1~1.5 L/d包含电解质的液体进入结肠被吸收，剩余大约100 mL经粪便排出[7]。因此，在回盲瓣水平进行改道约有1~1.5 L/d肠液丢失，其中包括大约200 mEq钠、100 mEq氯和10 mEq钾[8]。

回肠，尤其是末端回肠具有较强的适应黏膜变化的能力[9]。术后早期回肠造口液流出量约1~1.5 L/d[10]，但造口液流出量在随后的数天到数周内逐步下降，经历适应的过程[11]。回肠切除长度对适应程度影响明显。随着小肠切除的增加，回肠造口液流出量也增加，切除更多的回肠不仅使吸收面积减少，还失去“回肠减速”机制导致适应变差[9]。

2 肠造口术后的肠适应性

肠适应性被认为是由激素、管腔、机械等因素共同作用引起黏膜结构变化和肠蠕动、通透性、电解质运输和吸收能力的改变[12]。

2.1 肠黏膜形态学的适应

肠适应性通常认为是黏膜肥大和剩余小肠的增生。在动物模型中，剩余小肠出现循环速率增加和肠道多能干细胞增殖，绒毛长度和绒毛细胞增加[12-14]。回肠造口动物模型在术后几天内，回肠黏膜的重量和厚度增加[9]。

2.2 电解质转运的适应

醛固酮在适应过程中起到至关重要的作用[15-16]。盐皮质激素通过直接激活上皮钠通道（ENaC）受体介导的电解质转运调节[17-18]。人体的肠道灌注研究中，结肠切除术后早期钾排泄和钠潴留明显增加[19]。盐皮质激素虽主要诱导肾脏的钠潴留，也会促进小肠的钠吸收。急性肾上腺功能不全可导致回肠造口排出量大幅增加[20]。

2.3 肠动力的适应

肠道排空缓慢、增加吸收时间是结肠切除术后肠适应的另一机制。回肠造口患者回肠排空减慢了约4倍[19]。上消化道的动力降低可能是由于“回肠制动”的上调所致[21]。小肠平滑肌的改变也可导致排空力下降，结肠切除的大鼠显示纵向肌肉收缩频率降低，提示推进性蠕动减少，减慢排空并促进吸收[9]。

3 回肠造口性腹泻的原因

回肠造口性腹泻的原因分两种情况：无法适应的回肠造口性腹泻和适应后的患者表现出新的造口液排出量增加（图1）。

3.1 无法适应的回肠造口性腹泻

无法适应可能与回肠切除的程度有关。未治疗或亚临床肾上腺功能不全的患者在围手术期可能会因使用影响肾素-血管紧张素-醛固酮系统的药物使得适应性变差。反射性酸分泌过多也导致肠适应性降低[22]。

3.2 适应后的回肠造口性腹泻

腹泻的传统病因也适用于回肠造口患者，包括感染、摄入不易吸收食物、腹腔疾病和药物原因。因无法通过结肠吸收补充液体丢失，所以表现更加严重。除此以外，这些患者还应考虑其他病因，包括克罗恩病复发、小肠细菌过度生长（small intestinal bacferial overgrowth，SIBO）和胆汁

酸缺乏[23]。同时结肠切除并不能消除艰难梭菌感染可能，且病死率比艰难梭菌结肠炎更高，近期有抗生素暴露、质子泵抑制剂（proton pump inhibitor，PPI）使用的患者都应考虑这一点[24-26]。

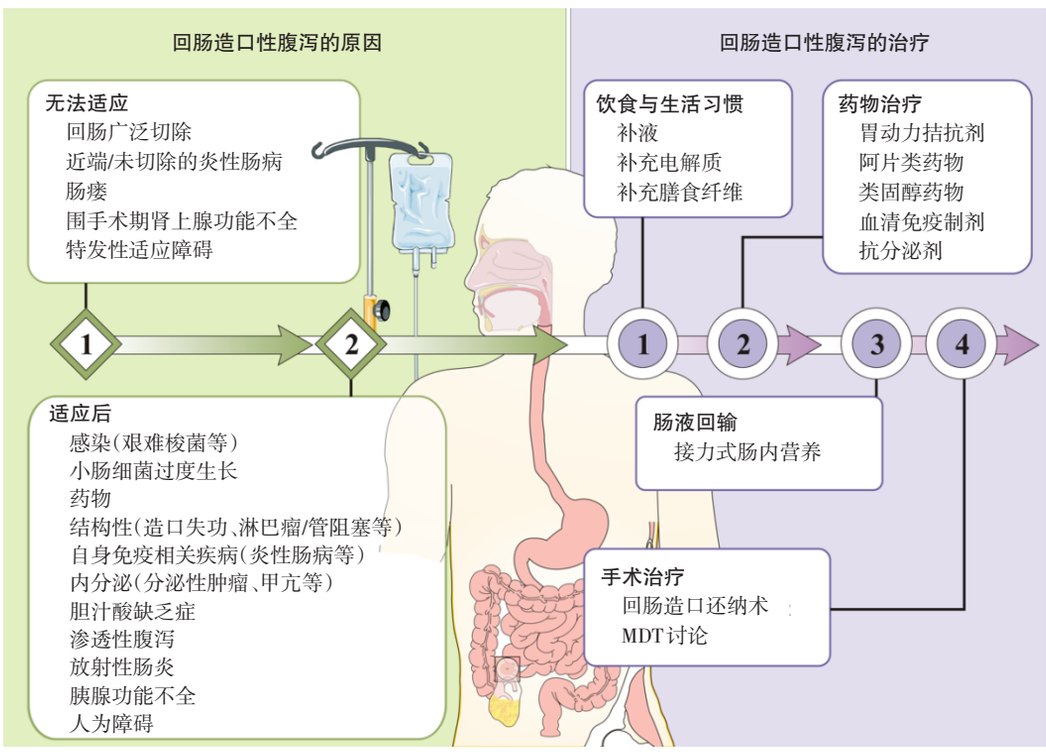

图 1　回肠造口性腹泻的原因和治疗

Figure 1　Causes and management of ileostomy diarrhea

4　回肠造口性腹泻的早期识别

4.1　病史和体格检查

首先全面了解病史、饮食记录和体格检查，同时获取患者的手术记录和影像学检查结果，准确了解解剖结构尤其是回肠切除的长度，分析切除标本的病理结果，以明确诊断并指导后续评估。体格检查应评估容量状态，判断患者是否需要静脉补液。评估血压、皮肤干湿和黏膜水合状态等生命体征。许多临床表现与饮食等因素有关，并不代表造口排出量增加，专业的肠造口护理师可通过记录造口排液的频次和体积统计造口液排出量，早期识别回肠造口液排出过多，并帮助解决问题[27-28]。去除造口袋后行回肠造口检查，评估是否有造口脱垂、疝或狭窄。造口患者出院后对其早期随访宣教可以显著降低再入院率[29]。

4.2　辅助检查

回肠造口性腹泻的早期评估与胃肠道完整患者的急慢性腹泻评估类似。实验室检查包括全血细胞计数、电解质、肌酐、IgA 抗组织转谷氨酰胺酶筛查乳糜泻、促甲状腺激素和早晨皮质醇水平。粪便检查应包括微生物学检查（粪便培养、艰难梭菌卵和寄生虫检查，贾第鞭毛虫和隐孢子虫的免疫学检查或多种生物的聚合酶链反应）、粪铁蛋白铁钙卫蛋白和隐血试验。

结构评估使用包括 CT、MRI 或小肠消化道造影判断狭窄或阻塞原因。如对远端梗阻的评估不确定，应行小肠逆行钡剂造影了解末端回肠和造口情况。对于有炎症性肠病（inflammatory bowel disease，IBD）病史或病因不明的患者，应行胃镜、回肠镜及黏膜活检。如果诊断仍不清楚，需定时收集粪便区别分泌性、渗透性、吸收不良和炎症原因，为诊断提供其他线索。严重的造口腹泻则应考虑是由广泛回肠切除的胆汁酸缺乏症、胰腺外分泌功能不全、黏膜病或 SIBO 引起[30]。

5　回肠造口性腹泻的治疗

5.1　补液、饮食调整

回肠造口性腹泻首先处理水、电解质紊乱及

相关并发症。根据脱水的严重程度，给予静脉或口服补液。有造口周围皮炎或渗漏的患者，添加膳食纤维丰富的食物，可增加造口液体黏稠度。研究[31]表明，添加15 g果胶使回肠造口液黏度增加约100%，而酒精、水果和蔬菜等则容易导致造口液增多。

5.2 肠液回输

尽可能地应用更多的肠管[32]。对于有条件行肠液回输的患者，积极地行肠液回输，有助于结肠对消化液的吸收，避免水电解质的丢失，维持内环境的稳定。肠液回输可提高营养物质的吸收量，缩短补充性肠外营养的应用周期，避免结肠的废用性萎缩，为后续进一步治疗创造条件。笔者单位近年来积极推广接力式肠内营养，不仅做到了全程封闭、连续回输和引流，保证了消化液的新鲜和时效，还能促进肠黏膜的生长与修复，维持肠黏膜结构和功能的完整性，减少肠源性感染的发生[33]。

5.3 适应失败的治疗

5.3.1 增加适应性药物 特杜鲁肽是一种皮下注射的GLP-2激动剂，已被批准用于短肠综合征所致的慢性肠衰竭[34]。GLP-2受体被认为是通过GLP-2肠细胞受体发挥作用，促进黏膜生长，减少胃动力和胃酸分泌，增加肠系膜血流[35-36]。

5.3.2 抗运动剂 抗运动剂包括洛哌胺、苯乙氧基化物/阿托品、可待因、吗啡和阿片类。洛哌丁胺和苯乙氧基化物是合成的具有抗运动作用的，尤其作用于小肠的阿片激动剂[37-38]。洛哌丁胺可使已建立的回肠造口排出量减少16%~30%，且洛哌丁胺通过血脑屏障的能力有限，与阿托品或阿片类药物相比，具有更小的中枢和抗胆碱能副作用[39-40]。

5.3.3 抗分泌剂 结肠切除术后1年，基础和刺激性的胃酸分泌大约增加1倍[22]，胃酸分泌过多可导致早期的高排出量。有报道[41]指出，应用抑酸剂可治疗回肠造口性腹泻。对于原因不明的高排出量回肠造口性腹泻可经验性应用PPI[42]。生长抑素类似物在多方面都显示其抑酸分泌的有效性[43-44]，回肠造口术后严重造口性腹泻患者使用生长抑素类似物能够降低造口液排出量并延缓小肠排空时间[45]。对已获得足够控制并确定适当剂量的回肠造口性腹泻患者，可每月给予长效生长抑素类似物注射剂。糖皮质激素也在肠适应过程中发挥重要作用，其通过减轻IBD的炎症反应，影响盐皮质激素活性改变离子和水的转运来改善造口液高排出量症状[46-47]。氟可的松是一种有效的盐皮质激素激动剂，可以减少无腹泻患者的回肠造口液量，但仍需要进一步研究探索其在高排出量患者中的益处[48]。

5.4 胆汁酸缺乏症

胆汁酸结合剂会加重脂肪吸收不良和脂肪泻，并不适用于末端回肠造口患者。在回肠切除较长的患者（通常>100 cm），胆汁酸的丢失可能超过肝脏的产量，并可能导致因胆汁酸缺乏而引发的脂肪泻，应该考虑补充胆汁酸[49]。

5.5 手术治疗

条件允许的前提下，可关闭转流或襻式回肠造口解决回肠造口性腹泻导致的问题。对于解剖不佳、远端吻合口存在问题的患者，应多学科讨论继续行粪便转流的必要性[50]。

6 小 结

回肠造口性腹泻具有复杂的病理生理，对患者生活质量和预后也具有较大影响。虽然病因较为常见，但仍需考虑复发性IBD、胆汁酸缺乏、SIBO和需要手术干预的解剖异常等特殊情况。强调尽早识别回肠造口性腹泻患者，及时补水和寻找病因，针对根本原因进行处理辅以药物治疗，减少造口液排出量，对提高患者生活质量、改善预后尤为重要。

利益冲突：所有作者均声明不存在利益冲突。

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