·综述·

脑肠互动异常在顽固性便秘诊治中的意义

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【摘要】 慢性便秘是影响人们生活质量的常见消化系统疾病，其病因复杂，治疗难度大。近一半的慢性便秘患者治疗效果并不理想，称为顽固性便秘。目前，临床对顽固性便秘的诊治主要集中在肠道局部治疗。然而近期研究发现，顽固性便秘的发生和发展是一个多因素参与的复杂过程，单一针对肠道症状治疗并不能取得理想的效果。基于罗马Ⅳ提出的脑肠互动障碍理念，我们发现在顽固性便秘患者中，脑肠互动障碍可能起到了关键作用。本文将从脑肠互动障碍的角度，围绕中枢神经系统，外周神经系统，内分泌系统及肠道4个方面，对顽固性便秘的病理机制、诊治思路进行综述。

【关键词】 顽固性便秘；　脑肠互动障碍；　慢性便秘；　脑肠轴

基金项目：国家自然科学基金项目（82100680）；西京医院助推项目（XJZT24CY45）；国家消化系统疾病临床医学研究中心临床应用研究课题（NCRCDD2020-05）；宁夏回族自治区科技惠民项目（2023CMG03017）；宁夏自然科学基金（2023AAC03471、2024AAC03470）

Significance of disorders of brain-gut interaction in the diagnosis and management of refractory constipation

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【Abstract】 Chronic constipation is a common digestive disease that affects people's quality of life, with complex causes and difficult treatment. Nearly half of the patients with chronic constipation do not achieve satisfactory treatment results, which is referred to as refractory constipation. Current clinical strategies for addressing refractory constipation have predominantly focused on the mitigation of intestinal symptoms. However, emerging evidence suggests that the pathogenesis and progression of refractory constipation are multifactorial and highly intricate, and that strategies targeted solely at symptom relief may be insufficient to yield optimal therapeutic outcomes. Based on the concept of disorders of brain gut interaction disorder (DBGI) proposed by Rome IV, we found that DBGI may play a key role in patients with refractory constipation. From the perspective of DBGI, this review synthesized the contemporary insights into the pathological mechanisms underlying refractory constipation, as well as diagnostic and therapeutic strategies focusing on four aspects: the central nervous system, the peripheral nervous system, the endocrine

DOI：10.3760/cma.j.cn441530-20240328-00113

收稿日期　2024-03-28　　**本文编辑**　万晓梅

引用本文：田博文，金涛，苏雪，等. 脑肠互动异常在顽固性便秘诊治中的意义[J]. 中华胃肠外科杂志，2024，27(12)：1301-1308. DOI：10.3760/cma.j.cn441530-20240328-00113.

system, and the intestinal environment.

【Key words】 Refractory constipation; Disorders of brain-gut interaction; Chronic constipation; Gut-brain axis

Fund programs: National Natural Science Foundation of China (82100680); Xijing Hospital Promotion Project (XJZT24CY45); Clinical Application Research Project of National Clinical Research Center for Digestive Diseases (NCRCDD2020-05); Ningxia Autonomous Region Science and Technology Benefiting People Project (2023CMG03017); Ningxia Natural Science Foundation (2023AAC03471, 2024AAC03470)

慢性便秘在我国发病率16%~20%，是最常见的胃肠道疾病之一[1]。顽固性便秘指标准治疗无效的慢性便秘，包括生活习惯改善、泻剂、益生菌和手术等[2-5]。顽固性便秘占慢性便秘50%以上，是一种独特便秘亚型，极大增加患者痛苦，自杀者时有发生[6]。

脑肠互动异常（disorders of brain-gut interaction，DBGI）是顽固性便秘重要的潜在病理机制之一。先前多使用功能性便秘定义不合并器质性异常的亚型。而罗马Ⅳ中用DBGI取代了功能性胃肠道疾病表述，以明确病因来源[7]。脑肠间存在的复杂相互作用，包括情绪、神经、免疫、内分泌和肠道微生态等多个系统[8]。DBGI代表的心理-社会-生物医学模式，让我们以更广的视野来观察、认识和处理顽固性便秘。

以躯体化症状为核心的精神障碍在DBGI中起关键作用。这在导致顽固性便秘的同时，也常引起多种不适，如睡眠障碍、躯体症状焦虑、抑郁、腹胀腹痛、消化不良、胃轻瘫和大便失禁等[9-10]。目前，国内外便秘诊治基于症状和生理检查分类，未能评估躯体化症状等精神障碍的作用。通过DBGI视角探讨精神障碍对顽固性便秘发病机制的影响，建立有效的评估筛查方法，将对提升便秘的诊疗效果产生深远意义。

一、便秘流行病学

便秘与精神症状发病率间存在密切的相互关系，心理因素可以引起便秘，便秘也可以引起极大的情绪障碍。精神障碍患者的便秘发生率为20%~37%，远高于一般人群[11]。一项纳入1 000多例为期12年的前瞻性研究提示，焦虑是新发功能性胃肠病的独立预测因子[12]。一项研究提示，慢性便秘患者的焦虑和抑郁患病率分别为34.6%和23.5%；另外，自杀倾向高（14.8%）也值得我们注意[13]。另一项关注于中国老年功能性便秘患者的研究同样也得到了类似的结果，分别有30.8%和21.7%的功能性便秘患者患有抑郁症和焦虑症，抑郁和焦虑的比例随着便秘症状的加重而增加[14]。本单位前期数据统计发现，顽固性便秘多合并焦虑（80.20%）、抑郁（72.77%）或躯体化症状（86.32%）等异常，可能构成顽固性便秘重要特征之一（数据未发表）。作为核心症状，合并躯体化症状的功能性便秘患者表现出了更严重的症状以及更差的治疗效果，与顽固性便秘存在紧密关系[15]。

二、便秘病理机制及影响因素

顽固性便秘肠道治疗效果不佳的原因，可能受到多种肠外因素影响，包括环境、大脑和脑肠轴等。环境因素包括早期生活经历、家庭、社会、文化和应激创伤等。大脑因素包括心理及中枢神经异常，并通过脑肠轴影响排便。脑肠轴包括自主神经及内分泌系统。最终肠道病理包括动力异常、内脏高敏感性、免疫屏障失调和肠道微生态改变等，并反过来影响精神及神经结构，构成复杂的相互作用网络，见图1。

家庭社会环境刺激可诱发大脑精神障碍改变，并导致便秘等肠道功能异常。有研究显示，很多类似的功能性胃肠病呈现家族聚集性[16]。其中父母信念及行为有重要作用，它影响了孩子面对便秘时的反应、处理及情绪。父母过度关注下，孩子可能学习强化而表现更强烈的腹痛等症状[17]。此外，有荟萃分析汇总11项研究后发现，儿童心理压力和负担也与功能性便秘风险显著相关[18]。社会关系或文化等其他压力事件也对便秘症状产生重要影响。在便秘及排便紊乱患者中，不良生活事件及创作后应激事件发生率分别达到了75.4%及27.5%[19]。不良生活事件可能诱发焦虑、抑郁等精神障碍，并进一步导致内脏超敏，增强大脑对肠道疼痛感知，形成顽固性便秘的重要病理机制[20]。55%的IBS患者表现出内脏超敏特征，是肠易激综合征（irritable bowel syndrome，IBS）主要症状之一[21]。而罗马Ⅳ标准将不同便秘亚型视为疾病谱不同阶段，暗示顽固性便秘也可能存在与IBS类似的机制[22]。

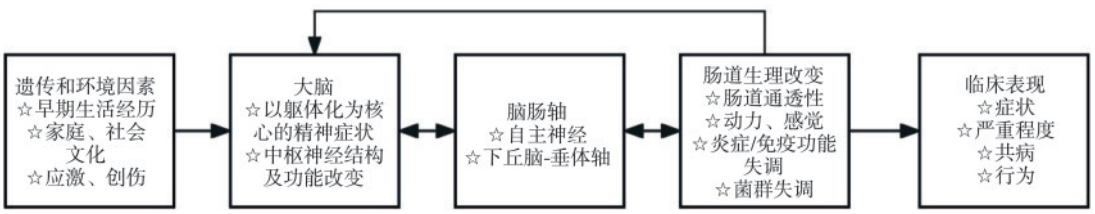

图1 社会心理因素通过脑肠轴与便秘症状相互影响

脑肠轴包括自主神经及内分泌系统，维系了中枢到内脏平滑肌末梢间的双向联系，是导致顽固性便秘的直接通路[23]。迷走神经为自主神经核心。应激等刺激引起的大脑改变通过迷走神经介导胃肠道功能障碍[24]。神经递质则以5-羟色胺（5-hydroxytryptamine，5-HT）为主。5-HT也称为血清素，主要由肠嗜铬细胞通过色氨酸羟化酶（tryptophan hydroxylase 1，TPH1）合成[25]。约有95%的5-HT存在于胃肠道在调控胃肠道运动中扮演核心作用[26-27]。血清素转运蛋白（serotonin transporter，SERT）可转运并降低5-HT浓度，抑制肠道蠕动，是顽固性便秘关键潜在靶点[28]。脑肠轴还受下丘脑-垂体轴（hypothalamic-pituitary axis，HPA）代表的内分泌系统调节。HPA轴启动激素级联反应，增加炎性反应因子，成为肠道症状来源之一[29-30]。微生物也在脑肠轴中扮演重要调控角色，我们称为微生物-脑肠轴[31]。肠道菌群通过代谢产物参与脑肠轴通讯包括色氨酸代谢物、短链脂肪酸（short-chain fatty acid，SCFA）、神经活性肽及胆汁酸等[32-33]。具体机制包括诱导肠嗜铬细胞分泌5-HT、改善神经元功能等，最终通过刺激局部神经系统（肠神经和迷走神经）影响大脑功能和认知，并影响肠蠕动[34-35]。除多种因素影响肠道蠕动外，长期顽固性便秘反过来也会影响精神及神经系统。与健康人群相比，慢性便秘患者合并焦虑及抑郁概率明显上升[36-37]。DBGI患者结肠黏膜炎性反应激活肥大细胞，刺激内脏传入神经元引发超敏反应。该反应放大了内脏传入信号，引起更明显的不适感，并反向加剧了精神及神经系统症状[38-39]。

三、便秘的筛查方法

首诊医生需要警惕以躯体化症状为核心的顽固性便秘[40]。目前，国内外指南根据症状和生理学检查确定便秘分类，缺乏以躯体化症状为核心的症状评估。需要注意的是，很多顽固性便秘并不伴排便次数减少和大便干燥，而是缺乏满意的完全性自发性排便不足。该概念指的是排便后的完全排空感，反映了排便后满意度。完全性自发性排便改善也是衡量顽固性便秘治疗有效的核心标准[41]。临床医生多对此缺乏认识，常致治疗失败，甚至接受不必要的手术。

顽固性便秘相关躯体化症状识别要点如下：（1）令人痛苦的便秘相关躯体症状，如消化不良、反酸嗳气、腹痛腹胀、睡眠障碍和头晕乏力等。此项对是否有明确病因不作要求。（2）对痛苦症状出现过度的想法、感觉或行为，经常把自己健康想象得极其糟糕。（3）躯体症状超过6个月。我们在问诊中要关注患者每天要花多少时间来处理身体问题，每天超过3 h思考或处理躯体不适可认为过度，这一指标可帮助医生快速筛查[42]。焦虑主要症状为担忧及紧张，抑郁特点为情绪低落、兴趣或愉悦感缺失、思维迟缓等。以上症状均可辅助专科医生在访谈中识别。

另外，自评量表也是重要的评估手段。焦虑、抑郁、躯体化症状分别通过广泛性焦虑自评量表-7（generalized anxiety disorde-7，GAD-7）、患者健康问卷-9（patient health questionnaire-9，PHQ-9）、患者健康问卷-15（patient health questionnaire-15，PHQ-15）进行评估。评分≤4一般为认为正常，4~10为中度，≥10为重度。目前精神症状评分与顽固性便秘尚无相关研究发表，根据本中心临床工作经验，精神症状重度（≥10）是顽固性便秘的独立危险因素。

压力、应激及睡眠障碍等生活不良事件是重要诱发或伴随因素，有助于全面评估顽固性便秘严重程度。慢性压力被认为是功能性胃肠病发展和维持风险因素[43]。生活质量受损也与焦虑和抑郁症状成为DBGI首次发病的独立预测因子[44-45]。应激事件同样导致异常精神状态，并在功能性便秘群体中拥有更高发生率[46-47]。睡眠障碍也会导致顽固性便秘并增加患胃肠道疾病的风险。Jiang等[48]对126例成年慢性便秘的患者统计分析发现，与睡眠正常的患者相比，有睡眠障碍的慢性便秘患者经常抱怨更多的躯体症状和情绪障碍，其中睡眠障碍、抑郁和焦虑都与便秘严重程度呈正相关，成为潜在的顽固性便秘病因。此外，休学、辞职或丧失社交功能也是常见心理异常特征。

大脑功能或结构MRI改变可作为潜在特征性改变，为顽固性便秘提供客观依据[49]。一项针对功能性便秘静息态功能性磁共振成像（resting-state functional magnetic resonance imaging，rs-fMRI）研究显示，情绪调节相关大脑区域发生改变[50]。结构MRI也被用于评估与脑功能变化相关结构改变。有研究调查29例功能性便秘患者，报告了该组患者背侧前扣带回皮层、眶额叶皮层、后扣带皮层、楔前叶和补充运动区的皮质厚度减少[51]。Duan等[52]发现，合并焦虑、抑郁的功能性便秘患者大脑出现特异性改变。本课题组也通过fMRI发现，顽固性便秘右侧部分小脑、右侧壳核、左侧丘脑和右侧脑岛的低频振荡振幅与正常对照组有显著差异，提示潜在机制同时，也为特征性评估提供了潜在依据（数据未发表）。Zhang等[53]使用弥散张量成像（diffusion tensor imaging，DTI）的研究反映了功能性便秘与丘脑和边缘（顶叶）皮层之间结构连接的改变有关，并突出了丘脑在功能性便秘患者结构连接变化中的综合作用。此外，近期一项研究利用rs-fMRI和Allen脑图谱，揭示了功能性便秘患者脑区改变与突触信号、中枢神经系统发育、脂肪酸代谢和免疫相关的基因表达模式的关联，这些基因表达与微生物-脑肠轴相关，这为功能性便秘的病理生理提供了新的视角[54]。

身心症状外，也须注意器质性病变对顽固性便秘的影响，包括继发性和原发性改变。继发性常见于机械性梗阻、肿瘤、代谢紊乱及药物等[55]。此类患者可通过病史、体格检查或辅助检查找到原发病。原发性便秘评估包括结肠运输时间、肛肠测压、球囊排出试验、排粪造影等，以确定局部解剖功能异常[56]。常见的局部解剖异常有：结肠冗长迂曲，结肠下垂、脾区综合征、结肠扩张、直肠内脱垂和直肠前突等。值得注意的是，很多轻中度解剖异常便秘手术效果不佳，提示非重度解剖异常可能并非便秘唯一病因。评估时需综合考虑精神、菌群等多方面影响。

综上，顽固性便秘诊治应基于生物-心理-社会医学模式展开，综合评估便秘症状、不良事件病史、排便功能和心理异常等心身要素。对于“四多一少”典型患者，须高度怀疑顽固性便秘可能，包括就诊医院多、症状多、检查多、使用药物多和异常检查结果少。排除器质性病变后，应重点关注躯体化症状等心身异常，对于每天思考或处理躯体不适超过3 h，持续6个月以上的患者，应高度怀疑并给予针对性干预。

四、便秘的干预策略

生物-心理-社会医学模式对顽固性便秘有重要治疗潜力。应充分考虑患者精神症状，以医患沟通为核心，辅以行为认知、神经调控以及肠道菌群等综合治疗。医患沟通首要环节是取得患者信任[57]。良好的医患关系可提高患者满意度及依从性。非语言信息中展示友善信号，如点头或友善的眼神接触、柔和的语气等。交谈中以患者为中心，关注患者以躯体化症状为核心的精神状态。怀疑顽固性便秘后，以简洁方式解释脑肠相互作用，为治疗奠定基础。最后参考慢病管理模式，通过互联网医院建立长期联系，以取得最佳治疗效果。DGBI相关顽固便秘诊治流程见图2。

顽固性便秘是一种跨越学科的复杂疾病，其治疗需要以患者为中心的个体化多学科治疗。参考罗马Ⅳ、躯体症状障碍专家共识及类似病理机制，顽固性便秘可分为轻中重三级[58-59]。轻者无明显心理问题。治疗重点以教育随访为主，指导患者意识到顽固性便秘症状源于心理异常；配合安慰、饮食控制及泻剂，指导患者树立正确观念。中度患者有明显心理困扰，部分丧失社交活动能力；建议记录排便日记，通过行为认知、肠道菌群、神经调节剂等方式干预。每3个月再评估复查。重度患者伴有严重焦虑、抑郁或躯体化症状等心理障碍，甚至有自杀风险，需转至心理科进一步治疗；消化专科可尝试神经调节剂、神经刺激等协同配合治疗[60-61]。

肠道菌群可通过干预神经系统缓解症状进展，具体包括饮食、益生菌、合生元、后生元和粪便微生物群移植(fecal microbiota transplant，FMT)等。高纤维素食物可增加SCFA并抑制致病菌定植，改善顽固性便秘症状[62-63]。近期一项研究发现，红心火龙果具有作为预防或改善早期便秘的安全食品补充剂的潜力[64]。指南推荐饮食调整作为初始治疗[65]。Zhang等[66]发现，不同比例的多菌种益生菌通过改变肠道菌群的组成和结构，增加SCFA和乙酸的含量降低肠道炎性反应来改善便秘。此外，据报道，益生菌对中枢神经系统疾病具有调节作用，包括焦虑和抑郁样行为的正常化和孤独症的减少[67-68]。Bazzocchi等[69]以车前草纤维为益生元结合乳酸菌和双歧杆菌，有效改善了排便频率和粪便稠度。丁酸盐作为后生元调节巨噬细胞功能，可影响便秘患者结肠蠕动[70]。FMT同样可改善便秘及结肠慢传输症状[71]；同时缓解IBS精神症状[72-73]。综上，肠道菌群有潜力缓解便秘症状的同时，改善精神障碍，有较好治疗前景。

神经调节剂被认为可沿整个脑-肠轴发挥中枢调节和外周消化道效应，具有广泛的干预效果[74]。选择性血清素受体再摄取抑制剂(selective serotonin reuptake inhibitor，SSRI)作为治疗焦虑和抑郁一线药物，应用最为广泛。有研究显示SSRI氟西汀可减轻IBS-C患者腹部不适及腹胀、增加排便频率并改善大便黏稠度[75]。我们在临床实践中发现，使用氟西汀治疗顽固性便秘，不仅可以显著改善精神障碍，还有效缓解顽固性便秘症状，预实验提示6个月综合有效率达到了47.1%。用药应坚持4周以上，如果有效需维持1年以上，再逐步减量。苯二氮卓类药物用于治疗精神疾病并与IBS共病，通过增强GABA受体在中枢神经系统发挥镇静作用[76]。尽管短期使用安全，长期使用可能导致认知障碍和出现其他不良反应，故仅推荐用于治疗伴有焦虑的IBS患者[77]。三环类抗抑郁药(tricyclic antidepressant，TCA)用于治疗IBS症状，其镇痛机制涉及抑制去甲肾上腺素和血清素再摄取，增加神经递质活性等。TCA由于其抗胆碱能作用可能使结肠运动更加缓慢，仅推荐在结肠传输试验正常患者中使用[78]。

认知行为疗法是一种由专业人员提供的心理干预，通过改变有害思维模式和促进有益行为来提升个体适应性。认知行为疗法广泛应用于多种心理和消化系统障碍，包括抑郁症、焦虑症、失眠和强迫症等[79]。认知行为疗法对愿意接受心理干预患者尤其有效[80]。举例来说，许多患者倾向于夸大腹胀等肠道刺激带来的感觉，并低估自己控制能力。认知行为疗法可帮助患者认识到自己的这种行为。大量证据支持认知行为疗法对IBS、嗳气和功能性消化不良等DBGI的疗效[81-82]。对于顽固性便秘患者，认知行为疗法也可能是有益的干预措施。

生物反馈也是潜在有效的方法，它使用设备记录或放大肌肉收缩和松弛的生理过程，将其转换为视觉或听觉信号并反馈给患者，让患者从反馈中学习并控制功能紊乱[83]。一项随机对照试验表明，在治疗功能性排便障碍方面，生物反馈比假治疗、泻药、饮食调整等更有效[84]。针对合并盆底失迟缓的顽固性便秘患者可以在进行生物反馈之前对患者进行认知行为疗法心理干预，这样不仅可以使患者充分地了解自身情况，并且可以充分地配合进行生物反馈治疗来学习如何控制或改变疾病。

神经电刺激可尝试用于保守治疗效果不佳的患者，包

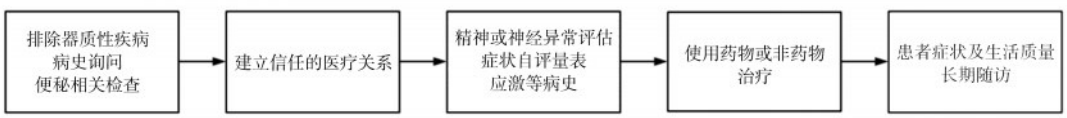

图2 脑肠互注异常相关顽固性便秘的诊治

括骶神经刺激、经皮骶神经刺激和胫后神经刺激等。骶神经刺激在第三骶椎孔放置电极，并在臀部皮下植入神经调控器[85]。骶神经刺激最初用于排尿障碍，后发现对肠道功能也有益处[86]。然而，骶神经刺激治疗便秘有效性存在争议[87-88]。尽管如此，欧洲神经胃肠病学与运动学会指南仍建议，顽固性便秘仍可尝试骶神经刺激，微创性是其潜在优势[89]。经皮骶神经刺激作为一种非侵入性骶骨神经调控方法，机制类似于骶神经刺激。研究表明，经皮骶神经刺激治疗4周后便秘评分和生活质量有所改善[90]。Kim和Yi[91]也观察到了经皮骶神经刺激对排便和便秘的积极影响。胫神经分支自坐骨神经，对盆底肌肉和胃肠道有刺激作用。胫后神经刺激通过脊髓受体传递冲动至骶神经，调节肠道和括约肌功能，并诱导中枢可塑性[92]。研究表明，胫后神经刺激可引起与骶神经刺激相似的肛直肠功能变化，并可显著改善便秘评分[93-94]。神经调控有潜力填补保守治疗无效至手术前空白，但其有效性仍需进一步验证。

上述治疗无效时可考虑手术策略，所有顽固性便秘患者在行手术治疗前，应行心理医生访谈，排除精神心理因素，合并有精神心理疾病或心理状态异常是外科手术的禁忌[95]。手术治疗时应严格掌握以下适应证：(1)经诊断和鉴别诊断，确诊慢传输型便秘；(2)排除胃或小肠传输功能障碍；(3)结肠切除手术前应排除直肠推动力不足及盆底痉挛型出口梗阻型便秘；(4)排除结直肠及腹腔内脏器的器质性疾病；(5)经非手术治疗效果差、长期依赖刺激性导泻剂或灌肠治疗，严重影响日常生活，有强烈的手术意愿；(6)无严重的精神心理障碍因素，常用的手术方式有：全结肠切除-回肠直肠吻合术、结肠次全切除联合不同肠道重建、回肠或结肠造口术、结肠旷置术、经阴道直肠前突术和腹腔镜腹侧补片直肠固定术等[96-98]。但手术通常是一种不可逆的选择，往往适用于严重解剖异常、保守治疗无效且症状严重影响生活质量的患者，需严格把握手术指征。

五、未来及展望

现有的便秘或者排便障碍分型不能很好地指导临床实践，因排便障碍自杀的患者时有发生，如何将此类患者鉴别出来，是现有指南急需解决的难题。临床医生需要特别重视生物-心理-社会医学模式在便秘中的作用。目前，便秘诊治大量依赖于症状及经验，相关病理生理机制研究尚处于早期。我们受罗马Ⅳ指南启发，根据临床观察及文献研究，猜想DGBI是重要的潜在便秘机制。DBGI中精神症状、神经-内分泌紊乱、肠道功能及菌群异常都可能导致便秘发生。及早关注DBGI相关危险因素并早期及时介入，可以避免过度治疗，并大大提高治疗有效率。

利益冲突　所有作者均声明不存在利益冲突

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