·指南与共识·

炎症性肠病营养支持治疗专家共识(第二版)

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自《炎症性肠病营养支持治疗专家共识(2013·深圳)》发表以来,营养支持治疗(nutrition support therapy,既往称为营养支持)在炎症性肠病(inflammatory bowel disease,IBD),尤其是克罗恩病(Crohn's disease,CD)治疗中的作用已得到国内消化科医生的广泛重视[1]。近5年来,关于IBD营养支持治疗的机制、适应证和实施方法等方面均有较多研究进展。为及时反映国内外最新认识和研究进展,中华医学会消化病学分会炎症性肠病学组、中华医学会肠外与肠内营养学分会胃肠病与营养协作组的专家联合对2013年版IBD营养支持治疗专家共识进行更新。

本共识采用Delphi程序进行制定。上述两学组专家对文献进行检索、筛选、评价,确定共识需阐明的问题及推荐方案,再经两组专家讨论修改,进行投票并由第三方计票。投票等级分为a完全赞成(必不可少);b部分赞成,但有一定保留;c赞成,但有较大保留;d不赞成,但有一定保留;e完全不赞成。本共识意见中的推荐等级根据投票结果分为A级指标(强烈推荐),即a得票数为80%及以上;B级指标(推荐),即a和b得票数相加为80%及以上;C级指标(建议),即a、b和c得票数相加为80%及以上;未达C级指标则删去。最终由专家审阅定稿形成本共识意见。根据证据级别高低及专家投票结果,本共识将推荐等级分为"强烈推荐"、"推荐"和"建议"3个等级。

一、IBD患者营养状况及其对疾病的影响

1. 营养不良是IBD患者的常见临床表现,并对病情变化产生不良影响。推荐等级:强烈推荐

营养不良是指患者现存的营养状况受损,分为营养不足、超重以及肥胖三大类[2]。但国内IBD患者多表现为营养不足,因此本共识中营养不良特指营养不足。IBD营养不良表现多样,以蛋白质热量型营养不良多见,表现为消瘦和体质量下降,病程久者多表现为混合型营养不良[3]。

国外文献报道,IBD营养不良发生率为16%~85%,85%~100%的儿童CD患者有营养不良史,疾病活动期营养不良比缓解期普遍[4-7]。国内因并发症住院手术的CD患者合并营养不良的发生率高达86.7%[8]。2017年我国IBD住院患者的营养状况调查结果表明,营养不良发生率为55%(结果待发表)。小肠尤其是回肠是消化和吸收营养的主要部位,由于CD病变常累及小肠,而溃疡性结肠炎(ulcerative colitis,UC)仅累及结直肠,所以CD患者营养不良比UC多见[9]。

营养不良增加IBD患者住院率,延长住院时间,降低患者抗感染能力,妨碍手术切口和肠吻合口愈合,增加手术并发症发生率和病死率,影响机体对药物治疗的反应,降低患者生活质量[9-13]。营养不良还是IBD患者发生静脉血栓事件和急诊手术的独立风险因素,是造成儿童和青少年患者生长发育迟缓或停滞的主要原因[10-11,14]。纠正营养不良有利于改善患者营养状况,提高治疗效果。

2. IBD患者常合并机体组成的变化。推荐等级:推荐

机体由骨质群(bone mass)、脂肪群(fat mass)和瘦组织群(lean body mass)3部分组成,瘦组织群又由细胞外总体与体细胞总体两部分构成。IBD患者由于营养摄入不足和疾病的影响,常出现机体组成改变,如骨骼肌减少、脂肪堆积或脂肪减少[15]。在上述情况下,虽然患者体质量和体质指数(body mass index,BMI)可能正常,但机体组成已经发生改变,通过分析机体组成能够更准确地反映患者的营养状况[16]。骨骼肌减少也称为少肌症(sarcopenia),包括骨骼肌质量、力量及功能的进行性下降[17]。少肌症在IBD患者中非常普遍,并受疾病活动度和治疗药物的影响[18-22]。BMI正常的IBD儿童中,有93.6%的CD和47.7%的UC患儿存在少肌症;60%的成人IBD患者合并少肌症[21,23]。由于骨骼肌减少和力量下降,少肌症患者的活动量减少,容易出现疲劳、骨质疏松和脂肪堆积,IBD手术率和术后并发症增加,生活质量降低[18,21,24-25]。

肥胖可能影响IBD病程,加重炎症反应,但目前研究结果不一致[26-27]。西方患者合并肥胖的比例可能更高,国内尚缺乏相应数据[28-30]。肥胖症或肥胖伴少肌症在儿童IBD较多见,成人的研究相对较少。内脏脂肪含量与CD疾病活动度及炎症反应水平显著相关,并影响机体对生物制剂的反应性[31-33]。内脏脂肪增多的CD患者术后并发症(尤其是感染并发症)发生率明显增加,且术后更易复发[34-36]。

3. IBD患者常合并微量营养素缺乏,应予关注。推荐等级:推荐

人体所需营养物质包括宏量营养素和微量营养素,宏量营养素包括水、电解质、碳水化合物、氨基酸和脂肪酸,微量营养素指维生素和微量元素。IBD患者受膳食摄入不足、肠道(尤其是回肠)炎症反应以及药物干扰等因素的影响,容易合并微量营养素缺乏,病史长或者手术后患者尤其明显[37-38]。微量营养素缺乏在IBD活动期和缓解期均可发生[39-40]。处于疾病缓解期或宏量营养素水平正常(营养状况正常)的患者亦可能存在微量营养素缺乏[3]。

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CD常累及回肠，甚至需要切除回肠，而回肠是脂肪和脂溶性维生素吸收的主要部位，所以IBD患者尤其是CD患者常合并脂溶性维生素缺乏，其中低维生素D水平十分常见[41-42]。一般将血清25-OH维生素D＜75 nmol/L定义为维生素D不足，＜50 nmol/L定义为维生素D缺乏[43]。研究证实，近70%结肠切除术后的UC患者血清25-OH维生素D_3水平低于31 ng/ml(77.5 nmol/L)[44]。高达30%的回肠造口的IBD患者骨密度降低[45]。除回肠因素外，使用糖皮质激素、日照时间、活动量、生活习惯、肥胖、吸烟等均影响维生素D水平[46]。目前尚不明确维生素D缺乏与IBD发病及严重程度之间的因果关系。

约22%的CD患者和25%的UC结肠切除患者存在维生素B_{12}缺乏，80%的IBD患者有叶酸缺乏[47-48]。回肠病变(＞30～60 cm)、末端回肠切除(＞20 cm)以及治疗药物等均可影响维生素B_{12}和叶酸吸收[49-50]。缺铁性贫血在IBD亦相当普遍[51]。由于结肠溃疡失血等原因，UC患者缺铁性贫血发生率较高；即使结肠切除术后，其贫血发生率也达20%以上；超过50%的合并储袋炎的UC患者有铁缺乏[52-53]。贫血会导致疲劳，影响患者生活质量[54]。

腹泻会造成IBD患者不同程度的钾、镁、钙和磷丢失。约10%的CD患者会出现锌缺乏[55]。儿童CD缺锌现象更普遍[56]。锌缺乏的IBD患者预后差，补充锌能够降低CD风险[57-58]。

二、IBD患者的营养及能量需求评估与供给

4. 对IBD患者要常规进行营养风险筛查和营养状况评定。推荐等级：强烈推荐

营养风险(nutritional risk)是指现存或潜在的与营养因素相关的导致患者出现不良临床结局的风险，对具有营养风险的患者进行营养支持治疗能够改善临床结局[59]。随着疾病严重程度的加剧，具有营养风险的IBD患者数量显著增加[60-61]。营养风险筛查工具有多种，最适合IBD患者的筛查工具尚不明确。本共识推荐目前应用最广泛的营养风险筛查工具2002(NRS2002)[62]。NRS2002评分≥3分提示有营养风险，需要进行营养支持治疗[62]。

营养状况评定包括主观与客观两个部分。本共识推荐患者整体营养状况评估表(scored patient-generated subjective global assessment，PG-SGA)作为营养状况主观评定工具。PG-SGA将营养状况分为重度营养不良(≥9分)、中度营养不良(4～8分)和营养正常(0～3分)[63]。客观部分包括静态和动态两类测定指标。静态指标指人体测量指标，包括身高、体质量、BMI、机体组成、三头肌皮褶厚度、上臂肌围及其他用于评估慢性营养不良的指标；动态测定指标包括氮平衡和半衰期较短的内脏蛋白如前白蛋白等。血浆总蛋白和白蛋白半衰期较长，结果受多种因素影响，作为疾病急性期机体营养状况的评价指标不够敏感[64-65]。氮平衡是可靠且常用的动态评价指标，有条件的医院可以使用[66]。

IBD患者在初诊时应常规进行营养风险筛查。对筛查出的有营养风险的患者应进行营养状况评定，确定营养治疗方案，并给予营养支持治疗。病情变化可以影响患者营养状况和代谢状态，合并感染或使用糖皮质激素、饥饿、肠梗阻或肠瘘等均能恶化患者的营养状况和代谢状态，因此在治疗期间应动态监测患者的营养状况，并根据监测结果调整营养支持治疗方案。

5. 根据患者的需求确定能量和蛋白质供给量。推荐等级：强烈推荐

有关IBD能量消耗的研究不多。有研究认为，IBD并不增加静息能量消耗(resting energy expenditure，REE)，虽然疾病活动期REE可能增加，但由于患者活动量减少，抵消了炎症反应活动增加的REE[67]。因此，对缓解期和轻中度活动期疾病，可以沿用正常人的能量供给[68-69]。但极度营养不良、重症UC或CD患者的REE有别于正常人：体温每升高1 ℃，CD患者的REE增加10%～15%，合并脓毒症时REE约增加20%[70]。活动期CD的能量消耗约高出缓解期8%～10%，因此对重症患者应采用间接能量测定的方法，个体化确定患者的能量需求[71]。动态评估REE能够为IBD患者的精准营养支持治疗提供依据[72]。

儿童和青少年IBD患者处于生长发育期，摄入的营养除满足正常代谢需要外，还要增加追赶同龄人身高和体质量的营养需求，因此，每日提供的能量应为正常儿童推荐量的110%～120%，以避免能量供给不足造成蛋白质分解供能[73]。

IBD患者蛋白质代谢受摄入量、肠道消化和吸收能力、肠道炎症反应、全身炎症反应和使用糖皮质激素等因素的影响。缓解期IBD患者蛋白质需要量与普通人相似($1.0\ g\cdot kg^{-1}\cdot d^{-1}$)，活动期蛋白供给应达到$1.2\sim1.5\ g\cdot kg^{-1}\cdot d^{-1}$)[74-75]。

6. 应定期检测并及时纠正IBD患者微量营养素水平。推荐等级：推荐

营养支持治疗能够提供一部分微量营养素，但可能不足，因此应定期评估患者微量营养素水平，对不足者予以针对性补充。每日口服复合维生素制剂能够纠正大部分患者的维生素缺乏，但对于维生素D、锌、铁缺乏可能需要有针对性的纠正[76]。

维生素D不但参与骨代谢，还与IBD肠黏膜炎症反应程度和并发症有关[77-79]。研究证实，纠正维生素D缺乏能改善IBD病程，抑制CD炎症反应甚至减少手术率，降低复发率，提高药物治疗效果[80-84]。富含维生素D和锌的膳食甚至可能预防CD发生[85]。上述观点虽然尚未得到普遍认同，但低维生素D现象不会因为IBD炎症反应得到控制而自行改善，必须通过补充维生素D纠正[86]。活动期及使用糖皮质激素的IBD患者更应定期监测并纠正25-OH维生素D和血钙异常，以保持正常的血清25-OH维生素D水平[67]。

CD患者应每年1次或必要时(如未使用硫唑嘌呤者出现巨红细胞增多症时)检测血清维生素B_{12}和叶酸水平[87]。末端回肠切除(或合并回盲部切除)＞20 cm时，应每月预防性补充1 mg维生素B_{12}；如果此类患者已有维生素B_{12}缺乏，应每天或隔天肌注维生素B_{12} 1 mg，7 d后改为每周肌注1 mg，持续4～8周，然后每月注射1 mg或每天口服1～2 mg，终生

补充[88]。服用柳氮磺吡啶或甲氨喋呤(methotrexate,MTX)的IBD患者建议补充叶酸。对使用甲氨喋呤的儿童,服药后24~72 h应口服叶酸5 mg,或每周连续5 d服用叶酸1 mg/d[89]。

缺铁性贫血的IBD患者都应补充铁剂,补充目标为血红蛋白(Hb)水平和铁贮备恢复正常。轻度贫血(女性Hb 100~119 g/L,男性Hb 110~129 g/L)、疾病缓解期、既往无口服铁剂不耐受的患者首选口服补铁;Hb<100 g/L、疾病活动期、既往对口服铁剂不耐受或正在使用促红细胞生成素的患者建议静脉补铁;静脉补铁能够快速纠正铁缺乏及贫血状态,并避免口服铁剂对肠道的刺激及潜在的加重或诱发疾病活动的不良反应[90-91]。对于慢性病贫血,在静脉补铁的同时可以使用促红细胞生成素。Hb<70 g/L时可以考虑输注红细胞,并静脉补铁[87]。每日补铁量不宜超过100 mg。动物实验结果表明,口服过量铁(尤其是硫酸亚铁)能够催化氧自由基形成,造成肠上皮氧化应激损伤,加重肠道炎症反应;过量的铁还会增加$CD8^+/CD4^+$ T细胞比例,诱导内质网应激,甚至改变肠道菌群[92]。红肉中含较多的铁,进食过多红肉对IBD的不利影响可能与上述机制有关[93]。

三、IBD患者营养支持治疗的目的和作用

7. 营养支持治疗能够诱导CD缓解,并可能有助于维持缓解。推荐等级:推荐

从上世纪70年代开始,随着太空饮食(space diet,也称为要素饮食,elemental diet)的发明并应用于临床,人们发现要素饮食具有和糖皮质激素相当的诱导CD缓解的临床效果[94]。后来进一步证实,无论是以氨基酸单体为氮源的肠内营养(enteral nutrition,EN)制剂(即要素饮食),还是以短肽或整蛋白为氮源的EN制剂均有类似的治疗作用[3]。目前认为,全肠内营养(exclusive enteral nutrition,EEN,也称total enteral nutrition,TEN)能够诱导成人CD缓解,但疗效不如糖皮质激素或生物制剂,可能是成人对EEN依从性差有关,同时,EEN对不同部位CD诱导缓解的效果可能有所差别[95-100]。但是,对于合并营养不良或有营养风险的患者,或不适于使用糖皮质激素或生物制剂的患者,以及围手术期患者,EEN是最佳选择[101-102]。部分患者考虑到药费和药物不良反应等因素,也倾向于选择使用EEN诱导CD缓解。

EEN诱导CD缓解的机制不明,可能与EN组成(如复杂碳水化合物、脂肪酸构成、维生素和微量元素)合理、抗原负荷少、有助于短链脂肪酸(short-chain fatty acid,SCFA)产生,以及调整肠道微生态平衡(如拟杆菌/普雷沃菌比例)改善菌群结构,保护肠黏膜屏障等机制有关[103-110]。近年来,内脏脂肪在CD发病过程中的作用日益受到重视。EEN能够减轻内脏脂肪堆积,改变系膜脂肪结构,或许与诱导CD缓解作用机制有关[31,111]。

长期EN能够维持CD缓解,延缓复发,但相关证据尚不充分[112-114]。EN维持CD缓解有效的证据多来自日本,来自西方的结果多为无效,其原因不明。

8. EEN能够促进肠黏膜溃疡愈合。推荐等级:推荐

EEN诱导CD缓解后,肠黏膜炎症反应消退,溃疡能够愈合,其疗效优于糖皮质激素[115-116]。黏膜愈合是EEN治疗后CD长期维持缓解的重要原因,通过EEN达到完全黏膜愈合的患者3年复发率显著低于没有达到完全黏膜愈合者[117]。EEN达到黏膜愈合至少需要8周以上[118],治疗12周的黏膜愈合率可达到47%[96];8周的完全黏膜愈合率为33%,接近完全黏膜愈合率达19%。与此相比,6~12周的生物治疗黏膜愈合率只有28%[117,119]。

9. 营养支持治疗能够促进CD儿童和青少年生长发育。推荐等级:强烈推荐

生长发育迟缓甚至停滞在儿童和青少年IBD(尤其是CD)中相当普遍,男孩多于女孩,约有30%的IBD儿童初发症状为生长发育迟缓。生长发育迟缓对儿童的主要影响在于身高增长较同龄人缓慢,其中约半数到成年时仍身材矮小[120]。

营养不良是生长发育迟缓的原因之一,但不是唯一原因,单纯纠正营养不良能够改善患儿营养状况,增加体质量,但不一定促进身高增长。活动性炎症反应造成的下丘脑-垂体-性腺轴和生长激素(GH)/胰岛素样生长因子(IGF)-1轴分泌减少是生长发育迟缓的直接原因,循环血中肿瘤坏死因子(TNF-α)也影响长骨骨骺端的生长[121-122]。EEN由于同时具有补充营养和诱导活动期CD缓解的作用,在改善营养状况的同时能够减轻CD炎症反应程度,促进生长发育,所以,EEN是儿童和青少年CD首选的治疗手段[123]。研究表明,12周EEN能够提升CD患儿骨小梁密度Z分值,增加骨骼肌含量,并增加BMI Z分值[124-125]。多项研究也表明,EEN促进儿童和青少年CD患者生长发育的效果优于糖皮质激素,具有不可替代的优势[126-128]。

预防IBD儿童和青少年生长发育迟缓的关键是对生长发育情况进行定期监测,及时发现并采用EEN甚至手术等有效措施积极控制活动性炎症反应,避免低IGF-1,纠正营养不良能够促进身高增长和生长发育[129-130]。

10. EN能够促进IBD手术患者康复。推荐等级:强烈推荐

加速康复外科(enhanced recovery after surgery,ERAS)理念在外科领域已深入人心,其核心是通过减轻手术患者应激反应,促进术后康复,这一理念同样适用于IBD手术患者[131-133]。影响IBD患者术后康复的关键是手术并发症的风险因素。需要手术的IBD患者由于病情重,肠道有狭窄或穿透性并发症等原因,营养状况较一般IBD患者更差[134-135]。营养不良是手术并发症的独立风险因素[136]。IBD患者在择期手术前应进行营养风险筛查和营养状况评定,对有营养风险或营养不良的患者先进行营养支持治疗,待营养风险下降、营养状况得到纠正后再手术,能够提高手术安全性,减少手术并发症[137-138]。对于存在营养不良、合并感染或使用糖皮质激素等免疫调节剂的患者,EEN在改善营养状况的同时,能够诱导CD缓解,有助于控制感染、撤除糖皮质激素以及消除糖皮质激素对手术的不利影响[139]。上述理念也称预康复[140]。

IBD患者术后肠麻痹(ileus)发生率高[141]。术后早期EN是ERAS的重要内容,不仅能够促进肠道运动功能恢复,改

善营养状况，而且有助于维护肠黏膜屏障功能，降低感染发生率，缩短术后住院时间[131]。

11. 营养支持治疗UC的目的在于改善营养状况。推荐等级：强烈推荐

营养支持治疗没有诱导或维持UC缓解的作用，但能够纠正UC患者营养不良或降低营养风险[67,142-143]。UC营养支持治疗首选EN，仅在EN失败或UC合并肠衰竭时使用肠道休息和全肠外营养（TPN）[144]。UC患者需要TPN治疗大多提示病情严重[145]。

四、IBD患者营养支持治疗的临床应用

12. IBD患者营养支持治疗首选EN。推荐等级：强烈推荐

EN不仅能够提供身体所需的营养物质，而且消化吸收途径符合生理状态，能增加门静脉血流量、维护消化道生理功能和肠黏膜屏障。通过EN提供的能量只要达到总能量需求的20%，即可发挥上述作用[146]。因此，营养学界有句名言："只要肠道有功能，就应该使用肠道；即使部分肠道有功能，也应该使用这部分肠道"[147]。所以，营养支持治疗首选EN。

根据摄入量占营养需求总量的比例，EN分为EEN和部分肠内营养（partial enteral nutrition，PEN）。EEN指患者所需的营养素完全由EN提供，没有其他营养来源。EEN可有效诱导活动期CD缓解。PEN指在进食的同时补充EN，以达到增加能量和营养素摄入的目的，多用于纠正营养不良[148-149]。如果通过肠道供能达不到总能量需求的60%，应给予补充性肠外营养（supplementary parenteral nutrition，SPN）。

13. 需要营养支持治疗的IBD患者如果EN禁忌或无法达到有效剂量，应予肠外营养（parenteral nutrition，PN）治疗；EN联合PN优于TPN。推荐等级：强烈推荐

营养支持治疗首选EN，如EN禁忌或无法达到有效剂量，应给予PN。考虑到TPN相关的并发症风险，营养风险高（NRS2002≥5分）或重度营养不良的IBD患者，如EN禁忌或无法实施，应在24～48 h内给予TPN；如EN能够实施，但在48～72 h后仍无法达到60%以上能量及蛋白质需求时，供给不足部分由SPN补充[67,150]，当EN提供的能量超过所需目标量的60%时可以停用PN[149,151-152]。营养风险低（NRS2002≤3分）或轻中度营养不良的IBD患者只有在预计营养摄入受限超过7 d时才给予TPN。

IBD患者给予TPN的常见临床情形：（1）CD继发短肠综合征早期有严重腹泻；（2）高流量小肠瘘（流量＞500 ml/d）且EN无法维持水电解质及营养平衡；（3）因肠梗阻无法实施EN；（4）高位肠内瘘（如胃或十二指肠-结肠内瘘）且无法实施EN；（5）肠瘘继发腹腔感染未得到控制；（6）不耐受EN的其他情形，如重症UC或其他原因造成的严重腹胀或腹泻，严重的肠动力障碍；（7）无法建立EN通路。

14. 治疗儿童和青少年CD首选EEN。推荐等级：强烈推荐

85%的CD儿童出现体质量下降，15%～40%的儿童出现生长发育停滞。EEN用于儿童和青少年CD，不但能够有效地纠正营养不良，而且可以促进骨密度增加和身高增长[127,153-155]。EEN诱导儿童和青少年活动期CD缓解的疗效与糖皮质激素相当，诱导缓解率可达80%。同时，EEN还能提高深度缓解率和黏膜溃疡愈合率，改善预后，其效果优于糖皮质激素，与生物制剂相仿或高于生物制剂，但不良反应更少[115,117,126,156-158]。

EEN的疗效与治疗时间有关，诱导儿童和青少年CD缓解的推荐疗程为6～8周，促进黏膜愈合至少为12周，治疗生长发育迟缓需时更长[115]。同时，EEN和PEN的疗效也有差别：EN占总能量摄入50%时，诱导缓解的效果只有EEN的1/3；占80%～90%时，诱导缓解率为65%，与EEN类似[159-160]。

鉴于EEN的疗效及糖皮质激素和免疫抑制剂的潜在不良反应，治疗儿童和青少年CD首选EEN[89,127]。

15. 诱导成人CD缓解或术前预康复时，应采用EEN。推荐等级：推荐

对于依从性良好的成人CD，EEN的诱导疾病缓解率与糖皮质激素相似，并可促进肠黏膜愈合[96,161]。EEN治疗过程中应及时评估CD疾病活动情况，适时添加维持缓解药物，从而转换到药物维持缓解。EEN诱导CD缓解后如何过渡到普通饮食尚无一致意见，通常在诱导缓解过程中开始服用维持缓解药物，药物起效后2～3周内逐渐撤减EN并过渡至普通饮食[143,162]。

CD术前预康复的目的包括改善营养状况、控制感染、诱导缓解、撤减糖皮质激素/生物制剂和戒烟等[135]。择期手术的CD患者多数需要进行预康复，并以C-反应蛋白（CRP）恢复正常（＜8 mg/L）、营养状况改善（血清白蛋白＞35 g/L）和停用可能增加手术并发症的药物作为预康复的终点[137,163]。如果患者使用生物制剂（如英夫利西单克隆抗体），术前需至少停用8周以上；使用糖皮质激素（连续使用20 mg/d或以上等效剂量泼尼松＞6周），术前至少停用4周以上[164-165]。为维持停用药物这段时间CD病情相对稳定，通常予EEN治疗[139,166]。单纯以改善营养状况为目的时，根据营养摄入量的大小可采用PEN或EEN，无法给予EN时可使用TPN，时间一般不少于7～14 d[136]。对合并肠梗阻或肠瘘的患者，口服EN可能加重肠道症状，应选择管饲。

16. 以维持CD缓解为目的时，可采用EEN或PEN。推荐等级：推荐

营养支持治疗可能有助于维持CD缓解，但长期EN受到患者依从性的影响，用于维持CD缓解的EN使用量、疗程及联合用药方案等也缺乏一致意见。在EN用于维持CD缓解的研究报道中以PEN居多。PEN方案要求患者每日需求的总能量的50%以上由PEN提供[167-168]。PEN方法：（1）在正常进食基础上口服营养补充（oral nutritional supplement，ONS）；（2）白天进食低脂饮食，夜间鼻饲；（3）每4个月中进行1个月的EEN；（4）EN联合英夫利西单克隆抗体维持CD缓解[169-171]。荟萃分析结果显示，与普通正常饮食相比，PEN可以有效减少CD复发，其作用优于某些药物（如糖皮质激素和5-氨基水杨酸制剂）[114]。

17. 长期营养支持治疗有助于短肠综合征患者维持 CD 疾病缓解。推荐等级：强烈推荐

CD 是导致短肠综合征的常见病因。禁食联合 TPN 虽然能够满足患者对营养的需求，减轻短肠患者腹泻，但不利于肠功能代偿。通过 EN 维持 CD 缓解的观点虽然没有得到普遍认可，但对于合并短肠的 CD 患者来说，这一思路值得借鉴。根据保留小肠长度、功能和患者的耐受情况适量管饲 EN，不但有利于维持患者营养状况，促进肠功能代偿，而且有助于诱导和维持 CD 缓解，推迟复发[172]。对于无法耐受 EEN 的短肠患者，可以采用 PEN 联合 SPN 的方案满足患者对营养的需求，如肠道完全无法使用时才给予 TPN[67]。

18. CD 合并肠狭窄时不应放弃 EN。推荐等级：强烈推荐

肠狭窄是 CD 最常见并发症。合并肠狭窄的 CD 患者多存在营养不良，需要进行营养支持治疗。肠狭窄分为炎性狭窄和纤维性狭窄。经营养支持治疗诱导缓解后，大多数炎性狭窄患者的症状可以改善，但纤维性狭窄仍需要外科处理。临床工作中很难界定炎性与纤维性狭窄，二者常共存，因此应充分评估，了解肠狭窄性质（炎性或纤维性）、程度（有无肠梗阻及梗阻程度）、部位及有无闭袢或肠绞窄等，再决定采用 EN 或 PN[173]。营养支持治疗过程中应动态观察病情变化，及时调整治疗方案。EN 诱导炎性狭窄症状缓解后，根据具体情况可以恢复进食并使用药物维持治疗；部分患者在营养状况改善后，可行内镜或手术治疗肠狭窄[174-175]。

轻度肠狭窄可以选择 ONS 或管饲 EN，中、重度肠狭窄推荐采用肠内营养输注泵持续管饲，以免加重梗阻症状。如果通过管饲仍无法达到 EEN，对于内镜可及的狭窄（如食道或幽门/十二指肠狭窄），可以将肠内营养管送至狭窄远端给予 EEN；对内镜不可及的狭窄，可以采用 PEN 联合 SPN 或者 TPN 联合药物（如糖皮质激素）诱导缓解，待狭窄症状改善后再向管饲 EEN 过渡；如 TPN 治疗 7 ~ 10 d 后肠梗阻症状仍不能缓解，应权衡继续 TPN 还是手术建立 EN 途径或解除肠梗阻的风险与获益。对于高位肠梗阻可以考虑在梗阻远端行空肠插管造口，低位梗阻可以在梗阻近端肠造口，为实施 EN 创造条件。待营养不良得到纠正、一般状况改善后再进行确定性处理，如内镜下狭窄扩张或手术[175-176]。

考虑到 PN 的潜在风险，对轻中度营养不良的患者，如果不能进行 EN，预计禁食时间 < 1 周可以不进行 PN，预计禁食时间 > 1 周则进行 TPN。对于严重营养不良、营养风险 > 5 分、或者一直依赖营养支持治疗的患者，如果无法实施 EN，应及时给予 TPN，待肠梗阻症状改善后，再通过 PEN 联合 SPN 逐渐向 EEN 过渡[177]。

19. CD 合并腹腔/腹膜后脓肿及肠外瘘时，积极引流脓肿和减少消化液丢失有利于实施 EN。推荐等级：强烈推荐

腹腔/腹膜后脓肿是 CD 的严重并发症。经皮脓肿置管引流是其最有效的治疗方法。无法置管或置管引流失败时，脓肿较大者应行手术引流，脓肿较小（< 3 cm）者可在密切观察下使用抗生素治疗[178-179]。此时应慎用 CD 的治疗药物如糖皮质激素及生物制剂，以免加重感染[96,180]。

腹腔/腹膜后脓肿大多是肠壁穿透性病变即肠瘘导致。肠外瘘不是 EN 的绝对禁忌证，其改善营养状况的疗效优于 PN，在充分引流的前提下应首选 EN[67]。如果瘘口较小，可以直接进行 EN，但应注意观察瘘口的变化，以免肠液增多加重腹腔感染。如果进行 EN 后感染加重，应调整脓腔引流管的位置、管径，或更改为负压吸引，做到充分引流脓肿，再开始 EN，并逐渐过渡至 EEN[181]。某些单纯性小肠瘘经 EN 或 PN 治疗后有可能自愈，避免手术[182]。

明确瘘口解剖部位和肠液漏出量对制定营养支持治疗方案至关重要。如果瘘口位置较高，可以将肠内营养管置入瘘口以下肠段进行 EN；如果瘘口位置较低，可经鼻置管使用瘘口以上的胃肠道进行 EN，原则是把尽可能多的肠管利用起来；如果瘘口在小肠中段，也可以经上消化道进行 EN，并及时将排出的肠液收集起来，经瘘口再回输入远端的消化道。肠液排出量≥500 ml/24 h 时应尽量回输，不但有利于维持水电解质平衡，而且能够有助于营养物质的消化和吸收[179,183]。如果消化液无法收集或丢失过多（≥500 ml/24 h），应在 EN 的同时密切关注水电解质平衡的变化，必要时给予 SPN。

轻中度营养不良的患者，如果预计感染可以在 1 周内得到有效引流，并耐受 EN，可不必给予 PN[152]。严重营养不良或消化液丢失量大的患者，如果不能耐受 EN，应禁食并及时给予 TPN，不仅可以改善患者营养状况，而且能减少肠液丢失，有利于肠瘘愈合[184]。

20. CD 合并肠内瘘的营养支持治疗方案取决于瘘口解剖部位、大小及旷置肠管长度。推荐等级：强烈推荐

CD 合并肠内瘘患者实施 EN 的关键在于克服肠道结构异常。通过影像学检查找到能够用于 EN 的肠段，再通过内镜等技术，采用适宜的管饲途径，多能够成功实施 EN。

高位内瘘（如胃或十二指肠-结肠内瘘）且瘘口较大引起短路症状者，推荐置营养管至瘘口以下空肠进行 EN[67]。旷置肠段较短或瘘口较小的肠-肠内瘘者，如果短路症状不明显，可以按照一般原则给予 EN。肠-膀胱瘘及肠-阴道瘘者，如漏出量不大，症状不严重，使用低渣肠内营养制剂进行 EN，同时口服喹诺酮或咪唑类抗生素可以改善感染症状；如果症状严重，可考虑先行转流性肠造口，既有助于进行 EN，又能有效控制感染和肠道症状[185]。肠内瘘和狭窄常同时存在，在实施 EN 过程中应避免加重肠梗阻，如果 EN 不能全量供能，可以进行 SPN。通过营养支持治疗纠正营养不良并诱导 CD 缓解后，部分肠内瘘患者瘘口能够闭合，但大多数患者仍需要手术治疗，此时进行确定性手术可显著改善手术结局[186]。

五、IBD 患者营养支持治疗的实施与监测

21. 由营养支持小组执行营养支持治疗。推荐等级：强烈推荐

营养支持小组（nutrition support team，NST）由多学科专业人员构成，包括医师、营养师、护士、药剂师等，其主要职责是承担营养风险筛查与评价，制定、实施营养支持治疗方案并监测治疗效果，指导家庭营养支持治疗等任务[187]。研究

显示，NST的参与可以降低营养治疗相关并发症，提高营养支持治疗效果[188]。

22. 长期营养支持治疗可以在家庭实施。推荐等级：强烈推荐

病情相对稳定且需要长期营养支持治疗的患者可以实施家庭营养支持治疗。家庭营养支持治疗可以让患者回归家庭，提高生活质量，减少医源性感染和医疗费用，提高医疗资源的使用效率[189]。

家庭营养支持治疗分为家庭肠内营养（home enteral nutrition，HEN）和家庭肠外营养（home parenteral nutrition，HPN）。HEN多采用管饲。导管管理不善是常见的HEN并发症。对于肠道耐受较好、使用PEN的患者也可以采用口服的方式，其优点是简便易行，符合生理，患者依从性好。HPN是肠衰竭（短肠综合征）患者长期营养支持治疗、维持生命的重要途径，由于其对技术和设施的要求较高，营养液配制过程应在设施完备的医疗机构进行，接受HPN的患者及家属需要进行严格的培训[190]。TPN相关并发症是患者死亡的主要原因，尤其在使用的最初2年要密切监测，积极处理[191-192]。为尽量减少营养支持治疗相关并发症，提高疗效，家庭营养支持治疗需要在NST的监督指导下进行[193]。

23. 应重视对医护人员和IBD患者的相关宣教，以提高患者对营养支持治疗的依从性和治疗效果。推荐等级：强烈推荐

依从性不高是IBD患者实施EN过程中经常遇到的问题。EN时间越久，依从性对疗效的影响越大。EEN诱导成人CD缓解疗效不好可能与依从性欠佳有关[194-195]。除患者对管饲的恐惧和长期禁食的抵触外，医护人员对EN重要性认识不足、对治疗过程缺乏耐心、对疗效缺乏信心也是导致患者依从性差的原因[196]。加强对医护人员和患者的相关宣教，促进医患及患者之间的相互交流，制定并严格执行标准化的营养支持治疗流程，有利于提高患者对EN的依从性。管饲能够提高患者对EEN的耐受性，改善治疗效果[197-198]。

24. 根据病情需要选用不同剂型的EN制剂。推荐等级：强烈推荐

要素饮食（氨基酸单体配方）、短肽（低聚配方）及整蛋白（多聚配方）EN制剂诱导及维持CD缓解的效果并无明显差别[67]。整蛋白EN价格低廉，口感好，但由于氮源来自于整蛋白，适用于消化吸收功能相对健全的患者。要素饮食或短肽EN的氮源来自于蛋白质分解，适用于消化吸收功能不全（如肠道吸收面积减少或各种原因引起的消化吸收功能减退）的患者，但由于其相对分子质量较小，对EN制剂的渗透压影响较大。膳食纤维不但能够给结肠黏膜提供SCFA，而且有助于改善粪便性状，但对合并肠狭窄的患者要慎用，以免加重肠梗阻症状[199]。

25. 根据EN摄入量和病情采取不同的给予方式。推荐等级：强烈推荐

EN摄入方式包括口服和管饲。口服最常用的方式是ONS，适用于添加营养改善营养状况或采用EN长期维持缓解患者。当EN摄入量＜900 ml/d时，多数患者可以耐受ONS；超过这一限度，患者往往出现胃肠道不耐受现象，需要管饲。管饲包括间歇推注、间断滴注和持续输注3种方式。IBD患者由于合并肠狭窄等原因，通常采取持续输注的方式，即在20~24 h内将每日所需的全量营养液持续输入胃肠道。管饲尤其适用于EEN（营养液输注量大）、肠腔狭窄或吸收面积不足的患者，如不全性肠梗阻、肠外瘘或短肠综合征患者[200]。

管饲方法包括鼻胃管、鼻肠管、内镜下胃/空肠造口（percutaneous endoscopic gastrostomy/jejunostomy，PEG/J），以及手术胃/空肠造口等，其中鼻胃管途径最常用。鼻饲管持续放置时间不宜超过4周，时间过长容易压迫鼻黏膜出现溃疡、压迫鼻旁窦开口造成堵塞以及鼻窦炎等并发症。如果持续管饲时间＞4周可选择PEG/J，这项操作并不增加胃瘘风险[201-202]。一般不推荐CD患者做空肠插管造口[203]。

26. 使用输注泵进行管饲能够提高患者的耐受性。推荐等级：强烈推荐

与间歇推注和间断滴注相比，使用输注泵持续输注EN不但减少管饲护理工作量，而且能够准确控制输注速度，按时完成输注量，改善肠道吸收情况，减少EN并发症，提高胃肠道耐受性[197]。

27. 采用“全合一”方式进行PN，并根据病情调整营养配方。推荐等级：强烈推荐

PN时应避免将碳水化合物、脂肪乳剂、氨基酸等分别输注，而应将所有营养成分放在同一容器内，同时输注给患者，此为“全合一”输注方式，其优点是能够提高机体对营养物质的利用效率，减少代谢并发症，降低营养液和输注管路污染的发生率。

“全合一”的总能量构成中，碳水化合物供能应占50%~70%，其余能量由脂肪乳剂供给，约为30%~50%。碳水化合物比例过高容易产生糖代谢紊乱、CO_2潴留、肝内胆汁淤积等并发症。脂肪乳剂的主要作用是提供能量和必需脂肪酸，主要成分为多不饱和脂肪酸（polyunsaturated fatty acids，PUFA），不同成分的脂肪酸具有不同的免疫调节功能。n-6 PUFA是脂肪乳剂的主要成分，但其代谢产物具有加剧炎症反应的作用，不宜做为脂肪酸的唯一来源，而应添加促炎作用很弱的鱼油脂肪乳剂（主要成分为n-3 PUFA）、橄榄油脂肪乳剂（主要成分为n-9单不饱和脂肪酸，n-9 monounsaturated fatty acids，n-9 MUFA）、或不影响炎症反应并且能够快速供能的中链甘油三酯（medium-chain triglyceride，MCT）[204]。研究证实，添加n-3 PUFA的TPN对活动期CD可能具有诱导缓解、减少术后感染风险、缩短术后住院时间的作用[205-209]。目前尚无证据支持静脉给予谷氨酰胺二肽对IBD活动度具有调节作用[136,210]。

对于高分解代谢或TPN早期（1周内）患者，建议采用容许性低热卡、高蛋白配方[总能量≤20 kcal·kg^{-1}·d^{-1}（1 kcal=4.184 kJ）或每日提供总能量只占预计需要量80%，蛋白质≥1.2 g·kg^{-1}·d^{-1}]，以免加重脏器代谢负担[67,211]。

28. 使用PICC或中心静脉导管输注TPN，SPN可由周围静脉通路输注。推荐等级：强烈推荐

与周围静脉通路相比，中心静脉管径粗，血流量大，不易产生静脉炎，适用于输注高浓度或大容量营养液如TPN。与锁骨下静脉穿刺置管术相比，经外周静脉穿刺中心静脉置管术（peripherally inserted central catheter，PICC）更安全，是输注TPN首选途径[212]。颈内静脉或股静脉穿刺置管术的穿刺口容易污染，股静脉置管易形成血栓，均不建议用于输注TPN。

在B超引导下放置中心静脉导管可提高置管安全性。置管成功后应进行X线检查，确定导管尖端位置合适并排除置管并发症后才可使用。建议采用单腔静脉导管输注营养液，其优点是内径粗、阻力小、接口少和污染机会少。SPN液体量一般较小，浓度低，使用时间较短（<10～14 d），可考虑经周围静脉输注，但也应警惕发生血栓性静脉炎[212]。

29. 营养支持治疗过程中应动态评估治疗效果。推荐等级：强烈推荐

IBD营养支持治疗的作用是多方面的，为达到不同的治疗目的，应动态评估治疗效果，及时调整治疗方案。以诱导CD缓解为目的时，应在治疗开始和终止时评价CD活动度，包括CD活动指数（Crohn′s disease activity index，CDAI）、血清CRP和粪便钙卫蛋白等，各项检测指标的临床意义有所不同，但均有利于明确营养支持治疗的终点，及时切换到其他治疗方案。活动期CD是手术后并发症的风险因素[213]。术前通过EEN进行预康复不但能够改善营养状况，而且能够诱导疾病缓解。动态监测疾病活动等相关指标有助于准确把握手术时机，减少术后并发症，推迟术后复发[163,214-215]。由于患者对EN的依从性和肠道耐受性不同、营养摄入途径不同（管饲或ONS）等原因，治疗效果有所差别。动态评估疗效有助于及时发现问题，提高疗效。

30. 营养支持治疗过程中应密切监测相关并发症。推荐等级：强烈推荐

EN并发症包括胃肠道并发症（腹泻、腹胀、恶心、呕吐等）、代谢并发症（水电解质平衡异常、血糖波动等）、感染并发症（吸入性肺炎、营养液污染等）及导管相关并发症（鼻窦炎、鼻咽部黏膜损伤、造口旁瘘、营养管堵塞或易位、营养管错误连接等）[216]。IBD患者因肠道炎症反应、肠狭窄及肠瘘等原因，出现EN并发症的风险高于普通患者。EN并发症重在预防，实施过程中必须遵循相关规范[217]。

管饲是常见的营养途径，盲法放置的鼻饲管应通过X线等影像学手段证实位置合适后才可使用。有胃排空障碍或误吸风险（如幽门、十二指肠或高位空肠狭窄）时，推荐将导管放到狭窄以远进行管饲，从较低速度（10～15 ml/h）开始输注，再根据患者耐受程度逐渐增加至目标量[218]。为避免返流，卧床重症患者应采取头高位（15°～30°）；高危患者应定时监测胃排空情况，以免发生误吸[219]。输注过程中缓慢增加输注量、保持营养液合适温度、防止营养液污染等措施能够减少胃肠道并发症，提高患者耐受性。

PN并发症包括导管相关并发症（穿刺损伤、导管异位、导管堵塞或折断、空气栓塞、血栓形成等）、感染并发症（导管相关感染、营养液污染等）、代谢并发症（血糖波动、水电解质紊乱、微量元素和维生素缺乏、脂代谢异常及高氨血症等）、脏器功能损害（如PN相关性肝损害）等。部分并发症可以通过严格遵循相关规范加以预防，但有些并发症如脏器功能损害原因尚不十分清楚，防范措施是积极使用EN[212]。

再灌食综合征（refeeding syndrome）是重度营养不良患者营养支持治疗初期的严重并发症，病死率高，治疗效果差。该并发症重在预防，具体措施是准确识别高危患者，对重度营养不良者要密切监测血磷、维生素B_1、烟酸等微量元素和维生素水平，在补充宏量营养素之前先重点纠正微量营养素和维生素缺乏[220]。IBD患者血栓发生率高，在实施营养支持治疗时应注意预防。

六、膳食及体育锻炼对IBD的影响及建议

31. 复杂碳水化合物、红肉或含添加剂的肉制品、含硫/硫酸盐/硫氨基酸的食品和饮料等可能与IBD有关。推荐等级：建议

膳食对IBD发病及临床症状的影响一直是关注的重点，即使是孪生兄弟或姐妹，膳食结构的差异也会显著影响IBD的发生[221]。但受到食物的多样性、食物成分相互作用以及食物与肠道微生态之间的复杂关系等多方面因素的影响，一直缺乏大规模高质量的临床研究。因此，目前对膳食与IBD关系的认识均是基于小样本或实验研究结果，临床证据尚不充分[222]。

复杂碳水化合物是关注的焦点之一，由于双糖和多糖在小肠难以吸收，渗透压高，进入结肠后迅速被细菌发酵，某些碳水化合物如精炼糖、麦胶及某些淀粉经发酵后还会产气，刺激肠道分泌黏液，促进致炎细菌生长，因此有学者提出了剔除饮食（exclusion diet）的膳食思路，避免食入除单糖以外的其他碳水化合物膳食[223]。文献提及较多的包括可发酵的低聚糖、双糖、单糖、多元醇（fermentable oligosaccharides，disaccharides，monosaccharides and polyols，FODMAP）含量低的饮食、特殊碳水化合物饮食（special carbohydrate diet，SCD）和旧石器时代饮食等，有研究证实上述膳食能减轻CD症状和炎症反应程度[224-225]。

膳食纤维分可溶性和不溶性两种。可溶性膳食纤维和抗性淀粉能在结肠发酵成SCFA，为结肠黏膜提供能量，并具有抗炎、调节免疫等作用，能够强化肠屏障，减少细菌易位，促进肠蠕动和益生菌生长，调节肠道免疫耐受，降低轻中度IBD疾病活动度及粪便炎症反应指标，预防疾病复发，膳食纤维摄入不足增加CD风险[226-240]。不溶性膳食纤维对IBD的影响尚不明确，虽然能增加粪便含水量，但有可能加重肠道梗阻症状，所以对于合并肠道狭窄的CD患者应予限制[241-243]。

有研究认为饮用牛奶可能会降低CD的发病率[244]，但摄入过多富含胱氨酸的食物如红肉、禽蛋类、奶类，以及含添加剂（如亚硫酸盐、卡拉胶、硫酸软骨素等）的食品和饮料会增加硫的摄入，硫经肠道内硫酸盐还原菌代谢后产生硫化氢，

可以加重肠黏膜炎症反应和临床症状[245]。

32. 适当控制脂肪的摄入。推荐等级:推荐

膳食脂肪的摄入量及脂肪成分是影响IBD发病的重要因素。近年来,饮食习惯的改变尤其是饮食西方化造成动物脂肪、食用油和人造奶油等富含n-6 PUFA的食品摄入过多,而n-3 PUFA摄入不足,这一现象与全球IBD发病率升高有关[246]。

n-6 PUFA是能量和必需脂肪酸的重要来源,但其代谢产物为促炎性介质,长期服用能够增加患IBD的风险,尤其是UC[247-248]。而n-3 PUFA的代谢产物促炎作用弱,中链甘油三酯代谢后对炎症反应没有影响。有研究表明,适当调高膳食当中n-3/n-6 PUFA的比例(比如服用富含n-3 PUFA的鱼油)可能降低患IBD的风险,降低UC的疾病活动度,下调活动期CD炎性因子表达,延长UC缓解时间,减少糖皮质激素的用量[249-258]。添加n-3 PUFA能够提高EN诱导活动期CD缓解的疗效[259]。

上述研究虽然在理论上成立,并且得到部分临床研究的证实,但尚缺乏大规模、高质量临床研究的支持,长期口服补充n-3 PUFA维持CD或UC缓解的研究也没有得到预想的结果,因此关于饮食中不同脂肪含量和成份对IBD的影响尚难下结论[67,260-262]。但限制脂肪尤其是n-6 PUFA的摄入量,少食红肉、人造脂肪和食用油可能会降低罹患IBD的风险,对IBD患者有益[85,230,263-264]。不过,长期严格限制脂肪摄入要当心必需脂肪酸缺乏。

虽然EN的成分并不影响其疗效,最新的荟萃分析也未证明脂肪含量与诱导缓解率之间存在明显相关性[262]。但EN中脂肪所提供的热量占总热量的比例不宜超过40%,低脂EN似乎能够取得更好的诱导CD缓解的效果[85,248,263-264]。

33. 增加新鲜水果和蔬菜的摄入。推荐等级:推荐

新鲜蔬菜和水果富含维生素、微量元素和膳食纤维。研究表明,蔬菜及水果摄入减少、糖和软饮料摄入过多可能与CD及UC的发病增加相关[85,264]。增加水果和蔬菜等富含可溶性膳食纤维食物的摄入量,少食红肉、人造脂肪和食用油可能降低IBD的发病风险[85,223,230,263-265]。

34. 肠道微生态与IBD关系密切。推荐等级:推荐

环境因素是决定IBD发生和发展的关键,膳食是环境因素中的最主要部分,它通过与肠黏膜长期的直接作用,也通过影响肠道微生态组成和功能对肠道发挥影响[110]。与健康人相比,IBD患者肠道微生态组成和功能的改变主要表现在肠黏膜菌群多样性减少(比如肠杆菌增加和梭菌减少)和微生态代谢产物的变化[110]。肠道微生态的大多数功能通过分解膳食纤维产生SCFA来实现。产生SCFA的主要细菌普拉梭菌(*Faecalibacterium prausnitzii*)丰度下降增加IBD发病[266]。

调整肠道微生态可以从饮食和微生态两方面着手,但从肠道微生态改变到IBD发病是个漫长的过程。基于目前对肠道微生态的认识和干预手段尚不足以取得治疗效果,通过口服益生菌治疗IBD的研究虽然很多,但疗效不确切。某些益生菌产品对轻中度UC诱导或维持缓解的效果得到了循证医学证据支持,明确有效的益生菌包括*E.coli Nissle 1917*和*VSL#3*[267-268]。*VSL#3*治疗抗生素治疗失败的储袋炎(pouchitis)和预防储袋炎复发有效[269]。益生菌制剂对诱导、维持CD缓解和预防复发均无效[270-271]。虽有研究结果显示益生元可以减轻CD活动度,但随机对照研究未能证实其对诱导CD和UC缓解有益[232-235]。合生元(益生菌和益生元的合剂)可能有利于控制UC活动度,提高生活质量,减轻内镜下肠黏膜炎症反应,但尚需更多研究证实[238,272-273]。

粪菌移植(fecal microbiota transplantation,FMT)对CD及储袋炎的治疗效果多来自于病例报道,缺乏高水平临床研究的证据支持[274]。尽管目前研究表明FMT对UC有诱导缓解作用,但FMT面临着菌液安全性和标准化、治疗方案的规范化等问题,因此,临床广泛应用尚需进一步研究支持[275-276]。

35. 营养支持治疗结合适度体能锻炼有利于改善IBD患者营养状况,提高生活质量。推荐等级:推荐

因为腹部症状和疾病的影响,IBD患者体能状况欠佳[277]。研究显示,体能状况可能与IBD活动度和抑郁状态存在一定的相关性;UC患者的体能状况与其年龄和抑郁状态独立相关;体育锻炼对IBD患者的情绪能产生积极影响[278-279]。

营养支持治疗能改善IBD患者营养状况,在此基础上进行适度的体能锻炼有助于提高营养支持治疗效果,增加骨密度和肌肉含量,延缓疾病复发。研究表明,经常从事体育锻炼的缓解期CD患者6个月内疾病复发的可能性明显降低[280]。体育锻炼还能够提高IBD患者的生活能力及社会适应力,有助于提高生活质量[281]。肥胖型IBD患者也应通过体育锻炼减少脂肪量,增加肌肉群,而不是通过限制蛋白质或能量的摄入来消耗脂肪[282-283]。

体育锻炼的强度要适当,高强度的体育运动可引起短暂的轻度全身炎症反应,增加促炎细胞因子的释放,对控制IBD病情不利[279]。目前尚缺乏适用于IBD患者的可推荐的运动方案,建议采取积极、自觉、量力而行的锻炼方案[284]。

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