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甘草泻心汤治疗溃疡性结肠炎机制及临床研究进展*

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摘要:溃疡性结肠炎(UC)是一种慢性、非特异性炎症性肠病,病因与环境、遗传、感染和肠道微生物等多种因素相关。甘草泻心汤出自《伤寒论》,由炙甘草、黄芩、黄连、干姜、人参、半夏、大枣组成,具有调和寒热,消痞止利的功效。现代研究发现,甘草泻心汤单独或联合西药治疗UC具有显著疗效,其作用机制包括调节炎症因子、保护肠道黏膜和改善肠道菌群。通过查阅相关文献,对近年来甘草泻心汤及其单体治疗UC的相关机制与临床研究进行梳理与总结,以期为中医及中西医结合治疗UC提供理论依据并拓展治疗思路。

关键词:甘草泻心汤;溃疡性结肠炎;炎症因子;肠道黏膜;肠道菌群
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溃疡性结肠炎(UC)是一种以腹痛、腹泻、里急后重及黏液脓血便为主要临床表现的慢性、非特异性炎症性肠病[1]。国内外研究主要认为其病因与环境、遗传、免疫、感染和肠道微生物等多种因素相关[2]。此病患病率在全球范围内逐年增高,在新兴工业化国家中尤为明显[3]。目前治疗UC以缓解症状为主,西医以对症治疗为主,存在易复发,不良反应发生率较高等问题[4],中西医结合治疗UC具有改善临床症状、加快溃疡愈合、降低复发率、提升患者生活质量等优势[5]。甘草泻心汤在临床上被大量应用于溃疡性结肠炎的治疗,并且现代药理研究发现甘草泻心汤具有抗溃疡、抑制炎症信号通路、修复肠道黏膜屏障和改善肠道菌群失调的作用[6]。笔者对甘草泻心汤治疗UC的机制及临床研究展进行综述,以期对治疗UC提供理论依据与新的思路。

1 溃疡性结肠炎的病因病机

溃疡性结肠炎由于其临床症状及特点,在中医归属于"久痢,休息痢,肠澼"的范畴。探其主要病因病机,大多由于素体虚弱,饮食不节或情志不畅等原因,致脾虚失运,水湿内生,阳虚寒盛,所致泄泻,《杂病源流犀烛》云:"湿盛则飧泄……是泄虽有风寒热虚之不同,要未有不源于湿者也。"此外病程日久,湿郁化热,湿热毒邪侵犯,寒热错杂清浊失分,热灼肠络,混杂而下,故见黏液脓血便。《景岳全书》云:"今之凡患泻痢者,正以五内受伤,脂膏不固,故曰剥而下。"本病以脾虚为本,湿热毒邪为标,凝聚成痈疡,故而本病虚实夹杂,寒热错杂[7]。

2 甘草泻心汤的方证探析

甘草泻心汤出自《伤寒论》158条:"伤寒中风,医反下之,其人下利,日数十行,谷不化,腹中雷鸣,心下痞硬而满,干呕心烦不得安,医见心下痞,谓病不尽,复下之,其痞益甚,此非结热,但以胃中虚,客气上逆,故使硬也,甘草泻心汤主之。"本方由炙甘草、黄芩、黄连、半夏、干姜、人参、大枣组成。《医宗金鉴》曰:"方以甘草命名者,取和缓之意。用甘草、大枣之甘温,补中缓急,治痞之益甚;半夏之辛,破客逆之上从;芩、连泻阳陷之痞热,干姜散阴凝之痞寒。缓急破逆,泻痞寒热,备乎其治矣。"故重用炙甘草补中缓急,胃虚得补,急利得缓,更可清热解毒,调和诸药。黄芩、黄连性味苦寒,清热燥湿,泻火散痞,降阳以复阴。半夏辛温,燥湿化痰,散结除痞以缓脘腹胀满,干姜辛热,温中散寒宣畅中焦气机,二

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者燥湿温脾，调畅脾胃气机，宣通上下之阴阳。人参、大枣甘温益气，以补脾胃，复其气机升降枢纽之职。《绛雪园古方选注》曰："甘草泻心，非泻结热，因胃虚不能调剂上下，致水寒上逆，火热不得下降，结为痞。故君以甘草、大枣和胃之阴，干姜、半夏启胃之阳，坐镇下焦客气，使不上逆；仍用芩、连，将已逆为痞之气轻轻泻却，而痞乃成泰矣。"可见本方诸药相合平调寒热，缓急补虚，燥湿消痞止利。宣降气机，调气以降破逆。

此外甘草泻心汤治疗范围广泛，《金匮要略百合狐惑病脉证并治》云："狐惑之为病……甘草泻心汤主之。"现代医家将其应用于白塞病、口腔黏膜病，肠易激综合征、溃疡性结肠炎、慢性结肠炎、失眠等疾病。陈士铎《石室秘录》云："同治者，同是一方，而同治数病也，异治者，一病而异治也。"本着异病同治的思想虽病相异而其病机相同则可同治[8]。故病机体现为"脾胃气虚，寒热错杂"者便可治之。

3 临床研究

甘草泻心汤作为治疗湿热蕴结证及寒热错杂证的经典方剂广泛应用于临床。赵秋枫等[9]将60例复发性UC患者分为甘草泻心汤组和美沙拉嗪组，3个月后两组在临床症状、肠镜下表现与肠道菌群构成方面均较前改善，但两者间差异无统计学意义，甘草泻心汤组的白介素(IL)-10水平更高，复发率与加重率明显降低。单纯使用甘草泻心汤治疗UC的疗效与美沙拉嗪相当，且复发率更低。

关于甘草泻心汤单独治疗UC的研究较少，临床上多与美沙拉嗪、柳氮磺吡啶等药物联合使用且疗效更为显著。甘草泻心汤联合美沙拉嗪或柳氮磺吡啶的研究周期为1.5~3个月，治疗后中西医结合治疗总有效率高于单纯西药治疗，前者中医证候积分、改良Mayo评分下降更明显，炎症性肠病问卷(IBDQ)评分更高，肿瘤坏死因子-α(TNF-α)、IL-6、IL-8、IL-17、IL-23降低，IL-10提高，差异有统计学意义($P<0.05$)[10-22]。见表1。

4 甘草泻心汤治疗UC的相关机制

4.1 抑制炎症 UC起病和发展与促炎和抑炎细胞因子的平衡被打破有关，促炎细胞因子在UC的病程中起主要作用，诱发炎症或加重炎症程度使肠道黏膜受损，从而破坏肠道免疫稳态。TNF-α、IL-6和IL-8等促炎细胞因子能诱发或加重UC；而IL-4、IL-10等抑炎细胞因子对维持肠道免疫稳态起关键作用[23]。

在硫酸葡聚糖钠(DSS)诱导的UC模型大鼠中[24-25]，甘草泻心汤联合血竭灌胃治疗能够抑制UC大鼠IL-6和TNF-α的表达。IL-6能够促进肠上皮细胞(IECs)分泌炎症介质，使黏膜通透性增加、中性粒细胞依附和聚集，引起炎症，诱发或加重UC[26]。TNF-α聚集中性粒细胞，诱导趋化因子，上调其他促炎因子的表达(如IL-6和IL-8)，这些促炎因子也可提高TNF-α的生物学活性，从而触发并进一步放大炎症反应；此外，TNF-α促进内皮细胞黏附因子分泌，提高上皮紧密连接的通透性从而抑制IECs生长[27-28]。

张建伟等[29]、陈少芳等[30]发现甘草泻心汤通过抑制Toll样受体4/核转录因子-κB(TLR4/NF-κB)炎症信号通路来降低TLR4活性，延缓NF-κB激活，从而减少IL-6、IL-8的产生并促进IL-10分泌。TLR4/NF-κB是UC的炎症信号通路之一，NF-κB为该通路下游核转录因子，激活后的NF-κB在炎症反应、细胞凋亡等过程中起关键作用。NF-κB可增加IL-6、IL-8等细胞因子的分泌，这些细胞因子进一步活化NF-κB，从而放大炎症反应[31]。IL-8在激活中性粒细胞、诱导其他炎症因子的同时增加过氧化物及溶酶体酶数量，加重UC的炎症反应[32]。在UC患者中，TNF-α、IL-6和IL-8的水平可反映病情严重程度[33]，而IL-10的水平通常较低，可能与病情严重程度呈负相关[34]。IECs过度应激反应常发生在IL-10水平较低的小鼠体内，发生机制与辅助性细胞介导的炎症反应相关，治疗后UC小鼠模型肠道黏膜内IL-10水平随症状好转而恢复至正常水平[35]。

陈浩等[36]发现甘草泻心汤可以改善UC模型大鼠结肠病态结构，并降低IL-6水平和转录激活因子(STAT3)基因表达。IL-6/STAT3通路在UC的发生和发展中扮演重要角色，IL-6及其受体形成的复合物与膜糖蛋白膜糖蛋白130(gp130)结合，激活并相继磷酸化Janus激酶(JAK)与STAT3，导致细胞核内抗凋亡基因表达提高，从而加重炎症程度[37]，而阻断或抑制IL-6/STAT3通路可以减轻UC模型大鼠的肠道损伤和炎症[38]。

综上，甘草泻心汤能够缓解肠道的过度炎症反应和预防炎症反复发作，与血竭联合运用有抗炎、抗溃疡作用。

4.2 保护肠道黏膜 肠道屏障功能异常和UC的发生与发展具有相关性，肠道黏膜屏障主要由IECs、黏液层、抗菌肽、分泌型免疫球蛋白A(sIgA)、肠黏

表1 甘草泻心汤治疗UC临床观察

Tab.1 Clinical observation on treatment of UC with Gancao Xiexin Decoction

疾病	方药组成及剂量	联合药物	例数	对照组	治疗时间	评价指标	疗效	参考文献
UC寒热错杂证	炙甘草12 g、黄芩10 g、党参10 g、半夏6 g、干姜6 g、黄连3 g、大枣10 g	美沙拉嗪肠溶片	60	美沙拉嗪肠溶片	8周	中医症状积分、改良Mayo评分、肠道菌群、TNF-α、IL-6、IL-10	总有效率联合组为86.67%（26/30），对照组为60.00%（18/30）	[10]
UC寒热错杂证	炙甘草12 g、黄芩9 g、党参9 g、半夏9 g、干姜9 g、黄连3 g、大枣3颗	美沙拉嗪肠溶片	77	美沙拉嗪肠溶片	8周	中医证候积分、肝功能、TNF-α、CRP	总有效率联合组为89.74%，对照组为71.05%	[11]
UC	炙甘草12 g、黄芩10 g、党参10 g、半夏9 g、干姜6 g、黄连3 g、大枣10 g	美沙拉嗪肠溶片	62	美沙拉嗪肠溶片	8周	临床症状消失时间、TNF-α、CRP、临床总体疗效、不良反应	总有效率联合组为93.55%，对照组为74.19%	[12]
UC	炙甘草12 g、黄芩9 g、党参9 g、半夏9 g、干姜9 g、大枣6 g、黄连3 g	美沙拉嗪缓释片	200	美沙拉嗪缓释片	2个月	中医证候积分、TNF-α、IL-6、IL-10	联合组中医证候积分改善、TNF-α、IL-6较对照组下降明显、IL-10上升更高	[13]
UC	炙甘草12 g、黄芩9 g、党参9 g、半夏9 g、干姜9 g、大枣6 g、黄连3 g	美沙拉嗪肠溶片	82	美沙拉嗪肠溶片	1.5个月	治疗有效例数、TNF-α、IL-6、IL-10	总有效率联合组为95.00%，对照组为80.00%	[14]
UC	炙甘草12 g、党参30 g、大枣30 g、黄芩9 g、半夏9 g、干姜6 g、黄连3 g	美沙拉嗪肠溶片	94	美沙拉嗪肠溶片	6周	改良Mayo评分、IBDQ评分、TNF-α、IL-6、IL-10	总有效率联合组为93.62%（44/47），对照组为76.60%（36/47）	[15]
UC寒热错杂证	炙甘草12 g、黄芩9 g、党参9 g、半夏9 g、干姜9 g、大枣6 g、黄连3 g	美沙拉嗪缓释颗粒	88	美沙拉嗪缓释颗粒	2个月	中医证候积分、肠镜下Baron评分、IBDQ评分、复发率、TNF-α、IL-8、IL-17、IL-23	总有效率联合组为95.45%，对照组为77.27%；复发率联合组为7.14%；对照组为29.41%	[16]
UC	炙甘草12 g、党参30 g、大枣30 g、黄芩9 g、半夏9 g、干姜6 g、黄连3 g	美沙拉嗪肠溶片	86	美沙拉嗪肠溶片	6周	TNF-α、IL-6、IL-10	总有效率联合组为95.35%，对照组为81.40%	[17]
UC	炙甘草12 g、黄芩9 g、人参9 g、半夏9 g、干姜9 g、大枣6 g、黄连3 g	美沙拉嗪肠溶片、龙血竭胶囊	88	美沙拉嗪肠溶片	3个月	复发率、改良Mayo评分、TNF-α、IL-8	总有效率联合组为95.45%，对照组为75.00%；复发率联合组为4.86%，对照组为39.39%	[18]
UC	炙甘草12 g、黄芩9 g、党参9 g、半夏9 g、干姜9 g、大枣6 g、黄连3 g	柳氮磺吡啶片	60	柳氮磺吡啶片	8周	中医症状积分、Mayo评分、IBDQ评分、TNF-α、IL-17、IL-23	总有效率联合组为93.33%，对照组为73.33%	[19]
UC	炙甘草12 g、黄芩9 g、党参9 g、半夏9 g、干姜9 g、大枣6 g、黄连3 g	柳氮磺吡啶片	60	柳氮磺吡啶片	8周	改良Mayo评分、IBDQ评分、IL-17、IL-23	总有效率联合组为93.33%，对照组为80.00%	[20]
UC	炙甘草12 g、黄芩9 g、党参9 g、半夏9 g、干姜9 g、大枣6 g、黄连3 g	柳氮磺吡啶片	74	柳氮磺吡啶片	8周	TNF-α、IL-6、IL-17	总有效率联合组为93.33%，对照组为80.00%	[21]
UC寒热错杂证	炙甘草12 g、黄芩9 g、党参9 g、半夏9 g、干姜9 g、大枣6 g、黄连3 g	柳氮磺胺吡啶肠溶片	60	柳氮磺胺吡啶肠溶片	3个月	中医证候积分、肠镜镜检结果变化	总有效率联合组为90.00%，对照组为73.33%	[22]
复发性UC	炙甘草12 g、党参30 g、大枣30 g、黄芩9 g、半夏9 g、干姜6 g、黄连3 g	–	60	美沙拉嗪胶囊	3个月	临床症状评分、肠镜下评分、肠道菌群、IL-6、IL-10	停药后甘草泻心汤组复发或加重率较对照组低	[9]

膜免疫系统和肠道菌群构成[39]，其功能的改变主要体现在肠道菌群的改变和肠黏膜通透性增加。研究发现，肠黏膜通透性增加常常发生在UC起病之前，肠道菌群位置和数量的改变会影响免疫系统而导致肠道黏膜功能的异常[40]。

IECs是肠道黏膜屏障构成的主要成分之一，其结构完整保障了肠屏障功能的稳定。沈雁等[41-42]发现甘草泻心汤可抑制蛋白激酶R样内质网激酶（PERK）-真核细胞起始因子2α（eIF2α）-C/EBP同源蛋白（CHOP）凋亡信号通路的活化和传导，减少IECs凋亡，改善肠黏膜通透性，保护肠黏膜屏障。内质网应激（ERS）是细胞中广泛存在的的一种自我稳定机制，在应激细胞的凋亡中起到关键作用[43]。UC患者及动物模型的IECs处于过度ERS状态[44]，被激活的PERK-eIF2α-CHOP信号通路将使IECs异常凋亡，引起肠上皮通透性增高进而破坏肠黏膜屏障[45]。

陈浩等[46]发现UC模型大鼠血清内D-乳酸和内毒素水平显著升高，紧密连接蛋白-1（Claudin-1）和其信使核糖核酸（mRNA）表达显著下降。使用甘草泻心汤后这一趋势得到抑制。D-乳酸由肠道固有细菌产生；内毒素是革兰氏阴性细菌细胞壁中的脂多糖，主要由肠道细菌分泌，两者皆可透过损伤的肠道黏膜屏障。Claudin-1是构成细胞间紧密连接（TJ）功能的主要成分之一，TJ维持肠上皮细胞的稳定、调控肠道屏障通透性以防止细菌及毒性物质进入结肠黏膜。还有研究发现甘草泻心汤通过促进肠黏膜浆细胞分泌sIgA，从而遏止病原体及其毒素对黏膜的入侵，增强肠道免疫[47]。

因此甘草泻心汤可通过降低上皮细胞凋亡、维持细胞建紧密连接和促进肠黏膜浆细胞分泌sIgA来保护和修复肠黏膜屏障。

4.3 调节肠道菌群 肠道菌群是人体中重要且数量和种类庞大的微生态系统，各个菌群之间共存并相互制约，形成动态平衡，其功能主要包括参与物质吸收、代谢、调节免疫系统和构成肠黏膜屏障等[48]。UC患者肠道菌群稳态失衡，乳杆菌及双歧杆菌等有益菌含量较低，而拟杆菌、肠球菌、梭杆菌及肠杆菌等致病菌含量过高[49-50]。肠道菌群平衡被破坏会导致肠道黏膜屏障受损和免疫稳态失衡，这与UC的发生和易反复发作等相关[51-53]。甘草泻心汤通过调节肠道菌群及其代谢物来改善小鼠的结肠炎症，具体机制为增加有益菌（如杜氏杆菌、双歧杆菌和乳杆菌等），减少致病菌（如大肠杆菌、肠球菌等），降低亚油酸并调节其代谢途径，从而改善结肠组织损伤并降低促炎细胞因子水平[47,54]。

5 甘草泻心汤中单体成分对于UC的治疗机制

甘草泻心汤由炙甘草、黄芩、黄连、干姜、人参、半夏、大枣组成，其中分别含有甘草酸苷、盐酸小檗碱、黄芩苷、6-姜烯酚等有效成分[55]对UC有较好疗效[56]。

甘草酸苷（GL）可通过降低过度的ERS水平来抑制半胱氨酸天冬氨酸蛋白酶（Caspase）-12信号通路活化，进一步减少IECs的过度应激凋亡，从而保护肠道黏膜屏障，降低UC小鼠模型的疾病活动指数（DAI）和组织损伤指数（TDI），缓解UC的炎症反应[57]。

在保护肠道黏膜方面，黄连提取物盐酸小檗碱（BBR）可以减轻肠道干细胞（ISCs）和紧密连接蛋白的损害[58-59]。BBR不仅下调ERS水平，减缓IECs过度凋亡[60-62]，而且与黄连粗多糖上调Claudin-1及mRNA表达水平有效缓解结肠炎症[63-64]。在调节炎症因子方面，BBR减少结肠组织内TNF-α和IL-1β水平的同时提高IL-10水平[65]。其过程可能是BBR通过抑制NF-κB调节一氧化氮合酶（iNOS）和精氨酸酶1（Arg1）的mRNA表达，促进M1向M2型逆极化[66]，升高M2/M1比例，从而抑制TNF-α和IL-6表达，提高抗炎因子IL-10和血清转化生长因子（TGF）-β的表达[67]。在UC小鼠模型结肠组织中M1型巨噬细胞比例显著升高，TNF-α和IL-6的水平亦相应升高[68]。通常M2/M1巨噬细胞比例相对稳定，M1型巨噬细胞具有促炎作用，M2型巨噬细胞具有抑炎作用并参与组织修复[69]。这些研究结果表明BBR能够改善肠道炎症反应，减轻肠道干细胞和紧密连接蛋白损害，保护修复肠道黏膜，从而缓解UC病情。

黄芩苷能抑制NF-κB的活化[70]，降低凋亡蛋白人凋亡相关因子配体（FasL）、结肠组织环氧合酶-Ⅱ（COX-2）、Caspase-9、β连环蛋白（β-catenin）的表达水平，阻断磷脂酰肌醇3激酶（PI3K）/丝氨酸苏氨酸蛋白激酶（AKT）信号通路，下调炎症因子和促凋亡因子水平，降低TNF-α、IL-1、IL-6和IL-8表达水平，从而抑制肠道免疫反应，减少IECs的凋亡[71]。

干姜提取物6-姜烯酚可抑制肠道TLR4/NF-κB信号通路的过度激活[72]。Notch信号通路是修复肠道黏膜的主要信号通路，能够调节IECs的增殖、分化与凋亡，但其过度激活则会导致黏膜屏障的受

损[73]。6-姜烯酚能抑制其过度激活，调节 IECs 向分泌细胞系（杯状细胞）分化，增加黏糖蛋白 2（MUC2）黏蛋白分泌，使得肠道受损黏膜得以修复[74-76]。

6 小结与展望

随着当代社会的发展和环境的变化，UC 的发病率在不断上升，UC 的病因病机也在不断变化，对其病因病机也具有更深入的认识，在现代更精确的诊断技术，更标准的评价指标和更规范化的临床试验下，现有的临床研究证明了甘草泻心汤治疗 UC 的有效性。此外实验表明，甘草泻心汤可以通过调节炎症因子，保护肠道黏膜，调节肠道菌群等途径治疗 UC。其单体有效成分治疗 UC 机制涉及缓解肠道内炎症反应、抑制过度应激凋亡、保护修复肠道黏膜屏障等。临床研究显示，单独使用甘草泻心汤与美沙拉嗪疗效相似，甘草泻心汤与美沙拉嗪或柳氮磺吡啶联合使用较单独应用总有效率高，在缓解症状，抑制炎症，改善肠镜下评分，降低复发率方面更具优势。

然而甘草泻心汤治疗 UC 作用机制尚不明确，近年来甘草泻心汤治疗 UC 的作用机制研究多集中在组织、细胞、分子层面，较为微观。大部分聚焦于单一靶点，平行关联不同靶点和 UC 的病理机制相结合的研究较为缺乏。此外，缺乏对甘草泻心汤从微观分子作用网络的分析及多中心，大样本，随机双盲的临床研究。接下来的研究应注重不同研究之间的关联性，加强文献整理及单一靶点之间的关联研究将已有研究归纳、整理、总结、提高，由一个个横向研究发展到纵向研究最后到整体化研究，构筑更加科学的，具体的规范化研究模式，为中医药科学化提供更多的依据，为临床治疗提供依据与参考。

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Mechanism and clinical research progress of Gancao Xiexin Decoction in the treatment of ulcerative colitis

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Abstract:Ulcerative colitis (UC) is a chronic,non-specific inflammatory bowel disease. The etiology may be related to environmental,genetic,immune,infectious and intestinal microbiological factors. Gancao Xiexin Decoction from treatise on febrile diseases. It is composed of prepared liquorice,scutellaria,coptis,dried ginger,ginseng,pinellia ternata and jujube,which has the effect of mediating cold and heat,eliminating ruffian and stopping profits. Modern studies have found that Gancao Xiexin Decoction alone or combined with Western medicine has significant curative effect on UC,and its mechanism involves regulating inflammatory factors,protecting intestinal mucosa and improving intestinal flora. Through reviewing relevant literature,this paper summarized and summarized the mechanism and clinical research related to the treatment of UC by Gancao Xiexin Decoction and its monomer in recent years,in order to provide theoretical basis for the treatment of UC by traditional Chinese medicine and integrated traditional Chinese and Western medicine and expand the treatment ideas.

Keywords:Gancao Xiexin Decoction;ulcerative colitis;inflammatory factor;intestinal mucosa;intestinal flora