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述评

- 1907 莫迪司、普美显增强磁共振与肝纤维化分期的相关性研究

肖曼君, 肖恩华

基础研究

- 1914 从多项行为学测评分析慢传输型便秘大鼠的精神心理异常及其意义

张桢, 贺平

临床研究

- 1920 综合预见性护理联合延续护理对脑卒中吞咽障碍患者出院后的护理效果分析

蔡文煊

- 1927 原发性胆汁性肝硬化的早期诊断运用

李建柱, 周琛, 王剑飞

- 1933 微生态制剂对老年肠道菌群失调相关性腹泻的影响分析

章科清, 江琴, 张海兵

文献综述

- 1939 胃癌新辅助治疗进展和展望

张顺, 蒋小华

- 1947 重症急性胰腺炎早期液体复苏

刘爱茹, 胡端敏

临床实践

- 1953 复方嗜酸乳杆菌预处理在根除幽门螺杆菌阳性胃溃疡的疗效评价

王立明, 尚惺杰

消 息

- 1913 《世界华人消化杂志》正文要求
1926 《世界华人消化杂志》修回稿须知
1932 《世界华人消化杂志》性质、刊登内容及目标
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Contents

Volume 26 Number 33 Nov 28, 2018

EDITORIAL

- 1907 Staging of liver fibrosis using Gd-EOB-DTPA and Gd-BOPTA enhanced magnetic resonance imaging

Xiao MJ, Xiao EH

BASIC RESEARCH

- 1914 Behavioral analysis of mental and psychological abnormalities and their significance in rats with functional constipation

Zhang Z, He P

CLINICAL RESEARCH

- 1920 Effect of comprehensive predictive nursing combined with continuous nursing on dysphagia in stroke patients

Cai WH

- 1927 Clinical value of inflammatory factors in early diagnosis of primary biliary cirrhosis with bacterial infection

Li JZ, Zhou C, Wang JF

- 1933 Therapeutic effect of microecological preparation plus Rifaximin on diarrhea associated with intestinal flora imbalance in elderly patients

Zhang KQ, Jiang Q, Zhang HB

REVIEW

- 1939 Neoadjuvant therapy in gastric cancer: Current status and future perspectives

Zhang S, Jiang XH

- 1947 Early fluid resuscitation in severe acute pancreatitis

Liu AR, Hu DM

CLINICAL PRACTICE

- 1953 Therapeutic efficacy of pretreatment with compound *Acidophilus lactobacillus* tablets followed by quadruple therapy in gastric ulcer patients with *Helicobacter pylori* infection

Wang LM, Shang XJ

Contents

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Volume 26 Number 33 Nov 28, 2018

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重症急性胰腺炎早期液体复苏

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Early fluid resuscitation in severe acute pancreatitis

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Abstract

Acute pancreatitis (AP) is a common clinical emergency disorder, and its morbidity is increasing gradually. Severe AP (SAP) often occurs with a sudden onset and high mortality. Microcirculation disturbance and hemodynamic abnormality is one of the main pathophysiologic mechanisms of SAP. Early fluid

resuscitation is the cornerstone of therapy. However, at present, the fluid type, the amount of fluid resuscitation, and the rehydration rate are still in dispute. Early goal-directed fluid therapy as an important individualized liquid resuscitation strategy has great significance to improve the prognosis of SAP. This article reviews the pathophysiological mechanisms of microcirculation disturbance, the related dispute of liquid resuscitation therapy, and the application of early goal-directed treatment strategy.

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Key Word: Severe acute pancreatitis; Fluid resuscitation; Early goal-directed therapy

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摘要

急性胰腺炎(acute pancreatitis, AP)是常见的临床急症, 发病率逐年升高, 尤其重症急性胰腺炎(severe AP, SAP), 起病凶险、病死率高。微循环障碍、血流动力学改变是SAP主要病理生理机制之一, 早期液体复苏是SAP治疗的基石。而目前对于早期补液类型、补液量、补液速率等仍处于争议中, 早期目标导向性液体治疗是一项重要的个体化液体复苏策略, 对改善SAP的预后具有重要意义。本文就SAP微循环病理生理改变的发生机制、液体复苏的相关治疗及早期目标导向性液体治疗策略的应用做一综述。

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关键词: 重症急性胰腺炎; 液体复苏; 早期目标导向性液体治疗

核心提要: 本文以重症急性胰腺炎(severe acute pancreatitis, SAP)早期液体复苏为重点, 对早期补液类型、补液量、补液速率等进行综述, 并结合最新文献提出早期目标导向性液体治疗作为一项重要的个体化液体复苏策略, 在SAP早期补液中的应用及对改善SAP预后的意义。

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0 引言

急性胰腺炎(acute pancreatitis, AP)是胰腺的急性炎症反应, 起病急, 病情变化快, 具有较高的发病率与病死率^[1]. AP临床表现多样, 2012年修订的亚特兰大标准分为轻症、中度重症和重症三类, 并将发病后1 wk定义为疾病的早期阶段^[2]. 轻症急性胰腺炎(mild acute pancreatitis, MAP)症状轻, 为自限性疾病, 通常1-2 wk内恢复, 中度重症急性胰腺炎(moderate severe acute pancreatitis, MSAP)伴有局部或全身并发症, 可出现一过性(48 h内)器官功能衰竭, 器官衰竭超过48 h, 即可进展为重症急性胰腺炎(severe AP, SAP), 病死率高达30%-50%^[3], 发病急性期是全身炎症反应综合征(systemic inflammatory response syndrome, SIRS)、多器官功能障碍综合征(multiple organ dysfunction syndrome, MODS)的高发期, 积极对症支持治疗是重要的治疗手段, 其中有效液体复苏是SAP急性期治疗的基石. 然而关于早期规范化的液体复苏治疗国内外尚未形成统一共识. 以个体为标准的早期目标导向性液体疗法, 在其他重症患者中的应用价值已经被证实^[4-8], 其中液体复苏是其治疗的核心, 因此目标导向性液体复苏策略在SAP中的应用应该引起临床医生的重视.

1 SAP微循环障碍的病理生理机制

微循环障碍是SAP发生发展的重要生理机制之一. 在胆道梗阻、高脂血症、酒精等始动因素下, 胰蛋白酶原异常激活, 消化自身胰腺组织, 同时促进机体大量炎症介质、血管活性因子等的释放^[9,10], 这些促炎介质可以激活血管内皮细胞, 引起血管收缩, 增加血管通透性, 引发毛细血管渗漏综合征(systemic capillary leak syndrome, SCLS), 血管内的大量液体渗漏到血管外, 机体血容量减少和组织间隙水肿, 导致组织器官缺血缺氧^[11]. 此外, 炎症介质与活化的粒细胞及血管内皮的相

互作用激活凝血与抗凝血系统, 促进弥散性血管内凝血(disseminated intravascular coagulation, DIC)的发生, 进一步加重组织器官的乏氧. 微循环障碍可引起肠黏膜缺血坏死及缺血再灌注损伤, 导致肠道黏膜屏障破坏, 可伴有麻痹性肠梗阻, 肠道细菌及内毒素易位即出现肠源性感染^[12].

AP时, 胰腺微循环改变明显加重了胰腺的缺血程度, 导致胰腺坏死. 在全身表现为血流动力学改变, 低血容量、低血压甚至休克. 同时, 瀑布式的炎症级联反应诱发SIRS^[11], 加重全身组织器官损害, 进而发展为MODS^[13,14], 增加患者病死率. 发病急性期是SIRS、MODS的高发期, 因此对于SAP发病早期及时有效的液体复苏对疾病的预后至关重要^[15].

2 补液类型

目前临床上最常用的两种液体类型即晶体和胶体. 最常用的晶体主要有生理盐水(normal saline, NS)、乳酸盐林格溶液(lactated ringer's solution, LR), 而丙酮酸乙酯林格溶液、高渗盐水为目前新的晶体类型. 最常用的胶体主要有低分子右旋糖酐、羟乙基淀粉、白蛋白、血浆等. 目前对于最佳补液类型的选择仍未有定论.

在胆源性胰腺炎的动物模型中发现, 大量LR的补充可以明显改善胰腺微循环状态^[16]. Wu等^[17]人比较了NS与LR补液组对AP预后的影响, 结果显示补液24 h后, LR补液组的SIRS的发生率、C反应蛋白(C-reactive protein, CRP)水平显著降低. 该试验也因此奠定了LR在未来补液治疗中的重要地位. 丙酮酸乙酯林格溶液是一种新型的液体类型, 丙酮酸盐具有潜在的抗氧化作用, 可以减少胰腺水肿或坏死, 通过下调炎症因子减少远隔脏器的损伤, 并在坏死性胰腺炎老鼠模型中, 显著提高其生存率^[18], 可见丙酮酸乙酯林格溶液可以代替乳酸钠林格溶液用于液体复苏治疗. 最近多项动物研究发现在AP中, 高渗盐水液体复苏可以调节细胞毒素反应^[18-20], 显著改善胰腺微循环, 促进心脏的收缩, 增加外周组织的血流灌注^[21], 高渗盐水补液可明显降低补液量, 进而降低肺水肿的发生风险. 然而高渗盐水补液导致脑桥脱髓鞘、肾脏衰竭等严重副反应已经被报道^[20], 其临床的应用也因此受到了挑战.

大部分动物研究表明胶体补液明显优于晶体. Schmidt等^[22]人在胰腺炎老鼠模型中发现, 大量右旋糖酐补液组的胰腺坏死发生率及病死率显著低于LR和NS补液组. 在一项纳入了13例非胆源性SAP患者的I期临床试验中, 采用右旋糖酐扩容6 h后, 胰腺微循环明显改善, 且胰腺坏死发生率为15%, 病死率只有7.7%^[23]. 此外, 羟乙基淀粉(hydroxyethyl starch, HES)作为另一种人

工合成胶体在临床中被广泛应用. Cheng等^[24]研究显示在SAP早期, HES补液组可明显降低白介素(Interleukin, IL)-1、IL-8的水平, 缩短液体负平衡出现的时间, 稳定机体免疫状态, 降低SAP腹腔内高压的发生风险^[25], 且HES联合NS补液组与单纯NS补液组相比, 器官衰竭和感染的发生率显著降低^[26]. 此外, 有研究指出HES的应用可以加重肾脏的损伤^[27]及凝血功能的障碍^[28], 而人血白蛋白的影响较小^[29]. 另一项研究发现, 采用新鲜冰冻血浆进行液体复苏可以减少血清-2巨球蛋白水平的下降^[30], 提示其在SAP早期可能具有潜在的治疗作用.

此外, 在血流动力学方面, 胶体可维持血管内胶体渗透压, 减少毛细血管渗漏, 降低第三间隙液体滞留^[31], 及抑制炎症因子, 预防炎症反应的进展^[32]. 晶体可分布于血管内和血管外间隙, 不仅补充血容量, 同时对体内的炎症因子起到稀释作用^[33]. 然而过多的晶体或胶体均不利于患者整体预后^[34], 早期以晶体为主的大量低渗透性补液可能增加毛细血管的渗漏, 加重第三间隙液体滞留和肺水肿的发生, 增加急性呼吸衰竭的发生率. 而过量的胶体则可能增加血管内的容量负荷, 引起血液黏滞, 凝血功能障碍及多器官氧合障碍, 增加病死率. 因此合适的晶胶比是取得最佳治疗效果的关键. 目前晶胶联合补液也被推荐优于单一补液治疗^[25,35], 而对于晶胶补液的最佳比例仍未有定论.

多项临床试验对SAP的晶胶补液比例进行探究, 一些研究专家指出3:1晶胶比早期补液效果更佳, 但缺乏高质量临床研究证据^[3]. 一项纳入47例SAP患者的回顾性研究分析^[36], 入院第一个24 h晶胶比1.5-3.0组患者的机械通气率、死亡率、液体滞留量均显著低于<1.5、>3.0晶胶比组. 冯永文等^[37]人研究指出, SAP患者液体复苏72 h后, 与低晶胶比组(<3:1)相比, 高晶胶比组(>3:1)的补液量、血管外肺水指数、膀胱内压、心房钠尿肽均显著增高, 氧合指数PaO₂/FiO₂显著降低.

综上, 在SAP早期选择何种液体类型进行液体复苏仍处于争议中. 美国胃肠协会推荐首选晶体补液, LR优于NS溶液, 且在红细胞比容<25%并且白蛋白<2 g/dL时考虑增加含红细胞的胶体补液^[38]. 我国共识意见中指出早期液体复苏应采取合适的晶胶比, 晶体首选LR, 次之NS, 胶体首选人血白蛋白或血浆, 关于羟乙基淀粉存在争议, 因其对肾脏和凝血功能有一定影响需慎用^[3].

3 补液量及速率

目前国内外研究对于在SAP早期选择积极性补液还是限制性补液仍各持己见. 积极性补液被定义为在入院24 h内接受的输液总量大于入院72 h内输液总量的1/3^[39]. 我国研究专家则将补液量超过15 mL/kg·h定义

为积极性补液, 补液量5-10 mL/kg·h则定义为限制性补液^[35].

Gardner等^[32]在2项回顾性研究中指出, 积极的补液可以减少患者器官衰竭的发生率, 提高生存率. Talukdar等^[40]认为在胰腺炎早期予以积极的液体治疗有助于维持血流动力学的稳定. Li等^[41]从动物实验中证实大量的液体灌注可以减少血液中的炎症递质, 进而延缓疾病进展.

然而其他研究对于积极性补液则持有相反的意见. 一项纳入247例AP患者的前瞻性研究指出, 入院24 h内补液量>4.1 L的液体复苏组持续器官衰竭的发生率显著高于补液量3.1-4.1 L液体复苏组^[41]. 瑞典学者Eckerwall等^[42]研究发现, 在发病初的24 h内输液大于4.0 L的SAP患者, 发生呼吸系统并发症的可能性增大(66%:53%, $P<0.001$), 且转入ICU继续治疗的患者比率也较高. 毛恩强等人的研究指出, SAP早期积极性补液组(15 mL/kg·h)的并发症的发生率及病死率均高于限制性补液组(5-10 mL/kg·h)^[35], 其另一项研究发现快速扩容组SAP患者的生存率显著低于缓慢扩容组^[43], 且28 d内脓毒症的发生率也明显增加.

鉴于SAP早期补液治疗的重要性, 目前部分指南推荐SAP早期积极性补液治疗^[32,38,44,45]. 我国共识意见^[3]则推荐, 补液速度 5-10 mL/kg·h, 特殊情况下可达到 12 mL/kg·h, 并评估复苏终点指导补液治疗. 综上, 由于SAP病情的严重性及复杂性, 补液治疗受多种复杂因素的影响, 早期规范化液体复苏共识尚较难达成. 而以个体化为基础, EGDFT可能成为今后最佳液体复苏治疗策略.

4 早期目标导向性液体治疗

EGDFT是以恢复血流动力学为目标的个体化液体复苏策略, 至今已经有超过15年的研究史. 该治疗策略的重要价值在高风险的手术^[5]或围手术期患者^[6-8], 严重脓毒血症或脓毒性休克^[4]以及高乳酸血症患者^[46]中的应用已经被证实. 并且在脓毒症治疗的国际指南中被推荐. 目前该策略在AP中的应用罕见报道, 大部分AP的发生具有相似的病理生理机制及危险因素, 尤其SAP早期快速血流动力学的改变是其病情发生发展的基础, 因此以血流动力学参数为导向, 采用该策略指导液体复苏可能有效改善SAP患者的预后.

首先基于对患者病情严重程度及潜在临床结局的评估, 快速明确是否应该选择EGDFT. 在严重脓毒血症及脓毒性休克患者中, 以20-30 mL/kg的补液速度扩容超过30 min, 收缩压仍持续低于90 mmHg, 或高乳酸血症(>4 mmol/L), 则需开始EGDFT^[46]. SAP患者ICU转入率

高达80%, 存在持续器官功能的衰竭, 病死率高, 预后差。实时分析反应组织器官氧合灌注的相关参数, 如心率、血压、血氧饱和度、尿素氮、乳酸水平等, 采用中心静脉置管监测中心静脉压(cetral venous pressure, CVP), 留置导尿监测尿量及腹腔内压力等措施对患者病情实时密切监测, 评估AP的严重程度及结局预后, 一旦符合SAP标准, 立即在治疗开始6-8 h内启动EGDFT。目前关于AP病情严重程度的早期预测指标还需进一步研究。其中难治性低血压、持续高乳酸血症仍是提示病情严重, 临床预后差的有效指标。

此外, 明确复苏终点, 密切监测血流动力学参数, 评估治疗的有效性。EGDFT旨在初始复苏6 h内达到以下目标^[47]: (1)中心静脉压达到8-12 mmHg(1 mmHg = 0.133 kPa); (2)平均动脉压 ≥ 65 mmHg; (3)尿量 ≥ 0.5 mL/kg·h; (4)上腔静脉血氧饱和度或混合静脉血氧饱和度 ≥ 0.7 或0.65。每30 min对复苏终点进行评估^[46], 同时在治疗过程中动态监测CVP、心输出量、中心静脉氧饱和度等反应循环功能等指标, 及时调整治疗方案。Constantin等^[48]人在SAP的动物模型研究中指出以左心室每搏量变异度指导液体复苏组, 氧合指数、存活率均明显优于CVP组。

EGDFT指导液体复苏, 可以有效降低重症患者的病死率, 改善患者的预后, 同时减少医疗资源的消耗。国内一项研究指出EGDFT组SAP患者48 h液体摄入量、48 h乳酸水平、血淀粉酶恢复时间、血管活性药物使用时间、机械通气时间均少于或小于传统补液组, 且并发症发生率和28 d死亡率均低于传统补液组^[49]。Yang等^[50]人指出SAP患者及时实施EGDFT方案可明显降低腹腔内高压, 改善组织器官的氧合, 减少脏器功能的衰竭。

综上, EGDFT是一种贯穿治疗始终的液体治疗理念, 实时监控, 并结合患者对液体治疗的敏感性和耐受性, 以血流动力学参数为指导, 综合考虑患者病情, 适时调整液体类型的选择、补液速率及补液量, 从而避免补液量不足导致的循环衰竭, 又可以避免补液过度引起的心衰、呼衰等并发症。然而目前EGDFT主要在高风险手术及脓毒性休克等重症患者中有所研究, 但基于其在重症患者早期复苏救治中的重要价值, 值得临床医生对其在SAP早期液体复苏中的应用开展广泛研究探讨。

5 结论

SAP早期的液体复苏是最有效的治疗方式, 正确有效的液体治疗可以改善胰腺的微循环障碍, 增加组织器官氧合灌注, 进而延缓病情进展, 改善预后。但基于SAP患者病情的多变性、影响因素的多样性及治疗过程的复杂性, 其早期补液类型、补液量、补液速率仍未有定论。

而以恢复血流动力学为目标的个体化治疗策略EGDFT在重症患者早期液体复苏治疗中有着重要价值, 通过动态实时监测血流动力学参数指导液体复苏方案, 其可能成为未来临床上重要的补液方式, 促使SAP早期液体复苏治疗进入一个新的时代。

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• 消息 •

《世界华人消化杂志》外文字符标准

本刊讯 本刊论文出现的外文字符应注意大小写、正斜体与上下角标。静脉注射iv, 肌肉注射im, 腹腔注射ip, 皮下注射sc, 脑室注射icv, 动脉注射ia, 口服po, 灌胃ig. s(秒)不能写成S, kg不能写成Kg, mL不能写成ML, lcpm(应写为1/min)÷E%(仪器效率)÷60=Bq, pH不能写PH或P^H, *H pylori*不能写成HP, T_{1/2}不能写成tl/2或T_{1/2}, V_{max}不能Vmax, μ不写为英文u. 需排斜体的外文字, 用斜体表示。如生物学中拉丁学名的属名与种名, 包括亚属、亚种、变种。如幽门螺杆菌(*Helicobacter pylori*, *H.pylori*), *Ilex pubescens* Hook, et Arn.var.*glaber* Chang(命名者勿划横线); 常数*K*; 一些统计学符号(如样本数*n*, 均数mean, 标准差SD, *F*检验, *t*检验和概率*P*, 相关系数*r*); 化学名中标明取代位的元素、旋光性和构型符号(如*N*, *O*, *P*, *S*, *d*, *l*)如*n*-(normal, 正), *N*-(nitrogen, 氮), *o*-(ortho, 邻), *O*-(oxygen, 氧, 习惯不译), *d*-(dextro, 右旋), *p*-(para, 对), 例如*n*-butyl acetate(醋酸正丁酯), *N*-methylacetanilide(*N*-甲基乙酰苯胺), *o*-cresol(邻甲酚), 3-*O*-methyl-adrenaline(3-*O*-甲基肾上腺素), *d*-amphetamine(右旋苯丙胺), *l*-dopa(左旋多巴), *p*-aminosalicylic acid(对氨基水杨酸)。拉丁字及缩写*in vitro*, *in vivo*, *in situ*; *Ibid*, *et al*, *po*, *vs*; 用外文字母代表的物理量, 如*m*(质量), *V*(体积), *F*(力), *p*(压力), *W*(功), *v*(速度), *Q*(热量), *E*(电场强度), *S*(面积), *t*(时间), *z*(酶活性, kat), *t*(摄氏温度, °C), *D*(吸收剂量, Gy), *A*(放射性活度, Bq), *ρ*(密度, 体积质量, g/L), *c*(浓度, mol/L), *φ*(体积分数, mL/L), *w*(质量分数, mg/g), *b*(质量摩尔浓度, mol/g), *l*(长度), *b*(宽度), *h*(高度), *d*(厚度), *R*(半径), *D*(直径), *T*_{max}, *C*_{max}, *V*_d, *T*_{1/2} *CT*等。基因符号通常用小写斜体, 如*ras*, *c-myc*; 基因产物用大写正体, 如P16蛋白。



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