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新进展综述

脓毒症与高血糖及其治疗的研究进展

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[**摘要**]　脓毒症及脓毒性休克是急危重症医学面临的重大难题,随着人口老龄化及医疗相关侵入性操作增加等,脓毒症的发病率逐渐增高。目前脓毒症的定义不仅局限于感染与全身炎症反应,更准确地强调机体对感染反应的失控及由此导致的器官功能障碍,其涉及的病理过程还包括神经内分泌、代谢障碍、凝血异常等非免疫方面。脓毒症及脓毒性休克的高血糖状态常见,高血糖得不到有效控制对患者预后不利。

[**关键词**]　脓毒症;　高血糖;　炎症

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The research progress of sepsis and hyperglycemia and their treatment　*WEI Li-hong, ZHANG Jie. Department of Endocrinology, the First Affiliated Hospital of Guangxi Medical University, Nanning* 530021, *China*

[**Abstract**]　Sepsis and septic shock are the major problems in acute and severe medical care. The incidence of sepsis is increasing as the population ages and the medical-related invasive procedures increase. At present, the definition of sepsis is not only confined to infections and systemic inflammatory response, but accurately stressed the body′s response to infections resulting in loss of organ dysfunction, whose pathological process involves the aspects of neuroendocrine, metabolic disorders, coagulation abnormalities and nonimmunological diseases. Hyperglycemia is common in the patients with sepsis and septic shock, whose prognoses are usually bad if high blood sugar is not effectively controlled.

[**Key words**]　Sepsis;　Hyperglycaemia;　Inflammation

脓毒症是指宿主对感染的反应失控导致危及生命的器官功能衰竭,脓毒性休克是指经血管活性药物维持平均动脉压在 65 mmHg 及以上或血清乳酸水平≥2 mmol/L,其为脓毒症亚型[1]。脓毒症以及脓毒症休克是危重症领域的重大难题,病情凶险。国内一项前瞻性观察性队列研究显示脓毒症院内病死率为 33.5%[2],另一项研究提示脓毒症院内病死率为 20.6%[3],国外学者的研究[4~6]显示包括脓毒性休克在内的脓毒症的病死率为 12.8% ~ 33.2%。目前脓毒症的发病机制尚未完全明确,其病理改变除早期的促炎及抗炎反应外,还包括凝血、神经内分泌、心血管、代谢等非免疫性机体改变[1],脓毒症可引起血脂异常、血糖代谢紊乱[7~9],脓毒症中血糖异常主要为高血糖、低血糖,前者包括非糖尿病患者中的应激性高血糖和糖尿病患者的高血糖。有研究[10]表明高血糖增加脓毒症炎症反应,对预后不利。但 Tiruvoipati 等[11]研究认为应激性高血糖可能不影响脓毒症患者的预后,且其在重症监护室(ICU)的病死

率较低。另有研究[12]显示,严格控制血糖在 4.4 ~ 5.6 mmol/L 与 8.3 ~ 10 mmol/L 组的病死率、器官功能障碍等方面无明显差异。因此,目前针对血糖升高与脓毒症关系及脓毒症血糖控制较确切的范围仍有争议,现就脓毒症与血糖代谢关系及治疗进行综述。

1 脓毒症与高血糖流行病学特点

脓毒症患者血糖升高常见。一项研究[13]包括 20 余万已诊断脓毒症患者中有 23.3% 合并 2 型糖尿病,2 型糖尿病患者脓毒症发病率也逐渐增加。另外,脓毒症患者中应激性高血糖亦常见。应激性高血糖,是脓毒症患者血糖异常的类型之一,指非糖尿病患者在疾病或创伤等应激情况下出现短暂性血糖升高,应激因素消失后血糖恢复正常,其表现为血糖高于正常糖代谢(空腹血糖 <6.1 mmol/L,非空腹血糖 <7.8 mmol/L),且糖化血红蛋白 <6.5%[14]。Tiruvoipati R 等[11]研究提示脓毒症的应激性高血糖可能与其预后无关,且其在 ICU 的病死率更低。但另有研究[14,15]认为,无论是否已诊断为糖尿病,脓毒症患者入院时的血糖升高水平与其院内不良预后有关。脓毒症患者入院血糖≥11.1 mmol/L 则增加 30 d 内的病死率[14]。

2 血糖代谢特点

2.1 正常血糖代谢及高血糖定义 正常人血糖水平保持相对恒定是由激素、神经系统、组织器官共同调节的结果。激素主要包括胰岛素、胰高血糖素、生长激素、糖皮质激素、肾上腺素及甲状腺激素;神经系统主要包括副交感神经和交感神经。当血糖升高时,副交感神经直接刺激胰岛 B 细胞释放胰岛素,并同时抑制胰岛 A 细胞分泌胰高血糖素,使血糖降低;当血糖降低时,交感神经刺激胰岛 A 细胞分泌胰高血糖素,血糖升高。人体中血糖主要来源于食物糖的消化道吸收、肝糖原分解及非糖物质异生,血糖主要去路包括氧化分解、糖原合成、参与脂肪及氨基酸合成等。以上过程需肝、肾、脂肪、肌肉组织等组织细胞参与。同时糖也是组成人体组织的重要成分,如糖蛋白和糖脂是细胞膜的构成成分,参与细胞间信息传递等。正常人血糖水平:空腹血糖为 3.9 ~ 6.0 mmol/L,餐后 2 h 血糖 <7.8 mmol/L;高血糖是指空腹血糖 >6.1 mmol/L,餐后 2 h 血糖 >7.8 mmol/L。

2.2 高血糖病理机制

2.2.1 参与炎症反应 高血糖可经各种途径影响机体的病理生理过程。高血糖参与机体炎症反应。有研究表明,肿瘤坏死因子(TNF-α)与血浆血糖升高程度相关[16],当血糖≥5.6 mmol/L 时胎盘中 TNF-α 升高[17],TNF-α、白介素-6(IL-6)与高血糖及胰岛素抵抗相关[18],另外,糖尿病组炎症标志物高敏 C 反应蛋白(hsCRP)、IL-8 及 IL-6 均高于对照组[19]。TNF-α 通过激活 NF-κB 后上调纤维细胞血管黏附分子-1(vascular cell adhesion molecule-1,VCAM-1)促进心脏炎症[20]。IL-6 为免疫细胞分泌的细胞因子,参与炎症反应,其信号功能丧失可减少嗜酸性粒细胞和中性粒细胞招募小鼠细胞因子、趋化因子和过敏原引起的气道炎症[21]。但有研究提示暴露于高糖状态下的外周血单核细胞 IL-6 下降,提示急性高血糖有多种缺陷免疫反应[22],IL-8 升高可能经 NF-κB 信号通路参与气道炎症反应[23],IL-8 受体基因敲除后可使结肠的中性粒细胞减少[24]。

2.2.2 参与内皮细胞损伤过程 在一项大鼠研究[25]中发现,高血糖及急性血糖波动时血管内皮细胞活性氧(reactive oxygen species,ROS)增加,血糖波动组增加更明显,循环中炎症因子 IL-6、TNF-α、细胞间黏附分子-1(intercellular adhesion molecule-1,ICAM-1)增加,血糖波动组更明显,结果使血管内皮细胞出现明显的炎症反应和氧化应激,增加单核细胞与内皮细胞粘附,促进内皮细胞凋亡,导致严重心血管损伤。

3 脓毒症病理生理机制

3.1 内皮细胞受损 脓毒症主要表现为宿主对感染的失控,病原微生物进入机体后可损伤组织或血管,如扰乱血管内皮细胞、平滑肌等组织,使血管功能紊乱[26],脓毒症时血小板活性增加,使血浆 5-羟色胺水平升高,内皮细胞通过 5-羟色胺受体摄取 5-羟色胺,随后激活蛋白激酶-1(PAK1)及下游变形蛋白分子磷酸化,使内皮细胞功能障碍[27],从而使血管通透性增加,血管内成分溢出,如巨噬细胞、自然杀伤(NK)细胞到达损伤或感染部位,直接吞噬、杀伤病原体或损伤组织。

3.2 炎症反应 在感染早期,如细菌细胞壁某些成分可直接经补体旁路途径激活补体系统产生血清过敏毒素,如活性最强的补体 C5a,脓毒症早期 C5a 途径被激活,C5a 调节 IL-12(+)树突细胞迁移以诱导脓毒症中的致病性 Th1 和 Th17 细胞[28]。脓毒症时 C5a 使其受体表达及 IL-8 水平下降,损害中性粒细胞功能,影响机体炎症反应过程[29],C5a 作为脓毒症炎症反应的中枢介质,其引起的主要病理改变包括全身炎症反应、弥散性血管内凝血(DIC)、菌血症、免疫抑制、脓毒性休克、心力衰竭等[30]。感染可

激活体内免疫系统,即中性粒细胞、巨噬细胞、单核细胞等激活,释放细胞因子及化学因子,使促炎因子及抗炎因子释放,当调控炎症反应的炎症因子调节失控时出现全身炎症反应[31]。

3.3 氧化应激损伤　细菌毒素脂多糖可增加一氧化氮合成酶及 NADPH 氧化酶-4 表达,使活性氮及活性氧增加,出现氧化应激,加重线粒体功能障碍[32],线粒体内增高的活性氧损伤线粒体 DNA,其突变可使细胞核 DNA 甲基转移酶活性降低,最终引起整个细胞 DNA 甲基化降低[33],从而使细胞凋亡增加。相反,抗氧化治疗可以减轻氧化应激、降低炎症因子 IL-6 水平,改善线粒体功能及器官功能障碍[34]。

4　脓毒症与高血糖的关系

4.1 脓毒症对糖代谢的影响　脓毒症时趋化素增加,损害糖代谢过程[7],脓毒症的全身炎症反应诱导胰岛素抵抗[35]。研究[36,37]表明,脓毒症高血糖与白介素-6 相关,白介素-6 可减少胰岛素基因的表达。脓毒症时机体为应激状态,肾上腺皮质激素增加、交感神经兴奋等升糖代谢活性增强,使血糖升高。脓毒症时炎症反应、氧化应激、微循环障碍等病理生理过程导致肝、胰岛、肾等糖代谢调节器官功能障碍,使血糖升高。另外,脓毒症治疗过程使用外源性儿茶酚胺有可能直接引起血糖升高。

4.2 高血糖及其控制水平对脓毒症预后的影响　高血糖增加脓毒症炎症反应,同时降低胰岛素敏感性及减少胰岛素分泌[38],使高血糖进一步加重。高血糖使脓毒症神经病理改变显著,损害胶质细胞活性,增加小胶质细胞[39];高血糖增加急性肾损伤及急性心肌梗死风险[40,41]。高血糖可抑制脓毒症血管内皮细胞功能,同时抑制机体免疫反应[14]。血糖增加鲍曼不动杆菌脂多糖合成,诱发巨噬细胞产生炎症细胞因子[42]。然而,高血糖可能是应激性代谢的特征[43],应激性高血糖可能对脓毒症的预后无害[11],一味地降血糖治疗可能增加脓毒症患者的低血糖风险,而低血糖是严重脓毒症患者院内死亡的独立危险因素[44]。有研究表明,控制脓毒性休克患者血糖能改善其凝血功能及炎症反应[45],血糖降至 4.4 ~ 6.1 mmol/L 能改善脓毒症患者的心功能[46],脓毒症患者入住 ICU 后 72 min 内血糖≤6.6 mmol/L 时是 14 d 内死亡的独立危险因素[47],故并非血糖越低预后越好。当脓毒症患者血糖浓度超过 10.0 mmol/L,应进行降糖治疗,使血糖值控制在 8 ~ 10 mmol/L。Sanches 等[45]研究提示脓毒性休克患者血糖控制 < 8.3 mmol/L 时可改善凝血功能,而在一项临床随机对照试验中,严重脓毒症与脓毒性休克患者中胰岛素强化组(血糖 4.4 ~ 6.1 mmol/L)和胰岛素常规组(血糖 10 ~ 12.2 mmol/L)的病死率并无区别[48]。因此,脓毒症患者的血糖控制水平尚有争议,为预防低血糖及避免高血糖的不利影响,血糖值控制在 10 mmol/L 左右可能对预后较有利,但仍需综合考虑个体差异,如年龄、基础病、并发症等因素。

5　结语

综上所述,脓毒症患者以全身炎症反应为病理基础,相关毒素及炎症介质引起全身血管内皮细胞功能障碍,血管通透性增加,激活免疫反应及相关补体途径,引发炎症风暴,使促炎及抗炎过程失控、氧化应激增加、线粒体功能障碍等,最终导致各器官功能障碍,而高血糖亦参与炎症反应过程、影响机体免疫细胞功能、增加机体氧化应激、影响线粒体功能等,两者有相互促进作用,因此,控制脓毒症患者高血糖状态并且维持在一定水平可能改善患者预后。

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新进展综述

中心静脉导管相关性血栓的研究进展

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[**摘要**]　深静脉导管广泛应用于急危重症抢救，导管相关性血栓为其重要并发症之一，然而仅5%病人有典型症状。该文就导管相关性血栓的流行病学特点、高危因素、临床诊断、防治要点等进行综述。

[**关键词**]　深静脉导管；　导管相关性血栓；　床旁多普勒超声；　代谢组学
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Research progress of central venous catheter-related thrombosis　*DENG Da-zhi, HUANG Xiang-hong. Department of Emergency, the People's Hospital of Guangxi Zhuang Autonomous Region, Nanning* 530021, *China*

[**Abstract**]　Central venous catheters(CVCs) are extensively used in the patients with severe diseases treated in Intensive Care Units. An important complication of CVCs is the development of catheter-related thrombosis(CRT) which becomes symptomatic in approximately 5% of the patients. The epidemiological characteristics, risk factors, symptoms, diagnosis, prevention and treatment of CRT are reviewed in this paper.

[**Key words**]　Central venous catheters(CVCs);　Catheter-related thrombosis(CRT);　Bedside Doppler ultrasound;　Metabonomics

约1/4的ICU患者接受中心静脉导管(central venous catheters, CVC)置入，解剖入路为颈内静脉、锁骨下静脉及股静脉，根据临床需要可选用单腔管、双腔细管、双腔血透管、三腔管等，通过其完成血流动力学监测、高浓度抢救性或刺激性药品输入、静脉营养支持及血液净化治疗等。导管相关性血栓是常