

应激性高血糖比值与急性心肌梗死患者预后关系的研究进展

王馨溢, 陈紫君*

重庆医科大学附属永川医院心血管内科, 重庆

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摘要

葡萄糖代谢异常是心血管疾病高发病率和高病死率的重要因素, 更是急性心肌梗死(AMI)患者再发心肌梗死及全因死亡的重要危险因素。尽早评估影响患者预后的相关危险因素有助于指导治疗及改善预后。自2015年Roberts等学者首次提出应激性高血糖比值(SHR)的概念, 越来越多的研究表明, SHR在危重患者的预后中有一定预测价值。现就SHR与AMI患者预后关系的临床研究进展进行综述, 并探讨其潜在的病理生理机制。

关键词

应激性高血糖比值, 急性心肌梗死, 预后

Research Progress on the Relationship between Stress Hyperglycemia Ratio and Prognosis in Patients with Acute Myocardial Infarction

Xinyi Wang, Zijun Chen*

Department of Cardiology, The Affiliated Yongchuan Hospital of Chongqing Medical University, Chongqing

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Abstract

Abnormal glucose metabolism is an important contributor to the high morbidity and mortality of

*通讯作者。

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cardiovascular disease, it is also a significant risk factor for recurrent myocardial infarction and all-cause mortality in patients with acute myocardial infarction (AMI). Early assessment of the risk factors associated with the patient's prognosis is helpful to guide treatment and improve outcomes. Since Roberts et al. first introduced the concept of the stress hyperglycemia ratio (SHR) in 2015, a growing body of research has demonstrated that SHR holds predictive value for the prognosis of critically ill patients. This review summarizes the recent advancements in clinical research concerning the relationship between SHR and the prognosis of patients with AMI, and explores its potential pathophysiological mechanisms.

Keywords

Stress Hyperglycemia Ratio, Acute Myocardial Infarction, Prognosis

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1. 引言

应激性高血糖比值(stress hyperglycemia ratio, SHR)是一种反映急性应激状态下相对高血糖水平的新型生物标志物,其在危急重症患者预后中的预测价值已被证实[1]。而急性心肌梗死(acute myocardial infarction, AMI)是心血管疾病中可致死致残的危急重症[2],因此及时进行风险评估有助于治疗方案的制定,同时对改善患者预后有一定作用。现结合目前最新发表的相关研究结果对 SHR 与 AMI 患者预后的关系及潜在的病理生理机制进行综述。

2. SHR 的定义及计算方法

SHR 是指在强烈刺激因素作用下应激血糖与平均血糖的比值[3],但其计算方法尚存在一定争议。一是基于入院血糖(admission blood glucose, ABG)和糖化血红蛋白(glycosylated hemoglobin, HbA1c)推导出的 SHR: $[ABG (\text{mmol/L})]/[1.59 \times HbA1c (\%) - 2.59]$ [4],二是根据 ABG 和糖化白蛋白(glycated albumin, GA)计算得出 SHR: $[ABG (\text{mmol/L})]/[GA (\%)]$ [5]。目前已证实上述两种算法得出的 SHR 对患者预后均有一定预测价值[6]。

与 GA 相比, HbA1c 揭示机体在检测前 2~3 月内的平均血糖水平,稳定性较好,不易受其他因素干扰[7],更能有效反映患者基础血糖情况。因此,目前大多数研究以 ABG 和 HbA1c 为基础推导出 SHR。

3. SHR 与 AMI 患者预后的关联

多项研究已证实 SHR 与 AMI 患者的不良预后存在相关性,现从主要不良心血管事件(Major adverse cardiovascular events, MACE)及死亡率、不同亚组人群间的异质性、并发症风险预测、剂量反应关系等四个方面进行分析。

3.1. MACE 和死亡率

有研究[8]显示接受冠状动脉旁路移植术(coronary artery bypass grafting, CABG)的 AMI 患者中, SHR 最高三分位组的 90 天和 180 天死亡率分别是最低组的 3.78 倍和 4.19 倍(P 均 <0.001 , P for trend <0.001),这表明 SHR 升高与因 AMI 接受 CABG 手术患者的短期死亡率升高相关。另一项多中心研究[9]表明,在接受 PCI 治疗的 AMI 患者中, SHR 与住院期间 MACE 独立相关,但随访 SHR 较高组($SHR > 1.45$)与出

院后 MACE 无关, 这提示 SHR 与患者长期预后关系有待进一步验证。通过奥格斯堡心肌梗死登记库[10]纳入 2311 例 AMI 患者, 中位随访 6.5 年后, 发现较高的 SHR 与 28 天死亡率呈显著非线性正相关, 且在无糖尿病患者中关联更强(SHR = 0.6912, 95% CI 0.6317~0.7496, P = 0.0351), 但对 5 年死亡率的预测能力有限。

一项基于多中心 OPTIMAL 研究[11]通过纳入 3384 例接受经皮冠状动脉介入治疗(percutaneous coronary intervention, PCI)治疗的 AMI 患者, 中位随访时间 24.1 个月, 发现随着 SHR 四分位数升高, 生存率逐渐降低(log-rank P < 0.001), 并运用多变量 Cox 回归分析证实, 与 Q2 组(SHR: 0.93~1.11)相比, SHR 是 Q4 组(SHR \geq 1.34)患者心血管死亡(HR = 1.56)、全因死亡(HR = 1.48)和 MACE (HR = 1.34)的独立预测因素。另一项研究[12]通过搜集来自 11 家医院接受 PCI 的急性冠脉综合征(acute coronary syndrome, ACS)患者数据, 中位随访 31.33 个月后, 结果显示 SHR 是接受 PCI 的 ACS 患者长期 MACEs 的独立预测因素(HR = 3.3479, 95% CI 1.4103~7.9475, P = 0.0062)。大型全国前瞻性队列研究[13]通过纳入 7662 例 ACS 患者中位随访 2.1 年后, 结果提示 SHR 最高三分位组的 ACS 患者与 MACE 的长期风险显著相关(HR = 1.53, 95% CI 1.24~1.88)。另一研究[14]通过中位随访 36 个月接受 PCI 的 AMI 患者, 发现当 SHR > 1.317 时, 主要不良心脑血管事件(major adverse cardiovascular and cerebrovascular events, MACCE)风险显著升高(HR = 2.279, 95% CI 1.569~3.311, P < 0.001), 且该关联在不同性别、年龄及糖尿病状态亚组中均稳定存在, 并能独立预测接受 PCI 的 AMI 患者的预后(HR = 1.998, 95% CI 1.299~3.074, P = 0.002), ROC 曲线下面积为 0.636 (95% CI 0.613~0.659, P < 0.05)。美国与中国多中心队列研究[15]也证实, SHR 升高与 AMI 患者 1 年及长期全因死亡率存在显著相关性, 尤其在非糖尿病患者中关联更为明显(MIMIC-IV 队列: aHR = 1.87, 95% CI 1.40~2.50; CIN-II 队列: aHR = 1.44, 95% CI 1.03~2.02)。一项纳入 87,974 例患者的荟萃分析[16]显示, SHR 处于上四分位数的 AMI 患者, 其 MACCE 发生风险较下四分位数患者升高 70% (HR = 1.7, 95% CI 1.42~2.03, P < 0.001)、长期风险增加 64% (HR = 1.64, 95% CI 1.49~1.8, P < 0.001)。

以上研究说明长期随访数据同样支持 SHR 与 AMI 患者预后存在显著相关性这一结论。

3.2. 不同亚组人群差异

SHR 对 AMI 患者预后的预测价值在不同临床亚组中存在异质性。

3.2.1. 阻塞性冠心病(Obstructive Coronary Artery Disease, OCAD)与非阻塞性冠心病(Non-Obstructive Coronary Artery Disease, NOCAD)

大型荟萃分析[13]提示 SHR 在阻塞性与非阻塞性冠心病患者中均呈现显著相关性。在平均 34 个月的随访期间[17], 冠状动脉非阻塞性心肌梗死(myocardial infarction non-obstructive coronary artery, MINOCA)患者中 SHR \geq 0.84 组的 MACE 发生率(35.3%)显著高于 SHR \leq 0.73 组(15.4%), 首次表明 SHR 与 MINOCA 患者的长期预后不良独立相关, 且 SHR = 0.86 是预测 MACE 的最佳临界值(HR = 2.465, 95% CI 1.461~4.159, P = 0.001)。另一项包含 1179 例 MINOCA 患者的前瞻性队列研究[18]中, 中位随访 3.5 年后, 发现随着 SHR 三分位水平升高, MACE 的发生率显著增加(8.1%、14.0%、20.5%, P < 0.001), 且与 MACE 风险增加独立相关(HR = 2.30, 95% CI 1.2~4.38, P = 0.011), 在预测 MACE 方面的曲线下 SHR 面积为 0.63。

3.2.2. 是否合并糖尿病(Diabetes Mellitus, DM)

多项研究显示无论患者是否患有 DM, SHR 是 AMI 患者短期及长期预后强有力的独立预测因子。中国急性心肌梗死中心研究[19]结果显示无论是否合并 DM, SHR 四分位数最高组的住院死亡率均显著高于最低组(DM 患者: 9.7% vs 2.0%, OR = 4.070, 95% CI 2.014~8.228; 非 DM 患者: 8.8% vs 2.2%, OR =

2.976, 95% CI 1.695~5.224)。

3.2.3. 是否合并低蛋白血症

在 AMI 患者中, 与低 SHR 且白蛋白正常的患者相比, 高 SHR 合并低蛋白血症患者的 MACEs 发生率更高(OR = 6.800, 95% CI 3.883~11.907, $P < 0.001$)。但无论基线白蛋白水平如何, 高 SHR 均与 MACEs (交互作用 $P = 0.866$)以及心脏休克或急性充血性心力衰竭的心血管风险增加相关[20]。

3.2.4. 是否合并心房颤动(Atrial Fibrillation, AF)

通过利用 MIMIC-IV (2.2 版)数据库[21]中 3233 例首次诊断为 AMI 患者的数据进行队列研究, 研究发现: 对于合并 AF 的 AMI 患者, SHR 与死亡率呈 U 型关联, 拐点为 1.09, 低于(OR = 0.30, 95% CI 0.10~0.94)或高于(OR = 3.28, 95% CI 2.01~5.34)该值均会增加死亡风险, 而在无 AF 的 AMI 患者中则未发现这种关系。这表明房颤是造成这种差异的一个因素, 它增加了低 SHR 的 AMI 患者的死亡风险。

3.2.5. 是否合并射血分数降低

在射血分数降低的 AMI 患者[22]中, 与血糖变异性(glycemic variability, GV)相比, SHR 与住院死亡率的相关性更强(OR = 1.51, 95% CI 1.24~1.82), 在非糖尿病患者中更为显著。

3.2.6. 是否合并斑块破裂(Plaque Rupture, PR)

按斑块表型分层发现[23], SHR > 1.2 对 PR 患者的 MACE 预测价值显著(HR = 2.09, 95% CI 1.17~3.73, $P = 0.013$), 但对斑块侵蚀但未破裂患者无显著预测意义。

3.3. 并发症风险预测

3.3.1. 支架内血栓形成

一项回顾性研究[24]显示, SHR 是 ST 段抬高型心肌梗死(ST-segment elevation myocardial infarction, STEMI)患者 PCI 术后院内支架内血栓(stent thrombosis, ST)形成的独立预测因子, 当 SHR ≥ 1.26 时, ST 发生风险显著升高(OR = 3.15, 95% CI 1.88~5.27, $P < 0.001$), 而入院高血糖(admission hyperglycemia, AH)无显著预测价值($P = 0.182$)。

3.3.2. 心功能损伤与微血管并发症

一项[25]纳入 357 例 STEMI 患者的研究发现, SHR ≥ 1.01 组的左心室射血分数(left ventricular ejection fraction, LVEF)和左心室整体功能指数(left ventricular global function index, LVGFI)显著低于 SHR < 0.85 组, 且 SHR 与 LVEF 呈负相关($r = -0.252$, $P < 0.001$), 是左心室功能障碍的独立决定因素, 在校正协变量后的回归分析中显示, SHR 与 AMI 患者的微血管阻塞(microvascular obstruction, MVO)存在一定程度相关。

此外, SHR 还是心肌内出血(intramycardial hemorrhage, IMH)的强预测因子, 一项[26]多中心 EARLY-MYO-CMR 注册研究数据表明, SHR 每升高 0.1 单位, IMH 风险增加 21% (aOR = 1.21, 95% CI 1.10~1.33, $P < 0.001$), 且该关联在糖尿病与非糖尿病患者中均成立。但值得注意的是, 即使在较低的 SHR 水平下, 微血管损伤的风险也会迅速升高。

同时, AMIPE 多中心前瞻性研究[27]表明, SHR > 1.14 与非 ST 段抬高型心肌梗死(non-ST segment elevation myocardial infarction, NSTEMI)患者 PCI 术后 4a 型心肌梗死风险增加相关(aOR = 2.73, 95% CI 1.70~4.42, $P < 0.001$), 其预测准确性(AUC = 0.69)优于 ABG 和 HbA1c。

3.3.3. 其他并发症

在危重症 AMI 患者中, SHR 与急性肾损伤(acute kidney injury, AKI)风险呈 J 型关联, 是 AKI 发生的

早期独立标志物。然而, Cox 回归分析表明, 无论是在总体队列还是在 AKI 亚组中, SHR 均不是医院死亡的独立预测因素[28]。

3.4. SHR 与预后关联的剂量反应关系

多数研究[1]显示, SHR 与 AMI 患者不良预后呈非线性关联, 主要表现为 J 型或 U 型曲线, 表明低 SHR 和高 SHR 均与死亡率和发病率增加相关。

在接受药物洗脱支架(drug-eluting stent, DES)植入的 ACS 患者[29]中, SHR 与 2 年随访期间的 MACCE 率和 MACE 率呈 U 型关联, 与住院期间和 2 年随访期间的心脏死亡呈 J 型关联, 拐点为 0.78, 低于或高于该值均会增加风险。在 PCI 术后 AMI 患者中[8], SHR 与长期不良结局呈 J 型关联, SHR 约为 1.0 时风险最低, 高于 1.34 时心血管死亡风险显著升高(HR = 1.56)。

而在无 AF 的 AMI 患者中, SHR 与死亡率呈线性关联, SHR 越高, 死亡风险越高(OR = 1.95, 95% CI 1.52~2.51) [18]。

4. SHR 与现有评分系统的关系

有研究[30]将 SHR 纳入新型预后评估系统, 结果显示对 AMI 患者短期 MACE 具有良好的预测价值。同时 SHR 可能有助于提高 TIMI 风险评分在 STEMI 患者中的预测效率(每增加 1 单位 SHR, AUC 增量 0.009, $P < 0.05$), 尤其是对于 DM 患者[31]。SHR 也显著提高了 GRACE 评分性能(C 统计量从 0.706 (95% CI 0.599~0.813)提高到 0.727 (95% CI 0.616~0.837) ($P < 0.01$)), NRI = 0.305, IDI = 0.042 [12], 另一研究[32]将 SHR 纳入 GRACE 评分同样证实其可显著提升预测效能(NRI = 0.184, IDI = 0.014)。

5. 潜在的病理生理机制

SHR 与 AMI 患者不良预后的关联机制尚未完全明确, 目前认为主要与以下途径相关: 1) 神经内分泌与炎症反应激活[1] [33]: ① 应激状态下儿茶酚胺、皮质醇释放增加, 导致胰岛素抵抗和肝糖异生增强, 从而引起血糖急剧升高; ② 高血糖和应激又共同刺激炎症因子释放, 一方面加重胰岛素抵抗, 另一方面直接损伤血管内皮细胞, 抑制一氧化氮(NO)合成, 加剧氧化应激, 从而促使冠状动脉内皮屏障功能受损, 引起血管内斑块不稳定甚至破裂; 2) 血栓形成风险增加: 在 STEMI 合并 DM 患者中, $SHR \geq 1.19$ 与冠状动脉内大量血栓负荷(large thrombus burden, LTB)独立相关(AUC = 0.669, 95% CI 0.604~0.730, $P < 0.001$, 灵敏度 71.4%, 特异度 64.7%), 提示高 SHR 可加重冠状动脉血栓负荷[34], 并通过促进血小板聚集和凝血因子激活参与血栓形成过程; 3) 微血管功能障碍[22] [23]: SHR 升高可削弱微血管完整性, 诱发 IMH 和微循环阻塞, 进而导致心肌灌注不足和心功能损伤; 4) 全身代谢紊乱[35]: 持续相对高血糖状态可影响能量代谢、加剧细胞内酸中毒, 同时高血糖可抑制缺血区心肌细胞及血管内皮细胞的修复, 并且全身性代谢紊乱可增加多器官并发症风险。

6. 总结

SHR 作为整合急性与慢性血糖状态的新型生物标志物, 在 AMI 患者的风险分层、预后评估及治疗指导中展现出独特优势[1]。大量循证医学证据表明, SHR 升高与 MACE、死亡率及多种并发症风险增加密切相关, 且在不同临床亚组中具有稳定的预测价值[13]。

然而, 目前仍存在诸多争议: SHR 的最佳临界值在不同人群中尚未统一[14], 虽然 SHR 与传统风险评分系统联合评估患者病情时可增强其评估效益, 但在不同临床情境下的应用及其与其他生物标志物的联合预测效能仍需进一步探索[29]。未来需开展大样本、长期随访的前瞻性研究, 明确不同临床场景下

SHR 的最佳阈值, 还可利用组学技术进一步阐明 SHR 介导心肌损伤的分子通路, 可为开发靶向药物治疗提供理论基础。另外, 通过设计随机对照试验, 探索基于 SHR 的个体化血糖管理方案对比常规降糖方案在降低 MACE 与死亡率的有效性, 进一步提升 AMI 患者的精准医疗水平。

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