

• 综 述 •

血常规衍生免疫炎症标志物与冠状动脉疾病的相关性研究进展

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[摘要] 炎症在冠状动脉疾病(CAD)的形成和进展中扮演着重要的角色。以往的研究广泛探讨了传统的炎症相关生物指标在评估 CAD 风险和预后方面的重要性。近年来,基于血常规衍生的免疫炎症标志物如系统性免疫炎症指数、全身炎症反应指数和全身炎症聚合指数等的研究逐渐受到关注,这些指标因简便、高重复性和独特地反映了炎症与免疫状态而成为研究热点。该文综述了这些炎症标志物的研究进展,探讨其在 CAD 预测和预后分析中的应用价值,并展望未来研究方向。

[关键词] 冠状动脉疾病; 动脉粥样硬化; 炎症; 免疫炎症标志物; 综述

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**Research progress on the correlation between immune inflammatory markers
derived from blood routine and coronary artery disease**

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[Abstract] Inflammation plays a critical role in the formation and progression of coronary artery disease (CAD). Previous studies have extensively explored the significance of traditional inflammation-related biomarkers in assessing CAD risk and prognosis. In recent years, the research on immune inflammatory markers derived from hematological parameters, such as the systemic immune-inflammation index (SII), systemic inflammation response index (SIRI), and aggregate systemic inflammation index (AISI), has gained increasing attention. These markers have become research hotspots due to their simplicity, high reproducibility, and unique ability to reflect inflammatory and immune states. This article reviews the progress in research on these inflammatory markers, discusses their application value in CAD prediction and prognosis analysis, and provides an outlook on future research directions.

[Key words] Coronary artery disease; Atherosclerosis; Inflammation; Immune inflammatory markers; Review

冠状动脉疾病(CAD)是导致残疾和死亡的主要因素之一。动脉粥样硬化(AS)作为 CAD 的核心病理过程,炎症在其发生、发展中至关重要。AS 是一种涉及众多炎细胞与因子相互影响的慢性炎症反应^[1]。在早期阶段,血管内皮损伤会释放出黏附分子包括血管细胞黏附分子 1(VCAM-1)、E-选择素和 P-选择素等,它们的表达上调促进了淋巴细胞和单核细胞与内皮细胞之间的黏附,并在血管壁中激发炎症反应^[2]。在进展过程中,巨噬细胞等炎症细胞浸润血管壁,活化的肥大细胞进一步促进炎症微环境的形成^[3],导致局部炎症反应加剧,加速动脉硬化进程。此外,内皮损伤和功能障碍还会引发先天和适应性免疫反应,促使白细胞、中性粒细胞和单核细胞释放到血液循环中,从而促进巨噬细胞分化、T 细胞增殖及细胞因子

的产生^[4]。在免疫反应过程中,多种细胞类型包括巨噬细胞、T 细胞、B 细胞等参与其中,它们构建了一个复杂的免疫调节网络,以保护机体不受外界威胁。

经典的炎症标志物已被证实与 CAD 的发生和预后显著相关。近年来,基于血常规免疫炎症标志物,因其简便、经济、可重复性强,且能反映免疫状态和炎症的独特优势而成为研究热点。这些新型炎症标志物的研究不仅补充了传统标志物的不足,也深化了对 CAD 炎症机制的理解。本文综述了血常规衍生免疫炎症标志物的研究进展,探讨其在 CAD 风险预测和预后评估中的应用价值。

1 血细胞与 AS

血细胞在 AS 中发挥着多方面的重要作用,其涉及免疫反应、内皮损伤、斑块形成及不稳定性。中性

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粒细胞、淋巴细胞、血小板和单核细胞通过复杂的相互作用,推动 AS 的进展并影响斑块的稳定性。

1.1 中性粒细胞 中性粒细胞是人体免疫系统的重要组成部分,在 AS 的形成、进展和斑块不稳定性中起关键作用。在 AS 的初始阶段,活化的中性粒细胞通过脱颗粒促进单核细胞的募集,并分泌活性氧(ROS)和各类蛋白酶,导致内皮细胞层失调,促使低密度脂蛋白胆固醇(LDL-C)外渗^[5]。随着病程进展,中性粒细胞还会产生髓过氧化物酶(MPO),参与 LDL 的氧化,进一步促进泡沫细胞形成^[6]。在晚期,活化的血管平滑肌细胞(VSMCs)分泌 CC 趋化因子配体 7(CCL7),进一步驱动中性粒细胞分泌有细胞毒性组蛋白 H4 的中性粒细胞胞外网(NETs)。NETs 通过多种机制促进 AS 的进展,包括促进血栓形成、加剧内皮损伤、诱导平滑肌细胞死亡、放大局部炎症反应,并且可能导致斑块的不稳定性和破裂^[7-8]。

1.2 淋巴细胞 淋巴细胞是 AS 相关免疫反应的关键组成部分,尤其是 T 淋巴细胞。不同 T 细胞亚群通过分泌细胞因子或直接与其他免疫细胞相互作用,对 AS 的进展和斑块的稳定性产生重要影响。在晚期病变中,CD4⁺ T 淋巴细胞通过分泌 IL-2、TNF,以及促进 IL-18、IL-12 的释放,与巨噬细胞和血管细胞相互作用,进而加速斑块形成^[9]。相反, T 调节细胞(Tregs)对 AS 具有保护作用。这些细胞通过抑制树突状细胞的抗原呈递功能,削弱先天免疫和适应性免疫之间的炎症桥接^[10-12]。B 淋巴细胞的作用在 AS 中表现出双重性。B1 细胞通过产生针对氧化低密度脂蛋白(ox-LDL)的 IgM 抗体,展现出保护作用;而 B2 细胞通过分泌 IgG 和 IgE,则可能促进 AS 的进展^[13-15]。

1.3 血小板 血小板在 AS 中既参与斑块形成,又在斑块损伤后的血栓形成中发挥重要作用。在 AS 过程中,血小板聚积于病变处并与活化的内皮细胞和白细胞交互作用,促进泡沫细胞形成和斑块的增大^[16]。在斑块破裂时,血小板与细胞外基质(ECM)和组织因子(TF)相互作用,激活凝血,促成血栓形成,从而加剧血管堵塞和心血管事件的发生^[17-20]。

1.4 单核细胞 单核细胞在 AS 中的作用也十分重要,血管内皮细胞的损伤会导致黏附分子(如 VCAM-1 和 ICAM-1)的表达上升,这一变化促进了单核细胞在血管内壁的黏附^[21]。在趋化因子(如 MCP-1)作用下,单核细胞迁移至内皮下,转化为巨噬细胞,吞噬 ox-LDL,进而形成泡沫细胞^[5,22]。此外,单核细胞和巨噬细胞还可以通过分泌基质金属蛋白酶(MMPs)削弱纤维帽的强度,从而导致斑块不稳定性^[23]。

2 血常规衍生免疫炎症标志物

2.1 中性粒细胞与淋巴细胞比值(NLR) NLR 来源于绝对中性粒细胞计数除以绝对淋巴细胞计数。其能够较好地反映体内的炎症状态和免疫功能失衡。

多项研究表明,NLR 的增高与 CAD 的严重性及其负面预后有着紧密的联系。在心肌梗死患者中,NLR 与冠状动脉病变程度呈正相关,提示 NLR 可以作为评估冠状动脉病变严重程度的指标^[24]。相关研究指出,NLR 与 TIMI 评分显著相关,高 NLR 组的 TIMI 评分显著升高,反映出更大的心肌损伤和更差的预后^[25]。多项荟萃分析和系统评价一致认为,NLR 升高的急性冠脉综合征(ACS)患者在短期内发生不良心脏事件(MACE)和全因死亡的风险显著增加,且在不同随访时长和研究设计中均表现出稳健的预测能力^[26-27]。

在不同人群中,NLR 的预测价值也得到验证。对于冠状动脉慢性完全闭塞(CTO)患者,高 NLR 水平预测了 32 个月内 MACE 发生率的显著增加,是其独立预测因子^[28]。在川崎病患者中,NLR 被证实是冠状动脉损害的早期独立预测因子^[29]。

此外,NLR 与其他炎症标志物或指数联合使用,可提高对 CAD 的预测能力。WANG 等^[30]发现 NLR 与甘油三酯-葡萄糖指数(TyG)联合使用,在 STEMI 患者 PCI 后 MACE 风险预测中效果优于单独使用的 GRACE 评分模型,增强了预测准确性。YUAN 等^[31]在 ACS 患者中,NLR 和 LDL-C/HDL-C 联合预测冠状动脉病变严重程度的敏感性和特异性较高,灵敏度为 87.1%,特异性为 90.9%。

2.2 血小板与淋巴细胞比值(PLR) PLR 融合了与血栓形成及炎症相关的指标,展示了血小板活化与炎症反应的协调状态。在预测 AS 斑块负荷及心血管结局方面,PLR 可能比单一的血小板或淋巴细胞计数更为有效。

对于冠状动脉病变的预测,一项针对接受冠状动脉造影心绞痛患者的研究发现,重度 CAD 组的 PLR 显著高于其他组,且 PLR 与 Gensini 评分及 CRP 呈正相关。即便是在控制年龄、性别等可能影响结果的因素之后,PLR 仍是判断重度 CAD 的一个独立预测指标^[32]。在不稳定型心绞痛(UA)和非 ST 段抬高型心肌梗死(NSTEMI)患者中,HEART 评分与 PLR 显著正相关,尤其在高风险 HEART 组,表明 PLR 在高危心脏事件预测中具有独立性和可靠性^[33]。

在 ACS 患者中的预后评估中,近期的系统评价表示高 PLR 与住院期间及长期的 MACE 风险增加相关^[34]。急性心肌梗死(STEMI)患者高 PLR 与血小板活化和炎症水平升高相关,是心肌再灌注效果差及冠状动脉血栓负担重的独立预测因子^[35]。

2.3 淋巴细胞与单核细胞比值(LMR)和单核细胞与淋巴细胞比值(MLR) LMR 和 MLR 通过反映淋巴细胞和单核细胞的平衡,提供了炎症状态的线索。近年来,大量研究与临床实践逐渐验证了 LMR 在慢性炎症过程中扮演着重要角色。SI 等^[36]研究显示,CAD 患者的 LMR 显著低于非 CAD 患者,且与冠状

动脉钙化得分(CAC)呈显著负相关,进一步分析表明,LMR ≤ 4.8 可作为CAD的独立风险因子,预测能力优于高血压和年龄等传统风险因素。YANG等^[37]的回顾性分析指出,LMR是孤立性冠状动脉扩张症中对慢血流(CSF)的独立负相关预测因子,强调低LMR与增强的炎症反应及血管内皮损伤相关。低LMR是冠心病的一个独立危险因素,KOSE等^[38]研究结果显示,高SYNTAX评分组(>32)的LMR进一步下降,成为高SYNTAX评分的独立预测因子。此外,低LMR与支架内再狭窄(ISR)发生有显著关系,尤其当LMR <2.86 时,ISR风险显著增加,提示LMR与冠状动脉支架术后炎症反应密切相关^[39]。在预后方面,在对STEMI患者的研究显示,入院时低LMR者在长期随访中发生MACE及全因死亡的风险显著增加^[40]。

关于MLR,有研究指出高MLR患者的全因死亡率及心脏死亡率显著增加,表明MLR是MACE的独立风险因素^[41]。近年来,VAKHSHOORI等^[42]的荟萃分析进一步验证,高MLR显著增加MACE风险,在ACS患者中风险更高。

2.4 系统性免疫炎症指数(SII) SII是一个通过白细胞计数得出的指标,是中性粒细胞和血小板的计数相乘,然后除以淋巴细胞计数。SII结合了血小板、中性粒细胞和淋巴细胞,提供整体炎症和免疫状态的信息。SHENG等^[43]研究发现,高SII水平是不稳定斑块(ISNA)及薄帽纤维斑块(TCFA)的独立预测因子,提示了其在评估斑块稳定性方面的潜力。在稳定型冠心病患者中,SII与SYNTAX评分显著相关,并作为独立预测因子^[44]。此外,SII的升高是增加ISR风险的独立因素,且为显著风险因子^[45]。近年来,SII作为一种新型的炎症指标,已经在冠心病、ACS等疾病的预后评估中展现出较强的预测能力。荟萃分析显示,SII升高的PCI患者后续MACE风险显著增加^[46]。在ACS患者中,SII是MACE的独立预测因子,并显著提高传统风险模型的预测效能^[47]。在特定患者群体,对于CTO患者,高SII与冠状动脉侧支循环(CCC)发育不良显著相关^[48]。在糖尿病或糖尿病前期患者中,SII与ACS风险显著相关,结合LDL-C和HDL-C的预测模型准确性显著提高^[49]。

2.5 全身炎症反应指数(SIRI) SIRI结合中性粒细胞、单核细胞和淋巴细胞计数,公式: $SIRI = (\text{中性粒细胞计数} \times \text{单核细胞计数}) / \text{淋巴细胞计数}$ 。作为一个更全面的指标,SIRI深化了对炎症反应的评估。在评估冠状动脉病变方面,SIRI与Gensini评分呈显著正相关,预测能力优于NLR、MLR和PLR,凸显了其在冠心病风险评估中的优势^[50]。SIRI在复杂CAD、多支病变患者和ISR中,均表现出较高的预测效能^[45,51]。在联合检测上,有研究显示SII联合单核细胞计数比高密度脂蛋白比值(MHR)检测,更能提高

对冠状动脉病变程度预测的准确性^[52]。

在预后评估中,针对STEMI患者的研究显示,SIRI高水平可预测30 d内MACE的发生,尤其在炎症状态较低的患者中预测效果更佳^[53]。在肥胖人群中,SIRI能独立预测全因和心血管相关的死亡风险,每增加一个标准差,死亡率分别增加16%和22%^[54]。此外,将SIRI与TyG结合,可增强对死亡风险的预测能力^[55]。

2.6 全身炎症聚合指数(AISI) AISI也称泛免疫炎症值(PIV)或全身免疫炎症反应指数(SIIRI),是中性粒细胞、单核细胞和血小板计数相乘除以淋巴细胞计数得出。AISI作为一种新型指标,综合反映中性粒细胞、单核细胞、淋巴细胞和血小板的水平,强调了炎症细胞与免疫细胞的综合影响,为炎症与免疫的相互作用研究提供了新视角。

在评估冠状动脉病变方面,对NSTEMI患者的研究发现,PIV与SYNTAX评分呈显著正相关,在预测高SYNTAX评分时表现出超高的敏感性和特异性,显示其评估冠状动脉病变严重程度的潜力^[56]。在ISR风险预测中,AISI在ISR组患者中显著升高,与ISR发生率呈正相关,通过随机森林模型分析,AISI在ISR风险预测中表现最佳^[57]。

在不良预后的预测中,一项回顾性分析纳入959例首次诊断为CAD的患者作为研究对象,研究显示SIIRI是MACE的独立预测因子,结合传统风险因素后,SIIRI的预测效能显著提升^[58]。在ACS患者中,AISI被证实为预测不良结局的重要独立因子,能够有效预测ACS患者的不良结局^[59]。在STEMI患者中,住院期间发生MACE的患者PIV显著高于未发生MACE者,且PIV在预测MACE方面优于SII、PLR、NLR^[60]。

3 小结与展望

炎症在CAD的发生、发展及急性事件触发中起核心作用。基于血常规衍生的免疫炎症标志物(如NLR、PLR、LMR、SII、SIRI、AISI等)因其易得、低成本,展示了一定的临床应用潜力。这些标志物不仅加深了对CAD中炎症作用机制的理解,还为CAD的早期筛查、风险预测和预后管理提供了新的参考方向。尽管这些标志物在临床研究中表现出较好的应用前景,但仍需通过更多的大规模、多中心临床研究来验证其广泛适用性。未来的研究应该进一步探索这些标志物的临床价值,尤其是将其与其他生物学指标或影像学参数相结合进行综合评估,以便更精准地预测CAD的短期和长期风险,指导临床决策。或借助大数据和人工智能分析寻找新的炎症标志物,深入揭示CAD的炎症驱动机制,推动个性化诊疗的实现。

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