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NLR、PLR与维持性血液透析患者合并心血管疾病的研究进展

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摘　要

近年来，慢性肾脏疾病(chronic kidney disease, CKD)发病率持续上升。血液透析(haemodialysis, HD)作为危重症或晚期肾脏病患者主要肾脏替代疗法，极大地延长了患者的生存期，但同时也伴随着一些并发症，特别是心血管疾病已成为终末期肾病(end-stage renal disease, ESRD)患者最常见的并发症和死亡的主要原因，中性粒细胞淋巴细胞比值(neutrophil to lymphocyte ratio, NLR)、血小板淋巴细胞比值(platelet to lymphocyte ratio, PLR)是近期发现的新型炎症标志物。研究表明，NLR是影响MHD患者合并心血管疾病发生的危险因素，而PLR则是MHD患者全因死亡率和心血管死亡率的独立预测指标。目前，在临床实践中，一些传统的炎症标志物，如IL-6和TNF-α，在MHD患者中的常规检测存在困难，不仅不方便，而且成本高昂，相比之下，NLR和PLR作为简单、易于获得且具有成本效益的新型炎症标志物，可以在缺乏其他昂贵炎症标志物测试的情况下，帮助临床医生评估MHD患者的微炎症状态和预后。深入研究NLR和PLR与MHD患者合并心血管疾病的相关性，可能为MHD患者并发症管理提供新的生物标志物和治疗靶点。

关键词

维持性血液透析，心血管疾病，中性粒细胞淋巴细胞比值，血小板淋巴细胞比值

Research Progress on Cardiovascular Diseases in Patients Undergoing NLR, PLR and Maintenance Hemodialysis

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Abstract

In recent years, the incidence of chronic kidney disease (CKD) has continued to rise. Hemodialysis (HD), as the main renal replacement therapy for critically ill or advanced kidney disease patients, has greatly prolonged the survival period of patients, but it is also accompanied by some complications. In particular, cardiovascular diseases have become the most common complication and the main cause of death for patients with end-stage renal disease (ESRD). The neutrophil to lymphocyte ratio (NLR) and the platelet to lymphocyte ratio (PLR) are novel inflammatory markers discovered recently. Studies have shown that NLR is a risk factor influencing the occurrence of cardiovascular diseases in MHD patients, while PLR is an independent predictor of all-cause mortality and cardiovascular mortality in MHD patients. At present, in clinical practice, the routine detection of some traditional inflammatory markers, such as 1L-6 and TNFa, in MHD patients is difficult. It is not only inconvenient but also costly. In contrast, NLR and PLR, as simple, easily accessible and cost-effective new inflammatory markers, It can help clinicians assess the micro-inflammatory status and prognosis of MHD patients in the absence of other expensive inflammatory marker tests. In-depth research on the correlation between NLR and PLR and cardiovascular diseases in MHD patients may provide new biomarkers and therapeutic targets for the management of complications in MHD patients.

Keywords

Maintenance Hemodialysis, Cardiovascular Diseases, Neutrophil-Lymphocyte Ratio, Platelet-Lymphocyte Ratio

1. 引言

慢性肾脏病(Chronic Kidney Disease, CKD)及其终末阶段——终末期肾病(End stage renal disease, ESRD)已成为全球严重的公共卫生问题[1]。据统计，1990 年至 2017 年间，全球的 CKD 死亡率增加了 41.5% [2]。而我国同期增长率高达 152.7%，远远高于全球 CKD 死亡率的增长速度[3]。按此趋势，CKD 预计将在 2040 年成为全球导致寿命损失的第五大常见原因[4]。CKD 是指肾脏损伤或不明原因导致的肾小球率过滤(glomerular filtration rate, GFR)小于 60 ml/min/1.73m^2 超过 3 个月的疾病[5]，如果患者未得到及时有效的治疗，随着肾实质不可逆性损害，可出现肾脏器萎缩，并逐渐发展为 ESRD [6]。当 GFR ≤ 15 ml/min/1.73m^2 时诊断为 ESRD。肾脏替代治疗是 ESRD 患者的有效治疗方法，通常包括血液透析

(Hemodialysis, HD)、腹膜透析和肾移植，其中血液透析是 ESRD 患者最常用、最安全的肾脏替代治疗方式，可显著延长患者生存期[7] [8]。然而，HD 不可避免会伴随某些并发症[9]，如心血管事件、感染甚至死亡等。其中心血管事件是 MHD 患者常见的并发症及死亡的首要原因[10]。占该类患者死亡原因的 40%~60%，是普通人群的 10~20 倍[11]。在 MHD 患者中，除传统危险因素外，“微炎症状态”被广泛认为是 CVD 发生与进展的重要推手[12]。是 MHD 患者并发心血管疾病的促进因素，也是肾衰竭患者因心血管并发症死亡的预测因子[13]。该状态表现为低水平、持续的炎症状态[14] [15]。通常表现为体内炎症标志蛋白和炎症因子水平持续升高[16]。临床上以 hs-CRP 在 3~10 mg/L 为常用判定标准[17]。MHD 患者中约 30%~65%存在此现象[18]-[20]。其与动脉粥样硬化、冠心病、心律失常、心力衰竭及静脉血栓形成密切相关，并可预测不良预后[16] [21]。如今 CRP、白细胞介素 1 和 6 以及 TNF-α 作为炎症标志物其效能已被认可[22] [23]。但是，在目前的社会经济状况下，我们需要寻找成本效益更高的生物标记物。近年来，中性粒细胞淋巴细胞比值(neutrophil to lymphocyte ratio, NLR)、血小板淋巴细胞比值(platelet to lymphocyte ratio, PLR)因检测简单、成本低廉，被视为新型炎症标志物。大量研究证实：NLR 与 CRP、IL-6 及 TNF-α 呈显著正相关，动态趋势高度一致[24] [25]。回顾性分析 43,272 例血液透析患者，发现 NLR 甚至可以作为 CRP 的潜在替代标志物，用于评估 CKD 患者全身炎症状态[26] [27]。PLR 同样与 CRP、白介素 6、肿瘤坏死因子等水平呈正相关[28] [29]。本文将综合目前的研究结果，阐述 NLR 及 PLR 在 MHD 并发心血管疾病患者中的研究进展，探讨 NLR 及 PLR 在 MHD 并发心血管疾病患者中的预测、诊断和管理中的潜在价值，为今后的研究提供依据。

2. NLR、PLR 的生物学特性和检测

NLR 定义为外周血中性粒细胞绝对计数与淋巴细胞绝对计数之比；PLR 则为血小板绝对计数与淋巴细胞绝对计数之比。NLR 升高提示急性或慢性炎症、氧化应激及肿瘤相关性炎症，PLR 升高则反应血栓性炎症及慢性持续性炎症，二者在感染、自身免疫病、心血管疾病和肿瘤领域中均具有重要的辅助诊断和预后评估价值。

在消化系统中，NLR 可以反映溃疡性结肠炎的活动度[30]。在肿瘤方面，高 NLR 与 NK/T 细胞淋巴瘤、泌尿系上皮癌、小细胞肺癌等不良预后相关[31]-[35]。PLR 在多种实体瘤中亦提示预后不佳。在代谢及心血管疾病方面，NLR 原发性高血压患者早期肾功能损害的独立危险因素[36]。NLR 与糖尿病肾病 DKD 患者的炎症程度、蛋白尿以及肾功能下降呈正相关[37] [38]。NLR、PLR 均与老年 DKD 进展密切相关[39]。NLR 升高是抗中性粒细胞胞浆抗体(antineutrophil cytoplasmic antibody, ANCA)相关性肾炎病情严重性及不良预后的独立预测因子[40]。在血管通路方面，NLR 与 HD 患者自体动静脉瘘(AVF)狭窄的独立相关[41]。可作为术后早期 AVF 再狭窄的预警指标。在自身免疫方面，狼疮性肾炎(LN)与系统性红斑狼疮(SLE)患者中，NLR、PLR 显著升高，并与活动指数(SLEDAI 评分)呈正相关[42]-[44]。NLR 有助于鉴别 SLE 患者疾病活动与感染[45]。在急危重症方面，高 PLR 与腹膜透析患者 CVD 事件独立相关[44]。在 DKD 患者中，PLR 升高与左心室肥厚(left ventricular hypertrophy, LVH)显著相关[46]。PLR 也可用于预测 AKI 危重患者的预后及造影剂相关肾功能进行性恶化[47]。

3. NLR 与 MHD 患者心血管疾病的临床关联

(1) NLR 与心脏结构损伤

1) 左心室重构一项纳入大样本的 MHD 队列发现，NLR 与左心室质量指数(LVMI)呈线性正相关，NLR 每升高一个四分位，LVMI 平均增加 9.4 g/m^2 [47]，Botu 等的研究推测持续微炎症状态促使心肌细胞肥厚、间质纤维化，最终表现为 MHD 患者的心肌不均匀性增厚[48]。Bal 等研究发现 NLR 与 ESRD 患

者的心血管疾病严重程度相关，NLR 升高与左室射血分数(LVEF)下降存在负相关趋势，虽未达统计学显著，但方向一致[49]。

2) 瓣膜钙化 HD 患者主动脉瓣和二尖瓣钙化发生率可达 25%~59%。心脏瓣膜钙化组 NLR 显著高于非钙化组($P < 0.05$)，且 NLR 与钙化积分呈等级递增关系[50]。瓣膜钙化不仅增加瓣膜狭窄或关闭不全的风险，还与感染性心内膜炎、猝死密切相关；NLR 升高为临床早期干预提供了窗口期。

3) 心房功能异常 Demirtas L 等首次报道，高 NLR 是左心房机械功能障碍及心房电机械延迟的独立危险因素[51]。

(2) NLR 与动脉粥样硬化及血管钙化

多中心研究一致发现，NLR 与颈动脉内膜中层厚度(CIMT)、胸主动脉钙化评分(TACS)呈显著正相关[47]。CIMT 与 TACS 时全身动脉粥样硬化的“窗口”，NLR 升高提示炎症介导的血管重塑正在加速。王血荣报道，在 CKD 3~5D 期患者中，冠脉动脉钙化(CAC)发生率高达 57.93%。NLR 是 CAC 的独立危险因素[52]。一项对 225 例 CKD 3~5 期患者进行前瞻性随访发现，NLR 与血流介导的血管舒张功能(FMD)呈强负相关；当 $NLR > 2.8$ 时，可独立预测后续心血管事件，其敏感度和特异度均优于传统炎症指标 CRP [53]。

(3) NLR 与心功能衰竭

陈婷研究显示，无论是否合并充血性心力衰竭(CHF)，NLR 均与 NYHA 分级呈正相关；分级越高，NLR 越高[54]。日本一项纳入 86 例透析患者的前瞻性队列研究亦发现，NLR 随心功能分级进展呈阶梯式上升，提示其可实时反映心衰的严重程度[55]。马贤玉指出，NLR 对 MHD 患者新发 HF 具有重要预测价值[56]。黄建槐等进一步证实，心血管事件组 NLR 显著高于对照组，高 NLR 患者因急性心衰再住院风险明显增加[57]。

(4) NLR 与心血管事件发生率

Solak 等随访 225 例 CKD 3~5 期患者发现，NLR 与内皮功能障碍独立相关，可预测复合心血管终点事件[58]。包括心梗、脑卒中、外周动脉闭塞等。Lano 等多中心研究证实，NLR 与 MHD 患者心血管事件发生率呈正相关；ROC 曲线提示，当 $NLR > 3.24$ 时，对 2 年内心血管事件或死亡的预测价值最佳(AUC 0.84) [59]-[61]。有研究将 NLR 与 PLR 联合构建炎症评分，发现高炎症评分与 MHD 患者全因死亡率独立相关[62]。与传统单一指标相比，复合评分显著提高了预测效能。

(5) NLR 与死亡率

相关研究在 83 例回顾性研究及国外 339 例 42 个月随访队列中得出一致结论：NLR 升高是血液透析患者全因死亡及住院率的独立预测因素[63] [64]。周亮亮与张凤芹指出，$NLR > 3.24$ 是连续性血液透析患者 2 年心血管死亡的独立危险因素[61]。Neuen 等 2007~2012 年单中心大样本研究亦发现，NLR 对血液透析患者的心血管并发症死亡具有独立的监测价值[65]。Ao 等荟萃分析纳入全球 12 项研究，证实高 NLR 与 CKD 患者全因死亡率和心血管死亡率显著相关。NLR 每升高一个单位，心血管死亡风险增加 12% [66]。

4. PLR 与 MHD 患者心血管疾病的临床关联

(1) PLR 与心脏及血管钙化

于平通过多层螺旋 CT 定量 386 例 MHD 患者的主动脉弓钙化体积，发现高 PLR 是主动脉弓钙化的独立危险因素(OR = 2.31, 95% CI 1.53~3.48)，钙化体积随 PLR 四分位增加而递增($P\ trend < 0.01$) [67]。王云丹等发现，PLR 与主动脉瓣、二尖瓣钙化积分呈正相关($r = 0.42$, $P < 0.001$) [68]。当 $PLR \geq 83.18$ 时，CKD 1~5 期患者 5 年全因死亡风险增加 1.6 倍，提示 PLR 可能是跨 CKD 各期均适用的潜在生物标志物[69]。卜希与刘沧桑观察到，PLR 升高是 MHD 患者并发左心室肥厚(LVH)的独立危险因素(OR = 1.57, 95% CI 1.12~2.20)，且与左心室质量指数呈线性正相关[70]，表明 PLR 可在结构性心脏病早期阶段即发出预警。

(2) PLR 与心血管事件发生率

王聪秀等前瞻性纳入 512 例 MHD 患者，中位随访 42 个月，结果显示 PLR 是心血管事件(心肌梗死、脑卒中、急性心衰等)的独立危险因素(HR = 1.46, 95% CI 1.21~1.77) [71]。研究发现，PLR 水平与动脉硬化程度呈线性相关；以 PLR ≥ 150 为切点，预测复合心血管终点的 AUC 达 0.79 [72]。此外，在一项包括 44,114 例 MHD 患者的早期大型队列中，淋巴细胞计数下降与中性粒细胞计数升高协同增加 ESRD 患者死亡风险[73]，进一步支持 PLR 作为联合炎症–免疫轴的综合指标具有独特优势。

(3) PLR 与死亡终点

一项研究对 360 例 HD 患者进行了长达 71 个月的前瞻性随访，发现 PLR 处于最高四分位者的全因死亡风险显著升高；多因素 Cox 回归证实，PLR 每升高一个四分位，死亡风险增加 28%。Mustafa Yaprak 团队的研究亦得出一致结论：高 PLR 是 HD 患者全因死亡的独立危险因素[74]。于源等在老年 MHD 亚群中观察到相同现象——高 PLR 组 3 年全因及心血管死亡累积发生率分别为 34.7%与 21.4%，显著高于低 PLR 组的 14.2%与 8.6% [75]。汇总多项单中心研究后指出，高 PLR 与 MHD 患者院内死亡风险呈剂量–反应关系，提示 PLR 可作为院内危重症早期预警指标[76] [77]。

5. NLR、PLR 影响心血管系统的机制探讨

(1) NLR——炎症–免疫失衡的简易窗口

慢性肾脏病(CKD)及维持性血液透析(MHD)患者普遍处于微炎症状态。NLR 作为中性粒细胞(促炎)与淋巴细胞(抗炎/免疫调节)两大系统的整合指标，可在常规血常规中直接获得，近年来被证明与肾功能恶化、血管内皮功能障碍及动脉粥样硬化(AS)进程密切相关。

1) NLR 与炎症反应的关系：促炎细胞因子的释放。中性粒细胞计数升高提示炎症激活[78]。活化的中性粒细胞释放 TNF-α、IL-1β、IL-6 及活性氧自由基，启动并放大全身炎症级联[79]。淋巴细胞计数下降反映免疫抑制状态；IL-6、TNF-α 诱导淋巴细胞凋亡，造成 Th1/Th2 失衡，进一步削弱炎症负调[80] [81]。NLR 升高即促炎–抗炎失衡的量化表现，可预测 4 期 CKD 患者进入终末期肾衰竭的独立风险[82]。

2) NLR 对血管内皮功能的影响：氧化应激、一氧化氮(NO)生成减少。活化的中性粒细胞黏附并穿透血管内皮，释放 ROS 及水解酶，直接损伤内皮基底膜，抑制 eNOS 活性，减少 NO 生成，导致血管舒缩功能紊乱[83] [84]。氧化应激与微炎症形成正反馈：内皮损伤部位进一步招募炎症细胞，炎症介质积聚，加剧内皮功能障碍并促发左心室肥厚(LVH) [85]-[87]。

3) NLR 与动脉粥样硬化：炎症细胞浸润、泡沫细胞形成。中性粒细胞释放的蛋白酶和细胞因子促进单核–巨噬细胞迁移并抑制胆固醇外流，加速泡沫细胞形成；泡沫细胞再次分泌 IL-1β、IL-6，推动斑块进展[88]-[91]。VCAM-1、ICAM-1 等黏附分子在炎症刺激下表达上调，介导白细胞黏附与跨内皮迁移，成为 AS 早期关键步骤[92] [93]。临床研究表明，NLR 与颈动脉 IMT、冠状动脉钙化评分及 MHD 患者主要心血管不良事件(MACE)呈正相关，可作为血管内皮损伤和 AS 负荷的独立预测因子[94] [95]。

因此 NLR 通过“促炎细胞因子释放–氧化应激–内皮功能障碍–动脉粥样硬化”的连锁反应，把 CKD 患者的微炎症状态与心血管风险紧密联系在一起。动态监测 NLR，有望为早期识别高危人群、评估干预效果及个体化抗炎治疗提供一条简便而有效的临床路径。

(2) 血小板/淋巴细胞比值(platelet-to-lymphocyte ratio, PLR)近年来被视为一种整合“血栓–炎症”双重信息的新型标志物。

大量证据表明，PLR 升高不仅反映血小板活化和血栓形成倾向，还提示淋巴细胞凋亡所致免疫失衡及炎症加剧，从而与动脉粥样硬化及其并发症(如心衰、瓣膜钙化等)密切相关。

1) 血小板激活与炎症放大 血流高剪切力可迅速诱导血小板活化并增强其与血管内皮细胞的黏附

[96]。活化的血小板大量释放 IL-1、IL-6、TNF-α 及血小板活化因子(PAF)，刺激巨噬细胞增殖并进一步升高循环血小板水平[97] [98]。血小板源性生长因子(PDGF)等分泌产物促进血管平滑肌迁移与内膜增生，加速粥样硬化进程[99]。血小板来源的细胞外囊泡携带 CD40L、TLRs 等免疫分子，可直接调节 T 淋巴细胞生成与功能，提示血小板兼具“免疫细胞”属性[100]-[102]。

2) 血小板–白细胞交互作用 活化血小板通过 P-选择素/PSGL-1 等配体与中性粒细胞、单核细胞及 T 淋巴细胞形成聚集体，诱导白细胞趋化、黏附并迁移至血管壁，触发局部炎症反应[103] [104]。

动物及体外研究显示，血小板分泌的基质细胞衍生因子-1 (SDF-1)可招募 CD34+干细胞分化为巨噬细胞/泡沫细胞，加速脂质条纹形成[105]。

3) 淋巴细胞减少与免疫失衡 慢性炎症状态下，淋巴细胞凋亡显著，导致 TNF-α、IL-6 等炎症介质进一步升高[106]。淋巴细胞减少不仅削弱宿主免疫监视，还反向促进动脉壁炎症与钙化[107] [108]。

因此 PLR 通过整合血小板活化(促栓、促炎)与淋巴细胞减少(免疫失衡)两大信息，为动脉粥样硬化及其并发症提供了简便、经济且可重复的“血栓–炎症”综合指标。

6. 当前局限

当前关于 NLR、PLR 与维持性血液透析(MHD)患者合并心血管疾病(CVD)的研究已证实二者具有独立预测价值，但尚存在以下局限：(1) 多数为单中心、横断面或回顾性设计，样本量有限，缺乏外部验证；(2) 仅采集单次血常规数据，未动态监测 NLR/PLR 变化及其与 CVD 事件的时序关系；(3) 混杂因素控制不足，感染、铁剂、免疫抑制剂等均可干扰结果；(4) 机制研究停留在细胞层面，缺乏基因、蛋白组学及单细胞测序数据支撑。

7. 结论

现有研究一致证实：在 MHD 患者中，NLR、PLR 升高与动脉粥样硬化、心衰、瓣膜钙化及心血管死亡呈独立正相关；它们不仅是 MHD 患者心血管风险的敏感预测指标，更是连接尿毒症微炎症状态与心血管损害的“病理枢纽”。其升高反映了“促炎–抑修复”的免疫失衡核心机制，整合了传统风险因素未能捕捉的全身应激状态，为风险分层提供了全局性视角。

未来研究应超越相关性验证，聚焦机制探索(如特定免疫细胞亚群和 NETs 的作用)和临床转化。关键在于建立动态监测体系，定义有预警价值的波动阈值，并开展以 NLR/PLR 为指导的干预性研究(如靶向抗炎治疗、营养运动方案)，验证其作为可操作治疗靶点的价值，最终实现 MHD 心血管并发症的精准防控。需要更加大规模、多中心、前瞻性研究，来探索 NLR 和 PLR 在不同心血管疾病亚型中的作用。以及 NLR 和 PLR 与其他新型生物标志物的联合应用。

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