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血小板/淋巴细胞比值在肝脏疾病中的研究进展

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摘　要

血小板/淋巴细胞比值(Platelet-to-Lymphocyte Ratio, PLR)作为一种反映全身性炎症标志物，因其易于获取、计算方法简便以及成本较低等优势，在肝脏疾病的研究中受到广泛关注。本文综述了PLR在病毒性肝炎、非酒精性脂肪性肝病、肝硬化以及原发性肝癌等方面的研究进展，重点关注其在疾病监测和预后评估中的潜在应用价值。

关键词

血小板/淋巴细胞比值，肝脏疾病

Research Advances of Platelet-to-Lymphocyte Ratio in Liver Disease

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Abstract

Platelet-to-lymphocyte ratio (PLR), as a marker of systemic inflammation, has attracted much

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attention in the study of liver diseases due to its easy accessibility, simple calculation method and low cost. In this article, we review the research progress of PLR in viral hepatitis, non-alcoholic fatty liver disease, liver cirrhosis, and primary liver cancer, focusing on its potential application in disease monitoring and prognostic assessment.

Keywords

Platelet-to-Lymphocyte Ratio, Liver Disease

1. 引言

肝脏在人体的物质合成、代谢、解毒等多个方面发挥重要作用。肝脏疾病已成为威胁人类生命健康的主要疾病之一，可造成每年约 200 万人死亡，给社会和经济发展带来沉重的负担[1]。先前的研究已证实，炎症反应在肝脏疾病的发生与发展过程中起到了关键作用。血小板/淋巴细胞比值(platelet-to-lymphocyte ratio, PLR)是一种新型炎症标志物，能够有效反映机体的炎症程度及免疫平衡状态，在心血管疾病[2]、肾脏病[3]、风湿免疫疾病[4]等疾病的诊断、预后评估和治疗监测中展现出重要的临床价值。近年来大量研究揭示了 PLR 与肝脏疾病的相关性，本文就 PLR 与病毒性肝炎、非酒精性脂肪性肝病、肝硬化以及原发性肝癌的关系进行综述。

2. PLR 概述

血小板和炎症之间存在相互调节的关系，当肝组织受到损伤时，血小板会迅速被激活，聚集到受损区域，释放出多种炎症因子，进而招募更多的炎症细胞进入受损的肝组织，形成炎症反应的加剧，形成恶性循环[5]。淋巴细胞作为免疫系统的重要组成部分，在正常情况下维持着人体免疫系统的平衡与调节。当病原体入侵或损伤信号出现时，淋巴细胞会被激活并参与炎症反应，发挥其在免疫应答中的关键作用[6]。淋巴细胞计数的降低通常提示机体处于应激状态，且与整体健康状况的下降密切相关[7]。血小板/淋巴细胞比值综合了血小板和淋巴细胞两种血液学参数，形成一个新的复合炎性指标，能够更全面地反映机体的炎症和免疫状态，比单独的血小板或淋巴细胞绝对值更加稳定可靠[8]。PLR 受多种因素的影响，包括性别、年龄、地区等。在中国健康成年人中，平均 PLR 值为 99。此外，女性的 PLR 值通常高于男性，而年轻人的 PLR 值则显著高于老年人[9]-[11]。作为一种可通过血常规检测得到的简单生物标志物，PLR 已被广泛应用于预测恶性肿瘤、心血管疾病和感染性疾病等的预后和发病率。

3. PLR 与病毒性肝炎

病毒性肝炎是由不同病毒引起的以肝脏炎症为主要特征的疾病，持续的炎症反应可导致肝细胞功能下降和全身性炎症反应增加，并伴有不同程度的肝纤维化。我国是病毒性肝炎的高发区，部分肝炎患者可演变成慢性肝炎，进而演变为肝硬化和原发性肝细胞癌，对人民健康构成威胁。

慢性乙型肝炎(Chronic hepatitis B, CHB)是我国常见的慢性传染病之一。肝脏炎症和纤维化的早期诊断和准确评估对于控制慢性乙型肝炎的进展很重要，近年有研究发现 PLR 与肝纤维化程度和肝脏炎症有一定联系。Ding 等[12]观察到在 457 例接受肝活检和常规实验室检查的 CHB 患者中，PLR 与肝组织学评分存在统计学上显著的负相关，对晚期纤维化(S3-4)和肝硬化(S4)具有良好的分期性能，最佳截断值分别

为 9.67 (AUC = 0.72)和 74.27 (AUC = 0.76)。这些与 Lu 等[13]之前的研究结果一致。Kosekli 等[14]发现 PLR 在慢性乙型肝炎患者中下降，中位值在健康对照组、轻度纤维化、晚期纤维化对照组的中位 PLR 分别为 122 (64~197)%、99.5 (36~259)%和 119 (61~1547)%，而且 PLR 与 ISHAK 肝纤维化评分显著正相关，但在诊断晚期肝纤维化方面表现不佳。国内一项回顾性研究显示与无肝损伤组相比，肝损伤组的 PLR 水平升高，但是 PLR 与肝功能指标无相关性[15]。然而另一项前瞻性多中心研究结果显示，PLR 与慢性肝炎严重程度无关。此外，PLR 与 CHB 患者的早期病毒学反应密切相关[16]。Zhao 等[17]回顾性分析了 172 名 HBV 感染者，结果显示 PLR 与血清 HBV DNA 血清和血清 HBeAg 之间存在显著相关性。同样的，徐等[18]研究结果显示，CHB 患者的基线 PLR 水平与病毒载量下降幅度呈正相关性，说明高基线 PLR 水平预示着较好的病毒学应答。

丙型肝炎病毒感染是一个重大的公共卫生问题，是主要的传染病之一。机体免疫相关细胞如淋巴细胞、血小板，在丙型肝炎病毒感染过程中发挥重要作用。与 NLR 不同，PLR 是评估丙肝患者肝纤维程度的良好标志物。Fayed 等[19]通过一项前瞻性病例对照研究揭示了 PLR 与纤维化分期和五个无创纤维化指数(APRI、CDS、Fib-4、GUCI、Lok 指数)呈负相关，对于晚期纤维化的诊断效能较低。Catanzaro 等[20]进一步探索出 PLR 值 ≥89.00 是肝纤维化患者(非 F4 纤维化组)和已进入肝硬化阶段的患者(F4 纤维化组)之间的分界点。Meng 等[21]首次报道了 PLR 与 HCV 相关肝病患者的疾病严重程度密切相关，而且 PLR 是 HCV 清除的良好指标，PLR 增加表明病毒学应答良好。然而邵等[22]在其研究中提出 PLR、NLR、与慢性 HCV 感染者病毒载量之间无相关性。Wróblewska 等[23]对 42 例隐匿性丙型肝炎状态的患者进行了长期随访，发现 PLR 升高与死亡率相关。

4. PLR 与非酒精性脂肪性肝病

非酒精性脂肪性肝病(non-alcoholic fatty liver disease, NAFLD)是最常见的慢性肝病，其疾病谱包括非酒精性肝脂肪变、非酒精性脂肪性肝炎、肝硬化和肝细胞癌等，是一种与肥胖、血脂异常、胰岛素抵抗和遗传易感密切相关的代谢性肝损伤[24]。Chen 等[25]研究发现，PLR 和 WBC/MPV 与阻塞性睡眠呼吸暂停低通气综合征患者 NAFLD 的发生呈负相关，且 PLR 联合 WBC/MPV 在 BMI < 28 kg/m^2 和年龄 ≥ 60 岁亚组中预测 NAFLD 效能优于传统指标，而在 BMI ≥ 28 kg/m^2 中则没有。Choe 等[26]也发现 NAFLD 患者的 PLR 显著升高，且仅在无 MS 的非肥胖(BMI < 25 kg/m^2)亚组中，PLR 与 NAFLD 独立相关，而 Duan 等[27]研究发现肥胖儿童的 PLR 和 NAFLD 无相关性。这些表明非代谢紊乱人群可能更适合 PLR 的应用。最新一项关于 NAFLD 患者的 PLR 的荟萃分析表明[28]，与非 NAFLD 对照组相比，NAFLD 患者的 PLR 水平明显降低，中国和美国患者更为明显，这与之前的研究结果一致，表明 PLR 可能是 NAFLD 的保护因素。

PLR 在 NAFLD 病情发展的不同阶段发挥动态调节作用。国外一项大样本横断面研究中[29]，PLR 值低于 184.74 时，与 NAFLD 的发生风险呈负线性相关，在非糖尿病人群中表现出更高的诊断准确性。研究进一步发现 PLR 与肝硬化风险呈 U 型关系：PLR ≤ 130.5 时，PLR 降低与肝硬化风险增加相关；PLR > 130.5 时，PLR 升高则风险上升。然而另一项研究显示 PLR 和 NAFLD 风险呈倒“U”形非线性剂量反应，分段回归和阈值分析中结果显示 ln(PLR)的拐点值为 4.64，当 ln(PLR)低于 4.64 时，ln(PLR)每增加一个单位，NAFLD 风险增加 0.55 倍。相反，当 ln(PLR)超过 4.64 时，ln(PLR)每增加一个单位与 NAFLD 风险降低 0.40 倍相关[30]。此外，该项研究结果还显示 PLR 基线对术后预测脂肪肝分级变化无效。PLR 在 NAFLD 中的临床意义需结合人群特征及疾病阶段综合评估，两者的因果关系以及潜在机制仍不清楚，值得进一步研究。

5. PLR 与肝硬化

肝硬化是各种慢性肝病的最后阶段，其病理阶段以肝脏慢性炎症、弥漫性纤维化、假小叶、再生结

节和肝内外血管增殖为特征。多项研究表明 PLR 可用于评估肝硬化严重程度和并发症。Pomacu 等[31]表明 PLR 在 HBV/HCV 肝硬化组高于酒精性肝硬化组，但和健康对照组无差异。然而 Zhu 等[32]研究发现 PLR 在肝硬化患者中显著低于健康人，可用于诊断肝硬化。施等[33]也报道 PLR 水平与肝硬化严重程度相关，CHB 肝硬化失代偿期患者的 PLR 水平明显低于肝硬化代偿期。Zhao [17]等报道 PLR 在区分 HBV-CC 和 HBV-AC 患者方面准确可靠，其灵敏度(79.4%)和特异性(82.0%)均高于 NLR (灵敏度 20.6%；特异性 88%)，包含 PLR 和年龄在内的逻辑回归模型能最准确地区分 HBV-CC 和 HBV-AC 患者。Metawea [34]等分析了 149 例丙型肝炎病毒(HCV)相关性肝硬化患者，发现 PLR 和 MELD-Na 评分相关，在 HCC 组和非 HCC 组以及 SBP 组和非 SBP 组之间没有差异。国内一项研究结果显示 PLR < 78.63 是肝硬化的独立危险因素，还发现合并消化道出血、自发性腹膜炎和肝性脑病的 CHB 肝硬化患者 PLR 水平比无并发症者比较更低[35]。Nie 等[36]发现 PLR 在肝硬化患者合并门静脉血栓水平较高，是门静脉血栓形成的独立危险因素，与 Xing 等[37]、Han 等[38]之前的研究结果一致。Wang 等[39]提出高 PLR 与肝硬化患者 EVL 术后早期出血独立相关，再出血风险随着 PLR 的增加而上升。

6. PLR 与原发性肝癌

原发性肝癌(hepatocellular carcinoma, HCC)是我国最常见的恶性肿瘤之一，早期难以被发现，具有潜伏期长、发病率和死亡率高的特点。PLR 最早被用来预测恶性肿瘤的预后，近年来研究证明 PLR 与肝癌预后密切相关。Chen 等[40]揭示 PLR 是诊断 HCC 的生物标志物，ROC 曲线显示 AUC、敏感性、特异性分别为 0.912%、81.2%、80.6%，均显著高于 NLR、PLT、NEU 和 LYM。Liu 等[41]在研究中探讨了 PLR、NLR、MLR 动态变化与原发性肝癌患者的关系，发现首次免疫治疗前基线 NLR < 3.38、MLR < 0.28 或 PLR < 227.18 患者的 PFS 显著更长，并且较低的 PLR、NLR 和 MLR 与 BCLC 较早分期、转移部位较少、肝外转移频率较低或表现状态较好相关。Dertli 等[42]前瞻性研究了 105 名肝硬化伴肝癌患者，发现 PLR、NLR 与肿瘤的晚期分期相关，且低 PLR 组(PLR < 100.29)、低 NLR 组(<2.7)生存率高于高 PLR 组(PLR > 100.29)、高 NLR 组(NLR > 2.7)。

此外，PLR 对肝癌术后的预后及复发有预测价值。Zheng 等[43]进行了一项荟萃分析，比较了肝切除术后 HBV 相关肝癌患者的不同 NLR 和 PLR 水平之间的总生存期和无进展生存期，结果显示，高 NLR 患者的总生存期和无进展生存期较差，而 PLR 降低与肝切除术后复发的低风险和更好的生存率显著相关。Hosoda [44]等招募了 338 名肝癌根治术后患者，其中 47 例患者(13.9%)在术后 6 个月内复发。PLR 与术后早期复发显著相关，最佳截断值为 133 (AUC = 0.85)，logistic 回归分析确定 PLR 为早期复发的独立预后因素。同时，高 PLR 与较高的 DCP、较大的肿瘤直径和较差的组织学分化明显相关。Cui 等[45]研究 PLR 与肝癌移植术后预后的关系，发现存活组 PLR 显著高于死亡组，cox 多因素分析显示 PLR (>82.15)是无病生存期的独立危险因素，生存分析中低 PLR 组的 5 年 DFS 率明显更高。

7. 结语

PLR 作为一种简单、有效、相对便宜的系统性炎症标志物，与病毒性肝炎、NAFLD、肝硬化、原发性肝癌等肝脏疾病存在密切联系，对这些疾病的严重程度、预后等具有一定的预测价值，但是仍存在一些不足。首先，PLR 的研究多为回顾性研究，缺乏大样本、多中心的前瞻性研究，这限制了其在临床实践中的广泛应用。其次 PLR 的最佳截断值在不同疾病和研究中存在差异，缺乏统一的标准，这可能影响其在临床应用中的准确性和可比性。此外，PLR 在肝脏疾病中的潜在机制也需要深入研究，以揭示其在肝脏疾病发生发展中的具体作用。未来的研究应致力于解决这些不足之处，进一步验证 PLR 在肝脏疾病监测和预后评估中的应用价值，以期为临床实践提供更有力的依据。

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