·综述·

血小板及其衍生相关比值与急性缺血性脑卒中相关的研究进展

刘荣荣[1]，杨荣礼[2]，朱士光[3]，卢海龙[2]，刘建[4]，平蕾[5]，孙勇[6]，石瑜[7]，翟羽佳[8]
1.徐州医科大学研究生院，江苏 徐州，221000；2.徐州医科大学附属医院老年科，江苏 徐州，221000；3.徐州医科大学附属医院神经内科，江苏 徐州，221000；4.徐州市第一人民医院神经内科，江苏 徐州，221116；5.徐州市中心医院神经内科，江苏 徐州，221000；6.连云港市第一人民医院神经外科，江苏 连云港，222061；7.徐州市肿瘤医院神经内科，江苏 徐州，221005；8.徐州矿务集团总医院神经内科，江苏 徐州，221006
通信作者：朱士光，E-mail:Sgzhuxyfy@126.com

【摘要】 急性缺血性脑卒中（AIS）是一种常见的卒中类型，具有高复发率和高致残率的特点，是导致死亡和残疾的主要原因之一。血小板在AIS的发病机制中扮演着重要角色，与动脉粥样硬化、血栓形成和神经炎症等多种病理生理过程密切相关。近年发现血小板及相关比值在预测AIS的预后和转归方面具有重要的临床价值。本文综述了血小板与淋巴细胞比值（PLR）、血小板与中性粒细胞比值（PNR）、平均血小板体积与血小板（MPV/PLT）等一系列血小板相关衍生比值与AIS关系的研究进展。探讨这些相关指标与AIS的发生、严重程度、预后及并发症之间的联系，对早期制定AIS的治疗策略和改善预后具有一定的意义。

【关键词】 急性缺血性卒中；血小板；动脉粥样硬化；炎症；预后；溶栓；取栓
【文章编号】 2095-834X（2025）06-61-08
DOI：10.26939/j.cnki.CN11-9353/R.2025.06.006
本文著录格式：刘荣荣，杨荣礼，朱士光，等. 血小板及其衍生相关比值与急性缺血性脑卒中相关的研究进展[J]. 当代介入医学电子杂志，2025，2(6)：61-68.

Research progress on the association of platelet parameters and platelet derived ratios with acute ischemic stroke

Liu Rongrong[1], Yang Rongli[2], Zhu Shiguang[3], Lu Hailong[2], Liu Jian[4], Ping Lei[5], Sun Yong[6], Shi Yu[7], Zhai Yujia[8]
1.Graduate School of Xuzhou Medical University, Xuzhou 221000, Jiangsu, China; 2.Department of Geriatrics, the Affiliated Hospital of Xuzhou Medical University, Xuzhou 221000, Jiangsu, China; 3. Department of Neurology, the Affiliated Hospital of Xuzhou Medical University, Xuzhou 221002, Jiangsu, China; 4. Department of Neurology, Xuzhou First People's Hospital, Xuzhou 221116, Jiangsu, China; 5. Department of Neurology, Xuzhou Central Hospital, Xuzhou 221000, Jiangsu, China; 6. Department of Neurosurgery, the First People's Hospital of Lianyungan, Lianyungang 222061, Jiangsu, China: 7. Department of Neurology, Xuzhou Cancer Hospital, Xuzhou 221005, Jiangsu, China; 8. Department of Neurology, General Hospital of Xuzhou Mining Group, Xuzhou 221006, Jiangsu, China
Corresponding author: Zhu Shiguang, E-mail: Sgzhuxyfy@126.com.

【**Abstract**】 Acute ischemic stroke (AIS), a common type of stroke characterized by high recurrence rate and high disability rate, stands as one of the primary causes of death and disability in patients. Platelets play a pivotal role in the pathogenesis of AIS, being closely associated with multiple pathophysiological processes such as

收稿日期：2025-04-21
基金项目：徐州市科技局科研项目 (KC22241)；徐州医科大学附属医院临床研究专项（LCZX202414）；中西医协同慢病管理研究项目（CXZH2024036-24）

atherosclerosis, thrombosis and neuroinflammation. In recent years, studies have revealed that platelet parameters and related ratios hold significant clinical value in predicting the prognosis and outcome of AIS. This paper presents a comprehensive review of research advancements concerning a series of platelet derived ratios, including the platelet-lymphocyte ratio (PLR), platelet-neutrophil ratio (PNR), and mean platelet volume/platelet count ratio (MPV/PLT), in the context of AIS. By exploring the associations between these indicators and the occurrence, severity, poor prognosis and complications of AIS, this review aims to highlight their critical significance for formulating early treatment strategies and improving patient outcomes.

【Keywords】 Acute ischemic stroke; Platelets; Atherosclerosis; Inflammation; Outcome; Thrombolytic therapy; Thrombectomy

急性缺血性脑卒中（acute ischemic stroke, AIS）以高发病率、高致残率及高死亡率为显著特征，给全球公共卫生及患者家庭带来沉重的负担。尽管血管再通治疗可以显著改善AIS患者的结局，但由于缺乏高特异性的早期诊断指标，大量患者仍因诊断延迟错失黄金治疗时间窗。因此，探索新型生物标志物已成为当前研究的重点。

血小板活化与AIS的发生发展关系密切，小鼠模型证实程序性死亡配体1（programmed death ligand 1，PD-L1）与血小板激活状态相关，抑制PD-L1可以抑制血小板活化，并通过下调Caspase-3/GSDME通路改善动脉血栓形成和中风，这表明靶向PD-L1可能是干扰血栓形成的一种潜在方法[1]。临床研究证实血小板活化程度与脑梗死体积及神经功能缺损程度呈正相关[2-3]。近年来发现，血小板衍生比值在心力衰竭、急性心肌梗死、缺血性脑卒中等心脑血管疾病中具有预测价值[4-6]，其通过整合血小板数量、体积及外周血细胞参数，可能为AIS风险分层、疗效评估及预后预测提供新维度。本文系统梳理相关研究进展，旨在为临床早期制定干预策略及精准治疗提供理论依据。

1 血小板与AIS

AIS主要是由于脑血管血液循环障碍引起的脑组织缺血性坏死及相应的神经功能损害，以中老年群体为主，特征包括起病急骤、病情进展快速等，起病前无明显征兆，致残率、致死率较高。近年更是呈现出持续增加、低收入群体快速增长、发病人群年轻化趋势。因此，及时进行救治、尽早恢复血流灌注减少脑组织进一步缺血损伤对患者预后非常重要，目前临床的治疗方式主要包括抗血小板聚集、抗凝、静脉溶栓、脑动脉机械取栓等方式。尤其对于急性大动脉闭塞所致AIS来说，静脉溶栓为首选的方式，但其血管再通率低，且时间窗要求局限（3~4.5 h），而脑动脉机械取栓虽能恢复80%以上的血管再通，但其术后仍有超过50%的患者死亡或终身残疾[7]。为提高治疗疗效，及时挽救缺血半暗带，需早期识别预后不良患者并行早期干预。

血管内皮受损时，血小板激活并释放炎性介质[8]，触发血小板聚集、血管痉挛等级联反应，加剧动脉粥样硬化[9]。动脉斑块处的血流湍流又进一步促进血小板活化，形成“炎症-血栓”的病理闭环，最终导致脑血管急性闭塞。血小板在卒中后的炎症反应和血栓形成中扮演着重要角色[10]。

血小板在延迟再灌注和介导再闭塞方面起着重要作用。小鼠模型的实验表明，血小板可能通过介导白细胞募集作为一种潜在机制，在疾病进展和严重程度中扮演重要角色。阻断血小板功能，疾病的严重程度可得到显著改善[11]。Schuhmann等[12]将小鼠大脑中动脉的细丝闭塞4 h，阻断其血小板黏附受体糖蛋白(glycoprotein, GP) Ibα，证实可在闭塞期间以及再通/再灌注之前延缓脑梗死进展，为超急性脑卒中再通前的超早期治疗策略提供了思路。Chen等[13]对寒冷暴露组和常温组的小鼠进行了血小板功能评估，发现低环境温度暴露会促进血小板活化，从而增加脑梗死的风险。

2 血小板衍生比值与AIS的发生

外周血血常规指标极易获取，血小板衍生比值是基于血常规参数计算的复合指标，主要包括血小板与淋巴细胞比值（platelet to lymphocyte ratio, PLR）、平均血小板体积与血小板（mean platelet volume to platelet ratio, MPV/PLT）、血小板/高密度脂蛋白胆固醇比值（platelet to high-density lipoprotein cholesterol ratio, PHR）、血小板与中性粒细胞比值（platelet to neutrophil ratio, PNR）、血小板与白细胞比率（platelet to white blood cell ratio, PWR）、平均血小板体积与淋巴细胞比值（mean platelet volume to lymphocyte ratio, MPVLR）、红细胞分布宽度与血小板比值（ red blood cell distribution width-platelet ratio, RPR）、白细胞计数与平均血小板体积比（white blood cell count to mean platelet volume ratio, WMR）、葡萄糖与血小板比值

（glucose to platelet ratio, G/P）等，目前已有国内外研究者聚焦于揭露以上血小板衍生比值与AIS之间的关系，以期早期进行危险分层以及提供个体化治疗方案提供依据。

2.1 PLR与卒中的发生 动脉粥样硬化在卒中的发病机制中起着关键作用，而炎症通过介导动脉粥样硬化发展的每个阶段，在动脉粥样硬化的发生、进展中起着关键作用。高血小板计数会促进血小板活化并加剧炎症介质的释放，淋巴细胞亚群可以参与多种炎症反应，减少梗死面积和改善AIS后的功能障碍。然而，应激相关的皮质类固醇激素释放会促进淋巴细胞凋亡，在动脉粥样硬化的发展中发挥抗炎反应，导致免疫功能受损，进一步促进血栓的形成[14-16]。高血小板计数和低淋巴细胞计数有助于微血栓形成[17]，增加了卒中发生率及预后不良的风险。

PLR是一种新的生物标志物，研究证实其与动脉粥样硬化及血栓形成相关。İdil等[18]试验纳入了150例患者，在PLR值较高的患者中，检测到颈动脉狭窄的发生率较高（P=0.010），且PLR与颈动脉狭窄百分比呈正相关（r=0.250，P=0.002）。另外，PLR可作为预测急性深静脉血栓形成的生物标志物[19]。在将PLR作为预测AIS发生的生物标志物进行研究时，发现AIS组的平均PLR高于对照组。研究表明卵圆孔未闭患者的血小板计数升高与PLR升高可能与隐源性卒中发生有关[20]。PLR还有助于识别非瓣膜性心房颤动患者发生心源性脑栓塞的风险[21]。

2.2 MPV/PLT与卒中的发生 多项体外研究模拟体内微环境后发现，机械应力通过糖蛋白和整合素的相互作用激活血小板受体，MPV越高，表面GPIIb/IIIa受体密度更高表达水平更强，更易黏附于受损血管内皮细胞，导致血小板聚集和血栓形成，血小板的高活化潜能加剧了缺血损伤[22]。MPV作为血小板活性的指标，已被证实和血栓栓塞并发症以及心血管不良事件具有很强的相关性[23]。Bath等[24]研究表明，MPV与卒中风险呈正相关，MPV每增加1 fL，缺血性卒中风险增加15%。卒中是急性心肌梗死后的严重并发症，虽然其中涉及的病理生理机制尚不完全清楚，但炎症和血小板反应性增加可能在急性心肌梗死后卒中的发生过程中起重要作用。法国地区一项以急性心肌梗死患者为研究对象的调查发现，入院时较高的MPV/PLT有助于识别院内卒中风险增加的患者[25]，这一发现或能为探究炎症和血小板反应性增加在急性心肌梗死后卒中发生的病理生理机制提供新见解。Ma等[26]提出MPV/PLT可预测血液透析患者AIS的发生，MPV/PLT值越大，血液透析患者AIS发生率越高，且功能预后较差。

2.3 PHR与卒中的发生 高密度脂蛋白胆固醇与卒中之间的关系存在争议，而PHR已被证实是炎症和高凝状态的新型标志物，一项大型研究纳入2005年—2018年国家健康和营养检查调查(National Health and Nutrition Examination Survey，NHANES) 的横断面数据进行了分析，得出结论：PHR与脑卒中发生率之间的相关性表现出显著的阈值效应，PHR>223.684与卒中风险呈正相关，而HDL-C与卒中之间的相关性并不显著[27]。

3 血小板衍生比值与卒中严重程度、预后

高血小板反应性与不良预后相关。血小板参数及其组合可预测入院时和3个月后AIS和短暂性脑缺血发作的严重程度[28]。

3.1 PLR与卒中严重程度、预后 PLR不仅与卒中发生相关，现有研究支持其与患者入出院时NIHSS评分之间在统计学上存在显著正相关关系[29]。Zhu等[30]采用二元Logistic回归分析6种复合炎症比值对AIS患者30 d预后的独立影响，显示PLR是AIS患者短期预后不良的独立危险因素，且呈负相关关系。此外也有少量研究指出，PLR可能不是预测AIS患者功能结果的标志物[31]。

3.2 PNR与卒中预后 活化血小板和中性粒细胞之间的相互作用促进血栓性疾病中中性粒细胞外陷阱（neutrophil extracellular traps，NETs）的产生，且血小板来源的微囊泡（platelet microvesicles, PMPs）可能在AIS中的NETs形成中起着关键作用[32]。NETs的形成有助于动脉、静脉以及微血管中血栓的扩散[33]，早期干预NETs可能是改善患者预后的重要措施[32]。PNR水平与AIS患者3个月[34]及1年预后（包括死亡率）相关[35]。一项大型研究纳入了基线时没有卒中病史的27 811名参与者，平均随访14.3年，记录了838例卒中死亡病例，发现较高的中性粒细胞计数增加致命性卒中发生风险，而较高的PNR则降低该风险[36]。

3.3 PWR与卒中严重程度、预后 Amalia等[37]以503例卒中患者为研究对象，并将其依据病因学特征分为大动脉粥样硬化卒中组和心源性栓塞组，经Logistic回归明确动脉粥样硬化卒中组患者平均PWR显著高于心源性栓塞组，而平均NIHSS评分则相对较低，PWR与NIHSS评分呈现显著负相关关系。此外，针对接受血管内治疗的急性前循环闭塞性卒中群体，高PWR患者临床结局更佳，症状性颅内出血的风险更低。上述研究结果从不同维度共同揭示，PWR与AIS疾病严重程度之间存在紧密关联，可作为评估AIS病情进展及预后的潜在生物学指标。

3.4 MPV与卒中严重程度、预后 MPV是评估卒中严重程度的标志物，研究表明入院时mRS评分高的患

者其MPV水平亦较高[38]，且与卒中后 1 年的死亡率相关[39]。MPV/PLT对AIS的严重程度及预后有重要评估价值[40]，Zhu等[41]动脉粥样硬化卒中组和心源性栓塞组卒中患者研究显示MPV/PLT与非瓣膜性心房颤动性卒中的病变体积呈负相关。入院时的MPVLR同样与卒中严重程度呈正相关，MPVLR 升高是 AIS 后短期死亡率和不良预后的独立预测因素[42]。另外，WMR亦被证实与入院时卒中严重程度显著相关，可作为评估AIS患者预后的关键指标[43]。

4 血小板及衍生比值与药物治疗

在冠状动脉疾病患者中，已检测到个体血小板反应性随时间的变化而变化[44]。血小板对阿司匹林随时间的反应性增加也是AIS患者的早期功能恶化的独立危险因素[45]。无论病因如何，卒中治疗中血小板高反应性均与较差的晚期预后相关[46]。

AIS急性期治疗的关键在于尽早恢复脑血流，减少脑组织损伤。在缺血性卒中的早期阶段，包括静脉溶栓及溶栓桥接机械取栓等在内的再灌注治疗是指南推荐的恢复血流方法，在发病 4.5 h内给予阿替普酶或替奈普酶溶栓是临床常用的治疗措施，可显著改善预后。Sun等[47]研究纳入 741 例连续接受静脉溶栓的AIS患者，分别在入院时和卒中后 24 h采集血样用于PLR测量，并依据mRS评分评估结局，证实了溶栓后 24 h较高的PLR与不良结局风险增加独立相关。Xu等[48]的研究同样证实了接受静脉溶栓治疗的AIS患者，较高的PLR水平与不良预后（包括 3 个月内死亡率）相关。

溶栓后早期神经系统结局包括早期神经功能改善(early neurological improvement，ENI) 和早期神经功能恶化（early neurological deterioration，END）。多因素Logistic 回归分析表明，相较于ENI组，END组患者PLR水平更高[49]。研究已证实，MPV对穿支动脉疾病型脑梗死患者END的发生具有预测价值[50]。临床功能不良组患者入院时及溶栓后 18~24 h的MPVLR水平均高于功能结果良好组，提示高MPVLR值可预测溶栓后END[51]。Jiang等[52]发现溶栓前RPR水平升高或是AIS患者溶栓后END的独立危险因素，对溶栓后END的发生具有预测作用。

再灌注治疗后出血转化（hemorrhagic transformation，HT）在最初 24 h内发生率较高。HT是缺血性卒中的一种常见并发症，指AIS后缺血区血管重新恢复血流灌注导致的出血，包括出血性梗死（hemorrhagic infarction，HI）和脑实质血肿（parenchymal hemorrhage，PH）。研究发现HI后抗血小板治疗不会加重出血转化和神经功能恶化，尽管指南建议可以启动或继续抗血小板治疗，但临床中为规避出血风险，会限制抗栓再灌注治疗的强度，致使再灌注治疗不充分，则可能影响脑卒中的恢复。PH后积极抗血小板治疗或可加重患者出血转化，从而加重脑损伤，应延迟抗血小板治疗，在出血 2~4 周后个体化进行抗血小板治疗[53-54]。高PLR与AIS伴大动脉粥样硬化患者较高的HT风险相关，同时预示着其院内死亡率较高[55]。炎症反应在卒中和房颤的病理生理学中起关键作用，PLR作为新型炎症标志物可预测心源性栓塞型卒中患者院内HT、END的发生[56]，密切监测炎症标志物有助于指导临床治疗。

Desilles JP等采用短暂性大脑中动脉闭塞（transient monofilament middle cerebral artery occlusion，tMCAO）联合高血糖大鼠模型进行研究发现，高血糖可触发小鼠血栓炎症级联反应，破坏神经血管及内皮细胞的完整性，损伤星形胶质细胞，诱发神经血管损伤，致使血脑屏障破坏，血管通透性增加，最终诱发HT的发生[57]。入院时的G/P水平与未经溶栓或取栓治疗的AIS患者HT风险独立相关[58]。同时，另有研究表明PNR与静脉溶栓后AIS患者HT 和延迟性神经功能恶化（定义为出院 24 h至 7 d mRS评分 3~6 分）存在关联，PNR降低显著预示不良结局[59-60]。

AIS严重威胁患者的生存和生活质量，推动该领域的治疗技术不断革新。糖蛋白VI（glycoprotein VI，GPVI）作为血小板表面胶原蛋白/纤维蛋白（原）特异性受体，已成为血栓形成和血栓炎症性疾病（尤其是缺血性卒中）治疗的新兴靶点。基于此开发的新型人源化抗GPVI抗体Fab片段（EMA601）在小鼠模型中，已证实可有效防治动脉血栓形成和血栓炎症病变[61]，展现出良好的抗血小板治疗潜力。此外，纤溶酶原激活剂uPA与靶向活化血小板表面糖蛋白IIb/IIIa的单链抗体融合而成的重组融合构建体（SCE5-scuPA）作为新型溶栓剂，在小鼠AIS模型研究中表现优异，其依据血小板靶向与纤溶酶原激活两种功能协同作用，形成一种具备既能改善神经功能缺损、减少梗死体积、降低脑出血风险、保护血脑屏障，且对小鼠卒中后免疫功能、血小板数量无显著影响，提示其作为AIS前瞻性溶栓药物的临床应用价值[62]。

5 血小板衍生比值与机械取栓术

颅内动脉狭窄闭塞病变是卒中的主要病因，机械取栓治疗作为新兴疗法，可显著改善急性大动脉闭塞所致AIS患者预后。Li等[63]发现血小板分布宽度升高是经机械取栓术的前循环AIS患者 3 个月不良功能结局的独立预测因子。高PLR比值会增加血管再通不足

率和梗死面积，Ma等[64]证实了较高的PLR与取栓后成功再通的AIS患者不良功能结局独立相关，其潜在机制仍待进一步研究。此外，取栓后的患者中，较低的PLR、RPR和较高的PNR与术后3个月良好功能结局相关[65-66]，且低PNR、高WMR与大血管闭塞引起的AIS患者发生无效再通风险相关[67-68]，可能有助于识别无效再通。

6 血小板衍生比值与AIS并发症

预测卒中相关性肺炎（stroke-associated pneumonia，SAP）对于加强预防措施和降低发病率和死亡率至关重要。荟萃分析表明，高PLR与SAP发生相关，表明它可能是预测SAP的有价值的血液生物标志物[69-70]。血小板在抗菌宿主防御中起着至关重要的作用，MPV/PLT可被视为AIS合并肺炎患者的30 d死亡率的重要实验室预测指标[71]。

卒中后抑郁（post-stroke depression，PSD）是卒中患者最常见的心理并发症，发病率约39.8%[72]。发病机制可能与脑损伤导致的去甲肾上腺素和5-羟色胺失衡有关，病灶累及相关神经元及传导通路，导致神经递质水平下降。炎性细胞因子被视为PSD的危险因素，其中PLR在PSD的预测中具有重要价值[73-74]。有研究纳入363例AIS患者并进行1个月的随访，通过17项汉密尔顿抑郁量表评估发现入院时高PLR是PSD发生的重要且独立的预测标志物，且与95岁后的PSD风险呈线性关系（$P<0.001$）[75]。与未出现抑郁的卒中患者相比，PSD患者的PLR明显更高[76]。

7 小结与展望

随着我国老龄化进程加速，AIS已成为严重威胁中老年人健康的重大疾病。尽管再灌注治疗和神经影像学等诊疗技术取得显著进展，但早期诊断与精准预测仍是临床诊疗的关键瓶颈。“时间就是大脑”的治疗理念凸显了快速、精准干预的重要性。因此，寻找可靠的生物标志物对早期评估疾病进展、神经功能损伤及预后具有重要意义。本综述系统总结了国内外血小板及衍生比值在AIS研究中的最新进展，并探讨其作为新型生物标志物的潜力，以期推动AIS诊疗向精准化、个体化方向发展。未来，亟需构建大规模卒中数据库，结合人工智能技术挖掘整合临床、影像及治疗数据，开发个性化诊疗方案与预测模型，同时拓展血小板相关指标在其他系统疾病中的预测价值研究。

利益冲突 所有作者均声明不存在利益冲突

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