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单核细胞血小板聚集体的研究进展

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[摘 要] 循环中单核细胞血小板聚集体(monocyte-platelet aggregates，MPA)是血小板活化和炎症反应的敏感且有力的标志，MPA是介于炎症与血栓形成的桥梁。血小板激活后与单核细胞上的P-选择素糖蛋白配体-1(P-selectin glycoprotein ligand-1，PSGL-1)结合，形成MPA，在促进血液高凝及血栓形成、参与炎症反应调节炎症过程、监测肿瘤病情等方面发挥重要作用。本文回顾近年来MPA相关文献，围绕MPA的形成，对其功能以及在疾病中的作用进行综述，为血栓栓塞及炎症相关疾病的治疗提供新的靶点。

[关键词] 单核细胞血小板聚集体；血栓；炎症；高凝状态；动脉粥样硬化

Research progress in monocyte-platelet aggregates

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Abstract Monocyte-platelet aggregates (MPA) in circulation is a sensitive and powerful sign of platelet activation and inflammatory response, and MPA is a bridge between inflammation and thrombosis. After activation, platelets bind to p-selectin glycoprotein ligand-1 (PSGL-1) on monocytes to form MPA, which plays an important role in promoting blood hypercoagulation and thrombosis, participating in inflammatory response and regulating inflammatory process, and monitoring tumor disease. In this paper, we will review literatures about MPA in recent years, and focus on the formation of MPA, its function and progress in diseases, so as to provide new targets for the treatment of thromboembolism and inflammation-related diseases.

Keywords monocyte-platelet aggregates; thrombosis; inflammation; hypercoagulability; atherosclerosis

血小板激活后与单核细胞上的P-选择素糖蛋白配体-1(P-selectin glycoprotein ligand-1，PSGL-1)结合，形成单核细胞-血小板聚集体(monocyte-platelet aggregates，MPA)，作为血小板及单核细胞活化的标志，在促进血栓形成以及炎症反应中发挥重要作用，是炎症与血栓形成的桥梁[1-3]。近年来，有关MPA的相关研究日益增多，本文将系统综述MPA的形成、分类、功能以及在不同疾病中MPA潜在的诊断和治疗意义。

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1 MPA

MPA的形成主要依赖于血小板的活化，部分依赖于单核细胞的活化[3-4]。血小板在某些刺激下被激活并脱颗粒，P-选择素转移到血小板表面，与单核细胞上的PSGL-1结合，形成MPA[1]。目前，根据脂多糖(lipopolysaccharide，LPS)受体CD14和Fcγ受体III(Fc gamma receptor III，FcγRIII)CD16的差异表达，人类单核细胞分为3个亚群：经典$CD14^{++}CD16^{-}$细胞、中间$CD14^{++}CD16^{+}$细胞和非经典$CD14^{+}CD16^{++}$细胞亚群，$CD16^{+}$单核细胞传统上被称为“促炎”单核细胞[1,5-6]。血小板可以与上述三种类型的单核细胞结合形成MPAs。经典的单核细胞具有吞噬功能，没有炎症属性，而非经典单核细胞亚型在体内具有免疫监视和促炎功能。单核细胞高表达CD16被认为是完全成熟且更具促炎作用，低表达CD16的中间型单核细胞处于短暂分化阶段，同时具有促炎和抗炎表型[1,4]。研究[7]表明与中间型单核细胞相关的MPA可能是斑块易损性的标志，与冠状动脉粥样硬化进程有关。Allen等[1]评估血小板是否优先聚集到特定的单核细胞亚型，结果显示：与经典单核细胞相比，血小板优先与中间型和非经典单核细胞亚型相结合。

2 MPA的作用

对血小板活化指标的研究，从1980年代初开始的测定血小板成分的释放到1989年Larsen等最先开始的血小板表面CD62p的测定，再到近年来MPA的测定经历了漫长的过程。通过全血流式细胞术分析，白细胞-血小板聚集体，尤其是MPA，可能是循环中血小板活化更敏感的标志物[8-9]。活化血小板通过P-选择素、血小板糖蛋白IIb/IIIa(platelet glycoprotein IIb/IIIa，GPIIb/IIIa)复合物和CD154与单核细胞表面PSGL-1、CD11b/CD18和CD40 等结合，形成MPA[10]。P-选择素/PSGL-1介导的血小板结合导致单核细胞核因子-κB(nuclear factor-κB，NF-κB)活化/易位、超氧阴离子产生及促进单核细胞趋化蛋白-1(monocyte chemotactic protein-1，MCP-1)、肿瘤坏死因子-α(tumor necrosis factor-α，TNF-α)、白介素-8(interleukin-8，IL-8)、白介素-1β(interleukin-1β，IL-1β)和组织因子(tissue factor，TF)的释放[11-12]。TF是外源性凝血级联反应的关键因子，血小板和单核细胞与凝血有关，这与TF促凝活性的调节有关。当TF与因子VII及其活性形式VIIa结合时，会触发凝血，导致纤维蛋白的形成。CD40L-CD40相互作用可用于连接MPA的形成，也可通过诱导单核细胞表达TF触发促凝反应。人们普遍认为单核/巨噬细胞能够合成TF的细胞，后来发现中性粒细胞及血小板中也存在TF，但中性粒细胞和血小板TF在体内的作用仍有待确定[12]。P-选择素还可能刺激单核细胞增加血小板活化因子的合成，从而形成一个恶性循环，导致血小板进行性活化失去控制。单核细胞占血小板血栓结合白细胞的16%，这几乎是循环血液中单核细胞比例的4倍，进一步支持了MPA在血栓形成中的重要性[13]。

血栓形成和炎症之间互相促进，血栓的存在可能是炎症的病灶，感染和相关炎症可以触发血小板和白细胞活化和血栓形成[12]。MPAs除了作为血小板活化的敏感指标在促进血液高凝及血栓形成方面发挥作用外，在炎症反应中同样具有重要作用。MPA可以促进炎症反应，C-反应蛋白(C-reactive protein，CRP)孵育全血可使MPA形成倍增，注射LPS可使小鼠MPA计数增加4倍，说明炎症也可以促进MPA的形成[13]。通过流感免疫及体外实验证实，MPA的形成导致循环中单核细胞向CD16+细胞的表型转变，使其促炎作用增强，内皮黏附性增加[14]，另有数据[15-16]证明MPA增强抗炎细胞因子白介素-10(interleukin-10，IL-10)的表达。单核细胞与血小板的聚集伴随着单核细胞的激活，导致炎性细胞因子和趋化因子的表达增加，而且与无血小板结合单核细胞相比，MPA的迁移能力明显增强，这些都在炎症时的器官功能障碍中起重要作用[12,17]。MPA与缺血-再灌注损伤(ischemia-reperfusion injury，IRI)有关[18]，大鼠心肌IRI模型不同时间点血小板白细胞聚集体的表达水平逐渐上升并显著高于假手术组，提示炎症血栓标志物血小板白细胞聚集体参与心肌IRI发生。MPA的形成还可以使血小板同源聚合增加阻止出血[10]。此外，MPA与血小板介导的止血功能受损有关，血小板与循环白细胞的相互作用可能是严重创伤后血小板反应性受损的另一个机制[19]。

3 MPA与疾病的关系研究进展

3.1 MPA 与心、脑血管疾病

动脉粥样硬化性心脑血管疾病在世界范围的发病率逐渐上升。循环MPA的形成导致单核细胞在小鼠动脉粥样硬化斑块中的浸润增加。此后Rutten等[20]也证明了MPA和单核细胞浸润在动脉

粥样硬化斑块进展中起促进作用。MPAs在动脉粥样硬化性血栓形成疾病中发挥重要作用。与健康人相比，稳定性冠心病患者形成MPAs的倾向增加[21]。不稳定心绞痛患者中间型单核细胞、中间型单核细胞相关MPAs和总MPAs水平升高[22]。心肌梗死患者循环中MPAs水平的升高先于肌酸激酶同工酶MB(creatine kinase-MB，CK-MB)水平的升高，因此认为MPAs水平可能是心肌梗死的更早期标志物[21]。血栓形成和动脉粥样硬化是缺血性卒中发生的主要原因，单核细胞-血小板聚集物和单核细胞与血小板聚集率在缺血性脑卒中后第2天显著升高，可能反映了对脑缺血的急性反应[23]，MPAs对于脑梗死患者早期诊断及预测病情具有重要的临床应用价值[24]。严重肢体缺血患者的MPA较高，MPA可能与外周动脉疾病(peripheral artery disease，PAD)显著相关，较长的下肢血管长度使炎性单核细胞更容易进入内皮下层，这一过程因MPAs的存在而增强[1]。活化的血小板和单核细胞之间的相互作用可以启动导致动脉粥样硬化的促炎和黏附分子的释放，并促进单核细胞向内膜下间隙的浸润[25]。

研究[26]表明：MPA与心肌梗死后心肌组织炎症有关，高MPA百分比与6个月后左心室收缩功能恶化有关。多支病变心肌梗死患者的$CD14^{++}CD16^{+}$(中间单核细胞，MON2)型MPA的百分比更高，表明MON2型MPA与心血管预后密切相关[21]。ST段抬高心肌梗死早期MPAs增加且持续时间较长，甚至持续到单核细胞计数正常化后[27-28]。Di Serafino等[29]也证明了MPA可以被认为是动脉粥样硬化的细胞生物标志物，不仅可以促进斑块的生长，而且可以代表冠状动脉疾病的发展和不稳定。在其他心血管疾病中，MPA与疾病也具有一定关系，例如：心力衰竭患者的MPA生成增加[30]；MPA与心房颤动的血栓形成程度有关，并可作为左房血栓的潜在生物标志物[31]。

3.2 MPA 与炎症和感染性疾病

高血压被广泛认为与促炎环境有关，与健康的志愿者相比，高血压患者显示出更高的MPA水平[2]。2型糖尿病患者中观察到单核细胞和血小板聚集水平升高，推测MPA是糖尿病的早期炎症标志[32]。有血管病变的糖尿病患者MPA的水平显著高于无血管病变患者，强调MPA与糖尿病并发症之间的关系[33]。肝硬化患者的MPAs水平显著增高，这种增加在$CD16^{+}$单核细胞中更加显著[34-35]。此外，在溃疡性结肠炎[36]、抗磷脂综合征、系统性红斑狼疮、类风湿性关节炎等自身免疫性炎症疾病中也显示循环中MPAs水平的增加[34]。

在某些细菌及病毒感染性疾病中也可观察到升高的MPA。研究[3]表明1型人体免疫缺陷病毒(human immunodeficiency virus 1，HIV-1)感染者3个单核细胞亚群中MPAs的百分率均高于健康对照组，并且非经典和中间单核细胞中的MPA水平与HIV-1病毒复制密切相关，推测HIV-1复制活跃的患者中潜在冠状动脉疾病的风险更高。在人免疫缺陷病毒(human immunodeficiency virus，HIV)感染者中，通过血小板-单核细胞相互作用可能导致$CD16^{+}$单核细胞跨越血脑屏障的迁移增强，增加艾滋病病毒相关性脑炎的风险[37]。在肺结核患者肺肉芽肿的活检中发现了血小板标志物，表明MPAs可作为血小板进入肺的载体发挥作用[4]。脓毒症可诱导MPA百分比的迅速扩张，脓毒症患者的28天病死率与MPAs显著相关，推测MPA升高可作为预测脓毒症病死率的生物标志物[17]。

3.3 MPA 参与了肿瘤的发生与发展

MPA在不同阶段影响肿瘤的炎症过程：血小板改变内皮的激活状态，将单核细胞募集到肿瘤部位，调节原发和转移肿瘤部位的炎症环境；肿瘤相关的血小板活化促进凝血级联反应的启动，并构成血栓形成的重大风险；肿瘤激活的血小板通过促进血管生成和转移等关键过程进一步促进疾病进展等[38]。肺癌患者MPA、纤维蛋白原(fibrinogen，FIB)、*D*-二聚体及CD62p水平均高于良性肺病和正常对照，MPA的敏感度可达到81.7%，且与血栓前状态指标 FIB、*D*-二聚体尤其是CD62p都有良好的相关性，证实MPA与肿瘤患者血栓前状态的密切联系[39]。有转移的结肠癌患者MPA显著升高，远处转移是肿瘤患者病情恶化的表现，说明检测MPA水平对监测结肠癌患者的病情具有重要意义[40]。血小板及其与单核细胞的相互作用在肿瘤的各个阶段都至关重要。然而，MPA在肿瘤发生和发展中的确切机制和影响仍不清楚[38]。

3.4 MPA 与其他疾病

可溶性血管内皮生长因子受体-1(soluble fms-like tyrosine kinase 1，sFlt-1)是子痫前期的重要介质，有关文献[41]报道先兆子痫患者活化的血小板与单核细胞结合产生sFlt-1，首次将sFlt-1的产生与先兆子痫常见的MPA联系起来。正常妊娠晚期孕妇组MPA表达百分率明显增高，提示晚期妊娠孕妇血小板活化

程度增强[42]。高脂喂养的小鼠表现出代谢的异常并伴随着MPAs水平升高，说明高脂饮食会促进不良的高凝状态，使心血管疾病的风险增加[43]。

4 结语

单核细胞与血小板相互作用形成MPA，参与炎症反应，使血液处于高凝状态，并且越来越多的证据已经表明MPA参与不同疾病的发生与发展，在不同疾病中具有潜在的诊断和判断预后的价值。针对MPA的治疗可能对疾病具有一定的作用，早期测定MPA水平及对其形成过程进行有效干预，可能为血栓栓塞及炎症相关疾病的治疗带来新希望。

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