Journal of Autacoids

Open access Perspective

Kinin Inhibitor Targets Thromboinflammation along Proinflammatory Processes

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INTRODUCTION

Accumulating evidence suggests that ischemic stroke is a thromboinflammatory disease in which the contact kinin pathway plays a central role by activating procoagulant and proinflammatory processes. Blocking specific members of the contact kinin signaling pathway is a promising strategy for controlling ischemic stroke. Here, plasma kallikrein and active inhibitors were identified for the first time from tree flukes. Tests have shown that sylvestin increases activated partial thromboplastin time without affecting prothrombin time. Thromboelastography and clot retraction assays were also shown to prolong clotting time in whole blood and inhibit clot retraction in platelet-rich plasma. The potential role of sylvestine in ischemic stroke was evaluated by temporary and permanent middle cerebral artery occlusion models. Importantly, sylvestine showed no signs of bleeding tendency. This study identifies sylvestine as a promising contact kinin signaling pathway inhibitor that may provide comprehensive protection against ischemic stroke without increasing the risk of bleeding. Despite decades of promising preclinical validation and clinical implementation, ischemic stroke remains a leading cause of death and disability worldwide. Their complex pathophysiological features include thrombosis and inflammation.

DESCRIPTION

Thrombosis, cerebrovascular occlusion, inflammatory response, and highly interconnected processes leading to severe neuronal damage after ischemic events. Therefore, here thrombosis-dependent mediators relevant after stroke, focus on recent findings on platelet regulation, potential regulation of the innate and adaptive immune system in the thromboinflammatory process, and provide a comprehensive provide the most up-to-date information.

Recent findings indicate that ischemic stroke is a thromboinflammatory disease. Plasma kallikrein cleaves high molecular weight kininogen to release bradykinin and is a key component of the proinflammatory contact kinin system. In addition, Plasma kallikrein can activate clotting factor, the origin of the endogenous coagulation cascade. Thus, Plasma kallikrein triggers important pathological pathways of stroke formation, thrombosis, and inflammation. Various mediators balance vascular tone, inflammation, coagulation and thrombosis in severe inflammatory and infectious diseases. In addition, we also discuss the therapeutic implications of these complex interactions, as modulation of one system can affect other systems, with beneficial or detrimental consequences. Plasma prekallikrein is the liver-derived precursor of plasma kallikrein, a trypsin-like serine protease, which circulates in plasma bound to high-molecular-weight kininogen. The zymogen is converted to prekallikrein by an activator. Prekallikrein regulates several proteolytic cascades in the cardiovascular system, including the intrinsic coagulation pathway, kallikrein-kinin system, fibrinolytic system, renin-angiotensin system, and alternative complement pathway. Here, we review the biochemistry and cell biology of prekallikrein and highlight recent *in vivo* studies that have identified critical roles for proteases in procoagulant and inflammatory disease states. Targeted use of prekallikrein offers a new strategy to influence thrombosis and vascular inflammation in a variety of previously unappreciated diseases.

CONCLUSION

The risk of thrombosis is an increasing challenge and one of the leading causes of death worldwide, and is influenced by multiple factors in intravascular coagulation, vessel walls, and cellular systems. Mass stimulation and/or increased expression thereof help maintain healthy vascular homeostasis by producing a graded increase in plasma prostacyclin that reduces thrombosis. Recognition of these subtle mechanisms will help develop new antithrombotic strategies by targeting vascular receptors for renin-angiotensin.

Received:	31-January-2023	**Manuscript No:**	JAC-23-15816
Editor assigned:	02-February-2023	**PreQC No:**	JAC-23-15816 (PQ)
Reviewed:	16-February-2023	**QC No:**	JAC-23-15816
Revised:	21-February-2023	**Manuscript No:**	JAC-23-15816 (R)
Published:	28-February-2023	**DOI:**	10.35841/jac.4.1.01

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Citation Masahiro M (2023) Kinin Inhibitor Targets Thromboinflammation along Proinflammatory Processes. Autacoids J. 4:01.