Platelet Aggregation: Action of Human Plasma Kallikrein and the Effect of the Recombinant Kallikrein Inhibitor, rBbKI


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Platelets are circulating anuclear cell fragments from megakaryocytes with a central role in haemostasis. These fragments have an important participation in pathological processes, mainly thrombosis and tumour metastasis. Human plasma kallikrein (HuPK) is a glycoprotein that participates in the activation of blood coagulation and fibrinolysis. The action of HuPK on platelet function is still unclear. The objective of this study was to evaluate the action HuPK on platelets from healthy human volunteers, in the presence and absence of platelet agonists, such as ADP (10 µM), arachidonic acid (0.5 µM), collagen (4.0 µg/mL), epinephrine (60 µM) and thrombin (1.0 UI/ml), and the effect of a recombinant plasma kallikrein inhibitor (Bauhinia bauhinioideas kallikrein inhibitor, rBbKI). HuPK in concentration up to 1.05 µM had no direct action on platelet aggregation. However, it potentiated the action of ADP and arachidonic acid, agonists that per se are not able to induce platelet aggregation at low concentrations (0.83 µM and 0.20 µM, respectively). HuPK increaseg of 44% and 59% the aggregation induced by ADP and arachidonic acid, respectively, leading to a maximum of aggregation. The kallikrein inhibitor (rBbKI, 3.8 µM) did not interfere on platelet aggregation induced by the five agonists tested, but it completely inhibited the potentiating action of HuPK. In conclusion, the data suggest a new physiological action of plasma kallikrein and further studies are necessary to explain the mechanism involved.

Word Keys: Platelets, inhibitors, kallikrein

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