The antiplatelet activity of magnesium sulfate involved suppression of protein kinase C and Na+/H+ exchanger.

許準榕
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Abstract

Magnesium sulfate is widely used to prevent seizures in pregnant women with hypertension. The aim of this study was to examine the inhibitory mechanisms of magnesium sulfate in platelet aggregation in vitro. In this study, magnesium sulfate concentration-dependently (0.6-3.0 mM) inhibited platelet aggregation in human platelets stimulated by agonists. Magnesium sulfate (1.5 and 3.0 mM) also concentration-dependently inhibited phosphoinositide breakdown and intracellular Ca+2 mobilization in human platelets stimulated by thrombin. Rapid phosphorylation of a platelet protein of M(r) 47,000 (P47), a marker of protein kinase C activation, was triggered by phorbol-12-13-dibutyrate (PDBu, 50 nM). This phosphorylation was markedly inhibited by magnesium sulfate (3.0 mM). Magnesium sulfate (1.5 and 3.0 mM) further inhibited PDBu-stimulated platelet aggregation in human platelets. The thrombin-evoked increase in pHi was markedly inhibited in the presence of magnesium sulfate (3.0 mM). In conclusion, these results indicate that the antiplatelet activity of magnesium sulfate may be involved in the following two pathways: (1) Magnesium sulfate may inhibit the activation of protein kinase C, followed by inhibition of phosphoinositide breakdown and intracellular Ca+2 mobilization, thereby leading to inhibition of the phosphorylation of P47. (2) On the other hand, magnesium sulfate inhibits the Na+/H+ exchanger, leading to reduced intracellular Ca+2 mobilization, and ultimately to inhibition of platelet aggregation and the ATP-release reaction. Copyright 2004 National Science Council, ROC and S. Karger AG, Basel