Mechanisms involved in the antiplatelet activity of magnesium in human platelets.

許準榕
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Abstract

In this study, magnesium sulphate dose-dependently (0.6-3.0 mmol/l) inhibited platelet aggregation in human platelets stimulated by agonists. Furthermore, magnesium sulphate (3.0 mmol/l) markedly interfered with the binding of fluorescein isothiocanate-triflavin to the glycoprotein (GP)IIb/IIIa complex in platelets stimulated by collagen. Magnesium sulphate (1.5 and 3.0 mmol/l) also inhibited phosphoinositide breakdown and intracellular Ca+2 mobilization in human platelets stimulated by collagen. Magnesium sulphate (3.0 mmol/l) significantly inhibited thromboxane A2 formation stimulated by collagen in platelets. Moreover, magnesium sulphate (1.5 and 3.0 mmol/l) obviously increased the fluorescence of platelet membranes tagged with diphenylhexatriene. In addition, magnesium sulphate (1.5 and 3.0 mmol/l) increased the formation of cyclic adenosine monophosphate (AMP) in platelets. Phosphorylation of a protein of Mr 47 000 (P47) was markedly inhibited by magnesium sulphate (1.5 mmol/l). In conclusion, the antiplatelet activity of magnesium sulphate may involve the following two pathways. (1) Magnesium sulphate may initially induce membrane fluidity changes with resulting interference of fibrinogen binding to the GPIIb/IIIa complex, followed by inhibition of phosphoinositide breakdown and thromboxane A2 formation, thereby leading to inhibition of both intracellular Ca2+ mobilization and phosphorylation of P47. (2) Magnesium sulphate might also trigger the formation of cyclic AM, ultimately resulting in inhibition of the phosphorylation of P47 and intracellular Ca+2 mobilization.