Inhibitory mechanisms of gabapentin, an antiseizure drug, on platelet aggregation

周敦穗

Pan CF;Shen MY;Wu CJ;Hsiao G;Chou DS;Sheu JR

摘要

Abstract

Gabapentin (Neurontin) is an analogue of gamma-aminobutyric acid (GABA) that is effective against partial seizures. Gabapentin has been reported to modulate serotonin release from platelets, but the effects of gabapentin on platelet activation have not been explored. In this study, gabapentin concentration-dependently (60-240 microM) inhibited platelet aggregation in washed platelets stimulated by collagen (1 microg mL(-1)), ADP (20 microM) and arachidonic acid (60 microM). Gabapentin (120 and 240 microM) also concentration-dependently inhibited collagen (1 microg mL(-1))-induced phosphoinositide breakdown, intracellular Ca(2+) mobilization, thromboxane A(2) formation, and p38 MAPK phosphorylation in human platelets. In conclusion, the most important findings of this study suggest that gabapentin inhibits platelet aggregation, at least in part, through the phospholipase C-inositol 1,4,5-trisphosphate-thromboxane A(2)-Ca(2+) pathway. Thus, it is possible that gabapentin treatment, alone or in combination with other antiplatelet drugs, may induce or potentiate inhibition of platelet aggregation, which may affect haemostasis in-vivo.