Mechanism involved in the antiplatelet activity of naloxone in human platelets.

許準榕

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Abstract

In this study, naloxone was tested for its antiplatelet activity in human platelet suspensions. In platelet suspensions (4.5 x 10(8)/ml), naloxone (0.1-0.5 mM) significantly inhibited platelet aggregation and ATP-release stimulated by various agonists (i.e., thrombin, collagen, U46619, and ADP). Furthermore, naloxone (0.5 and 0.8 mM) dose-dependently inhibited the intracellular free Ca2+ rise of Fura 2-AM loaded platelets stimulated by collagen. Additionally, naloxone (0.5 and 1.0 mM) did not influence the binding of FITC-triflavin to platelet glycoprotein (GP) IIb/IIIa complex. On the other hand, naloxone (0.5 mM) markedly decreased the fluorescence of platelet membranes tagged with diphenylhexatriene (DPH). In addition, naloxone (0.1-0.5 mM) did not significantly affect cyclic-AMP levels in human washed platelets. It is concluded that the antiplatelet activity of naloxone may possibly be due to the induction of conformational changes in the platelet membrane and the inhibition of the intracellular Ca2+ ([Ca2+]i) mobilization as well as the release reaction of platelets stimulated by agonists.