

Inhibitory mechanisms of naloxone on human platelets

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摘要

Abstract

1. In the present study, naloxone was tested for its antiplatelet activities in human platelet-rich plasma (PRP). In human PRP, naloxone (0.1-0.5 mmol/L) inhibited aggregation stimulated by a variety of agonists (i.e. collagen, adenosine diphosphate (ADP), U46619 and adrenaline). 2. Naloxone (0.1-0.5 mmol/L) did not significantly affect cyclic adenosine monophosphate and cGMP levels in human washed platelets, whereas naloxone (0.5 mmol/L) significantly inhibited thromboxane B₂ formation stimulated by collagen (5 micrograms/mL) in human washed platelets. 3. Naloxone (0.5 mmol/L) significantly inhibited [³H]-inositol monophosphate formation of [³H]-myoinositol-loaded platelets stimulated by collagen and U46619. Moreover, naloxone did not influence the binding of ¹²⁵I-triflavin to platelet membranes. Triflavin is an Arg-Gly-Asp-containing specific fibrinogen receptor antagonist. 4. Addition of naloxone (0.5 mmol/L) to platelet preparations tagged with diphenylhexatriene (DPH) resulted in a considerable decrease in relative fluorescence intensity. 5. It is suggested that the anti-platelet effects of naloxone may be caused, at least partly, by the induction of conformational changes in the platelet membrane initially, followed by the inhibition of thromboxane A₂ formation and phosphoinositide breakdown of platelets stimulated by agonists.