Mechanisms involved in agonist-induced hyperaggregability of platelets from normal pregnancy

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摘要

Abstract

There is substantial evidence of increased platelet reactivity in vivo and in vitro during pregnancy. Platelet activation occurs in pregnancy with a risk of the development of preeclampsia. In this study, platelet behavior was studied during 28-40 weeks of gestation in a group of women who remained normotensive and a group of nonpregnant female controls. Platelet aggregation and ATP release stimulated by agonists (i.e. collagen and adenosine 5'-diphosphate) were markedly enhanced in washed platelets from pregnant subjects. Furthermore, the collagen-evoked increase in intracellular Ca2+ ([Ca2+]i) mobilization in fura-2-AM-loaded platelets was also enhanced in pregnant subjects. Moreover, the binding activity of fluorescein isothiocyanate-triflavin toward the platelet glycoprotein IIb/IIIa complex did not significantly differ between the nonpregnant and pregnant groups. In addition, the amount of thromboxane A2 (TxA2) formation from pregnant subjects was significantly greater than that from nonpregnant subjects in both resting and collagen-activated platelets. On the other hand, prostaglandin E2 formation in the presence of imidazole in either resting or arachidonic acid (100 μM)-treated platelets did not significantly differ between these two groups. The levels of cyclic AMP formation in both resting and prostaglandin E1 (10 μM)-treated platelets from pregnant subjects were significantly lower than those in nonpregnant subjects. Nitric oxide production was measured by a chemiluminescence detection method in this study. The extent of nitrate production in either resting or collagen-stimulated platelets from pregnant subjects did not significantly differ from that of platelets from the nonpregnant group. We conclude that the agonist-induced hyperaggregability of platelets from normal pregnancy may be due, at least partly, to an increase in TxA2 formation and a lowering of the level of cyclic AMP formation, which leads to increased [Ca2+]i mobilization and finally to enhanced platelet aggregation and ATP release.