

IONOPHORETIC ACTION OF PHOSPHOLIPIDS ON HUMAN PLATELETS. Y. Ikeda, M. Kikuchi, K. Watanabe, Y. Ando and M. Hasegawa. School of Medicine, Keio University, Tokyo, Japan.

The effects of phospholipids on calcium uptake by platelets were examined by using $^{45}\text{CaCl}_2$. Platelet suspensions ($3-5 \times 10^8/\text{ml}$) were prepared in PIPES buffer at pH 6.8 and were incubated with $0.2 \text{ mM } ^{45}\text{CaCl}_2$ ($10 \mu\text{Ci}/\text{ml}$) in the presence or absence of various kind of phospholipids. The mixtures were centrifuged through silicone oil for 1 min. at $7,000g$ to separate the pellet. The radioactivity in the pellet was measured with liquid scintillation spectrometer.

Among the phospholipids tested, cardiolipin and phosphatidic acid exerted a significant increase of calcium uptake. The amount of calcium translocated into platelets after 60 min. incubation with cardiolipin, phosphatidic acid, sphingomyelin and phosphatidylcholine (0.4 mM) was 2.58 , 1.33 , 0.30 and $0.32 \text{ n mol}/10^9 \text{ plts}$, respectively, while that without phospholipids was $0.31 \text{ n mol}/10^9 \text{ plts}$. For the sake of comparison, the effect of A23187 or thrombin on calcium uptake was examined. The amount of calcium taken up in the presence of $1 \mu\text{M}$ A23187 and $1 \text{ u}/\text{ml}$ of thrombin was 1.84 and $1.33 \text{ n mol}/10^9 \text{ plts}$, respectively. Calcium uptake was dependent on cardiolipin concentrations and incubation time. LaCl_3 (1 mM) completely inhibited cardiolipin or A23187 induced calcium influx. However, when platelets were initially incubated with $^{45}\text{CaCl}_2$ for 60 min. in the presence of 0.4 mM cardiolipin followed by addition of 1 mM LaCl_3 , 50 % of total calcium translocated still remained in platelets indicating that cardiolipin exerted an increase of calcium uptake into both lanthanum-displaceable and non-displaceable pool. Both cardiolipin (0.4 mM) and A23187 ($1 \mu\text{M}$) induced a similar enhancement of platelet aggregation by ADP ($2 \mu\text{M}$) or epinephrine ($1 \mu\text{g}$). LaCl_3 (1 mM) also inhibited such enhanced aggregabilities. The results suggest that phospholipids may potentiate platelet aggregabilities by their ionophoretic activities.

Published online: 2019-04-16

INVITED SYMPOSIUM III

Immune Disorders of Hemostasis.

POST-TRANSFUSION PURPURA. Richard H. Aster, M.D., Milwaukee Blood Center, Inc. and The Medical College of Wisconsin, U.S.A.

Post-transfusion purpura is characterized by severe, destructive thrombocytopenia with bleeding manifestations occurring 7-8 days after blood transfusion. In nearly all cases, patients have been women with a past history of pregnancy who received their first blood transfusion. A potent platelet antibody detectable by a variety of methods is invariably present in serum at the onset. In all but two cases studied, this antibody has been directed toward the platelet-specific antigen, PIA^1 , present in 98% of the general population. Patients untreated or given prednisone recover spontaneously in 10-48 days unless fatal hemorrhage supervenes. Exchange transfusion with whole blood or by plasmapheresis appears to have shortened the duration of thrombocytopenia significantly in numerous reported and non-reported instances. Following recovery, patients whose antibody had PIA^1 specificity are found to have platelets that lack this antigen. It is not yet clear how an isoantibody, apparently provoked by blood transfusion, causes fulminant destruction of autologous platelets. Possible explanations include a) cross-reactivity between the isoantibody and an autoantigen, b) transfusion of soluble PIA^1 antigen in the plasma of certain unusual donors with subsequent formation of immune complexes having anti-platelet activity, and c) coating of recipient platelets with transfused PIA^1 , causing transient conversion of platelet type to PIA^1 -positive and permitting destruction of such platelets by anti- PIA^1 antibody. Evidence for these pathogenetic mechanisms will be summarized.