Time
10.15

0852 PLATELET RELEASE REACTION - A PREDICTOR OF DEATH FOLLOWING MYOCARDIAL INFARCTION?

S. Heptinstall*, G. P. Mulley, Patricia M. Taylor and J. R. A. Mitchell, Department of Medicine, University of Nottingham, England.

A comparison has been made of the extent to which ADP can induce the release reaction in platelets from 26 patients who, within 5 days, had sustained a myocardial infarction and from 54 age-matched ambulant control subjects. Platelet rich plasma was prepared from blood collected into heparin (10 u/ml), samples were incubated with ^{3}H-serotonin, aggregation was then induced by adding ADP (10 µM, final concentration) and the extent to which ADP induced the release reaction was determined by measuring the amount of the accumulated ^{3}H-serotonin that was released.

The results obtained for the group of patients who had experienced a myocardial infarction were not significantly different from those obtained for the controls. However, a significant difference ($P = 0.02$) was observed between the results obtained for those patients who died within 12 months of the infarction (mean 29.4% ± 5.6 of accumulated ^{3}H-serotonin released by ADP) and those who survived (mean 12.0% ± 3.3). Of the 8 patients who died, 7 had platelets that released ≥ 20% of the ^{3}H-serotonin that had been accumulated; of the 18 patients who survived only 4 had platelets that released ≥ 20% ^{3}H -rotonin.

Platelet Fibrinogen

Purcell Room

09.00

0853 RELATIONSHIP BETWEEN AGGREGATION AND BINDING OF ^{125}I-FIBRINOGEN AND ^{45}CALCIUM TO HUMAN PLATELETS

E.I. Peerschke, R.A. Grant and M.B. Zucker*, Department of Pathology, New York University Medical Center, New York, New York, U.S.A.

Since calcium and fibrinogen are essential cofactors for ADP-induced aggregation, their mechanisms of action were investigated. Aspirin-treated platelets were filtered through Sepharose 2B equilibrated with cation-poor Tyrode's solution. After adding the radioactive compounds at 22°, platelets were centrifuged through silicone oil. Trapping was assessed in separate samples with ^{14}C-sorbitol. Calcium binding was maximal at 1 hr and with 200 uM $CaCl_2$. Two binding sites could be demonstrated on normal and thrombasthenic platelets and on platelets which had lost their ability to aggregate (but not to change shape or promote clot retraction) after treatment with EDTA (8 min, 37°, pH 7.8). ADP did not alter calcium binding in the presence or absence of fibrinogen. Fibrinogen, however, bound to normal gel filtered platelets in the presence of ADP and ionized calcium but not to thrombasthenic or EDTA-treated platelets or to normal platelets in the presence of EDTA or at pH 6.5. Binding of fibrinogen increased with concentration but saturation was not observed even at physiologic levels. Fibrinogen binding was similar in gel filtered platelets and citrated platelet-rich plasma. These studies indicate that stimulation of platelets with ADP under conditions favorable to aggregation is associated with binding of fibrinogen but not of additional calcium.

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