Haemostatic Changes in Cardiovascular Disease

Level 6 Red Side (Princes Buffet)

Free Poster Session 11.30 – 12.45

Poster Board P6-094

0337 PLATELET INVOLVEMENT AFTER MYOCARDIAL INFARCTION

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After Myocardial Infarction (MI) the Heparin Thrombin Clotting Time (HTCT) of platelet (plt) poor plasma is short, indicating an increase in Heparin Neutralizing Activity (HNA). Plt. Factor 4 (PF4) released in vitro also neutralizes heparin. Does the plasma HTCT in MI reflect PF4 released in vivo? Fifteen patients with MI were compared with 23 controls. The mean HTCT was 43.9 secs. in controls and 14.8 secs. in patients. Plasma PF4 measured by RIA was abnormal in 3 patients but strictly normal in the other 12 (n = 12, mean 3.96 ng/ml; controls n = 22, mean 3.54). There was no correlation between the plasma PF4 and the HTCT. The plts. were frozen and thawed and the patients' plts. released less HNA (0.17 units/10^9 plts.) relative to the controls (0.70 units/10^9 plts.) and there was a tight inverse correlation between the plasma HTCT and the intra-plt. HNA. Plts. were isolated and stimulated maximally with thrombin; then malondialdehyde (MDA) production reflecting PG synthesis was monitored fluorometrically. Patients liberated less MDA (415 ng/10^9 plts.) than the controls (911 ng/plts.). All differences are significant except the PF4. Plasma fibrinogen and α_1 acid glycoprotein were also measured. Thus after an MI and presumably as a result of it, plts. are damaged or "exhausted" as reflected both by a decrease in an enzymatic process - PG synthesis and by a decrease in the content of HNA (? PF4). This interim report also clearly demonstrates that the plasma HTCT does not reflect the same attribute as plasma PF4 detected by RIA.

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0338 PLATELET FUNCTION STUDIES IN NORMAL VOLUNTEERS AND PATIENTS WITH ANGINA PECTORIS

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In 30 normal subjects (group I) and in 89 patients with angina pectoris we studied: the platelet survival time (PST), the platelet aggregation test I (PAT I) acc. to Breddin, the platelet aggregation ratio (PAR) acc. to Wu and Hoak and the Filtragometer log TA acc. to Hornstra. The patients were divided in two groups: 46 patients had already been treated for 6 months with clofibrate (group II) and 43 patients with placebo (group III) in a double blind trial. The average PST ($T_{\frac{1}{2}}$) was within the normal range (group I 99 hrs. group II 105,7 hrs.; group III 102,0 hrs.). About 20% of patients of group II and III had abnormally shortened $T_{\frac{1}{2}}$. The PAT I was on average abnormal in group II and III (PAT I in group II 2,3; group III 2,7), but group II normalized after 12 months treatment (PAT I 1,85). The PAR was abnormal in group III, while group II was within the normal range (group I 0,87; group II 0,82; group III 0,69). The log TA results were abnormal in group II and III (group I 2,45; group II 2,1; group III 2,1), after 12 months treatment the patient group remained abnormal (group II 2,2; groip III 2,1). We failed to find a correlation between the four platelet function tests, nor with these tests and basic laboratory values. The PAT I, the PAR and the Filtragometer seems to be valuable in the detection of abnormal platelet behavior in vitro, but it does not mean than an abnormal platelet survival in vivo occurs in the same individuals.