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PLASMA β-THROMBOGLOBULIN AND PLATELET FACTOR 4 IN PATIENTS WITH CHRONIC RENAL FAILURE AND EFFECT OF HEMODIALYSIS. Y. Endo, S. Mamiya, M. Satoh and K. Takahashi. The Third Department of Internal Medicine, Akita University School of Medicine, Akita, 010, Japan.

We have reported that β-thromboglobulin (β-TG) and platelet factor 4 (PF_4) increased in chronic renal failure. The purpose of the current study is to reveal a correlation between plasma β-TG (Amersham Corp. England) and renal function, a correlation between plasma β-TG and PF_4 (Abbott Lab., USA) and the effect of hemodialysis on patients with chronic renal failure.

Significantly increased levels of plasma β-TG (76.8±25.5 ng/ml, $p<0.01$) were observed in 24 patients with chronic renal failure (BUN>20mg/dl), compared to normal subjects (13.2±5.6ng/ml). The increase in β-TG was highly correlated with BUN ($r=0.651$, $p<0.01$), creatinine ($r=0.778$, $p<0.01$) and creatinine clearance ($r=-0.723$, $p<0.01$). Although plasma PF_4 (normal 5.0±2.0ng/ml) increased also, no statistical significance could be found. Statistical correlation between β-TG and PF_4 was not found in these patients. This reason is thought to be due to the difference of molecular weight (PF_4 8000MW, β-TG 36000MW) and half-life (PF_4 30min, β-TG 100min). The high levels of β-TG (89.4±3.4ng/ml) showed a further increase (109.4±5.8ng/dl, $p<0.01$) after dialysis. This is thought to be due to hemoconcentration, because of no adhesion of platelet to cellulose membrane but about 20% elevation in mean of other blood factors such as RBC, WBC, platelet, fibrinogen etc. The PF_4 levels (before, 7.7±1.3ng/ml) which increased at 15min (55.2±19.6ng/ml, $p<0.01$) and 1 hr (23.7±8.4ng/ml, $p<0.01$) are thought to be due to the influence of heparin infusion. The change in PF_4 was not accompanied by the change in β-TG. During hemodialysis the decrease of other platelet functions such as adhesiveness, aggregation induced by ADP, collagen and PF_3 remained unchanged.

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THE PLATELET SPECIFIC PROTEINS DURING CARDIO PULMONARY BYPASS. G. CELLA, V.GALLUCCI, O. VITTADELLO AND A. GIROLAMI. "Semeiotica Medica" and Dep. of Cardiovascular Surgery, University of Padova, Italy and VA Hospital, West Roxbury, and Harvard Med. School, Boston, USA.

Cardiopulmonary bypass is extremely damaging to platelets. In fact, it causes a quantitative and qualitative alteration in their function. When platelets adhere and aggregate or are broken up on foreign surfaces they release their granular contents. We evaluated the release of two platelet specific proteins B-thromboglobulin (BTG) and Platelet factor 4 (PF4) in 20 patients who underwent extracorporeal circulation for open heart surgery. All the patients were perfused with Pemco modular pump and a Bently disposable oxygenator. BTG and PF4 were estimated using commercial radioimmunoassay kits. A parallel release (basal value, under anesthesia and sternotomy, BTG:119.6 ng/ml,PF4:30 ng/ml) was present for both proteins in time dependent fashion until the end of extracorporeal circulation. High average levels were observed in patients in whom the bypass was stopped after about one hour (BTG 1606 ng/ml, PF4 745 ng/ml) and similarly in those in whom bypass was stopped after about two hours (BTG 1540 ng/ml, 745 ng/ml). No correlation was found either between the level of PF4 and the additional heparin administered after the initial standard dose ($r=0.29$. $p>0.10$) and between the level of PF4 and the amount of heparin consumed during the bypass ($r=0.05$, $p>0.47$).

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MORPHOLOGY OF VITAL AVIAN THROMBOCYTOID CELLS H.Janzarik, Department of Internal Medicine, University of Giessen, Germany.

The morphologic criteria of vital avian thrombocytoid cells are not well established. In the present study the different forms of chicken and duck thrombocytoid cells have been analyzed by phase and interference contrast microscopy and compared with those of erythrocytes, erythrocyte ghosts, granulocytes, monocytes, and lymphoid cells.

Since the nucleated thrombocytoid cells do not differ in size from the other white blood cells, they can easily be confused with them and even with the nucleated erythrocyte ghosts which also are capable of adhesion and aggregation but do not spread. According to previous investigations using membrane immunofluorescence tests, thrombocytoid cells may be closely related or identical with B lymphocytes (Janzarik et al. Developmental and Comparative Immunology 1980, 4,123). However, thrombocytoid cells are the only cells with platelet-like spreading and strong adhesion to collagen fibrils. In fresh blood specimens they are spherical and cannot be differentiated morphologically from lymphoid cells. After aging in vitro, suspended thrombocytoid cells elongate to spindle forms.

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THE EFFECT OF STORAGE ON PLATELET MORPHOLOGY. A. Sturk[*], L.M. Burt[o], T. Hakvoort[*], J.W. ten Cate[*], N. Crawford[o]. * Department of Haematology, University Hospital "Wilhelmina Gasthuis", Amsterdam, The Netherlands. o Department of Biochemistry, Royal College of Surgeons, London, England.

Platelet concentrates were stored for one, two or three days at 4°C (unagitated) or room temperature (unagitated and linearly agitated). The morphology of platelets in platelet concentrates, directly after twice washing at room temperature and after 60 min incubation of the washed platelets at 37°C was investigated by both scanning and transmission electron microscopy.

Platelets in the freshly prepared concentrates are slightly activated, i.e. show some pseudopod formation. At 4°C platelets rapidly loose their discoid shape. After three days their surface membrane shows extensive folding, they are slightly vacuolated and have lost most of their granules. Incubation of these cold-stored platelets at 37°C does not induce reversal to the discoid shape. Room temperature storage results in reversal of the slight initial platelet activation. After three days unagitated platelets are slightly more vacuolated than platelets stored with agitation. Room temperature storage usually results in remarkably well preserved, discoid platelets. Occasionally however, agitated platelet concentrates contain a high proportion of odd shaped cells. As platelets stored at 4°C did not became discoid after incubation at 37°, the altered membrane structure could provide an explanation for their short survival upon transfusion. Our results also provide a morphological correlation with the slightly better recovery and survival of platelets stored agitated vs.- non-agitated platelets at room temperature.