

PLATELET ADHESION AND AGGREGATION IN BEHCET'S SYNDROME. J.G. Brook, M. Aviram, S. Haim, A. Markel, A. Weiner, A. Marmur, Y. Berkowitz, I. Bornstein, and M. Youdim. Lipid Research Unit, Rambam Medical Center, Faculty of Medicine, Technion, Haifa, Israel.

Both venous and arterial thrombosis can be a feature in some patients with Behcet's Syndrome. The pathogenesis of the coagulopathy is as yet ill understood. We have examined platelet adhesion and platelet aggregation in 14 patients with proven Behcet's Syndrome and compared them with matched controls. Platelet adhesion was measured by two techniques recently developed in our laboratory. In the first platelet adhesion is determined when platelet rich plasma is applied to a glass slide while applying a predefined centrifugal force. In the second method citrated whole blood is pumped through an inert silicone tube and applied to a glass slide in the form of a stagnation point flow field. The number of platelets adhering to the slide per unit area is determined. The conditions of blood flow through this system were similar to those prevailing in arteries of similar size. Adhesion is found to be significantly greater in Behcet's patients by both methods. There was a 30% increase in platelet adhesion in the Behcet patients. Platelet aggregation was determined by the Born method. Platelet rich plasma was used and 5HT, epinephrine, ADP, and collagen were the aggregating agents. In our hands platelets from the Behcet's patients are more sensitive to aggregation induced by epinephrine and ADP than platelets from our controls.

It appears that disturbed platelet function may play an important role in the coagulopathy of Behcet's Syndrome.

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PLATELET THROMBOXANE INCREASE DURING CELL SEPARATION IN HEALTHY BLOOD DONORS. H. Riess, E. Hiller, W. Schramm, J. Howe. Department of Medicine, University of Munich and Bavarian Red Cross Munich, FRG.

An increasing number of healthy persons is subjected to extracorporeal blood systems in order to obtain platelet and granulocyte concentrates. To assess the influence of extracorporeal circulation on hemostasis we determined a spectrum of parameters in 20 blood donors before and after cell separation.

Obviously anticoagulation with ACD (acid citrate dextrose) is effective in preventing the activation of the plasmatic clotting system:

There were no significant changes in the levels of fibrinogen, plasminogen, soluble fibrin monomer complexes (SFMC, measured by quantitative gel filtration), antithrombin III (AT III, immunological and functional) and fibrin degradation products (FDP, staphylococcal clumping test).

On the other hand we observed a significant rise in the plasma levels of Thromboxane-B₂ (TxB₂, RIA) at the end of cell separation. This value exceeded the starting level more than twice. Serum levels of TxB₂ were unchanged.

From an additional five blood donors blood samples were examined simultaneously from both the input and the output line of the cell separator 30 min after the beginning of extracorporeal circulation. No changes were seen in the fibrinogen-, SFMC-, AT III-, plasminogen- and FDP-levels. There was a persistent but not significant increase in the plasma levels of TxB₂ in the output line of the cell separator whereas there was no such trend in the serum levels of TxB₂.

F CB 3:R-AG and fibrinopeptide A of all 25 blood donors² were measured and will be reported.

The increased levels of plasma TxB₂ at the end of cell separation may express contact activation of platelets.

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PLATELET ACTIVATION IN SEVERE BURNS. P. Hourdille, P. Bernard, P. Fialon, R. Sanchez, M.R. Boisseau, C. Soria J. Soria. Laboratoire d'Hémobiologie, Hôpital Cardiologique 33600 BORDEAUX PESSAC - FRANCE - Hôtel Dieu PARIS - Hôpital Lariboisière PARIS.

Acquired storage pool disease occurs in severe burns from the first week post-burn and during a period which depends on the severity of the burn and the presence of complications. As in congenital storage pool disease we observed a decrease of platelet ADP and serotonin content, abnormal aggregation, impaired ¹⁴C serotonin uptake. But we did not find a good correlation between the content of very dense bodies and the number of granules stained with mepacrine. In the majority of cases, the number of fluorescent granules per platelet remained normal although the ADP and 5-HT levels decreased markedly. These results suggest that mepacrine was able to visualize granular structures even if the granular content was very reduced. For this reason the mepacrine labelling test is of no use in detecting this acquired storage pool deficiency. Coagulation factors have been shown to be involved after burn injury. The decrease of prekallikrein and some time of antithrombin III which occur from the plasmatic coagulation activation is also correlated to the decrease of granular content.

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ACTIVATION OF PLATELETS IN CHRONIC GLOMERULONEPHRITIS (CGN) AND NEPHROTIC SYNDROME (NS). H. Yamazaki, T. Motomiya, C. Sakakibara, S. Ariga, T. Ida, R. Kuriyama, Y. Chida, J. Takeuchi and S. Tomura. Tokyo Metropolitan Institute of Medical Science and Tokyo Medical and Dental University, Tokyo

Platelet count, volume, aggregation, adenine nucleotide contents, plasma β -thromboglobulin (β -TG), plasma platelet factor 4 (PF-4) and urine β -TG were measured in 31 patients with CGN, 20 patients with NS and 51 healthy controls. Platelet counts were higher and volumes were smaller in NS than in controls significantly. Platelet aggregation induced by ADP, adrenaline and collagen was significantly higher in the patients than in controls and it markedly increased in the cases of progressive glomerular lesions. Plasma levels of β -TG were significantly elevated in the patients (65.8±7.5(SE) ng/ml) compared with controls (22.2±1.6 ng/ml) and NS showed markedly elevated plasma β -TG levels (124.3±28.6 ng/ml). Plasma levels of PF-4 were also significantly higher in the patients (28.6±3.6 ng/ml) than in controls (13.2±1.1 ng/ml). There was a significant inverse correlation between plasma β -TG and creatinine clearance (r=-0.47, P<0.01). Urine β -TG excretion was higher in patients with decreased renal function than in those with normal renal function. Plasma β -TG remarkably decreased in 3 of 4 patients with markedly increased β -TG levels when they were given anti-platelet drugs. Platelet ATP contents were 6.98±0.24 in the patients and 8.06±0.42 μ moles/10¹¹ platelets in controls. There was a significant difference (P<0.05). Platelet ADP contents were 3.08±0.15 in the patients and 3.27±0.19 in controls.

The results suggest that platelet aggregation and release reaction increased in patients with CGN and NS. However the decrease in adenine nucleotides in platelets was more marked in ATP than in ADP content. There may be an abnormal metabolism of adenine nucleotides in platelets related to the energy metabolism in addition to a decrease in storage pool in such diseases. Also it is suggested that activated platelets may be an important factor in the genesis of thrombotic tendency in NS.