1330

INDIUM LABELED PLATELET KINETICS IN ASCITIC CIRRHOSIS. J. Reiffers, P. Couzigou, L. Vuillemin, M. Amouretti, C. Beraud, D. Ducassou. Service d'Hématologie, Service de Médecine Nucléaire, Service d'Hépato-Gastro-Entérologie. C.H.R. Bordeaux. (Groupe Hospitalier Sud) FRANCE.

Using Indium-oxine as platelet label, we performed platelet kinetic studies in 30 patients with ascitic cirrhosis. Spleen size was also assessed. All results were compared to that found in 10 controls.

Seven patients had platelet kinetics not different from controls. Five of them were thrombopenic, and presented probably a platelet production insufficiency. Eighteen patients had a shortened platelet survival (less than 7.5 days). These patients were thrombocytopenic and presented a platelet destruction. The major site of destruction was splenic in ten out of these 18 patients. Only five patients had a normal platelet lifespan and an increased splenic pooling. No correlation was found between the following parameters: platelet count, platelet survival, splenic uptake of the radioactivity, spleen size.

It is concluded that in patients with liver cirrhosis and portal hypertension:

1) Hypersplenism (normal platelet survival and high splenic pooling) is a rare cause of thrombopenia.

2) Thrombopenia is probably due to several mechanisms which are associated in a same patient and which are independent of the spleen size.

1331

INDIUM-111-TROPOLONE: A NEW HIGH-AFFINITY TRACER FOR PLATELET LABELING, PREPARATION AND EVALUATION. M. K. Dewanjee, S. A. Rao, and P. Didisheim. Mayo Clinic and Mayo Foundation, Rochester, Minnesota, U.S.A.

Platelets have been labeled with a new, neutral, lipid-soluble metal-complex of Indium-111-tropolone (In-TPL). Unlike oxine, tropolone is soluble in isotonic saline. Oil/saline partition of In-111-oxine and In-TPL are 0.5 and 23.7 respectively. Platelet labeling with In-TPL was performed in both ACD-plasma and ACD-saline media within two hours' time. Increasing concentration of tropolone, citrate ion, and plasma proteins decreases platelet-labeling efficiency. Effects of pH, temperature, platelet concentration, and calcium ion concentration on platelet labeling were studied. In optimum platelet-labeling conditions, canine, rabbit, porcine, and human platelets have been labeled with a consistent labeling efficiency of 80-90%. The optimum labeling conditions are In-TPL in ACD-saline and ACD-plasma at tropolone concentration of 5 and 10 µg/ml respectively and 30 minutes' incubation of platelets with the tracer at room temperature. A kit formulation for convenient routine preparation of In-111-labeled platelets has been developed. The function of the In-TPL-labeled platelets has been studied by their aggregability with adenosine diphosphate. The capacity of In-111-labeled platelets to aggregate in vitro does not correlate well with their ability to circulate in vivo. No adverse effect of In-TPL on platelets has been observed by studies of biodistribution, recovery, and platelet survival in dogs. Comparable mean platelet survival times of four repeated studies in five dogs were obtained in both ACD-saline and ACD-plasma. These results indicate that Indium-111 platelets labeled by tropolone carrier may be preferable to oxine carrier for studies of platelet survival, imaging sites of endothelial damage, and thrombus formation.

1332

THE EFFECT OF A PROTEINASE INHIBITOR ON PLATELET FUNCTION IN STORED BLOOD. S.Haas,P.Wendt,G.Blümel. Institute of Experimental Surgery of the Technical University Munich, FRG

Blood platelets are markedly traumatized by the withdrawal of blood from the donor. Hereby the function of thrombocytes is activated and this platelet stimulation is closely related with the early formation of microaggregates in stored blood. Therefore it is clinically desired to stabilize the platelets but without inhibiting their function irreversibly and causing hemorrhage.Concerning this question an experimental study was carried out.

Blood was drawn from 10 volunteers under blood bank conditions and was stored in presence of 200 KIU aprotinin per ml ACD-blood resp.the same volume of saline at a temperature of +4°C.Immediately after the withdrawal of blood and 1,2,3 and 7 days later blood samples were taken and the following parameters were studied: platelet aggregation induced by ADP, aggregate ratio, PF 4, beta-thromboglobulin and tx B_2 in plasma and serum.

In the aprotinin blood the aggregability was slightly diminished and was longer present than in the control group. In addition, the aggregate formation was significantly decreased.The release reaction of platelets was not effected by aprotinin;the increase of PF 4 and beta-thromboglobulin was similar to that of the control group. Also the thromboxane formation was not effected.Thus the protective effect of aprotinin is independent from the prostaglandin metabolism.

This study shows that a high dose of aprotinin has a stabilizing effect on platelets in stored blood and that the thrombocyte function is not irreversibly inhibited by this substance. Therefore,aprotinin can be regarded to be effective in the prophylaxis of disseminated platelet aggregation without causing a bleeding risk.

1333

PLATELET PHAGOCYTOSIS AS A MODEL FOR PLATELET ADHESION

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It has been suggested that platelet phagocytosis might be a useful model to provide insight into platelet adhesion to polymer substrates commonly employed in biocompatibility studies. To test this supposition the present study of platelet engulfment of four strains of bacteria (opsonized as well as non-opsonized) under well defined in vitro physical conditions was undertaken. In physiologic conditions, platelet adhesion is maximum on the more hydrophilic polymers and minimum on the more hydrophobic surfaces; bacterial engulfment under the same conditions follows an identical pattern in that the more hydrophilic bacteria are more readily engulfed. The experimental data further suggest that, unlike phagocytosis by neutrophils platelet interaction with bacteria is non-specific in that it does not appear to be antibody receptor modulated. Opsonization of the bacteria does however play an important role in that it serves to increase the hydrophobicity of the bacteria thereby influencing the degree of bacterial engulfment. A striking correlation between the extent of bacterial engulfment and the Helmholtz Free Energy of Engulfment exists. Platelet adhesion to polymer substrates and platelet engulfment of bacteria appear to follow the same thermodynamic model.