MIXED POSTERS IV

Platelets

A COMPARATIVE STUDY OF THE EFFECTS OF HEPARIN AND A LOW MOLECULAR WEIGHT SEMI-SYNTHETIC HEPARIN ANALOGUE UPON PLATELET FUNCTION. R. Michalski, D.A. Lane and V.V. Kakkar. Thrombosis Research Unit, King's College Hospital Medical School, London.

We have already reported (1) some in vitro and in vivo properties of a low molecular weight glycosaminoglycan polysulphate. It was found that while this semi-synthetic heparin analogue (SSHA) was virtually inactive in a number of in vitro clotting assays, following intravenous or subcutaneous injection it has a more specific anti-Xa potentiating effect than heparin. In the present communication a comparison has been made of some effects of SSHA and heparin upon platelet function. In several of the in vitro tests performed, such as their potentiating effect on ADP and adrenaline induced aggregation and their effects on the aggregation of washed platelets by Factor Xa, heparin proved to be far more potent than SSHA. It was found that after intravenous injection of both drugs, PRP samples containing comparable anti-Factor Xa activities responded differently to the addition of thrombin as SSHA barely inhibited thrombin induced aggregation. Similarly, SSHA had little effect on the dilute thrombin clotting time of plasma, following intravenous injection. Heparin and analogue were neutralised to approximately the same degree by a crude PF₄ preparation, and similar transient thrombocytopenia effects were observed with both drugs.

1. Thomas, D.P. et al, Lancet, i, 120, 1977.

CHANGES IN DISTRIBUTION OF PLATELET MEMBRANE GLYCOPROTEINS IN PATIENTS WITH MYELOPROLIFERATIVE DISORDERS.

R.B. Bolin, T. Okumuru and G.A. Jamieson, The American National Red Cross, Blood Research Laboratory, Bethesda, Maryland 20014 USA.

A tendency towards hemorrhage can occur in patients with myeloproliferative diseases and in many patients abnormal platelet function can be detected by various techniques. Platelet aggregation studies have shown decreased response to aggregating agents and a few patients may have no response to ADP, collagen and epinephrine and thus may mimic the platelet defect seen in patients with thrombasthenia. We have studied membrane glycoprotein patterns in 16 patients with various myeloproliferative disorders and compared the results to normal controls since glycoproteins have been implicated as being important to platelet function both in normal and pathological states. Although no abnormal glycoproteins were present, there was an abnormal ratio compared to the controls with a relative decrease in glycoprotein I (M.W. 150,000) and an increase in glycoprotein IV (M.W. 87,000) that was statistically significant ($P < 0.005$). There was no discernible correlation between glycoprotein pattern and aggregation response or platelet count but those patients with megathrombocytes did have higher values for glycoprotein IV than those without megathrombocytes. These experiments suggest that patients with myeloproliferative disorders may have alterations in membrane glycoproteins that could alter platelet function.