681

PLATELET DEPOSITION AFTER CAROTID ENDARTERECTOMY DECREASES WITH TIME. AC Meek, P Jarvis, CM Backhouse, CN McCollum, RM Greenhalgh. Department of Surgery, Charing Cross & Westminster Medical School, London, UK.

Platelets are deposited on the exposed media following carotid endarterectomy and will continue to accumulate until neointima covers this thrombogenic surface. Radiolabelled platelet uptake was measured to assess the time to intimal repair.

Autologous 111Indium labelled platelets were infused 2 days and 2 months postoperatively in 10 patients undergoing unilateral carotid endarterectomy. Platelet accumulation was measured daily by gamma camera images counting radioactivity over the operated artery and comparing it to the contralateral side as Carotid Uptake Ratio (CUR).

Mean (±sem) counts per gamma camera cell over the operated side at 24 hours were 46.3±4.3 compared to 38.6±3.9 on the unoperated side ($p<0.001$). At 2 months this difference had disappeared with counts of 38.8±3.2 and 39.1±3.2 over the operated and reference arteries respectively. Early post-operative CUR at 1.22±0.04 was significantly higher than 1.01±0.06 at 2 months which equates to no radiolabelled platelet uptake ($p<0.01$). Radiolabelled platelet uptake was visible on 8 of the 10 early scans, but this was seen in only 2 patients at 2 months, both of whom had a persistently high CUR indicating continued platelet accumulation at that time.

Early postoperative platelet deposition decreases in the weeks following carotid endarterectomy presumably due to the development of a neointima. Those cases with persistently high platelet accumulation may have luminal thrombus which could lead intimal hyperplasia and restenosis.

PLATELET ACTIVATING FACTOR

682

Effect of Platelet Activating Factor(PAF) on the collagen induced platelet aggregation in whole blood. Y.Oura, N.Sakiyama, R.Ueshima, M.Higuchi, E.Kakishita and K.Nagai .The Second Department of Internal Medicine, Hyogo College of Medicine, 1-1 Mukogawa-cho, Nishinomiya, HYOGO, 633 JAPAN

Studies of platelet aggregation are generally performed in platelet-rich plasma(PRP) by the transmittance method. Recently, impedance aggregometry has been introduced which shows the platelet aggregability in whole blood. We compared the impedance aggregometry in whole blood with the transmittance method in PRP, with regard to collagen induced platelet aggregation. The aggregation rate in whole blood increased with increasing concentration of collagen, but remained unchanged in PRP. The factors which influence the platelet aggregation rate in whole blood were studied. CV-3988, that is the specific antagonist of PAF, acetylsalicylic acid (ASA) and phosphocreatine / creatine phosphokinase (CP/CPK) were used in order to evaluate the contribution of PAF, thromboxane and ADP in whole blood. CV-3988 dose-dependently inhibited platelet aggregation induced by collagen in whole blood, but did not inhibit the aggregation in PRP. ASA(10mM) inhibited the aggregation in whole blood incompletely too, but completely in PRP. And the inhibition of CP/CPK (CP/CPK : 1.5mM/50U/ml) was very weak in whole blood compared to that of other antagonists. The inhibitory effect of CV-3988 was investigated on the collagen induced platelet aggregation in whole blood which was pretreated with ASA (10mM) and CP/CPK (1.5mM/50U/ml), resulting in a collagen induced aggregation in whole blood that was not completely inhibited. We conclude that there are some other different factors, which influence platelet aggregation in whole blood, in addition to thromboxane, ADP and PAF.

683

HUMAN POLYMORPHONUCLEAR LEUKOCYTE ACTIVATION INDUCED BY PLATELET ACTIVATING FACTOR (PAF). A. Del Maschio, M. Albors, F. Bucchi, M. Tomasiak, V. Bertelé, C. Cerletti and G. de Gaetano. Istituto "Mario Negri", Milano, Italy.

Human polymorphonuclear leukocytes (PMNs) loaded with the photoprotein Aequorin, were exposed to PAF in the presence of extracellular Ca^{2+} (1 mM). PMNs aggregation measured in the "Platelet Ionized Calcium Aggregometer" (P.I.C.A.) was dependent on the concentration of the stimulus. Ca^{2+} cytoplasmatic increase was monitored in parallel at concentrations of PAF which did not modify cellular integrity (10^{-7}-10^{-5}M). The intracellular Ca^{2+} flux (up to 19±3 µM) triggered by PAF was also concentration-dependent. In order to establish the role played by this intracellular messenger, we studied some cellular responses possibly related to Ca^{2+} mobilization: enzymatic release, oxygen radicals production, and arachidonic acid metabolism. PAF induced release of both lysozyme and β-glucuronidase (15% to 20% of the total enzyme content at the maximal concentration). However PAF (10^{-12}-10^{-4}M) stimulated the production of only small amounts of oxygen radicals as compared to Phorbol Myristate Acetate (PMA). Leukotriene B_4 (LTB_4), the main arachidonic acid metabolite in PMNs and the products of its catabolism (20-OH and 20-COOH LTB_4) were assayed by two different technics (HPLC and RIA) in the same cellular suspensions. PAF (10^{-4} M)-stimulated PMNs ($0.5\text{-}2\times10^{7}$ cells/ml) did not produce any detectable amount of these arachidonic acid metabolites. In contrast, calcium ionophore A 23187 (2 µM)-stimulated PMNs (in the same range of cellular concentration) produce up to 170 ng/ml of LTB_4. In conclusion, cytoplasmatic Ca^{2+} increase in PAF-stimulated PMNs was not accompanied either by oxygen radicals production or by activation of arachidonic acid metabolism catalyzed by 5-lipoxygenase.