Platelets in Diabetes Mellitus

Purcell Room

Time
14.00

0791 THE EFFECT OF ALLOXAN-INDUCED DIABETES ON TISSUE PLASMINOGEN ACTIVATOR ACTIVITY OF THE RAT

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Subcutaneous injection of alloxan (10mg/100g) induced severe diabetes in Sprague-Dawley rats (hyperglycemia, glucosuria, weight loss, polydypsia, polyphagia and polyuria). The effect on tissue plasminogen activator activity (PAA) was studied histochemically in key organs (heart, kidney, lung, aorta, and caudal vena cava) 4 days, 2, 3, 4, 6, and 8 weeks after induction of diabetes. After an initially increased vascular PAA (release reaction) seen in lung, kidney, aorta, and myocardium, but not in caudal vena cava, a decreased PAA was found in arteries of the renal medulla and particularly of the renal cortex and in arteries of the myocardium two to eight weeks after the induction of diabetes. Compared to the normal PAA level, the intima of the aorta showed after the initial rise, a fall in two to three weeks, a second rise by four to six weeks, followed by a fall to the normal value at the eighth week. In the lung the initially increased PAA continued to be slightly elevated until the sixth week; by the eighth week it was normal. In the caudal vena cava no changes in the PAA were seen. Of interest are the observed differences in PAA patterns: (1) between arteries and veins, (2) large and small arteries, and (3) arteries in different tissues up to at least eight weeks post induction of diabetes.

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14.15

0792 PLATELET ABNORMALITIES IN EXPERIMENTAL DIABETES

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Many abnormalities of platelet function occur in patients with diabetes mellitus, particularly those with angiopathy. We have previously demonstrated that prostacyclin (PGI_2) is decreased in streptozotocin-diabetic rats, and have now investigated platelet reactivity in these animals. Responsiveness to ADP and arachidonic acid was increased, and platelet cyclo-oxygenase and thromboxane synthetase activities were significantly elevated ($p < 0.05$) in diabetic animals (5.5 ± 0.7 and 5.9 ± 0.9 arbitrary units/10^9 platelets) when compared with control animals (3.0 ± 0.4 and 3.9 ± 0.3 arbitrary units/10^9 platelets). Malondialdehyde synthesis was 1.5 and 0.9 n moles per 10^8 platelets in diabetic and control rats respectively. Diabetic platelets were also less sensitive to the anti-aggregating effects of PGI_2 (IC_{50}: diabetic, 2.3 ng/ml; control, 1.3 ng/ml. Survival of ^{111}indium-labelled autologous platelets was significantly reduced, indicating that platelet function is abnormal in vivo, in diabetic animals. Platelet hyper-reactivity, possibly associated with depressed PGI_2, could be related to the vascular complications of diabetes.