Tuesday, July 14, 1981

Poster Presentations

Thrombosis, Clinical – V

Platelet Activation

11:00–12:30 h

Grand Ballroom Lobby Boards 249–260

0433

PLATELET FUNCTION CHANGES IN ACUTE MYOCARDIAL INFARCTION. A. Strano, A. Raineri*, S. Novo, G.L. Piraino*, A. Mazzola, M. Traina*, G. Davì. Institute of Clinical Medicine and *Institute of Cardiovascular Physiopathology, University of Palermo, Italy.

Whether the thrombotic component of myocardial infarction is primary or secondary in a given patient, platelet function alterations can influence many mechanism from which depends if the thrombotic lesion grows or sends platelet emboli to the smaller myocardial vessels. Recently in some cases of infarction, coronary artery spasm has been demonstrated angiographically; thromboxanes, vasoconstrictive and platelet-aggregating substances, are released by platelets during myocardial ischemia. The local release of these substances may modify the myocardial cell viability and regional blood flow.

The aim of the present study was to investigate changes in platelet function in relation to the time interval after the beginning of the chest pain in 8 patients suffering from acute myocardial infarction.

The following parameters were estimated: plasmatic PF4 and BTG levels, thromboxane B_2 formation by platelets stimulated with thrombin, plasmatic levels of thromboxane B_2, MDA formation by platelets stimulated with thrombin, platelet sensitivity to exogenous prostacyclin; factor XIII activity was also determined.

The tests showed, in the first three days, an augmented release of platelet constituents together with a "platelet exhaustion", demonstrated by a reduced formation of MDA and thromboxane B_2; in the following days the platelets changed to a state of hyperactivity. The platelet sensitivity to prostacyclin was reduced during the whole period after the onset of the acute myocardial infarction; this provides an additional mechanism involved in increased platelet aggregation.

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PLATELET ACTIVATION DURING TREADMILL EXERCISE IN PATIENTS WITH CHRONIC PERIPHERAL ARTERIAL DISEASE. G. Baele, H. Bogaerts, D. L. Clement, R. Pannier and F. Barbier. Coagulation Laboratory and Angiology Unit, Department of Medicine, University Hospital, Gent, Belgium.

β-Thromboglobulin (β-TG) and platelet factor 4 (PF-4) are specific platelet proteins released during in vivo platelet activation. An increase in PF-4 after exercise-induced myocardial ischemia was reported by Green, L.H. et al.. This observation prompted us to measure β-TG and PF-4 in patients with chronic occlusive arterial disease of the lower limbs and to look for increments during treadmill exercise.

β-TG was measured using the Amersham test kit and PF-4 using the radioimmunoassay kit of Abbott Diagnostics Division. Plasma levels in 28 normal individuals ranged for β-TG from 7 to 39 ng/ml with a mean value of 21.0 ng/ml, for PF-4 from 1 to 19 ng/ml with a mean value of 6.0 ng/ml.

β-TG and PF-4 were measured in 59 patients with chronic peripheral arterial disease before and 5 min. after treadmill exercising till occurrence of claudication. Plasma levels of β-TG before treadmill exercising ranged from 24 to 260 ng/ml with a mean of 77.9 ng/ml, PF-4 levels ranged from 2 to 240 ng/ml with a mean of 30.4 ng/ml. These levels were significantly higher than those measured in normal individuals.

After treadmill exercise β-TG levels showed a statistically significant increase to a mean value of 87.3 ng/ml but PF-4 did not rise significantly (mean value : 32.4 ng/ml). The supplementary increase of already elevated β-TG levels may be explained by enhanced in vivo platelet activation during treadmill exercising till occurrence of claudication. As the clearance of PF-4 from human plasma has been shown to be much faster than the clearance of β-TG, increases in PF-4 levels may be more difficult to detect during dynamic explorations of the vascular system.