EFFECTS OF ACUTE AND CHRONIC INFLAMMATION ON HAEMOSTASIS IN RAT MODELS

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Haemostasis is an integral system that one of the first reacts on inflammatory process sometimes conducting it. Most of the clotting factors interact with inflammatory mediators acting on different stages of inflammatory response. Our work aimed to estimate and compare the responses of haemostasis on acute or chronic inflammation in rat models. Acute inflammation was modeled by intraperitoneal injection of LPS (0.5 mg/kg). Rats were scarified on the third day after the injection. Chronic inflammation was observed while developing the model of insulin resistance induced by the high-fat diet containing 58 % of fats of the total diet during 6 months. Blood of rats was collected by heart puncture using 0.38 % Sodium Citrate as an anticoagulant. Platelet-rich plasma (PRP) was obtained by the centrifugation at 100 rpm for 30 min. Platelet aggregation was induced by 12.5 μM of ADP and measured using Aggregometer Solar 2110. The concentration of fibrinogen was determined by the modified spectrophotometric method using thrombin-like enzyme from the Agkistrodon halys venom. Level of protein C was measured using specific chromogenic substrate S2366 (pyroGlu-Pro-ArgpNA) and activating enzyme from the Agkistrodon halys venom. The activated partial thromboplastin time (APTT) was measured using Coagulometer Solar.

Both studied models led to the inflammatory response that was approved by the decreasing of the level of protein C. Its level was 63±15 % in acute inflammation and 72±8 % at the chronic process. However, the concentration of fibrinogen that was prominently increased during acute inflammation (4.6±1.8 mg/ml against 2.1±0.2 mg/ml in control) was only slightly increased during chronic inflammation (2.8±0.6 mg/ml). The most interesting, we observed the adverse effects of acute and chronic inflammation on the blood plasma clotting in APTT-test and the rate of ADP-induced platelet aggregation. Clotting time in APTT-test was prolonged in acute inflammation (48±10 s) and shortened in chronic process (25±9 s) in comparison to control meanings (35±3 s). Platelets reactivity was also suppressed in acute inflammation (aggregation rate 28±10 %) and enhanced in chronic inflammation (65±9 %) in comparison to control meanings (43±6 %).

Thus the response of haemostasis to acute and chronic inflammation differed much and sometimes was opposite. We can assume the predisposition of rats with chronic inflammation to thrombogenesis. However, acute inflammation resulted in a dramatic decrease of platelet aggregation and prolongation of clotting time in the APTT test. Those data most likely were the evidence of disseminated intravascular coagulation and consumption coagulopathy. We detected ‘slow’ response to chronic inflammation that was characterized by preparation of the system to effective coagulation; and ‘fast’ response, when the coagulation system was over-activated. In both cases, the effective work of the hemostatic system was impaired valuably.

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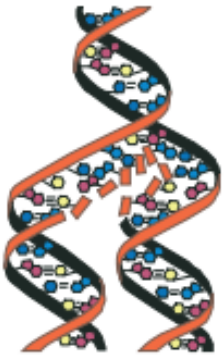

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