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Effect of Amino Acid Complex on Blood, Liver and Kidneys in Rats with Chronic Cadmium Intoxication: Biochemical and Morphological Study

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ABSTRACT

The aim of this work is to study the effect of cadmium on plasma hemostasis, liver and kidneys, since, being a heavy metal, it accumulates in these tissues, causing several pathologies (1). To neutralize the pathological effect of cadmium, the effect of an amino acid complex (AAc) consisting of γ-aminobutyric acid, β-alanine, glutamine and ethanolamine-O-sulfate was studied. Biochemical methods characterizing plasma hemostasis (general coagulability, prothrombin time, thrombin time, activated partial thromboplastin time (aPTT), fibrinogen level, etc.) and histological methods (hematoxylin and eosin staining, Giemsa staining) were used. An increase in general blood coagulability by 30% was established, compared with control animals, which decreased by 57% as a result of intravenous administration of AAc (2). Similar changes occur when determining thrombin and prothrombin time and aPTT, with the exception of factor XII, the activity of which is suppressed by 34-50%. Fibrinogen and calcium levels also change. In rats poisoned with cadmium, sinusoids between the liver plates of the morphologically abnormal hepatocytes filled mainly with morphologically altered red (erythrocytes) and white (leukocytes) blood cells in contrast to intact and treated with AAc rats. In intact rats, both the cortical and juxtamedullary nephrons involved in urine formation contain normal capillary tufts (glomeruli), which are largely absent upon exposure to cadmium, probably as a result of high blood clotting, but which are restored after exposure to AAc.

Keywords: amino acid complex, hemostasis, cadmium intoxication, anticoagulant, hepatocytes

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