THE ASSESSMENT OF CARBOHYDRATE BLOOD METABOLISM IN EARLY PERIOD OF BURN

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Background. The pathogenesis of burn disease is accompanied by an energy and protein deficiency reducing adaptable body's resistance and its compensatory abilities. Lactate dehydrogenase (LDH, EC 1.1.1.27) is involved in carbohydrate metabolism and the regulation of anaerobic and aerobic glycolysis. The products and substrates of lactate dehydrogenase reaction (lactate and pyruvate) play an important role in the control on the relation of the oxidized and reduced forms of NAD in the cell. Therefore, the evaluation of LDH activity and carbohydrate metabolism helps to evaluate early disorders in oxidative-energy reactions in thermal injury.

The aim of this work was to study the activity of LDH, level of lactate and glucose in blood the early period after thermal injury.

Materials and methods. For studies we used blood of 20 patients with thermal injury (burn more than 15% of the body surface, II–III degree, 1 – 4 days after lesion). As control was used the blood of healthy individuals (n=15). The concentration of glucose and lactate was measured on the device Super GL ambulance (Germany). In erythrocytes the LDH activity in the direct and reverse reaction was determined in the spectrophotometer Power Wave XS by Kochetov G. A. [1]. We calculated the coefficient of balance of energy reactions (CBER) according to the formula: CBER = (LDHdir/LDHrev) / (LDHrev/LDHdir)×100, where LDHdir – LDH activity in direct reaction (nmol NADH/min); LDHrev – the activity of LDH in reverse reaction (nmol NADH/min); 100 – the correction factor [2]. The results were processed using the Statistica-6.0.

Results. The results showed that thermal trauma is accompanied by significant changes in carbohydrate metabolism. The activity of LDHdir decreased by 13%, LDHrev – by 49% in comparison with healthy people. CBER increased in 3 times when the burn (CBER is 3,10+0,07 at healthy people and CBER is 9,15+0,18 at patients with burns). The increasing of CBER indicates a decrease in metabolism in patients, the decrease in the rate of the oxidation-energy reactions. The reduction of LDH activity is associated with deficit of oxygen and contributes to the accumulation of blood lactate. The high degree of negative correlation between LDHdir and lactate concentration (r= -0,76, p=0,035) was detected.

The concentration of glucose increased in the burn by 23% compared to the norm. It probably was as a result of acceleration of glycogenolysis and gluconeogenesis, leading to hypermetabolism in the organism. At the same time, the cell in hypoxia spends glucose with the formation of

lactate. We found the increase in the concentration of lactate in 4 times in comparison with healthy people (p=0,023). It characterizes the intensity of anaerobic glycolysis and deficiency of oxygen. It is proof of the development of endogenous intoxication. All patients had a high degree of positive correlation between glucose and lactate (r=0,74, p=0,031). Under hypoxic conditions the cell supports energy needs at the expense of activation of anaerobic glycolysis that partially compensates for the lack of ATP, however, quickly causes an accumulation of lactate.

Conclusion. Thus, to study the concentration of lactate and glucose, the activity of lactate dehydrogenase in direct and reverse reaction and the calculation of CBER will reveal violations of energy metabolism in blood and possible complications in patients with thermal injury in the early period after injury.

References:

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