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CLINICAL STUDY

Can a single lactate value predict adverse outcome in critically ill newborn?

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ABSTRACT

OBJECTIVES: The aim of this study was to investigate the role of the lactic acidosis, as an early predictor of significant consequences and/or a fatal outcome in term neonates after a perinatal asphyxia.

BACKGROUND: Severe perinatal asphyxia can generate multiple organ dysfunction and neonatal mortality.

METHODS: In routine clinical practice, after an admission to the Intensive Care Unit, lactate concentration was determined in capillary blood sa+mples during the first one to six hours after birth in 55 term newborns with the post-asphyxial hypoxic-ischemic encephalopathy. The control group consisted of 36 healthy term neonates randomly selected in the maternity ward at the Gynecology and Obstetrics Clinic.

RESULTS: Significantly higher concentrations of lactate ($p < 0.0005$) were observed in term neonates with post asphyctic syndrome (8.63 ± 4.43 mmol/L) if compared to the control group subjects (1.04 ± 0.36 mmol/L). The increase in lactate level > 8.7mmol/L with 80 % sensitivity and 82 % specificity indicated the development of the hypoxic-ischemic encephalopathy stage II/III, while the lactate level > 9.95 mmol/L was a predictor of death, with 75 % sensitivity and 74.4 % specificity.

CONCLUSION: Determination of lactate concentrations in serum of term newborns associated with risk factors for the perinatal asphyxia is a useful tool in diagnosing metabolic disorders and ischemic damage, particularly severe clinical forms *(Tab. 2, Fig. 3, Ref. 34)*. Text in PDF *www.elis.sk.*

KEY WORDS: oxidative stress, respiration, artificial, respiratory distress syndrome, newborn.

Introduction

Perinatal asphyxia (PA) is the medical condition of impaired gas exchange before, during or shortly after birth, which – if continuing – may cause a progressive decrease in the partial pressure of oxygen, and an increase in partial pressure of carbon dioxide, associated with a drop in blood pH. It is the second leading cause of neonatal morbidity and mortality, after prematurity (1– 3). The PA incidence varies depending on diagnostic criteria. According to the research in developed countries, the incidence of severe PA, (which leads to significant neurological sequelae or death) is about 1/1000 live births (4, 5), while the PA occurs more often in underdeveloped countries. In hospitalized children, it ranges from 5–10/1000 live births, but the data come from developed countries and probably underestimate the actual incidence of perinatal asphyxia in underdeveloped areas (5, 6).

Pathophysiological basis of post-asphyctic lesions in some organs and tissues resulted from an exhausted mechanism of circulatory and metabolic adjustments of newborn to hypoxic-ischemic stress. Fundamental initial vascular changes in hypoxia are vasoconstriction, reducing blood flow through so-called 'peripheral' vascular regions (kidneys, gastrointestinal tract, skin and muscles), increased systemic blood pressure, and maintenance of the blood supply to the brain, myocardium and the adrenal glands. Later, during circulatory decompensation, there are myocardial workload and a reduced blood inflow to the right atrium, reduction in cardiac output, systemic hypotension and a development of ischemia in all organs (7, 8).

In addition to the vascular compensatory response to hypoxia, there are also metabolic changes. Anoxia activates anaerobic glycolysis with a consequently reduced synthesis of adenosine triphosphate and lactate accumulation. In the event of depletion of the glycogen stores, an accelerated catabolism of energy-rich purine bases occur (8, 9) and therefore, hypoxanthine and uric acid accumulate. Reduction of the adenosine triphosphate stores produces severe effects for the weakening of energy-dependent cation transport system (sodium, calcium, potassium), membrane phospholipid disorder and free fatty acids disorder, and the accumulation of various harmful metabolites such as prostaglandins, leukotrienes and free radicals, which lead to further cell damage (9).

In recent years, there has been a great interest in the application of biochemical markers in the assessment of post-asphyctic lesions. In this sense, a so-called 'Biochemical Apgar score' has been suggested for an immediate evaluation of hypoxic-ischemic insult (8, 9). In addition, more and more attention is paid to their

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