METABOLIC STATUS AS AN INDICATOR OF POST-HYPOXIC COMPLICATIONS IN NEWBORNS BORN IN ASPHXIA

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Abstract: The protection of motherhood and childhood is a priority area of health care in our Republic; this provision is enshrined in fundamental government documents, programs for reforming the health system, and a program for creating a healthy generation. According to the International Consensus on Resuscitation of the Newborn, more than a million newborns die every year in the world, and in 19% of cases the cause of death is asphyxiation.

Keywords: newborns, premature infants, hypoxia, asphyxiation, metabolic status.

Introduction

Complications in the posthypoxic period are explained by the accumulation in the blood and tissues of an excess of free radicals, products of lipid peroxidation, proteolytic enzymes, calcium ions in the cytosol of neurons, i.e. changes in metabolic status [5,6]. Moreover, all the symptoms appear only in the structure of multiple organ failure. It is difficult to understand the mechanism of development of this pathology and requires a deep clinical, instrumental and laboratory analysis for the timely diagnosis of posthypoxic syndrome and the correct correction of complex treatment to prevent complications and provide follow-up. Therefore, the allocation of lesions of individual functional systems is relatively arbitrary and requires in-depth analysis to substantiate the diagnosis and pathogenetic treatment [4,8]. Hence the extreme importance of careful medical supervision of these children using mandatory laboratory and instrumental examination of the main parameters of their life.

The air of the study was to identify the components of the formation of posthypoxic pathology in newborns with a history of high risk factors for the development of antenatal and intrapartum hypoxia.

Material and methods: 64 matured newborns were examined in hospital treatment at the neonatal pathology department of the regional children's multidisciplinary clinical medical center. All patients were transferred from maternity hospitals.

Research methods: clinical anamnestic, instrumental diagnostic, laboratory. Clinical, hardware and laboratory monitoring was carried out. Biochemical examination using the KONE apparatus (Analytical systems t/0 Analyticae Systems) 2000

Results

The history of the main causes of the risk of developing posthypoxic pathology in the newborns observed by us was established:

1. the interruption of blood flow through the umbilical cord-20%;
2. violation of gas exchange through the placenta-42%;
3. inadequate hemoperfusion of the maternal part of the placenta-22%;

4. deterioration in oxygenation of the blood of the mother-62%;
5. respiratory failure of the newborn - 30%

More than half of the observations have a combination of 2-3 high-risk factors for the development of posthypoxic complications.

Analysis of metabolic status indicates the development of hyperbilirubinemia during the adaptation period in the first days of inpatient treatment to 225.0 ± 10.5 μmol/l, with indirect bilirubin at the level of 192.6±10.1 μmol/l, with a tendency to increase to 220.8±7.8 μmol/l, this creates a risk of developing neurological symptoms against hypoxia as an aggravated background for the penetration of toxic indirect bilirubin into the tissue. The presence of a history of intrauterine infection risk does not exclude hepatocellular etiology of an increase in direct bilirubin upon admission and in dynamics, respectively 336±12.0 and 362±3.8 with a norm of 0 to 34 μmol/l, which is statistically significant in relation to healthy newborns. Against this background, a combination with a moderate increase in liver enzymes, namely ALT to 0.716±0.085, a marked increase in cholesterol by almost 2 times, alkaline phosphatase to 79.3 + 18.6 μmol/l indicates a tendency to development of cholestatic jaundice, which is against the background intrauterine hypoxia may be associated with immaturity of the enzyme systems of the liver, delayed postnatal maturation of the bile ducts and their differentiation, increased cholesterol is usually combined with hypothyroidism, which is alarming due to the prevailing environmental iodine deficiency sky situation.

Phosphorus, in turn, plays a significant role in myelination of nerve fibers, and hypophosphatemia can exacerbate neurological symptoms. The prevailing hypophosphatemia in combination with a decrease in protein levels indicates a limitation of the participation of phosphorus in protein synthesis, a decrease in the composition of ATP and ADP [3,16]. Hypophosphatemia is combined with a decrease in the level of serum protein from 56.2±12.5 ml mol/l to 51.5±11.5 ml mol/l in dynamics at a normative level of 65 to 80 ml mol/l.

Against the background of imperfection of protein metabolism and insufficiency of the liver synthesizing function of the iron, the level of iron in the observed newborns remains at the level of the lower bounds of indicators in healthy children (F = 8.13 ± 1.46), but with such metabolic changes in liver function, a decrease in the content of macroergic compounds will not provide intensity plastic processes for complete energy metabolism.

The catabolic direction of metabolism in hypoxic complications was expressed in shifts of nitrogen metabolism with a moderate increase in urea level upon admission to 6.5± 0.72 with a norm of 2.5 to 4.5 ml mol/l with a slow tendency to decrease against the background of the ongoing treatment in dynamics.

It is possible that such an increase in urea level with normal creatinine levels is one of the indicators of biochemical shifts in the adaptation period with urate infarction in newborns, but the presence of signs of impaired liver function does not exclude the risk of developing posthypoxic hepatorenal syndrome [13,17]. If we analyze the observed metabolic changes in posthypoxic syndrome in conjunction with the anamnesis, the previous indicators of the laboratory examination carried out by treatment, we can distinguish a causal relationship.

As you know, changes in the neurological status in case of hyperbilirubinemia do not so much depend on the level of bilirubin as on the background state of a burdened history of hypoxia. These pathological factors facilitate the toxic effects of indirect bilirubin on nerve cells and penetration into tissues.

These features of the relationship of hyperbilirubinemia and hypophosphatemia during hypoxic complications in newborns should be taken into account in the differential diagnosis of neurological symptoms of the period of adaptation of newborns. Intrauterine infection of the fetus is not always manifested by specific clinical symptoms, but the change in liver samples in our observations, such as: ALT, cholesterol, alkaline phosphatase,

bilirubin and its fractions, is probably the laboratory phenomenon or reaction, an indicator of predisposition to certain pathological conditions.

A decrease in protein level, in turn, promotes the release and accumulation of toxic indirect bilirubin, and the long-term preservation of prolonged neonatal hyperbilirubinemia with an increase in the neurological symptoms of bilirubin encephalopathy.

An analysis of the same biochemical tests of metabolic status in preterm infants with chronic intrauterine hypoxia or acute asphyxia in history shows a relatively moderate hyperbilirubinemia due to indirect bilirubin, which is statistically significant. In particular, the total bilirubin in the first days of hospitalization is increased to 204.0± 33.4 and for 2 weeks remains at the level of 208.4 ± 12.8 μmol/l; indirect bilirubin from 174.5 ± 25.8 in the first days rises to 243.6 ± 12.4 and direct bilirubin without particular dynamics from 31.83 ± 14.6 to 37.4 ± 16.8.

Indicators of liver tests, in particular, ALT, upon admission, increased to 0.77 ± 0.12 and in dynamics decreased, there was also a significant increase in alkaline phosphatase to 100.4 ±16.2 ml mol/l, which is higher than in full-term newborns. It is known that an increase in alkaline phosphatase occurs at the height of the inflammatory process, at the same time it is observed with rickets, obstructive jaundice, hypothyroidism. Hypophosphatemia in premature infants is moderately reduced to 1.13 ± 0.53 ml mol/l, but decreases in dynamics to 0.84 ± 0.16 ml mol/l, which is almost 2 times less than in healthy children. Hypercholesterolemia to 5.5 ± 0.52 in the early days, in dynamics gradually decreases to 4.6 ± 0.6 ml mol/l.

The revealed shifts in the metabolic indicators of liver function in a premature newborn indicate an aggravation of the state of functional immaturity of the liver cells by the pathological effect of prolonged hypoxia, which, in combination with a number of factors, could contribute to prematurity. Biochemical signs of cholestatic jaundice were expressed, although no pathological changes were detected with ultrasound of the liver and biliary tract.

The level of total serum protein remained at 59.0 ± 5.0 and 58.0±1.0 ml mol/l, which is relatively higher than that of the full-term, but lags behind the standards, which against the background of hyperbilirubinemia can contribute to an increase in neurological symptoms due to bilirubin intoxication. The condition is exacerbated by a parallel increase in indirect bilirubin and hypophosphatemia in dynamics i.e. the inhibition of myelination of nerve fibers coincides with the toxic effects of indirect bilirubin.

An increase in urea level to 6.4 ± 0.6 ml mol/l without any significant changes in dynamics compared to full-term infants with normal creatinine levels indicates an indirect relationship with hyperammonemia, characteristic of premature infants during adaptation. But the lack of a downward trend in dynamics indicates the possibility of developing posthypoxic complications of the urinary system.

Conclusions:

In the absence of appropriate clinical symptoms, these laboratory phenomena serve as an indicator of a specific pathological process. These changes can be identified at the preclinical stage of the disease as a risk factor and therefore can serve as a sensitive marker of predisposition to membrane pathology.

Metabolic predictors of posthypoxic complications during early adaptation are the laboratory phenomenon or an indicator of predisposition to certain pathological conditions.

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