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Functional Activity Of Plasma Hemostasis In Neonatal Calves With Iron Deficiency, Who Received Ferroglucin And Glycopin.

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ABSTRACT

In conditions of iron deficiency anemia, hypoxia phenomena inevitably arise, which increase lipid peroxidation. This can stimulate the coagulating system of blood and weaken its anticoagulant and fibrinolytic system, thereby initiating intravascular fibrinogenesis. In the study, the features of plasma hemostasis in newborn calves with iron deficiency anemia were studied. Also an attempt was made to find an approach to its adequate and most complete correction. A combination of ferroglucin and glycopin, which had a pronounced positive effect on the processes anabolism, hematopoiesis, growth and development of young animals. The effect of this combination on coagulation disorders in newborn calves with iron deficiency anemia was evaluated. It was found that calves with iron deficiency anemia are characterized by increased lipid peroxidation and activation of blood coagulation processes. The combined use of ferroglucin and glycopin in new-born calves with iron deficiency can completely eliminate excessive plasma lipid peroxidation and existing hemocoagulation disorders.

Keywords: plasma, hemostasis, hemocoagulation, calves, iron deficiency, ferroglucin, glycopin.

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INTRODUCTION

Successful development of the economy in any country in the world is possible if the food base is sufficient [1,2,3]. In this connection, livestock breeding is of particular importance in many regions of the world [4]. His active development based on modern scientific information on the possibility of rapid recovery of the organism of productive animals is able to provide the population with a sufficient volume of dairy and meat products [5].

Special attention still deserves iron deficiency anemia. It is still noted in many cattle-breeding farms in Russia in new-born calves. In a large number of cases, it is the cause of their general weakening, growth retardation and death [6]. Under conditions of anemia, hypoxia phenomena naturally arise, which inevitably increase lipid peroxidation, stimulating coagulation, weakening anticoagulant and fibrinolytic system, thereby initiating intravascular fibrinogenesis [7,8,9]. At the same time, the state of plasma hemostasis in newborn calves with deficiency of iron deficiency anemia, approaches to its adequate and most complete correction. It was suggested that the combination of ferroglucin and glycopin, which has a pronounced positive effect on the processes anabolism, hematopoiesis, growth and development of young growth can affect coagulation violations in newborn calves with iron deficiency anemia.

The goal of the work is formulated: to evaluate the effectiveness of the effect of the combination of ferroglucin and glycopin on the functional activity of coagulation hemostasis in neonatal calves with iron deficiency anemia.

MATERIALS AND METHODS

Research was conducted in strict accordance with ethical principles established by the European Convent on protection of the vertebrata used for experimental and other scientific purposes (adopted in Strasbourg March 18, 1986, and confirmed in Strasbourg June 15, 2006) and approved by the local ethic committee of Russian State Social University (Record №12 dated December 3, 2015).

45 newborn calves were examined at which iron deficiency anemia was detected at birth (the number of erythrocytes in the blood was $4.3 \pm 0.24 \times 10^{12}/l$, the hemoglobin concentration was 96.1 ± 0.24 g/l, the iron content in the plasma was 12.6 ± 0.17 $\mu\text{mol}/l$). The control group consisted of 29 healthy newborn calves.

In the calves included in the study, the activity of peroxide oxidation of plasma lipids was determined taking into account the level of acyl hydroperoxides and thiobarbituric acid-active products by the Agat-Med set with an evaluation of the antioxidant activity of the plasma.

For each monitored calf, a determination was made of the level of coagulation factors (I, II, V, VII, VIII, IX, X, XI, XII), duration of activated partial thromboplastin time, prothrombin and thrombin time.

All calves with iron deficiency anemia received a correction of ferroglucose -75 at a rate of 15 mg of iron per 1 kg of body weight of the calf by one injection intramuscularly in combination with glycopin digestion at 6 mg / day in the morning in combination 6 days, starting simultaneously with injection of ferroglucin. Assessment of the state of animals was carried out at the end and after the end of the correction. The statistical processing of the results was carried out by Student's t-test.

RESULTS OF THE STUDY

In the initial state, a decrease in the antioxidant activity of plasma ($22.4 \pm 0.19\%$ versus control - $33.7 \pm 0.14\%$) was observed in calves with iron deficiency leading to activation of blood lipid peroxidation. Thus, their level of primary products of lipid peroxidation of acyl hydroperoxides reached 3.38 ± 0.17 $D_{233}/1$ ml (in the control 1.44 ± 0.09 $D_{233}/1$ ml). The plasma content of the anemic calves of the secondary products of free radical lipid oxidation-thiobarbituric acid-active compounds (5.16 ± 0.31 $\mu\text{mol}/l$) also significantly exceeded the same value in the control (3.46 ± 0.14 $\mu\text{mol}/l$).

As a result of the correction of ferroglucin and glycopin in calves with iron deficiency, an increase in the antioxidant activity of the plasma ($33.5 \pm 0.11\%$) and a decrease in the intensity of blood lipid peroxidation,

estimated by a decrease in the amount of acyl hydroperoxides (1.45 ± 0.18 D₂₃₃/1 ml) and thiobarbituric acid-active compounds (3.43 ± 0.21 μ mol/l to the control level).

Table. Dynamics of plasma coagulation activity in calves with iron deficiency anemia that received ferroglucin and glycopin

Registered parameters	Ferroglucin and glycopin, n=45, M \pm τ		Control, n=29, M \pm τ
	exodus	after correction	
Coagulation factor I, g/l	2.2 \pm 0.08	1.8 \pm 0.11 $p_1 < 0.01$	1.9 \pm 0.10 $p < 0.01$
Coagulation factor II, %	77.4 \pm 0.19	72.5 \pm 0.24 $p_1 < 0.01$	73.9 \pm 0.29 $p < 0.05$
Coagulation factor V, %	126.4 \pm 0.32	87.5 \pm 0.19 $p_1 < 0.01$	87.7 \pm 0.24 $p < 0.01$
Coagulation factor VII, %	71.9 \pm 0.20	71.9 \pm 0.18	71.5 \pm 0.12
Coagulation factor VIII, %	135.5 \pm 0.27	92.8 \pm 0.23 $p_1 < 0.01$	92.9 \pm 0.17 $p < 0.01$
Coagulation factor IX, %	96.9 \pm 0.33	86.4 \pm 0.28 $p_1 < 0.01$	86.7 \pm 0.28 $p < 0.01$
Coagulation factor X, %	61.9 \pm 0.24	61.2 \pm 0.26	61.3 \pm 0.15
Coagulation factor XI, %	93.8 \pm 0.38	92.3 \pm 0.18	92.5 \pm 0.19
Coagulation factor XII, %	91.2 \pm 0.32	90.3 \pm 0.24 $p_1 < 0.01$	90.1 \pm 0.17
Activated partial thromboplastin time, s	28.2 \pm 0.38	39.4 \pm 0.42 $p_1 < 0.01$	39.7 \pm 0.34 $p < 0.01$
Prothrombin time, s	12.4 \pm 0.24	17.2 \pm 0.36 $p_1 < 0.01$	17.4 \pm 0.23 $p < 0.01$
Thrombin time, s	16.0 \pm 0.15	17.1 \pm 0.19 $p_1 < 0.01$	17.2 \pm 0.21 $p < 0.01$

Legend: p - reliability of differences in the initial state of the indicators in calves with anemia in the control, p_1 - the reliability of the dynamics of the calves in the result of correction.

In the initial state in calves with iron deficiency, a significant increase in the activity of I, II, V, VIII and IX coagulation factors was found. This inevitably led these animals to accelerate the clotting time along the external pathway (thrombin time of 12.4 ± 0.24 s), the internal pathway (activated partial thromboplastin time of 28.2 ± 0.38 s) and in the final stage of coagulation-the transfer of fibrinogen to fibrin (thrombin time 16.0 ± 0.15 s). By the end of the correction, the calves had a decrease in activity to the level of the norm of all initially activated coagulation factors while maintaining intact factors at a normal level.

The revealed activity of coagulation testes in the calves receiving correction reflected the significant dynamics of the activity of the individual factors of the coagulation system in these animals (Table). So, as a result of correction, gradual inhibition of activated partial thromboplastin time by 39.7% was established with a simultaneous retardation of prothrombin time by 38.7%. At the same time, thrombin time, reflecting the intensity of the fibrinogen transition to fibrin, in those receiving ferroglucin and glycopin calves, who had iron deficiency anemia at birth, increased by 6.9%.

Thus, the use of a combination of ferroglucin and glycine is able to completely and quickly normalize the activity of plasma hemostasis in newborn calves with iron deficiency anemia.

DISCUSSION

Initially, active peroxidation of blood plasma lipids in calves with iron deficiency anemia in the case of ferroglucin and glycine has experienced a pronounced weakening before its complete normalization due to the emerging antioxidant protection activity of the liquid part of the blood [10-20]. The observed newborn calves showed a normalization of blood activity in all initially coagulated (I, II, VIII, and IX) clotting factors [21,22]. This became possible due to normalization against the background of correction of metabolic and synthetic disorders in the liver [23,24], characteristic of iron deficiency. The gradual slowing of prothrombin time reflected the weakening of mechanisms of activation of plasma hemostasis along the external pathway and was largely due to normalization as a result of the correction in the anemized calves of the intensity of formation and activity that triggers the clotting of thromboplastin [25-35].

The revealed inhibition against the background of the correction of the initially accelerated activated partial thromboplastin time reflected a decrease in the activity of the internal clotting path when the final stage of haemocoagulation estimated by thrombin time slows down, is a consequence of normalization of the content of all clotting factors in the blood plasma of animals [36,37]. The achieved dynamics of plasma coagulation activity ensures that the calves that receive calves correction necessary for this stage of their development have a level of liquid blood properties and an optimal degree of perfusion of the internal organs, supporting the necessary intensity of metabolism for its further growth and development in calf tissues [38-40].

CONCLUSION

For newborn calves with iron deficiency anemia is characterized by increased lipid peroxidation and activation of blood coagulation processes. Combined use of ferroglucin and glycine in new-born calves with iron deficiency completely eliminates excessive plasma lipid peroxidation and existing hemocoagulation disorders.

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