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Physiology Of Antiaggregatory Manifestations Of The Vascular Wall In Newborn Calves With Iron Deficiency, Receiving Metabolic Significant Effects.

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ABSTRACT

One of the often encountered conditions that violate homeostasis in newborn calves is iron deficiency, which can lead to anemia, growth retardation and development, weakening of their resistance. Iron deficiency in the body of newborn calves leads not only to a decrease in hemoglobin in the blood, but also to a decrease in the activity of iron-containing enzymes, including the vascular wall, which inevitably affects its functions. In view of the fairly widespread prevalence of iron deficiency in newborn calves in livestock farms, it still has great practical significance for modern animal husbandry and can be considered as a model of disruption of the hemostatic activity of the vascular wall during the neonatal phase, which allows searching for an approach to eliminate it. In modern times, krezacin, a derivative of aroxyalkyl carboxylic acids, is increasingly used as an effective stimulant for vital activity in calves, exhibits an effective stimulating effect on living organisms, increases survival in adverse and extreme conditions and normalizes life processes, very significantly stimulating hemoglobin synthesis in animals. The work assessed the antiaggregatory activity of the vascular wall in newborn calves with iron deficiency who received ferroglucin and krezacin. In the work applied biochemical, hematological and statistical methods. In the outcome of newborn calves with iron deficiency, a marked decrease in vascular control over platelet aggregation was observed. The possibilities of combining ferroglucin and krezacin in terms of correcting the antiaggregatory activity of the vascular in newborn calves with iron deficiency and signs of anemia are revealed. It was established that the combined correction after 3 days after its completion can significantly weaken the lipid peroxidation in the plasma of the observed animals, greatly enhancing the antiaggregation control of the vascular wall over platelet activity. It can be considered that the combined use of ferroglucin and krezacin in newborn calves with iron deficiency 3 days after the end of exposure brings the antiaggregatory properties of the vascular wall to the control level.

Keywords: anti-aggregation activity, vascular wall, newborn calves, iron deficiency, anemia.

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INTRODUCTION

The newborn phase is the initial stage of early ontogenesis, in which all organs and systems of productive animals, including cattle, adapt to extrauterine existence [1-5]. The optimality of its course ensures the success of the deployment of the genetic program of the calf with the possibility of achieving the most pronounced productive qualities [6-10].

An important element of the homeostasis of a growing organism is the state of the functional ability of the vascular wall [11,12], the development of which many functions of the organism during the entire early ontogenesis [13,14] largely depend on the activity of the growing body. Penetrating all organs and tissues [15], the vessels are closely connected with all systems [16,17], organs, affecting the state of aggregation of the blood [18-20]. In the event of deviations from the physiological state in endotheliocytes, the formation of various hemostatically significant substances [21,22], such as nitric oxide, prostacyclin and von Willebrand factor [23-28], is often disturbed.

One of the frequently encountered conditions that disturb homeostasis in newborn calves is iron deficiency, which can lead to anemia [29,30], retard growth and development of animals, weaken their resistance and provide conditions for the occurrence of many pathological processes, often leading to death [31]. Iron deficiency in the body of newborn calves leads not only to a decrease in hemoglobin in their blood [32,33], but also to a decrease in the activity of iron-containing enzymes [34,35], including the vascular wall [36,37], which inevitably affects its functions [38]. Due to the fairly high prevalence of iron deficiency in newborn calves in livestock farms, it still has great practical significance for modern animal husbandry and can be considered as a model of disruption of the hemostatic activity of the vascular wall during the neonatal phase, which allows searching for an approach to eliminate it [38].

In modern biological science, great importance is attached to the study of the age aspects of hemostasis [39] and the prevention of its disorders, including in case of iron deficiency anemia in newborn calves [40].

In modern times, krezacin, a derivative of aroxyalkyl carboxylic acids, is increasingly used as an effective stimulant for vital activity in calves, exhibits an effective stimulating effect on living organisms, increases survival in adverse and extreme conditions and normalizes life processes, very significantly stimulating hemoglobin synthesis in animals [41]. However, the impact of this drug on developing vasopathy and the mechanisms of their implementation in newborn calves with iron deficiency against the background of the iron preparation was not previously assessed.

The possibilities of this combination of drugs in terms of a quick correction of the antiaggregatory capabilities of the vascular wall of newborn calves with iron deficiency remain unclear.

The aim of the work is to establish the antiaggregatory activity of the vascular wall in newborn calves with iron deficiency against the background of ferroglucin and krezacin.

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).

In the conducted study, 38 newborn calves of black-and-white breed with iron deficiency and signs of anemia (serum iron 12.4±0.25 μmol/l, siderocytes 1.6±0.10%, hemoglobin count 82.4±0.19 g/l, erythrocytes $4.0\pm0.12\times10^{12}$/l). The control group included 31 healthy newborn calves of black and motley breed.

The expression of lipid peroxidation (LPO) in plasma was determined by the level of thiobarbituric acid in it - active products using the Agat-Med kit and the acyl hydroperoxide (AGP) with the elucidation of the level of antioxidant activity (AOA) of the liquid part of blood. The antiaggregation potential of the vessel wall was determined according to the degree of inhibition of platelet aggregation (AP) with all tested inducers during

temporary venous occlusion with calculation of the index of antiaggregatory activity of the vascular wall (IAAVW) resulting from dividing the time of AP after temporary venous occlusion without its time. Counting the number of platelets in the blood was carried out in the chamber Goryaeva. AT was recorded by visual micromethod with ADP (0.5×10^{-4} M), collagen (dilution 1:2 of the main suspension), thrombin (0.125 U/ml), ristomycin (0.8 mg/ml), adrenaline (5×10^{-6} M), as well as with combinations: ADP and adrenaline, ADP and collagen, adrenaline and collagen.

In all animals, iron deficiency and anemia were corrected with ferroglucin in 150 mg (2 ml) intramuscularly, twice with an interval of 4 days and krezacin 5 mg/kg once a day in a feeding scheme for four days, starting simultaneously with the first injection of ferroglucin. All laboratory parameters taken into account were determined before the start of the correction and 3 days after its completion. Statistical processing of results carried out by t-student criterion.

RESULTS AND DISCUSSION

With the use of ferroglucin and krezacin, optimization of the red blood characteristics was achieved however, for the rest of the clarified indicators, the severity of the correction was incomplete.

Newborn calves with iron deficiency when included in the study showed increased plasma LPO (AHP 3.41±0.15 D_{233}/1 ml, tmobarbituric acid-active products 5.59±0.09 µmol/l, AOA 21.5±0.19%). Against the background of the use of ferroglucin and krezazin, a decrease in plasma LPO processes was noted - AHP decreased to 1.92 ± 0.03 D233 / 1 ml., thiobarbituric acid-active products to 4.11±0.07 µmol/l, AOA was 29.4±0.17% (in the control 1.43±0.10 D_{233}/1 ml, 3.47±0.17 µmol/l and 34.1±0.29%, respectively). Obviously, the weakening of LPO in plasma in calves with iron deficiency, receiving ferroglucin and krezacin, improves metabolic processes in the vascular endothelium, enhancing its hemostatic functions.

In the initial state in calves with iron deficiency, a pronounced depression of the IAAVW was detected in relation to all agonists evaluated and their combinations. The maximum value of the IAAVW belonged to adrenaline due to the greatest inhibition of AP with this agonist in the sample with temporary venous occlusion. To a small extent, it was inferior to the value of IAAVW with ristomycin and ADP. IAAVW with thrombin was 1.20±0.04 (1.49±0.11 in the control) and 1.19±0.06 in the control (1.60±0.07 in the control). Low values of IAAVW in the case of simultaneous use of two inductors indicated significant suppression of the production of the vascular wall of antiplatelet agents in conditions close to real.

The use of ferroglucin and krezacin in iron-deficient calves was accompanied by an increase in IAAVW for all inductors used and their combinations (see table). The highest at that was the value of IAAVW with ADP, it was slightly inferior to the values of IAAVW with adrenaline and collagen. IAAVW with thrombin (1.45 ± 0.07) and with ristomycin (1.44±0.05) were even less. The values of IAAVW, when two inductors were applied at once, also increased, but did not reach the level of control. The increase in IAAVW in calves with iron deficiency on the background of ferroglucin and krezacin was largely due to the positive effect of the correction on the intensity of lipid peroxidation, reducing violations of receptor and postreceptor mechanisms in the vascular wall. The increase in IAAVW with ristomitsin in calves with iron deficiency, receiving estimated correction, indicated a weakening to the level of close to control synthesis in her von Willebrand factor.

Being one of the most important stages of ontogenesis, the neonatal phase largely determines the adaptation of the organism to environmental conditions. It is in the neonatal phase that all organs and systems form with an adequate activation of the genetic program of a living being under the influence of environmental factors [42]. An important system that binds together the body of a newborn animal is the vascular system. It is multifunctional and through a number of mechanisms is connected with other systems, organs, in turn, affecting the state of aggregation of blood. The activity of the vessel wall, including in young productive animals, determines the level of factors in the blood that support the optimal blood rheology and, thus, the homeostasis of the growing organism [43].

Congenital iron deficiency anemia is accompanied by impaired functioning of many organs and systems [44], including the walls of blood vessels with the formation of vasopathy. Depression of antioxidant protection of plasma of newborn calves with anemia against the background of hypoxia developing in them contributes to the activation of lipid peroxidation in the plasma, disrupting the integrity of the endothelium

and increasing endotheliocytemia. At the same time, the activation of plasma LPO in newborn calves with iron deficiency also causes a pronounced weakening of the antiaggregation ability of the vascular wall, apparently due to a decrease in the synthesis activity of prostacyclin and NO in it, which lowers the proper level of microcirculation in the tissues necessary for the organism in the beginning of extrauterine the existence of [45].

So, against the background of temporary ischemia of the venous wall in newborn calves with iron deficiency, there was a lack of decrease in the adhesive ability of the blood plates, which was provided by the weakness of at least two mechanisms. The first is insufficient control from the side of the vascular wall over the density of collagen receptor glycoproteins Ia - IIa and VI on the platelet membrane, which is indirectly established by the non-expression of inhibition of AP with collagen during transient venous ischemia. The second mechanism of depression of control over platelet adhesion in anemic newborn calves is associated with a significant increase in von Willebrand factor production by vascular structures and its intensive binding to its receptors - (GPI b) on the surface of blood plates with insufficient ability of physiological antiplatelet agents secreted by the vessel wall to limit this process [46].

In addition, in conditions of low vascular release of physiological antiaggregants, excessive fixation of strong aggregation agonists – collagen and thrombin to receptors on the platelet membrane – is ensured, enhancing the activity of phospholipase C in them, stimulating the phosphoinositol pathway of activating blood platelets, causing phospholirination of the contractile system proteins in them [47]. Under conditions of lack of education in the vessels of PGI_2 and NO, the effect of weak aggregation inducers - ADP and adrenaline on platelet receptors is also excessive, which causes high expression of fibrinogen receptors (GPII-IIIa) and significant activity of phospholipase A_2, regulating the release of phospholipids arachidonic acid [48].

The weakening of the antiaggregatory activity of the vascular wall in newborn calves with anemia was also confirmed on the model with the combined use of aggregation inducers, showing the insufficiency of the production of vascular disaggregating substances, largely simulating the actual blood flow conditions in animals in which a number of agonists are simultaneously present.

As a result of the use of ferroglucin and krezacin in newborn calves with iron deficiency, vascular control of the adhesion ability of the blood platelets as a result of activation of the initially impaired mechanisms was enhanced. One can speak of increased control from the vascular wall over the density of collagen receptor glycoproteins Ia-IIa and VI on the platelet membrane, detected indirectly by marked inhibition of AT with collagen during transient venous ischemia. In addition, as a result of the correction, depression of the control of platelet adhesion in anemic newborn calves was eliminated as a result of a significant weakening of von Willebrand factor production by the vascular structures and weakening of its binding to its receptors (GPIb) on the surface of blood platelets while enhancing the ability of physiological antiaggregants released the vessel wall to limit this process [49,50,51].

The above correction stimulated the release of physiological antiplatelet agents from the vessels, limiting the fixation of strong aggregation agonists – collagen and thrombin to the receptors on the platelet membrane, weakening the activity of phospholipase C in them, inhibiting the phosphoinositol pathway of activation of blood plates, causing defospholiation of proteins of the contractile system in them. The growth of education in the vessels PGI_2 and NO action is weak inducers of aggregation - ADP and epinephrine on the receptors of the platelets is also limited, which leads to a decrease in the expression of fibrinogenic receptors (GPIIв-IIIa) and decrease the activity of phospholipase A_2 regulate the release from phospholipids of arachidonic acid [52,53].

The increase in newborn calves with anemia of the antiaggregatory activity of the vascular wall was confirmed on the model with the combined use of aggregation inducers, showing a significant increase in the production of vascular disaggregating substances, largely simulating the actual blood flow conditions in animals.

CONCLUSION

n the case of the development of iron deficiency and signs of anemia in newborn calves, there is a marked weakening of the antiaggregatory capacities of the vessels. The combined use of ferroglucin and

krezacin in newborn calves with iron deficiency 3 days after the end of exposure brings the antiaggregatory properties of the vascular wall to the control level.

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