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Physiological Control Of The Vascular Wall Over Platelet-Induced Aggregation In Newborn Calves With Iron Deficiency.

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ABSTRACT

Being highly vulnerable in the neonatal phase, the vascular wall in case of negative changes in the body can weaken the antiaggregatory control of platelets. One of such negative and fairly common conditions in newborn calves is iron deficiency, which still causes significant economic damage to farms of various forms of ownership. The formation of an anemic state contributes to the development of vascular dysfunctions with a decrease in the production of biologically active substances in them, leading to the deterioration of various functions of tissues and organs. In newborn calves with iron deficiency, an increase in platelet aggregation functions and a weakening of the antiaggregation activity of the vessel wall were detected. The basis of these disorders in calves is the activation of plasma lipid peroxidation and impaired synthesis in the vessel wall of prostacyclin. It can be considered that the development of iron deficiency can cause a number of complex disorders in the body, accompanied by the development of thrombocytopathy and degeneration of the vascular endothelium. Vascular dysfunction entails the activation of platelets, which, together with other metabolic disorders characteristic of iron deficiency, contribute to the weakening of the antiaggregatory activity of the vascular wall, leading to a deterioration of microcirculation in the tissues.

Keywords: newborn calves, platelets, vascular wall, antiaggregation, iron deficiency.

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INTRODUCTION

Having a great length, the vessels are closely connected with all systems and organs and largely determine many of their functions [1,2,3]. Through the synthesis of biologically active substances in their walls, the vessels regulate the severity of aggregation of blood corpuscles [4,5], including platelets [6], considered to be the trigger element and substrate for all hemostasis in biological objects [9]. Being very vulnerable in the neonatal phase, the vascular wall with negative changes in the body can weaken the antiaggregation control of platelets [10,11]. One of these negative and fairly common conditions in newborn calves is iron deficiency [12,13], which, despite great advances in research on livestock [14,15], causes significant economic damage to farms of various forms of ownership due to negligence and negligence of performance of their functions by lower-level employees [16.17].

The formation of the anemic state contributes to the development of vascular dysfunctions with a decrease in the production of biologically active substances in them, leading to the deterioration of various functions of tissues and organs [18-20]. It is of great scientific and practical interest to study the dynamics of the antiaggregation possibilities of the vascular wall in newborn calves with iron deficiency. In this regard, the present study was planned and conducted.

The aim of the work is to evaluate the antiaggregation potential of the vascular wall in newborn calves with iron deficiency.

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).

A total of 154 newborn calves of black-and-white iron-deficient breeds that had impaired erythropoiesis and changes in blood parameters were examined. Their hemoglobin count was 82.6±0.24 g/l, erythrocytes 4.1±0.12x10^{12}/l, siderocytes 1.5±0.08% with serum iron levels of 12.9±0.16 µmol/l. The control group consists of 31 healthy newborn calves.

The activity of lipid peroxidation (LPO) in the plasma of animals was estimated by the content of thiobarbituric acid-active products in it in the Agat-Med kit and the acyl hydroperoxide (AHP) with the registration of the antioxidant activity (AOA) of the liquid blood. Anti-aggregation activity of the vessel wall was detected by the effect of temporary venous occlusion on platelet aggregation (AP) with inductors and their combinations by calculating the anti-aggregation index of the vascular wall (IAAVW) while dividing the AP time against the background of venous stagnation by its duration without it. AP was determined using a visual micromethod using a series of inductors: ADP (0.5×10^{-4} M), collagen (dilution 1:2 of the main suspension), thrombin (0.125 units / ml.), Ristomycin (0.8 mg / ml.), adrenaline (5×10^{-6} M.) and their combinations: ADP and adrenaline, ADP and collagen, adrenaline and collagen, which allowed to create conditions close to intravascular. Statistical processing of the results obtained was carried out using Student's t-test.

RESULTS

As a result of the examination of newborn calves with iron deficiency, the intensification of LPO processes in plasma was found. Thus, the concentration of thiobarbituric acid-active products in it was 5.54±0.09 µmol/l, in the control - 3.47±0.17 µmol/l. The content of AHP in the plasma of calves with iron deficiency was 3.42±0.07 D_{233}/1 ml (in the control 1.43±0.10 D_{233}/1 ml). The revealed activation of LPO in plasma in animals with iron deficiency was possible as a result of a significant weakening of its antioxidant activity - 21.6±0.12% (in the control - 34.1±0.29%).

In the observed calves with iron deficiency, the AP acceleration was found, most pronounced under the influence of collagen - 22.8±0.05 s (in the control 30.8±0.11 s). Somewhat slower AT developed under the action of ADP (24.7±0.14 s) and ristomycin (24.0±0.10 s). Thrombin and adrenaline antibodies also appeared faster than in control and occurred in 38.2 ± 0.08 s and 66.6 ± 0.15 s, respectively. The development time of

AP against the background of a combination of inductors was also shortened: ADP + adrenaline - 18.4±0.07 s, ADP + collagen - 17.4±0.11 s, adrenaline + collagen - 13.0±0.09 s.

In newborn calves with iron deficiency on the background of venous occlusion, the most pronounced inhibition of AP was noted with adrenaline - IAAVW 1.32±0.08 (in the control 1.63±0.06). Slightly lower, the IAAVW is registered with ristomycin (1.28±0.12) and ADP (1.26±0.05). IAAVW for thrombin and collagen antibodies were even less - 1.21 ± 0.04 and 1.20±0.03, respectively. The indices of the aggregation activity of the vascular wall with the combination of inductors were also below the control: for ADP + epinephrine 1.21 ± 0.02, ADP + collagen - 1.19±0.03, epinephrine + collagen - 1.20±0.04.

DISCUSSION

The development of iron deficiency causes a number of complex disorders in the body, accompanied by the development of thrombocytopathy and degeneration of the vascular endothelium [21,22]. Vascular wall dysfunction entails the activation of platelets [23-25], which, together with other metabolic disorders characteristic of iron deficiency [26-30], contribute to the weakening of antiaggregatory activity of the vascular wall [31], leading to an increase in AP [32,33]. High platelet aggregation under the influence of various in vitro inducers indicates an increased activity of platelets in vivo [34,35]. One of the most important mechanisms of this enhancement may be the activation of thromboxane formation in the blood platelets [36,37]. In addition, there is an increase in the concentration of von Willebrand factor involved in the aggregation process [38–40], indirectly registered to accelerate AP with ristomycin [41.42]. At the same time, arachidonic acid metabolism weakens in the vessel wall with a reduction in the formation of the main thromboxane antagonist, vasodilator, and antiplatelet agent - prostacyclin [43,44].

Evaluation of AP with a combination of two aggregation inducers allows simulating the conditions of platelet activity in newborn calves with iron deficiency, the aggregation process controlled by the vascular wall [45,46] that actually occurs in their bloodstream.

The effect of two inductors at once on the AP process without venous occlusion and against its background in newborn calves with iron deficiency revealed the mutually potentiating effect of plate agonists at their low sensitivity to disaggregating signals of the vascular wall in actual blood flow conditions [47-50]. The low level of control from the vascular wall over AP with a combination of inductors detected during its temporary ischemia indicates a high risk of microthrombus formation in newborn calves with iron deficiency [51-53].

CONCLUSION

The development of iron deficiency causes a number of complex disorders in the body, accompanied by the development of thrombocytopathy and degeneration of the vascular endothelium. Vascular dysfunction entails the activation of platelets, which, together with other metabolic disorders characteristic of iron deficiency, contribute to the weakening of the antiaggregatory activity of the vascular wall, leading to an increase in AT. In newborn calves with iron deficiency, a significant weakening of the antiaggregatory activity of the vascular wall in relation to one and two inductors is observed.

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