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Restoration of Physiological Activity of Platelets in New-Born Calves with Iron Deficiency

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ABSTRACT

New-born calves are still often found with iron deficit. This state negatively influences their growth and development to some extent due to thrombocytopathy development. In this connection great scientific and practical significance for veterinary science and physiology belongs to the search of approaches to effective correction of new-born calves' thrombocytopathy in lack of iron conditions. It seemed to be perspective to evaluate the degree of influence on new-born calves' platelet activity of traditionally applied at iron deficit ferroglukin in combination with metabolism stimulators (polyson and cresacin). During our research it was established that new-born calves with iron deficit also have lowering of plasma antioxidant protectability, intensification of lipids' peroxidation processes, increase of thrombocytes' hemostatic activity. As a result of application of ferroglukin, polyson and cresacin combination to new-born calves with iron deficit we managed to get evident increase of blood plasma antioxidant protectability, significant decrease in it of lipids' peroxidation processes at normalization of thrombocyte activity.

Keywords: new-born calves, iron deficit, hemostasis system, ferroglukin, polyson, cresacin.

INTRODUCTION

It becomes clear that the phase of cattle new-born state is a very important development stage for the whole subsequent ontogenesis[1,2]. Well-being and ill-being of environmental conditions at this life stage can seriously influence subsequent phases of early ontogenesis including realization processes of hereditary information during growth, development and reproduction[3,4]. At the same time, at many Russian farms new-born calves are often found to have different abnormalities negatively influencing their metabolism processes and finally – their growth and development[5]. In previous researches it was found that at homeostasis deviations especially in case of a young organism there can happen quick increase of hemostasis components' activity able to lead to microcirculation disturbance[6,7]. The most numerous researches in this field were made with human beings[8,9,10]. Being based on their results we managed to make a conception of the presence of age-specific dynamics of hemostasis components' activity[11,12], most vulnerable its mechanisms and the potential of different influencing varients on the organism aiming at hemostatic processes optimization. Because of great social significance of thrombosis at cardial pathology[13,14] modern researchers firmly retain their attention at the aspects of hemostatic changes appearing at the given category of patients [15,16,17]. Exactly while studying hemostasiopathy pathogenesis at cardial pathology there was formulated the understanding of its correction possibility not only with the help of usual cardiological means[18,19,20] but there was

shown their minimization possibility with the help of traditional applications [21,22,23] what is really important for biological researches.

As the state of iron deficit is spread enough among new-born calves[1,5] and it is rather often accompanied by development of disturbances in hemostasis system[24] there is a great practical demand in their quick and effective removal among calves at farms. At the same time effective approaches aimed at simultaneous reduction of iron deficit and hemostasiopathy signs are still worked out unsatisfactorily[25].

That's why investigations led with new-born calves with the aim of finding approaches to early and effective hemostasiopathy correction on the model of iron deficit state keep their great scientific and practical significance. Worked out at the given state varients of evidence decrease of hemostasis disturbances can serve the basis for the following creation of correction complexes able to be effective in the field of hemostasiopathy reduction of new-born calves at many diseases. Great interest should exist to the evaluation of influence on the whole hemostasis system of the combination of traditionally applied at iron deficit ferroglukin[25], and earlier shown their high biological activity and ability to influence hemostasis system separate components metabolically active means – polyson[26] and cresacin[27].

In this connection we put the following aim for our investigation – to find the evidence of platelets activity correction of new-born calves with iron deficit with the help of ferroglukin, polyson and cresacin combination.

MATERIALS AND METHODS

The work was fulfilled with 37 new-born calves having the signs of erythrogenesis and decrease of iron content in their organisms (serum iron 13,1±0,09mkmol/l, siderocytes 1,5±0,05%, haemoglobin 98,2±0,25g/l, erythrocytes 4,2±0,18x10^{12}/l). The control group contained 29 healthy new-born calves.

The state of lipids' peroxidation (LPO) in animals' plasma was found out according to the quantity in it of thiobarbituric acid – active products with the help of a set by the firm "Agat-Med" (Russia) and acylhydroperoxides with the account of antioxidant activity level of the liquid part of blood[28]. Thrombocytes' number in calves' blood was found out by their calculation in Gorjaev's chamber. Thrombocytes aggregation was registered by visual micromethod[29] with some inductors: with ADP (0,5x10^{-4} M), with thrombin (0,125un/ml), with collagen (dilution 1:2 of the main suspension), with rhystomicin (0,8 mg/ml), with adrenalin (5x10^{-6} M) in plasma with standardized quantity of thrombocytes in it (200x10^{9} tr.).

The correction of iron deficit state of new-born calves was realized by ferroglukin intramuscularly, once from the calculation of 15mg of iron on 1kg of body mass, polyson 5mg/kg in the morning in the scheme of liquid feeding during 6 days and cresacin – every day 3mg/kg in the scheme of liquid feeding during 6 days beginning simultaneously with ferroglukin application. Evaluation of healthy animals' state was made two times – at their birth and on the 7th day of life. Because of the absence of reliable differences between the results of both investigations control values of each index are presented by one figure – a simple average between them. Examination of calves having iron deficit was fulfilled twice – at their birth and on the next day after correction finish (the 7th day of life). Statistical processing of received data was fulfilled by Student's t-criteria.

RESULTS

Examined new-born calves with iron deficit were found to have characteristic for the given state weakness, limpness, absence of interest to the environment, paleness of rhinoscope and slime layers. These animals were noted to have increased LPO activity in plasma (acylhydroperoxide 3,41±0,022 D_{233}/1ml, thiobarbituric acid- active products 5,20±0,027 mkmol/l at value depression of blood liquid part antioxidant activity 22,2±0,15%). The values of these indices under control were equal to 1,45±0,010 D_{233}/1 ml, 3,46±0,012 mkmol/l and 33,7±0,15% correspondingly.

Thrombocytes' quantity in new-born calves' blood corresponded to norms. Besides,

Table : Parameters of hemostasis in newborn calves with iron deficiency treated with ferroglukin, polyson and cresacin

Consider indicators control,	Calves with iron deficiency, n=37, M±m		
	Outcome	After the correction	n=29, M±m
platelet aggregation with ADP, s	26,0±0,16	40,1±0,12 @$_1$<0,01	40,2±0,08 @<0,01
platelet aggregation with collagen, s	19,2±0,21	31,3±0,08 @$_1$<0,01	31,4±0,08 @<0,01
platelet aggregation with thrombin, s	36,5±0,12	54,2±0,20 @$_1$<0,01	53,8±0,07 @<0,01
platelet aggregation with rystomicin, s	21,0±0,19	48,1±0,14 @$_1$<0,01	48,0±0,12 @<0,01
platelet aggregation with adrenalin, s	67,9±0,23	97,4±0,16 @$_1$<0,01	97,6±0,06 @<0,01

Legend: p - reliability of differences of indicators between the control and the initial state of the calves with iron deficiency, p_1 – reliability of dynamics of indicators in calves with iron deficiency against the background of correction

thrombocytes' aggregation of animals with iron deficit turned out to be reliably increased (table). Their earliest thrombocytes' aggregation appeared in response to collagen (19,2±0,21s), a bit later it developed with ADP and with rhystomicin, still later in response to thrombin (36,5±0,12s). The latest thrombocytes' aggregation of calves with iron deficit appeared under adrenalin influence (67,9±0,23s).

Realized state correction provided examined calves with iron deficit improvement of the common state and their activity, increase of their serum iron level to the control values (23,2±0,21 mkmol/l). On the background of ferroglukin, polyson and cresacin combination examined calves were found to have evident plasma content decrease of acylhydroperoxides (1,70±0,014 D_{233}/1 ml, p<0,05) and thiobarbituric acid-active products (3,87±0,019 mkmol/l, p<0,05) at the increase of antioxidant activity to 28,6±0,16% (p<0,05).

Correction realization of animals having at the beginning iron deficit was accompanied by invariability of thrombocytes' quantity in their blood and slowdown of thrombocytes' aggregation to the control level. Besides, most actively animals' thrombocytes responded by aggregation to collagen, ADF and rhystomicin, less actively – to thrombin and adrenalin addition into plasma (table).

DISCUSSION

Realization of genetically defined growth and development processes of living organisms takes place at constant influence on organism of numerous factors of environment and internal environment[4,30]. Physiological peculiarities of their influence are mostly expressed by the optimum of living beings' blood content[31,32] especially as far as hemostasis system components' activity is concerned[33,34]. Besides, any disturbances in an organism are accompanied by negative dynamics of hematological indices[9,35] including parameters of hemostasis system[34,36]. It becomes clear, that in the basis of hemostasiopathy development in case of examined new-born calves we have not only iron deficit but also found during investigation depression of plasma antioxidant defence which as previous works showed causes LPO activation in it. Increase of peroxidation in plasma damages structures of blood platelets and vessels and affects their functions[37,38]. Found in new-born calves with iron deficit thrombocytes' aggregation acceleration points at the increase of their receptors' sensibility to stimulating influences from the outside[39]. Besides, active development of thrombocytes' aggregation in response to rhystomicin in case of calves with iron deficit should be regarded as consequence of their sensibility increase to Willybrand's factor[10]. Besides,

acceleration of thrombocytes' aggregation coming of these animals indirectly tells about the increase in their blood platelets of exchange processes of arachidonic acid with surplus thromboxan A_2 formation[40].

Application of ferroglukin, polyson and cresacin combination made new-born calves with iron deficit state feel saturation of their organisms with iron, positive dynamics of red blood and common animals' state indices. Fulfilled impact on examined calves' organisms was accompanied by lowering of their LPO processes intensity in plasma what weakened its damaging influence on endothelium and liver thrombocytes. Found normalization of thrombocytes aggregation of calves with iron deficit state after getting of ferroglukin, polyson and cresacin combination is mostly the consequence of these means combination positive impact on innerthrombocyte LPO, receptor and postreceptor thrombocytes' functioning mechanisms[10]. Developing in these conditions time increase of thrombocytes aggregation coming in response to rhystomicin pointed at lowering in these calves' blood of adhesion cofactor – Willybrand's factor[20].

CONCLUSION

New-born calves having iron deficit are characterized by lowering of blood plasma antioxidant defence, intensification in it of LPO processes, increase of thrombocyte hemostatic activity. With the help of application to new-born calves with iron deficit of the combination of ferroglukin, polyson and cresacin we can really strengen plasma antioxidant defence, weaken LPO activity in it, normalize thrombocyte activity.

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