Correction of hemostasis physiological indices of newborn calves with iron deficit as a result of ferroglukin, polyson and cresacin application

ABSTRACT

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

Keywords: newborn calves; iron deficit; ferroglukin; polyson; cresacin.

1. INTRODUCTION

Nowadays cattle breeding is one of the main sources of wholesome and socially-significant foodstuff [1]. For its further intensive development are demanded serious diversified researches able to make scientific basis for subsequent increase of its effectiveness [2]. It becomes clear that in the phase of cattle, newborn state is a very important developmental stage for the whole subsequent ontogenesis [3]. 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 [4,5]. At the same time, at many Russian farms newborn calves are often found to have different abnormalities negatively influencing their metabolism processes and finally – their growth and development [6]. 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] but there was shown their minimization possibility with the help of traditional applications [20,21] what is really important for biological researches.

As the state of iron deficit is spread enough among newborn calves [6] and it is rather often accompanied by development of disturbances in hemostasis system [22] 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.

That’s why investigations led with newborn 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 newborn 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 ferrogulkin [23], and earlier shown their high biological activity and ability to influence hemostasis system separate components metabolically active means – polyson [24] and cresacin [25].

In this connection we put the following aim for our investigation –to find the evidence of hemostasis system activity correction of newborn calves with iron deficit with the help of ferrogulkin, polyson and cresacin combination.

2. MATERIALS AND METHODS

2.1 Materials

The work was fulfilled with 37 newborn 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 newborn calves.

All the investigations in the present work were conducted in full correspondence with ethical norms and recommendations on humanization of work with laboratory animals containing "The European Convent on the protection of vertebrate animals used for experiments or in other scientific purposes" (Strasbourg, 1986).
2.2 Methods

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 [26].

Thrombocytes’ number in calves’ blood was found out by their calculation in Gorjaev’s chamber. Thrombocytes aggregation was registered by visual micromethod [10] 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.).

Antiaggregation capabilities of a vascular wall were defined with the help of a probe with temporal venous occlusion on the basis of visual micromethod of thrombocyte aggregation registration [10] with all the applied inductors. We calculated the value of vascular wall antiaggregation activity index (VWAAI) while dividing thrombocytes aggregation period on the background of venous deadlock on the time of thrombocytes aggregation appearance without it. Examined calves were also calculated the index value of vascular wall anticoagulation activity by dividing of antithrombin III activity after venous occlusion on its value before it [27]. Vascular control over fibrinolytic blood activity was found out by calculating of index value of vascular wall fibrinolytic
activity while dividing the time of euglobulin lysis before occlusion on lysis time after it [27].

The state of plasma hemostasis was evaluated according to duration of activated partial thromboplastinic time (APTT), prothrombinic time and thrombinic time with the help of generally accepted methods [27]. The correction of iron deficit state of newborn calves was realized by ferroglukinin 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.

3. RESULTS AND DISCUSSION

Examined newborn 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_{239}/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 D233/1 ml, 3.46±0.012 mkmol/l and 33.7±0.15% correspondingly.

Thrombocytes’ quantity in newborn calves’ blood corresponded to norms.

Besides, 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).

Newborn calves with iron deficit were found to have VWAAI decrease in relation to all the applied inductors (table). The lowest VWAAI value belonged to collagen, a bit higher VWAAI value was for thrombin, still higher VWAAI value was with rhystomicin, ADP and adrenalin. Examined animals with iron deficit were noted to have decrease of vascular wall anticoagulant capabilities found according to the index value decrease of vascular wall anticoagulant activity. Fibrinolytic features of these animals’ vessels were also weakened – the index of vascular wall fibrinolytic activity turned to be decreased on 13.9%.

Newborn calves with iron deficit were also characterized by APTT increase (on 41.8%) and prothrombin time (on 42.6%) combining with some
152 intensification of thrombin time (on 6.8%).
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$_{233}$/m l, 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).

Examined calves in the result of realized application were noted to have evident VWAAI increase in relation to all the applied inducers (table). The minimum value was VWAAI value with thrombin. Other VWAAI values were a bit higher and had a tendency of approaching the control values. Newborn calves with iron deficit having received iron preparation in combination with metabolically active means were noted to have a tendency to the index increase of vascular wall anticoagulant activity and the index rise of vascular wall fibrinolitic activity on 9.0%.
Because of realization of complex application we reached APTT slowdown on 25.0% at simultaneous decrease of prothrombin time on 31.9% what, though, didn’t allow them to normalize. Besides, the value of thrombin time, defining the activity of fibrinogen transition into fibrin, of these calves increased on 3.7% and had a tendency to reach the control values.

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 [28]. Physiological peculiarities of their influence are mostly expressed by the optimum of living beings’ blood content [29] especially as far as hemostasis system components’ activity is concerned [30]. Besides, any disturbances in an organism are accompanied by negative dynamics of hematological indices [31,32] including parameters of hemostasis system [33,34]. It becomes clear, that in the basis of hemostasiopathy development in case of examined newborn 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 [35,36]. Found in newborn calves with iron deficit thrombocytes’ aggregation acceleration points at the increase of their receptors’ sensibility to stimulating influences from the outside. 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. 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 surplustromboxan A2 formation [37].

Weakening of vascular hemostasis functional capabilities of animals with iron deficit became apparent as lowering of vessels’ antiaggregation features what evidently was caused by generation decrease in their walls of prostacyclin and nitric oxide molecules [38]. At the same time examined calves were noted to have weakening of vessels’ anticoagulant and fibrinolytic capabilities because of production depression in them of anticoagulant – antithrombin III and tissue activators of plasminogen.

Found acceleration of prothrombin time of newborn calves with iron deficit pointed at their evident activation intensification of plasma hemostasis outer mechanism’s starting and had as its basis activity increase of coagulation factors participating in it [39]. Early APTT coming was connected with their activation of coagulation factors participating in the inner way of hemocoagulation. Acceleration of blood coagulation final stage pointed at the intensive change of fibrinogen into fibrin in case of examined calves [17].

Application of ferroglukin, polyson and cresacin combination made newborn 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 [38]. 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 [19].

In the result of applied impact animals having at the beginning iron deficit got strengthened antiaggregation, anticoagulant and fibrinolyticvessels’features. In the basis of found changes there was not reached control level intensification of production in vascular endothelium of these calves of prostacyclin, nitric oxide, antithrombin III and tissue activator plasminogen [29].

Found in examined animals on the background of applied impact slowdown of prothrombin time reflected positive dynamics of hemocoagulation processes along the outer way mainly on behalf of production lowering in liver of factors participating in it [1]. Noted on the background of correction slowdown of initially accelerated APTT told about weakening of generation activity of inner ways coagulation factors and especially factor XII. Found duration slowdown of hemocoagulation final stage which state was judged according to thrombin time
pointed at weakening of fibrinogen transformation into fibrin in examined calves.

In the result of fulfilled investigation it becomes clear that in case of ferroglukin, polyson and cresacin combination application to newborn calves with iron deficit we can provide normalization of thrombocyte activity and evident positive dynamics of the rest hemostasis system components.

4. CONCLUSION

Newborn calves having iron deficit are characterized by lowering of blood plasma antioxidant defence, intensification in it of LPO processes, increase of thrombocyte hemostatic activity and hemocoagulation at depression of vascular wall capabilities to slowdown these processes. With the help of application to newborn calves with iron deficit of the combination of ferroglukin, polyson and cresacin we can really strengthen plasma antioxidant defence, weaken LPO activity in it, normalize thrombocyte activity and produce positive dynamics of hemostatic features of vascular wall and plasma hemostasis.

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Table. Parameters of hemostasis in newborn calves with iron deficiency treated with ferroglukin, polypson and cresacin

<table>
<thead>
<tr>
<th>Consider indicators</th>
<th>Calves with iron deficiency, n=37, M±m</th>
<th>Control, n=29, M±m</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>outcome after the correction</td>
<td></td>
</tr>
<tr>
<td>Platelet aggregation with ADP, s</td>
<td>26,0±0,16</td>
<td>40,1±0,12</td>
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<tr>
<td>Platelet aggregation with collagen, s</td>
<td>19,2±0,21</td>
<td>31,3±0,08</td>
</tr>
<tr>
<td>Platelet aggregation with thrombin, s</td>
<td>36,5±0,12</td>
<td>54,2±0,20</td>
</tr>
<tr>
<td>Platelet aggregation with rystomicin, s</td>
<td>21,0±0,19</td>
<td>48,1±0,14</td>
</tr>
<tr>
<td>Platelet aggregation with adrenalin, s</td>
<td>67,9±0,23</td>
<td>97,4±0,16</td>
</tr>
<tr>
<td>VWAAI with ADP</td>
<td>1,44±0,002</td>
<td>1,58±0,004</td>
</tr>
<tr>
<td>VWAAI with collagen</td>
<td>1,33±0,010</td>
<td>1,47±0,004</td>
</tr>
<tr>
<td>VWAAI with thrombin</td>
<td>1,37±0,007</td>
<td>1,45±0,005</td>
</tr>
<tr>
<td>VWAAI with rystomicin</td>
<td>1,43±0,010</td>
<td>1,49±0,002</td>
</tr>
<tr>
<td>VWAAI with adrenalin</td>
<td>1,44±0,008</td>
<td>1,55±0,006</td>
</tr>
<tr>
<td>Index value of vascular wall anticoagulation activity</td>
<td>1,24±0,006</td>
<td>1,28±0,003</td>
</tr>
<tr>
<td>Index Value of Vascular Wall</td>
<td>Fibrinolytic Activity</td>
<td>p&lt;0.05</td>
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<td>------------------------------</td>
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<tr>
<td>1.22±0.005</td>
<td>1.33±0.004</td>
<td>1.39±0.010</td>
</tr>
<tr>
<td>Activated Partial Thromboplastic Time, s</td>
<td>28.0±0.23</td>
<td>35.0±0.11</td>
</tr>
<tr>
<td>Prothrombinic Time, s</td>
<td>12.2±0.26</td>
<td>16.1±0.15</td>
</tr>
<tr>
<td>Thrombinic Time, s</td>
<td>16.1±0.19</td>
<td>16.7±0.14</td>
</tr>
</tbody>
</table>

Legend: p - reliability of differences of indicators between the control and the initial state of the calves with iron deficiency, p1 - reliability of dynamics of indicators in calves with iron deficiency against the background of correction.