© Ivanov DO., Shabalov NP., Petrenko YV., Shabalova NN.,Treskina N.A.

Ivanov DO[1]., Shabalov NP[2]., Petrenko YV[1]., Shabalova NN[2].,Treskina N.A[1].

THE SPECIFIC CHARACTERISTICS OF DIC-SYNDROME VARY WITH DIFFERENT CLINICAL SETTINGS IN THE NEWBORN

[1]*Federal Center of blood, heart and endocrinology by V.A. Almazov, St-Petersburg, Russia*
[2]*Military Medical Academy, Department of pediatrics, St-Petersburg, Russia;*

Резюме

СПЕЦИАЛЬНЫЕ ХАРАКТЕРИСТИКИ ДВС СИНДРОМА В ЗАВИСИМОСТИ ОТ КЛИНИЧЕСКИХ ПРОЯВЛЕНИЙ У НОВОРОЖДЕННЫХ

Мы обследовали 6 групп детей (всего 214 детей) с тяжелой патологией неонатального периода (137 доношенных и 80 недоношенных новорожденных, рожденных на сроке 32-36 недель). В контрольную группу включены здоровые доношенные новорожденные в возрасте 5-6 дней.

Группа 1 - здоровые доношенные новорожденные в возрасте 5-6 дней — 49 детей; группа 2 — новорожденные с тяжелой асфиксией в родах — 40 новорожденных; группа 3 - новорожденные с тяжелым постгипоксическим синдромом, осложненным пневмонией — 32 ребенка; группа 4 — новорожденные после абдоминальных оперативных вмешательств, связанных с мальформациями ЖКТ — 35; группа 5 — новорожденные с сепсисом (А- гипоэргический вариант - 33, Б — гиперэргический вариант — 35); гр. 6 - новорожденные с гемолитической болезнью (ГБН) -39 человек.

Анализ гемостаза проводился на основании оценки уровня 10 прокоагулянтов в плазме крови (I, II, V, VII, VIII, IX, X, XI, XII, XIII факторы); коагуляционных тестов: АЧТВ, протромбиновое время, тромбиновое время, плазменные концентрации 5 ингибиторов сывороточных протеиназ: антитромбина-III (АТ-III), α1-антитрипсина, протеина С, α2-макроглобулина, С1- ингибитора, содержания в плазме фибронектина, ф-ра Виллебранта, высокомолекулярного кининогена, плазминогена и ПДФ. Использовались реагенты компании Behring (Германия). Динамика агрегации тромбоцитов на адреналин и АДП оценивалась при помощи агрегометра THROMLITE 1006.

Статистическая обработка проводилась с использованием критерия Стьюдента, непараметрических методов Фишера и Колмогорова.

Introduction. Disseminated intravascular coagulation (DIC) – is one of the causes of high mortality in severe neonatal diseases of different etiologies. Etiology and trigger mechanisms of neonatal DIC-syndrome are well known: severe hypoxia, trauma, shock of any etiology, generalized infection, and surgical intervention. However, the specific contribution each of these factors in the pathogenesis, peculiarities of development and hemostasiological characteristics of DIC-syndrome in various forms of neonatal pathology are not studied. The role of initial reactivity, aseptic surgical trauma, type of agent (in the case of septic DIC) in hemostasiological and clinical characteristics of the syndrome are not sufficiently studied [1-4].

Our observations of newborns for years [5 - 7] at the neonatal intensive care unite (NICU) had confirmed the view that DIC is more common than diagnosed. Also there are differences in the frequency and nature of the clinical manifestation of thrombohemorrhagic syndrome in DIC of various geneses [8,9]. It was the basis for the study of hemostasiological features of DIC-syndrome of various etiologies. We have previously identified two different patterns of DIC-syndrome in sepsis, "decompensated" and "overcompensated" [8].

The aim of this work was to study the clinical and hemostasiological characteristics of DIC-syndrome with severe posthypoxic syndrome, complicated by pneumonia, sepsis and extensive abdominal surgery, associated with malformations of the gastrointestinal tract.

Materials and methods. We examined six groups of children (214 infants in total) with severe neonatal pathology (137 term and 80 preterm newborns with gestational age 32 - 36 weeks). The control group was healthy term infants 5 - 6th day of life.

There were 5 groups of newborns. Group I - healthy full-term newborns 5-6th day of life - 49 infants; II group – newborns with heavy intrapartum asphyxia - 40 infants; III group- infants with severe posthypoxic syndrome complicated by pneumonia - 32 infants; IV-group -newborns after abdominal operations in connection with malformations of the gastrointestinal tract - 35 infants; V-group -neonates with sepsis ("A" - hypoergic variant - 33 infants and "B" - hyperergic variant -35 infants); VI group - neonates with hemolytic disease of the newborn (HDN) - 39 infants.

Analysis of hemostasis was performed on the basis of the level of 10 procoagulants in the blood plasma (I, II, V, VII, VIII, IX, X, XI, XII, XIII factors); coagulation tests: activated partial thromboplastin time (APTT), prothrombin time (PT), thrombin time (TT), plasma concentrations of 5-inhibitors of serine proteinase: antithrombin-III (AT-III), a1-antitrypsin (a1-AT), protein C (Prot. C), a2 macroglobulin (a2-MG), C1-inhibitor (C1-IN), levels in plasma of fibronectin (FN), von Willebrand factor (vWF), a high-molecular weight kininogen (CMC); plasminogen (PG) and fibrin degradation products (FDP). All studies were performed using hemostatic agents from Behring production (Germany). The dynamics of platelet aggregation to adrenaline and ADP was assessed using aggregometer (THROMLITE 1006).

Statistical analysis was performed using Student's t-test, nonparametric methods of Fisher and Kolmogorov.

Results. The diagnosis of DIC-syndrome was set in 128 out of 214 examined newborns. Clinical criteria for diagnosis of DIC-syndrome was the severity of the initial disease of the newborn at risk (ie, situational assessment), accompanied by multiple organ dysfunction: the combination of different disorders of the central nervous system, lungs, kidneys, liver, systemic hemodynamic and often by thrombosis and hemorrhage. Hemostasiological criteria of DIC-syndrome were: hyper-and hypocoagulation shifts of APTT, PT, consumption of procoagulants, antithrombin III, high molecular weight kininogen, plasminogen, FDP increasing, thrombocytopenia with failure of the aggregation platelet activity.

The frequency of DIC-syndrome varied in different forms of severe perinatal pathology. DIC-syndrome ever happened in sepsis. Hemostasiologically DIC-syndrome was revealed in all children after surgical intervention for atresia of any part of gastrointestinal tract with prolonged anesthesia. DIC-syndrome was seen in one third of children with severe intrapartum asphyxia and posthypoxic syndrome. The frequency of hemorrhagic disorder manifestations in DIC-syndrome depended on its origin.

Hemostasiological characteristics of DIC-syndrome in newborns with severe asphyxia and hypoxia in the early neonatal period (group II) are presented in Table 1: the presence of consumption coagulopathy that appeared in increase of the APTT, TT, PT, reduction of factor IX, AT-III, PG, CMC, fibrinogen (FG), thrombocytopenia, increase of FDP that are the signs of activation of coagulation and fibrinolysis.

The main features of the DIC-syndrome in posthypoxic syndrome were: significantly increased aggregation activity of platelets to adrenaline, which possibly was a compensative mechanism in the hemostatic system, and an exceptionally high level of vWF - a marker of blood vessels damaging. Clinical improvement was accompanied by a shift in the coagulation toward the normalization of coagulation parameters. VWF decreased in 3.6 times. Simultaneously there was a significant increase of fibrinogen (FG) concentration, VIII and X factors, CMC, a1-AT and inhibition of fibrinolysis. The functional activity of platelets to adrenaline decreased, still remaining above normal.

Despite significant changes in hemostasiogram, the effectiveness of treatment allowed all children to survive. However, there was a high percentage of moderate or severe posthypoxic encephalopathy in these children during the first month of life (85%).

DIC-syndrome in newborns of group III manifested by bleeding disorders in 7 out of 9 children and 2 of them had intraventricular hemorrhage (IVH). Analysis of hemostasis in children of group III with complicated posthypoxic syndrome showed a wave-like nature of coagulation with the presence of sharp changes in direction of hypo-and hypercoagulation (in group II - without pneumonia, DIC had only hypocoagulation changes), but its degree of severity in group II was less than in group III. Platelet aggregation of group III in whole was characterized by hyperaggregation, which gave some similarities with group II. In the hypocoagulation phase all tests were significantly elongated and PG level was reduced. Positive correlations between the PG and XIII factor ($r = 0,78$), as well as PG - FG and PG - FN ($r = 0,80$ and 0.72, respectively) were noted in children of group III. There was relatively high level of procoagulants in the phase of hypercoagulation (on the bases of APTT and PT). The level of FDP was high during the flame period. Health improvement was accompanied by significant increase of FG, VIIIf, CMC and α1-AT (same as in group II) compared with the normal concentrations.

The parameters of hemostasis in children operated in the neonatal period in connection with developmental disabilities (without infectional complications) are shown in Table 1 (Group IV). Analysis of hemostasis revealed DIC with coagulopathy of consumption (with high level of VIIIf), thrombocytopenia of consumption, lack of FN, AT-III and a high content of the FDP. Our observation shows that surgery, even in children with reference indicators lead to the activation of both coagulation and platelet hemostasis. Intraoperative complications leading to the development of DIC were not registered. APTT was increased 5-fold as compared with the control group and PT – 2-fold. The main feature of homeostasis in this group was significantly increased platelet aggregation, especially pronounced at the height of hypocoagulation, which allowed us to be regarded as a mechanism of adaptation the hyperaggregation in the hemostatic system.

The peculiarity of postoperative DIC was brief hypercoagulation phase that occurred even at the operating table. Hypocoagulation crisis was followed by

Table 1.

The parameters of hemostasis in neonates with severe posthypoxic syndrome, complicated and uncomplicated by pneumonia

Group		I Healthy newborns n = 49	II Heavy posthypoxic syndrome with DIC n = 13		III Posthypoxic syndrome complicated by pneumonia and DIC n = 9		
Parameters		5-6 days	Stage of height	Stage of improvment	Phase of hyper-coagulation	Phase of hy-pocoagulation	Phase of im-provement.
APPT (sec)		51,,6 ±± 0,,2	125,4±7,2*	55,9±4,6	39,9±1,2*	173,2±5,6*	59,9±3,2
PT(sec)		16,,8 ±± 2,,5	25,8±2,1*	14,4±1,6	15,6±1,4*	35,8±2,9*	14,4±2,1
TT (sec).		17,2 ±± 1,2	21,3±2,0*	19,9±1,8	19,2±2,2*	27,9±3,1*	19,8±2,0
FG (g/l)		2,0 ±± 0,05	1,2±0,2*	3,85±0,8*	2,1±0,4	1,3±0,3*	3,3±0,6*
VII f (%)		67,6± 1,3	74,3±3,1*	86,5±6,2*	78,3±2,8	71,6±2,5	84,2±3,1
VIII f (%)		81,0 ± 1,1	107,1±3,5*	132,3±4,9*	98,2±4,5*	99,8±5,7*	127,2±7,5
IX f (%)		85,4 ± 1,6	71,3±2,5*	85,6±5,3	78,8±2,2	71,9±2,1	103,8±4,3
X f (%)		60,3 ±± 2,2	73,2±4,6*	94,3±4,3*	89,9±2,6*	82,9±2,3*	89,3±3,5*
vWF (%)		121,8± 15,2	616,2±11,3*	168,2±28,6*	229,6±13,7*	241,6±12,1*	231,1±11,2*
PG (mkg/l)		45,0 ± 3,0	39,1±3,7	71,3±4,4*	42,6±4,3	37,0±2,1*	63,8±3,8*
FDP (mkg/l)		5,2± 0,6	14,0±2,7*	7,0±1,0	23,5±2,1*	22,1±2,4*	7,4±2,1
FN (g/l)		0,16 ±± 0,01	0,2±0,045	0,21±0,04*	0,08±0,01*	0,1±0,01*	0,15±0,01
AT-III (g/l)		0,17± 0,02	0,08±0,03*	0,18±0,02	0,1±0,01*	0,12±0,01*	0,2±0,03
α1-AT (g/l)		1,8 ±± 0,02	2,05±0,21	3,7±0,19*	2,0±0,13	2,59±0,24*	2,43±0,21*
CMC (%)		100,8 ±±1,1	62,6±3,6*	89,4±3,7	73,5±2,1*	61,2±4,5*	94,2±3,5
Platelets (thousands $\times 10^9$)		245,0 ±± 30,0	98,3±12,1*	233,3±23,5	94,1±14,5*	101,6*±8,9*	198,9±16,1
Aggregation to adrenaline (%)							
Min.	2	12,7 ±2,5	32,3±2,1*	11,2±2,3	19,9±1,9*	19,9±1,9*	27,9±2,3*
	5	20,9 ± 3,4	40,1±2,2*	13,9±2,9*	27,9±3,7*	27,9±3,7*	21,0±1,2
	8	25,2 ± 3,3	40,5±2,6*	22,0±1,6	29,2±4,1	29,2±4,1	23,8±1,9
	10	28,7 ± 3,2	45,5±2,5*	23,2±2,5	31,8±2,5	31,8±2,5	23,8±2,8

* statistically significant differences compared with healthy newborns by the Student t-test ($p < 0.05$)

a significant increase in VIIIf, FN, PG, and growth in concentration of anticoagulants, presenting current aseptic inflammation. Increasing of vWF was minimal, and the platelet count in the lower limit was persistent.

All newborns with neonatal sepsis had clinical and hemostasiological characteristics of DIC-syndrome but they were different in hypoergic and hyperergic variants (Table 2).

In 92.9% of newborns with hypoergic variant of sepsis (group V variant "A") there were no hypercoagulation crises in the height of DIC i.e. shortening of the APTT and PT. The data presented by Table 2 indicate hypocoagulation in hemostasis. The duration of this period ranged from 5 to 21 days (for dead children during the whole period of follow-up). There were low levels of the majority of procoagulants: AT-III, a2-MG, PG and CMC in hypoergic variant "A", which were used during coagulation and fibrinolysis with inadequate synthesis and thus significantly differed from hyperergic variant "B". Low level of VIIIf was the main marker of "A" variant which for all other investigated variants of DIC was high, including sepsis "B". Platelets aggregation was characterized by low activity to adrenaline in sepsis "A".

In 100% of patients with sepsis "A" there were increased level of α1-AT, C1-inhibitor (2-fold higher than in variant "B") vWF, that reflects, in our view, a higher level of proteolytic activity of plasma, endothelial damage and increased permeability of the vascular wall in this variant of septic DIC.

In hyperergic variant (group V-"B" variant) hypercoagulative crises occurred in 100% of patients while their duration ranged from 5 to 30 days, i.e. there was a general hypercoagulation in hemostasis. However, this group of infants later experienced hypocoagulable states, in the majority of newborns (85.6 %) it was only once and usually coincided with the emergence of new foci of infection. At the same time, there was reduced aggregation activity of platelets to adrenaline. The difference in values of APTT and PT as compared to the norm, respectively, was 144,8 seconds and 27,5 seconds which is 2 times lower than in variant "A". Aggregation activity of platelets to adrenaline was

Table 2

Parameters of hemostasis in children operated in the neonatal period in connection with developmental malformations with two variants of sepsis (hypoergic variant "A" and hyperergic variant "B").

Parameters		IV Operated		V Sepsis		
		Crisis of hypocoagulation (n = 35)	The period of stabilization (n = 26)	Group "A" (the height of the infection process) (n =33)	Group "B" (the height of the infection process) (n =35)	Improvement (n =30)
APPT (sec)		264,9±20,7*	42,2±5,3	281,0±54,0*	29,4±6,4*	44,2±3,4
PT(sec)		33,5± 2,7*	15,9±0,9	28,4±3,3	15,2±0,7*	13,0±0,3
TT (sec).		26,5± 2,1*	19,1±0,4	24,4±1,5*	19,4±1,3*	19,9±0,8
FG (g/l)		1,2±0,2*	3,1±0,2*	1,0±0,2*	3,0±0,2*	2,8±0,3*
Vf (%).		-	-	53,3±4,8*	88,3±6,4*	89,6±6,4
VII f (%).		64,5±3,3	75,3±4,0*	59,5±5,4*	87,2±2,6*	76,2±3,7
VIIIf (%).		102,0±8,3*	110,0±15,5*	63,0±5,5*	137,1±15,1*	116,5±15,1*
IXf (%).		69,2±2.9*	77,4±5,0	62,7±2,5*	100,3±6,5*	80,2±5,4
X f (%).		83,3±4,5*	66,9±2,5	59,0±5,4	94,1±3,5*	85,0±3,9*
vWF (%)		241,0±21,8*	186,0±27,2	309,0±27,5*	336,1±27,2	84,0±9,0
PG (mkg/l)		34,3±1,8*	62,7±8,7	32,0±5,5*	67,9±4,8*	69,8±10,3
FDP (mkg/l)		39,7±10,2*	10,2±4,0*	31,0±5,8*	5,7±1,1	11,6±2,9*
FN (g/l)		0,13±0,01*	0,29±0,0*4	0,12±0,01*	0,26±0,02*	0,29±0,01*
AT-III (g/l)		0,09±0,01*	0,30±0,02*	0,08±0,01*	0,24±0,01	0,27±0,01*
α1-AT (g/l)		3,3 ±0,3*	2,3 ±0,3*	2,94±0,19*	2,3±0,26*	2,40±0,29
α2-MG (g/l)		-	-	1,50±0,17	2,32±0,29	3,4±0,06*
C1-IN (g/l)		-	-	0,62±0,12*	0,31±0,09*	0,28±0,09
CMC (%)		-	-	53,0±4,0*	65,1±7,4*	94,8±7,1
Platelets (thousands $\times 10^9$)		135,0±11,5*	254,3±12,6	79,0±5,7*	120,0±8,2*	340,0±31,1
Aggregation to adrenaline (%)						
Min.	2	31,7±5,0*	16,8±4,2	4,15±1,5*	6,6±0,9*	14,6±2,6
	5	47,7±11,4*	22,2±5,1	6,8±2,1*	13,9±1,2*	24,6±4,6
	8	59,2±8,2*	25,5±5,3	11,4±2,4*	18,6±1,7	29,0±4,8
	10	47,8±14,0*	27,8±5,3	9,9±1,7*	22,3±2,3	31,0±5,7

* statistically significant differences compared with healthy newborns by the Student t-test ($p < 0.05$)

lower than normal but higher than in variant "A". Attention is drawn to the high (even compared to healthy) content of FG, VIIIf and anticoagulants in spite of the current active DIC. The peculiarity of hemostasis in hyperergic variant of the sepsis was the high content of PG regarded as the inhibition of fibrinolysis.

As in both septic groups there were newborns after abdominal operations due to developmental disabilities, we analyzed coagulation tests in these subgroups separately. On the basis of our observations of postoperative DIC (group 4) we concluded that surgical intervention with sepsis was accompanied by elongation of APTT in group "B" average at 24% and PT - 70%.

As for the group "A», there was also increased clotting time from the baseline in the early postoperative period (in some cases - 3 – 3,5 times). But the overall shift of hemostasis to hypocoagulation had led to impossibility to assess the degree of shear caused by the operation itself.

DIC with hemolytic disease of newborn (HDN) (VI group) always manifested by hemorrhagic syndrome with symptoms of disorders in all components of hemostasis. The main feature of hemostasiogram was reduced platelet aggregation for all types of used agregants (ADP, ristomitsin, adrenaline).

Discussion. Increased sensitivity of newborns to all factors initiating DIC is associated with physiological characteristics of the hemostatic system in newborns, particularly, by low mononuclear phagocytes ability to clean the blood from clotting products, immaturity of the liver and inability to provide adequate compensatory synthesis of pro-and anticoagulants [1,2]. On the other hand, the interpretation of hemostatic test in newborn is difficult due to their lability in the first days of life. Studying specific characteristics of homeostasis in healthy infants during the 1st week of life, we concluded that high lability of coagulation parameters and platelet hemostasis with relatively

stable X key factor of proteolytic cascade and the absence of clinical thrombosis and hemorrhages, reflects the processes of adaptation to the transition states of hemodynamics, respiratory tract and hematopoiesis [5]. However, the same circumstance predisposes to an increased tendency of newborns to thrombosis and hemorrhagic disorders.

A detailed analysis of the significant adverse factors of ante-and intrapartum periods of life in the development of fetal hypoxia, malnutrition, malformations and sepsis are described previously. The study of the mechanism of DIC development in these conditions and recognition of differing characteristics in various clinical settings may allow optimization of therapy and better assessment of prognosis.

Figure 1 shows the major factors of DIC development in various forms of neonatal disease. The data of histogram shows that in groups II and III DIC was induced by the combination of heavy asphyxia and traumatic factors in childbirth. Trigger mechanisms of DIC with severe intrapartum hypoxia (II and III groups) were severe posthypoxic syndrome with respiratory insufficiency, hemodynamic disorders, acidosis, and hyperbilirubinemia. Peculiarities of hemostasiogram in children of these groups are defined by damage of the vascular wall and membranes of blood cells due to severe acidosis as evidenced by 5.5 times higher concentration of vWF and hemodynamic disturbances. Certainly, «absence» of hypercoagulative phase (apparently that took place during delivery) points to a low ability of liver to synthesize proteins in these children which is easily explained by maternal preeclampsia and presence of chronic fetal hypoxia in one third of children. Improving of the clinical status was accompanied by an increase in the content of FG, VIIIf, CMC, PG and α1-antitrypsin that was significantly higher than in healthy newborns. These acute-phase polyfunctional proteins show the recovery process after severe asphyxia and high content of Xf show maintaining of tension in the system of hemostasis.

Newborns in group III had a number of similarities in the height stages with group II in platelet hemostasis and similarities with the group V in the coagulation link of hemostasis (in the phase of hypooagulation with a version «A» and in hypercoagulation with the version «B»). This circumstance confirms different contributions of hypoxic and infectious factors in the character of DIC. Infectious factor gives the clinical picture and hemostatic picture wave-like character with deviations to hypo-and/or hypercoagulation.

The reason for the development of DIC in operated newborns is the operation itself. Hemodynamic disturbances, anemia, respiratory failure and acidosis play an important role in the early postoperative period. Attention is drawn to the fact that hypo-coagulation crisis is detected in all children who underwent abdominal surgery that is observed in the first 4 days after the intervention, and in the absence of additional aggravating factors (such as repeated surgery or infection) is not repeated. This short duration of the crisis and the ability to self-compensation in the hemostatic system indicates the presence of plastic and energy reserves as a result of less severe antenatal suffering and infectious components (ie, there was aseptic SIRS). That was the main feature of post-operative DIC in comparison with DIC of other origin (in compared with groups II, III, V-A variant) in which there was a wave-like nature of most of the parameters of hemostasis in the dynamics and hypocoagulation periods.

Sepsis is the most common cause of DIC in the neonatal period, but as shown by our data [10] pattern of "hypoergic" variant of DIC («A») and "hyperergic" variant («B») differs and we associate it with a type of pathogen, peculiarities of the initial reactivity reflecting the degree of antenatal suffering. The predominance of gram-negative bacteria as the causative agents of sepsis in the group «A» requires a higher level of endotoxemia. Endotoxin leading to a reduction of marginal pool of leukocytes, exposure of vascular endothelialium, activation of endothelial cells triggers a cascade of proteolytic systems of plasma (thrombin, kinin, fibrinolitic and the complement system) [11-14]. The high content of α1-AT providing 88% of contrprotease activity of plasma and C1-IN, suppressing contact activation of thrombin and callikrein-kinin systems is regarded by us as a compensative mechanism. However, in variant «A» it is not sufficient, probably due to lack of contr mediators of eosinophil (eosinopenia is typical or complete absence of eosinophils). This variant is also characterized by lymphopenia and a tendency to monocytopenia due to lack of mediators produced by these cells.

Concluding the discussion of the DIC syndrome characteristics in different clinical settings we would like to say that the overall hemostatic parameters determined on the basis of coagulation tests (venous blood parameters) not always reflect hemostatic situation in a certain organ / region. The local cellular factors (macrophages, endothelial cells, etc) also affect the general hemostatic situation resulting in thrombosis of kidney, brain vessels in general hypocoagulation.

References

1. Pugh M. DIC screening in the newborn. Neonatal Netw. 1997 Oct;16(7):57-60.

2. Veldman A, Fischer D, Nold MF, Wong FY. Disseminated intravascular coagulation in term and preterm neonates. Semin Thromb Hemost. 2010; 36(4):419-28.

3. Wynn J, Cornell TT, Wong HR et al. The host response to sepsis and developmental impact. Pediatrics. 2010 May;125(5):1031-41.

4. Grover SB, Mahato S, Chellani H. et al. Disseminated intravascular coagulation with intracranial haematoma in neonatal congenital syphilis. J Trop Pediatr. 2011 Aug;57(4):315-8.

5. Ivanov DO, Shabalov NP, Shabalova NN. Hemostasis in the dynamics of the first week of life as a reflection of the mechanisms of adaptation to extrauterine life of newborn. Pediatriia. 2000, 3 : 22 - 32.

6. Shabalov NP, Diukov EV, Veber IN, Chumakova GN. Features of the thrombocytic component of hemostasis in newborns and mechanisms of its disorders. Gematol Transfuziol. 1991 May;36(5):10-4.

7. Sergeeva VA, Nesterenko SN, Shabalov NP. Fetal inflammatory response in the development of multiple organ dysfunction in newborn. Anesteziol Reanimatol. 2010 Jan-Feb;(1):30-4

8. Stanworth SJ, Bennett C. How to tackle bleeding and thrombosis in the newborn. Early Hum Dev. 2008 Aug;84(8):507-13.

9. Williams MD, Chalmers EA, Gibson BE; Haemostasis and Thrombosis Task Force, British Committee for Standards in Haematology. The investigation and management of neonatal haemostasis and thrombosis. Br J Haematol. 2002 Nov;119(2):295-309.

10. Ivanov DO, Shabalov NP, Shabalova NN. Sepsis in the newborn. . Pediatriia. 2003, 5 : 46 - 56.

11. Antonelli M. Sepsis and septic shock: pro-inflammatory or anti-inflammatory state? J. Chemother 1999 Dec; Vol. 11(6) :536-540.

12. Boldt J, Papsdorf M, Rothe A et al Changes of the hemostatic network in critically ill patients--is there a difference between sepsis, trauma, and neurosurgery patients? Crit Care Med., 2000 Feb; 28 (2): 445-50.

13. Xu J, Lupu F, Esmon CT. Inflammation, innate immunity and blood coagulation. Hamostaseologie. 2010 Jan;30(1):5-6, 8-9.

14. Suzuki S., Morishita S. Hypercoagulability and DIC in high-risk infants. Semin. Thromb. Hemost. 1998; Vol. 24(5), P. 463-476.

Receptionat 18.09.2013

© NY Smedyk, VV Ryazanov, GE Trufanov, IA Vikhtinskaya, DO Ivanov, VV Ipatov

NY Smedyk[1], VV Ryazanov[1,2], GE Trufanov[1,2], IA Vikhtinskaya[1,2], DO Ivanov[2], VV Ipatov[1]

MAGNETIC RESONANCE PELVIMETRY IN SUBCLINICAL FORMS OF NARROW PELVIS AND SHOULDER DYSTOCIA RISK ASSESSMENT

[1]Military Medical Academy, Department of radiology, St-Petersburg, Russia;
[2]Federal Center of blood, heart and endocrinology by V.A. Almazov, St-Petersburg, Russia

Summary

ПЕЛЬВИОМЕТРИЯ С ПОМОЩЬЮ ПРИМИНЕНИЯ ЯДЕРНО-МАГНИТНОГО РЕЗОНАНСА ПРИ СУБКЛИНИЧЕСКИХ ФОРМАХ УЗКОГО ТАЗА И ОПРЕДЕЛЕНИЯ РИСКА В СЛУЧАЕ ДИСТОЦИИ ПРЕЧИКОВ

Во многих странах точность измерений размеров наружного таза сомнительна. Распространенность подтипов субклинически узкого костного таза еще более затрудняет диагностику.

Цель: *установить акушерские МР пельвиометрические и фетометрические референсные значения при оценке риска дистоции плечиков плода перед родами и сравнить МР пельвиометрию с наружными методами измерения.*

Методы: *обследованы 40 женщин с одноплодной беремонностью в сроке 38-39 недель. МР пельвиометрия производилась на высокопольном 1,5 T MRT. Все субклинические типы узкого костного таза были разделены на 3 степени. Проводилась также фетометрия. Риск дистоции определялся как минимальный, возможный и высокий.*

Результаты: *Абсолютно узкий таз был выявлен у 3 женщин. Диаметр костного таза, измеренный на МРТ, был нормальным у 14 (35%) женщин. Двенадцати женщинам (30%) со 2 степенью узкого таза и/или крупным плодом (>4000 г) при отсутствии других противопоказаний было рекомендовано родоразрешение через естественные родовые пути, но 6 из них выполнено КС.*

Заключение: *низкая частота выявления субклинически узкого костного таза при наружных измерениях может приводить к запоздалой интранатальной диагностики этой патологии. МР пельвиометрия выявляла субклиническое сужение костного таза. Дополнительные данные фетометрии помогли оценить риск дистоции плечиков перед родами.*

Introduction. In many countries accuracy of external pelvis measurements is considered to be questionable because of the high mistake value (up to 1.5 - 5 cm). Subclinical narrow bone pelvis subtypes predominance make the diagnostics even more complicated. Assessment of pelvical cavity form and detection of all pelvical distances, distances of fetus head and chest-with-shoulders circumference followed with conclusion about their relevarion or discrepancy can be provided by complex data of rentrenopelvime-