Original article

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VALUE OF HAEMATOLOGICAL AND SERUM BIOCHEMICAL PARAMETERS IN THE PREDICTION OF PERINATAL OUTCOME IN PREECLAMPSIA

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Preeclampsia is a serious disorder characterized by a generalized maternal inflammatory response associated with diffuse endothelial cell dysfunction. Preeclampsia has a long preclinical phase before it manifests. The possibility of predicting complications in preeclampsia is clinically very significant, as it could contribute to the reduction of maternal and neonatal morbidity and mortality. The aim of this study was to examine whether haematological and serum biochemical parameters may be of use in predicting more severe clinical picture and worse perinatal outcome in preeclampsia.

The prospective observational study included the study group consisted of 30 singleton pregnancies with preeclampsia completed by caesarean section (CS). This study group was divided into two subgroups with respect to severity of preeclampsia (mild and severe). The control group consisted of 20 healthy pregnant women delivered by elective CS. Clinical characteristics of pregnant women, haematological and serum biochemical parameters, as well as perinatal outcome were analyzed. In preeclampsia, the higher values of hematocrit and hemoglobin are noted, and lower platelet count, as well as the higher values of aspartate aminotransferase (AST), alanine aminotransferase, lactate dehydrogenase (LDH), gamma-glutamyl transferase, cholesterol, triglycerides, uric acid, urea and creatinine. Laboratory parameters associated with a severe clinical picture of preeclampsia in our study, as well as with a worse perinatal outcome were thrombocytopenia and increased AST and LDH levels. However, despite being indicators of a poorer outcome, they cannot be used with absolute certainty and in isolation from other indicators to predict poor perinatal outcome in preeclampsia. Deciding the delivery time in relation to an expectative approach should be based on a comprehensive consideration of gestational age, fetal condition, clinical and laboratory maternal indicators.

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Key words: *biochemical parameters, haematological parameters, perinatal outcome, preeclampsia*

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Introduction

Hypertensive disorders in pregnancy occur in 2-10% of pregnancies worldwide (1). Preeclampsia is a serious disorder, unique in human pregnancy, characterized by a generalized systemic maternal inflammatory response associated with diffuse endothelial cell dysfunction. It is a complex disease in which numerous genetic, immunological and environmental factors interact. It is characterized by widespread systemic vascular endothelial dysfunction and microangiopathy in mothers, but not fetuses. The fetus does not develop any clinical manifestations similar to maternal syndrome, and fetal morbidity and mortality are solely the result of placental insufficiency. Preeclampsia also leads to an increased risk of neonatal morbidity and mortality, especially in relation to neonatal prematurity (2).

The prevailing view is that preeclampsia has its cause in abnormal placentation, which in turn leads to widespread maternal endothelial effects and clinical manifestation of the disease (3). Unusual amounts of placental debris, syncytiotrophoblastic microparticles, products of oxidative damage, pro-inflammatory cytokines and angiogenic factors are assumed to be released into the intervillous space, and then interact with the maternal endothelium and immune cells, causing maternal symptoms (4).

Significant changes in the structure and function of the maternal vascular endothelium lead to altered vascular reactivity, activation of the coagulation cascade, and multisystem damage that occurs in preeclampsia. One of the pathological changes is also in the endothelial cells of the renal glomerular capillaries (glomerular endotheliosis), which results in some clinical manifestations.

Preeclampsia is known to have a long preclinical phase before symptoms and signs of disease become apparent in the second half of pregnancy. The severity of clinical picture in preeclampsia can range from milder to extremely severe forms, and life-threatening pregnancies. The main threats to a fetus are intrauterine growth retardation (IUGR), fetal distress, and even perinatal death of the fetus. On the other hand, while premature termination of pregnancy is always good for the safety of the mother, it can further compromise the fetus due to prematurity. Therefore, it is of utmost importance to adequately assess when the harm of an expectative approach may be greater than the risk of premature termination of pregnancy.

The possibility of predicting complications in preeclampsia is clinically very significant, as it could contribute to the reduction of maternal and neonatal morbidity and mortality.

The aim

The aim of this study was to examine whether haematological and serum biochemical parameters may be of use in predicting more severe clinical picture and worse perinatal outcome in preeclampsia.

Patients and methods

A prospective case-control observational study was conducted at the Clinic of Obstetrics and Gynecology, Clinical Center Niš. The study group consisted of 30 pregnant women whose pregnancies were complicated by preeclampsia, terminated by cesarean section, spontaneously conceived, singletons, with no fetal anomalies and preexisting clinical disorders, as well as without complicating actual pregnancy by diabetes and chorioamnionitis. The criteria for diagnosis of preeclampsia were new-onset arterial hypertension, or diastolic pressures of ≥ 90 mmHg and systolic pressures of ≥ 140 mmHg, measured on two separate occasions within 24h, more than 6 hours apart, and proteinuria of ≥ 300 mg of protein in 24-hour urine samples which were developed after the 20th week of pregnancy in previously normotensive women (5). In the analysis of clinical parameters, the highest recorded values of arterial blood pressure were used. The study group was divided into two subgroups: severe and mild preeclampsia, based on the presence of criterion for severe preeclampsia. The criterion for the diagnosis of severe preeclampsia was the presence of one of the following criteria: systolic blood pressure ≥ 160 mmHg or diastolic ≥ 110 mmHg, proteinuria ≥ 2 g/24h, increased serum creatinine, persistent headache or cerebro-visual disorders, persistent epigastric pain, platelet count < 100,000/mm^3 and/or findings of microangiopathic hemolytic anemia (with increased lactate-dehydrogenase) (6). The neonatal birth weight that was below the tenth percentile for a given gestational age was taken as a criterion for setting the diagnosis of IUGR. The control group consisted of 20 healthy pregnant women with singleton pregnancies, spontaneously conceived, with no fetal anomalies, delivered by elective cesarean section due to obstetric indications that cannot be linked to the etiology of tested disorders (previous cesarean section and breech presentation). The examined and the control group consisted of Caucasian women. The research has been approved by the Ethical Committee of the Medical Faculty, University of Niš and with the informed consent of the involved participants.

The results are systematized, and grouped in the data base. Statistical analysis was performed by using Statistical Package for Social Sciences software (SPSS version 15.0, Inc., Chicago, IL, USA). Continuous variables are presented as mean values, standard deviations and median, while the qualitative variables are presented by their frequency and percentage. Determination of the normality of distribution of continuous variables was performed by Shapiro-Wilk test. If the distribution of continuous data were normal, comparison of arithmetical mean values of two independent samples was performed by Student's t-test for independent samples, and if not, Mann-Whitney U-test was used. Comparison of absolute frequencies of categorical variables was performed by Chi-square test and his variants according to the size of the samples.

Results

Table 1 shows the clinical characteristics of pregnancies complicated by preeclampsia (severe and mild preeclampsia) compared to the control group. Comparisons and establishing the existence of statistical significance of differences were made between the unified study group with preeclampsia and the control group, while the table also shows the values of the parameters in the examined subgroups with severe and mild preeclampsia. The mean age of patients with preeclampsia was 31.3 years and was slightly higher than the control group (29.5 years), but no statistically significant difference in the age of pregnant women was reported. Although patients with severe preeclampsia were on average slightly older, no statistically significant difference in age was reported compared to patients with mild preeclampsia. A striking fact is that in the subgroup with severe preeclampsia, as many as 40.91% of patients were over 35 years old. Parity in pregnant women was significantly higher in the control compared to the study group (1.85 vs 1.40), by $p < 0.01$.

In preeclampsia, the mean gestational age at birth was significantly lower than the control group (36.60 gestational weeks (gw) vs 39.25 (gw) ($p < 0.001$). The proportion of preterm neonates in the

study group was 43.3%, and in 16.7% of the women in the study group the pregnancy had to be terminated before the 34th week of gestation. Proteinuria in severe preeclampsia was 2.81 g/24h with a median as a measure of central tendency of 0.57 g and only a few extremely high values. Severe preeclampsia within the study group was reported in 73.3% of patients, in 63.3% preeclampsia was associated with intrauterine growth retardation, and in 36.67% with oligoamnion.

The mean neonatal birth weight and the weight of the placenta in preeclampsia were significantly lower, the perinatal outcome worse and there were no perinatal deaths.

Table 1. The clinical characteristics of pregnancies complicated by preeclampsia (severe or mild) compared to the control group

Clinical parameters	**Preeclampsia** (n = 30)†				**Control group** (n = 20)†	
	Severe (n = 22)		**Mild** (n = 8)			
Age (years)	31.91 ± 6.55	34.00	29.63 ± 3.54	30.50	29.50 ± 4.54	28.50
Parity	1.32 ± 0.65	1.00	1.63 ± 0.52	2.00	1.85 ± 0.37**	2.00
Gestational age (weeks)	36.32 ± 2.63	36.50	37.50 ± 2.51	38.00	39.25 ± 0.97***	39.00
Systolic blood pressure	172.73 ± 15.69	170.00	147.50 ± 3.53	150.00	106.50 ± 11.93***	110.00
Diastolic blood pressure	110.77 ± 9.17	110.00	96.88 ± 3.34	97.50	65.75 ± 5.91***	70.00
Proteinuria (grams/24h)	2.81 ± 4.01	0.57	0.40 ± 0.10	0.39	0***	
Incidence of IUGR‡	15 (68.18%)		4 (50.00%)		0***	
Incidence of oligoamnion	7 (31.28%)		4 (50.00%)		0**	
Birthweight (grams)	2244.5 ± 773.2	2100.0	2731.2 ± 997.1	2675.0	3425.0 ± 451.4***	3475.0
APGAR score 1 min	7.68 ± 1.17	8.00	7.13 ± 2.42	8.00	8.80 ± 0.41***	9.00
APGAR score 5 min	8.14 ± 0.71	8.00	8.00 ± 1.60	9.00	8.95 ± 0.22***	9.00
Placental weight (grams)	436.3 ± 120.8	395.0	441.2 ± 119.2	485.0	585.5 ± 82.1***	580.0

† Data are presented as mean values ± standard deviation, median, or as incidences and percentages
‡ IUGR – Intrauterine growth retardation
** - $p < 0.01$; *** - $p < 0.001$

Table 2. Haematological parameters in preeclampsia compared to the control group

Haematological parameters	**Preeclampsia**† (n = 30)		**Control group**† (n = 20)	
Total leukocyte count, x 10^9/L	9.85 ± 2.68	9.30	9.29 ± 2.49	8.83
Red blood cells count, x 10^{12}/L	4.17 ± 0.38	4.20	4.01 ± 0.48	3.94
Hemoglobin, g/L	121.40 ± 12.92	126.00	108.75 ± 18.10 **	107.50
HCT - Hematocrit (%)	36.89 ± 3.38	38.00	34.28 ± 5.13 *	33.90
Platelets count, x 10^9/L	201.03 ± 51.52	207.50	253.10 ± 71.87**	238.50
Incidence of thrombocytopenia (< 150 x 10^9/L)	3 (10.00%)		1 (5.00%)	
Neonatal hemoglobin, g/L	152.4 ± 40.30	138.90	116.90 ± 11.50 ***	117.60
Neonatal hematocrit (%)	65.86 ± 6.70	68.00	56.72 ± 3.94 ***	57.00
Neonatal poycytemia (HCT ≥6 5%)	18 (60.00%)		0 (0.00%)***	
Neonatal platelets count, x 10^9/L	168.60 ± 32.25	162.50	189.50 ± 22.02 *	187.50
Incidence of neonatal thrombocytopenia	5 (16.67%)		0 (0.00%)	

† Data are presented as mean values ± standard deviation, median, or as incidences and percentages
* - $p < 0.05$; ** - $p < 0.01$; *** - $p < 0.001$

The study group reported statistically significant higher hematocrit ($p < 0.05$) and hemoglobin concentrations ($p < 0.01$), as well as lower platelet counts ($p < 0.01$) (Table 2). There were no statistically significant differences in haematological parameters between the two subgroups of the study group with respect to the severity of preeclampsia. However, all cases of thrombocytopenia were in the subgroup with severe preeclampsia.

Neonates in the study group reported significantly higher values of hematocrit and hemoglobin ($p < 0.001$), and significantly lower values of platelet count than the control group ($p < 0.05$). All cases of neonatal thrombocytopenia were in the group with preeclampsia, and were reported in 16.67% of cases. As many as 60% of the newborns in preeclampsia were polycythemic (with hematocrit ≥ 65), while none were reported in the control group ($p < 0.001$).

Table 3 shows the biochemical parameters in pregnant women with preeclampsia compared to the control group. The study group reported statistically significant higher values of aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma glutamyl transferase (γ-GT), triglyceride and urea by $p < 0.01$; lactate dehydrogenase (LDH), total cholesterol, creatinine and uric acid by $p < 0.001$ compared to the control group, and lower values of albumin ($p < 0.01$). The neonates in the study group reported statistically significant lower values of neonatal glycemia than the control group ($p < 0.05$).

Table 3. Biochemical parameters in preeclampsia compared to the control group

Biochemical parameters	**Preeclampsia**† (n = 30)		**Control group**† (n = 20)	
AST - aspartate aminotrans. (U/L)	23.63 ± 8.57	21.00	18.26 ± 6.24 **	17.45
ALT - alanine aminotrans. (U/L)	18.51 ± 7.75	16.30	12.28 ± 5.03 **	9.65
LDH - lactate dehydrogenase (U/L)	467.97 ± 229.61	416.30	308.07 ± 80.04 ***	304.50
γ-GT - gamma-glutamyl trans. (U/L)	12.60 ± 5.24	10.45	8.71 ± 3.80 **	7.45
Total bilirubin (μmol/L)	6.34 ± 2.74	5.95	9.69 ± 3.55 ***	8.75
Direct bilirubin (μmol/L)	0.94 ± 0.48	0.80	1.37 ± 0.60 **	1.10
Serum proteins (g/L)	59.67 ± 7.06	59.10	61.39 ± 3.76	61.40
Serum albumin (g/L)	30.32 ± 4.37	29.75	33.3 ± 4.32 **	33.60
Total cholesterol (mmol/L)	8.18 ± 1.75	8.73	6.40 ± 1.82 ***	6.00
Triglycerides (mmol/L)	4.07 ± 1.17	4.00	3.08 ± 0.99 **	2.94
Urea (mmol/L)	3.85 ± 1.68	3.40	2.59 ± 0.80 **	2.45
Creatinine (μmol/L)	71.62 ± 9.81	70.85	62.32 ± 6.80 ***	61.70
Uric acid (μmol/L)	329.38 ± 82.03	327.35	232.02 ± 39.58 ***	225.15
CRP - C-reactive protein (mg/L)	6.59 ± 3.73	6.15	5.20 ± 4.23	3.25
Neonatal glycemia (mmol/L)	2.49 ± 1.13	2.30	3.27 ± 1.07 *	3.40

† Data are presented as mean values ± standard deviation, median, or as incidences and percentages
* - $p < 0.05$; ** - $p < 0.01$; *** - $p < 0.001$

Discussion

The average age of pregnant women in our study group of 31.3 years is approximate to the average age of patients with preeclampsia in many other studies (7, 8). Although there are studies reporting the prevalence of younger pregnant women among those affected by preeclampsia (9, 10), our study showed that patients with severe preeclampsia were on average slightly older than those with mild preeclampsia. It is striking that in the subgroup with severe preeclampsia, as many as 40.9% of patients were of advanced age (35 years and older). Other studies have also found that advanced age is a risk factor for severe preeclampsia (11).

There is a statistically significant difference in the parity of the patients in our study and control group. This information is not surprising and it is predominantly due to the way in which the control group was formed, with the highest frequency of pregnant women with previous birth by caesarean section as the main indication for a repeated caesarean section, aimed at avoiding pathological conditions that could impair the quality of the control group, as well as at excluding the effects of vaginal birth on perinatal outcome. However, within the study group itself, primiparae dominated by 66.7%

in proportion, with an average parity of 1.4, which is in agreement with the findings of other authors (7, 12). This has been explained by the fact that the maternal immune system responds to the genetically strange fetus, based on the hypothesis that the mother's immune system "learns" to adapt to the fetus and that preeclampsia results from failure of maternal tolerance to paternal alloantigens (13). Within the study group, there was a statistically significant difference in the parity of the patients in relation to the severity of preeclampsia. Significantly higher number of primiparae compared to multiparae was in the subgroup with severe preeclampsia compared to those with the mild form of the illness. Thus, primiparae are at greater risk of developing severe preeclampsia.

In the study group, the average gestational age at birth was 36.6 gestational weeks. The research by Aviram et al. reported the approximate average age of pregnancies complicated by preeclampsia, at 36.2 gestational weeks (8), and in the study by Kumari et al. it was 35.5 weeks (10). The proportion of preterm neonates in the study group was 43.3%, which is also in agreement with the findings of other authors (9).

Proteinuria is one of the two essential criteria for defining preeclampsia. It is caused by damage to the endothelium of renal glomeruli as one of the manifestations of generalized endothelial damage that exists in preeclampsia. By the values of proteinuria, our study group is extremely inhomogeneous. Quite divergent results are reported among the existing studies examining whether there is an association between the degree of proteinuria and maternal and fetal perinatal outcomes. While some negate the influence of proteinuria degree on the presence of maternal complications and perinatal outcome (14, 15), others indicate an association between the degree of proteinuria and these complications (10, 16, 17).

Neonates from pregnancies complicated by preeclampsia, and especially preeclampsia associated with IUGR, are known to be at greater risk of various complications (18). The newborn parameters we analyzed, such as birth weight, and Apgar score at the 1^{st} and 5^{th} minutes, were all significantly lower in the study group. Our results are consistent with those of other authors suggesting an association of fetal growth restriction and preeclampsia (8, 19). The existence of cases associated with IUGR and cases with eutrophic growth within preeclampsia indicates at least two etiopathogenetic modalities in relation to the presence of placental dysfunction (20-22).

Of haematological parameters among our patients, the most significant is the statistically lower platelet count in the study group compared to the control group. All cases of thrombocytopenia were from the subgroup with severe preeclampsia. According to the findings of most other authors, low platelet count is one of the most important laboratory indicators associated with poor maternal outcome (23, 24). In pregnant women with a platelet count lower than 50 x 10^9/L, the risk of coagulation disorders is 7.78 times higher, and in those with a platelet count of 50 to 99 x 10^9/L 2.69 times higher than in pregnant women with a platelet count greater than 150 x 10^9/L (24).

In the study group significantly higher are the values of hematocrit and hemoglobin concentration compared to the control group, which is in agreement with the reports of some other authors indicating the association between hemoconcentration and preeclampsia (25). Hemoconcentration leads to the reduction of uterine perfusion. Negative correlation between hemoglobin values and neonatal birth weight was reported in both normotensive women (26) and women with preeclampsia (25, 27).

Of the neonatal haematological parameters, the registered elevated values of hemoglobin, hematocrit, and incidence of polycythemia in neonates in preeclampsia can be explained by chronic hypoxia. Chronic tissue hypoxia induces an increase in plasma erythropoietin levels during fetal life resulting in stimulation of fetal erythropoesis and polycythemia. Polycythemia, due to blood hyperviscosity, further burdens the neonatal hemodynamics and results in impaired cardiopulmonary and metabolic adaptation of the newborn with the deepening of hypoxia. It is often present in preeclamptic newborns, and especially those with impaired growth (28). In our study, the average platelet count in the neonates of the study group was significantly lower compared to the control group, and thrombocytopenia was present in 16.7% of the neonates in the study group, and was not reported in the control group. All of our registered cases of neonatal thrombocytopenia reported mild thrombocytopenia, which is a characteristic of most chronic intrauterine hypoxia - induced neonatal thrombocytopenia (29).

In pregnancies complicated by preeclampsia, thrombocytopenia is usually identified at birth or within the first 72h with resolution within the first 10 days of life in most cases (30). The pathogenesis of neonatal thrombocytopenia in preeclampsia is not completely clarified. One possible mechanism is that chronic hypoxia has a direct depressive effect on megakaryocytic proliferation, which is supported by a study showing that IUGR fetuses have a significant megakaryocytopoetic defect without evidence of increased platelet destruction (31).

The Benoit and Rey study did not determine that decreased plasma albumin levels in pregnant women with preeclampsia could be an independent marker of the severity of preeclampsia (32). The abnormality of any parameter of liver function increases the risk of poor maternal outcome (24), but do some parameters affect more than others?

Laskin et al. indicate a positive correlation of elevated AST, ALT, and LDH levels, decreased albumin levels, and poor maternal outcome (23), whereas von Dadelszen et al. report elevated AST values as one of the major predictors of poor maternal outcome (33). An increase in LDH levels is associated with an increase in the severity of the disease (34). In our study, all AST and LDH values above the reference values were reported in the subgroup with severe preeclampsia, so we considered these two parameters to be the most significant indicators of the severity of preeclampsia among all biochemical parameters.

Changes in lipid status that occur in normal pregnancy are accentuated in preeclampsia (35-37). Our study group, too, reported increased cholesterol and triglyceride levels. There are also data of increased triglyceride levels in preeclampsia with normal cholesterol levels (38). However, numerous studies suggest that there is no difference in the levels of lipid parameters in preeclampsia compared to normal pregnancy (39).

Of the other laboratory-biochemical parameters we analyzed, the values of urea, creatinine and uric acid were significantly higher in the study group. Renal dysfunction is primarily reflected in increased serum uric acid levels, which is the most sensitive laboratory indicator of preeclampsia and its specific marker (40). Elevation of uric acid levels in preeclampsia often precedes hypertension and proteinuria, i.e. precedes the clinical manifestations of this disorder. Although there are studies suggesting that uric acid levels may be a predictor of poor perinatal outcome in preeclampsia (11, 41-43), most have shown that its value in predicting poor maternal and fetal outcome has not yet been confirmed (44, 45). The decrease in its clearance is due to decreased glomerular filtration in preeclampsia, and its elevated values are also a consequence of its increased production under oxidative stress (40). Uric acid is the end product of purine metabolism and the involvement of xanthine oxidase enzyme is important for its synthesis. The oxidative damage to the placenta and the resulting cytokines accelerate the synthesis of this enzyme and thus increase uric acid production. During normal pregnancy, serum concentrations of uric acid drop by 25-30% in early pregnancy due to increased renal clearance resulting from increased glomerular filtration and decreased proximal tubular reabsorption and changes in its production. Later, during pregnancy, the levels of serum uric acid rise, especially due to increasing fetal production and decreased binding to albumin, up until the end of pregnancy when they reach prepregnancy values (40). The most widely accepted explanation for hyperuricemia in preeclampsia is an increase in proximal tubular reabsorption, a decrease in tubular excretion, and a consequence of increased xanthine oxidase activity. The study by Dong et al. reported that serum uric acid levels were approximately equal and not elevated in normal pregnancy and pregnancy with isolated gestational hypertension, and significantly higher in preeclampsia (46), while Williams et al. found that its values were elevated even in pregnancy-induced hypertension without proteinuria (47). Creatinine clearance was decreased in most patients with severe preeclampsia. However, serum creatinine levels were not very helpful because of the wide range of normal values, also shown in our study, with the study group reporting significantly higher urea and creatinine levels, but not beyond the referential ones. Changes in urea clearance are accompanied by changes in creatinine clearance. However, if serum creatinine values rise so much that they fall outside the reference range, a predictor of poor maternal outcome has been confirmed (24, 33).

Regarding the biochemical parameters of new-borns in preeclampsia, dominant is the finding of decreased values of glycemia. The cause of hypoglycaemia in these neonates should be sought in the reduced glycogen reserves (glycogenolysis is the major source of glucose in neonates in the first hours after birth), decreased fetal hepatic gluconeogenesis due to impaired hepatic flow, and decreased maternal glucose transplacental transport. Numerous studies confirm that hypoglycemia is one of the most commonly present biochemical parameters of neonates in preeclampsia (48).

Conclusion

The laboratory parameters in our study, associated with a severe clinical picture of preeclampsia and a worse perinatal outcome, were thrombocytopenia and elevated AST and LDH levels. However, despite being poorer outcome indicators, they cannot be used with absolute certainty and in isolation from other parameters to predict poor perinatal outcome in preeclampsia. Deciding the delivery time in relation to an expectative approach should be based on a comprehensive consideration of gestational age, fetal condition, and clinical and laboratory maternal indicators.

Conflict of Interest

The authors declare that they have no any actual or potential conflict of interest including any financial, personal or other relationships with other people or organizations.

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VREDNOST HEMATOLOŠKIH I SERUMSKIH BIOHEMIJSKIH PARAMETARA U PREDIKCIJI PERINATALNOG ISHODA KOD PREEKLAMPSIJE

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Preeklampsija je ozbiljan poremećaj, koji se karakteriše generalizovanim maternalnim inflamatornim odgovorom, udruženim sa difuznom disfunkcijom endotelnih ćelija. Preeklampsija ima dugu pretkliničku fazu, pre nego postane manifestna. Mogućnost predviđanja komplikacija kod preeklampsije je klinički veoma značajna, jer bi mogla doprineti smanjenju morbiditeta i mortaliteta majki i neonatusa.

Cilj ovog rada je da ispita da li hematološki i serumski biohemijski parametri mogu biti od koristi u predikciji teže kliničke slike i goreg perinatalnog ishoda kod preeklampsije.

Prospektivna opservaciona studija fokusirala se na ispitivanu grupu od 30 jednoplodnih trudnoća sa preeklampsijom završenom carskim rezom. Ova ispitivana grupa podeljena je na dve podgrupe. Ispitanice su podelje u grupe shodno težini preeklampsije (umerena i teška). Kontrolnu grupu činilo je 20 zdravih trudnica, porođenih elektivnim carskim rezom. Analizirane su kliničke karakteristike trudnica, hematološki i serumski biohemijski parametri, kao i perinatalni ishod. Kod preeklampsije, povišene su vrednosti hematokrita i hemoglobina, a umanjen je broj trombocita. Takođe, povišene su vrednosti aspartat aminotransferaze (AST), alanin aminotransferaze, laktat dehidrogenaze (LDH), gama-glutamil transferaze, holesterola, triglicerida, mokraćne kiseline, uree i kreatinina. Laboratorijski parametri, koji su u našem istraživanju bili udruženi sa teškom kliničkom slikom preeklampsije i gorim perinatalnim ishodom, bili su trombocitopenija i povišeni nivoi AST i LDH. Međutim, uprkos tome što su pokazatelji goreg ishoda, ne mogu se sa apsolutnom sigurnošću i izolovano od drugih pokazatelja koristiti u predikciji lošeg perinatalnog ishoda kod preeklampsije. Donošenje odluke o trenutku za porođaj, u odnosu na ekspektativni pristup, trebalo bi da bude bazirano na sveobuhvatnom sagledavanju gestacijske starosti, stanja fetusa, kliničkih i laboratorijskih maternalnih pokazatelja.

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Ključne reči: *biohemijski parametri, hematološki parametri, perinatalni ishod, preeklampsija*