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A Comparison of Maternal Procalcitonin Concentrations in Preeclamptic and Normotensive Patients

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ABSTRACT

Background: Preeclampsia is a specific multisystemic disorder in pregnancy characterized by hypertension and proteinuria after 20 weeks of gestation. Several studies have found that pro-inflammatory cytokine levels were higher in preeclampsia patient than women with normotensive pregnancy, one of which was procalcitonin. Aim: To determine the relationship between procalcitonin level in severe preeclampsia and women with normotensive pregnancy. Methods: Sampling collection in this study was obtained using the executive sampling method. There were 50 study samples, consisting of 25 pregnant patients with severe preeclampsia and 25 normotensive pregnant patients who underwent delivery at Prof. Dr. R. D. Kandou Manado Hospital and Related Hospital in Manado City. Measurement of Procalcitonin level in severe preeclampsia and normotensive pregnancy was performed. Data were then analyzed using SPSS software version 20.0. Results: Procalcitonin levels in pregnant women with severe preeclampsia was 0.100 (0.020.00–0.900) ng/mL. Procalcitonin level in normotensive pregnant women was 0.040 (0.0150–0.045) n/mL. There was a significant relationship (p <0.001) between PCT level and severe preeclampsia events. Severe preeclampsia patients had a higher PCT level than normotensive pregnant women. Patients with PCT level≥0.095 ng/mL had a relative risk of experiencing severe preeclampsia of 4.125 (Confidence interval [CI] 95%, 2,257-7,540); and had a specificity and sensitivity of 100% and 68%, respectively. Conclusion: There was a significant relationship between PCT levels and the incidence of severe preeclampsia. Severe preeclampsia patients had a higher PCT level than normotensive pregnant women.

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ABSTRAK

Latar Belakang: Preeklamsia adalah kelainan multisistemik spesifik pada kehamilan yang ditandai oleh timbulnya hipertensi dan proteinuria setelah umur kehamilan 20 minggu. Beberapa studi menemukan bahwa kadar sitokin proinflamasi pada pasien preeklamsia lebih tinggi dibandingkan dengan wanita hamil

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normotensi, salah satunya adalah prokalsitonin. Tujuan: Mengetahui hubungan antara kadar prokalsitonin pada preeklamsia berat dan wanita hamil normotensi. Metode: Pengambilan sampel pada penelitian ini menggunakan metode consecutive sampling Jumlah sampel penelitian 50 sampel terdiri dari 25 pasien hamil dengan preeklampsia berat dan 25 pasien hamil normotensi yang menjalani persalinan di RSUP Prof. Dr. R. D. Kandou Manado dan RS Jejaring di Kota Manado. Dilakukan pemeriksaan kadar Prokalsitonin pada kehamilan dengan PEB dan kehamilan dengan normotensi. Data kemudian dianalisis dengan menggunakan software SPSS versi 20.0. Hasil: Kadar prokalsitonin pada wanita hamil dengan preeklamsia berat adalah sebesar 0,100 (0,020,00–0,900) ng/mL. Kadar prokalsitonin pada wanita hamil normotensi adalah sebesar 0,040 (0,0150–0,045) ng/mL. Terdapat hubungan yang signifikan ($p < 0,001$) antara kadar PCT dengan kejadian PEB, dimana pasien PEB memiliki kadar PCT yang lebih tinggi dibandingkan wanita hamil normotensi. Pasien dengan kadar PCT ≥0,095 ng/mL memiliki risiko relatif untuk mengalami PEB sebesar 4,125 (IK 95%, 2,257–7,540); serta memiliki spesifisitas dan sensitivitas 100% dan 68%, secara berturut-turut. Kesimpulan: Terdapat hubungan yang signifikan antara kadar PCT dengan kejadian PEB, yang mana pasien PEB memiliki kadar PCT yang lebih tinggi dibandingkan wanita hamil normotensi

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INTRODUCTION

Preeclampsia is a specific multisystemic disorder in pregnancy characterized by hypertension and proteinuria after 20 weeks of gestation.[1] More than 4 million pregnant women have preeclampsia every year, and an estimation of 700,000 women died due to preeclampsia and 50,000 fetal deaths. Preeclampsia was held responsible for 15-20% of deaths of pregnant women worldwide and was a major cause of mortality and morbidity in the fetus.[2] In Indonesia, preeclampsia was the third-highest etiology (13%) for maternal mortality after bleeding (45%) and infection (15%).[3]

In a normal pregnancy, there was an activation of the innate immune system so that the maternal inflammatory response was also stimulated, but in preeclampsia patients, there was an excessive maternal systemic inflammatory response resulting in a general intravascular inflammatory reaction. This excessive systemic inflammatory response caused endothelial dysfunction and increased vascular reactivity resulting in clinical symptoms of preeclampsia.[4]

Several studies have found that pro-inflammatory cytokine levels in preeclampsia patients were higher than in normotensive pregnant women, one of which is procalcitonin.[5] Procalcitonin is a precursor of calcitonin, which plays a role in the calcium homeostasis process in the body. Until now, procalcitonin has often been used as an indicator of systemic inflammation caused by bacterial infections.[6] Study by Agostinis et al. (2018) and Artunc-Ulkumen et al. (2015) found that procalcitonin levels in maternal blood serum and placenta of preeclampsia patients were significantly higher than normal pregnant women.[7,8] However, another study by Duckworth et al. (2016) and Birdir C et al. (2015) found no significant differences in procalcitonin levels of preeclampsia patients and normotensive pregnant women.[9,10]

Based on different studies results related to the role of procalcitonin as a pro-inflammatory cytokine in the diagnostic examination of preeclampsia patients, the researcher aimed to determine the difference of procalcitonin levels in preeclampsia patients and normotensive pregnant women.

METHODS

This was an observational analytic study that evaluated differences in procalcitonin level in patients with severe preeclampsia with normotensive pregnant women. A cross-sectional study design was used. This research was conducted at Prof. dr. R. D. Kandou Manado General Hospital and related hospital in Manado City, Pancaran Kasih Hospital and Bhayangkara Hospital. Sample selection was done by executive sampling. The study samples were 25 pregnant women with severe preeclampsia and 25 normal pregnant women who visited the Prof. General Hospital. dr. R. D. Kandou Manado, Smooth Hospital, and Bhayangkara Hospital met the research inclusion criteria and were willing to participate in research.

The inclusion criteria in this study were: pregnant women in 20 weeks or more gestation age who were treated to the emergency department and Obstetrics and Gynecology Outpatient Clinic at Prof. dr. R. D. Kandou Manado General Hospital, Pancaran Kasih Hospital, and Bhayangkara Hospital; Severe preeclampsia pregnant women with a single live intrauterine fetus; Pregnant women in 20 weeks or more gestation age who were willing to take part in the study were marked by a signed informed consent sheet. The exclusion criteria were: Patients with chronic diseases including diabetes melitus, heart disease, kidney disease, malignancies, autoimmune disorders, or other chronic diseases; Not willing to participate in this research.

After history taking and physical examination, a blood sample was taken from patients for procalcitonin levels measurement. The blood sample was then sent to the Pramita Manado laboratory clinic. Then, an immunochromatographic analysis was performed using the Siemens BRAHMS KRYPTOR device that has been calibrated according to standards. Data analysis was carried out using SPSS software version 20.0. This research was conducted after obtaining approval and recommendations from the Health Research Ethics Committee, Sam Ratulangi University School of Medicine, Prof. dr. R. D. Kandou Manado.

RESULTS

This study recruited 25 patients into each case and control group (Table 1). The average age of the normotensive patient was 29± 8,261 years. This number was relatively higher than

the average age of severe preeclampsia patients (31.40± 8,332 years). However, researchers did not find a significant difference in the age distribution for each group.

Table 1. Study subject's baseline characteristics

Characteristic	Normotensive		Severe Preeclampsia	
	N	%	N	%
Maternal age				
<20 years	3	12	3	12
20–35 years	17	68	12	48
≥35 years	5	20	10	40
Parity				
Primiparity	10	40	6	24
Multiparity	15	60	19	76
Gestation age				
20–34 weeks	4	16	7	28
34–37 weeks	6	24	11	44
>37 weeks	15	60	7	28
Education				
No formal education	0	0	0	0
Elementary	0	0	0	0
Junior high school	5	20	3	12
Senior high school	16	64	19	76
Bachelor	4	16	3	12
Occupation				
Housewive	19	76	19	76
Civil officer	3	12	2	8
Private	3	12	4	16
Body mass index				
<18.5	0	0	0	0
>18.5- ≤ 25	1	4	11	44
> 25	24	96	14	56

In this study, researchers found the median PCT plasma level of patients with severe preeclampsia was 0.100 (0.020.00–0.900) ng/mL. This result was higher than the median PCT plasma level of patients with normal blood pressure (normotensive) was 0.040 (0.0150–0.045) ng/mL. A statistical test using the Mann-Whitney test found a significant difference in plasma PCT levels between severe preeclampsia patients and normotensive patients (p <0.001).

Table 2. Procalcitonin levels difference in severe preeclampsia and normotensive pregnant women

Group	Procalcitonin level (ng/mL)	p-value
Severe preeclampsia	0,100 (0,020,00–0,900)	<0,001
Normotensive pregnant women	0,040 (0,0150–0,045)	

Mann-Whitney test (CI 95%)

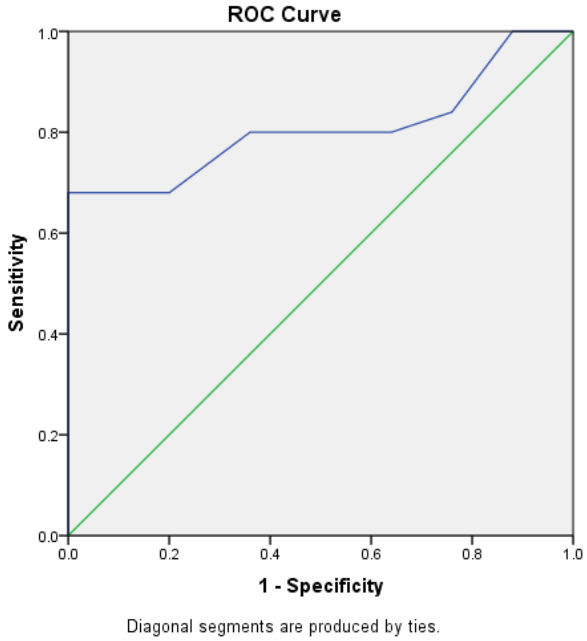

Figure 1. ROC curve of diagnostic value of PCT levels in severe preeclampsia prediction

The area under the curve (AUC) value was 0.807 (CI 95%, 0.676–0.938). This value indicated that the PCT level had a moderate predictive ability to determine severe preeclampsia events. The optimum cut point was recorded at a threshold ≥ 0.095 ng/mL with a Youden index of 1.680. Procalcitonin level's specificity and sensitivity values in predicting severe preeclampsia were 100% and 68%, respectively (Table 3).

Tabel 3. PCT diagnostic value at ≥0,095 ng/mL threshold to predict severe preeclampsia

Parameter	Value	CI 95%
Spesificity	100.00%	86.28%–100.00%
Sensitivity	68.00%	46.50%–85.05%

For the effect of PCT level on severe preeclampsia, the relative risk of patients with PCT ≥0.095 ng/ml to experience severe preeclampsia was 4.125 (CI 95%, 2.257–7.540). This indicates that PCT level ≥0.095 ng/ml was a significant risk factor for the occurrence of severe preeclampsia (table 4).

Table 4. Proportion/analysis test of the relationship of PCT level and severe preeclampsia

PCT level (ng/mL)	Diagnosis		RR (CI 95%)	P
	Severe preeclampsia (%)	Normotensive(%)		
≥0.095	17 (100)	0 (0)	4,125 (2,257–7,540)	<0,001*
<0.095	8 (24,24)	25 (75,76)		

Total	25 (50)	25 (50)

Chi-square test

DISCUSSION

The relationship between patient characteristics and severe preeclampsia events

In this study, we found that severe preeclampsia patients had older age than normotensive patients. However, this age difference was not statistically significant. The findings of this study were strengthened by Shen et al. who found that at the age ≥35 years, the risk of severe preeclampsia had not increased significantly compared to age <35 years.11 An article and practical guide related to hypertensive disease in pregnancy stated that older age was associated with an increased risk of severe preeclampsia, and age ≥40 years was a significant risk factor for severe preeclampsia in pregnant women, regardless of their parity history.12,13 Increased risk of severe preeclampsia in older age was associated with the uterine arteries aging triggering a failure of spiral arterial remodeling.14

The relationship between PCT level and severe preeclampsia events

The relationship between PCT levels and severe preeclampsia events was the main outcome of this study. In this study, the median PCT level for the normotensive group was found at 0.040 (0.0150–0.045) ng/mL. This number was lower than the PCT level in the severe preeclampsia group, 0.100 (0.020.00–0.900) ng/mL. Statistically, this median difference was found to be significant ($p < 0{,}0001$). The same findings were reported by Jannesari and Kazemi who conducted a case-control design study of 50 normotensive patients and 59 patients with severe preeclampsia. This study found a mean PCT level for the severe preeclampsia and normotensive group were 0.06±0.04 ng/mL and 0.04±0.01 ng/mL ($p = 0.001$).15 A case-control study in Turkey also found that PCT and endothelin-1 were related to the severe preeclampsia incidence and predicted pregnancy outcome.16 There were also studies in Indonesia with 65 samples, 40 samples with severe preeclampsia and the remaining 25 normotensive samples, showed a significant difference of PCT levels from preeclampsia and normotensive mothers with a value of $p = 0.005$. The severe preeclampsia samples PCT levels were ranged from 0.17192-0.43498 ng/mL, the mean value was 0.24134 ng/mL, while the normotensive sample PCT levels were ranged from 0.17592–0.26807 ng/mL.17

Researchers also tried to calculate the cut-off value used as a standard value of increased PCT that significantly related to severe preeclampsia events. The optimum cut-off point was obtained at 0.095 ng/mL, with the sensitivity and specificity of PCT levels recorded at 68% and 100%, respectively. Jannesari and Kazemi reported a cut-off value at 0.042 ng/mL. However, recorded specificity and sensitivity are too low, 71% and 54%, respectively.15 A study in Italy reported the relationship between PCT level and SEVERE preeclampsia events and found the optimum cut-off point at 50 ng/L (90.9% sensitivity and 40.3% specificity).18 A recent study by Artunc-Ulkumen et al. reported increased PCT levels in both mild and severe preeclampsia.18,19 Differences in cut points and diagnostic values (sensitivity and specificity) between studies and the findings of this study may be influenced by different patient

characteristics. However, it has been proven that PCT level was a significant predictor factor for severe preeclampsia events.

From the research of Duckworth et al. (2016) in 143 severe preeclampsia and 280 normotensive women, PCT did not represent a useful diagnostic test to determine the development of severe preeclampsia in 14 days (P> 0.001). The result differed from Montagnana et al.'s (2008) research that found PCT level in the severe preeclampsia group was significantly higher than in the mild preeclampsia and hypertension groups. They concluded that PCT was an inflammatory mediator (such as cytokines) instead of being a simple marker.5 Preeclampsia pathogenesis is still not entirely known, even though experts have carried out various theoretical approaches. A common inflammatory process is the most accepted hypothesis that can explain the pathogenesis of preeclampsia. The systemic inflammatory response of the mother is normal in pregnancy. However, this systemic inflammatory response is milder than severe preeclampsia patients. Therefore, inflammatory markers, such as CRP and PCT, will increase during pregnancy.

As long as the inflammation continues, PCT is released as an acute phase reactant to inflammatory stimulation, and serum PCT levels increase rapidly.20 For this reason, PCT is generally accepted as a good predictive marker related to the inflammatory process and an additional tool to guide antibiotic prescribing.21.22 Serum PCT level was increased faster than C-reactive protein levels (CRP) and reached its peak in a very short time. In addition, if the patient responds appropriately to treatment, the PCT level will return to the normal range faster than CRP, which makes it a more effective biomarker for life-threatening systemic inflammatory diseases.23

Excessive inflammation in pregnancy has been linked to pregnancy complications such as premature labor, intrauterine growth restriction, and preeclampsia. Preeclampsia is a multisystemic disorder in human pregnancy characterized by expanded vascular endothelial malfunction and vasospasm after 20 weeks of gestation and can appear up to 4-6 weeks postpartum. Clinically, preeclampsia is defined by hypertension and proteinuria, often characterized by increased liver enzymes and thrombocytopenia with or without pathological edema.24 The idea that the placenta is the origin of preeclampsia pathogenesis has been universally accepted. An uncomplete spiral arteries remodeling was considered an initial trigger in hypertension, proteinuria, and other fetomaternal complications.25

Increased pro-inflammatory cytokines that circulate in preeclampsia can be responsible for increasing systemic PCT level; Furthermore, increased PCT level induced the production of pro-inflammatory cytokines that stimulated PCT release, which triggered the production of the PCT, thus causing positive curvature of PCT secretion.26 Another role that PCT contributed in preeclampsia pathogenesis was related to its cytotoxic activity in hepatocytes and endothelial,27.28 which the typical characteristic of preeclampsia are endothelial dysfunction and liver damage.29 Regarding the local role of PCT in the placenta in preeclampsia, Agostinis et al. detected a substantial increase in mRNA expression of PCT in preeclampsia patients compared with normal placenta.30 Although trophoblasts, stromal cells, and decidua endothelial produce PCT under normal physiological conditions, this production did not reach the limits that can be detected in plasma. The main pro-inflammatory cytokine whose levels have been shown to increase in preeclampsia patients is TNF-α.31,32

The various pathomechanisms described have explained the researchers' findings in this study. PCT was related to the incidence of severe preeclampsia, where an increase in PCT ≥0.095 ng/mL became a significant predictor factor for severe preeclampsia events. The role of PCT in predicting severe preeclampsia is inseparable from severe preeclampsia's pathophysiology, which involves placenta inflammation triggering PCT release that can be detected in plasma.

This research has successfully linked the patient's demographic factors and PCT to severe preeclampsia incidence in pregnant women. Apart from the findings mentioned by the researcher, there were several weaknesses of the study that researchers want to underline. First, this study had a case-control design that had a high risk of biased selection. The selection of the study design was carried out based on the availability of limited severe preeclampsia cases. Second, this study did not evaluate other factors that cause an increase in PCT level. As PCT is also produced in various inflammatory conditions, including bacterial infections, the assessment of whether or not there are signs of infection is deemed necessary to ensure that the increase in PCT is solely caused by severe preeclampsia.

CONCLUSION

Based on the findings in this study, it can be concluded that there was a significant relationship between PCT level and severe preeclampsia events. Severe preeclampsia patients had higher PCT levels than normotensive pregnant women. Patients with PCT levels ≥0.095 ng/mL had a relative risk of experiencing severe preeclampsia of 4.125 with 100% specificity and 68% sensitivity.

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