Decrease Rate of Platelet Count as a Marker for Complications of Preeclampsia: A Cross-Sectional Study

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Abstract

Background

Thrombocytopenia is considered a sign of maternal organ damage of preeclampsia. However, platelet count distribution during pregnancy shifts downward even in uncomplicated women, and platelet counts in some uncomplicated women falls below the cutoff value of thrombocytopenia. Thus, not only absolute platelet counts number but also platelet count reduction rate may relate with adverse outcomes of preeclampsia. This study aimed to evaluate whether the overall platelet count reduction rate was associated with adverse outcomes of preeclampsia.

Methods

This retrospective study included 240 singleton pregnant women with preeclampsia. Overall platelet count reduction $\geq 30.0\%$ was used as a cut-off value because 10–20% platelet counts reduction can be observed in uncomplicated pregnancy. The association between the overall platelet count reduction from the first trimester $\geq 30.0\%$ and adverse outcomes; severe hypertension, uteroplacental dysfunction, maternal organ damage except for thrombocytopenia, and preterm delivery <34 gestational weeks were analyzed. Moreover, subgroup analysis was conducted in women without thrombocytopenia.

Results

A total of 95/240 (39.6%) of women had $\geq 30.0\%$ of overall platelet count reduction from the first trimester. The incidences of maternal organ damage except for thrombocytopenia (12.4%, 22.1%; $p = 0.047$), and preterm delivery <34 gestational weeks (30.3%, 48.4%; $p = 0.005$) were higher in women with $\geq 30.0\%$ of overall platelet count reduction than those otherwise. Even in women without thrombocytopenia (platelet counts $\geq 150 \times 10^9/L$), severe hypertension (66.2%, 85.4%; $p = 0.018$), and preterm delivery <34 gestational weeks (30.8%, 51.2%; $p = 0.017$) were more frequently observed in women with $\geq 30.0\%$ of overall platelet count reduction than those otherwise.

Conclusions

A $\geq 30.0\%$ of overall platelet count reduction from the first trimester was associated with adverse outcomes of preeclampsia even in women without thrombocytopenia.

Introduction

Failure of trophoblast invasion and remodeling of spiral arteries followed by imbalance of angiogenic and antiangiogenic factors cause systemic endothelial damage, which results in preeclampsia [1, 2]. Injured endothelium can activate platelets, increasing the consumption of platelets and causing thrombocytopenia in preeclampsia [3]. Therefore, thrombocytopenia is considered a sign of maternal organ damage of preeclampsia [4, 5].

Platelet count (PC) in pregnant women decreases throughout pregnancy [6], and gestational thrombocytopenia (defined as $PC < 150 \times 10^9/L$ without any other etiology) occurs in 4.4%–11% of pregnancies [7, 8]. While gestational thrombocytopenia is not associated with maternal and neonatal complications, severe thrombocytopenia in preeclampsia including hemolysis, elevated liver enzymes, low platelet syndrome increases the risk of adverse maternal and fetal outcomes [9]. Expectant management can be chosen in cases with stable maternal and fetal condition [4, 5]. However, thrombocytopenia can get worse during expectant management because preeclampsia is a progressive disease. PC distribution during pregnancy shifts downward even in uncomplicated women, and PC in some uncomplicated women falls below the cutoff value of thrombocytopenia ($< 150 \times 10^9/L$). Therefore, the evaluation of not only the absolute PC value but also the overall reduction rate of PC from the baseline may give further information for predicting adverse outcomes of preeclampsia. This study aimed to examine whether the overall reduction rate of PC from the first trimester is associated with adverse outcomes of preeclampsia.

Materials And Methods

Study Population

Women who were diagnosed with preeclampsia and delivered at Hokkaido University Hospital and Japan Community Health Care Organization Hokkaido Hospital between April 2010 and May 2019 were enrolled for this study. Women who were < 18 years old at delivery or transferred to other hospitals before delivery were excluded. In addition, women were excluded if their thrombocytopenia was not associated with preeclampsia, the baby had severe congenital anomaly, and data of PC levels in the first trimester were not available.

Data Collection

Diagnosis of preeclampsia was conducted based on According to the International Society for the Study of Hypertension in Pregnancy and Japan Society for the Study of Hypertension in Pregnancy guidelines, preeclampsia was defined by the new onset of hypertension (systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg) accompanied by proteinuria (protein/creatinine ratio ≥ 0.3) and/or a sign of maternal organ damage and utero-placental dysfunction, as listed in Table 1 [4, 10]. The results of blood tests conducted during 9–13 gestational weeks (GW) were used as the value of the PC levels at the first trimester. The overall reduction rate of PC was defined with the formula: (PC levels at the first trimester – PC levels at delivery) / PC levels at the first trimester. The women were divided into the three groups based on the PC levels at delivery: normal ($PC > 150 \times 10^9/L$), mild thrombocytopenia ($\geq 100 \times 10^9/L$ and $< 150 \times 10^9/L$), and severe thrombocytopenia ($< 100 \times 10^9/L$).

The incidence of severe hypertension (systolic blood pressure, ≥ 160 mmHg; diastolic blood pressure, ≥ 110 mmHg), maternal organ damage, uteroplacental dysfunction, and preterm delivery < 34 GW were analyzed. Maternal organ damage and uteroplacental dysfunction were defined according to Table 1.

Statistical Analyses

Statistical analyses were conducted using Stata/SE version 15.1 (StataCorp, College Station, TX, USA). The normality of the data was analyzed using histograms in terms of skewness and kurtosis. Continuous data are reported as mean $\pm$ standard deviation. Categorical variables are expressed as frequency and percentage. Statistical significance was calculated using independent *t*-test and chi-square test for continuous data and categorical variables, respectively. PC decrease is part of the natural course of pregnancy and 10%–20% reduction is observed in noncomplicated pregnancy [6, 11]. Therefore, a 30.0% reduction was used as the cutoff value of overall reduction of PC decrease from the first trimester. Odds ratio calculated with logistic regression analysis often overestimates relative risk (RR) for common outcomes (incidence >10.0 %) [12]. Therefore, the Poisson regression analysis with a robust error variance was used to estimate the RR and 95% confidence interval (CI).

Ethical Approval

The institutional review board of Hokkaido University Hospital (019-0070) and Japan Community Health Care Organization Hokkaido Hospital (2020-4) approved this study. Hokkaido University Hospital Clinical Research Administration Center and Japan Community Health Care Organization Hokkaido Hospital Ethics Review Board did not require informed consent for this retrospective study. Information about this study was placed on the home pages of Hokkaido University Hospital and Japan Community Health Care Organization Hokkaido Hospital with the opportunity to opt out. All methods were carried out in accordance with the Declaration of Helsinki.

Results

During the study period, 329 women were diagnosed with preeclampsia. Of these women, 89 fulfilled the exclusion criteria and were not included in the analysis. Therefore, 240 women were evaluated in this study. Among them, 44 (18.3%) of women developed mild thrombocytopenia and 22 (9.2%) of women developed severe thrombocytopenia (Fig 1). In addition, 95/240 (39.6%) of women had $\geq 30.0\%$ of overall platelet count reduction from the first trimester.

Clinical characteristics based on the overall reduction rate of PC from the first trimester in women with preeclampsia are shown in Table 2. PC in the first trimester was similar between the two groups (<30.0% of reduction, $\geq 30.0\%$ of reduction; $249 \pm 53 \times 10^9/L$, $254 \pm 59 \times 10^9/L$, $p = 0.442$). At delivery, 95/240 (39.6%) of women had a $\geq 30.0\%$ of overall PC reduction from the first trimester and developed mild and severe thrombocytopenia more frequently than those with <30.0% of overall PC reduction ($p < 0.001$). The incidences of maternal organ damage except for thrombocytopenia (12.4%, 22.1%; $p = 0.047$), and preterm delivery <34 GW (30.3%, 48.4%; $p = 0.005$) were also higher in women with $\geq 30.0\%$ of overall PC reduction than those otherwise. The RR of $\geq 30.0\%$ of overall PC reduction for the adverse outcomes in adjusting maternal age are presented in Table 3. A $\geq 30.0\%$ of overall PC reduction was significantly associated with the increased risk of preterm delivery <34 GW (RR, 1.58; 95% CI, 1.14–2.18).

The clinical characteristics based on the overall reduction of PC from the first trimester in preeclamptic women without thrombocytopenia are shown in Table 4. Even in the women whose PC levels were normal, severe hypertension (66.2%, 85.4%; $p = 0.018$), and preterm delivery <34 GW (30.8%, 51.2%; $p = 0.017$) were more frequently observed in women whose overall reduction of PC was $\geq 30.0\%$ than those otherwise. On the other hand, the incidence of uteroplacental dysfunction was higher in women with <30.0% of overall PC reduction (33.1%, 12.2%; $p = 0.009$). PC in the first trimester ($256 \pm 50 \times 10^9/L$, $297 \pm 49 \times 10^9/L$; $p < 0.001$) was higher in women with $\geq 30.0\%$ of overall PC reduction, while PC at delivery ($226 \pm 47 \times 10^9/L$, $179 \pm 25 \times 10^9/L$; $p < 0.001$) was lower in women with $\geq 30.0\%$ of overall PC reduction. The RR of $\geq 30.0\%$ of overall PC reduction for the adverse outcomes in preeclamptic women without thrombocytopenia in adjusting maternal age are presented in Table 5. a $\geq 30.0\%$ of overall PC reduction was significantly associated with the increased risk of severe hypertension (RR, 1.29; 95% CI, 1.08–1.54), and preterm delivery <34 GW (RR, 1.66; 95% CI, 1.12–2.46). By contrast, a $\geq 30.0\%$ of overall PC reduction was negatively associated with uteroplacental dysfunction (RR, 0.37; 95% CI: 0.16–0.87).

Discussion

This study revealed that the overall reduction rate of PC was associated with the adverse outcomes of preeclampsia, and it was also observed in women without thrombocytopenia. Close monitoring of both mother and baby is essential especially after developing preeclampsia [4, 5]. Although serum biomarkers, such as the ratio of soluble fms-like tyrosine kinase-1 (sFlt-1) to placental growth factor (PlGF) have a predictive value for the adverse outcomes of preeclampsia [13], these biomarkers are not widely available especially in developing countries. On the other hand, a complete blood count is a more globally accessible test. Therefore, assessing the risk of adverse outcomes of preeclampsia with PC has clinical benefits.

Physiological changes during pregnancy decrease PC. Hemodilution caused by increased plasma volume contributes to lower PC during pregnancy [14]. In nonpregnant women, one-third of circulating platelets are trapped within the splenic sinusoids [15]. The size of the spleen increased by 50% during pregnancy [17], and the placental circulation is similar to the splenic circulation [18]. Because of these changes, PC gradually decrease by 17% at the time of delivery from a nonpregnant situation in uncomplicated pregnancy [6]. This study used PC in the first trimester as a baseline because PC in nonpregnant situation is often unavailable in clinical practice. The PC level at the first trimester is below the level of nonpregnant women [6]. Therefore, the overall reduction of PC $\geq 30.0\%$ from the first trimester exceeds the physiological change. Thus, $\geq 30.0\%$ of overall PC reduction may be a sign of elevated consumption of the platelets caused by endothelial damage with preeclampsia. In addition, 7.6% of women with <30.0% of overall PC reduction developed mild thrombocytopenia in this study, and this rate is compatible with the incidence of gestational thrombocytopenia (4.4%–11%) in all pregnancy [7, 8]. This finding implies that thrombocytopenia in women with <30.0% of overall PC reduction is not associated with preeclampsia.

A previous study reported that little variation of the PC reduction existed in women with uncomplicated pregnancy. Therefore, women whose PC are at the lower end of the normal range before pregnancy tend to have thrombocytopenia during pregnancy [6]. Actually, the recurrent risk of gestational thrombocytopenia was 14.2 times higher in women with a history of gestational thrombocytopenia than those who have no history of gestational thrombocytopenia [6]. In this study, a $\geq 30.0\%$ of overall PC reduction was associated with severe hypertension and preterm delivery $< 34\text{GW}$ even in the women whose PC levels were within normal limits. This may be because the PC levels of these women before pregnancy were at the higher end of the normal range. Thus, their PC did not reach the cutoff value for thrombocytopenia although the PC reduction was a result of elevated consumption of the platelets caused by endothelial damage with preeclampsia.

Uteroplacental dysfunction in this study was negatively correlated with the overall reduction of PC. Inadequate trophoblast invasion and following uteroplacental malperfusion cause both preeclampsia and fetal growth restriction [18, 19]. However, not all cases of fetal growth restriction are associated with preeclampsia. It is difficult to assess the effect of preeclampsia on fetal growth restriction because the etiology of fetal growth restriction varies [20]. In this study, hypertensive women with fetal growth restriction were classified as preeclampsia even if they did not have proteinuria or sign of maternal organ damage. The symptoms of preeclampsia may not be severe in some of those women because fetal growth restriction was not a result of preeclampsia; therefore, those women less likely to develop a $\geq 30.0\%$ of overall PC reduction. This might be a reason why uteroplacental dysfunction was observed more frequently in women with $< 30.0\%$ of overall PC reduction in this study.

This study has several limitations. First, this is a retrospective study and thrombocytopenia is a sign of maternal organ damage. Thus, the incidence of preterm delivery can be biased. However, the association between adverse outcomes of preeclampsia and overall reduction of PC was also detected in women without thrombocytopenia. Second, PC in the first trimester may be affected by hemoconcentration because of hyperemesis gravidarum, and the severity of hyperemesis gravidarum could not be assessed from the medical records in this study. However, PC in the first trimester was not different based on the overall reduction of PC. Thus, hyperemesis gravidarum would not affect the results of this study. Finally, sFlt-1 and PlGF were not widely available in Japan during the study period. Therefore, the relationship between the overall reduction of PC and these biomarkers could not be analyzed.

Conclusion

This study implies that a decrease rate of PC was an independent risk factor of the complications of preeclampsia irrespective of PC levels despite these limitations. Women who develop a $\geq 30.0\%$ PC decrease need to be considered high risk for developing complications of preeclampsia.

Abbreviations

CI: confidence interval

GW: gestational weeks

OR: odds ratio

PC: platelet count

PlGF: placental growth factor

RR: relative risk

sFlt-1: soluble fms-like tyrosine kinase-1

Declarations

Ethics approval and consent to participate

Hokkaido University Hospital Clinical Research Administration Center (019-0070) and Japan Community Health Care Organization Hokkaido Hospital Ethics Review Board (2020-4) approved this study. Hokkaido University Hospital Clinical Research Administration Center and Japan Community Health Care Organization Hokkaido Hospital Ethics Review Board did not require informed consent for this retrospective study. Information about this study was placed on the home pages of Hokkaido University Hospital and Japan Community Health Care Organization Hokkaido Hospital with the opportunity to opt out. All methods were carried out in accordance with the Declaration of Helsinki.

Consent for publication

Not required

Competing interests

The authors declare that they have no competing interests.

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Availability of data and materials

All data generated or analysed during this study are included in this published article as a supplementary information file.

Author contributions

All listed authors meet criteria for authorship. MM1 made substantial contributions to conceptualization, design of the work, and drafting the work. MM2 made substantial contributions to conceptualization, the

acquisition, and the analysis. TY and TU made substantial contributions to interpretation of data. KN1 and KN2 made substantial contributions to the acquisition, and the analysis. YS and KC made substantial contributions to design of work. HW made substantial contributions to drafting and revising work. All authors approved the submitted version and agreed to be personally accountable for their contributions to the work.

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References

1. Nagamatsu T, Fujii T, Kusumi M, et al. Cytotrophoblasts up-regulate soluble fms-like tyrosine kinase-1 expression under reduced oxygen: an implication for the placental vascular development and the pathophysiology of preeclampsia. *Endocrinology*. 2004;145:4838–4845.
2. Maynard SE, Min JY, Merchan J, et al. Excess placental soluble fms-like tyrosine kinase 1 (sFlt1) may contribute to endothelial dysfunction, hypertension, and proteinuria in preeclampsia. *J Clin Invest*. 2003;111:649–658.
3. Juan P, Stefano G, Antonella S, Albana C. Platelets in pregnancy. *J Prenat Med*. 2011;5:90-92.
4. Brown MA, Magee LA, Kenny LC, et al. Hypertensive Disorders of Pregnancy: ISSHP Classification, Diagnosis, and Management Recommendations for International Practice. *Hypertension*. 2018;72:24-43.
5. American College of Obstetricians and Gynecologists; Task Force on Hypertension in Pregnancy. Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. *Obstet Gynecol*. 2013;122:1122-1131.
6. Reese JA, Peck JD, Deschamps DR, et al. Platelet Counts during Pregnancy. *N Engl J Med*. 2018;379:32-43.
7. Gernsheimer T, James AH, Stasi R. How I treat thrombocytopenia in pregnancy. *Blood*. 2013;121:38-47.
8. Burrows RF, Kelton JG. Thrombocytopenia at delivery: a prospective survey of 6715 deliveries. *Am J Obstet Gynecol*. 1990;162:731-734.
9. Cines DB, Levine LD. Thrombocytopenia in pregnancy. *Blood*. 2017;130: 2271-2277.
10. Makino S, Takeda J, Takeda S, et al. New definition and classification of "Hypertensive Disorders of Pregnancy (HDP)". *Hypertens Res Pregnancy*. 2019;7:1-5.
11. Boehlen F, Hohlfeld P, Extermann P, PernegerTV, de Moerloose P. Platelet count at term pregnancy: a reappraisal of the threshold. *Obstet Gynecol*. 2000;95:29-33.
12. McNutt LA, Wu C, Xue X, Hafner JP. Estimating the relative risk in cohort studies and clinical trials of common outcomes. *Am J Epidemiol*. 2003;157:940-943.

13. Droge LA, Perschel FH, Stutz N, et al. Prediction of Preeclampsia-Related Adverse Outcomes With the sFlt-1 (Soluble fms-Like Tyrosine Kinase 1)/PlGF (Placental Growth Factor)-Ratio in the Clinical Routine: A Real-World Study. *Hypertension*. 2021;77:461-471.
14. Abduljalil K, Furness P, Johnson TN, Rostami-Hodjegan A, Soltani H. Anatomical, physiological and metabolic changes with gestational age during normal pregnancy: a database for parameters required in physiologically based pharmacokinetic modelling. *Clin Pharmacokinet*. 2012;51:365–396.
15. Aster RH. Pooling of platelets in the spleen: role in the pathogenesis of “hypersplenic” thrombocytopenia. *J Clin Invest*. 1966;45:645–657.
16. Maymon R, Strauss S, Vaknin Z, Weinraub Z, Herman A, Gayer G. Normal sonographic values of maternal spleen size throughout pregnancy. *Ultrasound Med Biol*. 2006;32:1827–1831.
17. Jønsson V, Bock JE, Nielsen JB. Significance of plasma skimming and plasma volume expansion. *J Appl Physiol(1985)*. 1992;72:2047–2051.
18. Genbacev O, Joslin R, Damsky CH, Polliotti BM, Fisher SJ. Hypoxia alters early gestation human cytotrophoblast differentiation/invasion in vitro and models the placental defects that occur in preeclampsia. *J Clin Invest*. 1996;97:540-550.
19. Khong TY, De Wolf F, Robertson WB, Brosens I. Inadequate maternal vascular response to placentation in pregnancies complicated by preeclampsia and by small-for-gestational age infants. *Br J Obstet Gynaecol*. 1986;93:1049-1059.
20. Burton GJ, Jauniaux E. Pathophysiology of placental-derived fetal growth restriction. *Am J Obstet Gynecol*. 2018;218:S745-S761.

Tables

Table 1. The criteria of maternal organ damage and utero-placental dysfunction

Liver involvement without underlying disease

AST or ALT >40 IU/L with or without right upper quadrant or epigastric abdominal pain

Acute kidney injury

Serum creatine level ≥ 90 $\mu\text{mol/L}$; 1.0 mg/dL

Neurological complications

Eclampsia, altered mental status, blindness, stroke, clonus, severe headache, or persistent visual scotoma

Blood coagulation disorders

Thrombocytopenia: platelet count $< 150 \times 10^9/\text{L}$, disseminated intravascular coagulation, or hemolysis

Utero-placental dysfunction

Fetal growth restriction^a, abnormal umbilical artery doppler waveform^b, or stillbirth^c

^a Estimated fetal weight < -1.5 standard deviation without chromosomal abnormality and multiple congenital anomaly syndrome

^b Absent or reversal of end diastolic flow, or extremely high pulsatility index or resistance index

^c The cases of stillbirth with chromosomal abnormality or multiple congenital anomaly syndrome are excluded

AST, aspartate transaminase; ALT, alanine transaminase

Table 2. Clinical characteristics based on the overall reduction of PC from the first trimester in women with preeclampsia

	Overall (n = 240)	Decrease (<30.0%) (n = 145)	Decrease (≥30.0%) (n = 95)	p value
Maternal age ^a	34.1 ± 5.1	33.8 ± 5.2	34.5 ± 5.0	0.312
Primipara [†]	153 (63.8)	96 (66.2)	57 (60.0)	0.328
Previous history of preeclampsia ^b	18 (7.5)	11 (7.6)	7 (7.4)	0.950
Low-dose aspirin for prevention ^b	4 (1.7)	2 (1.4)	2 (2.1)	0.667
Severe hypertension ^b	166 (69.2)	94 (64.8)	72 (75.8)	0.072
Uteroplacental dysfunction ^b	67 (27.9)	47 (32.4)	20 (21.1)	0.055
Maternal organ damage except for thrombocytopenia ^b	39 (16.3)	18 (12.4)	21 (22.1)	0.047
Preterm delivery <34 GW [†]	90 (39.5)	44 (30.3)	46 (48.4)	0.005
PC at first trimester ^a (× 10 ⁹ /L)	251 ± 56	249 ± 53	254 ± 59	0.442
PC at delivery ^a (× 10 ⁹ /L)	187 ± 64	219 ± 52	138 ± 47	<0.001
PC levels at delivery ^b				
Normal	174 (72.5)	133 (91.7)	41 (43.2)	<0.001
Mild thrombocytopenia	44 (18.3)	11 (7.6)	33 (34.7)	
Severe thrombocytopenia	22 (9.2)	1 (0.7)	21 (22.1)	

^a Mean ± standard deviation, statistical significance was calculated with independent t-test

^b n (%), statistical significance was calculated with chi-square test

PC, platelet count; GW, gestational weeks

PC levels, Normal, >150 × 10⁹/L; mild thrombocytopenia, < 150 × 10⁹/L and ≥100 × 10⁹/L; severe thrombocytopenia, < 100 × 10⁹/L

Table 3. Relative risk of $\geq 30.0\%$ of overall PC reduction for adverse outcomes of preeclampsia in adjusting maternal age

	RR	95% CI	<i>p</i> value
Severe hypertension	1.16	0.99–1.37	0.073
Uteroplacental dysfunction	0.64	0.41–1.02	0.059
Maternal organ damage except thrombocytopenia	1.77	0.99–3.15	0.052
Preterm delivery <34 GW	1.58	1.14–2.18	0.015

PC, platelet count; GW, gestational week; RR, relative risk; CI, confidence interval

Table 4. Clinical characteristics based on the overall reduction of PC in preeclamptic women without thrombocytopenia

	Overall (n = 174)	Decrease (<30.0%) (n = 133)	Decrease ($\geq 30.0\%$) (n = 41)	<i>p</i> value
Maternal age ^a	34.0 $\pm$ 5.2	34.0 $\pm$ 5.1	34.0 $\pm$ 5.5	0.538
Primipara ^b	110 (63.2)	88 (66.2)	22 (53.7)	0.147
Previous history of preeclampsia ^b	14 (8.1)	9 (6.8)	5 (12.2)	0.264
Low-dose aspirin for prevention ^b	1 (0.6)	1 (0.8)	0	0.578
Severe hypertension ^b	123 (70.7)	88 (66.2)	35 (85.4)	0.018
Uteroplacental dysfunction ^b	49 (28.2)	44 (33.1)	5 (12.2)	0.009
Maternal organ damage except for thrombocytopenia ^b	22 (12.6)	16 (12.0)	6 (14.6)	0.661
Preterm delivery <34 GW ^b	62 (35.6)	41 (30.8)	21 (51.2)	0.017
PC at first trimester ^a ($\times 10^9/L$)	265 $\pm$ 53	256 $\pm$ 50	297 $\pm$ 49	<0.001
PC at delivery ^a ($\times 10^9/L$)	215 $\pm$ 47	226 $\pm$ 47	179 $\pm$ 25	<0.001

^a Mean $\pm$ standard deviation, statistical significance was calculated with independent t-test

^b n (%), statistical significance was calculated with chi-square test

PC, platelet count; GW, gestational weeks

Table 5. Relative risk of $\geq 30.0\%$ of overall PC reduction for adverse outcomes of preeclamptic women without thrombocytopenia in adjusting maternal age

	RR	95% CI	<i>p</i> value
Severe hypertension	1.29	1.08–1.54	0.005
Uteroplacental dysfunction	0.37	0.16–0.87	0.023
Maternal organ damage except thrombocytopenia	1.21	0.50–2.92	0.666
Preterm delivery <34 GW	1.66	1.12–2.46	0.012

PC, platelet count; GW, gestational week; RR, relative risk; CI, confidence interval

Figures

Figure 1

Schematic illustration of the patient selection criteria

Supplementary Files

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- [dataBMC.csv](#)