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Risk Factors of Early and Late Onset Preeclampsia among Thai Women

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Abstract

Background—Little research has been conducted to specifically identify risk factors of early and late onset preeclampsia among Thai women.

Objective—To examine risk factors of early and late onset of preeclampsia among Thai women.

Methods—A case-control study of 150 preeclampsia cases with an equal number of normotensive controls was conducted among women who delivered live born singleton infants at King Chulalongkorn Memorial Hospital, Rajavithi Hospital, and Police General Hospital in Bangkok, Thailand from July 2006 to November 2007. Multivariable logistic regression analysis procedures were used to calculate odds ratios (OR) and 95% confidence intervals (CI) of potential risk factors associated with preeclampsia.

Results—Pre-pregnancy body mass index >30 kg/m2 (OR=5.25, 95%CI: 1.80, 15.32) and failure to use prenatal care services (OR=6.37, 95%CI: 1.26, 32.27) were associated with increased risk of preeclampsia. OR's of similar magnitude were observed when risk factors of early and late onset preeclampsia were assessed separately.

Conclusion—Advanced maternal age, obesity, and no utilization of prenatal care were covariates identified as risk factors for preeclampsia.

Keywords

Epidemiology; hypertension; preeclampsia; pregnancy; risk factors

Introduction

Preeclampsia is a pregnancy-specific hypertensive disorder that significantly affects maternal morbidity and mortality worldwide [1]. It occurs in 5–7% of all pregnancies, and is a leading cause of maternal deaths in developing countries [2,3]. Preeclampsia is also an important determinant of perinatal morbidity and mortality, in part, because of its considerable association with preterm delivery and intrauterine growth restriction (IUGR) [1,3]. Preeclampsia has been the subject of extensive research in recent years. A number of

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studies have identified advanced maternal age, nulliparity, high maternal body mass index (BMI), and pre-existing hypertension or diabetes as likely risk factors. Study results have been consistent across continents for these particular risk factors [4–6]. However, findings have been far less consistent for putative risk factors such as maternal race/ethnicity and socioeconomic status [5,7,8]. Furthermore, little research has been conducted to specifically identify risk factors of the early and late onset preeclampsia among Thai women.

In this study, we sought to identify risk factors of preeclampsia within this Southeast Asian population. This study was undertaken as an initial step towards better understanding the etiology of preeclampsia within a population that is undergoing rapid social, economic, and demographic changes.

Materials and methods

Study population and data collection

A case-control study using one control for each case of preeclampsia was conducted among women who delivered live born singleton infants at King Chulalongkorn Memorial Hospital, Rajavithi Hospital, and Police General Hospital, Bangkok, Thailand between July 2006 and November 2007. Cases were women with a confirmed diagnosis of preeclampsia. Preeclampsia cases were identified by daily monitoring of all new admissions to antepartum, labor and delivery wards of participating hospitals. Of the 158 eligible cases approached, 154 (97.5%) agreed to participate in the study. Controls were women who delivered an infant at term (≥37 weeks of gestation) and who had no evidence of preeclampsia or any hypertensive disorder during pregnancy. Controls were selected from the same hospital of delivery as cases. An eligible control, delivering immediately after a case patient, was approached and recruited for the study. Of the 161 controls approached, 154 (95.7%) agreed to participate in the study.

After excluding women found to have pre-gestational hypertension or diabetes mellitus, a total of 150 preeclampsia cases and 150 controls remained for analysis. All participants provided informed consent. The research protocol was reviewed and approved by the ethical committees of the Faculty of Medicine, Chulalongkorn University, Rajavithi Hospital, Police General Hospital, and the Institutional Review Board, Division of Human Subjects Research, University of Washington.

After obtaining informed consent, enrolled participants were asked to participate in a 45-minute in-person interview in which trained research personnel used a structured questionnaire to elicit information regarding maternal socio-demographic, lifestyle, medical and reproductive characteristics. Participants' labor and delivery medical records and prenatal medical records were also reviewed by trained obstetric research nurses who used a standardized abstraction form. Information abstracted from medical records included participants' pre-pregnancy weight, height, blood pressure, pregnancy complications and condition of the newborn.

Analytical variable specification

Preeclampsia—The diagnosis of preeclampsia was made using the current American College of Obstetricians and Gynecologists (ACOG) guidelines [2]. These guidelines define preeclampsia as sustained pregnancy-induced hypertension with proteinuria. Hypertension was defined as sustained blood pressure readings of >140/90 mmHg (with reading taking place >6 hours apart). ACOG defines proteinuria as urine protein concentrations of >30 mg/dL (or 1+ on a urine dipstick) on two or more random specimens collected > four hours apart. It has recently been suggested that early (<34 completed weeks gestation) and late

(>34 completed weeks gestation) onset preeclampsia may have different etiologies. We therefore categorized preeclampsia cases according to gestational age at onset using the cut-point suggested by von Dadelszen et al. [9].

Other covariates—Covariates considered in this analysis included maternal socio-demographic and behavioral characteristics including maternal age, marital status, educational attainment, employment status, cigarette smoking and alcohol consumption during pregnancy. Also considered were maternal reproductive and medical histories including parity, prior history of hypertensive disorders during pregnancy, history of abortion, maternal height, weight and infant gender. Parity was reported as the number of previous pregnancies lasting more than 22 weeks gestation. Pre-pregnancy body mass index was calculated as weight (in kilograms) divided by the square of height (in meters).

Statistical analysis

Multivariable logistic regression procedures were employed to calculate odd ratios (OR) of potential risk factors associated with preeclampsia. Confidence intervals, at the 95% level were also reported for each unadjusted and adjusted OR. Confounding was assessed by entering potential cofounders into a logistic regression model one at a time, and by comparing the adjusted and unadjusted ORs. Final logistic regression models included covariates that altered unadjusted ORs by at least 10% [10]. We considered the following covariates as possible confounders in these analyses: maternal age, parity, marital status, and maternal educational attainment. Backward logistic regression modeling procedures combined with the change-inestimate approach were used to select the final models reported in this manuscript. Variables of *a priori* interest (e.g., age, parity and pre-pregnancy body mass index) were forced into final models. In order to determine the extent to which the risk factors of preeclampsia are most strongly associated with early onset disease (as cited by von Dadelszen et al.[9]), we repeated analyses for early onset preeclampsia (n=29) and late onset preeclampsia (n=121) cases. All analyses were completed using SPSS, version 16.0 statistical software (SPSS Inc., Chicago, IL).

Results

Socio-demographic and behavioral characteristics of preeclampsia cases (in aggregate and according to early/late onset) and normotensive controls are presented in Table 1. We observed evidence of a strong and statistically significant increased risk of preeclampsia among women who were 30–34 years of age (OR=2.40; 95% CI 1.19–4.83) and ≥35 years of age (OR=2.75; 95% CI 1.35–5.61). Women who were 30–34 years of age (OR=2.23; 95% CI 1.06–4.71) and ≥35 years of age (OR=2.69; 95% CI 1.27–5.70) were more likely to have late onset preeclampsia as compared with women 25–29 years of age. Associations of similar magnitude were observed when analyses were limited to early onset preeclampsia cases. However, these associations did not reach statistical significance. We observed no evidence of associations of preeclampsia with maternal educational attainment, marital status, or employment during pregnancy. Women who reported smoking during pregnancy had a reduced risk of preeclampsia, as compared with non-smokers; however, this association did not reach statistical significance (OR 0.19; 95% CI 0.02–1.69). Maternal alcohol consumption during pregnancy was inversely and statistically significantly associated with preeclampsia risk (OR=0.09; 95% CI 0.01–0.74), although only 11 women in the study population reported consuming alcohol during pregnancy (10 controls, 1 case, respectively). Women who reported exercising during pregnancy had a 23% reduced risk of preeclampsia, though this association did not reach statistical significance. Similar reductions were observed for early (OR=0.58; 95% CI 0.16–2.06) and late onset (OR=0.81; 95% CI 0.42–1.57) preeclampsia.

Table 2 summarizes reproductive and medical history characteristics of preeclampsia cases and normotensive controls. Apparently, women with a prior history of pregnancy-associated hypertensive disorders had a statistically significant increased risk of preeclampsia (OR=10.17, 95%CI: 1.26, 82.02). This association was particularly strong for early onset preeclampsia (OR=21.45, 95%CI: 2.19, 210.57). A family history of hypertension was also associated with increased preeclampsia risk (OR=2.12, 95%CI: 1.06, 4.22). Women with a family history of hypertension had a 2.14-fold increased risk of late onset of preeclampsia (95%CI: 1.04, 4.40). An association of similar magnitude was observed for early onset preeclampsia (OR=2.01, 95%CI: 0.66, 6.09), though because of the small sample size the association did not reach statistical significance.

We observed a positive linear trend of preeclampsia risk in relation to maternal pre-pregnancy BMI (Table 3). Women who were underweight (<18.5 kg/m^2) had a 62% reduced risk of preeclampsia (OR=0.38; 95% CI 0.18–0.81) as compared with their counterparts who had a normal pre-pregnancy BMI (18.5–24.9 kg/m^2). Conversely, obese women (≥30 kg/m^2) had a 4.8-fold increased risk of preeclampsia (OR=4.76; 95% CI 1.73–13.12) compared with women who have a normal pre-pregnancy BMI. Associations of similar directions and magnitudes were observed when analyses were repeated for early and late onset preeclampsia, respectively. We observed a modest, though non-significant increased risk of preeclampsia for women who received no prenatal care during pregnancy (OR=2.66; 95% CI 0.79–8.91). Maternal height and infant gender were not associated with increased preeclampsia risk.

Results from three multivariable logistic regression models are summarized in Table 4. Patterns of associations were similar regardless of whether we assessed preeclampsia in aggregate or stratified into early and late onset subgroups. However, adjusted ORs were less precise (as reflected by relatively wider 95% CIs) for subgroup analyses of early onset preeclampsia. For all models, young (<20 years) and advanced (30–34 years and ≥35 years) maternal age were risk factors of preeclampsia. Also, a significant linear trend between pre-pregnancy BMI and risk of preeclampsia was observed for all subcategories of preeclampsia. Women who did not receive prenatal care during the index pregnancy had 6.37-fold increased risk of preeclampsia as compared with those women who initiated prenatal care during the first trimester (95% CI 1.26–32.27). An association of similar magnitude was observed for late onset preeclampsia cases (OR=7.19; 95% CI 1.36–38.11). Prior history of hypertensive disorders of pregnancy (OR=7.83; 95% CI 0.88–69.44) and a positive family history of hypertension (OR=1.92; 95% CI 0.87–4.23) were positively associated with preeclampsia risk, though these covariates did not reach statistical significance.

Discussion

This case-control study indicates that advanced maternal age, pre-pregnancy obesity status, and no utilization of prenatal care are associated with a significantly increased risk of preeclampsia in Thai women. These results are concordant with findings from earlier studies that evaluate the risk factors of preeclampsia in other populations [4,5,11,12]. Moreover, results from our analyses of early and late onset preeclampsia case groups support the position held by Huppertz [3] who recently noted no clear difference between the early and late onset preeclampsia.

Maternal age at extremes (<20 and >40 years) was identified as a risk factor of preeclampsia in a Saudi Arabian population [13]. Advanced maternal age (>35 years) had also been shown in other populations to be associated with increased risk of preeclampsia [4]. Similar patterns of risk were observed in our study of Thai women, where young (<20 years) and

older (>30 years) women were at an increased risk of preeclampsia regardless of whether the condition was classified as early or late onset.

Obesity is perhaps the most consistently reported modifiable risk factor of preeclampsia [4,11,14–17]. Mahomed et al. reported that Zimbabwean women with preeclampsia had a higher BMI (27.6±4.4 kg/m2) compared with normotensive women (25.2±3.8 kg/m2) [16]. Mittendorf et al. reported a 2.7-fold increased risk of preeclampsia in obese women compared to women with a pre-pregnancy BMI between 18–30 kg/m2 [11]. We observed a statistically significant linear trend of increased preeclampsia risk with increasing BMI. Similar linear trends have been reported by other investigators [16–18]. Although precise biologic mechanisms for associations of preeclampsia risk with maternal adiposity are unknown, several possible mechanisms have been proposed. Investigators have speculated that hyperlipidemia, oxidative stress and diffuse endothelial dysfunction are more common among obese versus lean pregnant women [17–19]. Others have postulated that the association is secondary to alterations in cardiac output [20]. Although compelling, these hypothesized mechanisms have yet to be evaluated in carefully-designed cohort studies of pregnant women.

Positive personal histories of hypertensive disorders of pregnancy [12,14,15] and family history of hypertensive disorders [21,22] are well known risk factors of preeclampsia. Preeclampsia is reported to recur in 13–18% of subsequent pregnancies [23,24]. In accordance with previous studies [12,14,15], we observed increased preeclampsia risk among women with a prior history of pregnancies complicated by hypertensive disorders. Odegard et al. [12], in their study of Norwegian women, reported that those with a prior history of preeclampsia, compared with women without such a history, had a 21.5-fold increased risk of the disorder in another pregnancy (95%CI: 9.8,47.2). The high recurrence risk of preeclampsia, and evidence of familial aggregation of hypertensive disorders [22] supports the concept that a subgroup of women may be predisposed to developing this very dangerous complication of pregnancy.

Nulliparity has been identified as a risk factor for preeclampsia in several populations [4,5,11,12]. However, we observed only a weak positive association of preeclampsia risk with this covariate. Additionally, although numerous authors have observed that smoking confers a decreased risk of preeclampsia [4,5,11,12,25], we did not identify maternal cigarette smoking during pregnancy as a statistically significant preeclampsia risk factor in the present study. The low frequency of mothers who reported smoking during pregnancy prohibited careful evaluation of this covariate among Thai women enrolled in our study.

von Dadelszen et al. [9] and Ilekis et al. [26] have recently supported the concept that preeclampsia is a heterogeneous disorder to be subdivided into the early and late onset case-groups. Using the criteria proposed by von Dadelszen et al. [9], we classified preeclampsia cases into the early and late onset subgroups and sought to empirically explore the possibility of heterogeneity in risk factors for the two groups of preeclampsia cases. Consistent with prior reports [3], we noted that the vast majority of preeclampsia cases (81%) were classified as the late onset cases while 19% were classified as early onset cases. Consistent with the literature summarized by Huppertz [3], we noted little evidence to support the thesis that there may be substantial differences in the epidemiology of the early and late onset preeclampsia. Although it would be highly desirable to define homogeneous subgroups of preeclampsia, great care must be taken to avoid introducing non-biological factors (e.g., health services factors such as variations in maternal utilization patterns of prenatal care or variations in clinical management protocols of the disorder).

In the present study, we were able to empirically assess risk factors of clinical subtypes of preeclampsia, using well-trained personnel to collect information from all participants. All interviewers were blinded to participants' case-control status and a well structured questionnaire was used to systematically collect risk factor information. The high participation rates for cases and controls (97.5% and 95.7%) also served to attenuate concerns about the selection bias. However, a number of limitations should be considered when interpreting results from our study. First, our study was limited in evaluation of several risk factors previously reported to influence the risk of preeclampsia. In particular, we did not have information pertaining to maternal mood and anxiety disorders [27], migraine disorders [28] or maternal dietary habits before and during pregnancy [29]. Second, the low prevalence of certain maternal risk factors (i.e., cigarette smoking and alcohol consumption during pregnancy) among women limited our estimation of precise odds ratios for these covariates. Third, since we studied women residing in metropolitan Bangkok, our observations may not be generalizable to Thai women residing in rural settings. Nevertheless, our findings are largely consistent with those reported from other more diverse populations. Lastly, the case-control study design does not allow us to dismiss the possibility of recall bias.

Our findings and those by other investigators point to public health and clinical measures that may be taken to potentially attenuate the incidence of preeclampsia and mitigate associated maternal-fetal complications resulting from the disorder.

In conclusion, advanced maternal age, obesity, and no utilization of prenatal care were covariates identified as risk factors for preeclampsia.

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References

1. Sibai B, Dekker G, Kupferminc M. Pre-eclampsia. Lancet. 2005; 365:785–99. [PubMed: 15733721]
2. ACOG practice bulletin. Diagnosis and management of preeclampsia and eclampsia. Obstet Gynecol. 2002; 99:159–67. [PubMed: 16175681]
3. Huppertz B. Placental origins of preeclampsia: challenging the current hypothesis. Hypertension. 2008; 51:970–5. [PubMed: 18259009]
4. Conde-Agudelo A, Belizan JM. Risk factors for preeclampsia in a large cohort of Latin American and Caribbean women. BJOG. 2000; 107:75–83. [PubMed: 10645865]
5. Jacobs DJ, Vreeburg SA, Dekker GA, Heard AR, Priest KR, Chan A. Risk factors for hypertension during pregnancy in South Australia. Aust N Z J Obstet Gynaecol. 2003; 43:421–8. [PubMed: 14712944]
6. Sun Y, Yang H, Sun WJ. Risk factors for pre-eclampsia in pregnant Chinese women with abnormal glucose metabolism. Int J Gynaecol Obstet. 2008; 101:74–6. [PubMed: 18082749]
7. Haelterman E, Qvist R, Barlow P, Alexander S. Social deprivation and poor access to care as risk factors for severe pre-eclampsia. Eur J Obstet Gynecol Reprod Biol. 2003; 111:25–32. [PubMed: 14557007]
8. Irwin DE, Savitz DA, Hertz-Picciotto I, St Andre KA. The risk of pregnancy-induced hypertension: black and white differences in a military population. Am J Public Health. 1994; 84:1508–10. [PubMed: 8092384]

9. von Dadelszen P, Magee LA, Roberts JM. Subclassification of preeclampsia. Hypertens Pregnancy. 2003; 22:143–8. [PubMed: 12908998]
10. Rothman, KJ.; Greenland, S. Modern epidemiology. 2. Philadelphia, PA: Lippincott-Raven; 1998. p. 255-9.
11. Mittendorf R, Lain KY, Williams MA, Walker CK. Preeclampsia. A nested, case-control study of risk factors and their interactions. J Reprod Med. 1996; 41:491–6. [PubMed: 8829061]
12. Odegard RA, Vatten LJ, Nilsen ST, Salvesen KA, Austgulen R. Risk factors and clinical manifestations of pre-eclampsia. BJOG. 2000; 107:1410–6. [PubMed: 11117771]
13. Al-Mulhim AA, Abu-Heija A, Al-Jamma F, El-Harith el HA. Pre-eclampsia: maternal risk factors and perinatal outcome. Fetal Diagn Ther. 2003; 18:275–80. [PubMed: 12835589]
14. Anorlu RI, Iwuala NC, Odum CU. Risk factors for preeclampsia in Lagos, Nigeria. Aust N Z J Obstet Gynaecol. 2005; 45:278–82. [PubMed: 16029292]
15. Catov JM, Ness RB, Kip KE, Olsen J. Risk of early or severe pre-eclampsia related to pre-existing conditions. Int J Epidemiol. 2007; 36:412–9. [PubMed: 17255351]
16. Mahomed K, Williams MA, Woelk GB, Jenkins-Woelk L, Mudzamiri S, Longstaff L, et al. Risk factors for preeclampsia among Zimbabwean women: maternal arm circumference and other anthropometric measures of obesity. Paediatr Perinat Epidemiol. 1998; 12:253–62. [PubMed: 9690261]
17. Stone JL, Lockwood CJ, Berkowitz GS, Alvarez M, Lapinski R, Berkowitz RL. Risk factors for severe preeclampsia. Obstet Gynecol. 1994; 83:357–61. [PubMed: 8127525]
18. Frederick IO, Rudra CB, Miller RS, Foster JC, Williams MA. Adult weight change, weight cycling, and prepregnancy obesity in relation to risk of preeclampsia. Epidemiology. 2006; 17:428–34. [PubMed: 16755262]
19. Kaaja R, Tikkanen MJ, Viinikka L, Ylikorkala O. Serum lipoproteins, insulin, and urinary prostanoid metabolites in normal and hypertensive pregnant women. Obstet Gynecol. 1995; 85:353–6. [PubMed: 7862371]
20. Paulson DJ, Tahiliani AG. Cardiovascular abnormalities associated with human and rodent obesity. Life Sci. 1992; 51:1557–69. [PubMed: 1435063]
21. Mahomed K, Williams MA, Woelk GB, Jenkins-Woelk L, Mudzamiri S, Madzime S, et al. Risk factors for preeclampsia-eclampsia among Zimbabwean women: recurrence risk and familial tendency towards hypertension. J Obstet Gynaecol. 1998; 18:218–22. [PubMed: 15512062]
22. Qiu C, Williams MA, Leisenring WM, Sorensen TK, Frederick IO, Dempsey JC, et al. Family history of hypertension and type 2 diabetes in relation to preeclampsia risk. Hypertension. 2003; 41:408–13. [PubMed: 12623936]
23. Ananth CV, Peltier MR, Chavez MR, Kirby RS, Getahun D, Vintzileos AM. Recurrence of ischemic placental disease. Obstet Gynecol. 2007; 110:128–33. [PubMed: 17601907]
24. Mostello D, Kallogjeri D, Tungsiripat R, Leet T. Recurrence of preeclampsia: effects of gestational age at delivery of the first pregnancy, body mass index, paternity, and interval between births. Am J Obstet Gynecol. 2008; 199:55, e51–57. [PubMed: 18280450]
25. Marcoux S, Brisson J, Fabia J. The effect of cigarette smoking on the risk of preeclampsia and gestational hypertension. Am J Epidemiol. 1989; 130:950–7. [PubMed: 2816902]
26. Ilekis JV, Reddy UM, Roberts JM. Preeclampsia-a pressing problem: an executive summary of a National Institute of Child Health and Human Development workshop. Reprod Sci. 2007; 14:508–23. [PubMed: 17959880]
27. Qiu C, Sanchez SE, Lam N, Garcia P, Williams MA. Associations of depression and depressive symptoms with preeclampsia: results from a Peruvian case-control study. BMC Womens Health. 2007; 7:15. [PubMed: 17900360]
28. Sanchez SE, Qiu C, Williams MA, Lam N, Sorensen TK. Headaches and migraines are associated with an increased risk of preeclampsia in Peruvian women. Am J Hypertens. 2008; 21:360–4. [PubMed: 18202669]
29. Qiu C, Coughlin KB, Frederick IO, Sorensen TK, Williams MA. Dietary fiber intake in early pregnancy and risk of subsequent preeclampsia. Am J Hypertens. 2008; 21:903–9. [PubMed: 18636070]

Table 1

Odds ratios (OR) and 95% confidence intervals (CI) for socio-demographic and behavioral characteristics of preeclampsia cases and normotensive controls, Bangkok, Thailand, 2006–2007.

	Controls (n = 150)		Preeclampsia (n = 150)			Early Onset Preeclampsia (n = 29)		Late Onset Preeclampsia (n = 121)	
Covariates	**n**	**%**	**n**	**%**	**Unadjusted OR (95% CI)**	**n**	**OR (95% CI)**	**n**	**OR (95% CI)**
Maternal age (years)									
<20	11	7.3	14	9.3	1.91 (0.78, 4.67)	3	2.10 (0.47, 9.43)	11	1.86 (0.72, 4.81)
20–24	47	31.3	35	23.3	1.12 (0.61, 2.05)	4	0.66 (0.18, 2.38)	31	1.23 (0.65, 2.33)
25–29	54	36.0	36	24.0	1.00 (Reference)	7	1.00 (Reference)	29	1.00 (Reference)
30–34	20	13.3	32	21.3	2.40 (1.19, 4.83)	8	3.09 (0.99, 9.62)	24	2.23 (1.06, 4.71)
≥35	18	12.0	33	22.0	2.75 (1.35, 5.61)	7	3.00 (0.93, 9.72)	26	2.69 (1.27, 5.70)
Maternal education (years)									
≤6	45	30.0	55	36.7	0.79 (0.34, 1.86)	7	0.34 (0.09, 1.29)	48	0.98 (0.39, 2.44)
7–12	94	62.7	78	52.0	0.54 (0.24, 1.21)	17	0.40 (0.12, 1.29)	61	0.59 (0.25, 1.43)
>12	11	7.3	17	11.3	1.00 (Reference)	5	1.00 (Reference)	12	1.00 (Reference)
Marital status									
Married	76	50.7	84	56.0	1.00 (Reference)	14	1.00 (Reference)	70	1.00 (Reference)
Unmarried	69	46.0	61	40.7	0.80 (0.50, 1.27)	15	1.18 (0.53, 2.62)	46	0.72 (0.44, 1.19)
Separated	5	3.3	5	3.3	0.90 (0.25, 3.25)	0	-	5	1.09 (0.30, 3.91)
Smoked in pregnancy									
No	145	96.7	149	99.3	1.00 (Reference)	28	1.00 (Reference)	121	1.00 (Reference)
Yes	5	3.3	1	0.7	0.19 (0.02, 1.69)	1	1.04 (0.12, 9.21)	0	-
Alcohol use in pregnancy									
No	140	93.3	149	99.3	1.00 (Reference)	29	1.00 (Reference)	120	1.00 (Reference)
Yes	10	6.7	1	0.7	0.09 (0.01, 0.74)	0	-	1	0.12 (0.01, 0.92)
Employed during pregnancy									
No	46	30.7	55	36.7	1.31 (0.81, 2.12)	6	0.59 (0.23, 1.55)	49	1.54 (0.93, 2.54)
Yes	104	69.3	95	63.3	1.00 (Reference)	23	1.00 (Reference)	72	1.00 (Reference)
Exercise during pregnancy									
No	120	82.2	126	85.7	1.00 (Reference)	24	1.00 (Reference)	102	1.00 (Reference)
Yes	26	17.8	21	14.3	0.77 (0.41, 1.44)	3	0.58 (0.16, 2.06)	18	0.81 (0.42, 1.57)

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Table 2

Odds ratios (OR) and 95% confidence intervals (CI) for reproductive and medical history characteristics of preeclampsia cases and normotensive controls, Bangkok, Thailand, 2006–2007.

	Controls (n = 150)		Preeclampsia (n = 150)			Early Onset Preeclampsia (n = 29)		Late Onset Preeclampsia (n = 121)	
Covariates	**n**	**%**	**n**	**%**	**Unadjusted OR (95% CI)**	**n**	**OR (95% CI)**	**n**	**OR (95% CI)**
Parity									
Nulliparous	90	60.0	82	54.7	0.80 (0.51, 1.27)	14	0.62 (0.28, 1.38)	68	0.86 (0.53, 1.39)
Multiparous	60	40.0	68	45.3	1.00 (Reference)	15	1.00 (Reference)	53	1.00 (Reference)
History of hypertensive disorders during pregnancy									
Nulliparous	90	60.0	82	54.7	0.93 (0.58, 1.48)	14	0.83 (0.35, 1.96)	68	0.95 (0.58, 1.56)
Parous-no prior history	59	39.3	58	38.7	1.00 (Reference)	11	1.00 (Reference)	47	1.00 (Reference)
Parous-prior history	1	0.7	10	6.7	10.17 (1.26, 82.02)	4	21.45 (2.19, 210.57)	6	7.53 (0.88, 64.75)
Number of previous abortion									
0	113	75.3	115	76.7	1.00 (Reference)	21	1.00 (Reference)	94	1.00 (Reference)
1	32	21.3	31	20.7	0.95 (0.54, 1.66)	7	1.18 (0.46, 3.02)	24	0.90 (0.50, 1.64)
≥2	5	3.3	4	2.7	0.79 (0.21, 3.00)	1	1.08 (0.12, 9.68)	3	0.72 (0.17, 3.10)
Family history of hypertension									
No	135	90.0	123	82.0	1.00 (Reference)	24	1.00 (Reference)	99	1.00 (Reference)
Yes	14	9.3	27	18.0	2.12 (1.06, 4.22)	5	2.01 (0.66, 6.09)	22	2.14 (1.04, 4.40)
Missing	1	0.7	0	0.0		0		0	

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Table 3

Odds ratios (OR) and 95% confidence intervals (CI) for current pregnancy characteristics of preeclampsia cases and normotensive controls, Bangkok, Thailand, 2006–2007.

	Controls (n = 150)		Preeclampsia (n = 150)			Early Onset Preeclampsia (n = 29)		Late Onset Preeclampsia (n = 121)	
Covariates	**n**	**%**	**n**	**%**	**Unadjusted OR (95% CI)**	**n**	**OR (95% CI)**	**n**	**OR (95% CI)**
Pre-pregnancy BMI (kg/m^2)									
<18.5	31	20.7	11	7.3	0.38 (0.18, 0.81)	2	0.38 (0.08, 1.72)	9	0.39 (0.17, 0.86)
18.5–24.9	93	62.0	86	57.3	1.00 (Reference)	16	1.00 (Reference)	70	1.00 (Reference)
25–29.9	16	10.7	25	16.7	1.69 (0.85, 3.38)	7	2.54 (0.90, 7.16)	18	1.49 (0.71, 3.14)
≥30.0	5	3.3	22	14.7	4.76 (1.73, 13.12)	3	3.49 (0.76, 16.05)	19	5.05 (1.80, 14.18)
Missing	5	3.3	6	4.0		1		5	
Maternal height (m)									
Low stature (≤145 cm)	3	2.0	2	1.3	0.65 (0.11, 3.97)	0	-	2	0.81 (0.13, 4.94)
Normal (>145 cm)	145	96.7	148	98.7	1.00 (Reference)	29	1.00 (Reference)	119	1.00 (Reference)
Missing	2	1.3	0	0.0		0		0	
Prenatal care onset									
Care initiated in 1st trimester	67	44.7	63	42.0	1.00 (Reference)	15	1.00 (Reference)	48	1.00 (Reference)
Care initiated after 1st trimester	79	52.7	77	51.3	1.04 (0.65, 1.65)	13	0.74 (0.33, 1.65)	64	1.13 (0.69, 1.86)
No prenatal care	4	2.7	10	6.7	2.66 (0.79, 8.91)	1	1.12 (0.12, 10.72)	9	3.14 (0.91, 10.80)
Infant gender									
Female	68	45.3	76	50.7	1.00 (Reference)	17	1.00 (Reference)	59	1.00 (Reference)
Male	82	54.7	74	49.3	0.81 (0.51, 1.27)	12	0.59 (0.26, 1.31)	62	0.87 (0.54, 1.41)

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Table 4

Adjusted odds ratios (OR) and 95% confidence intervals (CI) according to selected factors, Bangkok, Thailand, 2006–2007.

	All Preeclampsia (n = 150)	Early Onset Preeclampsia (n = 29)	Late Onset Preeclampsia (n = 121)
Covariates	**Adjusted OR* (95% CI)**	**Adjusted OR* (95% CI)**	**Adjusted OR* (95% CI)**
Maternal age (years)			
<20	1.99 (0.70, 5.68)	1.80 (0.27, 12.01)	1.94 (0.64, 5.86)
20–24	1.11 (0.57, 2.18)	0.67 (0.17, 2.70)	1.13 (0.56, 2.27)
25–29	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
30–34	2.30 (1.03, 5.17)	1.55 (0.38, 6.33)	2.23 (0.97, 5.13)
≥35	2.04 (0.89, 4.67)	1.87 (0.45, 7.74)	1.99 (0.85, 4.68)
Trend test p-value	*0.229*	*0.314*	*0.249*
Alcohol use in pregnancy	0.10 (0.01, 1.06)	-	-
Smoked in pregnancy	0.14 (0.01, 2.21)	-	-
Exercise during pregnancy	-	0.55 (0.11, 2.76)	-
Family history of hypertension	1.92 (0.87, 4.23)	-	1.83 (0.83, 4.03)
History of hypertensive disorders during pregnancy			
Nulliparous	1.31 (0.71, 2.39)	0.98 (0.34, 2.80)	1.45 (0.78, 2.70)
Parous-no prior history	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Parous-prior history	7.83 (0.88, 69.44)	24.30 (2.05, 287.87)	4.77 (0.50, 45.34)
Pre-pregnancy BMI (kg/m^2)			
<18.5	0.31 (0.13, 0.73)	0.38 (0.07, 2.24)	0.29 (0.12, 0.73)
18.5–24.9	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
25–29.9	1.71 (0.81, 3.58)	2.70 (0.82, 8.91)	1.64 (0.75, 3.57)
≥30.0	5.25 (1.80, 15.32)	4.07 (0.81, 20.42)	5.33 (1.83, 15.51)
Trend test p-value	*<0.001*	*0.009*	*<0.001*
Prenatal care onset/utilization			
Care initiated in 1st trimester	1.00 (Reference)	-	1.00 (Reference)
Care initiated after 1st trimester	0.99 (0.57, 1.70)	-	1.04 (0.59, 1.84)
No prenatal care	6.37 (1.26, 32.27)	-	7.19 (1.36, 38.11)

*Each column represents a different logistic regression model. Covariates not included in final models are indicated as (-). Each OR and 95% CI is adjusted for all other covariates listed in this table.

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