
OBSTETRICS

Preeclampsia and Pregnancy outcome in Pregnant Women with Gestational Hypertension at Chonburi Hospital

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ABSTRACT

Objectives: To determine the incidence of preeclampsia (PE) and pregnancy outcome in women diagnosed with gestational hypertension (GH) at Chonburi Hospital.

Materials and Methods: This combined retrospective-prospective cohort study collected the data of pregnant women diagnosed GH between January 2019 and April 2025. Pregnant women with chronic hypertension, abortion, or multifetal gestation were excluded. The data predicting progression from GH to PE were analyzed by logistic regression analysis.

Results: 238 pregnancies with GH were enrolled and followed until delivery. 12 of these (5.0%) lost to follow-up or delivery at other hospitals. 58 pregnant women (25.7%, 95% confidence interval (CI) 20.10, 31.88%) developed preeclampsia, and 38 of these cases (16.8%, 95% CI 8.39, 17.41%) were classified as having severe features. Logistic regression reveals that the patient diagnosed with GH before 34 weeks had a higher risk of developing PE (adjusted odds ratio (OR) 3.30, 95% CI 1.65, 6.61). Additionally, women with diastolic blood pressure ≥ 100 mmHg at diagnosis also had a higher risk of PE progression (adjusted OR 3.16, 95% CI 1.58, 6.34). The pregnant women with PE progression had higher rate of cesarean section (relative risk (RR) 1.69, 95% CI 1.44, 1.98), early preterm birth (RR 8.69, 95% CI 1.80, 41.86), late preterm birth (RR 5.07, 95% CI 2.66, 9.65) and low birth weight (RR 4.34, 95% CI 2.37, 7.97).

Conclusion: PE progression occurred for a quarter number in GH pregnant women. The risks of PE progression are early gestational age of GH diagnosis and high diastolic blood pressure at diagnosis. Adverse pregnancy outcomes in preeclampsia women are preterm delivery and high cesarean section rate.

Keywords: gestational hypertension, preeclampsia, pregnancy induced hypertension, hypertensive disorder of pregnancy, diastolic blood pressure, preterm delivery, cesarean section.

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อุบัติการณ์การเกิดภาวะครรภ์เป็นพิษในหญิงตั้งครรภ์ที่มีภาวะความดันโลหิตสูงขณะตั้งครรภ์ในโรงพยาบาลชลบุรี

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บทคัดย่อ

วัตถุประสงค์: เพื่อศึกษาอุบัติการณ์การเกิดภาวะครรภ์เป็นพิษและครรภ์เป็นพิษรุนแรงในหญิงตั้งครรภ์ที่มีภาวะความดันโลหิตสูงขณะตั้งครรภ์ในโรงพยาบาลชลบุรี

วัสดุและวิธีการ: เป็นการศึกษาเชิงพรรณนาแบบย้อนหลังโดยศึกษาจากเวชระเบียนของโรงพยาบาลชลบุรี ร่วมกับการศึกษาเชิงพรรณนาแบบไปข้างหน้า รวบรวมผู้ป่วยที่วินิจฉัยภาวะความดันโลหิตสูงขณะตั้งครรภ์ในช่วงเดือนมกราคม พ.ศ. 2562 - เดือนเมษายน พ.ศ. 2568 โดยตัดกลุ่มผู้ป่วยที่เคยได้รับการวินิจฉัยว่าเป็นความดันโลหิตสูง ผู้ที่มีภาวะแท้ง หรือมีภาวะครรภ์แฝดออกจากการศึกษา ข้อมูลในเรื่องของปัจจัยต่างๆที่อาจส่งผลต่อการเกิดภาวะครรภ์เป็นพิษในหญิงตั้งครรภ์ที่มีภาวะความดันโลหิตสูงถูกวิเคราะห์เพิ่มเติมโดยใช้การวิเคราะห์การถดถอยโลจิสติก

ผลการศึกษา: จากจำนวนหญิงตั้งครรภ์ที่ได้รับการวินิจฉัยเรื่องของภาวะความดันโลหิตสูงขณะตั้งครรภ์ทั้งหมด 226 ราย พบว่ามีการเกิดภาวะครรภ์เป็นพิษ 58 ราย (ร้อยละ 25.7, 95% CI 20.10, 31.88) โดย 38 เคสจากกลุ่มผู้ป่วยข้างต้นเกิดภาวะครรภ์เป็นพิษแบบรุนแรง (ร้อยละ 16.8, 95% CI 8.39, 17.41) จากการวิเคราะห์การถดถอยโลจิสติกพบว่าปัจจัยที่เกี่ยวข้องกับการเกิดภาวะครรภ์เป็นพิษอย่างมีนัยสำคัญมีด้วยกัน 2 ตัวแปรคืออายุครรภ์ที่วินิจฉัยภาวะความดันโลหิตสูงขณะตั้งครรภ์และค่าความดันโลหิตตัวล่างในวันที่วินิจฉัย ซึ่งพบว่าในผู้ป่วยที่วินิจฉัยภาวะความดันโลหิตสูงขณะตั้งครรภ์ก่อนอายุครรภ์ 34 สัปดาห์จะเพิ่มความเสี่ยงในการเกิดภาวะครรภ์เป็นพิษ (adjusted odds ratio (OR) 2.79, 95% CI 1.40, 5.54) และในกลุ่มผู้ป่วยที่มีค่าความดันโลหิตตัวล่างตอนวินิจฉัย ≥ 100 มิลลิเมตรปรอทจะเพิ่มความเสี่ยงต่อการเกิดภาวะครรภ์เป็นพิษเช่นกัน (adjusted OR 2.91, 95% CI 1.24, 6.82) โดยผู้ป่วยกลุ่มที่พัฒนาไปสู่ภาวะเป็นครรภ์เป็นพิษจะพบว่าจะมีอัตราการผ่าคลอดที่สูงกว่า (relative risk (RR) 1.69, 95% CI 1.44, 1.98), อัตราคลอดก่อนกำหนดก่อน 34 สัปดาห์สูงกว่า (RR 8.69, 95% CI 1.80, 41.86), อัตราคลอดก่อนกำหนดตั้งแต่ 34 สัปดาห์เป็นต้นไปสูงกว่า (RR 5.07, 95% CI 2.66, 9.65) และอัตราคลอดบุตรน้ำหนักตัวน้อยสูงกว่ากลุ่มที่ไม่ได้พัฒนาสู่ภาวะครรภ์เป็นพิษ (RR 4.34, 95% CI 2.37, 7.97) **สรุป:** หญิงตั้งครรภ์ที่มีภาวะความดันโลหิตสูงระหว่างตั้งครรภ์พบว่ามีกว่า 1 ใน 4 ที่พัฒนาไปสู่ภาวะครรภ์เป็นพิษ ซึ่งจากงานวิจัยนี้พบว่าปัจจัยที่เกี่ยวข้องนั้นจะเป็นเรื่องของการวินิจฉัยภาวะความดันโลหิตสูงขณะตั้งครรภ์ตั้งแต่อายุครรภ์น้อยกว่า 34 สัปดาห์ และความดันโลหิตตัวล่างตอนวินิจฉัย ≥ 100 มิลลิเมตรปรอท

คำสำคัญ: ภาวะความดันโลหิตสูงขณะตั้งครรภ์, ภาวะครรภ์เป็นพิษ, ภาวะความดันโลหิตสูงที่เกิดจากการตั้งครรภ์, กลุ่มโรคความดันโลหิตสูงในระหว่างตั้งครรภ์, ความดันโลหิตตัวล่าง, การคลอดก่อนกำหนด, การผ่าตัดคลอด

Introduction

Hypertensive disorders of pregnancy (HDP) are a significant global health concern, with an incidence of up to 10%⁽¹⁾ and rising continuously. Global statistics show that the incidence of HDP increased from 16.3 million cases per year in 1990 to 18.1 million cases per year in 2010. That's rising up to 10.9% over two decades⁽²⁾. A study by Urquia found that the incidence of HDP varies across different ethnicities, weight, and behavioral factors⁽³⁾. In Thailand, the incidence of HDP is reported as 27.5 per 1,000 pregnant women⁽⁴⁾. Data from the Thai Ministry of Public Health in 2018 reported that there were 15 maternal deaths per 100,000 live births during pregnancy, labor, and postpartum that were caused by HDP, accounting for 29% of direct maternal deaths⁽⁵⁾.

The exact cause of HDP is still unknown, but several hypotheses have been proposed, including chronic placental ischemia, genetic predisposition, and the maternal immune response to placental cells in the bloodstream. It is likely a multifactorial condition⁽⁶⁾. According to the American College of Obstetricians and Gynecologists (ACOG) 2020 classification, HDP is divided into four main categories⁽⁶⁾ including gestational hypertension, chronic hypertension, preeclampsia-eclampsia, and chronic hypertension with superimposed preeclampsia. Globally, the incidence of these conditions in pregnant women is 2-17%, 3-5%, 5-8%, and 0.75-1.25%, respectively^(1, 7).

Gestational hypertension (GH) is generally considered the mildest form of HDP. Typically, it does not require treatment, but close monitoring of maternal blood pressure and clinical symptoms are required. Blood pressure usually returns to normal within six weeks after delivery. If blood pressure does not reach the severe range ($\geq 160/110$ mmHg), fetal outcomes are not significantly different from normal pregnancy⁽⁸⁾. However, women with GH are at a 3-5 times higher risk of developing preeclampsia or eclampsia compared to the general pregnant population^(1, 9). This progression can lead to severe maternal and fetal morbidity and mortality. Therefore, early identifying

and preventing risk factors of the progression from GH to preeclampsia could improve pregnancy outcomes.

Several research studies have focused on this progression. In Northern Ethiopia, the GH incidence was 6%. Among women with GH, 17.1% developed preeclampsia⁽⁹⁾. Similarly, the study from Australia showed that 15-25% progression rate from GH to preeclampsia⁽¹⁰⁾. While a prospective cohort study in Taiwan showed that the GH incidence was 5.4%. Up to 42.1% of GH developed preeclampsia⁽¹¹⁾. This is consistent with a study from the U.S. which found that 46% of GH pregnancies progressed to preeclampsia, with 9.6% of these cases being severe⁽¹²⁾.

The previous studies revealed several predictive risk factors associated with the progression of GH to preeclampsia such as maternal age, body mass index (BMI), parity, history of miscarriage, excessive maternal weight gain during pregnancy, and grade III preterm placental calcification (PPC) from ultrasound⁽⁹⁻¹²⁾.

Due to the diversity of previous evidence about the progression rate from GH to preeclampsia and there is lacking data in Thai population. Our study focused on the incidence of progression from GH to preeclampsia in Chonburi Hospital. The data of suspected factors associated with preeclampsia progression such as baseline characteristics at the time of presentation and pregnancy outcomes also had been collected and analyzed.

Materials and Methods

This combined retrospective and prospective cohort study was conducted at Chonburi Hospital, Tertiary public hospital of Eastern Thailand. This study included women diagnosed GH in antenatal care clinic between January 2019 and April 2025. The sample size was calculated using Cochran's formula with the incidence of 17% from previous literature review, a margin of error of 0.05, and a 95% confidence level. The required sample size was 217 participants. To mitigate the risk of subject attrition, the initial sample size of 217 was inflated by 10%, establishing a final

requirement of 239 participants. The study was approved by the local Institutional Review Board of the hospital.

GH was defined as the new onset of hypertension (systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg) after 20 weeks of gestation without the presence of proteinuria or end-organ damage. Preeclampsia without severe features was defined as the new onset of hypertension after 20 weeks of gestation with the presence of proteinuria. In this study, a urinary protein-to-creatinine ratio (UPCR) of ≥ 0.3 was used to assess for proteinuria. New onset of hypertension detected after 20 weeks of gestation was classified as preeclampsia with severe features if any clinical sign of end-organ damage was present, including; SBP ≥ 160 mmHg, DBP ≥ 110 mmHg, Thrombocytopenia (platelet count $< 100,000$ cell/mm³), renal insufficiency (creatinine ≥ 1.1 mg/dL or a doubling of serum creatinine baseline), impair liver function (an aspartate aminotransferase / alanine aminotransferase rising ≥ 2 x of normal concentration), pulmonary edema, and new-onset persistent headache or visual disturbances⁽⁶⁾.

Exclusion criteria included a history of essential hypertension or secondary hypertension due to their known underlying diseases (e.g. hyperthyroid, pheochromocytoma, known case chronic kidney disease; creatinine > 1.1 mg/dl), multiple gestation, and termination of pregnancy before 24 weeks. Moreover, patients who did not deliver at Chonburi Hospital or had incomplete data about the onset of hypertension and laboratory test were excluded.

Clinical and laboratory data were recorded at the time of presentation. These data included patient age, body weight (both prepregnancy and at delivery), BMI, blood pressure at diagnosis, history of HDP, and history of abortion. The patient's diabetes mellitus (DM) status, including overt DM and gestational diabetes mellitus (GDM), and aspirin prescription during this pregnancy were also documented. Laboratory data consisting of complete blood count, creatinine, aspartate aminotransferase, alanine

aminotransferase, UPCR, and international normalized ratio, were also collected. Excessive weight gain was defined according to prepregnancy BMI⁽¹⁾.

In our study, patients who were at moderate to high risk of preeclampsia, according to the American College of Obstetricians and Gynecologists Committee Opinion on low-dose aspirin use during pregnancy⁽¹³⁾, were prescribed aspirin.

Blood pressure monitoring in our study was standardized by using the same model of digital sphygmomanometer, calibrated annually by iTRIS Medical Co., Ltd. If a patient's blood pressure is found to be in the hypertensive range, the patient will be advised to rest and wait for a repeated measurement in four hours, according to criteria diagnosis of hypertension.

All pregnant women with GH were followed-up at Chonburi Hospital every 1 to 2 weeks until delivery. At every visit, blood pressure was measured as described and urine protein screening was performed using a dipstick. If the blood pressure measurement at any visit shown in hypertensive range, the patient underwent laboratory blood testing and UPCR testing. All participants were advised regarding the signs of preeclampsia with severe features and were instructed to perform home blood pressure monitoring and record the results.

The patients were followed-up until delivery. Pregnancy outcomes such as route of delivery, gestational age at delivery, Neonatal birth weight, APGAR score, serious pregnancy outcomes were collected.

Data were collected and analyzed using STATA/MP version 16.0. Participant characteristics and outcomes were compared using a Pearson chi-squared test for categorical variables and two-tailed student's t-test for continuous variables. A $p < 0.05$ was considered significant. Multiple logistic regression analysis was performed to assess the effects of the significant variables as predictors of preeclampsia progression. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated after adjusting for potential confounding factors discovered in this study.

Results

The study period was January 2019 to April 2025. A total of 238 pregnant women with GH were enrolled. Initial data were obtained from a retrospective cohort study of 198 participants. The sample size was enhanced by the extension of data collection through a prospective cohort study, yielding an additional 40 participants. All of them were followed-up until delivery. 12 of these (5.0%) were lost to follow-up and delivery at other hospitals. The remaining 226 participants were collected the data for analysis. 58 cases progressed to preeclampsia (25.7%, 95% CI 20.10, 31.88%), with 38 cases (16.8%, 95% CI 8.39, 17.41%) developing severe features. The mean of gestational

age at which patients progressed to preeclampsia was 36.05 ± 2.13 weeks.

The baseline characteristics of the women with GH alone (GH-GH group) and GH who progressed to preeclampsia (GH-PE group) are presented in Table 1. There were significant differences in gestational age at diagnosis and DBP at first time diagnosis between these two groups. The GH-PE group had a significantly earlier diagnosis of GH (33.63 ± 3.18 weeks) compared to the GH-GH group (35.46 ± 4.21 weeks, $p = 0.003$). Additionally, the mean DBP at the time of initial diagnosis of GH was significantly higher in the GH-PE group (99.14 ± 5.78 mmHg) compared to the GH-GH group (96.14 ± 4.32 mmHg, $p < 0.001$).

Table 1. Baseline characteristic at the presentation between GH-GH group and GH-PE group.

Variables	GH-GH (n = 168)	GH-PE (n = 58)	p value
Maternal age (years), mean $\pm$ SD	30.75 ± 7.02	31.64 ± 6.28	0.395
Prepregnancy BMI (kg/m^2), mean $\pm$ SD	29.92 ± 6.47	28.21 ± 4.73	0.066
< 25 kg/m^2 , n (%)	39 (23.2%)	16 (27.6%)	0.504
25-30 kg/m^2 , n (%)	54 (32.1%)	21 (36.2%)	0.571
≥ 30 kg/m^2 , n (%)	75 (44.6%)	21 (36.2%)	0.262
Nulliparous, n (%)	60 (35.7%)	23 (39.6%)	0.591
History of abortion, n (%)	29 (17.3%)	9 (15.5%)	0.759
History of HDP, n (%)	5 (3.0%)	3 (5.2%)	0.435
Diabetes mellitus status			
GDM (diet control), n (%)	20 (11.9%)	10 (17.2%)	0.302
GDM (medication control), n (%)	18 (10.7%)	5 (8.6%)	0.649
Known case DM, n (%)	4 (2.4%)	4 (6.9%)	0.109
Aspirin prescription, n (%)	23 (13.7%)	8 (13.8%)	0.984
Gestational age at 1st diagnosis GH (weeks), mean $\pm$ SD	35.46 ± 4.21	33.63 ± 3.18	0.003
SBP at 1 st diagnosis (mmHg), mean $\pm$ SD	149.34 ± 5.78	150.38 ± 6.13	0.246
DBP at 1 st diagnosis (mmHg), mean $\pm$ SD	96.14 ± 4.32	99.14 ± 5.78	< 0.001
Excessive weight gain, n (%)	116 (69.0%)	41 (70.7%)	0.8153
Hemoglobin at diagnosis (g/dL), mean $\pm$ SD	12.19 ± 1.10	12.34 ± 1.07	0.365

SD: standard deviation, BMI: body mass index, HDP: hypertensive disorders of pregnancy, GDM: gestational diabetes mellitus, DM: diabetes mellitus, GH: Gestational hypertension, SBP: systolic blood pressure, DBP: diastolic blood pressure.

* Excessive weight gain was defined according to prepregnancy BMI and weight gain in second and third trimester⁽¹⁾: - Prepregnancy BMI < 25 kg/m^2 : > 0.45 kg/week , Prepregnancy BMI 25-24.99 kg/m^2 : 0.27 kg/week , Prepregnancy BMI ≥ 30 kg/m^2 : 0.23 kg/week

There were no significant differences between two groups in terms of maternal age, prepregnancy BMI, nulliparity, history of HDP in previous pregnancy, history of abortion, DM status, aspirin prescription, SBP at first diagnosis, excessive weight gain, or

hemoglobin at diagnosis.

Subgroup analysis was conducted to compare women turned to preeclampsia without severe feature (GH-MPE group, n = 20) and women turned to preeclampsia with severe feature (GH-SPE group, n

= 38). The results are shown in Table 2. The gestational age at the time of GH diagnosis was not significantly different in GH-MPE group (34.99 ± 2.64) when compared with GH-GH group (35.46 ± 4.21 , $p = 0.627$). However, a significant difference was found in GH-SPE group (32.92 ± 3.24) compared with GH-GH group ($p = 0.001$). Furthermore, the DBP at the time

of diagnosis was significantly higher in the GH-SPE group (99.87 ± 5.84 mmHg) compared to the GH-GH group (96.14 ± 4.32 mmHg, $p < 0.001$). Conversely, there was no significant difference between GH-GH and GH-MPE group (96.14 ± 4.32 , $p = 0.128$). Other variables from Table 1 were also analyzed, but no significant difference was detected.

Table 2. Subgroup analysis of baseline characteristics at the presentation between GH-GH group and GH-PE group.

Variables	GH-GH (n = 168)	GH-MPE (n = 20)	p value*	GH-SPE (n = 38)	p value*
Gestational age at diagnosis GH (weeks)	35.46 ± 4.21	34.99 ± 2.64	0.627	32.92 ± 3.24	0.001
DBP at 1st diagnosis (mmHg)	96.14 ± 4.32	97.75 ± 5.52	0.128	99.87 ± 5.84	< 0.001
Prepregnancy BMI					
< 25 kg/m ² , n (%)	39 (23.2%)	6 (30%)	0.501	10 (26.3%)	0.677
25-30 kg/m ² , n (%)	54 (32.1%)	4 (20%)	0.266	17 (44.7%)	0.140
≥ 30 kg/m ² , n (%)	75 (44.6%)	10 (50%)	0.649	11 (28.9%)	0.076

MPE: mild preeclampsia, SPE: superimposed preeclampsia, GH: gestational hypertension, DBP: diastolic blood pressure, BMI: body mass index

* p value compared with GH-GH group

The comparison of delivery outcomes between women GH-GH group and GH-PE group are shown in Table 3. Women with preeclampsia had a markedly higher rate of cesarean section compared with those with gestational hypertension (91.4% vs 54.2%,

relative risk (RR) 1.69, 95% CI 1.44, 1.98). After exclusion of the other obstetric causes of cesarean section such as abnormal fetal lie, previous cesarean section, the data still in the same direction (81.0% vs 42.9%, RR 1.89, 95%CI 1.52, 2.34)

Table 3. Comparison of pregnancy outcome between GH-GH group and GH-PE group.

Outcomes	GH-GH (n = 168)	GH-PE (n = 58)	RR	95% CI
Route of delivery				
Normal labor, n (%)	74 (44.0%)	5 (8.6%)	0.19	0.08, 0.46
Cesarean section, n (%)	91 (54.2%)	53 (91.4%)	1.69	1.44, 1.98
Others (V/E, B/A), n (%)	3 (1.8%)	0 (0%)	-	
Gestational age at delivery				
Early preterm, n (%)	2 (1.2%)	6 (10.3%)	8.69	1.80, 41.86
Late preterm, n (%)	12 (7.1%)	21 (36.2%)	5.07	2.66, 9.65
Birth weight (g)				
1,501-2,500 g, n (%)	14 (8.3%)	21 (36.2%)	4.34	2.37, 7.97
1,000-1,500 g, n (%)	1 (0.6%)	2 (3.4%)	5.79	0.53, 62.71
< 1,000 g, n (%)	0 (0%)	0 (0%)	-	
APGAR score at 1 min				
> 7	166 (98.8%)	54 (93.1%)	0.94	0.88, 1.01
≤ 7	2 (1.2%)	2 (3.4%)	2.90	0.42, 20.10
< 4	0 (0%)	2 (3.4%)	-	
Admit NICU	1 (0.6%)	3 (5.2%)	8.69	0.92, 81.90

GH: gestational hypertension, PE: preeclampsia, RR: relative risk, CI: confidence interval, V/E: variant/effect, B/A: before/after, NICU: neonatal intensive care unit

The mean gestational age at delivery was significantly lower in the GH-PE group than in the GH-GH group (36.20 ± 1.85 vs 38.04 ± 1.52 weeks, $p < 0.001$). As a result, preeclampsia had a significant impact on preterm delivery rates, leading to an increased risk of both early preterm birth (10.3% vs 1.2%, RR 8.69, 95% CI 1.80, 41.86) and late preterm birth (36.2% vs 7.1%, RR 5.07, 95% CI 2.66, 9.65).

Significant differences were also found in neonatal outcomes. Infants born to mothers with preeclampsia had lower mean birth weights ($2,748.17 \pm 735.68$ g vs $3,100.23 \pm 486.03$ g, $p < 0.001$). The risk of low birth weight (1,501–2,500 g) was significant higher in this group (36.2% vs 8.3%, RR 4.34, 95% CI 2.37, 7.97). However, there were no significant differences in the rates of very low birth weight (1,000–1,500 g) or extremely low birth weight ($< 1,000$ g), Apgar scores, or neonatal intensive care unit admission between the groups. No case of abruptio placenta or postpartum intensive care unit

admission was detected in this study.

From the result above, gestational age at diagnosis and DBP at first diagnosis were significantly different in both groups. Multiple logistic regression was conducted to identify predictors for the progression to preeclampsia. We used the variables with $p < 0.1$ from Table 1, which included gestational age at diagnosis, DBP at the first time of diagnosis, and the prepregnancy BMI, for multiple logistic regression analysis and calculation of adjusted OR.

After analysis with multiple logistic regression, we found the risk of progressing to preeclampsia was significantly higher in women diagnosed with GH before 34 weeks (adjusted OR 3.304, $p = 0.001$) (Table 4). Patients with a DBP at diagnosis equal to or higher than 100 mmHg also had a significant higher risk for preeclampsia progression (adjusted OR 3.316, $p = 0.001$). For the prepregnancy BMI, we found that lower prepregnancy BMI was associated with a modest increase in the risk for preeclampsia progression (adjusted OR 0.943, $p = 0.048$).

Table 4. Results of univariate and multivariate analysis of predictors of progression to preeclampsia in gestational hypertension.

Variables	Crude OR (95% CI)	p value	Adjusted OR* (95% CI)	p value
GA at diagnosis of GH				
GA < 34 week	2.415 (1.279, 4.559)	0.007	3.304 (1.653, 6.607)	0.001
DBP at diagnosis				
DBP ≥ 100 mmHg	2.410 (1.268, 4.580)	0.007	3.159 (1.576, 6.336)	0.001
Prepregnancy BMI				
< 25 kg/m ²	Reference		Reference	
25 - 30 kg/m ²	0.948 (0.439, 2.047)	0.892	0.990 (0.448, 2.188)	0.981
≥ 30 kg/m ²	0.683 (0.320, 1.455)	0.323	0.662 (0.303, 1.445)	0.301

PE: preeclampsia, GH: gestational hypertension, OR: odds ratio CI: confidence interval, GA: gestational age, DBP: diastolic blood pressure, BMI: body mass index

* We used the variables with $p < 0.1$ from Table 1, which included GA at diagnosis, DBP at the first time of diagnosis, and the prepregnancy BMI, for multiple logistic regression analysis and calculation of adjusted OR.

Discussion

This study demonstrated that 25.7% of patients with GH at Chonburi Hospital developed preeclampsia. This finding was consistent with the results reported in previous research, where the rate of progression from GH to preeclampsia has been documented to range widely from 10% to 46%⁽⁹⁻¹²⁾. The variability in

these rates may be attributed to differences in study populations, ethnicity, and regional healthcare practices. Additionally, the data showed that an earlier diagnosis of GH, particularly before 34 weeks of gestation, was associated with a higher risk of developing preeclampsia. This supported previous findings that the timing of GH onset was important

predictors of the progression to preeclampsia, emphasizing the importance of early screening and close monitoring of pregnant women diagnosed with GH before 34 weeks⁽¹⁰⁻¹²⁾.

From previous studies, the underlying pathophysiological mechanisms of GH and preeclampsia reported that both are different. In preeclampsia, the primary causative factor involves placental abnormalities, such as abnormal placental development and pathology leading to placental ischemia. This placental insufficiency triggers a cascade of events, including endothelial dysfunction, oxidative stress, and an imbalance in angiogenic factors, which contribute to systemic hypertension and multiple organ dysfunction. In contrast, GH is thought to result predominantly from maternal factors, including altered vascular tone, increased vascular resistance, and an imbalance between cardiac output and vascular compliance, without the significant placental pathology characteristic of preeclampsia⁽¹⁴⁾.

Based on the timing of onset, preeclampsia can be classified into two main categories: early-onset preeclampsia, diagnosed before 34 weeks of gestation, and late-onset preeclampsia, diagnosed at or after 34 weeks of gestation. In this study, the mean gestational age at progression to preeclampsia was 36.05 weeks (95% CI 35.50, 36.61), which corresponded to late-onset preeclampsia. Previous studies have suggested that the development of late-onset preeclampsia may not be related to placental factors but could also involve maternal factors. Therefore, the detection of GH at an earlier stage, which reflects underlying maternal predisposition, could be a predictive factor for the subsequent development of late-onset preeclampsia⁽¹⁵⁾.

Not only was the gestational age at the diagnosis of GH identified as a key predictive factor for progression to preeclampsia in our study, but DBP at first diagnosis was also an associated factor. An increase in DBP is caused by increased systemic vascular resistance, which can serve as a reflection of maternal vascular health. From our literature review, there is no previous study directly discussed

this specific association. However, some findings were consistent with our results. Maria et al (2014)⁽¹⁶⁾ reported that DBP could serve as a risk indicator for preeclampsia in normal population. They demonstrated that a baseline DBP ≥ 80 mmHg at 12 weeks of gestation, combined with an increase in DBP of ≥ 15 mmHg, could identify women at risk of preeclampsia, with a sensitivity of 92% and a specificity of 44%. But the study in GH population is still limited.

From Table 1, the baseline prepregnancy BMI was likely different between the GH-GH and GH-PE groups ($p = 0.066$). However, when prepregnancy BMI was classified according to the World Health Organization classification of overweight/obesity⁽¹⁷⁾, it seemed to be no statistical difference. Multiple logistic regression analysis indicated that patients with obesity tended to have a decreased risk of preeclampsia progression, although this finding was not statistically significant. This observation was consistent with the study by Badon Sylvia et al⁽¹⁸⁾, they found that chronic hypertension women with obesity with low gestational weight gain, compared to those with normal weight and overweight, were associated with 27–54% lower risk of several adverse pregnancy outcomes, including preeclampsia and cesarean delivery. Therefore, the association between prepregnancy BMI and gestational weight gain should be further investigated.

Comparison of perinatal outcomes in this study demonstrated that the GH-PE group had a higher incidence of preterm delivery, cesarean section, and neonatal low birth weight. However, no significant differences were observed in serious neonatal outcomes, such as birth asphyxia or the need for neonatal intensive care unit admission. These findings were consistent with previous studies^(11, 12) and corresponded with both the underlying pathophysiology of the disease and the current management strategies for preeclampsia.

The progression from GH to preeclampsia is associated not only with the immediate perinatal morbidity and mortality but also with an increasing

risk of long-term cardiovascular disease⁽¹⁹⁾. Therefore, identifying key risk factors for this progression is crucial for enabling early detection and prevention. These strategies could effectively reduce the long-term health complications linked to preeclampsia.

A key limitation of this study was its limited external validity arising from the single-center cohort design. Since the study was conducted exclusively at Chonburi Hospital, the findings may be subject to single-center bias, clinical protocols, and resource availability specific to this institution. Furthermore, the patient cohort may not fully represent the general population with pregnant women with GH due to geographic, socio-economic, or ethnic homogeneity within the center's catchment area. Consequently, the generalizability of these findings to other clinical settings or diverse populations remains uncertain. However, a significant strength of this study was an adequate sample size to answer the research question about incidence of preeclampsia progression in GH pregnancy. This study had picked up the interesting factors, DBP at the first time of GH diagnosis and prepregnancy BMI, that could be associated with progression of preeclampsia in GH pregnancy. Further studies focusing on this topic may be required.

Conclusion

The findings of this study showed that pregnant women with GH had a higher risk of progressing to preeclampsia than the general pregnant population. This was reflected in an incidence of 25.7% in this study's cohort, which was approximately 3-5 times higher than in the general population⁽¹⁾. This progression was associated with a significantly increased risk of adverse perinatal outcomes, including preterm birth, a higher rate of cesarean sections, and a lower neonatal birth weight. Another important finding was that an early diagnosis of GH and a higher DBP at diagnosis were significantly associated with the development of preeclampsia.

Thus, the patient who was diagnosed with GH prior to 34 weeks of gestation or those with a DBP

≥ 100 mmHg at diagnosis had a higher risk of progression to preeclampsia, especially at approximated 36 weeks of gestation. Consequently, counselling and close follow-up are required. A notable limitation of this research was its single-center cohort design, which resulted in a lack of population diversity. Therefore, further large-scale multicenter studies are recommended to confirm these findings and explore additional risk factors.

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Potential conflicts of interest

The authors declare no conflicts of interest.

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