Original Article

The Different Outcomes in Pregnant Women with Severe Features of Preeclampsia between New Onset Hypertension Group and Pre-Existing Hypertension Group

Pornchanit Santamonkunrot MD[1], Pimpika Tansupswatdikul MD[1]

[1] Department of Obstetrics and Gynecology, Chonburi Hospital, Chonburi, Thailand

Background: There are extensive evidence that preeclampsia is the reason for many maternal and perinatal morbidities. However, there are no previous study on the different outcomes in severe preeclamptic women between new onset hypertension and chronic hypertension.

Objective: To compare the rate of low Apgar score at 5 minutes and other adverse pregnancy outcomes of preeclamptic women with severe features between those who had new onset hypertension in pregnancy and those with chronic hypertension before pregnancy.

Materials and Methods: A retrospective cohort study was conducted. The medical records of pregnant women diagnosed with preeclampsia with severe features and delivered at Chonburi Hospital between January 2017 and June 2020 were reviewed. The authors categorized these pregnant women into two groups, new onset hypertension and chronic hypertension. Descriptive statistics were used for the data analyses.

Results: Of the 526 women diagnosed with preeclampsia with severe features, 290 met the inclusion and exclusion criteria. Eighty-nine had superimposed preeclampsia on chronic hypertension and 201 patients had new onset hypertension. The rate of low Apgar score at 5 minutes was not statistically different between the two groups (p=0.258). The incidence of impaired liver function and HELLP syndrome were increased in the new onset hypertension group at 18.9% versus 6.7% (p=0.008) and 9.5% versus 1.1% (p=0.010), respectively.

Conclusion: There was no significant difference in the rate of low Apgar score between the two groups. Regarding the severity of maternal outcomes, new onset preeclampsia with severe features seems to be more severe than superimposed preeclampsia on chronic hypertension.

Keywords: Low Apgar score; Preeclampsia with severe features; Chronic hypertension; Superimposed preeclampsia; Pregnancy outcomes

Received 28 December 2020 | Revised 17 March 2021 | Accepted 22 March 2021

J Med Assoc Thai 2021;104(6):951-8

Website: http://www.jmatonline.com

Hypertensive disorders complicate 3% to 10% of all pregnancies worldwide[1]. These include gestational hypertension, chronic hypertension, superimposed preeclampsia on chronic hypertension (SPCH), preeclampsia, and eclampsia syndrome. Preeclampsia does not only cause maternal morbidity, but perinatal outcomes are also affected. Both new onset hypertension (NOH) and chronic hypertension (CH) can restrict fetal growth, leadings to newborns that are small for their gestational age (GA) and have low birth weight because of uteroplacental insufficiency. Preeclampsia usually leads to iatrogenic preterm birth and causes the sequelae of preterm neonate. The Apgar score is a rapid method for assessing the status of a newborn[2]. A low Apgar score can imply intrapartum asphyxia and may leads to poor neonatal outcomes.

A population study with 30,000 pregnant women with CH found that preeclampsia increases the risk of cerebrovascular disease, pulmonary edema, and renal failure five-to six folds. SPCH occurs in 20% to 50% of cases[3]. A previous study has reported an incidence of superimposed preeclampsia in Thailand of 43.3% (95% confidence interval [CI] 37.8 to 48.9)[4]. Hence, CH in pregnancy is a consequential factor that needs consideration because it leads to high rates of maternal and perinatal morbidity.

Preeclampsia develops from the abnormal trophoblastic invasion of uterine vessels, immunological maladaptation, maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy, and genetic factors. Precipitating etiologies are the following, exposed to chorionic villi for the first time, exposed to a superabundance of

Correspondence to:

Santamonkunrot P.

Department of Obstetrics and Gynecology Chonburi Hospital, 69 Sukhumvit Road, Ban Suan, Chonburi 20000, Thailand.

Phone: +66-64-5169369, +66-38-931000

Email: spornchanit@gmail.com

How to cite this article:

Santamonkunrot P, Tansupswatdikul P. The Different Outcomes in Pregnant Women with Severe Features of Preeclampsia between New Onset Hypertension Group and Pre-Existing Hypertension Group. J Med Assoc Thai 2021;104:951-8.

doi.org/10.35755/jmedassocthai.2021.06.12379

chorionic villi, have preexisting conditions associated with endothelial cell activation or inflammation, and genetically predisposed to hypertension during pregnancy[5]. In patients with NOH, their predisposing factors are mostly from the first three. Patients with CH develop superimposed preeclampsia from suffering damaged endothelial cells damaged and being predisposed to hypertension.

The risk of morbidity and mortality rises if pregnant women with preeclampsia present with any of the following severe features, a systolic blood pressure of 160 mmHg or more, a diastolic blood pressure of 110 mmHg or more on two occasions at least four hours apart unless antihypertensive therapy is initiated before this time, thrombocytopenia with a platelet count less than 100,000/μL, impaired liver function as indicated by abnormally elevated blood concentrations of liver enzymes to twice the upper limit of the normal concentration, severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by alternative diagnoses, renal insufficiency with serum creatinine concentration more than 1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease, pulmonary edema, new-onset headache unresponsive to medication and not accounted for by alternative diagnoses, or visual disturbances.

While many studies have examined preeclampsia and CH, none have compared the pregnancy outcomes among patients with preeclampsia with severe features (PSF) between those with CH and those with NOH. Due to their different pathophysiology, the maternal and perinatal outcomes between the two groups might differ. The present study compared the rate of low Apgar score at 5 minutes and other adverse pregnancy outcomes between the new onset hypertension with preeclampsia with severe features (NOHPSF) and chronic hypertension with superimposed preeclampsia with severe features (CHSPSF).

Materials and Methods

This retrospective cohort study was approved by the Institutional Review Board of Chonburi Hospital (REC no. 24/63/R/h3). The medical records of pregnant women diagnosed with PSF who gave birth at Chonburi Hospital between January 2017 and June 2020 were reviewed. The diagnosis of PSF was based on the American College of Obstetrics and Gynecologist Practice Bulletin[6]. Preeclampsia was diagnosed if a pregnant woman had systolic blood pressure of 140 mmHg or higher or diastolic blood pressure of 90 mmHg or higher after GA of 20 weeks in previously normotensive women with one of the following:

• Proteinuria: urine dipstick 2+, urine protein to urine creatinine ratio (UPCR) at or more than 0.3, or urine protein 24 hours at or more than 300 mg

• Thrombocytopenia: platelet count of less than 100,000/μL

• Renal insufficiency: serum creatinine level of more than 1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease

• Impaired liver function: serum transaminase (SGOT or SGPT) levels twice the normal level

• Cerebral symptoms: new-onset headache unresponsive to medication, visual disturbances, and convulsions

• Pulmonary edema

Severe features include systolic blood pressure of 160 mmHg or more or diastolic blood pressure of 110 mmHg or more, thrombocytopenia with a platelet count of less than 100,000/μL), impaired liver function with elevated liver enzymes more than twice the upper limit of the normal level, renal insufficiency with serum creatinine of more than 1.1 mg/dL or twice the baseline level, pulmonary edema, new-onset headache unresponsive to medication, and visual disturbances[6]. Patients were considered to have CH if they had a history of CH, systolic blood pressure of 140 mmHg or more, or diastolic blood pressure of 90 mmHg or more before 20 weeks of gestation[5]. They were diagnosed with CHSPSF if they presented with any of the severe features mentioned above.

The authors excluded patients whose medical records were incomplete, GA at delivery less than 24 weeks, did not receive antenatal care, had their first antenatal care visit at more than 20 weeks of gestation, had a multifetal pregnancy, had other causes of proteinuria such as chronic renal disease or systemic lupus erythematosus (SLE), placenta previa, non-vertex presentation, preterm premature ruptured of membranes, fetus with a congenital anomaly or abnormal chromosome, had fetal distress, or a fetal non-reassuring status. Exclusion of patients with poor antenatal care records as mentioned would not confuse patients with chronic hypertension but no history of or record of hypertension. Patients were divided into two groups, CHSPSF and NOHPSF.

The primary outcome of the present study was to compare the rate of low Apgar score at 5 minutes as a score of less than 7, in neonates between the two

groups. A low 5-minutes Apgar score increased the relative risk of cerebral palsy by 20 to 100-folds[7-9]. The Apgar score was provided at 1 and 5 minutes after birth by either a pediatric resident or an experienced midwife.

Secondary outcomes were to compare the adverse maternal and perinatal outcomes of both groups. Adverse maternal outcomes included the rates of cesarean delivery, postpartum hemorrhage, placental abruption, involvement of other organs such as pulmonary edema, renal insufficiency, and impaired liver function, HELLP syndrome, eclampsia, postdelivery intensive care unit (ICU) admission, and maternal death. Perinatal outcomes included GA at delivery, a low Apgar score at 1 minute, birth weight, small for gestational age (SGA) rate, neonatal intensive care unit (NICU) and sick newborn ward (SNB) admission rate, and stillbirth rate. Pulmonary edema was diagnosed from signs and symptoms such as dyspnea, desaturation, or lung crepitations, and chest X-rays. Renal insufficiency was defined as serum creatinine greater than 1.1 mg/dL or more than twice the baseline level. Impaired liver function was defined as SGOT or SGPT greater than twice the normal upper limit. HELLP syndrome is diagnosed using the Tennessee Classification: hemolysis with serum LDH at or above 600 IU/L, elevated liver enzymes with AST at or above 70 IU/L, and low platelets at less than 100,000/μL[10]. Patients with HELLP syndrome were also included in patients with impaired liver function. The neonate is SGA if birth weight was less than the tenth percentile of their GA.

Since no previous study has compared the rate of low Apgar score between these two groups, a pilot study was performed. The authors collected 40 medical records with 20 in each group. None of the CHSPSF patients gave birth to a neonate with a low Apgar score. Two of the 20 NOHPSF women gave birth to a neonate with a low Apgar score. The sample size was calculated using the two independent proportions formula[11].

Where type I error with alpha=0.05, the ratio was 0.5, p_1 (NOHPSF group) was 0.1, and p_2 (CHSPSF group) was 0.01. The sample of group 1 (NOHPSF group) was 162 and that of group 2 (CHSPSF group) was 81. Data were collected from medical records between January 2017 and June 2020.

Data were presented as percentage, mean ± standard deviation (SD), median, and interquartile range (IQR). Independent t-tests were used for the continuous variables with a normal distribution, chi-square tests were used for the categorical data, and Mann-Whitney U tests were used for the skewed data. A p-value of less than 0.05 was considered to be statistically significant.

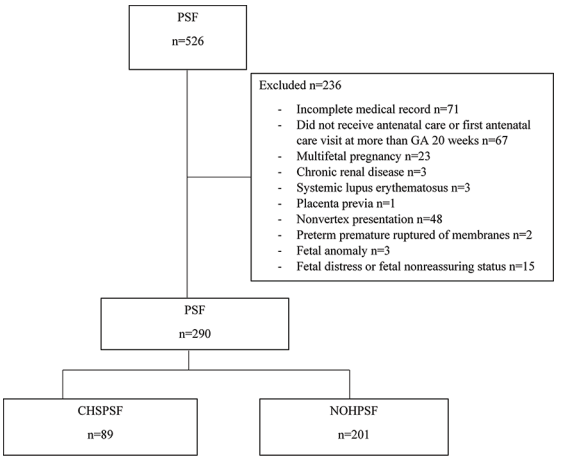

Figure 1. Overview flow of study participants.

PSF=preeclampsia with severe features; CHSPSF=chronic hypertension superimposed preeclampsia with severe features; NOHPSF=new onset hypertension with preeclampsia with severe features

Results

Five hundred twenty-six pregnant women were diagnosed with PSF in Chonburi Hospital between January 2017 and June 2020 (Figure 1).Two hundred thirty-six were excluded from the present study because 71 had incomplete medical records, 67 pregnant women did not receive antenatal care or their first antenatal care visit was at more than 20 weeks of gestation, 23 women had multifetal pregnancy, three had chronic renal disease, three had SLE, one had placenta previa, 48 had non-vertex presentation, two had preterm premature ruptured of membranes, three had a fetal anomaly or chromosomal abnormality, and 15 had fetal distress or a fetal non-reassuring status. The remaining 290 pregnant women were divided into two groups based on their diagnosis with 201 diagnosed with NOHPSF and 89 diagnosed with CHSPSF.

The baseline characteristics in Table 1 include maternal age, gravida, parity, history of abortion, history of preterm birth, diabetes status, number of antenatal care visits, any of the severe features, and the UPCR.

According to these baseline characteristics, there was no statistically significant differences between the two groups in terms of gravida, history of abortion, history of preterm birth, the rate of gestational diabetes (type 1 and 2), and UPCR. The severe features used

Table 1. Baseline characteristics

	CHSPSF group (n=89); n (%)	NOHPSF group (n=201); n (%)	p-value
Age			<0.001*
15 to 19	1 (1.1)	13 (6.5)	
20 to 34	54 (60.7)	137 (68.1)	
35 to 49	34 (38.2)	51 (25.4)	
Mean±SD	32.88±5.3	29.84±6.5	
Gravida			0.053
1	31 (34.9)	95 (47.3)	
2	25 (28.1)	61 (30.3)	
3	19 (21.3)	29 (14.4)	
>3	14 (15.7)	16 (8.0)	
Parity			0.036*
Nulliparity	37 (41.6)	111 (55.2)	
Multiparity	52 (58.4)	90 (44.8)	
History of abortion	22 (24.7)	37 (18.4)	0.218
History of preterm birth	6 (6.7)	11 (5.5)	0.671
Diabetes status			
GDMA1	10 (11.2)	25 (12.4)	0.772
GDMA2	4 (4.5)	4 (2.0)	0.230
Overt DM	18 (20.2)	12 (6.0)	<0.001*
Number of ANC; mean±SD	9.66±3.3	8.12±2.6	<0.001*
Diagnose severe features from			
BP ≥160/110 mmHg	78 (87.6)	147 (73.1)	0.006*
Impaired liver function	7 (7.9)	19 (9.5)	0.663
Renal insufficiency	5 (5.6)	9 (4.5)	0.676
HELLP syndrome	1 (1.1)	19 (9.5)	0.010*
Thrombocytopenia	0 (0.0)	8 (4.0)	0.056
UPCR; median (IQR)	1.66 (0.48 to 3.35)	1.98 (0.62 to 6.36)	0.075

CHSPSF=chronic hypertension superimposed preeclampsia with severe features; NOHPSF=new onset hypertension with preeclampsia with severe features; GDM=gestational diabetes mellitus; DM=diabetes mellitus; ANC=antenatal care; BP=blood pressure; UPCR=urine protein to urine creatinine ratio; SD=standard deviation; IQR=interquartile range

A p-value corresponds to independent t-test or Mann-Whitney U and chi-square test, * p<0.05 is statistically significant

to make the diagnosis were not statistically different for impaired liver function, renal insufficiency, and thrombocytopenia. The average age of the pregnant women in CHSPSF group was 32.88 years old, which was significantly higher than that in the NOHPSF group (p<0.001). Nulliparity was found more in the NOHPSF group than the CHSPSF group (p=0.036). Pregestational diabetes was found in 20.2% of the pregnant women with CHSPSF, significantly more than for NOHPSF at 6.0% (p<0.001). The average number of ANC visits by pregnant women with CHSPSF was more than those for NOHPSF patients at 9.66±3.28 versus 8.12±2.6 (p<0.001). The severe features used to diagnose were different in terms of severe range blood pressure with systolic blood pressure at or above 160 mmHg or diastolic blood pressure at or above 110 mmHg. This was found in CHSPSF group more than the NOHPSF group at 87.6% and 73.1% (p=0.006). Patients diagnosed with severe features from severe range blood pressure meant that all other laboratory values were normal and blood pressure was the only severe feature present. Other severe features such as impaired liver function, renal insufficiency, or thrombocytopenia may or may not have severe range blood pressure. The rate of HELLP syndrome increased in the NOHPSF group at 9.5% versus 1.1% (p=0.01), which concurred with the data on maternal outcomes in Table 2.

The rate of low Apgar score at 5 minutes in CHSPSF group was 1.1% compared with 3.5% in

Table 2. Maternal outcomes

	CHSPSF group (n=89); n (%)	NOHPSF group (n=201); n (%)	p-value
Route of delivery			
New cesarean section	63 (70.8)	134 (66.7)	0.488
Vaginal delivery	14 (15.7)	38 (18.9)	0.516
Repeat cesarean section	12 (13.5)	29 (14.4)	0.831
Postpartum hemorrhage	4 (4.5)	11 (5.5)	0.729
Placental abruption	0 (0.0)	1 (0.5)	0.505
Other organs involvement			
Pulmonary edema	4 (4.5)	12 (6.0)	0.612
Renal insufficiency	6 (6.7)	13 (6.5)	0.931
Impaired liver function	6 (6.7)	38 (18.9)	0.008*
HELLP syndrome	1 (1.1)	19 (9.5)	0.010*
Eclampsia	0 (0.0)	7 (3.5)	0.075
Postdelivery ICU admission	4 (4.5)	20 (10.0)	0.120
Maternal death	0 (0.0)	2 (1.0)	0.345

CHSPSF=chronic hypertension superimposed preeclampsia with severe features; NOHPSF=new onset hypertension with preeclampsia with severe features; ICU=intensive care unit

A p-value corresponds to chi-square test, * p<0.05 is statistically significant

Table 3. Perinatal outcomes

	CHSPSF group (n=89); n (%)	NOHPSF group (n=201); n (%)	p-value
GA at delivery (weeks)			
<34	29 (32.6)	42 (20.9)	0.033*
≥34	60 (67.4)	159 (79.1)	
Apgar at 1 minute			
<7	14 (15.7)	26 (12.9)	0.524
≥7	75 (84.3)	175 (87.1)	
Apgar at 5 minutes			
<7	1 (1.1)	7 (3.5)	0.258
≥7	88 (98.9)	194 (96.5)	
Birth weight (g)			
500 to 1,000	0 (0.0)	10 (5.0)	
1,001 to 1,500	23 (25.8)	22 (10.9)	
1,501 to 2,000	15 (16.9)	41 (20.4)	
2,001 to 2,500	13 (14.6)	43 (21.4)	
>2,500	38 (42.7)	85 (42.3)	
Mean±SD	2,304.5±856.8	2,367.6±838.7	0.557
SGA	20 (22.5)	46 (22.9)	0.938
Ward admission			
NICU	25 (28.1)	53 (26.4)	0.760
Sick newborn care units	30 (33.7)	67 (33.3)	0.950
Returned to their mother	33 (37.1)	80 (39.8)	0.661
Stillbirth	1 (1.1)	1 (0.5)	0.552

CHSPSF=chronic hypertension superimposed preeclampsia with severe features; NOHPSF=new onset hypertension with preeclampsia with severe features; GA= gestational age; SGA=small for gestational age; NICU=neonatal intensive care unit; SD=standard deviation

A p-value corresponds to chi-square test and independent t-test, * p<0.05 is statistically significant

NOHPSF group (p=0.258). The incidences of a low Apgar score at 5 minutes were not statistically different between the two groups.

For maternal outcomes, as shown in Table 2, there were no significant differences between the two groups in terms of delivery, postpartum hemorrhage, placental abruption, pulmonary edema, acute kidney injury, eclampsia, postdelivery ICU admission, and maternal death rate. However, two maternal deaths were found in the present study. One had thyroid storm and the other had valvular heart disease and pulmonary hypertension. Pregnant women NOHPSF had an increased rate of elevated liver enzymes at 18.9% versus 6.7% (p=0.008) and HELLP syndrome at 9.5% versus 1.1% (p=0.010).

As shown in Table 3, the perinatal outcomes were not statistically different for the rate of low Apgar score at 1 minute, mean of birth weight, rate of SGA, NICU admission, and stillbirth. GA at delivery was lower in CHSPSF group. Delivery at a GA less than 34 weeks in CHSPSF group was 32.6% compared with 20.9% in NOHPSF group (p=0.033).

Discussion

No previous study has compared the maternal and perinatal outcomes between CH and NOH among PSF patients. There is no significant difference in the rate of low Apgar score at 5 minutes between pregnant women in CHSPSF and those in NOHPSF group. This finding is different from a previous study in Jakarta[12] that reported higher rates of low Apgar score at 5 minutes in the NOH group than CH group with 58.3% versus 4.2%. These contradictory findings may be because of the difference in participants, as that study included all cases of preeclampsia with any severity and excluded cases of HELLP syndrome. HELLP syndrome was not excluded from the present study because it could be a consequence of both CH and new onset preeclampsia.

CH is found in 1% to 5% of pregnancies and the incidence increases with the age of the pregnant woman[13], which correlates with the findings in the present study. As mentioned before, CHSPSF and NOHPSF have different pathophysiology. One risk factor for preeclampsia is nulliparity because preeclampsia frequently occurs in women

exposed to chorionic villi for the first time[5]. Superimposed preeclampsia occurs because of endothelial cell activation or inflammation from CH. This pathophysiology explains why the rate of nulliparity is higher in NOHPSF group.

Tracey and Lanay reviewed the association of preeclampsia and pregestational and gestational diabetes[14]. The incidence of preeclampsia in type 1 diabetes mellitus is 15% to 20%[15-17] and type 2 diabetes mellitus is 10% to 14%[17,18] compared with the normal population of 2% to 7%[19,20]. Pregestational diabetes is found more in patients with CH; indeed, they usually coexist[21], as also found in the present study. Although coexisting pregestational diabetes in CHSPSF group is higher, the outcomes of vascular consequences such as birth weight, Apgar score, and rate of SGA are similar. Studies have shown that the odds of preeclampsia are increased in women with gestational diabetes[22-24]. After controlling for age, nationality, job status, smoking, parity, multifetal pregnancy, pre-pregnancy weight status, and gestational weight gain, the adjusted odds ratio is 1.29 (95% CI 1.19 to 1.41)[22]. According to ACOG, gestational diabetes is not considered to be a risk factor for preeclampsia and is not included in the high or intermediate risk that needs aspirin prophylaxis[25]. Further research is thus needed on whether low dose aspirin will benefit in preventing preeclampsia in pregnant women with gestational diabetes.

For adverse maternal outcomes, the present study showed significantly different outcomes in the rate of impaired liver function and HELLP syndrome, which were found more in NOHPSF group. HELLP syndrome is a severe form of preeclampsia that can cause severe maternal and neonatal complications. These consequences include the spontaneous rupture of liver subcapsular hematoma, abruptio placenta, DIC, postpartum hemorrhage, prematurity, neonatal thrombocytopenia, and severe fetal growth restriction[10]. This was found more in NOHPSF group than the CHSPSF group. In addition, the rate of eclampsia in the NOHPSF group was more than that in the CHSPSF group at 3.5% and 0.0%, respectively, although there was no statistical significance, but tend to be increased. If more patients were recruited, the rate of eclampsia could be significantly higher in the NOHPSF group. Therefore, NOHPSF patients lean toward having more severe maternal morbidity than CHSPSF patients. At antenatal care, doctors tend to be more cautious with CH women. These patients have closer follow-up visits, their blood pressure is monitored more closely, and they follow laboratory values more often. On the contrary, the NOHPSF group usually present when their clinicals are already severe, which may explain why severity is less in the CHSPSF group. Although no data on aspirin use were not collected, Chonburi Hospital has prescribed low dose aspirin to all high-risk patients since the ACOG recommendation in July 2018[25]. The use of low dose aspirin might lower the severity of preeclampsia in the CHSPSF group. However, there are insufficient data to support this.

In comparing the perinatal outcomes between the two groups, the only difference was GA at delivery. More patients delivered at a GA less than 34 weeks in the CHSPSF group than the NOHPSF group with 32.6% versus 20.9% (p=0.033). However, it cannot be concluded that perinatal outcomes are more severe in CHSPSF patients because the other perinatal outcomes (Apgar score at 1 and 5 minutes, birthweight, rate of SGA, NICU admission, stillbirth) were similar.

One strength of the present study is that it is the first to compare the outcomes in PSF between pregnant women with CH and NOH. It thus acknowledges which type of severe preeclampsia tends to be more severe and might need tertiary care. In addition, more antenatal care visits might be needed in low-risk patients to detect preeclampsia at earlier onset. Since this was a retrospective study, there were limitations in data collection. Many were excluded due to incomplete medical records, which might affect the results. No data were collected on aspirin prophylaxis in high-risk patients, the onset of severe features, and antihypertensives used, which might affect the maternal and perinatal outcomes. A prospective study at King's College Hospital in London found that in patients with CH, whether the patient can control her blood pressure to be less than 130 to 140 over 80 to 90 mmHg with medication throughout pregnancy does not change the pregnancy outcomes[26]. Therefore, antihypertensive drugs usage might not change the outcomes of the present study.

The authors do not know about the long-term morbidity and mortality difference of the two. Preeclampsia increases the risk of maternal cardiovascular diseases in the future, especially preterm preeclampsia[27]. It also increases the risk of ischemic heart disease at 24% versus 15% and stroke at 9.5% versus 6.5%, compared with the normal population[28]. Fifteen percent of previous preeclamptic women have renal disease[29]. To the best of the authors' knowledge, there are only studies on

long term sequelae in women with preeclampsia in general and no subgroup analysis has been conducted on the types of preeclampsia. Further studies of whether CHSPSF or NOHPSF increases the risk of subsequent chronic diseases should be carried out. In addition, maternal outcomes should be studied as primary, and more data should be collected on whether aspirin prophylaxis affects perinatal and maternal outcomes.

In conclusion, NOHPSF tends to lead to more severe maternal outcomes than CHSPSF. There are higher rates of elevated liver enzymes, HELLP syndrome, and eclampsia in NOHPSF group and these can lead to higher rates of maternal morbidity and mortality. There is no difference in the severity of perinatal outcomes between the two groups.

What is already known on this topic?

Preeclampsia increases the risk of cerebrovascular disease, pulmonary edema, and renal failure in CH women. Many studies examined between the outcomes of hypertensive disorders of pregnancy, gestational hypertension, and preeclampsia have highest morbidity of all. No study was done to compare outcomes between chronic hypertension superimposed preeclampsia and new onset preeclampsia.

The closest study to the present study was an observational study in Jakarta. It found that higher rates of low Apgar score at 5 minutes in NOH group than CH group. However, they included all severity of preeclampsia. In this study, the authors only studied severe preeclampsia groups.

What this study adds?

This is the first study to compare the outcomes in PSF between pregnant women with CH and NOH. There are higher rates of impaired liver function and HELLP syndrome in NOHPSF group. No difference in perinatal outcomes between the two groups. This study acknowledges which type of severe preeclampsia tends to be more severe and might need tertiary care. Future trials to investigate long term complications between the two groups is motivated.

Acknowledgment

The authors acknowledge the support from the department of Obstetrics and Gynecology at Chonburi Hospital.

Conflicts of interest

The authors declare no conflict of interest.

References

1. Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. Obstet Gynecol 2013;122:1122-31.
2. American Academy of Pediatrics, Committee on Fetus and Newborn; American College of Obstetricians and Gynecologists and Committee on Obstetric Practice. The Apgar score. Pediatrics 2006;117:1444-7.
3. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. ACOG Practice bulletin No. 203: Chronic hypertension in pregnancy. Obstet Gynecol 2019;133:e26-50.
4. Boriboonhirunsarn D, Pradyachaipimol A, Viriyapak B. Incidence of superimposed preeclampsia among pregnant Asian women with chronic hypertension. Hypertens Pregnancy 2017;36:226-31.
5. Cunningham FG, Leveno K, Bloom S, Spong C, Dashe J, Hoffman B, et al. Williams obstetrics. 25th ed. New York: McGraw-Hill Education; 2018.
6. Gestational hypertension and preeclampsia: ACOG practice bulletin, Number 222. Obstet Gynecol 2020;135:e237-60.
7. Moster D, Lie RT, Irgens LM, Bjerkedal T, Markestad T. The association of Apgar score with subsequent death and cerebral palsy: A population-based study in term infants. J Pediatr 2001;138:798-803.
8. Nelson KB, Ellenberg JH. Apgar scores as predictors of chronic neurologic disability. Pediatrics 1981;68:36-44.
9. Lie KK, Grøholt EK, Eskild A. Association of cerebral palsy with Apgar score in low and normal birthweight infants: Population based cohort study. BMJ 2010;341:c4990.
10. Haram K, Svendsen E, Abildgaard U. The HELLP syndrome: clinical issues and management. A Review. BMC Pregnancy Childbirth 2009;9:8.
11. Rosner BR. Fundamentals of biostatistics. 5th ed. Pacific Grove, CA: Duxbury; 2000.
12. Susilo S, Pratiwi K, Fattah A, Irwinda R, Wibowo N. Determinants of low APGAR score among preeclamptic deliveries in Cipto Mangunkusumo Hospital: A retrospective cohort study in 2014. Med J Indones 2015;24:183-9.
13. Panaitescu AM, Syngelaki A, Prodan N, Akolekar R, Nicolaides KH. Chronic hypertension and adverse pregnancy outcome: a cohort study. Ultrasound Obstet Gynecol 2017;50:228-35.
14. Weissgerber TL, Mudd LM. Preeclampsia and diabetes. Curr Diab Rep 2015;15:9.
15. Jensen DM, Damm P, Moelsted-Pedersen L, Ovesen P, Westergaard JG, Moeller M, et al. Outcomes in type 1 diabetic pregnancies: a nationwide, population-based study. Diabetes Care 2004;27:2819-23.
16. Persson M, Norman M, Hanson U. Obstetric and perinatal outcomes in type 1 diabetic pregnancies: A large, population-based study. Diabetes Care

2009;32:2005-9.
17. Knight KM, Thornburg LL, Pressman EK. Pregnancy outcomes in type 2 diabetic patients as compared with type 1 diabetic patients and nondiabetic controls. J Reprod Med 2012;57:397-404.
18. Groen B, Links TP, van den Berg PP, Hellinga M, Moerman S, Visser GH, et al. Similar adverse pregnancy outcome in native and nonnative Dutch women with pregestational type 2 diabetes: a multicentre retrospective study. ISRN Obstet Gynecol 2013;2013:361435.
19. Knuist M, Bonsel GJ, Zondervan HA, Treffers PE. Intensification of fetal and maternal surveillance in pregnant women with hypertensive disorders. Int J Gynaecol Obstet 1998;61:127-33.
20. Hauth JC, Ewell MG, Levine RJ, Esterlitz JR, Sibai B, Curet LB, et al. Pregnancy outcomes in healthy nulliparas who developed hypertension. Calcium for Preeclampsia Prevention Study Group. Obstet Gynecol 2000;95:24-8.
21. Mancia G. The association of hypertension and diabetes: prevalence, cardiovascular risk and protection by blood pressure reduction. Acta Diabetol 2005;42 Suppl 1:S17-25.
22. Schneider S, Freerksen N, Röhrig S, Hoeft B, Maul H. Gestational diabetes and preeclampsia--similar risk factor profiles? Early Hum Dev 2012;88:179-84.
23. Nerenberg KA, Johnson JA, Leung B, Savu A, Ryan EA, Chik CL, et al. Risks of gestational diabetes and preeclampsia over the last decade in a cohort of Alberta women. J Obstet Gynaecol Can 2013;35:986-94.
24. Ostlund I, Haglund B, Hanson U. Gestational diabetes and preeclampsia. Eur J Obstet Gynecol Reprod Biol 2004;113:12-6.
25. ACOG Committee Opinion No. 743: Low-dose aspirin use during pregnancy. Obstet Gynecol 2018;132:e44-52.
26. Nzelu D, Dumitrascu-Biris D, Nicolaides KH, Kametas NA. Chronic hypertension: first-trimester blood pressure control and likelihood of severe hypertension, preeclampsia, and small for gestational age. Am J Obstet Gynecol 2018;218:337.e1-7.
27. Bokslag A, Teunissen PW, Franssen C, van Kesteren F, Kamp O, Ganzevoort W, et al. Effect of early-onset preeclampsia on cardiovascular risk in the fifth decade of life. Am J Obstet Gynecol 2017;216:523.e1-7.
28. Arnadottir GA, Geirsson RT, Arngrimsson R, Jonsdottir LS, Olafsson O. Cardiovascular death in women who had hypertension in pregnancy: a case-control study. BJOG 2005;112:286-92.
29. Lopes van Balen VA, Spaan JJ, Cornelis T, Spaanderman MEA. Prevalence of chronic kidney disease after preeclampsia. J Nephrol 2017;30:403-9.