Clinical and Laboratory Parameters Associated with Eclampsia in Thai Pregnant Women

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Objective: *Determine the risk factors and pregnancy outcomes of eclampsia in our institution.*
Material and Method: *The authors conducted a case-control study of 80 Thai pregnant women with eclampsia and 240 controls with mild preeclampsia who delivered at King Chulalongkorn Memorial Hospital, Bangkok, Thailand between 1995 and 2011. Information was abstracted on established and hypothesized risk factors for eclampsia documented in the medical records.*
Results: *The risk factors that were significantly associated with eclampsia compared to mild preeclampsia after using multivariate logistic regression analysis were maternal age <20 years [adjusted odd ratio (aOR) 4.8, 95% confidence interval (CI) 1.7 to 14), antenatal care (ANC) <4 visits (aOR 3.4, 95% CI 1.2 to 9.1), deep tendon reflex (DTR) ≥3+ (aOR 15.1, 95% CI 5.3 to 42.7), serum uric acid ≥6 mg/dL (aOR 8.3, 95% CI 3.5 to 19.8), serum creatinine ≥0.9 mg/dL (aOR 18, 95% CI 4.8 to 67.5), and serum glutamate oxaloacetate transaminase (SGOT) ≥44 IU/L (aOR 15.9, 95% CI 5.6 to 45.3).*
Conclusion: *The risk factors of the development of eclampsia compared to mild preeclampsia are maternal age <20 years, ANC <4 visits, DTR ≥3+, serum uric acid ≥6 mg/dL, serum creatinine ≥0.9 mg/dL, and serum SGOT ≥44 IU/L. This information may be useful for obstetricians to predict which mild preeclamptic patients are at great risk for eclampsia and to consider administration of magnesium sulfate to prevent convulsion in these patients.*

Keywords: *Mild preeclampsia, Eclampsia, Risk factors*

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Eclampsia is one of the serious complications of hypertensive disorders of pregnancy. The incidence of eclampsia varies from 0.05% to 1% of total deliveries[1,2].

It is estimated that in developing countries 10% of all maternal deaths are associated with eclampsia[1,2].

Current management schemes designed to prevent eclampsia are based on early detection of gestational hypertension or preeclampsia and subsequent use of preventive therapy including close monitoring, use of antihypertensive drugs, timely delivery and prophylactic use of magnesium sulfate[3].

The risk factors of preeclampsia and eclampsia were usually studied together due to limited number of eclamptic patients. However, pregnancy induced hypertension in each patient has its own clinical course and only some cases of preeclampsia progress to eclampsia. This is why we need to study the characteristics to differentiate eclampsia from mild preeclampsia in order to provide better patient care.

It would be helpful if there were criteria to predict which mild preeclampsia are at great risk for eclampsia. A few studies were performed to find the risk factors including clinical and laboratory parameters that were associated with eclampsia. However, most results are incongruent. In addition, no reports in Thai populations study the risk factors of eclampsia.

The primary objective of this study is to identify the risk factors for eclampsia compared to mild preeclampsia in our population. The secondary objective is to study the outcomes and complications of pregnancy with eclampsia.

Material and Method

A case-control study was conducted at the Department of Obstetrics and Gynecology, King Chulalongkorn Memorial Hospital, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand between 1995 and 2011. This study has been approved by the Research Ethics Committee of the Faculty.

The studied cases were those diagnosed with eclampsia ascertained from all deliveries with a

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gestational age of 22 weeks or longer. Medical records of the patients with a discharge diagnosis of eclampsia were reviewed. Women with gestational epilepsy, a previous history of noneclamptic seizures, seizures attributed to encephalitis, meningitis, cerebral tumor, and women without a definite diagnosis of eclampsia were excluded.

Controls were those diagnosed with mild preeclampsia who delivered during the same period. Three cases of the control were matched to each study case on the basis of both groups having a disorder of similar etiology represent in two ends of spectrum. Exclusion criteria for controls were the same as those for cases.

Eclampsia and mild preeclampsia were defined according to criteria of the American College of Obstetricians and Gynecologists and the National High Blood Pressure Education Program Working Group on high blood pressure[4].

Data were collected regarding general information, pregnancy information, antenatal care, medical history, laboratory findings, and pregnancy outcomes.

Sample size calculation was based on risk factors for eclampsia from a previous study[1]. Serum creatinine >100 micromole/L (1.13 mg/dL) was the risk factor that gave the largest sample size. Thus, 80 woman in the present study group and 240 women in the control group were needed for detection of a statistical difference ($\alpha = 0.05$ and $\beta = 0.10$).

Hypothesized risk factors for eclampsia documented in the medical records were evaluated[1,2,5-11]. These included age, gravida, parity, gestational age of delivery, total weight gain (kg), pregestational body mass index (BMI) (kg/m^2), number of prenatal visits, history of pregnancy induced hypertension, presence or absence of medical disease, e.g., diabetes mellitus (DM), hypertension, systemic lupus erythematosus (SLE), hyperthyroidism, presence or absence of gestational DM, tobacco use before and during pregnancy, presence or absence of clinical headache, blurred vision, epigastrium pain, systolic and diastolic blood pressure (BP) levels, presence or absence of pitting edema, grading of deep tendon reflex (DTR), laboratory findings including proteinuria, hematocrit (Hct), platelet counts, serum blood urea nitrogen (BUN), serum creatinine, uric acid, serum glutamate oxaloacetate transaminase (SGOT), serum alkaline phosphatase (ALP), prothrombin time (PT) and partial thromboplastin time (PTT).

Statistical analysis

Data were presented as mean ± standard deviation, number, and percentage. Student t-test was used for continuous variables. Chi-square test and Fisher's exact test were used for categorical variables. Univariate analysis was performed to determine the relationship of a single risk factor and the occurrence of eclampsia. Then multivariate logistic regression analysis was used to evaluate the association between mild preeclamptic and eclamptic risk factors. The risk factors that produced a point estimate at a p-value of <0.05 on the univariate analysis, well used in the literatures and interesting were entered into a multivariate regression analysis. Adjusted odds (aOR) with 95% confidence interval (CI) were calculated. A p-value <0.05 was considered statistically significant. Statistical analysis was performed using SPSS 17.0.

Results

Of 138,261 births, the rate of eclampsia during the study period was 0.66 per 1,000 deliveries. Eighty pregnancies complicated by eclampsia were reviewed. Twenty-eight women had antepartum seizures (35%); 21 had seizures at home. Twenty-eight women had intrapartum seizures (35%); two of them had seizures while receiving magnesium sulfate. Twenty-four women had postpartum seizures (30%); two of them had seizures while receiving magnesium sulfate. Ten women were diagnosed with mild preeclampsia; two of them progressed to severe preeclampsia and then to eclampsia and the remaining had seizures without prodromal signs of severe preeclampsia. Laboratory findings were evaluated before the onset of seizure in 17 women. Serum uric acid was ≥6 mg/dL in 12 women, serum creatinine was ≥0.9 mg/dL in five women and SGOT was ≥44 IU/L in four women.

Demographic data of the study population are shown in Table 1. Maternal age, pregestational BMI, and gestational age were significantly lower in the eclamptic patients than in the controls. The proportion of gravidity, nulliparous was significantly higher in women with eclampsia than in the controls.

Table 2 provides the information regarding risk factors in women with eclampsia and mild preeclampsia through univariate analysis. Gravitdiy, parity, age <20 y, BMI <18.5 kg/m^2, antenatal care (ANC) <4 visits, hydrops fetalis, smoking, headache, blurred vision, epigastric pain, early onset preeclampsia (gestational age (GA) <34 weeks), systolic BP ≥160 mmHg, diastolic BP ≥110 mmHg,

presence of edema, DTR ≥3+, proteinuria ≥3+, Hct ≥40 vol%, platelet count <100,000 cells/mm^3, serum creatinine ≥0.9 mg/dL, serum BUN ≥10 mg/dL, serum uric acid ≥6 mg/dL, serum SGOT ≥44 IU/L, serum ALT ≥201 IU/L, prolonged PT, and prolonged PTT were significant associated with eclampsia compared to mild preeclampsia.

Table 3 shows the results of multivariate logistic regression analysis. The risk factors that were significant associated with eclampsia compared to mild preeclampsia were maternal age <20 years [adjusted odd ratio (aOR) 4.8, 95% confidence interval (CI) 1.7 to 14), ANC <4 visits (aOR 3.4, 95% CI 1.2 to 9.1), DTR ≥3+ (aOR 15.1, 95% CI 5.3 to 42.7), serum uric acid ≥6 mg/dL (aOR 8.3, 95% CI 3.5 to 19.8), serum creatinine ≥0.9 mg/dL (aOR 18, 95% CI 4.8 to 67.5) and serum SGOT ≥44 IU/L (aOR 15.9, 95% CI 5.6 to 45.3).

Maternal outcomes are shown in Table 4. Rate of cesarean section, maternal mortality, rate of maternal admission to ICU, length of maternal hospital stay and maternal complications including abruption placentae, HELLP (Hemolytic anemia, Elevated Liver enzyme, Low Platelet count) syndrome, pulmonary edema, intracranial hemorrhage, neurological deficits, aspiration pneumonia, and cardiopulmonary arrest were statistically higher in women with eclampsia.

Table 5 shows perinatal outcomes; perinatal mortality, rate of neonatal admission to NICU, length of neonatal hospital stay and perinatal complications including respiratory distress syndrome, intrauterine growth restriction (IUGR), sepsis, preterm delivery, low birth weight infants, stillbirth and neonatal death were statistically higher in women with eclampsia, and APGAR scores at 1 and 5 minutes, mean of birth weight and were statistically lower in women with eclampsia.

Discussion

A few studies examined risk factors for the development of eclampsia. However, no study investigates the risk factors for eclampsia in Thai population. In the present study, numerous risk factors for eclampsia were suggested by univariate analysis, but only some of these were actually established in multivariate models that control for possible confounders. In the multivariate analysis, the present study shows that maternal age <20 years old, ANC <four visits, DTR ≥3+, serum uric acid ≥6 mg/dL, serum creatinine ≥0.9 mg/dL, and serum SGOT ≥44 IU/L are significant risk factors associated with the development of eclampsia compared to mild preeclamptic women.

In the present population, young age <20 years was one of the risk factors for the development of eclampsia after adjustment for gravidity. Young maternal age is well-accepted risk factor for eclampsia[7].

Inadequate antenatal supervision was associated with a significantly increased risk of the development of eclampsia, which was consistent with the previous study[7]. Early detection of mild preeclampsia appears to play a major role in the prevention of eclampsia. Frequent prenatal visits result in prompt identification of preeclampsia and effective management of the disease through initiation of therapy such as magnesium sulfate prophylaxis.

Hypereflexia is a known clinical sign found associated with the development of eclampsia. Our results are invariable from previous studies[1,6]. Preeclampsia causes changes in the brain, which disrupts the equilibrium of the impulses between the cerebral cortex and the spinal cord. Although hyperreflexia is given much clinical attention and is found in many women before seizures, its adverse events still occur in the absence of hyperreflexia[12].

Table 1. Demographic characteristics of women in eclamptic (study) and mild preclamptic (control) groups

Variable	Eclampsia (n = 80), n (%)	Mild preeclampsia (n = 240), n (%)	p-value
Age (years, mean ± SD)	25.1±7.0	29.3±5.9	<0.001
Gravity	55 (68.75)	123 (51.25)	
Primigravida	25 (31.25)	117 (48.75)	0.006
Multigravida			
Parity			
Nulliparity	63 (78.75)	150 (62.50)	0.014
Multiparity	17 (21.25)	90 (37.50)	
Total weight gain (kg, mean ± SD)	15.4±5.8	16.8±6.5	0.078
Pregestational BMI (kg/m^2, mean ± SD)	20.2±4.0	23.9±5.1	<0.001

BMI = body mass index

Table 2. Risk factors for eclampsia compared to mild preeclampsia

Variable	Eclampsia (n = 80), n (%)	Mild preeclampsia (n = 240), n (%)	p-value
Age (years)			
<20	24 (30.00)	16 (6.67)	<0.001
20-34	45 (56.25)	181 (75.42)	
≥35	11 (13.75)	43 (17.91)	0.94
Gravity			
Primigravida	55 (68.75)	123 (51.25)	0.006
Multigravida	25 (31.25)	117 (48.75)	
Parity			
Nulliparity	63 (78.75)	150 (62.50)	0.014
Multiparity	17 (21.25)	90 (37.50)	
Total weight gain (kg)			
<8	4 (5.00)	10 (4.17)	0.409
8-19	38 (47.50)	140 (58.33)	
≥20	38 (47.50)	90 (37.50)	0.453
Pregestational BMI (kg/m^2)			
<18.5	16 (20.00)	22 (9.20)	<0.103
18.5-24.9	52 (65.00)	141 (58.80)	
25-29.9	5 (6.30)	38 (15.80)	0.008
≥30	7 (8.80)	39 (16.30)	0.061
Adequacy of prenatal care			
Poor ANC (<4 visits)	26 (32.50)	27 (11.25)	<0.001
Adequate ANC (≥4 visits)	54 (67.50)	213 (88.75)	
Gestational diabetes mellitus	2 (2.50)	14 (5.83)	0.195
Chronic hypertension	4 (5.00)	2 (0.83)	0.07
Diabetes mellitus	1 (1.25)	1 (0.42)	0.413
Systemic lupus erythematosus	1 (1.25)	2 (0.83)	0.738
Hyperthyroidism	2 (2.50)	7 (2.92)	0.845
Cigarette smoking	1 (1.25)	3 (1.25)	0.014
Multiplicity of pregnancy	3 (3.75)	12 (5.00)	0.647
Hydrops fetalis	4 (5.00)	1 (0.40)	0.004
Headache	35 (43.75)	1 (0.40)	<0.001
Blurred vision	24 (30.00)	0 (0)	<0.001
Epigastric pain	17 (21.25)	0 (0)	<0.001
Gestational age			
Early onset (<34 week)	30 (37.50)	6 (2.50)	<0.001
Late onset (≥34 week)	50 (62.50)	234 (97.50)	
Systolic BP			
<160 mmHg	20 (25.00)	214 (89.17)	
≥160 mmHg	60 (75.00)	26 (10.83)	<0.001
Diastolic BP			
<110 mmHg	38 (47.50)	233 (97.08)	
≥110 mmHg	42 (52.50)	7 (2.92)	<0.001
Pitting edema			
Absent	19 (23.75)	115 (47.92)	
Present	61 (76.25)	125 (52.08)	<0.001

BMI = body mass index; ANC = antenatal care; BP = blood pressure; DTR = deep tenton reflex; BUN = blood urea nitrogen; SGOT = serum glutamate oxaloacetate transaminase; ALP = alkaline phosphatase; PT = prothombin time; PTT = partial prothombin time

Table 2. (cont.)

Variable	Eclampsia (n = 80), n (%)	Mild preeclampsia (n = 240), n (%)	p-value
DTR			
1+	12 (15.00)	26 (10.83)	
2+	36 (45.00)	203 (84.58)	0.078
3+	32 (40.00)	11 (4.59)	<0.001
Proteinuria			
≤2+	34 (42.50)	231 (96.25)	
≥3+	46 (57.50)	9 (3.75)	<0.001
Hematocrit			
<40 vol%	42 (52.50)	191 (79.58)	
≥40 vol%	38 (47.50)	49 (20.42)	<0.001
Platelet			
<100,000/mm^3	16 (20.00)	3 (1.25)	<0.001
≥100,000/mm^3	64 (80.00)	237 (98.75)	
Serum creatinine			
>1.13 mg/dL	4 (5.00)	1 (0.42)	<0.001
0.9-1.13 mg/dL	17 (21.25)	6 (2.50)	<0.001
<0.9 mg/dL	59 (73.75)	233 (97.08)	
BUN			
<10 mg/dL	38 (47.50)	150 (62.50)	
≥10 mg/dL	42 (52.50)	90 (37.50)	0.003
Uric acid			
<6 mg/dL	14 (17.50)	150 (62.50)	
≥6 mg/dL	66 (82.50)	90 (37.50)	<0.001
SGOT			
≥76 IU/L	19 (23.75)	5 (2.08)	<0.001
44-75 IU/L	14 (17.50)	4 (1.67)	<0.001
<44 IU/L	47 (58.75)	231 (96.25)	
ALP			
<201 IU/L	37 (46.25)	178 (74.17)	
≥201 IU/L	43 (53.75)	62 (25.83)	<0.001
PT			
Normal	73 (91.25)	240 (100)	
Prolonged	7 (8.75)	0 (0)	<0.001
PTT			
Normal	75 (93.75)	240 (100)	
Prolonged	5 (6.25)	0 (0)	<0.001

BMI = body mass index; ANC = antenatal care; BP = blood pressure; DTR = deep tenton reflex; BUN = blood urea nitrogen; SGOT = serum glutamate oxaloacetate transaminase; ALP = alkaline phosphatase; PT = prothombin time; PTT = partial prothombin time

Table 3. Results of multivariate logistic regression analysis

Risk factors	Adjusted OR	95% CI	p-value
Age <20 years	4.8	1.7-14	<0.001
ANC <4 visits	3.4	1.2-9.1	0.017
DTR ≥3+	15.1	5.3-42.7	<0.001
Serum uric acid ≥6 mg/dL	8.3	3.5-19.8	<0.001
Serum creatinine ≥0.9 mg/dL	18.0	4.8-67.5	<0.001
SGOT ≥44 IU/L	15.9	5.6-45.3	<0.001

Table 4. Maternal outcomes in eclamptic (study) and mild preeclamptic (control) groups

Maternal outcomes	Eclampsia (n = 80), n (%)	Mild preeclampsia (n = 240), n (%)	p-value
Vaginal delivery	17 (21.30)	127 (52.90)	<0.001
Cesarean section	63 (78.80)	113 (47.10)	<0.001
Maternal hospital stay (days, mean ± SD)	8.0±3.0	4.9±2.9	<0.001
Maternal admission to ICU	80 (100)	0 (0)	<0.001
Maternal mortality	2 (2.50)	0 (0)	<0.001
Abruptio placentae	1 (1.25)	0 (0)	<0.001
HELLP syndrome	16 (20.00)	0 (0)	<0.001
Pulmonary edema	4 (5.00)	0 (0)	<0.001
Intracerebral hemorrhage	2 (2.50)	0 (0)	<0.001
Neurological deficits	2 (2.50)	0 (0)	<0.001
Aspiration pneumonia	1 (1.25)	0 (0)	<0.001
Cardiopulmonary arrest	2 (2.50)	0 (0)	<0.001

HELLP = hemolytic anemia, elavated liver enzyme, low platelet

Table 5. Perinatal outcomes in eclamptic (study) and mild preclamptic (control) groups

Perinatal outcomes	Eclampsia (n = 80), n (%)	Mild preeclampsia (n = 240), n (%)	p-value
Apgar score <7 (mean ± SD)			
1 min	6.4±2.8	8.9±0.3	<0.001
5 min	8.3±2.6	10.0±2.4	<0.001
Birth weight (gm, mean ± SD)	2,249±757	3,000±561	<0.001
Low birth weight (<2,500 gm)	50 (62.50)	35 (14.60)	<0.001
Admission to NICU	19 (23.80)	0 (0)	<0.001
Neonatal hospital stay (days, mean ± SD)	11.3±10.3	4.9±2.9	<0.001
Perinatal mortality	6 (7.50)	1 (0.40)	<0.001
Respiratory distress syndrome	11 (13.75)	4 (1.60)	<0.001
Intrauterine growth restriction	16 (20.00)	12 (5.00)	<0.001
Sepsis	5 (6.25)	5 (2.08)	<0.001
Preterm delivery	42 (52.50)	43 (17.90)	<0.001
Stillbirth	4 (5.00)	1 (0.40)	<0.001
Neonatal death	6 (7.50)	0 (0)	<0.001

NICU = neonatal intensive care unit

The authors found that hyperuricemia (serum uric acid ≥6 mg/dL) was a risk factor in the development of eclampsia compared to mildly preeclamptic patients. Although hyperuricemia appears to be a marker for renal tubular damage in women with severe preeclampsia, it may be a marker for cerebral ischemia in women with eclampsia. Bainbridge SA and Roberts JM[13] reported that hyperuricemia appears to identify a subset of preeclamptic women who are at greater risk for maternal and fetal morbidities. In addition, hyperuricemia in pregnant women without proteinuria is at least as good a predictor of fetal morbidity as hypertension and proteinuria. Several pathogenic effects of uric acid from in vitro culture studies and hyperuricemic animal models, including pro-inflammatory effects, stimulation of smooth muscle cell proliferation, inhibition of endothelial cell proliferation and migration, promotion of endothelial dysfunction and damage, may all play pivotal roles in the pathophysiology of preeclampsia[13].

In present study, serum creatinine ≥0.9 mg/dL was a significant risk factor associated with the

development of eclampsia compared to mild preeclamptic women. This was consistent with a former study[11]. According to renal physiology of pregnancy, a value of serum creatinine ≥0.9 mg/dL suggests an underlying renal disease and should prompt further evaluation[14]. In women with pregnancy-induced hypertension, if not properly managed, renal damage can occur as a consequence of prolonged renal ischemia and hypoperfusion.

The rising serum SGOT (≥44 IU/L) was also a significant risk factor associated with the development of eclampsia compared to mild preeclamptic women. This was also consistent with the previous study[10]. Liver cell damage due to vasospasm may be an explanation of the rising in severe case.

Primigravida was a significant risk factor of eclampsia in univariate analysis as in the previous study[7], but not in multivariate analysis in our study. Maternal first exposure to trophoblasts, which are of fetal origin is supposed to be one of the etiology of pregnancy-induced hypertension[9].

The clinical prodrome of seizures, which are headache, epigastrium pain, and blurred vision, were not the risk factors for development of eclampsia when analyzed by multiple logistic regression. Only one woman with mild preeclampsia did have these prodromes in contrast to the eclamptic cases; 43%, 21%, and 30% of eclamptic women had headache, epigastric pain, and blurred vision, respectively.

The criteria of the severity of hypertension and proteinuria, frequently diagnosed before the onset of eclampsia were not the risk factors for the development of eclampsia. However, in women with eclampsia, 75% had systolic BP ≥160 mmHg, 52% had diastolic BP ≥110 mmHg and 57% had proteinuria ≥3+.

Eclampsia is still responsible for significantly poor maternal and perinatal outcomes in both developed and developing countries. The present study demonstrates that maternal and perinatal outcomes include the rate of cesarean section, perinatal and maternal mortality, rate of neonatal admission to NICU, rate of maternal admission to ICU, length of neonatal hospital, length of maternal hospital stay, maternal and perinatal complications including abruption placentae, HELLP syndrome, pulmonary edema, intracranial hemorrhage, neurological deficits, aspiration pneumonia, cardiopulmonary arrest, respiratory distress syndrome, intrauterine growth restriction, sepsis, preterm delivery, low birth weight infants, stillbirth and neonatal death were statistically higher in women with eclampsia, and APGAR scores at 1 and 5 min, mean of birth weight are statistically lower in eclamptic women.

The strength of the present study is its large sample size that confers sufficient power to evaluate the risk factors for eclampsia. The large database allowed us to analyze risk factors with a high prevalence and those with low prevalence. The current study also had the ability to control for the influence of possible confounding factors.

Several limitations and potential biases of this study were unable to evaluate some risk factors for eclampsia previously reported such as change in paternity, family history of eclampsia, because these data were not available from the medical records.

In conclusion, maternal age <20 years, ANC < four visits, DTR ≥3+, serum uric acid ≥6 mg/dL serum creatinine ≥0.9 mg/dL, and serum SGOT ≥44 IU/liter are significant risk factors associated with the development of eclampsia compared to mild preeclamptic women. This information may be useful for obstetricians to predict whether mild preeclamptic patients are at great risk for eclampsia and to consider administration of magnesium sulfate to prevent convulsion in these patients.

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Potential conflicts of interest

None.

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อาการทางคลินิกและผลการตรวจทางห้องปฏิบัติการที่เกี่ยวข้องกับภาวะชักในภาวะครรภ์เป็นพิษในสตรีตั้งครรภ์ไทย

พรรณี ทวีสุข, เยื้อน ตันนิรันดร

วัตถุประสงค์: *เพื่อหาปัจจัยเสี่ยงของการชักและผลลัพธ์ของการตั้งครรภ์ในผู้ป่วยครรภ์เป็นพิษชนิดไม่รุนแรงในโรงพยาบาลจุฬาลงกรณ์*

วัสดุและวิธีการ: *ได้ทำการศึกษาแบบ case-control ในหญิงตั้งครรภ์ไทยที่มีภาวะครรภ์เป็นพิษแทรกซ้อนด้วยอาการชัก 80 ราย และหญิงตั้งครรภ์ที่มีภาวะครรภ์เป็นพิษไม่รุนแรง 240 ราย ที่คลอดในโรงพยาบาลจุฬาลงกรณ์ ระหว่าง พ.ศ. 2538 ถึง พ.ศ. 2554 โดยได้ทำการเก็บข้อมูลจากเวชระเบียนของผู้ป่วยในส่วนที่เกี่ยวข้องกับการเป็นปัจจัยเสี่ยงในการเกิดภาวะชักในภาวะครรภ์เป็นพิษ*

ผลการศึกษา: *ปัจจัยเสี่ยงของการชักในผู้ป่วยครรภ์เป็นพิษชนิดไม่รุนแรงที่ผ่านการวิเคราะห์การถดถอยพหุโลจิสติกส์ (multivariate logistic regression analysis) คือ อายุมารดาน้อยกว่า 20 ปี [adjusted odds ratio (aOR) 4.8, 95% confidence interval (CI) 1.7 to 14] จำนวนฝากครรภ์น้อยกว่า 4 ครั้ง (aOR 3.4, 95% CI 1.2 to 9.1) รีเฟล็กซ์เอ็นลึกตั้งแต่ระดับ 3 ขึ้นไป aOR 15.1, 95% CI 5.3 to 42.7) ระดับกรดยูริกในกระแสเลือดมากกว่าหรือเท่ากับ 6 มิลลิกรัมต่อเดซิลิตร (ORs 8.3; 95% CI 3.5-19.8) ระดับค่าซีรัมครีแอทินินในกระแสเลือดมากกว่าหรือเท่ากับ 0.9 มิลลิกรัมต่อเดซิลิตร (ORs 18; 95% CI 4.8-67.5) และระดับค่าซีรัมแกมม่า กลูตามิกออกซาโลอะซิติกทรานซามิเนส (SGOT) มากกว่าหรือเท่ากับ 44 ไอยูต่อลิตร (aOR 15.9, 95% CI 5.6 to 45.3)*

สรุป: *ปัจจัยเสี่ยงของการชักในผู้ป่วยครรภ์เป็นพิษชนิดไม่รุนแรง คือ อายุมารดาน้อยกว่า 20 ปี จำนวนฝากครรภ์น้อยกว่า 4 ครั้ง รีเฟล็กซ์เอ็นลึกตั้งแต่ระดับ 3 ขึ้นไป ระดับกรดยูริกในกระแสเลือดมากกว่าหรือเท่ากับ 6 มิลลิกรัมต่อเดซิลิตร ระดับค่าซีรัมครีแอทินินในกระแสเลือดมากกว่าหรือเท่ากับ 0.9 มิลลิกรัมต่อเดซิลิตร และระดับค่าซีรัมแกมม่า กลูตามิกออกซาโลอะซิติกทรานซามิเนส (SGOT) มากกว่าหรือเท่ากับ 44 ไอยูต่อลิตร ปัจจัยเสี่ยงดังกล่าวนี้จะเป็นประโยชน์ต่อสูติแพทย์ในการพยากรณ์ว่าผู้ป่วยครรภ์เป็นพิษชนิดไม่รุนแรงรายใดจะมีความเสี่ยงที่จะเกิดการชัก และทำการพิจารณาให้แมกนีเซียมเพื่อป้องกันการชักในผู้ป่วยครรภ์เป็นพิษชนิดไม่รุนแรงเหล่านี้*
