OUTCOME OF PREGNANCIES WITH HYPERTENSIVE DISORDERS

NATDHANAN PHATKRATHOK

A THESIS SUBMITTED IN PARTIAL FULFILLMENT

OF THE REQUIREMENTS FOR

THE DEGREE OF MASTER OF SCIENCES (EPIDEMIOLOGY)

FACULTY OF GRADUATE STUDIES

MAHIDOL UNIVERSITY

2000

ISBN 974-665-001-7

Thesis
entitled

OUTCOME OF PREGNANCIES WITH HYPERTENSIVE DISORDERS

Natdhanan Phatkrathok

Miss Natdhanan Phatkrathok
Candidate

Surapol Suwanagool

Prof. Surapol Suwanagool
M.D., M.P.H., F.A.C.P.
Major Advisor

Jayanton Patumanond

Asst. Prof. Jayanton Patumanond
M.D., M.P.H., D.Sc. (Clin Epidemiol)
Co-advisor

Chamaiporn Tawichasri

Asst. Prof. Chamaiporn Tawichasri
M.Sc. (Environmental Management)
Co-advisor

Soysaang Sethavanich

Dr. Soysaang Sethavanich
M.D., Dip Thai Brd Ob & Gyn
Co-advisor

Prakairat Sukumalchart

Asst.Prof. Prakairat Sukumalchart, M.S.
Acting Dean
Faculty of Graduate Studies

Surapol Suwanagool

Prof. Surapol Suwanagool
M.D., M.P.H., F.A.C.P.
Chairman
Master of Science Programme in
Epidemiology Faculty of Medicine,
Siriraj Hospital

Thesis
entitled

OUTCOME OF PREGNANCIES WITH HYPERTENSIVE DISORDERS

Was submitted to the Faculty of Graduate Studies, Mahidol University
for the degree of Master of Science (Epidemiology)
On
Nov 6, 2000

Natdhanan Phatkrathok

Miss Natdhanan Phatkrathok
Candidate

Lect. Wichai Techasathit
M.D., M.P.H. (Epidemiol)
Dip Thai Brd Int Med
Chairman

Jayanton Patumanond

Asst. Prof. Jayanton Patumanond
M.D., M.P.H., D.Sc. (Clin Epidemiol)
Member

Chamaiporn Tawichasri

Asst. Prof. Chamaiporn Tawichasri
M.Sc. (Environmental Management)
Member

Pharuhas Chanprapaph

Lect. Pharuhas Chanprapaph
M.D., Dip Thai Brd of Ob & Gyn
Member

Prakairat Sukumalchart

Asst.Prof. Prakairat Sukumalchart, M.S.
Acting Dean
Faculty of Graduate Studies
Mahidol University

P. Sakolsatayadorn

Prof. Piyasakol Sakolsatayadorn
M.D., Grad Dip Sc (Surg),
Dip Thai Brd Surg., F.R.C.S.T.
Dean
Faculty of Medicine, Siriraj Hospital
Mahidol University

ACKNOWLEDGEMENT

I would like to express my deepest gratitude and sincere appreciation to Asistant Professor Dr. Jayanton Patumanond and Asistant Professor Chamaiporn Tawichasri, Division of Epidemiology and Statistics, Department of Community Medicine, Faculty of Medicine, Chaingmai University, my principle supervisor for their invaluable advice, encouragement and continuous valuable guidance throughout this study. I am also grateful to Professor Surapol Suwanagool, Lecture Wichai Techasathit, Department of Preventive Medicine, Faculty of Siriraj Medicine, for their support, invaluable advice and guidance.

I am very grateful and indebted to Dr. Soysaang Sethavanich, the director of The Mother and Child Hospital, Chaingmai, and Lecture Pharuhas Chanprapap, Department of Obstetric & Gynecology, Faculty of Medicine, Chaingmai University, for their guidance and comment of this thesis.

Sincere gratitude is extended to the director of The Mother and Child Hospital, statistical record units, and all staff of The Mother and Child Hospital for their kind cooperation and friendly assistance, which made this study possible.

I would like to thank Lecture Wichai Techasathit, and Dr. Gulam Rasul, D.T.M. & H., M. P. H., for their advice in regard to English language usage.

Finally, my efforts for this thesis are dedicated to my parent and members in my family for their unending love, never lacking in kindness and support.

Natdhanan Phatkrathok

4137188 SIEP/M :MAJOR : EPIDEMIOLOGY ; M.Sc. (EPIDEMIOLOGY)
KEY WORDS :HYPERTENSIVE DISORDERS OF PREGNANCY / PREGNANCY OUTCOME / NEONATAL OUTCOME

NATDHANAN PHATKRATHOK: OUTCOME OF PREGNANCIES WITH HYPERTENSIVE DISORDERS. THESIS ADVISORS: SURAPOL SUWANAGOOL, M.D., F.A.C.P., JAYANTON PATUMANOND, M.D., D.Sc., CHAMAIPORN TAWICHASRI, M.Sc., SOYSAANG SETHAVANICH, M.D.
126 P. ISBN 974-665-001-7.

Hypertensive disorders of pregnancy are important causes of both maternal and neonatal morbidity and mortality worldwide. It is the second most common cause of maternal mortality countrywide in Thailand. The aim of this retrospective cohort study was to explore the outcome of pregnancy in hypertensive pregnancies as compared to normotensive pregnancies and their offspring. The subjects were 482 pregnant women who attended an antenatal care clinic and delivered at the Mother and Child Hospital, Chiang Mai from December 1,1997 to April 30, 1999. The hypertensive group included 164 with pregnancy-induced hypertension and 6 with chronic hypertension. Maternal and neonatal medical records were reviewed for demographic variables, antepartal, intrapartal, postpartal complication and neonatal variables. Data were presented in frequency, percentage, mean and standard deviation and analyzed with multivariate regression.

Chronic hypertensive pregnancies had 3.86 weeks shorter gestational duration (95% CI = 2.03 to 5.68), an increased risk of prematurity of 5.70 times (95% CI = 1.90 to 17.10), abnormal mode of delivery of 3.10 times (95% CI = 1.08 to 8.94), indication for abnormal delivery of 3.49 times (95% CI = 1.33 to 9.13). Pregnancy-induced hypertensive pregnancies were at an increase in risk of abnormal mode of delivery 1.52 times (95% CI = 1.10 to 2.08), indication for abnormal delivery 1.51 times (95 CI = 1.10 to 2.06), prolonged second stage in nullipara 2.81 times (95% CI = 1.12 to 7.01), 20.86 minutes longer duration of second stage labour (95%CI = 5.70 to 36.01), and 1.62 minutes in the third stage (95% CI = 0.11 to 3.13), 30.32 cc more estimated blood loss (95% CI = 5.17 to 55.47). The infants born to chronic hypertensive mothers weighted 518.30 grams or less (95% CI = 844.24 to 192.32), 2.05 cm. smaller head circumferences (95 %CI = 3.29 to 0.76), and 4.15 times increased in risk of low birth weight (95%CI = 1.01 to 17.10), and 78.0 times congenital anomalies (95% CI = 13.03 to 466.80).

Women with hypertensive pregnancies should be advised to attend antenatal care and an appropriate intervention should be given early, to avoid an undesirable outcome of pregnancy.

4137188 SIEP/M : สาขาวิชา : วิทยาการระบาด : วทม. (วิทยาการระบาด)

ณัฏฐนันท์ แพทย์กระโทก : ผลการตั้งครรภ์ของหญิงที่มีภาวะความดันโลหิตสูง (OUTCOME OF PREGNANCIES WITH HYPERTENSIVE DISORDERS). คณะกรรมการควบคุมวิทยานิพนธ์ : สุรพล สุวรรณกูล, M.D., F.A.C.P., ชยันตร์ธร ปทุมานนท์, M.D., D.Sc., ชไมพร ทวิชศรี, M.Sc., สร้อยสอาง เศรษฐวานิช, M.D. 126 หน้า ISBN 974-665-001-7

ภาวะความดันโลหิตสูงในหญิงตั้งครรภ์เป็นสาเหตุสำคัญของอัตราการป่วยและการตายของมารดาและทารกทั่วโลก และเป็นสาเหตุการตายอันดับที่สองของมารดาในประเทศไทย การศึกษาไปข้างหน้าแบบย้อนหลังเพื่อศึกษาผลของการตั้งครรภ์และสภาวะสุขภาพของทารกของหญิงที่มีภาวะความดันโลหิตสูงเปรียบเทียบกับหญิงปกติ กลุ่มตัวอย่างเป็นหญิงตั้งครรภ์จำนวน 482 ราย ที่มารับบริการฝากครรภ์และคลอดในโรงพยาบาลแม่และเด็กจังหวัดเชียงใหม่ระหว่างวันที่1 ธันวาคม พ.ศ. 2540 ถึง 30 เมษายน พ.ศ. 2542 หญิงตั้งครรภ์ที่มีภาวะความดันโลหิตสูงในการศึกษาประกอบด้วย หญิงที่มีภาวะความดันโลหิตสูงขณะตั้งครรภ์ 164 ราย และหญิงที่มีภาวะความดันโลหิตสูงเรื้อรัง 6 ราย รวบรวมข้อมูลของมารดาและทารกจากบันทึกการแพทย์ ซึ่งประกอบด้วยข้อมูลพื้นฐานภาวะแทรกซ้อนระหว่างตั้งครรภ์ ระยะคลอด ระยะหลังคลอด และสภาวะสุขภาพของทารก วิเคราะห์ข้อมูลโดยใช้ความถี่ สัดส่วน ค่าเฉลี่ย ค่าเบี่ยงเบนมาตรฐาน และการวิเคราะห์ถดถอยพหุ

หญิงตั้งครรภ์ที่มีภาวะความดันโลหิตสูงเรื้อรังมีระยะเวลาการตั้งครรภ์สั้นกว่า 3.86 สัปดาห์ (95%CI = 2.03 ถึง 5.68) มีโอกาสคลอดก่อนกำหนดเพิ่มขึ้น 3.10 เท่า (95%CI = 1.90 ถึง17.10) มีโอกาสคลอดผิดปกติ 3.10 เท่า (95% CI = 1.08 ถึง 8.94) มีข้อบ่งชี้ทำให้ไม่สามารถคลอดปกติ 3.49 เท่า (95%CI = 1.33 ถึง 9.13) หญิงตั้งครรภ์ที่มีภาวะความดันโลหิตสูงขณะตั้งครรภ์มีโอกาสคลอดผิดปกติ 1.52 เท่า (95%CI = 1.70 ถึง 2.08) มีข้อบ่งชี้ทำให้ไม่สามารถคลอดปกติ 2.81 เท่า (95% CI = 1.12 ถึง 7.01) นอกจากนี้ยังมีระยะเวลาการคลอดในระยะที่ 2 นานกว่า 20.86 นาที (95% CI = 5.70 ถึง 36.05) และระยะที่ 3 นานกว่า 1.62 นาที (95% CI = 0.11 ถึง 3.13) มีปริมาณการเสียเลือดมากกว่า 21.34 ลูกบาศก์มิลลิเมตร (95% CI = 2.18 ถึง 40.50) และมีน้ำหนักรกมากกว่า 30.32 กรัม (95% CI = 5.17 ถึง 55.47) ทารกที่คลอดจากมารดาที่มีภาวะความดันโลหิตสูงเรื้อรังมีน้ำหนักแรกคลอดน้อยกว่า 518.30 กรัม (95% CI = 844.24 ถึง 192.32) ความยาวรอบศีรษะน้อยกว่า 2.02 ซม. (95% CI = 3.29 ถึง 0.76) เป็นทารกน้ำหนักน้อย 4.15 เท่า (95% CI = 1.01 ถึง 17.10) และมีความผิดปกติแรกคลอดมากกว่า 78.0 เท่า (95% CI = 13.03 ถึง 466.80)

ควรแนะนำให้หญิงตั้งครรภ์ที่มีภาวะความดันโลหิตสูงมารับบริการฝากครรภ์ตั้งแต่เริ่มตั้งครรภ์และให้การดูแลอย่างเหมาะสม เพื่อลดปัญหาผลการตั้งครรภ์ที่ไม่พึงประสงค์

CONTENTS

LIST OF TABLES

LIST OF TABLES (CONTINUED)

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Background

More women die under the child-bearing each year in South Asia than in any other part of the world. Each time a women in South Asia becomes pregnant; she faces a risk of dying that is 31 times higher than the developed world and that one out of ever 38 South Asian women die due to pregnancy related causes (1).

In Thailand, maternal and child health is a major public health problem, because they also face a risk of dying due to pregnancy and obstetrics-related complications. As a result of improved public health care, the maternal mortality rate had declined from 0.36 to 0.17 per 1,000 live births during the period 1990-1994. It was the success of the 7th public health plan (1992-1996), which was one of the plan that reduced maternal death rate at pregnancy and delivery from 0.4 to 0.3 per 1,000 live births (2). Study conducted in each region found that the maternal death rate in the southern and northern region were higher than the other region (table1), and the fetal death rate in the northern region was the highest rate in 1997(table 2) (3,4).

Table 1 The number and maternal death rate per 100,000 live births in 1994-1997.

Region	1994		1995		1996		1997	
	n	ratio	n	ratio	n	ratio	n	ratio
Central	16	10.1	19	11.9	20	12	16	8.5
North-eastern	39	13.8	20	10.6	16	10.7	25	9.0
Northern	27	23.9	34	29.2	15	14.4	31	24.3
Southern	34	29	24	21.7	34	28.6	29	25.1
Total	116	17.28	97	16.8	85	15.8	101	14.2

Source: Kanshana S. etal. Maternal mortality in Thailand, 1994-1997.

Table 2 The fetal death rate per 1,000 total births in 1994-1997.

Region	1994	1995	1996	1997
Central	9.2	9.8	10.2	8.9
North-eastern	12.7	11.2	10.8	9.5
Northern	13.6	13.2	11.4	12.3
Southern	11.0	11.2	9.7	9.3
Country	11.7	11.2	10.01	9.0
Total	7,949	6,500	5,426	7,03

Source: Kanshana S. etal. Maternal mortality in Thailand, 1994-1997.

Although the maternal mortality rate has declined, the main causes of maternal death remained the same throughout 1989-1993. The Ministry of Public Health reported that obstructed labour, haemorrhage and pregnancy-induced hypertension (PIH), are the 3 leading causes of direct maternal death in Thailand (5). Hypertensive disorders of pregnancy are important causes of both maternal and neonatal morbidity and mortality worldwide. It is the second most common cause of maternal mortality countrywide in Thailand (1).

Pregnancy-induced hypertension occurs in approximately 6-7% and chronic hypertension in 0.5-3% of all pregnancies (1). However, the causes and pathology of maternal hypertension are largely unknown, and the classification and diagnosis of hypertensive disease in pregnancy are complex. Hypertension appears to be an important determinant of maternal and neonatal outcome. Other maternal complications of hypertension included pulmonary oedema, abruptio placenta, hemolysis, elevated liver enzymes, low platelet count (HELL) syndrome, maternal death, acute renal failure, coma with cerebral pathology and disseminated intravascular coagulation (DIC) (6-9). The perinatal complications include preterm delivery, low birth weight, intrauterine growth retardation (IUGR), still birth, and perinatal morbidity and mortality (7,8,10-26). Besides, the effect of hypertensive disorders on pregnancy and the outcome of such pregnancies are well established, while such adverse outcomes were not consistent and still remain debatable among studies.

Therefore, a strategy for reducing maternal and neonatal mortality and morbidity should be developed to prevent the effect of hypertensive disorders on pregnancy outcome. The preventive strategy should be applied from early pregnancy to postpartum period. The result obtained from this study is likely to provide information for health care providers to use in the planning of appropriate and effective intervention programs for the prevention and management of hypertensive pregnant mothers and their children. An effective intervention program for hypertensive pregnant mothers will contribute to the reduction of maternal and child mortality.

Objective

1. To explore the effect of hypertensive disorders of pregnancy on maternal outcome such as pregnancy progression, obstetric outcome, pregnancy outcome in postpartum period, etc.

2. To study the effect of hypertensive disorders of pregnancy on neonatal outcome such as birth weight, neonatal length, head circumferences, neonatal complications, hyperbilirubinemia, neonatal morbidity, etc.

Hypotheses

1. The outcome of pregnancy in hypertensive pregnant women is different from normotensive women.

2. Health status of the infants born to hypertensive mothers are different from those born to normotensive mothers.

Variables

Independent variable: Hypertensive disorders of pregnancy

Dependent variables:

- Pregnancy progression
- Pregnancy outcome
- Obstetric outcome
- Neonatal outcome
- Maternal health status in postpartum period
- Neonatal health status in postpartum period

Confounding variables:

- Maternal age
- Gravida
- Gestational age
- Maternal weight gain
- Birth weight, etc.

Definition of terms

The operational definitions of the terms used in this study are as follow:

Hypertensive disorders of pregnancy(HDP): are characterized by hypertension, oedema, and proteinuria, and, in some cases convulsion and coma. The classification and terminology of the hypertensive disorders of pregnancy are different and confusing. In clinical practice, however, only two distinct conditions are commonly recognized: chronic hypertension and pregnancy-induced hypertension (PIH) (27). These are also used in this study. The definition of hypertension in this study was guided by the committee on terminology of American College of Obstetrician and Gynecologists (ACOG), means an elevation of systolic blood pressure of 140 mmHg and above; or a diastolic blood pressure of 90 mmHg and above (6,28).

Chronic hypertension: is diagnosed when hypertension is known to be present before or during early 20 weeks of gestation (29).

Pregnancy-induced hypertension (PIH): defined as a blood pressure of 140/90 or above accounting for the first time during pregnancy (after 20 weeks of gestation); may be associated with generalized oedema, and/or proteinuria in which cases the criteria for a diagnosis of preeclampsia are present (30).

Gestational age: is the duration from the first day of the last menstruation to the time of labour or termination of pregnancy (31).

Primary postpartum hemorrhage: blood loss in excess of 500 ml (or blood loss of 1 percent of body weight) during the third stage of labor (32-33).

Puerperium infection: An infection of the genital tract arising as a complication of childbirth. The definition of a puerperium infection is when the temperature reaches 38°C on any 2nd of the first 10 days after delivery (postpartum), exclusive of the first 24 hours (32,34).

Pre-term infant: defined as an infant born before 37 completed weeks of gestation.

Term infant: defined as an infant born anytime after 37 completed weeks of gestation through 42 completed weeks of gestation (32,34-36).

Low birth weight infant: defined as an infant who has a birth weight less than 2,500 grams (32,34-36).

Normal birth weight infant: defined as an infant who has a birth weight more than 2,500 grams (32,34-36).

Live birth: An infant who breathes after birth or shows any other sign of life such as heartbeat or definite spontaneous voluntary movement (34).

Still birth: An infant born weighing at least 500 gm at delivery who did not breathe after birth or show any other sign of life (34).

Birth asphyxia: Newborn who has Apgar score of 7 or less after these first few minutes of life will be considered of having birth asphyxia (37-39).

Hyperbillirubinemia: Condition of increased billirubin level to more than 12 mg/100ml in premature birth, and 14 mg/100ml in term infants (37-39).

CHAPTER II

LITERATURE REVIEW

This research studies the outcome of pregnancies with hypertensive disorders. The topics covered in this literature review are:

1. Pregnancy progression
2. Pregnancy complication
3. Birth outcome
4. Neonatal outcome
5. Maternal and neonatal outcome in postpartum period

1. Pregnancy progression

During pregnancy the pregnant women have anatomical, chemical and physiological changes. Physiological changes during prenatal care were review as follow:

Weight Gain during Pregnancy

In general, weight gain increases about 1 pound per week in normal (40), when weight gain exceeds more than 2 pounds in any given week, or 6 pounds in a month, chances of developing preeclampsia should be suspected. The suddenness of excessive weight gain is a characteristic of preeclampsia rather than an increase distribution throughout gestation. A sudden increase in weight may precede the development of preeclampsia, and indeed, excessive weight gain in some women is the first sign. (32)

In chronic hypertension, Anyaegbunam AM, et al. studied the influence of chronic hypertension on outcome of pregnancy in women with gestational diabetes. They found the significant difference ($p < 0.005$) in mean maternal weight when hypertensive gestational diabetes women were compared to all nonhypertensive gestational diabetes women (90 ± 21.2 vs 70.6 ± 14.9 kg) (10). Ravi Thadhani, et al. found the risk of gestational hypertension increased as pregravid BMI increased ($p < 0.01$) (42).

Cardiovascular changes

During normal pregnancy, arterial blood pressure and vascular resistance decrease while blood volume, maternal weight, and basal metabolic rate increase (32,43). Cardiac output begins to rise during the first 10 weeks of pregnancy, reaching a peak of 30-50% above resting, nonpregnant levels at around the 20^{th} week (43-45). Even though cardiac output is elevated during pregnancy, the reduced systemic vascular resistance results in either a fall or no change in blood pressure, although the pulse pressure widen (44). Blood pressure is a product of cardiac output and total peripheral resistance. Blood pressure in the brachial artery varies when sitting or lying in the lateral recumbent supine position. Usually, arterial blood pressure decreases to a nadir at about mid-pregnancy and rises thereafter (Fig.1) (32,46-47).

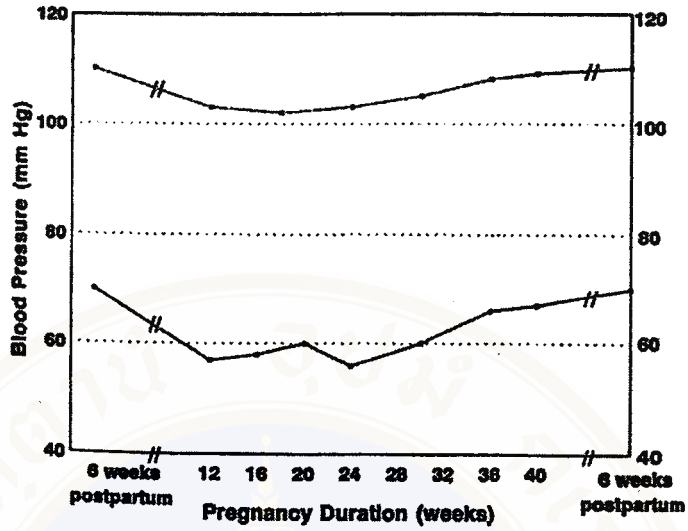

Figure 1. Blood pressure change as a result of pregnancy. Pressures of 6 weeks are shown before and after pregnancy.

During normal pregnancy total peripheral resistance decreases by 25% whereas in PIH it increases. This increase peripheral vascular resistance appears to be the main cause for the blood pressure elevation seen in PIH (28,45). In most women with chronic hypertension, the blood pressure falls by the second trimester, but the decrement is usually temporary (Fig.2) (32,44,48).

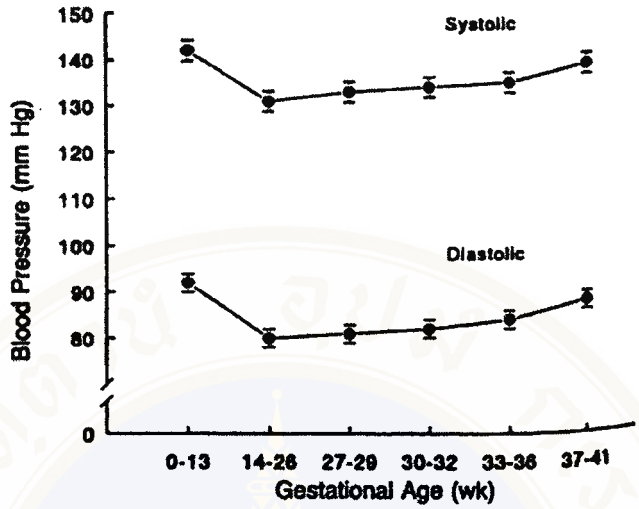

Figure 2. Systolic and Diastolic blood pressure changes during pregnancy in 90 chronically hypertensive women who were given no antihypertensive treatment. (Taken from Cunningham and Lowe, 1991;data from Saibai and colleagues, 1990b).

Hematological changes

Women of average size should have a blood volume of nearly 5000 ml. during the last several weeks of a normal pregnancy, compared with about 3500 ml when nonpregnant. With eclampsia, however, much or all of the anticipated 1500 ml of blood normally present late in pregnancy is absent (30,32,33). During the first trimester, blood volume begin to increase, reaching a level about 40% above nonpregnant values at 30^{th} week of gestation. There after, blood volume remains stable or even decrease slightly until term. Part of the increase in volume results form an increase in the number of erythrocytes, which rises by 20% - 40%. But a larger portion of that increase is caused by an expansion of plasma volume, which climbs to

a level about 50% greater than nonpregnant values by the 32nd week of pregnancy. The relative difference in red blood cells and plasma volume changes accounts for the " *physiologic anemia of pregnancy*" (44). In PIH this expansion in blood volume is reduced (16%). The hemoconcentration seen in PIH can result in decreased regional perfusion. In these patients, in the absence of hemorrhage, the intravascular compartment is not under filled due to the increase in vascular tone and vasospasm. Clinically this is reflected by an increasing hematocrit as the severity of PIH rises (32,44). During normal pregnancy the hematocrit falls from around 40% to 33% and then rises in the third trimester. The hematocrit in severe preeclampsia and eclampsia tends to increase, indicating a possible decrease in plasma volume and sequestration of fluid into the third space. Serial hematocrits in selected patients may be helpful in determining the severity and progression of disease (6,28,43).

2. Pregnancy Complication

Severe pre-eclampsia and ecampsia are still responsible for considerable maternal morbidity and mortality. Maternal morbidity includes severe bleeding from abruptio placenta with its resultant coagulopathy, pulmonary edema, aspiration pnenmonia, acute renal failure, cerebrovascular hemorrhage, liver rupture and retinal detachment (27,45). Derham RJ, et al (1989) showed that 106 patients with severe hypertension in pregnancy requiring delivery between 26 and 34 weeks for cardiotocographic fetal distress or unstable maternal blood pressure (7). In South Africa, Buga GA and Lumu SB studied the incidence, clinical features, complications and periantal outcome in patients with hypertensive disorders of pregnancy.

They found that the maternal complications of hypertension included, pulmonary oedema (3.9%), abruptio placentae (1.7%), HELL syndrome (1.2%), maternal death (1.0%), acute renal failure (0.9%), coma with cerebral pathology (0.5%) and DIC (0.5%). In addition, eclamptic patients had significantly more maternal complications than noneclamptic hypertensive women (9).

Maternal risks associated with chronic hypertension include superimposed preeclampsia or eclampsia, deterioration of renal function, cerebrovascular accident, congestive heart failure, and hemorrhage secondary to placental abruption. (48). Haddad B, and Sibai M found that pregnancies in women with chronic hypertension are at increased risk of superimposed pre-eclampsia, and abruptio placentae (8).

3. Birth Outcome

Induction of labor

Induction of labor is reserved for patient with preeclapsia that have reached 37 weeks of gestation, where the fetus is mature, and the condition of the cervix is favorable (45). In New Delhi, Yadar S, et al concluded in their case controlled prospective study that labor induction rate (52.8% vs 3.25%) were higher in hypertensive pregnant women compared to normal pregnant women (14). Omu AE, et al. also found that more hypertensive patients required induction 49.6% compared to 5.4% in normotensive women ($p < 0.001$) (16). Anyaegbunam AM, et al. showed that the only significant difference was a higher rate of induction of labor (36.7% vs 66%, $p < 0.005$) in hypertensive diabetic pregnant women (10).

Mode of delivery

The patient with pre-eclampsia at term whose intake / output is balanced, and whose blood pressure is being controlled deserves a trial of vaginal delivery. Cesarean section is reserved for adverse and deteriorating maternal and fetal conditions or in the preterm gestation with an unripe cervix (45). Many studies found that the maternal hypertension was significantly associated with a higher rate of cesarean section (14, 16, 22, 25, 45-50). A longitudinal cohort study was conducted by Rey E and Couturier A. in Montreal, Quebec, Canada, to assess the pregnancy outcome in women with chronic hypertension from a population with a perinatal mortality of 12 in 1000. They concluded that the women with chronic hypertension both with or without superimposed preeclampsia had a higher rate of Cesarean section than the general population (29.6 % vs 14.2%) (24). Kiss I, et al found that the prevalence of delivery by Cesaream section was significantly higher in the hypertensive group than in the normotensive one (13). In Thailand, the Rajavithee Hospital found that the hypertensive pregnancies had Cesaream section rate 2.5 times of the normotensive (51). Omu AE, et al. showed that there was an significant difference in the mode of delivery in terms of spontaneous vaginal delivery, vacuum and forceps delivery ($p > .05$). However, there was a significant association between the hypertensive pregnancies and cesarean section ($p < 0.001$) (16).

Prematurity

Yadar, et al. showed that preterm delivery (28.8% vs 3%) was higher in hypertensive complicated pregnancy as compared to normal pregnant women (14). Pietrantoni M and O' Brien WF examined the effect of the various hypertensive

disorders of pregnancy on perinatal outcome. They found that the transient and chronic hypertension accounted for 32.8% and 24.2% of preterm deliveries respectively. (15). Alexander E OM demonstrated that the evaluation of gestational age at delivery; 21.4 percent of the hypertensive pregnant women had preterm delivery, that is delivery before 37 complete weeks of gestation, compared to 5.4 percent in normal pregnant women. Of the normotensive women, there was obvious association between hypertension in pregnancy and preterm delivery ($p < 0.002$) (16). Jain L, et al. examined racial differences in the outcome of pregnancies complicated by hypertension using data obtained from a large perinatal data base with 109,428 consecutive deliveries from 1982 to 1987. Black women had a higher prevalence of hypertension than white women. However, when compared to normotensive women of similar race, white hypertensive women showed a higher risk for adverse pregnancy outcome than black hypertensives as indicated by the higher odds ratio for prematurity (OR = 1.7 for white, 1.2 for black) (21).

Ray E and Couturier concluded in their study that premature delivaries were statistically more frequent in the hypertensive women (34.4% vs 15.0%) than in the general population ($p < 0.01$) (24). Several studies found that the pregnancies in women with chronic hypertension are at increased risk of prematurity (8, 12,17-20,23).

4. Neonatal Outcome

Fetal and neonatal complications include intrauterine growth retardation, fetal death, hypoxia, abruptio placenta, and pre-term delivery (45,48).

Intrauterine growth retardation (IUGR)

Pregnancy-induced hypertension (PIH) effects on fetus reflects vasospasm with regard to placental perfusion: a decrease in uteroplacental perfusion may result in abruption, IUGR, or oligohydramnios (52). In normal pregnancy, the trophoblast migrates down the intima of the spiral arteries. The musculo-elastic tissue of the vessel wall gradually erodes and is replaced by fibrinoid material. The spiral ateries are converted from small muscular end arterioles to wide, tortuous uteroplacental arteries that empty into the intervillous space. Brosens and colleague termed these process *"physiologic changes"*. It has been suggested that the formation of uteroplacental arteries occur in two stages. Conversion of the decidual segments takes place in the first trimester, whereas the myometrial segments are transformed by a second wave of trophoblast invasion in the second trimester (43, 45, 53).

Chronic hypertension, pregnancy-induced hypertension, and preeclampsia have common effect of limiting trophoblast invasion, placental growth and development, uteroplacental blood flow, and fetal oxygen and nutrient deficiency. High-altitude hypoxia also can limit fetal growth. (52). Fetal risks include acute and chronic uteroplacental insufficiency. In the most severe case, this may result in intrapartum fetal distress or stillbirth. Chronic uteroplacental insufficiency increases the risk of an asymmetric (cranial – sparing) or symmetric SGA fetus (43, 54, 55). Xiong x, et al showed that after adjustment for duration of gestation and other confounders,

preeclampsia and severe preeclampsia increased the risk of intrauterine growth restriction. The adjusted ORs for intrauterine growth restriction were 1.49 (95%CI = 1.14 – 1.93) for gestational hypertension, 1.97 (95%CI = 1.43 – 2.73) for preeclampsia, and 1.95 (95%CI = 1.14 – 3.37) for severe preeclampsia (11). Omu AE, et al concludes that in linear regression analysis, hypertension in pregnancy gave rise to more intrauterine growth retardation than the normotensive control (16). In Chicago, 1997, a retrospective study of the effect of pregnancy – induced hypertension and chronic hypertension on pregnancy outcome was done by Jain L (20) demonstrated that chronic hypertension was more likely to result in an adverse outcome as showed by increased odds ratio (OR) for intrauterine growth retardation (OR 2.8 for PIH and 3.7 for hypertension) when compared with PIH. In 1998, Jain L, et al found the white hypertensive women had a higher risk for intrauterine growth retardation than black hypertensive women (OR 4.4 for white, 1.6 for black) when compared to normotensive of similar race (21). Misra DP concluded that the pregnancy-induced hypertension affects fetal growth could suggest a particular biological mechanism leading to intrauterine growth retardation (IUGR) (55).

Low Apgar score

Fernandez Jonusas S and Ceriani cernadas JM found that the frequency of low Apgar score at 5 minutes (OR = 3.63; 95%CI = 1.12 – 15.3) was significantly increased in infants born to hypertensive mothers (22). Whereas, Anyaeg bynam, et al (10) and Morgan MA, (25) concluded that neonates from hypertensive women were on more likely to have low 5 – minute Apgar scores than those from normotensive women.

Birth weight

Kiss I, et al concluded in their retrospective study that the birth weight of the newborn was lower in the hypertensive women than in the normotensive women and there was a negative correlation ($p < 0.001$) between the maximal pretreatment diastolic blood pressure and the birth weight of newborns in the hypertensive group (13). Chronic hypertension was associated with a 184 gm reduction in birth weight compared with 168 gm reduction with PIH, which was reported by Jain L (20). In 1998 they studied the racial differences in outcome of pregnancies complicated by hypertension. They found that hypertension was associated with a 156 gm reduction in the birth weight of newborns in whites as compared to a 63 gm reduction in hypertension on pregnancy outcome in women with gestational diabetes.

However, there was one study found that the mean birth weight for the hypertensive gestational diabetes group was significantly higher than that of the nonhypertensive gestational diabetes (3,360± -578 vs 3,293± -581; $p < 0.05$) (10).

Low birth weight infant

There was much studied report that hypertensive disorders in pregnancy were associated with low birth weight infant (12, 18, 21-23, 46-48). Women noted to have eclampsia during pregnancy had a substantially greater risk of delivering very-low-birth weight infants (birth weight ≤ 1,499 gm; risk difference (RD = 6.7%) and moderately-low-birth weight infants (1,500 – 2,499 gm; RD = 14.6%) compared with women without hypertension (17). Alexander E. OMU, et al found the mean birth weight of 3.24 ± 0.54 kilograms in the hypertensive women was significantly lower than the 3.64±0.61 kilograms in the normotensive group ($x^2 = 27.1$,8DF, $p < 0.01$)

(16). But after adjusted for gestational age and other confounders, there were no differences in mean birth weight between normotensive and those with pregnancy-induced hypertension. The risk of low birth weight was not increased significantly for gestational hypertension (adjusted OR 1.56; 95% CI = 1.73 – 4.39) for preeclampsia and 2.53 (95% CI = 1.19 – 4.93) for severe preeclampsia (11).

Small for gestational age (SGA)

Several studies found that maternal hypertension was significantly associated with a higher rate of SGA infant (10, 22, 24) or a higher risk than those in normotensive women (8, 17, 22, 46, 56-59).

Neonatal complication

Fernandez Jonvsas S and Ceriani cernadas IM showed that the frequency of necrotizing enterocolitis (OR = 3.33; 95% CI = 1.23 – 10.30) and polycythemia (OR = 3.63; 95% CI = 1.12 – 15.3) was significantly increased in infants born to hypertensive mothers (22). In addition, other neonatal complication in infant born to severe preeclampsia was respiratory distress syndrom, apnea, ventilation therapy, and oxygen requirements and bronchpulmonary dysphasia. (46). The prevalence of fetal asphyxia during delivery was significantly higher in the hypertensive group than the normotensive one. (13).

Maternal and neonatal mortality

There were many studies demonstrated that perinatal mortality, perinatal death, fetal death, still birth and maternal mortality were significantly increase in the hypertensive group (7, 13 – 15, 18, 20-21, 24-26, 49)

Maternal and neonatal complication in postpartum period

Alexander E. OMU, et al found that there was significant evidence that hypertension in pregnancy gave rise to more hospitalization than in normotensive control (16). For the other condition, there was no evidence about this.

CHAPTER III

MATERIALS AND METHODS

Study design

The study is a retrospective cohort study.

Study population

The target population is pregnant women in of upper northern region.

Study subjects

The subjects were pregnant women with singleton pregnancies, who attended at an antenatal clinic (ANC) and delivered at the Mother and Child Hospital, Chiang Mai, during 1st December 1997 to 30th April 1999. They were divided into two groups as following:

The index group was selected to be all hypertensive pregnant women who attended ANC during the study period, and we included 164 pregnant women with developed PIH (pregnancy-induced hypertension) and 6 pregnant women with chronic hypertension.

Controls were 312 normotensive pregnant women who attended ANC during the same time and were selected by simple random sampling.

Sample size

The sample size was calculated from the review of research literature that contained information regarding hypertensive disorders of pregnancy on obstetric outcomes and neonatal outcomes (Appendix B).

The effect of hypertensive disorders of pregnancy on	sample size
1. gestational age	455
2. birth weight	103

Due to the limitation of time and funding, total of 170 index cases and 312 controls were selected for this study.

Instrument

The researcher collected all necessary information for the study using a constructed data recording form. All information regarding study subjects were compiled from the hospital records (ANC records, labour records and admission charts) which was then classified into two parts:

Part I: Maternal data consists of:

- Demographic and reproductive characteristics
- Pregnancy outcomes
- Obstetric outcomes
- Maternal health status in postpartum period

Part II: Neonatal data consists of

- Neonatal outcomes
- Neonatal health status in postpartum period

(Appendix A)

Data collection

The data was collected using a data record form and performed by the researcher. Each pregnant woman's record was traced form the first antenatal visits until the end of pregnancy period. Their infant's information was also traced until discharge from the hospital. After the data were checked, verified and cleaned to improve the quality.

Data analysis

The collected data were encoded and edited using Epi Info versions 6.04 programme and then analyzed by using the STATA version 5.0 for windows programme.

Statistical analysis

1. Descriptive statistics: Qualitative data were presented by mean and standard deviation, and qualitative data were presented by frequency and percentage.

2. Analytic statistics:

2.1 Univariate analysis: The differences of important characteristics among three groups were analyzed by t-test or Mann-Withney *U* test (for numerical outcomes) and Chi-square or exact probability test (for categorical outcome).

2.2 Multivariate analysis: Multiple regression (for continuous outcomes) and log risk regression (for categorical outcomes) were used to control for potential confounders.

CHAPTER IV

RESULTS

The study was conducted at the Maternal and Child Hospital, Chiang Mai. There were 482 study subjects that included 170 index cases of hypertensive disorders of pregnancy and 312 control cases of normotensive pregnancy. The hypertensive group was divided into two groups classified by clinical criteria: 164 cases of pregnancy-induced hypertension (PIH) and 6 cases of chronic hypertension.

The proportion of pregnancy-induced hypertension cases are as follow:

89 gestational hypertension	54.3%
46 preeclampsia	28.0%
29 severe preeclampsia	17.7%

The proportion of chronoc hypertension cases are as follow:

4 chronic hypertension with superimposed preeclampsia	66.7%
2 chronic hypertension	33.3%

The studied results are presented as follow:

1. Demographic and reproductive characteristics
2. Pregnancy progression
3. Obstetric outcome
4. Pregnancy outcome of maternal and neonatal in postpartum period

1. Demographic and reproductive characteristics

Demographic and reproductive characteristics were shown in Table 3 and Table 4.

1.1 Demographic characteristics

Age

The mean age in normotensive pregnancy was 24.98 years (SD = 5.47), 27.65 years (SD = 7.10) in PIH pregnancy, and 32.67 years (SD = 5.75) in chronic hypertensive pregnancy. The mean ages of the three groups showed a statistically significant difference ($p < 0.001$).

Height

The distributions of height among the three groups were similar ($p = 0.688$). The average height in normotensive pregnancy was 152.86 centimeters (SD = 5.69), 153.42 centimeters (SD = 5.87) in PIH, and 151.67 centimeters (SD = 6.09) in chronic hypertensive pregnancy.

Pre-pregnancy weight

The mean pre-pregnancy weight of the normotensive pregnancy was 48.37 kilograms (SD = 7.14) which was significantly ($p < 0.001$) lower than that of the PIH pregnancy (55.07 kilograms, SD = 9.78).

Race

Most of the subjects were Thai: 97.8% in normotensive, 99.4 % in PIH, 100% in chronic hypertensive pregnancy, and they were no significant differences ($p = 0.343$).

Religion

The proportions of religion among the three groups were not different ($p = 0.571$). The most common religion among the subjects was Buddhist (99.0% of

normotensive, 100% of PIH, and chronic hypertensive pregnancy). Christian accounted for 1.0% in normotensive pregnancy.

Marital status

Most of the subjects were married (100% of normotensive, 99.4% of PIH and 100% of chronic hypertensive pregnancy).

Occupation

The percentage of occupation among the three groups were not difference ($p = 0.871$). More than half of the normotensive (51.6%), PIH (56.7%), and chronic hypertensive pregnancy (50.0%) were employee or farmer. The next most common was housewives (26.9% vs 27.4% vs 33.3%) and business (17.6% vs 11.6% vs 16.7%).

Education

The levels of education among the three groups were similar ($p = 0.070$). The majority of the normotensive pregnancy group (39.8%) finished secondary school, while the majority of PIH group (45.7%) and chronic hypertensive pregnancy group (83.3%) finished only primary school.

Family history of illness

Family history of illness was positive in 7.1% of normotensive pregnancy, 18.3% of PIH and 33.3% of chronic hypertensive pregnancy. The proportion of family history of hypertension was the highest in chronic hypertensive pregnancy. Other family history of illness included diabetes mellitus, unspecified hematologic diseases, heart disease, asthma, thyroid, diabetes mellitus / hypertensoin and epilepsy. The differences were not statistically significant ($p = 0.173$, $p = 0.284$, $p = 0.563$, $p = 0.622$, $p = 0.339$, $p = 0.284$, and $p = 0.339$, respectively).

1.2 Reproductive characteristics

The reproductive characteristics were not significant different in number in terms of previous pre-term delivery, previous low birth weigh delivery, gravidity, parity, past abortion, living children, except for past history of hypertensive pregnancy. The results found that the proportion of past history of hypertensive pregnancy in chronic hypertensive pregnancy was 33.3%, which was the highest among the three groups. There was a significant difference ($p < 0.001$).

Table 3 Demographic characteristics according to type of hypertensive disorders of pregnancy.

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Age (years)							
15 – 19	46	14.7	23	14.0	0	0.0	0.002
20 – 34	248	79.5	107	70.1	4	66.7	
35 – 45	18	5.8	22	13.4	2	33.3	
≥46	0	0.0	4	2.5	0	0.0	
Mean (SD)	24.98	(5.47)	27.65	(7.10)	32.67	(5.75)	< 0.001[a]
Range	15-40		16-47		25-40		
Race							
Thai	305	97.8	163	99.4	6	100.0	0.343
Other	7	2.2	1	0.6	0	0.0	

*Exact probability Test
[a] Kruskall-Wallis Test

Table 3 Demographic characteristics according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Height (cm)	(n = 312)		(n = 159)		(n = 6)		
≤ 145	32	10.2	14	8.8	0	0.0	0.688
146 – 160	257	82.4	128	80.5	6	100.0	
≥ 161	23	7.4	17	10.7	0	0.0	
Mean (SD)	152.86	(5.69)	153.42	(5.87)	151.67	(6.09)	0.633 [a]
Range	138-170		138-168		146-160		
Pre-pregnancy weight (kg)	(n = 73)		(n = 37)		(n = 0)		
36 – 50	55	75.4	14	37.8	na		< 0.001
51-65	14	19.2	19	51.4			
66-80	4	5.4	3	8.1			
81-95	0	0.0	1	2.7			
Mean (SD)	48.37	(7.14)	55.07	(9.78)			< 0.001 [b]
Range	36.0-73.0		43.7-88.0				

*Exact probability Test
[a] Kruskall-Wallis Test
[b] Mann Withney *U* Test

Table 3 Demographic characteristics according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Religion							
Buddhism	309	99.0	164	100.0	6	100.0	0.571
Christ	3	1.0	0	0.0	0	0.0	
Marital Status							
Married	312	100.0	163	99.4	6	100.0	0.353
Widowed	0	0.0	1	0.6	0	0.0	
Occupation							
House wife	84	26.9	45	27.4	2	33.3	0.871[c]
Government official / Private	10	3.2	5	3.1	0	0.0	
Employee / Farmer	161	51.6	93	56.7	3	50.0	
Commercial	55	17.6	19	11.6	1	16.7	
Other	2	0.7	2	1.2	0	0.0	
Education							
No education	21	6.7	9	5.5	0	0.0	0.070[c]
Primary school	120	38.5	75	45.7	5	83.3	
Secondary school	124	39.8	52	31.7	0	0.0	
Vocational or equivalent beyond	40	12.8	21	12.8	0	0.0	
No information	7	2.2	7	4.3	1	16.7	

*Exact probability Test, [c] Chi-square Test

Table 3 Demographic and reproductive characteristics according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Family history of illness	(n = 312)		(n = 153)		(n = 6)		
No	290	92.9	125	81.7	4	66.7	< 0.001
Yes	22	7.1	28	18.3	2	33.3	
Diabetes mellitus	9	2.9	10	6.6	0	0.0	0.173
Hypertension	8	2.6	9	5.9	2	33.3	0.010
Unspecified hematologic diseases	1	0.3	2	1.3	0	0.0	0.284
Heart disease	1	0.3	1	0.6	0	0.0	0.563
Asthma	2	0.7	2	1.3	0	0.0	0.622
Thyroid	0	0.0	1	0.6	0	0.0	0.339
Diabetes mellitus and Hypertension	1	0.3	2	1.3	0	0.0	0.284
Epilepsy	0	0.0	1	0.6	0	0.0	0.339

*Exact probability Test

Table 4 Reproductive characteristics according to type of hypertensive disorders of pregnancy.

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Past history of hypertensive pregnancy	(n = 312)		(n = 153)		(n = 6)		
No	311	99.7	149	97.4	4	66.7	< 0.001
Yes	1	0.3	4	2.6	2	33.3	
No. of previous pre-term delivery							
0	305	97.8	156	95.1	6	100.0	0.310
1	7	2.2	8	4.9	0	0.0	
No. of previous low birth weight delivery							
0	296	94.9	151	92.1	5	83.3	0.139
1	16	5.1	12	7.3	1	16.7	
2	0	0.0	1	0.6	0	0.0	

*Exact probability Test

Table 4 Reproductive characteristics according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Gravida							
nullipara	140	44.9	67	40.9	2	33.3	0.510[c]
2	126	40.4	66	40.2	1	16.7	
3	35	11.2	22	13.4	2	33.3	
4	8	2.6	7	4.3	1	16.7	
5	2	0.6	2	1.2	0	0.0	
6	1	0.3	0	0.0	0	0.0	
No. of parity							
0	183	58.7	88	53.7	2	33.3	0.123 [c]
1	111	35.6	61	37.2	2	33.3	
2	17	5.4	12	7.3	2	33.3	
3	1	0.3	1	0.6	0	0.0	
4	0	0.0	2	1.2	0	0.0	

*Exact probability Test
[c] Chi-square Test

Table 4 Reproductive characteristics according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
No. of past abortion							
0	237	75.9	126	76.9	5	83.3	0.232
1	68	21.8	34	20.7	0	0.0	
2	3	0.9	3	1.8	1	16.7	
3	4	1.2	1	0.6	0	0.0	
No. of living children							
0	184	59.0	92	56.1	3	50.0	0.647 [c]
1	112	35.9	60	36.7	2	33.3	
2	15	4.8	9	5.4	1	16.7	
3	1	0.3	1	0.6	0	0.0	
4	0	0.0	2	1.2	0	0.0	

*Exact probability Test
[c] Chi-square Test

2. Pregnancy progression

2.1 The results of laboratory in pregnancy progression

A positive result for HIV infection, VDRL, Hepatitis B virus, One tube osmotic fragility test (OF test) among the three groups were not difference ($p = 0.215$, $p = 0.559$, $p = 0.919$, and $p = 0.307$, respectively). A positive result for HIV infection, VDRL, or Hepatitis B virus was found in the normotensive group less than in the PIH pregnancy group, while a positive result of one osmotic fragility test was identified in normotensive group were more than PIH and chronic hypertensive pregnancy group. The averages of hematocrit level at first visit of normotensive and chronic hypertensive pregnancy (37.33%, SD = 3.32 and 37.33%, SD = 3.39) were higher than those of PIH pregnancy (37.31%, SD = 3.71), whereas the average of hematocrit level at third trimester of normotensive pregnancy (36.93%, SD = 3.30) was less than those of PIH (37.30% SD = 3.17) and chronic hypertensive pregnancy(37.50%, SD = 3.54). There was no significant differences ($p = 0.158$). However, the mean difference of hematocrit level during pregnancy in normotensive pregnancy was significantly increased from the first visit (0.76%, $p = 0.009$).

2.2 Pregnancy progression

Maternal weight at first visit (kilograms)

The means maternal weight at first visit among the three groups were significantly different ($p < 0.001$). The mean maternal weight at first visit in normotensive pregnancy (52.22 kilograms, SD = 8.54) was less than that in PIH pregnancy (59.01 kilograms, SD = 12.22) and in chronic hypertensive pregnancy (57.00 kilograms, SD = 9.06).

Body mass index at first visit (kg/m^2)

The mean BMI at first visit of normotensive pregnancy was 22.02 kg/m^2 (SD = 3.41), lower than that in PIH (24.66 kg/m^2, SD = 4.87) and in chronic hypertensive pregnancy (24.78 kg/m^2, SD = 3.43). The differences were statistically significant ($p < 0.001$).

Maternal weight gain (kilograms/week)

The mean maternal weight gain of normotensive pregnancy (0.42 kg/wk, SD = 0.20) was more than that in PIH pregnancy (0.41 kg/wk, SD = 0.10). There was no statistical difference ($p = 0.672$). The comparison between normotensive and chronic hypertensive pregnancy was not available, because of missing data.

Underlying disease/Complication during pregnancy

Approximately, half of each group (40.1% vs 42.6% vs 50.0%) had underlying disease/complication during pregnancy. The underlying diseases/complications during pregnancy among the three groups were not significantly different ($p = 0.735$). A major complication of pregnancy in the three groups was unspecified hematologic diseases. There were 113(36.2%) cases in normotensive pregnancy, 62(40.2%) cases in PIH pregnancy and 2(33.3%) cases in chronic hypertensive pregnancy. However,

their unspecified hematologic diseases, asthma, thyroid, diabetes mellitus, hypertension/diabetes mellitus, ectopic pregnancy, asthma/unspecified hematologic diseases and hypertension/diabetes mellitus/unspecified hematologic diseases were also similar among the three groups. The only significant difference was the intrauterine growth retardation ($p = 0.029$). The percentages of the intrauterine growth retardation was 0.3% in normotensive pregnancy less, when compared to 16.7% in chronic hypertensive pregnancy.

Completion of antenatal care

There was no significant difference for completion of antenatal care ($p = 0.739$). The percentage of completion of antenatal care were 47.6% in normotensive, 50.0% in PIH pregnancy, and 33.3% in chronic hypertensive pregnancy.

Table 5 Pregnancy progression according to type of hypertensive disorders of pregnancy.

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Screening test for HIV infection	(n = 190)		(n = 108)		(n = 6)		
Negative	183	96.3	99	91.7	6	100.0	0.215
Positive	7	3.7	9	8.3	0	0.0	
Serology test for syphilis	(n = 301)		(n = 159)		(n = 6)		
Negative	296	98.4	155	97.5	6	100.0	0.559
Positive	5	1.6	4	2.5	0	0.0	
Screening test for Hepatitis virus B	(n = 211)		(n = 124)		(n = 6)		
Negative	191	90.5	111	89.5	6	100.0	0.919
Positive	20	9.5	13	10.5	0	0.0	
One tube osmotic fragility test	(n = 202)		(n = 123)		(n = 6)		
Negative	87	43.1	61	49.6	4	66.7	0.307
Positive	155	56.9	62	50.4	2	33.3	

*Exact probability Test

Table 5 Pregnancy progression according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Anemia at first visit	(n = 273)		(n = 135)		(n = 6)		
No	261	95.6	124	91.9	5	83.3	0.115
Yes	12	4.4	11	8.1	1	16.7	
Mean (SD)(%)	37.33	(3.32)	37.31	(3.71)	37.33	(3.39)	0.941[a]
Range	26-46		27-46		31-40		
Anemia at third trimester	(n = 169)		(n = 74)		(n = 2)		
No	156	92.63	69	93.2	2	100.0	1.000
Yes	13	7.7	5	6.8	0	0.0	
Mean (SD)(%)	36.93	(3.30)	37.30	(3.17)	37.50	(3.54)	0.158[a]
Range	24-35		30-44		35-40		
Difference of hematrocrit level during pregnancy (%)							
Mean (SE)	0.76	(0.28)	-0.04	(0.41)	2.5	(2.5)	
p-value[d]	0.009		0.927		0.500		

*Exact probability Test
[a] Kruskall-Wallis Test
[d] Student t-Test

Table 5 Pregnancy progression according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Maternal weight at first visit (kg)							
35-54	209	67.0	63	39.4	3	50.0	< 0.001 [c]
55-74	97	31.1	81	50.6	3	50.0	
75-94	6	1.9	15	9.4	0	0.0	
95-114	0	0	1	0.6	0	0.0	
Mean (SD)	52.22	(8.54)	59.01	(12.22)	57.00	(9.06)	< 0.001 [a]
Range	36.0-93.0		35.5-108.3		47.5-73.0		
Body mass index at first visit (kg/m^2)							
≤ 18.50	34	10.9	7	4.4	0	0.0	< 0.001 [c]
18.51-24.9	223	71.5	84	53.5	4	66.7	
25-29.9	49	15.7	48	30.6	2	33.3	
≥ 30	6	1.9	18	11.5	0	0.0	
Mean (SD)	22.02	(3.41)	24.66	(4.87)	24.78	(3.43)	< 0.001 [a]
Range	15.61-377.25		15.78-47.50		19.03-28.52		

*Exact probability Test
[a] Kruskall-Wallis Test
[c] Chi-square Test

Table 5 Pregnancy progression according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Maternal weight gain (kg/wk)	(n = 73)		(n = 35)		(n = 0)		
0.01-0.49	54	74.0	27	77.1	na		1.000
0.50-0.99	17	23.3	8	22.6			
1.00-1.49	2	2.7	0	0.0			
Mean (SD)	0.42	(0.20)	0.41	(0.10)			0.672[b]
Range	0.12-1.15		0.25-0.58				

*Exact probability Test
[b] Mann Withney *U* Test

Table 5 Pregnancy progression according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Underlying diseases/Complication during pregnancy	(n = 312)		(n = 155)		(n = 6)		
No	187	59.9	89	57.4	3	50.0	0.735
Yes	125	40.1	66	42.6	3	50.0	
Unspecified hematologic diseases	113	36.2	62	40.2	2	33.3	0.888
Asthma	5	1.7	1	0.6	0	0.0	0.693
Thyroid	2	0.6	1	0.6	0	0.0	1.000
IUGR	1	0.3	0	0.0	1	16.7	0.029
Diabetes mellitus	0	0.0	1	0.6	0	0.0	0.342
Hypertension and DM	0	0.0	1	0.6	0	0.0	0.342
Ectopic pregnancy	1	0.3	0	0.0	0	0.0	1.000
Asthma and unspecified hematologic disease	1	0.3	0	0.0	0	0.0	1.000
Hypertension/DM/unspecified hematologic diseases	1	0.3	0	0.0	0	0.0	1.000

*Exact probability Test

Table 5 Pregnancy progression according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Completion antenatal care	(n = 311)		(n = 144)		(n = 6)		
Yes	163	52.4	72	50.0	4	66.7	0.739
No	148	47.6	72	50.0	2	33.3	
No. of antenatal care visit	(n = 311)		(n = 144)		(n = 6)		
0	0	0.0	1	0.7	0	0.0	0.363 [c]
1	11	3.6	2	1.39	0	0.0	
2	52	16.7	33	23.0	2	33.3	
3	100	32.1	36	25.0	2	33.3	
4	148	47.6	72	50.0	2	33.3	

*Exact probability Test
[c] Chi-square Test

2.3 The effect of hypertensive disorders on pregnancy progression

Table 4 shows the multivariate analysis of the effect of hypertensive disorders on pregnancy progression.

Hematocrit at first antenatal visit and third trimester

After adjusting for weight gain during pregnancy, the mean hematocrit level at first antenatal visit in PIH pregnancy was lower than the normotensive pregnancy. The mean difference was 0.02% (95%CI = -0.74 to -0.69, p = 0.951).

For the hematocrit level at third trimester, the mean hematocrit levels at third trimester in PIH and chronic hypertensive pregnancy were higher compared to the normotensive pregnancy. The mean differences were 0.77% (95%CI = -0.13 to 1.66, p = 0.093) and 0.56% (95%CI = -4.00 to 5.13, p = 0.808), respectively.

Weight gain during pregnancy

The mean weight gain during pregnancy of PIH pregnancy was less than normotensive pregnancy after controlling for age, gravida, HIV infection, complication during pregnancy, attending ANC and BMI at first visit. The mean difference was 0.02 kilograms (95%CI = -0.12 to 0.07, p = 0.649).

Table 6 The effect of hypertensive disorders on pregnancy progression from multiple regression.

Characteristics	Pregnancy-induced hypertension				Chronic hypertension			
	Regression Coef.	95% CI		p-value	Regression Coef.	95% CI		p-value
Hematrocrit level at first antenatal visit (%) [a]	-0.02	-0.74	0.69	0.951				
Hematrocrit level at third Trimester (%) [a]	0.77	-0.13	1.66	0.093	0.56	-4.00	5.13	0.808
Weight gain during pregnancy [b]	-0.02	-0.12	0.07	0.649				

[a] Adjusted for maternal weight gain

[b] Adjusted for age, attending ANC, gravida, complication during pregnancy, HIV infection, BMI at first visit

3. Pregnancy outcome

3.1 Outcome of pregnancy

Table 7 shows the outcome of pregnancy such as gestational age and neonatal status at delivery. The mean gestational age in normotensive pregnancy (38.49 week, SD = 1.91) was more than that in PIH pregnancy (38.05 week, SD = 2.21) and chronic hypertensive pregnancy (34.00 week, SD = 3.79). The differences were statistically significant (p = 0.001). All babies were live birth.

3.2 The effect of hypertensive disorders on pregnancy outcome

Table 8 shows the effect of hypertensive disorders on outcome of pregnancy compared with normotensive pregnant women.

Gestational age

After controlling for age, gravida, abortion, previous preterm delivery, past history of hypertensive pregnancy and HIV infection, PIH pregnancy had gestational age 0.17 week shorter than normotensive pregnancy (95%CI = -0.67 to 0.33, p = 0.500). Chronic hypertensive pregnancy had gestational age 3.86 week shorter than normotensive pregnancy (95%CI = -5.68 to -2.03, p < 0.001).

Prematurity

After adjusting for gravida, abortion, previous preterm delivery, complication during pregnancy, HIV infection, PIH pregnancy and chronic hypertensive pregnancy were at an increased risk of prematurity 1.02 and 5.70 times (95%CI = 0.52 to 1.98, p = 0.960 and 95%CI = 1.90 to 17.10, p = 0.002).

Table 7 Outcome of pregnancy according to type of hypertensive disorders of pregnancy.

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Gestational age (weeks)							
28-36	38	12.2	27	16.5	4	66.7	0.013
37-42	270	86.6	136	82.9	2	33.3	
≥ 43	4	1.2	1	0.6	0	0.0	
Mean (SD)	38.49	(1.91)	38.05	(2.21)	34.00	(3.79)	0.001 [a]
Range	32-43		28-45		28-38		
Neonatal status at delivery							
Live birth	312	100.0	164	100.0	6	100.0	
Stillbirth	0	0.0	0	0.0	0	0.0	

*Exact probability Test
[a] Kruskall-Wallis Test

Table 8 The effect of hypertensive disorders on outcome of pregnancy from multiple regression.

Characteristics	Pregnancy-induced hypertension				Chronic hypertension			
	Regression Coef.	95% CI		p-value	Regression Coef.	95% CI		p-value
Gestational age (week) [c]	-0.17	-0.67	0.33	0.500	-3.86	-5.68	-2.03	< 0.001
Prematurity [d]	[@]1.02	0.52	1.98	0.960	[@]5.70	1.90	17.10	0.002

[c] Adjusted for age, gravida, abortion, previous preterm delivery, past history of hypertensive pregnancy, HIV infection

[d] Adjusted for gravida, abortion, previous preterm delivery, complication during pregnancy, HIV infection

[@] Risk ratio

3.3 Obstetric outcome

The obstetric outcomes are summerized in Table 9.

Premature rupture of membrane

The means of duration of premature rupture of membrane among the three groups were not different (p = 0.404). It was 266.84 minutes (SD = 296.23) in normotensive, 323.42 minutes (SD = 411.31) in PIH, and 34.00 minutes (SD = 0) in chronic hypertensive pregnancy.

Type of membranes rupture

According to type of membranes rupture, the artificial rupture of membrane was 62.2% in normotensive, 48.9% in PIH, and 100% in chronic hypertensive pregnancy. The spontaneous rupture of membrane was 26.3% in normotensive and 42.1% in PIH pregnancy. The type of membranes rupture among the three groups had statistical differences (p = 0.012).

Characteristics of amniotic fluid

The most common characteristics of amniotic fluid among the three groups were clear (61.7% of normotensive, 55.8% of PIH, and 100% of chronic hypertensive pregnancy, respectively). The next most common was unknown (20.1% of normotensive and 26.3% of PIH pregnancy). It was not a statistical different (p = 0.786).

Mode of delivery

There were significant differences in mode of delivery among the three groups (p = 0.004). Most common mode of delivery in normotensive and PIH pregnancy were vaginal delivery (70.8% vs 55.5%), cesarean section (16.7% vs 28.0%), and vacuum extraction (4.8% vs 10.4%), whereas the most common mode of delivery in

chronic hypertensive pregnancy were vaginal delivery (33.3%) and cesarean section (33.3%).

Indication for abnormal delivery

More than half of the normotensive (69.9%) and PIH pregnancy (54.3%) had no indication for abnormal delivery, while the most common indication for abnormal delivery in chronic hypertensive pregnancy were maternal causes (83.3%). There was a statistically difference ($p < 0.001$).

Duration of labour

First stage labour

The average duration of first stage labour in normotensive pregnancy was 393.64 minutes (SD = 155.55), longer than those in both PIH (369.11 minutes, SD = 150.25) and chronic hypertensive pregnancy (365 minutes, SD = 225.66). There was no significant difference ($p = 0.357$).

Second stage labour in nullipara

The average duration of second stage labour in nullipara was 24.47 minutes (SD = 17.76) for normotensive pregnancy, shorter than those in PIH (39.19 minutes, SD = 34.14) and chronic hypertensive pregnancy (25.5 minutes, SD = 12.02). There was a significant difference ($p = 0.023$).

Second stage labour in multipara

The average duration of second stage labour in multipara was 21.44 minutes (SD = 15.93) of normotensive, also shorter than those in PIH (24.83 minutes, SD = 18.92) and chronic hypertensive pregnancy (32.00 minutes, SD = 0). The difference was not significant ($p = 0.391$).

Third stage labour

The average duration of third stage labour among three groups had no significant difference (p = 0.073). The average duration of third stage labour in normatersive pregnancy was 5.41 minutes (SD = 5.52), 7.03 minutes (SD = 9.17) in PIH pregnancy, and 10.67 minutes (SD = 10.69) in chronic hypertensive pregnancy.

Total duration of labour

The distributions of total duration of labour among the three groups were similar (p = 0.710). The average of total duration of labour in normotensive pregnancy was 421.88 minutes (SD = 157.96), 409.13 minutes (SD = 160.56) in PIH pregnancy, and 403.33 minutes (SD = 210.27) in chronic hypertensive pregnancy.

Estimated blood loss

The average of estimated blood loss in normotensive pregnancy was 346.15 cc (SD = 93.87), less than those in PIH pregnancy (381.10 cc, SD = 123.51) and chronic hypertnssive pregnancy (366.67 cc, SD = 103.28). The difference was statistically significant (p = 0.035).

Primary postpartum hemorrhage

Most of subjects had no primary postpartum hemorrhage (98.1% in mormotensive, 96.3% in PIH, 100.0% in chronic hypertensive pregnancy respectively), and there were no significant difference (p = 0.446).

Placenta weight

The averages of placenta weight among the three groups were significantly different (p = 0.008). The average placenta weight was 578.24 grams (SD = 97.62) in normotensive pregnancy, 596.83 grams (SD = 123.19) in PIH pregnancy, and 460.00 grams (SD = 89.44) in chronic hypertensive pregnancy.

Placenta characteristics

The characteristics of placenta in the three groups were similar (p = 0.283). The most common was completed placenta (92.7% in normotensive, 97.5 in PIH, and 100.0% in chronic hypertensive pregnancy).

Cord length

The mean cord length was 52.04 centimeters (SD = 9.52) in normotensive pregnancy, 52.06 centimeters (SD = 10.38) in PIH, and 45.83 centimeters (SD = 8.01) in chronic hypertensive pregnancy. There was no significant difference (p = 0.206).

Antihypertensive treatment

The percentage of hypertensive treatment used in PIH pregnancy were less than chronic hypertensive pregnancy (15.2% vs 83.3%). The difference was statistically significant (p < 0.001).

Table 9 Obstetric outcomes according to type of hypertensive disorders of pregnancy.

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Premature rupture of membrane	(n = 246)		(n = 126)		(n = 1)		
No	194	85.09	87	79.82	1	100.0	0.394
Yes	34	14.91	22	20.18	0	0.0	
Mean (SD) (min)	266.84	(296.23)	323.42	(411.31)	34.00	-	0.404 [a]
Range	2-1,885		1-2,566				
Type of membranes rupture	(n = 278)		(n = 133)		(n = 2)		
Spontaneous rupture of membrane	73	26.3	56	42.1	0	0.0	0.012
Artificial rupture of membrane	173	62.2	65	48.9	2	100.0	
Membrane leak	32	11.5	12	9.0	0	0.0	
Characteristic of amniotic fluid	(n = 214)		(n = 95)		(n = 1)		
Clear	130	61.7	53	55.8	1	100.0	0.786
Thin	26	12.2	9	9.5	0	0.0	
Thick	1	0.5	0	0.0	0	0.0	
Meconium stained	14	6.5	8	8.4	0	0.0	
Unknown	43	20.1	25	26.3	0	0.0	

*Exact probability Test
[a] Kruskall-Wallis Test

Table 9 Obstetric outcomes according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Mode of delivery							
Vaginal delivery	221	70.8	91	55.5	2	33.3	0.004 [c]
Vacuum extraction	15	4.8	17	10.4	1	16.7	
Forceps extraction	24	7.7	10	6.1	1	16.7	
Cesarean section	52	16.7	46	28.0	2	33.3	
Indication for abnormal delivery							
No	218	69.9	89	54.3	1	16.7	< 0.001 [c]
Maternal cause	58	18.6	63	38.4	5	83.3	
Neonatal cause	23	7.3	12	7.3	0	0.0	
Prophylaxis	13	4.2	0	0.0	0	0.0	

[c] Chi-square Test

Table 9 Obstetric outcomes according to type of hypertensive disorders of pregnancy(continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Duration of labour							
First stage labour (min)	(n = 258)		(n = 118)		(n = 3)		
Mean (SD)	393.64	(155.55)	369.11	(150.25)	365.00	(225.66)	0.357[a]
Range	50-960		60-865		220-625		
Second stage labour	(n = 258)		(n = 118)		(n = 3)		
Nullipara (min)	(n = 118)		(n = 42)		(n = 2)		
Mean (SD)	24.47	(17.76)	39.19	(34.14)	25.50	(12.02)	0.023[a]
Range	2-115		6-153		17-34		
Prolonged labour	(n = 140)		(n = 76)		(n = 1)		
No	110	93.2	34	80.9	2	100.0	0.058
Yes	8	6.8	8	19.1	0	0.0	

*Exact probability Test
[a] Kruskall-Wallis Test

Table 9 Obstetric outcomes according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Second stage labour	(n = 258)		(n = 118)		(n = 3)		
Multipara (min)							
Mean (SD)	21.44	(15.93)	24.83	(18.92)	32.00	-	0.391 [a]
Range	2-88		1-90				
Prolonged labour							
No	110	78.6	53	69.7	1	100.0	0.384
Yes	30	21.4	23	20.3	0	0.0	
Third stage labour (min)							
Mean (SD)	5.41	(5.52)	7.03	(9.17)	10.67	(10.69)	0.073 [a]
Range	1-52		1-62		4-23		
Total duration of labour (min)							
Mean (SD)	421.88	(157.96)	409.13	(160.56)	403.33	(210.27)	0.710 [a]
Range	64-980		82-874		275-646		

*Exact probability Test
[a] Kruskall-Wallis Test

Table 9 Obstetric outcomes according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Estimated blood loss (cc)							
≤ 250	13	4.2	5	3.1	0	0.0	0.482 [c]
251-500	293	93.9	153	93.3	6	100.0	
501-750	6	1.9	4	2.4	0	0.0	
751-1000	0	0.0	2	1.2	0	0.0	
Mean (SD)	346.15	(93.87)	381.10	(123.51)	366.67	(103.28)	0.035 [a]
Range	150-700		200-1000		300-500		
Primary postpartum hemorrhage							
No	306	98.1	158	96.3	6	100.0	0.446
Yes	6	1.9	6	3.7	0	0.0	

*Exact probability Test
[a] Kruskall-Wallis Test
[c] Chi-square Test

Table 9 Obstetric outcomes according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Placenta weight (gm)	(n = 307)		(n = 158)		(n = 6)		
0-250	0	0.0	1	0.6	0	0.0	0.008
251-500	93	30.3	46	29.1	5	83.3	
501-750	200	65.1	94	59.5	1	16.7	
751-1000	14	4.6	17	10.8	0	0.0	
Mean (SD)	578.24	(97.62)	596.83	(123.19)	460.00	(89.44)	0.008 [a]
Range	330-1,000		200-1,000		360-600		
Placenta characteristics	(n = 307)		(n = 158)		(n = 6)		
Complete	300	92.7	154	97.5	6	100.0	0.283
Incomplete	4	1.3	0	0.0	0	0.0	
Infraction	3	1.0	4	2.5	0	0.0	

*Exact probability Test
[a] Kruskall-Wallis Test

Table 9 Obstetric outcomes according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Cord length (cm)	(n = 307)		(n = 158)		(n = 6)		
26-50	180	58.6	98	62.0	5	83.3	0.675
51-75	119	38.8	55	34.8	1	16.7	
76-100	8	2.6	5	3.2	0	0.0	
Mean (SD)	52.04	(9.52)	52.06	(10.38)	45.83	(8.01)	0.206 [a]
Range	28-93		30-90		40-60		
Antihypertensive treatment							
No	312	100.0	139	84.8	1	16.7	< 0.001
Yes	0	0.0	25	15.2	5	83.3	

*Exact probability Test
[a] Kruskall-Wallis Test

3.4 Neonatal outcome

Neonatal outcomes are presented in Table 10.

Neonatal gender

The gender of babies were also similar (p = 0.408). The proportions of male and female were nearly 50% in each group.

Birth weight

The mean birth weight of babies born to normotensive mothers was 3,083.05 grams (SD = 377.02), more than those of babies born to PIH pregnancy (3,039.45 grams, SD = 517.02) and chronic hypertensive pregnancy (2,225 grams, SD = 680.67). There was significant difference among these three groups (p = 0.007).

Birth length

The mean birth length was 51.41 centimeters (SD = 2.17) in babies born to normotensive mothers, more than those in babies born to PIH mothers (50.95 centimeters, SD = 2.71), and in chronic hypertensive mothers (46.60 centimeters, SD = 4.28). The means of birth length of the babies in each group were also significantly different (p = 0.008).

Head circumference

The mean head circumference was 32.72 centimeters (SD = 1.38) in babies born to normotensive mothers, 32.87 centimeters (SD = 1.65) in babies born to PIH mothers, and 30.1centimeters (SD = 2.35) in babies born to chronic hypertensive mothers. There was significant difference (p = 0.029).

Cord coiling

Most of babies had no cord coiling (93.3% in babies born to normotensive, 90.9% in babies born to PIH mothers, and 83.3% in babies born to chronic hypertensive mothers). The differences was not significant ($p = 0.422$).

Apgar score at 1 minutes and 5 minutes

There was no significant difference in Apgar score at 1 minutes and 5 minutes ($p = 0.438$ and $p = 1.000$, respectively). Most babies had Apgar score at 1 and 5 minutes of 8 – 10 score: 97.1% and 93.3% in babies born to normotensive mothers, 95.1% and 99.4% in babies born to PIH mothers, and 100% in babies born to chronic hypertensive mothers, respectively.

Birth complication

The most common birth complications of babies born to normotensive and PIH mothers were meconium aspiration (8.7% and 12.2%), birth asphyxia (2.9% and 4.9%) and fever (2.2% and 3.6%), while the birth complication of babies born to chronic hypertensive mothers was fever (16.7%). The birth complications among three groups were not significantly different ($p = 0.129$).

Congenital anomalies

The most of babies born to normotensive (99.4%) and PIH mother (99.4%) had no congenital anomalies, while 50.0% of babies born to chronic hypertensive mothers had congenital anomalies. Considering the subgroup analysis of congenital anomalies, the percentages of Down syndrome and ambigous genitalis in babies born to chronic hypertensive mothers were more than that in normotensive mothers and PIH mothers. There was significant difference ($p = 0.014$ and $p < 0.001$).

Table 10 Neonatal outcomes according to type of hypertensive disorders of pregnancy.

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Neonatal gender							
Male	159	50.9	73	44.5	3	50.0	0.408
Female	153	49.1	91	55.5	3	50.0	
Birth weight (gm)							
1000-2499	19	6.1	17	10.3	5	83.3	< 0.001
2500-4000	291	93.3	140	85.4	1	16.7	
4001-4500	2	0.6	7	4.3	0	0.0	
Mean (SD)	3,083.05	(377.02)	3,039.45	(517.02)	2,225.00	(680.67)	0.007 [a]
Range	2,120-4,200		1,180-4,400		1,310-3,310		

*Exact probability Test
[a] Kruskall-Wallis Test

Table 10 Neonatal outcomes according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Birth length (cm)	(n = 307)		(n = 163)		(n = 5)		
30-40	0	0.0	1	0.6	1	20.0	< 0.008
41-50	111	36.2	62	38.0	3	60.0	
51-60	196	63.8	100	61.4	1	20.0	
Mean (SD)	51.41	(2.17)	50.95	(2.71)	46.60	(4.28)	0.008 [a]
Range	44-57		38-58		40-51		
Head circumferences (cm)							
26-30	20	6.5	14	8.6	4	80.0	< 0.001
31-35	283	92.2	140	58.9	1	20.0	
36-40	4	1.3	9	5.5	0	0.0	
Mean (SD)	32.72	(1.38)	32.87	(1.65)	30.10	(2.35)	0.029 [a]
Range	27.0-37.0		29.0-36.9		28.0-34.0		

*Exact probability Test
[a] Kruskall-Wallis Test

Table 10 Neonatal outcomes according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Cord coiling							
0	291	93.3	149	90.9	5	83.3	0.422
1	14	4.5	10	6.1	1	16.7	
≥ 1	7	2.2	5	3.0	0	0.0	
Apgar score at 1 minute							
8-10	303	97.1	156	95.1	6	100.0	0.438
4-7	9	2.9	8	4.9	0	0.0	
Apgar score at 5 minute	(n = 304)		(n = 161)		(n = 6)		
8-10	302	93.3	160	99.4	6	100.0	1.000
4-7	2	0.7	1	0.6	0	0.0	

*Exact probability Test

Table 10 Neonatal outcomes according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Birth complication							
No	269	86.2	130	79.3	5	83.3	0.129
Yes	43	13.8	34	20.7	1	16.7	
Birth asphyxia	9	2.9	8	4.9	0	0.0	0.437
Meconium aspiration	27	8.7	20	12.2	0	0.0	0.456
Fever	7	2.2	6	3.6	1	16.7	0.114
Congenital anomalies							
No	310	99.4	163	99.4	3	50.0	< 0.001
Yes	2	0.6	1	0.6	3	50.0	
Congenital heart disease	1	0.3	0	0.0	0	0.0	1.000
Down syndrome	0	0.0	0	0.0	1	16.7	0.014
Ambigous genitalis	0	0.0	0	0.0	2	33.3	< 0.001
Flat foot	0	0.0	1	0.6	0	0.0	0.354
Multiple anomalies	1	0.3	0	0.0	0	0.0	1.000

*Exact probability Test

3.5 The effect of hypertensive disorders on obstetric outcome

Table 11 shows the effect of hypertensive disorders on obstetric outcomes compared with normotensive pregnant women.

Premature rupture of membrane

After adjusting for gestational age and occupation, the duration of premature rupture of membrane in PIH was longer than normotensive pregnancy, whereas that in chronic hypertensive pregnancy was shorter than normotensive pregnancy. The mean difference was 56.58 minutes (95%CI = -16.55 to 129.72, p = 0.129) for PIH pregnancy, and 232.84 minutes (95%CI = -901.78 to 436.11, p = 0.494) for chronic hypertensive pregnancy.

Type of membranes rupture

After adjusting for gestational age and complications during pregnancy, the overall risk for abnormal type of membranes rupture was 0.92 times for PIH (95%CI = 0.78 to 1.09, p = 0.313) and 1.23 times for chronic hypertensive pregnancy (95%CI = 0.46 to 3.30, p = 0.677).

Characteristic of amniotic fluid

Pregnancy-induced hypertension had higher risk of abnormal characteristics of amniotic fluid after adjusting for gestational age and duration of first stage labour. The adjusted RR was 1.13 (95%CI = 0.85 to 1.49, p = 0.452).

Abnormal mode of delivery

After adjusting for gestational age, complications during pregnancy and birth weight, PIH and chronic hypertensive pregnancy had increased risk of abnormal mode of delivery compared to normotensive pregnancy.

The adjusted risk ratio of abnormal mode of delivery were 1.52 (95%CI = 1.11 to 2.08, p = 0.009) for PIH pregnancy, and 3.10 (95%CI = 1.08 to 8.94, p = 0.036) for chronic hypertensive pregnancy.

Indication for abnormal delivery

After adjusting for gestational age, complication during pregnancy and birth weight, pregnancy-induced hypertensive pregnancy and chronic hypertensive pregnancy were at an increased risk of indication of abnormal delivery compared to normotensive pregnancy. The adjusted risk ratio of having indication for abnormal delivery was 1.51 (95%CI = 1.11 to 2.06, p = 0.009) for PIH pregnancy, and 3.49 (95%CI = 1.33 to 9.13, p = 0.011) for chronic hypertensive pregnancy.

The duration of first stage labour

The duration of first stage labour in PIH and chronic hypertensive pregnancy was shorter than normotensive pregnancy. The mean difference was 14.94 minutes (95%CI = -47.38 to 17.50, p = 0.366) for PIH pregnancy, and 31.98 minutes (95%CI = -200.68 to 136.71, p = 0.709) for chronic hypertensive pregnancy after controlling for gravida.

The duration of second stage labour

In nullipara, after adjusting for age, gravida, duration of membranes rupture and birth weight, PIH pregnancy had duration of second stage labour of 12.75 minutes (95%CI = 4.09 to 21.09, p = 0.003), significantly longer than normotensive pregnancy. Chronic hypertensive pregnancy also had duration of second stage labour of 10.90 minutes longer than normotensive pregnancy (95%CI = -33.97 to 55.76, p = 0.632).

In multipara, PIH pregnancy had duration of second stage labour of 2.61 minutes longer than normotensive pregnancy after adjusting for age, gravida, duration of

rupture membranes and birth weight. It did not show statistical significance (95%CI = -2.30 to 7.52, p = 0.295).

Prolonged labour in second stage

In nullipara, pregnancy-induced hypertension and chronic hypertensive pregnancy were at an increased risk of prolonged labour in second stage compared to normotensive pregnancy after controlling for age, gravida, duration of membrane rupture, and birth weight. The adjusted risk ratio was 2.81 (95%CI = 1.12 to 7.01, p = 0.028) for PIH and 7.37 (95%CI = 1.58 to 34.36, p = 0.021) for chronic hypertensive pregnancy.

In multipara, pregnancy-induced hypertension and chronic hypertensive pregnancy were at an increased risk of prolonged labour in second stage compared to normotensive pregnancy after controlling for age, gravida, duration of rupture membrane and birth weight. The adjusted risk ratio was 1.41 (95%CI = 0.87 to 2.25, p = 0.102) for PIH and 4.65 (95%CI = 3.40 to 6.41, p = 0.220) for chronic hypertensive pregnancy.

The duration of third stage labour

The durations of third stage in PIH and chronic hypertensive pregnancy were 1.62 minutes (95%CI = 0.11 to 3.13, p = 0.035) and 5.25 minutes (95%CI = -2.62 to 13.13, p = 0.191), longer than normotensive pregnancy after controlling for age and placenta weight.

Prolonged labour in third stage

After adjusting for age and placenta weight, pregnancy-induced hypertension and chronic hypertensive pregnancy increased the risk of prolonged labour in third stage compared to normotensive pregnancy. The adjusted risk ratio of prolonged

labour in third stage for PIH pregnancy was 3.28 (95%CI = 0.56 to 19.37, p = 0.181) and the estimate risk ratio for chronic hypertensive pregnancy was 43.0 (95%CI = 5.20 to 355.91, p < 0.001).

Estimated blood loss at delivery

After adjusting for parity, mode of delivery, duration of labour, placenta weight and birth weight, the estimated blood loss at delivery in PIH pregnancy was more than normotensive pregnancy. The mean difference was 21.34 cc (95%CI = 2.18 to 40.50, p = 0.029). The estimated blood loss in chronic hypertensive pregnancy was also more than those in normotensive pregnancy. The mean difference was 8.24 cc (95%CI = -92.13 to 108.61, p = 0.872).

Primary postpartum hemorrhage

After adjusting for parity, mode of delivery, duration of labour, placenta weight and birth weight, pregnancy-induced hypertension and chronic hypertensive pregnancy did not increase the risk of primary postpartum hemorrhage compared to normotensive pregnancy. The adjusted risk ratio of primary postpartum hemorrhage for PIH pregnancy was 1.74 (95%CI = 0.79 to 3.87, p = 0.181), and the estimated risk ratio for chronic hypertensive pregnancy was 4.33 (95%CI = 0.67 to 28.21, p = 0.116).

Placenta weight

After adjustment for gestational age, HIV infection, serology test for VDRL, placenta weight in PIH pregnancy was more than that in normotensive pregnancy. A significant mean difference of 30.32 grams (95%CI = 5.17 to 55.47, p = 0.018) still existed. On the other hand, placental weight in chronic hypertensive pregnancy was less compared to normotensive pregnancy, and there was no significant difference. The mean difference was 83.89 grams (95%CI = -172.49 to 4.70, p = 0.063).

Cord length

After controlling for gestational age, cord length of pregnancy-induced hypertension and chronic hypertensive pregnancy were longer than normotensive pregnancy. The mean differences was 0.26 centimeters (95%CI = -1.62 to 2.14, p = 0.786) and 3.58 centimeters (95%CI = -11.70 to 4.54, p = 0.387).

Placenta characteristic

Pregnancy-induced hypertension and chronic hypertensive pregnancy were not at increased risk of abnormal placental characteristics compared to normotensive pregnancy. The adjusted risk ratio of abnormal placental characteristics for PIH pregnancy was 1.11 (95%CI = 0.33 to 3.74, p = 1.000) and the estimated risk ratio for chronic hypertensive pregnancy was 7.31 (95%CI = 1.06 to 50.52, p = 0.027) after controlling for gestational age and complication during pregnancy.

Table 11 The effect of hypertensive disorders on obstetric outcome from multiple regression.

Characteristics	Pregnancy-induced hypertension				Chronic hypertension			
	Regression Coef.	95% CI		p-value	Regression Coef.	95% CI		p-value
Premature rupture of membrane (min) [e]	56.58	-16.55	129.72	0.129	-232.84	-901.78	436.11	0.494
Type of membrane rupture [f]	0.92	0.78	1.09	0.313	1.23	0.46	3.30	0.677
Characteristic of amniotic fluid [g]	#1.13	0.85	1.49	0.452				
Abnormal mode of delivery [h]	@1.52	1.11	2.08	0.009	@3.10	1.08	8.94	0.036
Indication for abnormal delivery [h]	@1.51	1.11	2.06	0.009	@3.49	1.33	9.13	0.011
First stage labour [i]	-14.94	-47.38	17.50	0.366	-31.98	-200.68	136.71	0.709

[e] Adjusted for gestational age, occupation
[f] Adjusted for gestational age, complication during pregnancy
[g] Adjusted for gestational age, duration of first stage labour
[h] Adjusted for gestational age, complication during pregnancy, birth weight
[i] Adjusted for age, gravida
@ Risk ratio
# Estimate risk ratio

Table 11 The effect of hypertensive disorders on obstetric outcome from multiple regression (continued).

Characteristic	Pregnancy-induced hypertension				Chronic hypertension			
	Regression Coef.	95% CI		p-value	Regression Coef.	95% CI		p-value
Second stage labour [j]								
Nullipara	12.75	4.09	21.09	0.003	10.90	-33.97	55.76	0.632
Multipara	2.61	-2.30	7.52	0.295				
Prolonged labour in second stage [j]								
Nullipara	@2.81	1.12	7.01	0.028	@7.37	1.58	34.36	0.021
Mutipara	@1.41	0.87	2.25	0.102	@4.67	3.40	6.41	0.220
Third stage labour [k]	1.62	0.11	3.13	0.035	5.25	-2.62	13.13	0.191
Prolonged labour in third stage [k]	@3.28	0.56	19.37	0.181	#43.0	5.20	355.91	< 0.001

[j] Adjusted for age, gravida, duration of membranes rupture, birth weight
[k] Adjusted for age, placenta weight
@ Risk ratio
# Estimate risk ratio

Table 11 The effect of hypertensive disorders on birth outcome from multiple regression (continued).

Characteristics	Pregnancy-induced hypertension				Chronic hypertension			
	Regression Coef.	95% CI		P-value	Regression Coef.	95% CI		P-value
Estimated blood loss (cc) [l]	21.34	2.18	40.50	0.029	8.24	-92.13	108.61	0.872
Primary postpartum hemorrhage [l]	[@]1.74	0.79	3.87	0.181	[#]4.33	0.67	28.21	0.116
Placental weight (gm) [m]	30.32	5.17	55.47	0.018	-83.89	-172.49	4.70	0.063
Cord length (cm) [n]	0.26	-1.62	2.14	0.786	-3.58	-11.70	4.54	0.387
Placental characteristic [f]	[@]1.11	0.33	3.74	1.000	[#]7.31	1.06	50.52	0.027

[l] Adjusted for parity, mode of delivery, duration of labour, placental weight, birth weight
[m] Adjusted for gestational age, HIV infection, serology test for VDRL
[f] Adjusted for gestational age, complication during pregnancy
[n] Adjusted for gestational age
[@] Risk ratio
[#] Estimate risk ratio

3.6 The effect of hypertensive disorders on neonatal outcome

Table 12 shows the effect of hypertensive disorders on birth outcomes, compared with normotensive pregnant women.

Birth weight

After adjusting for age, gestational age, BMI at first visit, maternal weight gain, gravida and OF test, the birth weight in babies born to PIH and chronic hypertensive mothers were less than that babies born to normotensive mothers. A significant mean difference was 518.30 grams (95%CI = -844.28 to –192.32, p = 0.002), and 81.93 grams (95%CI = -173.38 to 9.52, p = 0.018), respectively.

Low birth weight

After adjusting for age, gestational age, BMI at first visit, maternal weight gain, gravida and OF test, PIH and chronic hypertensive pregnancy increased risk of low birth weight. The adjusted risk ratio was 2.28 (95%CI = 0.97 to 5.36, p = 0.059) for PIH and 4.15 (95%CI = 1.01 to 17.10, p = 0.049) for chronic hypertensive pregnancy.

Birth length and Head circumferences

After controlling for age, gestational age, BMI at first visit, maternal weight, gravida, the average birth length of babies born to PIH mothers was shorter while mean head circumferences was larger compared to that born to normotensive mothers. The mean difference was 0.32 centimeters (95%CI = -0.78 to 0.14, p = 0.173) for birth length and 0.01 centimeters (95%CI = -0.29 to 0.30, p = 0.963) for head circumferences.

Babies born to chronic hypertensive mothers had shorter birth length and smaller head circumferences than babies born to normotensive mothers. The mean difference

was 3.41 centimeters (95%CI = -5.41 to –1.42, p = 0.001) for birth length and 2.02 centimeters (95%CI = -3.29 to –0.76, p = 0.002) for head circumferences.

Cord coiling

Cord coiling of babies born to PIH and chronic hypertensive mothers were more than those born to normotensive mothers. The mean difference was 0.03 (95%CI = -0.05 to 0.11, p = 0.508) for babies born to PIH mothers, and 0.10 (95%CI = -0.26 to 0.45, p = 0.595) for those born to chronic hypertensive mothers after controlling for placental length and birth weight.

Apgar score at 1 minute

After controlling for duration of labour, gestational age, birth weight and birth complication, babies born to PIH mothers and chronic hypertensive mothers had less Apgar score at 1 minute than babies born to normotensive mothers. The mean difference was 0.07 minutes (95%CI = -0.27 to 0.13, p = 0.489) in babies born to PIH mothers, and 0.60 minutes (95%CI = -1.64 to 0.45, p = 0.264) in babies born to chronic hypertensive mothers.

Apgar score at 5 minute

After controlling for duration of second stage labour, gestational age and birth weight, infant born to PIH and chronic hypertensive mothers had less Apgar score at 5 minute than babies born to normotensive mothers. The mean difference was 0.07 minutes (95%CI = -0.24 to 0.10, p = 0.409) for babies born to PIH mothers, and 0.56 minutes (95%CI = -1.43 to 0.32, p = 0.363) for babies born to chronic hypertensive mothers.

Birth complication

Pregnancy-induced hypertension and chronic hypertensive pregnancy were at an increased risk of birth complication compared to normotensive pregnancy. The risk ratio of birth complication for PIH pregnancy was 1.39 (95%CI = 0.85 to 2.28, p = 0.194) and 2.61 (95%CI = 0.33 to 20.56, p = 0.363) for chronic hypertensive pregnancy after controlling for mode of delivery, duration of membranes rupture, duration of second stage of labour, and characteristic of amniotic fluid.

Congenital anomalies

Pregnancy-induced hypertension did not increase the risk of congenital anomalies. The risk ratio of congenital anomalies for PIH pregnancy was 0.95 (95%CI = 0.09 to 10.49, p = 0.967) while chronic hypertensive pregnancy increased the risk of congenital anomalies. The risk ratio was 78.0 (95%CI = 13.03 to 466.80, p < 0.001).

Table 12 The effect of hypertensive disorders on neonatal outcomes from multiple regression.

Characteristics	Pregnancy-induced hypertension				Chronic hypertension			
	Regression Coef.	95% CI		p-value	Regression Coef.	95% CI		p-value
Birth weight (gm) [o]	-81.93	-173.38	9.52	0.079	-518.30	-844.28	-192.32	0.002
Low birth weight [o]	[@]2.28	0.97	5.36	0.059	[@]4.15	1.01	17.10	0.049
Birth length (cm) [p]	-0.32	-0.78	0.14	0.173	-3.41	-5.41	-1.42	0.001
Head circumferences (cm) [p]	0.01	-0.29	0.30	0.963	-2.02	-3.29	-0.76	0.002
Cord coiling [q]	0.03	-0.05	0.11	0.508	0.10	-0.26	0.45	0.595
Apgar score at 1 minute [r]	-0.07	-0.27	0.13	0.489	-0.60	-1.64	0.45	0.264
Apgar score at 5 minute [r]	-0.07	-0.24	0.10	0.409	-0.56	-1.43	0.32	0.210
Birth complication [s]	[@]1.39	0.85	2.28	0.194	[@]2.61	0.33	20.56	0.363
Congenital anomalies	[@]0.95	0.09	10.49	0.967	[@]78.0	13.03	466.80	< 0.001

[@] Risk ratio

[#] Estimate risk ratio

[o] Adjusted for age, gestational age, BMI at first visit, maternal weight gain, gravida, OF test

[p] Adjusted for age, gestational age, BMI at first visit, maternal weight gain, gravida

[q] Adjusted for cord length, birth weight

[r] Adjusted for duration of labour, gestational age, birth weight, birth complication

[s] Adjusted for mode of delivery, duration of membrane rupture, duration of second stage labour, characteristic of amniotic fluid

4. The maternal and neonatal outcomes in postpartum period

4.1 The maternal outcomes in postpartum period

The postpartum maternal outcomes are in Table 13.

Pureperium infection

The percentages of pureperium infection among the three groups were not statistically different ($p = 0.145$), 1.3% in normotensive, 4.3% in PIH pregnancy, whereas the chronic hypertensive pregnancy had no pureperium infection.

Abnormal conditions at discharge

Abnormal conditions at discharge among three groups were significantly different ($p = 0.001$). All chronic hypertensive pregnancy had abnormal conditions at discharge. The abnormal conditions at discharge of chronic hypretensive pregnancy were 83.3% of hypertension and 16.7% of hypertension and DM. There was significant difference ($p < 0.001$ and $p = 0.014$).

Maternal last status

All subjects in this study were alive.

Duration of maternal hospitalization

The mean duration of maternal hospitalization was 4.11 days (SD = 2.24) in normotensive pregnancy, 5.16 days (SD = 2.87) in PIH pregnancy, and 9.50 days (SD = 2.74) in chronic hypertensive pregnancy. The difference in mean duration of maternal hospitalization was statistically significant ($p < 0.001$).

Table 13 Postpartum characteristics of mothers according to type of hypertensive disorders of pregnancy.

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Pureperium infection							
No	308	98.7	157	95.7	6	100.0	0.145
Yes	4	1.3	1	4.3	0	0.0	
Abnormal conditions at discharge							
Normal	312	100.0	152	92.7	0	0.0	0.001
Abnormal	0	0.0	12	7.3	6	100.0	
Hypertension	0	0.0	11	6.7	5	83.3	< 0.001
Fever	0	0.0	1	0.6	0	0.0	0.354
Hypertension and DM	0	0.0	0	0.0	1	16.7	0.014

*Exact probability Test

Table 13 Postpartum characteristics of mothers according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Maternal last status							
Dead	0	0.0	0	0.0	0	0.0	
Alive	312	100.0	164	100.0	6	100.0	
Duration of maternal hospitalization (days)							
1-3	177	56.7	61	37.2	0	0.0	< 0.001
4-7	114	36.6	84	51.2	1	16.7	
≥ 8	21	6.7	19	11.6	5	83.3	
Mean (SD)	4.11	(2.24)	5.16	(2.87)	9.50	(2.74)	< 0.001 [a]
Range	2-17		2-23		7-13		

*Exact probability Test
[a] Kruskall-Wallis Test

4.2 The neonatal outcomes in postpartum period

Table 14 shows the postpartum neonatal outcomes.

Highest hematocrit level within 7 days

The highest hematocrit levels within 7 days among the three groups were not significantly different (p = 0.050). The mean hematocrit level within 7 days was 58.42% (SD = 6.60) in babies born to normotensive mothers, 58.98% (SD = 6.77) in babies born to PIH mothers, and 65.40% (SD = 3.85) in babies born to chronic hypertensive mothers.

Highest hematocrit level after 7 days

There was no significant difference (p = 0.713) in the highest hematocrit level after 7 days among these three groups. The mean hematocrit level after 7 days was 51.60% (SD = 6.75) in babies born to normotensive mothers, less than those born to PIH mothers (51.67%, SD = 1.53), and those born to chronic hyypertensive mothers (55.67%, SD = 11.06).

Highest bilirubin level within 7 days

The mean bilirubin level within 7 days in normal weight babies born to normotensive mothers was 14.24 mg% (SD = 3.87), more than babies born to PIH mothers (13.02 mg%, SD = 3.75). There was a significant difference (p = 0.034). In addition, the mean bilirubin level within 7 days was 14.26 mg% (SD = 3.91) in low birth weight infants born to normotensive mothers, 13.67 mg% (SD = 4.51) in those born to PIH mothers, and 16.62 mg% (SD = 2.57) in those born to chronic hypertensive mothers. There was a significant difference (p = 0.029).

Highest bilirubin level after 7 days

The bilirubin level after 7 days in low birth weight infants born to normotensive mothers was 12.20 mg% (SD = 0), 10.00 mg% (SD = 1.01) of those born to PIH and 13.27 mg% (SD = 3.69) of those born to chronic hypertensive mothers. The differences were not statistically significant (p = 0.193).

Neonatal fever within 7 days and after 7 days delivery

The neonatal fever within 7 days and after 7 days of delivery were not significantly different (p = 0.635 and p = 0.177). The percentage of neonatal fever within 7 days was 10.6% in babies born to normatensive mothers, 11.6% in those born to PIH mothers, and 16.7% in those born to chronic hypertensive mothers. Only 2.2% in babies born to normotensive mothers had fever after 7 days of birth.

Neonatal morbidity

According to neonatal morbidity, the most common babies born to normotensive and PIH mothers were birth asphyxia (5.5% and 3.7%), but the difference was not statistically significant (p = 0.612). The neonatal morbidity in those born to chronic hypertensive mothers was polycythemia (16.7%), and there was a significant difference (p = 0.043).

Abnormal conditions at discharge or referral

The most common abnormal conditions at discharge or referral were jaundice: 7.4% in babies born to normotensive mothers and 8.5% in babies born to PIH mothers. Whereas the abnormal conditions at discharge or referral of babies born to chronic hypertensive mothers were jaundice / polycythemia / congenital anomalies (16.7%). There was a significant difference (p = 0.014).

Duration of neonatal hospitalization

The mean duration of neonatal hospitalization was 4.39 days (SD = 2.35) in babies born to normotensive mothers, less than those of babies born to PIH mothers (5.58 days, SD = 4.67) and those of babies born to chronic mothers (21.50 days, SD = 15.03). The difference was statistically significant ($p < 0.001$).

Table 14 Postpartum characteristics of neonates according to type of hypertensive disorders of pregnancy.

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Hightest hematrocrit level within 7 days (%)	(n = 191)		(n = 111)		(n = 5)		
38-40	1	0.5	0	0.0	0	0.0	0.116
41-65	455	81.2	87	78.4	2	40.0	
66-76	35	18.3	24	21.6	3	60.0	
Mean (SD)	58.42	(6.60)	58.98	(6.77)	65.40	(3.85)	0.050 [a]
Range	38-76		40-74		61-70		
Hightest hematrocrit level after 7 days (%)	(n = 15)		(n = 3)		(n = 3)		
45-54	14	93.3	3	100.0	2	66.7	0.500
55-64	1	6.7	0	0.0	1	33.3	
Mean (SD)	51.60	(6.75)	51.67	(1.53)	55.67	(11.06)	0.713 [a]
Range	40-66		50-53		44-66		

*Exact probability Test
[a] Kruskall-Wallis Test

Table 14 Postpartum characteristics of neonates according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Hightest bilirulin level within 7 days (mg%)	(n = 171)		(n = 101)		(n = 5)		
Normal weight infant	(n = 158)		(n = 8)		(n = 0)		
≤ 14.0	80	50.6	53	61.6			0.094
14.1-17.9	65	41.2	31	363.1	Na		
≥18.0	13	8.2	2	2.3			
Mean (SD)	14.24	(3.87)	13.02	(3.75)			0.034[b]
Range	7.5-27.0		4.6-25.8				
Low birth weight infant							
≤ 12.0	3	23.1	6	40.0	0	0.0	0.211
121-17.9	8	61.5	7	46.7	2	40.0	
≥18.0	2	15.4	2	13.3	3	60.0	
Mean (SD)	14.26	(3.91)	13.67	(4.51)	16.62	(2.57)	0.029[a]
Range	9.2-23.7		4.7-19.0		13.0-18.7		

*Exact probability Test
[a] Kruskall-Wallis Test
[b] Mann Withney *U* Test

Table 14 Postpartum characteristics of neonates according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Hightest bilirubin level after 7 days (mg%)	(n = 13)		(n = 3)		(n = 3)		
Normal weight infant	(n = 12)		(n = 0)		(n = 0)		
≤ 14.0	10	83.3	na		na		
14.1-17.9	2	16.7					
Mean (SD)	12.76	(2.11)					
Range	10.5-17.8						
Low birth weight infant	(n = 1)		(n = 3)		(n = 3)		
≤ 12.0	0	0.0	3	100.0	2	66.7	0.571
12.1-17.9	1	100.0	0	0.0	1	33.3	
Mean (SD)	12.20	(0.00)	10.00	(1.01)	13.27	(3.69)	0.193 [a]
Range			9.1-11.1		10.7-17.5		

*Exact probability Test
[a] Kruskall-Wallis Test

Table 14 Postpartum characteristics of neonates according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Hyperbilirubinemia within 7 days	(n = 171)		(n = 101)		(n = 5)		
Normal weight infant	(n = 158)		(n = 86)		(n = 0)		
No	80	50.6	53	61.6	0	0.0	0.108[b]
Yes	78	49.4	33	38.4	0	0.0	
Low birth weight infant	(n = 13)		(n = 15)		(n = 5)		
No	3	23.1	6	40.0	0	0.0	0.288
Yes	10	76.9	9	60.0	5	100.0	
Hyperbilirubinemia after 7 days	(n = 13)		(n = 3)		(n = 3)		
Normal weight infant	(n = 12)						
No	10	83.3					
Yes	2	16.7					
Low birth weight infant	(n = 1)		(n = 3)		(n = 3)		
No	0	0.0	3	100.0	2	66.7	0.571
Yes	1	100.0	0	0.0	1	13.3	

*Exact probability Test
[b] Mann withney *U* Test

Table 14 Postpartum characteristics of neonates according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Neonatal fever within 7 days							
No	279	89.4	145	88.4	5	83.3	0.635
Yes	33	10.6	19	11.6	1	16.7	
Neonatal fever after 7 days							
No	305	97.8	164	100.0	6	100.0	0.177
Yes	7	2.2	0	0.0	0	0.0	
Neonatal morbidity							
No	290	92.9	154	93.9	5	83.3	0.425
Yes	22	7.1	10	6.1	1	16.7	
Birth asphyxia	17	5.5	6	3.7	0	0.0	0.612
Anemia	1	0.3	1	0.6	0	0.0	1.000
Skin infection	3	1.0	1	0.6	0	0.0	1.000
Respiratory distress syndrome	0	0.0	1	0.6	0	0.0	0.354
Polycythemia	1	0.3	1	0.6	1	16.7	0.043

*Exact probability Test

Table 14 Postpartum characteristics of neonates according to type of hypertensive disorders of pregnancy (continued).

Characteristics	Normotensive		Type of hypertensive disorders				p-value*
			Pregnancy-induced hypertension		Chronic hypertension		
	n = 312	%	n = 164	%	n = 6	%	
Abnormal conditions at discharge or referral							
Normal	279	89.4	147	89.6	5	83.3	0.669
Abnormal	33	10.6	17	10.4	1	16.7	
Fever	1	0.3	0	0.0	0	0.0	1.000
Jaundice	23	7.4	14	8.5	0	0.0	0.821
Dead	1	0.3	0	0.0	0	0.0	1.000
Fever and juandice	3	1.0	0	0.0	0	0.0	0.571
Anemia	1	0.3	1	0.6	0	0.0	1.000
Skin infection	3	1.0	2	1.2	0	0.0	1.000
Fever and	1	0.3	0	0.0	0	0.0	1.000
Juandice/polycythemia/congenital anomaly	0	0.0	0	0.0	1	16.7	0.014
Duration of neonatal hospitalization (days)							
1 – 3	158	50.6	57	34.8	0	0.0	< 0.001
4 – 7	122	39.1	84	51.2	1	16.7	
≥ 8	32	10.3	23	14.0	5	83.3	
Mean (SD)	4.39	(2.35)	5.58	(4.67)	21.50	(15.03)	< 0.001 [a]
Range	1-17		2-51		7-42		

*Exact probability Test, [a]Kruskall-Wallis Test

4.3 The effect of hypertensive disorders on postpartum maternal outcomes

Table 15 shows the effect of hypertensive disorders on postpartum maternal outcomes compared with normotensive pregnant women.

Pureperium infection

Pregnancy-induced hypertension and chronic hypertension were at an increased risk of pureperium infection compared to normotensive pregnancy. The adjusted risk ratio was 3.33 (95%CI = 0.99 to 11.21, p = 0.053) for PIH pregnancy, and the estimated risk ratio was 13.0 (95%CI = 1.70 to 99.68, p = 0.003) for chronic hypertensive pregnancy after controlling for mode of delivery, gestational age, type of membranes rupture, duration of rupture membrane, and duration of second stage.

Abnormal conditions at discharge

Pregnancy-induced hypertension and chronic hypertensive pregnancy were at an increase risk of abnormal conditions at discharge compared to normotensive pregnancy. The estimated risk ratio of abnormal conditions at discharge was 22.83 (95%CI = 2.99 to 174.04, p < 0.001) for PIH pregnancy, and 260.0 (95%CI = 35.57 to 1900.65, p < 0.001) for chronic hypertensive pregnancy.

Table 15 The effect of hypertensive disorders on postpartum maternal outcome from multiple regression.

Characteristics	Pregnancy-induced hypertension				Chronic hypertension			
	Regression Coef.	95% CI		p-value	Regression Coef.	95% CI		p-value
Pureperium infection [t]	@3.33	0.99	11.21	0.053	#13.0	1.70	99.68	0.003
Abnormal conditions at discharge	#22.83	2.99	174.04	< 0.001	#260.0	35.57	1900.65	< 0.001

[t] Adjusted for mode of delivery, gestational age, type of membranes rupture, duration of rupture membrane, duration of second stage

@ = Risk ratio

= Estimate risk ratio

4.4 The effect of hypertensive disorders on neonatal outcomes in postpartum period

Table 16 shows the effect of hypertensive disorders on postpartum neonatal outcomes compared with normotensive pregnant women.

Hematocrit level within 7 days

After controlling for gestational age and birth weigh, and complication during pregnancy, the hematocrit levels after 7 days in both babies born to PIH and chronic hypertensive mothers were more than those born to normotensive mothers. The mean differences were 0.63% (95% CI = -0.92 to 2.19, p = 0.425) and 5.69% (95% CI = -0.50 to 11.88, p = 0.071), respectively.

Hematocrit level after 7 days

After controlling for gestational age and birth weigh, and complication during pregnancy, the hematocrit levels after 7 days babies born to PIH and chronic hypertensive mothers were less than that born to normotensive mothers. The mean differences were 6.42% (95% CI = -20.16 to 7.32, p = 0.336) and 1.47% (95% CI = -14.70 to 11.77, p = 0.817), respectively.

Bilirubin level within 7 days

After controlling for gestational age, duration of second stage labour mode of delivery and birth weight, the bilirubin level within 7 days in normal weight infant born to PIH mothers was less than those born to normotensive mothers. The mean difference was 0.40 mg% (95CI = -1.60 to 0.81, p = 0.517), and it was not statistically significant.

Bilirubin level within 7 days in low birth weight infant born to PIH mothers was less than that born to normotensive mothers. But low birth weight infant born to

chronic hypertensive mothers has higher bilirubin level within 7 days than those born to normotensive mothers. The mean differences were 0.89 mg% (95%CI = -4.89 to 3.12, p = 0.649) and 5.70 mg% (95% CI = -5.36 to 16 76, p = 0.294). There was no significant difference.

Hyperbilirubinemia within 7 days

After adjusting for gestational age, duration of second stage labour, mode of delivery and birth weight, normal weight infant born to PIH mothers increased risk of hyperbilirubinemia within 7 days of 0.95 time (95% CI = 0.59 to 1.54, p = 0.845).

Low birth weight infant born to PIH and chronic hypertensive mothers increased risk of hyperbilirubinemia after 7 days of 0.68 times (95% CI = 0.21 to 2.17, p = 0.517) and 0.74 times (95% CI = 0.05 to 10.12, p = 0.824).

Hyperbilirubinemia after 7 days

After adjusting for gestation age, duration of second stage labour, mode of delivery and birth weight, low birth weight infants born to chronic hypertensive mothers increased risk of hyperbilirubinemia after 7 days. The adjusted risk ratio was 0.33 (95 CI = 0.07 to 1.65, p = 0.248).

Neonatal fever within 7 day

After adjusting for gestational age, and birth weight, both in babies born to PIH and chronic hypertensive mothers had higher risk of neonatal fever within 7 days, but there was no significant difference. The adjusted risk ratios were 1.07 (95% CI = 0.60 to 1.88,p = 0.826), and 1.75(95% CI = 0.14 to 9.47, p = 0.896), respectively.

Neonatal fever after 7 days

Babies born to PIH and chronic hypertensive mothers had risk of neonatal fever after 7 days of 0.27 times (95% CI = 0.03 to 2.19, p = 0.188, and estimated risk ratio was 7.43 times (95% CI = 1.07 to 51.35,p = 0.025), respectively.

Neonatal morbidity

After adjusting for gestational age, duration of second stage labour, the babaies born to PIH and chronic hypertensive mothers were an increased risk of neonatal morbidity. The adjusted risk ratio were 0.36 (95% CI = 0.42 to 1.78, p = 0.694), and 2.36 (95% CI = 0.38 to 14.79,p = 0.358), respectively.

Abnormal conditions at discharge or referral

Babies born to PIH and chronic hypertensive mothers had risk of abnormal conditions at discharge or referral of 0.98 time (95 CI = 0.56 to 1.71, p = 0.943), and 1.58 time (95 CI = 0.26 to 9.71, p = 0.632).

Table 16 The effect of hypertensive disorders on postpartum neonatal outcome from multiple regression.

Characteristics	Pregnancy-induced hypertension				Chronic hypertension			
	Regression Coef.	95% CI		p-value	Regression Coef.	95% CI		p-value
Hightest hematrocrit level in 7 days [u]	0.63	-0.92	2.19	0.425	5.69	-0.50	11.88	0.071
Hightest hematrocrit level after 7 days [u]	-6.42	-20.16	7.32	0.336	-1.47	-14.70	11.77	0.817
Hightest bilirubin level within 7 days [v]								
Normal weight infant	-0.40	-1.60	0.81	0.517				
Low birth weight infant	-0.89	-4.89	3.12	0.649	5.70	-5.36	16.76	0.294
Hyperbilirubinemia within 7days [v]								
Normal weight infant	[@]0.95	0.59	1.54	0.845				
Low birth weight infant	[@]0.68	0.21	2.17	0.517	[@]0.74	0.05	10.12	0.824

[u] Adjusted for gestational age, birth weight, complication during pregnancy

[v] Adjusted for gestational age, duration of second stage labour, mode of delivery, birth weight

[@] Risk ratio

Table 16 The effect of hypertensive disorders on postpartum neonatal outcome from multiple regression.

Characteristics	Pregnancy-induced hypertension				Chronic hypertension			
	Regression Coef.	95% CI		p-value	Regression Coef.	95% CI		p-value
Hyperbilirunemia after 7 days [v]								
Low birth weight infant					[@]0.33	0.07	1.65	0.248
Neonatal fever within 7 days [u]	[@]1.07	0.60	1.88	0.826	[@]1.15	0.14	9.47	0.896
Neonatal fever after 7 days [u]	[@]0.27	0.03	2.19	0.188	[#]7.43	1.07	51.35	0.025
Neonatal morbidity [w]	[@]0.86	0.42	1.78	0.694	[@]2.36	0.38	14.79	0.358
Abnormal conditions at discharge or referral	[@]0.98	0.56	1.71	0.943	[@]1.58	0.26	9.71	0.62

[v] Adjusted for gestational age, duration of second stage labour, mode of delivery, birth weight
[u] Adjusted for gestational age, birth weight
[w] Adjusted for gestational age, duration of second stage labour, birth weight
[@] Risk ratio
[#] Estimate risk ratio

5. The significant of effects of hypertensive disorders on maternal and neonatal outcomes.

Table 17 shows the significant effect of hypertensive disorders on maternal and neonatal outcomes, compared with normotensive pregnant women.

5.1 Maternal outcomes

5.1.1 Demographic and reproductive characteristics

- The mean age of chronic hypertensive pregnancy was more than PIH and normotensive pregnancy ($p < 0.001$).
- The mean pre-pregnancy weight of PIH pregnancy was lower than normotensive pregnancy ($p < 0.001$).
- Chronic hypertensive pregnancy had family history of hypertension more than PIH and normotensive pregnancy ($p = 0.001$).
- Chronic hypertensive pregnancy had past history of hypertensive pregnancy more than PIH and normotensive pregnancy ($p < 0.001$).

5.1.2 Preqnancy progression

- The mean maternal weight at first visit in PIH pregnancy was more than chronic hypertensive and normatensive pregnancy ($p < 0.001$).
- The mean BMI at first visit in chronic hypertensive pregnancy was more than PIH and normotensive pregnancy ($p < 0.001$).
- The intrauterine growth retardation (IUGR) occurred in chronic hypertensive pregnancy more than normotensive and PIH pregnancy ($p = 0.029$).

5.1.3 Pregnancy outcomes

- The gestational age of chronic hypertensive was shorter than normotensive pregnancy (mean difference 3.86, 95%CI = -5.68 to -2.03), whereas PIH pregnancy did not show statistically difference (mean difference 0.17, 95%CI = -0.67 to 0.33).
- Chronic hypertensive and PIH pregnancy increased risk of abnormal delivery 3.10 and 1.52 (95% CI = 1.08 to 8.94, and 95% CI = 1.11 to 2.08).
- Chronic hypertensive and PIH pregnancy increased risk of indication for abnormal delivery 3.49 and 1.51 (95% CI = 1.33 to 9.13, and 95% CI = 1.11 to 2.06).
- The duration of the second stage labour of PIH pregnancy was longer than normotensive pregnancy (mean difference 20.86, 95% CI = 5.71 to 36.01), whereas chronic hypertensive pregnancy did not show statistically difference (mean difference 6.77, 95% CI = -74.77 to 88.32).
- Chronic hypertensive and PIH pregnancy increased risk of prolonged second stage in nullipara 7.37 and 2.81 times (95% CI = 1.58 to 34.36, and 95% CI = 1.12 to 7.01).
- The estimated blood loss of PIH pregnancy was more than that in normotensive pregnancy (mean difference 21.34, 95% CI = 2.18 to 40.50), whereas chronic hypertensive pregnancy did not show statistically difference (mean difference 8.24, 95% CI = -92.13 to 108.61).
- The placenta weight of PIH pregnancy was more than normotensive pregnancy (mean difference 30.32, 95% CI = 5.17 to 55.47), while chronic hypertensive pregnancy did not show statistically difference (mean difference 83.89, 95% CI = -172.49 to 4.70).

5.2 Neonatal outcomes

- The birth weight of babies born to chronic hypertensive mothers was lower than babies born to normotensive mothers (mean difference 581.30, 95% CI = -844.28 to -192.32), whereas babies born to PIH mothers did not show statistically difference (mean difference 81.93, 95% CI = -178.38 to 9.52).

- The babies born to chronic hypertensive mothers increased risk of low birth weight 4.15 times (95% CI = 1.01 to 17.10), whereas PIH mothers did not show statistically difference (adjusted RR 2.28, 95% CI = 0.97 to 5.36).

- The birth length of babies born to chronic hypertensive mothers was shorter than that born to normotensive mothers (mean difference 3.41, 95% CI = -5.41 to -1.42), whereas babies born to PIH mothers did not show statistically difference (mean difference 0.32, 95% CI = -0.78 to 0.14).

- The head circumference of babies born to chronic hypertensive mothers was smaller than that born to normotensive mothers (mean difference 2.02, 95% CI = 95% CI = -3.29 to -0.76), whereas babies born to PIH mothers did not show statistically difference (mean difference 0.01, 95% CI = -0.29 to 0.30).

- The babies born to chronic hypertensive mothers increased risk of congenital anomalies 78.0 times (95% CI = 13.03 to 466.80), whereas PIH mothers did not show statistically difference (adjusted RR 0.95, 95% CI = 0.09 to 10.49).

Table 17 The significantly difference of effect of hypertensive disorders on pregnancy outcomes from multiple regression, classified by type of hypertension.

Characteristics	Pregnancy-induced hypertension				Chronic hypertension			
	Regression Coef.	95% CI		p-value	Regression Coef.	95% CI		p-value
Gestational age (week) [c]	-0.17	-0.67	0.33	0.500	-3.86	-5.68	-2.03	< 0.001
Prematurity [d]	@1.02	0.52	1.98	0.960	@5.70	1.90	17.10	0.002
Mode of delivery [h]	@1.52	1.11	2.08	0.009	@3.10	1.08	8.94	0.036
Indication for mode of delivery [h]	@1.51	1.11	2.06	0.009	@3.49	1.33	9.13	0.011
Second stage labour in nullipara [j]	20.86	5.70	36.01	0.007	6.77	-74.77	88.32	0.870
Prolonged labour in second stage [j] Nullipara	@2.81	1.12	7.01	0.028	@7.37	1.58	34.36	0.021
Estimated blood loss (cc) [l]	21.34	2.18	40.50	0.029	8.24	-92.13	108.61	0.872

[c] Adjusted for age, gravida, abortion, previous preterm delivery, history of hypertension pregnancy, HIV infection

[d] Adjusted for gravida, abortion, previous preterm delivery, complication during pregnancy, HIV infection

[h] Adjusted for gestational age, complication during pregnancy, birth weight

[j] Adjusted for age, gravida, duration of rupture membrane, birth weight

[l] Adjusted for parity, mode of delivery, duration of labour, placenta weight, birth weight

@ Risk ratio

Table 17 The significantly difference of effect of hypertensive disorders on pregnancy outcomes from multiple regression, classified by type of hypertension.

Characteristics	Pregnancy-induced hypertension				Chronic hypertension			
	Regression Coef.	95% CI		p-value	Regression Coef.	95% CI		p-value
Placental weight (gm) [m]	30.32	5.17	55.47	0.018	-83.89	-172.49	4.70	0.063
Birth weight (gm) [o]	-81.93	-173.38	9.52	0.079	-518.30	-844.28	-192.32	0.002
Low birth weight [o]	[@]2.28	0.97	5.36	0.059	[@]4.15	1.01	17.10	0.049
Birth length (cm) [p]	-0.32	-0.78	0.14	0.173	-3.41	-5.41	-1.42	0.001
Head circumferences [p]	0.01	-0.29	0.30	0.963	-2.02	-3.29	-0.76	0.002
Congenital anomalies	[@]0.95	0.09	10.49	0.967	[@]78.0	13.03	466.80	< 0.001

[m] Adjusted for gestational age, HIV infection, serology test for VDRL

[o] Adjusted for age, gestational age, BMI at first visit, maternal weight gain, gravida, OF test

[p] Adjusted for age, gestational age, BMI at first visit, maternal weight gain, gravida

[@] Risk ratio

CHAPTER V

DICUSSION

Discussion

The discussion in this study were presented as follow:

1. The effect of hypertensive disorders on maternal outcome
2. The effect of hypertensive disorders on neonatal outcome

1. The effect of hypertensive disorders on maternal outcome

1.1 Maternal weight and BMI at first antenatal visit

The finding at first ANC visit demonstrated that maternal weight and BMI in PIH and chronic hypertensive pregnancy increased more than normotensive pregnancy, which was similar to the result of Anyaegbunam AM, et al. (10). They reported that the significant difference in mean maternal weight when hypertensive gestational diabetes women were compared to all non-hypertensive gestational diabetes women. In addition, the result of Ravi Thadhani, et al showed the risk of gestational hypertension increased as pre gravida BMI increased (42). Therefore, assessment of maternal weight gain and BMI at the first ANC visit is important to provide appropriate care to pregnant mother with such conditions. This appropriate care will help to reduce maternal morbidity and mortality due to PIH.

1.2 Hematocrit level during pregnancy

The hematocrit level during pregnancy includes hematocrit level at first visit and third trimester. The difference of hematocrit levels among three groups during pregnancy did not vary to a significant level. In general, the first hematologic change noted in preeclampsia is hemoconcentration. The hematocrit in severe preeclampsia and eclampsia tends to increase (61). The chronic hypertensive pregnancy could be expected to show similar hematologic changes but the sample size of our study did not allow showing the difference. For PIH pregnancy, it was possible that the largest group in PIH pregnancy were gestational hypertensive that did not find an increasing hematocrit level.

1.3 Complication during pregnancy

Our study showed that intrauterine growth retardation (IUGR) occurred more frequently in chronic hypertensive pregnancy than normotensive pregnancy (16.7% vs 0.3%, $p = 0.029$), similar to many previous studies (8, 11, 16, 20-21, 55). It could be explained that hypertensive disorders of pregnancy could result in fetal hypoxia, fetal death, intrauterine growth retardation, low birth weight and SGA infant (28, 43-44, 54-55).

1.4 Type of membrane rupture

The proportion of artificial membrane rupture required in chronic hypertensive (16.7%) pregnancy was more frequent than normotensive pregnancy (0.3%). This result might be the consideration of artificial membrane rupture in order to decide immediately delivery (62). This finding was similar to the result of the previous studies (10, 14,16). But the multivariate analysis was not statistically significant difference.

1.5 Mode of delivery

The cesarean section rate in both PIH (28.0%) and chronic hypertensive pregnancy (33.3%) were significantly higher than normotensive pregnancy (16.7%). Furthermore, after controlling for potentially confounding, PIH and chronic hypertensive pregnancy had an increased risk of abnormal delivery (adjusted RR = 1.52, 95% CI = 1.10 to 2.08 and 3.10, 95% CI = 1.08 to 8.94, respectively). Our finding confirmed previous studies (13, 14, 16, 22, 24, 26, 51). However, there was one study reporting no difference in delivered by cesarean section (25).

1.6 The duration of second stage labour

Result from the univariate and multivariate analysis showed that the duration of second stage in nullipara of PIH pregnancy was significantly longer than normotensive pregnancy (mean difference 12.75, 95%CI = 4.09 to 21.09, p = 0.023, and mean difference 10.90, 95%CI = -33.97 to 55.76, p = 0.003). In addition, we found that the PIH pregnancy increased risk of prolonged second stage of labour in nullipara (adjusted RR 2.81, 95% CI = 1.12 to 7.01). This significant difference was probably caused by the larger number of primiparas had PIH during pregnancy. It is estimated that PIH is associated with prolonged second stage of labour in nulliparas.

1.7 Estimated blood loss at delivery

The averages of estimated blood loss at the time of delivery in PIH and chronic hypertensive pregnancy were more than normotensive pregnancy (381.10 cc vs 366.67 cc vs 346.15 cc), and were also significant after adjusting for parity, mode of delivery, duration of labour, placenta weight, and birth weight (95%CI = 2.18 to 40.50 and 95%CI = -92.13 to 108.61). The women with severe preeclampsia or eclampsia usually have hypervolemia, so they have much tolerance of blood loss than the

normotensive pregnant women (32). In addition, magnesium sulfate inhibits uterine contraction, so magnesium sulfate therapy appears to prolong bleeding time, and increase blood loss at delivery (63).

1.8 Gestational age

The mean gestational age was lower in chronic hypertensive pregnancy compared to normotensive pregnancy. After controlling for potentially confounding variables, the strongly significant difference was found in both gestational age and prematurity of chronic hypertensive pregnancy (mean difference 3.86, 95% CI = -5.68 to -2.03 and adjusted RR = 5.70, 95% CI = 1.90 to 17.10). This study result was similar to several previous studies (8, 10-14, 17-21, 23-24).

2. The effect of hypertensive disorders on neonatal outcome

2.1 Birth weight and low birth weight

The mean birth weight of infants born to chronic hypertensive mothers was significantly lower than the born to normotensive mothers. Moreover, infants born to chronic hypertensive mothers increased the risk of low birth weight, adjusted RR was 4.15 (95% CI = 1.01 to 17.10) which was similar to those of several other studies (10-13, 16-17, 20-23, 51). It estimated that chronic hypertension was associated with low birth weight infant. The small gestational age infant in this study could not be evaluated, because there was no available records during the study period.

2.2 Birth length and head circumference

Considering birth length and head circumference, the result showed that infants born to chronic hypertensive mothers had significantly shorter birth length and smaller head circumference than normotensive mothers after adjusting for potential confounding variables, especially gestational age (mean difference 3.41 cm, 95%CI = -5.41 to -1.42 and 95%CI = -3.29 to -0.76). The reason might be that IUGR occurred more frequently in chronic hypertensive mothers than normotensive mothers, so there was possibility of limitation to fetal growth. It estimated that chronic hypertension was associated with less birth length and head circumference.

2.3 Apgar score

The previous studies (22, 25) found that neonates born to hypertensive women were more likely to have low 5-minute Apgar scores than those from normotensive women. However, our study showed no significant difference in the Apgar score among the neonates of the three groups.

2.4 Congenital anomaly

The most common congenital anomaly in babies born to chronic hypertensive mothers was Down syndrome (16.7%) and ambiguous genitalis (33%). Statistical differences ($p < 0.001$) existed in congenital anomaly among babies born to chronic hypertensive mothers and normotensive mothers. But we did not conclude that the chronic hypertension associated with congenital anomaly, because these conditions have multifactorial inheritance (61).

2.5 The effect of hypertensive disorder on maternal and neonatal outcome in postpartum period

However, we did not find any difference in terms of maternal and neonatal outcome in postpartum period.

CHAPTER VI

CONCLUSION

Conclusion

The aim of this retrospective cohort study was to explore the effect of hypertensive disorders on maternal and neonatal outcome. The study compared outcome of hypertensive pregnant women to normotensive pregnant women and their babies. The participants of this study were 482 pregnant women, who attended an antenatal care clinic and delivered at the Mother and Child Hospital, Chiang Mai during December 1, 1997 to April 30, 1999. Data were collected from the hospital records, and traced from the first antenatal visit until they were discharged from the hospital.

These results could be concluded that hypertensive pregnant women are associated with more adverse pregnancy outcome than normotensive pregnant women. The adverse effects of hypertensive disorders of pregnancy include shorter duration of gestational age, increased risk of indication for abnormal delivery, prolongation of 2^{nd} and 3^{rd} stage of labor, primary postpartum hemorrhage, less placental weight, abnormal characteristics of placenta, and abnormal conditions at discharge. The women with chronic hypertension are at risk for adverse outcome than those with pregnancy-induced hypertension.

Furthermore, infants born to hypertensive mothers had an increase risk of low birth weight, shorter birth length, and smaller head circumferences than infants born to normotensive mothers. These results were significant different in hypertensive pregnant women and theirs infants.

Recommendation

1. Early antenatal care

The result of the study revealed that hypertensive pregnant women had more prepregnancy weight, BMI at the first visit, IUGR rate, family history of hypertension, and past history of hypertensive pregnancy than normotensive pregnant women in prenatal care. Therefore, the hypertensive pregnant women, especially with chronic hypertension should be concerned as high-risk pregnancy. When PIH is detected early during the time of antenatal care, the pregnant women should receive health education to control hypertension, and special care should be given as necessary. They should be advised that a well-balanced diet with high protein and salt intake should be restricted as indicated earlier and keep watching on daily fetal movement, occurrence of headache, visual disturbances and epigastric pain.

During pregnancy

Hypertensive pregnancy, especially chronic hypertension should have ANC at least every 2-3 weeks until 30^{th} weeks of gestation and then weekly thereafter. In addition, fetal status should be also evaluated. The fetal surveillance such as fetal movement charts, non-stress tests, biophysical profile and ultrasonographic measurement of fetal growth should be monitored.

2. Labour and delivery

The result indicated that the duration of second stage in hypertensive pregnancy was longer, and the estimated blood loss was more than those in normotensive pregnancy, so avoidance of prolonged second stage and reduction of estimated blood loss should be made by immediately placenta delivery.

3. Postpartum care

Blood pressure of mother should be monitored until 6 month after delivery. If possible, health education and premarital counseling services should be offered for early appropriate intervention, to avoid an undesirable outcome of pregnancy.

Recommendation for future study

1. Prospective study for more details of outcome of hypertensive disorders in pregnancy.

2. To compare the outcome according to type of hypertensive disorder in pregnancy.

3. Long-term study for the postpartum period of maternal and neonatal health status.

4. Study for the effect of antihypertensive medication on pregnancy outcome.

REFERENCES

1. Sadd Rana. Obstetric and Perinatal care for developing countries. Islamabad, Pakistan: SAF Publication; 1998.
2. กระทรวงสาธารณสุข. รายงานผลการพัฒนาสาธารณสุขในแผนพัฒนาฯฉบับที่ 7 (พ.ศ. 2535 – 2539).กรุงเทพมหานคร: องค์การสงเคราะห์ทหารผ่านศึก; 2541.
3. วิชัย เทียนถาวร. สถานการณ์งานส่งเสริมสุขภาพในอดีต ปัจจุบันและแนวโน้มในอดีต. การประชุมเชิงปฏิบัติการจัดทำแผนดำเนินงานส่งเสริมสุขภาพในระยะ 20 ปี; 22-23 มกราคม 2540; โรงแรม เอส ดี อเวนิว : ม.ป.ท.; 2540: 1-9.
4. บุญศรี จันทร์รัชชกูล, รสิก รังสิปราการ และบัญญ พันธ์บูรณะ. สูติศาสตร์ร่วมสมัย. กรุงเทพมหานคร: สำนักพิมพ์ข้าวฟ่าง; 2542.
5. สุรศักดิ์ ฐานีพานิชสกุล.การส่งเสริมสุขภาพมารดาใน 20 ปีข้างหน้า. การประชุมเชิงปฏิบัติการการจัดทำแผนดำเนินงานส่งเสริมสุขภาพในระยะ 20 ปี; 22 – 23 มกราคม 2540; โรงแรม เอสดี อเวนิว: ม.ป.ท; 2540: 1-9.
6. กำแหง จาตุรจินดาและคณะ. สูติศาสตร์รามาธิบดี . กรุงเทพมหานคร: OS.Printing House CO.LTD; 2530.
7. Derham RJ,Hawkins DF,DC Viries LS,Aber VR, Elder MG. Outcome of pregnancies complicated by severe hypertension and delivered before 34 week;stepwise logistic regression analysis of prognotic factor. Br J Obstet Gynecol 1989 OCT; 96(10): 1173-81.
8. Haddad B, Sibai BM. Chronic hypertension in pregnancy. Ann Med 1999AUG; 31 (4): 246-52.

9. Buga GA,Lumu SB. Hypertensive disorders of pregnancy at Umtata General Hospital: perinatal and maternal outcomes. East Afr Med J 1999 Apr; 76 (4): 217-22.

10. Anyaegbunam AM, Scarpelli S, Mikhail MS. Chronic hypertension in gestational diabetes: influence on pregnancy outcome. Gynecol Obstet Invest 1995; 39 (3): 167-70.

11. Xiong X, Mayes D, Demianczuk N, Olson DM, Davidge ST, Newburn-Cook C, et al. Impact of pregnancy-induced hypertension on fetal growth. Am J Obstet Gynecol 1999 Jan; 180(1pt1): 2007-13.

12. Himmeimann A, Svensson A, Hansson L. Relation of maternal and blood pressure during pregnancy to birth weight and blood pressure in children: The Hypertension in Pregnancy off spring study. J Intern Med 1994 Apr; 235 (4): 347-52.

13. Kiss I, Lazar A, Pauline F,Farsang C. Retrospective analysis of clinical data of normotensive and hypertensive pregnant woman and their newborns. J Hypertens Suppl 1989 Dec; 7(6): S336- 7.

14. Yadav S, Saxena U ,Yadar R, Gupta S. Hypertensive disorder of pregnancy and maternal and foetal outcome: A case controlled study .J Indian Med Assoc 1997 Oct; 95(10): 548-51.

15. Pietrantoni M, O Brien WF. The current impact of the hypertensive disorder of pregnancy. Clin Exp Hypertens 1994 Jul; 16(4): 479-92.

16. Omu AE, al- Othman S, al-Qattan, al-Falah FZ, Sharma P. A comparative study of obstetric outcome of patient with pregnancy induced hypertension :economic considerarion .Acta Obstet Gynecol Scand 1996May; 75(5): 443-8.

17. Ananth CV, Peedicayil A, Savitz DA. Effect of hypertensive disease in pregnancy on birth weight, gestational duration and small gestational age birth . Epidemiology 1995 Jul;6(4):391-5.

18. Mahomed K, Mudzamiri S, Munetsi J, Nyoni J, Gakuje P. Hypertension in pregnancy – a major hospital cause for concern .Cent Afr J Med 1997 Jan; 43(1):11-6.

19. North RA, Taylor RS, Schellenberg JC. Evaluation of a definition of pre-eclampsia. Br J Obstst Gynecol 1999 Aug ;106(8):767-73.

20. Jain L. Effect of pregnancy-induced and chronic hypertension of pregnancy outcome. J Perinatal 1997 Nov-Dec; 17(6):425-7.

21. Jain L , Ferre C, Vidyasagar D. Racial differences in outcome of pregnancies complicated by hypertension. J Matern fetal Med 1998 Jam-Feb; 7(1): 23-7.

22. Fernandez Jonusas S, Ceriani Cernadas JM. The effects of arterial hypertension during pregnancy on birth weight intrauterine growth retardation and neonatal evolution. A matched case control study. An Esp Pediatr 1999 Jan; 50(1): 52-6.

23. Delmis J, Drazancic A, Ivanisevic M, Pfeifer D, Ljubujevec N. Fetal growth in pregnant women with chronic hypertension. Lijec Vjesn 1993 Nov- Dec; 115(11-12): 329-35.

24. Rey E, Couturier A. The prognosis of pregnancy in women with chromic hypertension. Am J Obset Gynecol 1994 Aug;171(2):410-6.

25. Morgan MA, Berkowitz KM, Thomas SJ, Reimbold P, Quilligan EJ. Abruptio placentae: perinatal outcome in normotensive and hypertensive patients. Am J Obstet Gynecol 1994 Jun; 170(6) :1595-9.

26. Gupta KB, Randhawa I, Pal A , Premi HK, Ganeshan J. Perinatal outcome in pregnancy induced hypertension. J Indian Med Assoc 1996 Jan; 94(1):6-16.

27. Helain J.Landy. The impact of Maternal illness on the Neonate. In: Neonate Pathophysiology & Management of the Newborn 5th ed. Gordon B. Avery, Mary Ann Fletcher, Mhalri G, MacDonald editor. London: Lippincott William & Wilkins; 1999.

28. ธีระ ทองสง และ สุรีย์ สิมารักษ์. ความดันโลหิตสูงในสตรีตั้งครรภ์. ม.ป.ท. ; ม.ป.ป.

29. กนก สีจร, สมพร โพธินาม , ยุทธพงศ์ วีระวัฒนกูล, วิฑูรย์ ประเสริฐเจริญสุข. ภาวะฉุกเฉินทางสูตินรีเวช. ขอนแก่น: ขอนแก่นการพิมพ์; 2537.

30. จตุพล ศรีสมบูรณ์, อภิชาต โอฬารรัตนชัย และธเนนทร์ วนาภิรักษ์. คู่มือสูติศาสตร์และนรีเวชวิทยา. กรุงเทพมหานคร: แมคกรอ - ฮิล อินเตอร์เนชั่นแนล เอ็นเตอร์ไพรส์อิงค์; 2538.

31. วราวุธ สุมาวงศ์. คู่มือการฝากครรภ์และการคลอด.พิมพ์ครั้งที่ 12 . กรุงเทพมหานคร: ม.ป.ท.; 2527.

32. Cunningham, et al. William Obstetrics 20th ed.Philadephia: Appleton & Lange; 1997.

33. NA Beischer, EV Macky and PB Colditz. Obstetrics and the Newborn An Illustrated Textbook 3th ed. Philadephia: W.B. Saunders; 1997.

34. ธีระพร วุฒิยวณิช , ธีระ ทองสง , จตุพล ศรีสมบูรณ์. ตำราสูติศาสตร์ เล่ม 1. เรียบเรียงครั้งที่ 2 . เชียงใหม่: หน่วยวารสารวิชาการ มหาวิทยาลัยเชียงใหม่;2539.

35. ส่าหรี จิตตินันท์, เสาวนีย์ จำเดิมเผด็จศึก. ตำรากุมารเวชศาสตร์ เล่ม 3 .กรุงเทพมหานคร. กรุงเทพเวชสาร; 2532.

36. Behrman RE, Rliegman RW, Arvin AM. Nelso textbook of pediatrics. 15th ed. Philadelphia: WB. Sannders company ; 1996.

37. กฤษณา เพ็งสา, สุกัญญา ทักษพันธ์.คู่มือทารกแรกเกิด. พิมพ์ครั้งที่ 2 .ขอนแก่น: โรงพิมพ์คลังนานาวิทยา; 2540.

38. สุจิตรา นิมมานนิตย์, ประมวล สุนากร. ปัญหาโรคเด็กที่พบบ่อย. พิมพ์ครั้งที่ 11 . กรุงเทพฯ: ดีไซร์; 2539.

39. ส่าหรี จิตตินันท์, เสาวนีย์ จำเดิมเผด็จศึก. ตำรากุมารเชศาสตร์ เล่ม 3 ใ กรุงเทพฯ: กรุงเทพเวชสาร, 2532.

40. จิรศักดิ์ มนัสากร และ วิทยา ถิฐาพันธ์.เชศาสตร์มารดาและทารก.กรุงเทพฯ:หน่วยการศึกษาภาควิชาสูติศาสตร์นรีเวชวิทยา คณะแพทยศาสตร์ศิริราชพยาบาล มหาวิทยาลัยมหิดล; 2539.

41. Scott JR, Worley RJ. Hypertensive disorder of pregnancy In: Scott JR, Disaia PJ, Hammon CB, Spellacy WN eds. Danforth's Obstetrics and gynecology. 6th ed. Philadelphia: Lippincott; 1990.

42. Ravi Thadhani, Meir J. Stampfer, David J.Hunter , etal. High Body Mass Index and Hypercholesteroemia: Risk of Hypertensive Disorders of Pregnancy; Obstetrics & Gynecology 1999 Oct; 94(9): 543-9.

43. Ferderick P. Zuspan. Hypertensive Disorders of pregnancy. In:Neonatal-Perinatal Medicine Disease of the fetus and Infant 6th ed.St.Louis: Mosby-Year book; 1997.

44. Jay M.Sullivan. Hypertension and Pregnancy.USA: Year Book Medical Publisher; 1986. p.13-17.

45. W.B. saunders. High Risk Pregnancy Management Option. Modechai Hallak in Hypertension in pregnancy. Second edition. China: Harcourt Brace and company; 1999.

46. Ronald R. Magness, Norman F.Gant. Normal vascular adaptation in pregnancy: potenials dued for understanding pregnancy-indued hypertension.In: Hypertension in pregnancy. London: Chapman & Hall; 1997. p.5-6.

47. Redman CWG. Hypertension in pregnancy. In: Swict M. editor. Medical disorders in Obstetric practice 2nd ed. Oxord: Blackwell Scientific Publications; 1989. p. 249-60.

48. William M.Baron. Hypertension. In: Medical Disorder During Pregnancy. William M.Baron, Marshall D. Lindheimer editor. St louis: Mosby-Year book; 1991. p. 23-24.

49. Piper JM, Langer O, Xenakis EM, Mcfarland M, Elliott BD, Berkus MD. Perinatal outcome in growth-restricted fetuses: do hypertensive and mormotensive pregnancies differ?. Ostet Gynecol 1996 Aug; 88(2): 194-9.

50. Rankhethoa NM, Moodley J, Adhikari M, Gonws E. Perinatal outcome of babies to black south African women with hypertension . East Afr Med J1998 Jyl; 75(7): 388-91.

51. ประธาน จินายน, นิพัฒน์ ธีรตกุลพิศาล. ภาวะความดันโลหิตสูงในสตรีตั้งครรภ์ที่โรงพยาบาลราชวิถี. วารสารกรมการแพทย์. 2534; 16(11): 623-633.

52. Helain J. Landy. The impact of Maternal illness on the Neonate. Neonatology Pathology & Management of the newborn fifth edition. Gordon B. Avery, Mary Ann Fletcher, Mhairi G. MacDonald. London: Lippincott William Wilkins; 1999.

53. E. Ablbert Reece, John C. Hobbins. Medicine of the Fetus & Mother . Andrea G. Within and Bana M Sabai. Hypertensive Disease in Pregnancy. Second edition. Lipincott-Raven publishers; 1999.

54. Marianne S. Anderson and William W. Hay, Jr. Intrauterine Growth Restiction and the small for gestational age infant. In: William 20th ed.Obstetric. Cunningham, etal. editor. Philadelphia: Appleton & Lange; 1997.

55. John H. Mattox. Core Textbook of Obstetrics & Gynecology. St.Louis: Mosby-Year book; 1998.

56. Kim CR , Vohr BR, Oh W. Effects of maternal hypertension in very-low-birth-weight infants. Arch Pediatr Adolesc Med 1996 Jul; 150(7): 686-91.

57. Misra DP. The effect of the pregnancy-induced hypertension of fetal growth: a review of the literature. Paediatr Perinat Epidemiol 1996 Jul; 10(3):244-63.

58. Lang T, Delarocque E, Astagneau P, De Schampfeleire I, Jeannee E, Salem G. Hypertension during pregnancy in Africa and infant's health. A Cohort Study in an urban setting. J Perinat Med 1993; 21(1): 13-24.

59. Merz R, Said A, Bergestrom S,Bugalho A, Samucidine M. Pregnancy-associated hypertension in Maputo. A study on maternal characteristics and perinatal outcome in 1275 consecutive cases. Int J Gynaecol Obstet 1992 Sep; 39(1) :11-5.

60. Brown MA, Buddle ML. Hypertension in pregnancy: maternal and fetal outcomes according to laboratory and clinical features. Med J Aust 1996 Oct 7; 165 (7): 360-5.

61. Michele R. Foley, Thomas H. Strong. JR. Obstetric Intensive Care a practical manual. William H. Clewell. Hypertensive emergencies in pregnancy. Philadelphia, USA: W.B. Saunder company; 1997.

62. สุรีย์ สีมารักษ์ , ธีระ ทองสง. การตั้งครรภ์และการคลอด. เรียบเรียงครั้งที่ 2 .เชียงใหม่: ศุภลักษณ์การพิมพ์; 2535.

63. Alan H. Decherney & Martin L. Pernoll. Current Obstetric & Gynecologic Dignotic & Treatment. William C. Mabie & Baha M. Sabai. Hypertensive states of pregnancy. Edition 8. USA: Appleton & Lange; 1991.

64. ชยันตร์ธร ปทุมานนท์.ระบาดวิทยาการแพทย์.กรุงเทพฯ: สุขโสภา. 2541.

APPENDIX A

MEDICAL RECORD OF HYPERTENSIVE DISORDERS OF PREGNANCY

Identify number.....

[1] Hypertensive pregnant women [0] Normotensive pregnant women

---

Maternal data

Part I Demographic characteristics

1. Name..... Surname..... HN.....
2. Age.....years Pre-pregnancy weight.....kg Height.....cm
3. Race

   [1] Thai [2] Chinese [3] Others
4. Religion

   [1] Buddhist [2] Christian [3] Muslim [4] Others
5. Marital status

   [1] Couple [2] Divorced [3] Separated [4] Widowed
6. Occupation

   [1] House wife [2] Government official [3] Private

   [4] Employee [5] Commercial [6] Farmer

   [7] Others
7. Education level

   [1] No education [2] Primary school [3] Secondary school

   [4] Vocational or equivalent and beyond [5] No information

Part II Reproductive characteristics

8. LMP.....EDC.....

9. Gravidity..... Parity..... Abortion..... Living child.....

10. No. of previous pre-term delivery.....

11. No. of previous low birth weight delivery.....

12. Family history of illness.....

13. Complication during pregnancy/underlying diseases.....

Part III Pregnancy progression

14. Laboratory test for :

HIV infection	[1] Positive	[0] Negative	[.] No test
VPRL	[1] Positive	[0] Negative	[.] No test
Hepatitis B	[1] Positive	[0] Negative	[.] No test
OF test	[1] Positive	[0] Negative	[.] No test

15. Antenatal care visit

No.ANC	Date	BW	BP	Hb/Hct	Alb/Sugar	Edema	Gestation age	Special test
1								
2								
3								
4								
5								
Delivery								

16 Weight gain during pregnancy.....(kg/week)

Part IV Pregnancy outcome at delivery

17 Admission date.....

18 Birth outcome

[1] Abortion [2] Preterm birth [3] Term birth

19 Gestational age at delivery .....weeks

20 Mode of delivery

[1] Normal labour [2] Vacuum extraction due to.....

[3] Forceps extraction due to.....[4] Cesarean section due to.....

21 Premature rupture of membrane.....hours.....minutes.

22 Type of membrane rupture

[1] Spontaneous rupture of membrane [2] Artificial rupture of membrane

[3] Membrane leakage [4] Unknown

23 Characteristics of fluid

[1] Clear [2] Thick [3] Thin

[4] Maconium stained [5] Unknown

24 Duration of labour

24.1 First stage of labour.....hours.....minutes.

24.2 Second stage of labour.....hours.....minutes.

24.3 Third stage of labour.....hours..... minutes.

24.4 Total stage of labour.....hours.....minutes.

25 Placental weight.....gm

26 Cord length.....cm

27 Placental characteristics

[1] Complete [2] Incomplete [3] Infraction

28 Estimated blood loss.....cc.

29 Primary postpartum hemorrhage [0] No [1] Yes

30 Complication during delivery.....

31 Medical used during delivery

[0] No [1] Yes.....

32 Blood pressure level

- At delivery.....mmHg
- 24 hours after delivery.....mmHg
- 48 hours after delivery.....mmHg
- 72 hours after delivery.....mmHg

Part V Postpartum data

33. Puerperium infection [0] No [1] Yes

34. Maternal last status

[1] Dead [2] Alive

35. Abnormal conditions at discharge

[0] Normal [1] Abnormal

36. Discharge date.....

Neonatal data

Part VI Neonatal data at delivery

37. Infant sex HN……..

[1] Male [2] Female

38. Date of birth……

39. Birth weight ……gm Head circumferences……cm.

Birth length …… cm

40. Birth complication……

41. Apgar score at 1 minute…… at 5 minute……

42. Congenital anomaly [0] No [1] Yes……..

43. Highest hematocrit level within 7 days……..%

Highest hematocrit level after 7 days……%

44. Highest bilirubin level within 7 days……mg% After 7 days……mg%

45. Neonatal morbidity

Signs and symptom	In 24 hours		Within 7 days		After 7 days	
	No	Yes	No	Yes	No	Yes
Fever						
Hyperbilirubinemia						
Anemia						
Other						

46. Neonatal last status

[0] Alive [1] Dead

47. Abnormal conditions at discharge

[0] Normal [1] Abnormal

48. Discharge date……

APPENDIX B

SAMPLE SIZE ESTIMATION

Sample size was calculated by using cohort study formula had continuous outcome based on the following information (64):

$$n_1 = \frac{(Z_\alpha + Z_\beta)^2 2 \times \sigma^2 \times (r+1)}{(\mu_0 - \mu_1)^2 \times r}$$

when $r = \frac{n_0}{n_1}$

n_1 = The number of exposure

n_0 = The number of non-exposure

r = The ratio of number of non-exposure to the number of exposure

α = Level of significant

β = Power of test

σ = Standard variance

μ_0 = The mean of outcome in unexposed

μ_1 = The mean of outcome in exposed

Where

$r = 2:1$

$\alpha = 0.05$ $Z_\alpha = 1.96$

$\beta = 0.80$ $Z_\beta = 0.84$

The exposure of interest was gestational age and birth weight from the study in China (11).

1. The outcome of interest was "the effect of PIH on birth weight". The mean birth weight in offspring born to PIH women was 3,172.52 grams (SD = 533.74), and 3,241.75 grams (SD = 430.79) for offspring born to normotensive women.

$$n_1 = \frac{(1.96 + 0.84)^2 \times (430.79)^2 \times (2+1)}{(3{,}172.52 - 3{,}241.75)^2 \times 2}$$

$$= 455.35$$

$$= 455$$

2. The outcome of interest was "the effect of PIH on gestational age"(11). The mean gestational age in preeclampsia women was 38.70 weeks (SD = 1.69), and 39.27 weeks (SD = 1.68) for normotensive women.

$$n_1 = \frac{(1.96 + 0.84)^2 \times (1.69)^2 \times (2+1)}{(38.70 - 39.27)^2 \times 2}$$

$$= 103.37$$

$$= 103$$

The sample size of exposure required was 455. In this study, a total of 170 exposed and 312 unexposed were obtained.

BIOGRAPHY

NAME	Miss Natdhanan Phatkrathok
DATE OF BIRTH	28 May, 1972
PLACE OF BIRTH	Nakhon ratchasima, Thailand
INSTITUTIONS ATTENDED	Baromarajchonanee College of Nakhon ratchasima, 1991-1994: Diploma of nursing science equivalence of Bachelor of Science in nursing Mahidol, University, 1998-2000: Master of Science (Epidemiology)
POSITION& OFFICE	1994-1995, In-patient department of Banleuam hospital Nakhon ratchasima, Thailand 1996-1997, In-patient department of Noonthai hospital Nakhon ratchasima, Thailand Position: Registered nurse