Global Academic Journal of Medical Sciences

Available online at www.gajrc.com
DOI: 10.36348/gajms.2021.v03i03.011

OPEN ACCESS

ISSN: 2706-9036 (P)
ISSN: 2707-2533 (0)

Original Research Article

"Risk Factors of Gestational Hypertension-Preeclampsia in Pregnant Women Patients Aged within 20-35 Years with Fetomaternal Outcome & Its Perioperative Management: A Study in Shaheed Ziaur Rahman Medical College Hospital, Bogra, Bangladesh"

Muhammad Mahmudul Haque[1*], Nitai Chandra Sarkar[2], Debashis Chowdhury[3]

[1]Assintant Prof. Department of Anaesthesiology and ICU, Shaheed Ziaur Rahman Medical College & Hospital, Bogra, Bangladesh
[2]Associate Prof. & Head, Department of Anaesthesiology and ICU, Shaheed ziaur Rahman Medical College & Hospital, Bogra, Bangladesh
[3]Senior Consultant (Anaesthesia), Chattogram Maa-O-Shishu Hospital Medical College, Chattogram, Bangladesh

***Corresponding Author**
Muhammad Mahmudul Haque

Article History
Received: 13.04.2021
Accepted: 18.05.2021
Published: 26.06.2021

Abstract: ***Background:*** Preeclampsia (PE) especially severe or early PE, is a leading cause of morbidity and mortality among the mothers and infants. To determine the maternal risk factors and fetomaternal outcome of severe preeclampsia. ***Objective:*** To find out the Risk factors of Gestational Hypertension-Preeclampsia in Pregnant Women Patients aged within 20-35 Years with Fetomaternal Outcome & Its Perioperative Management. ***Methods:*** It was a case control study, done in a Department of Anaesthesiology and ICU, Shaheed ziaur Rahman Medical College & Hospital, Bogra, Bangladesh during a period of six months from January to Jun-2018. Among 131 patients with severe preeclampsia and normal pregnant women admitted. Sampling technique were consecutive sampling methods. Singleton pregnancy between 28 to 40 weeks of gestation with severe preeclampsia was selected as study patients. Written informed consent was obtained. A questionnaire was completed for each patient including patient's age, gestational age, and parity, History of hypertension in family, weight and Body Mass Index (BMI) and PIH time level. ***Results:*** This study was out of 131 pregnant women with PIH 51 (38.93%), severe PIH 45 (34.35%), Eclampsia 24(18.32%) and Ch. Hypertension 11(8.39%) were age variation. Among the 81 cases and 50 controls regarding different risk factors age 20-35 Yrs. BMI, history of precelampsia, were found significant ($p<0.05$) between two groups. Among the case group, patients developed eclampsia 2(4.0%) abruptio placenta 3(6.0%) HELLP syndrome 2(4.0%) ascites 4(8.0%) and oliguria1 (2.0%). Among the 81 cases and 50 controls regarding different risk factors age 20-35 Yrs. BMI, history of precelampsia, were found significant ($p<0.05$) between two groups. Among the case group, patients developed eclampsia 2(4.0%) abruptio placenta 3(6.0%) HELLP syndrome 2(4.0%) ascites 4(8%) and oliguria1 (2.0). 64 (48.5%) pregnant women with PIH were having gestation time less than 28 weeks, 52(39.6%) of pregnant women with PIH were gestation time between 28-37 weeks, 11 (8.3%) of pregnant women with PIH were gestation time between 37-40 weeks, 4 (3.0%) of women with PIH were P1L1. Most 56.0% of the neonates had APGAR score 4-6 at 1 minute in case group and 12(24.0%) in control group. Showing different risk factors where regarding age 35> years, 15 patients found in case group and 4 patients in control group. Significant ($p<0.05$) difference was found between two groups. Patients had 4.93 times more likely to developed preeclampsia. Take baby in home safely in 35(70.0%) in cases group and 50(100.0%) in control group. Early neonatal death was found in

5(10.0%) in case group and not found in control group. Still birth was 10(20.0%) case group and not found control group. Statistically significant ($p<0.05$) difference was between two groups. There was found to be that still eclampsia and severe PIH contribute significantly to foetal and maternal morbidity and mortality. ***Conclusion:*** Preeclampsia is a leading cause of both fetal and maternal morbidity and mortality in the developing countries. Maternal and fetal outcome are worse in severe preeclampsia.

Keywords: Fetomaternal outcome; Preeclampsia; Pregnant women; Morbidity and mortality.

I INTRODUCTION

Preeclampsia is hypertension associated with proteinuria greater than 0.3g/L after 20 wks of gestation [1]. Pre-eclampsia may be mild and severe. Mild pre-eclampsia- if the systolic blood pressure is less than 160 mmHg and the diastolic blood pressure is less than 110 mmHg and the patient does not have any of the signs and symptoms associated with severe preeclampsia. Preeclampsia affects 2-10% of pregnant women worldwide and eclampsia 0.03-0.05% [2]. Pregnancy Induced Hypertension (HIP): It encompasses a range of disorders collectively & formerly known as toxemia of pregnancy which includes gestational hypertension, Preeclampsia & eclampsia. Gestational Hypertension is characterized by the onset of systemic hypertension within proteinuria or oedema during the last few weeks of gestation or during the immediate post-partum period. Preeclampsia may be defined as a syndrome exhibited after 20 weeks of gestation manifests as systemic hypertension, proteinuria & generalized oedema. Systemic Blood Pressure higher then 140/90 mmHg with daily urine protein losses of more than 2/gram are sufficient for the diagnosis of Preeclampsia. The reported incidences of eclampsia in developing countries are between 0.1 to 0.2 per 100 deliveries while in the Western world is 1 in 2000 to 1 in 3000 [3, 4]. It is estimated that worldwide 13% of maternal mortality is due to hypertensive disorders of pregnancy but it is much higher in developing countries where the estimates are between 20-80% in Africa and Latin America [5, 6]. Medical condition such as renal disease, chronic hypertension or high blood pressure at booking and chronic autoimmune disease are risk factors for pre-eclampsia. Other factors are thrombophilias and insulin resistance [7]. Change of paternity in multiparous women has been associated with preeclampsia and eclampsia [8]. In low socio economic status of women doubled the risk of pre-eclampsia and eclampsia [9]. A study in Australia found working women compared to non-working ones had a higher risk of developing pre-eclampsia and eclampsia (Najman et al., 1989). This may be related to the stress that women get during work. Black ethnicity has been reported as risk factor for pre-eclampsia in USA and UK [10]. Pre-eclampsia, though preventable to some extent when severe it leads to feto-maternal death. Although pre-eclampsia is not totally preventable, its early detection and proper treatment can prevent it from its complications. However, in Bangladesh antenatal care service is provided by various levels of health care providers, though their knowledge and skill may vary. For women with preexisting hypertension and/or proteinuria, the diagnosis of severe pre-eclampsia can be more difficult, but new-onset severe hypertension or proteinuria or development of other clinical or laboratory findings of severe pre-eclampsia are suggestive of preeclampsia in this setting. Preeclampsia and eclampsia are still an important cause of maternal and perinatal morbidity and mortality in the developing countries [19]. Gestational hypertension is usually defined as having blood pressure higher than 140-90 mm of Hg without the presence of proteins in urine and diagnosed after 20th week of gestation. Preeclampsia is gestational hypertension (blood pressure higher than 140-90 mm of Hg) plus protienuria (> 300 mg of protein in a 24 hr urine sample). Severe preeclampsia involves a blood pressure higher than 160-110 mm of hg, with additional medical signs and symptoms. It is referred to eclampsia when tonic clonic seizures appear in pregnant women with high blood pressure and protienuria. HELLP syndrome is referred to as a dangerous combination of three medical conditions: Haemolytic anaemia, elevated liver enzymes and low platelet count and it can complicate PIH [20].

In order to classify the hypertensive conditions of pregnancy, an arbitrary divide is made around 20th week of gestation. The woman who is found to be hypertensive prior to 20th weeks is said to have chronic or pre-existing hypertension in the absence of other pathology unrelated to pregnancy. Pregnancy induced hypertension (PIH) more common lt occurs during first pregnancies, it can also occur in subsequent pregnancies. PIH is more common in pregnant teens and in woman over age of 40 yrs. Many a times, PIH develops during second half of pregnancy, usually after 20th week, but it can also develop at the time of delievery or right after delivery. The incidence of Pregnancy induced

hypertension (PIH) in India ranges from 5-15%. The incidence of PIH in primigravidae is 16% and 7% in multigravidae. Primary pre-eclampsia occurs in 70% of PIH cases and secondary pre-eclampsia occurs in 30% in all PIH cases.(4)Foetal complications of preeclampsia and eclampsia include the risk of preterm delivery, oligohydramnios (low fluid volume within the uterus), and sub-optimal foetal growth. Maternal complications of preeclampsia and eclampsia include bleeding and clotting disorders, and HELLP syndrome.The exact cause of preeclampsia and eclampsia is not fully understood (5), but it is believed to be a disorder of the lining of blood vessels.

II. OBJECTIVE

To find out the Risk factors of Gestational Hypertension-Preeclampsia in Pregnant Women Patients Aged Within 20-35 Years with Fetomaternal Outcome & Its Perioperative Management.

III. MATERIALS AND METHODS

It was a case control study, done in Shaheed Ziaur Rahman Medical College Hospital, Bogra, during a period of six months from January to Jun-2018. The study population was 81 women with severe preeclampsia and 50 women with normal pregnancy admitted in the Department of Anaesthesiology and ICU, Shaheed ziaur Rahman Medical College & Hospital, Bogra, Bangladesh. Singleton pregnancy between 28 to 40 weeks of gestation with severe preeclampsia were selected as study patients. Those preeclampsia patients are included whose, systolic blood pressure is [3]160/mmHg and or diastolic blood pressure is [3] 110mmHg or proteinuria >5 gm/day, or oliguria of less than 500 ml in 24 hours, cerebral or visual disturbances, pulmonary edema or cyanosis, epigastric or right upper quadrant pain, impaired liver function, thrombocy-topenia, fetal growth restriction were selected as case and nor-motensive patients with above gestational age were selected as control. This study was carried out in 131 pregnant women suffering from pregnancy induced hypertension admitted through antenatal clinic as well as in emergency and data analysed for maternal and foetal outcome. Patient with chronic hypertension, diagnosed case of chronic renal failure, diagnosed case of hepatic disease, patient with cardiovascular disease, patient with haemorrhagic disor-ders, psychotic patients and patients of systemic lupus erythro-matosis were excluded from the study. A questionnaire was completed for each patient including patient's age, gestational age, parity, hypertension in family, previous h/o preeclampsia weight and Body Mass Index (BMI). Then measurment of blood pressure and proteinuria was recorded in data sheet. Ma-ternal complications before or after delivery as well as perinatal outcome were also recorded. Statistical analyses were carried out by using the Statistical Package for Social Sciences version 20 for Windows SPSS.

Defining Hypertension in Pregnancy

Hypertension during pregnancy can occur in one of three forms: chronic hypertension, GH and PE. Chronic hypertension is defined as elevated blood pressure known before conception or diagnosed before 20 weeks of gestation. GH is hypertension that develops any time after 20 weeks of gestation without proteinuria. PE is defined as hypertension (blood pressure >140/90 mm Hg) with organ damage that develops after 20 weeks of gestation and has the potential to result in serious adverse consequences for the mother and fetus. Target organ damage can be manifested by proteinuria, thrombocytopenia, elevated creatinine or liver transaminases, pulmonary edema or cerebral or visual symptoms. Severe hypertension is defined as systolic blood pressure >170 mm Hg or diastolic blood pressure >110 mm Hg.

Traditional and Nontraditional ASCVD Risk Factors in Women

Increasing among women and more impactful traditional risk factors for atherosclerotic cardiovascular disease (ASCVD) include diabetes, hypertension, dyslipidemia, smoking, obesity and physical inactivity. Emerging, nontraditional ASCVD risk factors include preterm delivery, hypertensive pregnancy disorders, GD, breast cancer treatments, autoimmune diseases and depression [29].

Steps to Assess Gestational Risk

Lack of physician awareness is one of the main reasons women who have been pregnant are lost to follow-up and fall through the cracks. Gestational risk factors should be stressed during obstetric and internal medicine training. More focused patient education during pregnancy may help in reducing the knowledge gap. Increasing awareness among primary care providers, cardiologists and cardiovascular team members as well as gynecologic-obstetrical team members of the long-term risks of this unique population is a start. Importantly, all clinicians throughout the care continuum should engage a postpartum woman in a detailed conversation about her own health and discuss the potential health of her future pregnancies. Focused questions with patients can lead to easily identifying some of the pregnancy-related complications, such as PE, GH, preterm delivery and small-for-gestational-age birth.

Source: Google

Screening for cardiovascular diseases should begin soon after a pregnancy complicated by hypertensive disorder. A large study published in BMJ last year showed that women with a hypertensive disorder of pregnancy had rates of hypertension that were 12 to 25 times higher than women with a normotensive pregnancy in the year after delivery and an increased risk persisted for decades [31]. Recently ACOG released a postpartum toolkit to address the care of women after delivery. They call the postpartum period "The Fourth Trimester" and the toolkit provides resources on postpartum weight management, postpartum follow-up of chronic disease of the cardiovascular and renal systems and long-term follow-up of pregnancy-related complications. High-risk co-management clinics for postpartum cardiovascular disease counseling need to be created, with the participation of the obstetrical team, cardiovascular providers and primary care provider. The increasing evidence of the impact of pregnancy on a woman's future cardiovascular health – and the increasing awareness by clinicians and women – will likely bring a paradigm shift in the next decade to early screening in pregnancy for ASCVD risk factors, which will require substantial changes to current models of antenatal care. Severe Preeclampsia is present when systemic blood pressure exceeds 160/110 mmHg with daily urine protein losses of more than 5gram.

Symptoms

- Headache
- Visual disturbances
- Epigastric pain
- Altered consciousness etc.

Risk factors for Preeclampsia

- Obesity
- Nulliparity
- Maternal age > 40 Years
- Previous Preeclampsia
- Chronic hypertension
- Diabetes
- Renal disease
- Multiple gestations etc.

Perioperative management of anesthesia

Preanaesthetic Assessment Airway assessment Facial oedema may indicate airway oedema which may lead to difficulty intubation. Preeclemptic patients are hypovolemic & prone to hypotension with neuroaxial anaesthesia. Preeelamptic patients are also at risk of pulmonary Oedema. A judicious hydration is indicated 500 ml – 1000 ml crystalloid preload is appropriate before Neuro axial anaesthesia.

Invasive central monitoring may be needed Laboratory investigations

- Hb%
- T/C of WBC
- DIC of WBC
- ESR
- Platlet count
- Hematocript
- Liver Function Test
- Blood Urea
- Serum Creatinine
- Arterial Blood Gas analysis
- Chest X-ray in case of pulmonary oedema etc.

Epidural analgesia is the preferred technique for labor analgesia if not contra indicated. Epidural analgesia reduces maternal catecholamine levels & can facilitate blood pressure control in labor. Epidural analgesia may improve intervillous blood flow in preeclampsia and thus improving utero placental performance and as a result fetal well-bing.

Continuous Monitoring of fetal heart rate Anesthesia for Delivery

Veginal delivery in presence of Gestational Hypertension / preeclampsia and in the absence of fetal distress is an acceptable anesthetic plan. Caesarean section is necessary in presence of fetal distress. General Anesthesia is indicated for preeclamptic mothers undergoing cesarean section who refuse regional anesthesia or who are coagulopathic. General Anesthesia is selected when hemorrhage or sepsis is the reason for an emergency cesarean section. Regional Anesthetic Techniques are selected to avoid the risks of general anesthesia in parturients with preeclampsia. The risks of general anesthesia include potentially difficult tracheal intubation owing to laryngeal oedema. Potential aspiration of gastric contents increased sensitivity to non-depolarizing muscle relaxants. Exaggerated pressor response to direct laryngoscopy and tracheal intubation.

Impaired placental blood flow

It is essential to restore intravascular fluid volume and control bollod pressure before induction of G A or starting neuroaxial anesthesia:

- Hydralazine 5-1-mg I.V Every 20-3- min
- Labetalol 20-50 mg/hr. I.V
- Nitroglycerin 10Mg/min I.V
- Nitroprusside 0.25 Mg/kg/min I.V

Spinal Anesthesia has traditionally been discouraged parturients with Preeclampsia due to risk of severe hypotension.

HELLP syndrome

This may occur in upto 20% of parturients who develop severe Preeclempsia.

Characteristic Feature

Hemolysis Elevated liver transaminase enzyme Low Platelet count Sign/Symptoms:

- Epigastric Pain
- Upper abdominal tenderness
- Systemic hypertension
- Proteinuria
- Nausea
- Vomiting
- Jaundice
- Pulmonary Oedema
- Pleural effusion
- Cerebral Oedema
- Hematuria
- Oliguria
- Acute Tubular Necrosis
- D/C

The definitive treatment of (HELLP) syndrome is the delivery of the fetus often by cesarean section. Platelet treatment before delivery packed RBC transfusion monitoring of urine output Central venous pressure monitoring.

Post anesthetic management

Monitoring of pt's pules, B P urine output, colour of urine, central venous pressure; Oxygen inhalation, Fluid resuscitation, Adequate analgesia with opioid.

IV. RESULTS

This study was out of 131 pregnant women with PIH 51 (38.93%), severe PIH 45 (34.35%), Eclampsia 24(18.32%) and Ch. Hypertension 11(8.39%) were age variation. Among the 81 cases and 50 controls regarding different risk factors age 20-35 Yrs. BMI, history of precelampsia, were found significant ($p<0.05$) between two groups. Among the case group, patients developed eclampsia 2(4.0%) abruptio placenta 3(6.0%) HELLP syndrome 2(4.0%) ascites 4(8.0%) and oliguria1 (2.0%).

Table-1: Pregnancy induced hypertension and various parameters (n=131)

		mild PIH(51)	severe PIH (45)	Eclampsia (24)	Ch. Hypertension(11)	total	% age
Age of patient	< 20 yrs.	9(17.65%)	5(11.11%)	Nil (%)	Nil (0%)	14	10.69%
	21-25 yrs.	7(13.73%)	2(4.44%)	8(33.33%)	3(27.27%)	20	15.27%
	26-30 yrs.	15(29.41%)	14(31.11%)	5(20.8%)	2(18.18%)	36	27.48%
	31 -35yrs	20(39.23%)	24(53.33%)	11(45.83%)	6(54.55%)	61	46.56%
	total	51(38.93%)	45(34.35%)	24(18.32%)	11(8.40%)	131	100%

Age of patients 46.56% of the patients with PIH fell in the age group of 31-35 yrs.

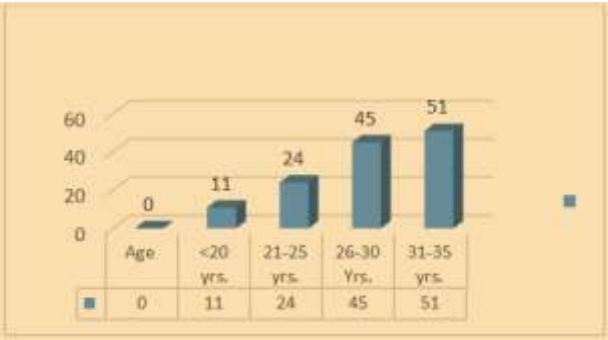

Fig-1: Age distribution of the patients

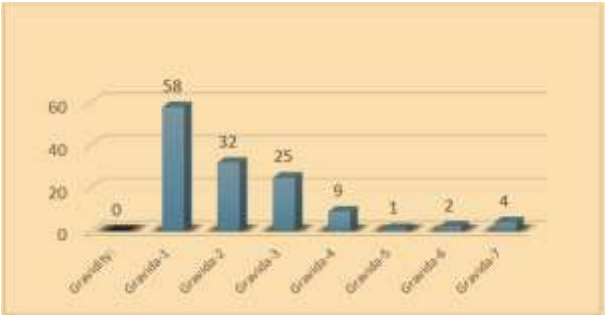

Fig-2: Gravidity: 94(47%) pregnant women with PIH were primigravidae, 56(28%) were 2nd gravida, 34(17%) were 3rd gravida, 9(4.5%) 4th gravid, 3(1.5%) more than G-4

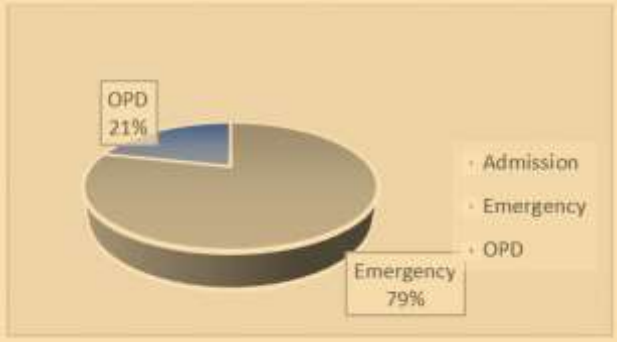

Fig-3: Admission (Emergency/O.P.D): Most of the pregnant women with PIH were admitted through emergency. 103 out of 131 (78.72%) and 28(21.37%) women admitted through O.P.D

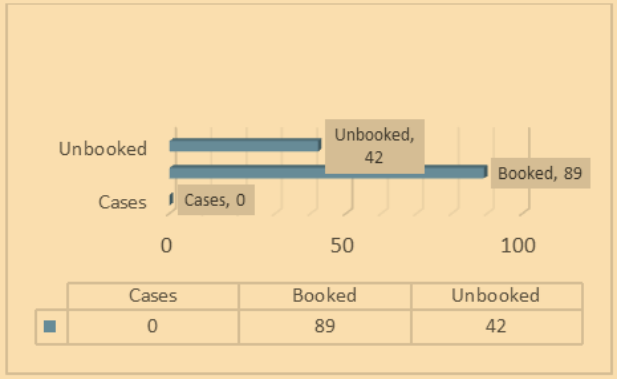

Fig-4: Booked or unbooked cases: 89 (67.93%) of the pregnant women with PIH came directly in emergency and antenatal clinic as unbooked cases and 42(32.06%) cases were booked or registered cases at various institutions

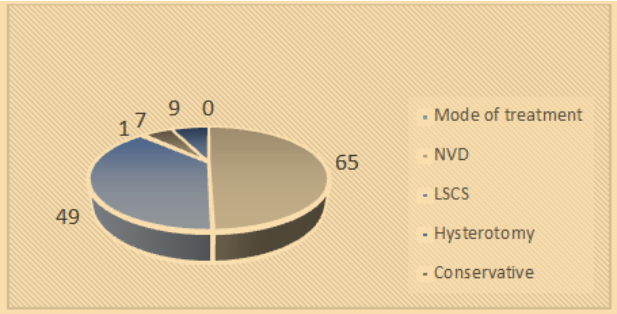

Fig-5: Mode of treatment: 65 (49.6%) of pregnant women with PIH were delivered by normal vaginal delivery, 49 (37.42%) of pregnant women with PIH underwent caesarean section for various causes, 1 pregnant patient with PIH underwent hysterectomy, 7 patients were treated conservatively and 9 patients did not report in the ward or left against medical advice

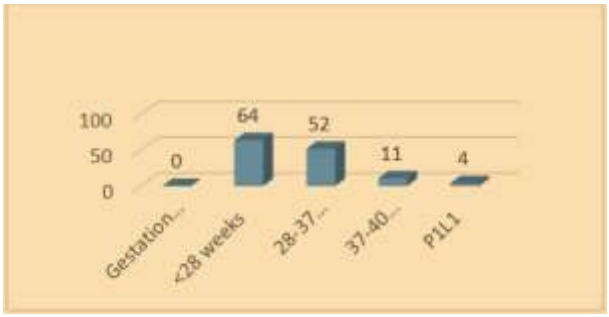

Fig-6: Gestation time: 64 (48.5%) pregnant women with PIH were having gestation time less than 28 weeks, 52(39.6%) of pregnant women with PIH were gestation time between 28-37 weeks, 11 (8.3%) of pregnant women with PIH were gestation time between 37-40 weeks, 4 (3.0%) of women with PIH were P1L1

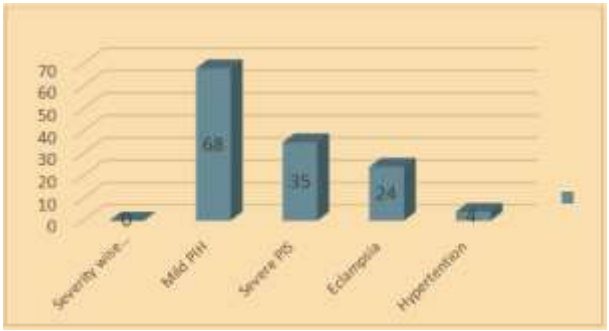

Fig-7: Severity wise Distribution: Out of 131 pregnant women admitted for PIH, 68(51.9%) were categorised as having mild PIH, 35(26.7%) were labelled as cases of severe PIH, 24(18.3%) women presented with eclampsia and 4(3.0%) had hypertension. Gestation: 54% of the women with mild PIH had gestation time between 29-40 weeks. 45% of the women with severe PIH had gestation time between 29-40 weeks and 21% of the women with eclampsia had gestation time between 29-40 weeks

Table-1.1

Gestation	Weeks	Mild pih(54)	Severe pih (45)	Eclampsia (21)	Ch. Hypertension (11)	Total	% age
	<28weeks	2(28.5%)	3(42.8%)	2(28.5%)	nil	7	5.34%
	29-37 weeks	31(43.66%)	27(38.02%)	6(8.45%)	7	71	54.19%
	38-40 weeks	21(39.62%)	15(28.30%)	13(24.52%)	4	53	40.45%

Maternal and foetal outcome: Out of 117 pregnant women with mild PIH, 8 (6.84%) of the patients had IUD and there was no maternal death, Out of 55 pregnant women with severe PIH, 15 (27.27%) of the patients had IUD and there was one (1.82%) maternal death, Out of 24 pregnant women with eclampsia, 7 (29.16%) of the patients had IUD and there was 3 (12.5%) maternal death.

Table-1.2

Maternal and foetal outcome	Weeks	Mild pih(51)	Severe pih (45)	Eclampsia (24)	Ch. Hypertension (4)	Total	% age
	IUD	8(15.68%)	15(33.33%)	7(29.16%)	Nil	30	15%
	MAT. DEATH	NIL (0 %)	1(2.22%)	3(12.5%)	Nil	4	2%

Table-2: Distribution of the study patients according to maternal risk factors (n=131)

Maternal risk factors	Case(n=50)	Control(n=50)	OR	95% CI		p Value
	n	n		Lower	Upper	
Age 35> years	15	4	4.93	1.36	19.43	0.005^{s}
BMI(>24.9kg/m^2)	38	25	3.17	1.25	8.16	0.007^{s}
History of preeclampsia	13	3	5.50	1.32	26.44	0.006^{s}
Diabetes	6	4	1.57	0.36	7.19	0.504^{ns}
Low socioeconomic status	24	17	1.79	0.74	4.35	0.154^{ns}

s=significant, ns=not significant, p value reached from chi square test

[Table 2] showing different risk factors where regarding age 35> years, 15 patients found in case group and 4 patients in control group. Significant (p<0.05) difference was found between two groups. Patients had 4.93 times more likely to developed pree-clampsia. Regarding BMI, 38 patients had BMI more than 25 in case group and 25 patients in control group. Significant (p<0.05) difference was found between two groups. Patients had 3.17 times more likely to developed preeclampsia. Regarding history of precelampsia, 13 patients found in case group and 3 patients in control group. Significant (p<0.05) difference was found be-tween two groups. Patients had 5.50 times more likely to devel-oped preeclampsia. Other results are mentioned in the table.

Table-3: Distribution of the study patients according to maternal outcome (n=131)

Maternal Outcome	Case(n=50)		Control(n=50)		p Value
	n	%	n	%	0.001^{s}
No complication	28	56.0	44	88.0	
Complication	22	44.0	6	12.0	
Eclampsia	2	4.0	0	0.0	
Abruptio placenta	3	6.0	0	0.0	
PPH	7	14.0	5	10.0	
Pulmonary oedema	3	6.0	1	2.0	
HELLP syndrome	2	4.0	0	0.0	
Ascites	4	8.0	0	0.0	
Oliguria	1	2.0	0	0.0	

s= significant, p value reached from chi-square test.

[Table 3] shows maternal complications of the patients. It was observed that 22(44.0%) and 6(12.0%) had complication in case and control group respectively. The difference was statistically significant (p<0.05) between two groups. Among the case group, 2(4.0%) patients developed eclampsia, 3(6.0%) patients developed abruptio placenta, 2(4.0%) patients developed HELLP syndrome, 4(8%) patients developed ascites and 1(2.0) patient developed oliguria. But none of the control group had developed these types of complications. The difference was statistically significant (p<0.05) between two groups. In case group 7(14.0%) patients developed PPH and 3(6.0%) developed pulmonary oedema. In control group 5(10.0%) developed PPH and 1(2.0%) developed pulmonary oedema, these difference were not so significant between two groups.

Table-4: Distribution of the study patients according to perinatal outcome (n=131)

Perinatal outcome	Case(n=50)		Control(n=50)		p value
Live birth	N%		N%		
Take home alive	35	70.0	50	100.0	0.001s
Early neonatal death	5	10.0	0	0.0	0.028s
Still birth	10	20.0	0	0.0	0.001s

s=significant, ns=not significant, p value reached from chi-square test

The above [Table 4] shows the perinatal outcome of the study patients. Take baby in home safely in 35(70.0%) in cases group and 50(100.0%) in control group. Early neonatal death was found in 5(10.0%) in case group and not found in control group. Still birth was 10(20.0%) case group and not found control group. Statistically significant (p<0.05) difference was between two groups.

Table-5: Distribution of the study patients according to APGAR score (n=100)

APGAR Score	Case(n=81)	Control(n=50)	P-Value
At 1 minute	N%	N%	0.001s
0	21-20.0%	0-0.0%	
0-4	9-8.0%	0-0.0%	
4-6	38-56.0%	12-24.0%	
7-10	13-16.0%	38-76.0%	
At 5 minute			0.002s
4-6	12-30.0%	3-6.0%	
7-10	28-70.0%	47-94.0%	

*10 babies drop out due to still birth, s= significant, p value reached from unpaired t-test.

The above [Table 5] shows the APGAR score at 1 min and 5 min of the delivered babies of the study patients. Most 56.0% of the neonates had APGAR score 4-6 at 1 minute in case group and 12(24.0%) in control group. Twelve (30.0%) babies had APGAR score 4-6 at 5 minutes in case group and 3(6.0%)%) babies had APGAR score 4-6 at 5 minutes in control group. There was statistically significant (p<0.05) difference was found between two groups.

V. DISCUSSION

This case control study was carried out with an aim to find out the demographic characteristics of patient with severe preeclampsia and of control group to evaluate the maternal risk factors of severe preeclampsia as well as to assess the foetomaternal outcomes associated with severe preeclampsia and normal pregnant patients. In this present study it was observed that majority (40.0%) of the cases were in age group 20-29 years and 50.0% in control group. Maternal age 35> years was found 15 (30%) in case group, and 4 (8%) in control group. The mean age was found 25.8±5.26 years with range from 17 to 38 years and 24.15±3.69 years with range from 18 to 35 years in case and control group respectively. The mean age difference was not statistically significant (p>0.05) between two groups. Similarly, Roudsari et al. showed the mean maternal age was 28.2±6.6 years in severe preeclampsia and 26.3±5.2 years in controls, which is closely resembled with the present study [11]. Maternal age 35 years was found by the authors was 28.6% in cases and 21.2% in controls. Amorim et al. reported that a significantly increased average age between cases 30.6 years versus 23.7 years old of the control group, which is higher with the current study [12]. Most of the patients were admitted through emergency as compared to admission through OPD. Gravidity: 94(47%) pregnant women with PIH were primigravidae, 56(28%) were 2nd gravida, 34(17%) were 3rdgravida, 9(4.5%) 4th gravid, 3(1.5%) more than G-4. This study was out of 131 pregnant women with PIH, 51 (38.93%) women were between 21-25 yrs, 45 (34.35%) were between 26-30 yrs, 24(18.32%) were less than 20 yrs, 11(8.39%). Among the 81 cases and 50 controls regarding different risk factors age 20-35 Yrs. BMI, history of precelampsia, were found significant (p<0.05) between two groups. Among the case group, patients developed eclampsia 2(4.0%) abruptio placenta 3(6.0%) HELLP syndrome 2(4.0%) ascites 4(8%) and oliguria1 (2.0). 64 (48.5%) pregnant women with PIH were having gestation time less than 28 weeks, 52(39.6%) of pregnant women with PIH were gestation time between 28-37 weeks, 11 (8.3%) of pregnant women with PIH were gestation time between 37-40 weeks, 4 (3.0%) of women with PIH were P1L1. Most 56.0% of the neonates had APGAR score 4-6 at 1 minute in case group and 12(24.0%) in control group. Showing different risk factors where regarding age 35> years, 15 patients found in case group and 4 patients in control group. Significant (p<0.05) difference was found between two groups. Patients had 4.93 times more likely to developed

preeclampsia. Take baby in home safely in 35(70.0%) in cases group and 50(100.0%) in control group. Early neonatal death was found in 5(10.0%) in case group and not found in control group. Still birth was 10(20.0%) case group and not found control group. Statistically significant ($p<0.05$) difference was between two groups. There was found to be that still eclampsia and severe PIH contribute significantly to foetal and maternal morbidity and mortality. Admission (Emergency/O.P.D): Most of the pregnant women with PIH were admitted through emergency i.e. 103 out of 131 (78.72%) and 28(21.37%) women admitted through O.P.D. Booked or unbooked cases: 89 (67.93%) of the pregnant women with PIH came directly in emergency and antenatal clinic as unbooked cases and 42(32.06%) cases were booked or registered cases at various institutions. Maternal and foetal outcome: Out of 117 pregnant women with mild PIH, 8 (6.84%) of the patients had IUD and there was no maternal death, Out of 55 pregnant women with severe PIH, 15 (27.27%) of the patients had IUD and there was one (1.82%) maternal death, Out of 24 pregnant women with eclampsia, 7 (29.16%) of the patients had IUD and there was 3 (12.5%) maternal death. The above table 4 shows the perinatal outcome of the study patients. Take baby in home safely in 35(70.0%) in cases group and 50(100.0%) in control group. Early neonatal death was found in 5(10.0%) in case group and not found in control group. Still birth was 10(20.0%) case group and not found control group. Statistically significant ($p<0.05$) difference was between two groups. It could be due to geographical variations, racial and ethnic differences, genetic causes and different lifestyle had significant impact to developed severe preeclampsia in their country. Many investigators Lee et al. Marviel et al. reported that obese women, prepregnancy body mass index of more than 24.2kg/m^2 are risk factors for development of severe preeclampsia [13, 14]. In this series it was observed that majority 38(76%) patients had BMI >25 in case group. The mean BMI was 31.4±7.5 kg/m^2 and 28.7±6.0 kg/m^2 in case and in control group respectively. The mean BMI was significantly ($p<0.05$) higher in case group. Manna done a study in BSMMU and found obese 35.6% and 22.2% in case and control group respectively, which is comparable with the current study [15]. The risk factors for developing preeclampsia are primigravid, age < 20 yrs or > 35 yrs, multiple gestations, family history of preeclampsia, and a prior history of preeclampsia, body mass index at or above 35 at first contact, preexisting hypertension or diabetes [13]. Regarding the maternal risk factors in this current study it was observed that, age >34 years significantly 4.93 times increased with 95% CI 1.36 – 19.43% to develop preeclampsia. BMI (>24.9 kg/m^2) had 3.17 times with 95% CI 1.25 – 8.16% more likely to developed preeclampsia. Previous history of precelampsia had 5.50 times with 95% CI 1.32 – 26.44% more likely to developed preeclampsia. However, diabetes, Low socioeconomic status was higher in case group but not statistically significant. Marviel et al. have been reported that age more than 34 years, obese women, and pregnancy body mass index of more than 24.2kg /m2 and urinary tract infection are significantly associated with preeclampsia [14]. The rate of neonatal complications is markedly increased in those who developed severe preeclampsia in second trimester whereas it is minimal in those with severe preeclampsia beyond 35 weeks gestation. Severe preeclampsia is also associated with increased risk of maternal mortality (0.2%) & increased risk of maternal morbidities (5.0%) such as convulsion, pulmonary oedema, acute renal failure, hepatic failure, disseminated intravascular coagulopathy & stroke. In 10% cases it leads to HELLP syndrome [15]. In another study Stone et al. (1994) obtained that history of preeclampsia OR=11.52 with 95% CI 5.09 - 26.09%, BMI 32.3 kg/m^2 OR=4.26 with 95% CI 2.06 - 8.81%. The above findings are comparable with the current study. In this present study it was observed that 44.0% of cases and 12.0% of control had complication, which was significantly ($p<0.05$) higher in case group. Among the case group, 2(4.0%) patients developed eclampsia, 3(6.0%) patients developed abruptio placenta, 2(4.0%) patients developed HELLP syndrome, 4(8%) patients developed ascites and 1(2.0) patient developed oliguria. But none of the control group had developed these types of complications. The difference was statistically significant ($p<0.05$) between two groups. In case group 7(14.0%) patients developed PPH and 3(6.0%) developed pulmonary oedema. Similarly, Stone et al. [34] found low (< 2500g) birth weight was 62.9% in cases and 6.9% in controls, which was also significantly ($p<0.05$) higher in case group, thus sup-port the present study. Similarly, Mansour et al. [36] mentioned that the babies born to severe preeclampsia group had lower APGAR score at 1 and 5 minutes, than control group ($p<0.001$), which support the current study. In this present study it was observed that 80.0% newborn needed admission in neonatal care unit in case group, due to prematurity, severe perinatal asphyxia and their complications. Eight (20.0%) newborn needed admission in neonatal care unit in control group, due to mild perinatal asphyxia and prematurity. The difference was significantly ($p<0.05$) higher in case groups. A study done by Das [32] at BSMMU, Bangladesh found that 79.2% newborn needed admission in neonatal care unit in case group and 16.0% in control group. This study finding in consistent with the current study. In this current study it was observed that take baby in home safely

was 70.0% in case group and 100.0% in control group. Early neonatal death was found in five (10.0%) of the case group due to severe perinatal asphyxia, prematurity and their complications. Ten (20.0%) babies were found still birth in case group and 5(10.0%) babies were found neonatal death in case group, but there were not still birth and neonatal death in control group. The difference was statistically significant ($p<0.05$) between two groups. In this study 80.0% babies were admitted in neonatal care unit in case group and 24.0% of babies were admitted in neonatal care unit in control group. Stone et al. [33] reported in their study that neonatal death was found 4.3% in cases and 0.3% in controls. The difference was statistically significant ($p<0.05$) between two groups in this study. Buchbinder et al. and Hauth et al. reported that admission to NI-CU varied from 38.0 to 43.0% babies born to severe preeclampsia [34]. In another study Buchbinder et al. showed admission to NI-CU was 38.1% in severe preeclampsia [36, 37]. In this study, there was no maternal death [37].

VI CONCLUSION

Preeclampsia is a leading cause of both fetal and maternal morbidity and mortality in the developing countries. Regular ante-natal checkup to detect the rapid wt gain or rising BP is very important to prevent preeclampsia. Beside this low dose aspir-in & low molecular wt heparine is useful in high risk women with thrombophilia. Calcium supplement & other antioxident like Vit E & C & balanced diet rich in protein may also reduce the risk of preeclampsia. There is no way known to prevent preeclampsia and eclampsia. However, the outcome can be improved with prompt recognition and management, so it is important for pregnant women to have routine health screenings. Most women with mild preeclampsia have good pregnancy outcomes.

REFERENCE

1. Fernando, A. (2007). Practical Guide to high-risk pregnancy & delivery, 417.
2. Lew, M., & Klonis, E. (2003). Emergency management of eclampsia and severe pre-eclampsia. Emergency Medicine, 15(4), 361-368.
3. Liskin, L. S. (1992). Maternal morbidity in developing countries: a review and comments. International Journal of Gynecology & Obstetrics, 37(2), 77-87.
4. Sibai, B. M. (2003). Diagnosis and management of gestational hypertension and preeclampsia. Obstetrics & Gynecology, 102(1), 181-192.
5. Fernando, A. (2007). Practical Guide to high-risk pregnancy & delivery. 3rd edition, 417.
6. Khan, K. S., Wojdyla, D., Say, L., Gülmezoglu, A. M., & Van Look, P. F. (2006). WHO analysis of causes of maternal death: a systematic review. The lancet, 367(9516), 1066-1074.
7. Kaaja, R. (2008). Predictors and risk factors of pre-eclampsia. Minerva ginecologica, 60(5), 421-429.
8. Broughton, P.F. (2001). Risk factors for pre - eclampsia. N Engl J Med; 344:925-926.
9. Cerón-Mireles, P., Harlow, S. D., Sanchez-Carrillo, C. I., & Nunez, R. M. (2001). Risk factors for pre-eclampsia/eclampsia among working women in Mexico City. Paediatric and perinatal epidemiology, 15(1), 40-46.
10. Ødegård, R. A., Vatten, L. J., Nilsen, S. T., Salvesen, K. Å., & Austgulen, R. (2000). Risk factors and clinical manifestations of pre-eclampsia. BJOG: An International Journal of Obstetrics & Gynaecology, 107(11), 1410-1416.
11. VAHID, R. F., Ayati, S., AYAT, E. H., ESMAEILI, H. E., Hasanzadeh, M., Shahabian, M., & POURALI, L. (2009). Comparison of maternal serum tumor necrosis factor-alpha (tnf-β) in severe and mild preeclampsia versus normal pregnancy.
12. Amorim, M. M. R. D., Santos, L. C., Porto, A. M. F., & Martins, L. K. D. (2001). Risk factors for maternal death in patients with severe preeclampsia and eclampsia. Revista Brasileira de Saúde Materno Infantil, 1(3), 237-247.
13. Lee, C. J., Hsieh, T. T., Chiu, T. H., Chen, K. C., Lo, L. M., & Hung, T. H. (2000). Risk factors for pre-eclampsia in an Asian population. International Journal of Gynecology & Obstetrics, 70(3), 327-333.
14. Merviel, P., Touzart, L., Deslandes, V., Delmas, M., Coicaud, M., & Gondry, J. (2008). Risk factors of preeclampsia in single pregnancy. Journal de gynecologie, obstetrique et biologie de la reproduction, 37(5), 477-482.
15. Manna, F. N., Khanam, N. N., Chowdhury, K. A., Das, S. N., Kabir, M. A., Zubyra, S. J., ... & Akhter, K. (2015). Study on Association of Maternal Serum Triglyceride with Pre-eclampsia. Mymensingh medical journal: MMJ, 24(3), 578-584.
16. Dittakarn Boriboonhirunsarn, M., Talungjit, P., & Sunsaneevithayakul, P. (2006). Adverse pregnancy outcomes in gestational diabetes mellitus. J Med Assoc Thai, 89(4), S23-8.
17. Buchbinder, A., Sibai, B. M., Caritis, S., MacPherson, C., Hauth, J., Lindheimer, M. D., ... & Thurnau, G. (2002). Adverse perinatal outcomes are significantly higher in severe gestational hypertension than in mild preeclampsia. American journal of obstetrics and gynecology, 186(1), 66-71.
18. Hauth, J. C., Ewell, M. G., Levine, R. J., Esterlitz, J. R., Sibai, B., Curet, L. B., ... & Calcium for Preeclampsia Prevention Study Group. (2000). Pregnancy outcomes in healthy nulliparas who

developed hypertension. Obstetrics & Gynecology, 95(1), 24-28.
19. Singhal, S.R., Deepika, A., Nanda, S. (2009). Maternal and perinatal outcome in severe Preeclampsia and eclampsia. JSAFOG, 1(3):25-8.
20. Walker, J.J. (2000). Preeclampsia. Lancet, 356:1260-5.
21. Robson, S.C. Hypertension and renal disease in pregnancy. In: Dr. Keith Edmond, editors. Dewhurdt's textbook of Obstetrics and
22. Gynaecology. (1986). 4th edition. Orient Longman ltd., London, 4:203-9.
23. Raddi, S.A., Nayak, B.S., Ratna, P., (2009). Stress, Coping Strategies, Quality of life and Livid Experiences of Women with Pregnancy induced Hypertension. JSAFOG, 1(1):65-8.
24. "40". Williams's obstetrics (24thed.). (2014). McGraw-Hill Professional.
25. "Hypertension in pregnancy. (2013). Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy." (PDF). Obstet Gynecol, 122(5): 1122–31.
26. World Health Organization. (2014). WHO recommendations for prevention and treatment of pre-eclampsia and eclampsia: implications and actions (No. WHO/RHR/14.17). World Health Organization.
27. Arulkumaran, N., & Lightstone, L. (2013). Severe pre-eclampsia and hypertensive crises. Best Practice & Research Clinical Obstetrics & Gynaecology, 27(6), 877-884.
28. GBD 2013 Mortality and Causes of Death, Collaborators. (17 December 2014). "Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013." Lancet 385:11771.
29. Garcia, M., Mulvagh, S. L., Bairey Merz, C. N., Buring, J. E., & Manson, J. E. (2016). Cardiovascular disease in women: clinical perspectives. Circulation research, 118(8), 1273-1293.
30. Abbasi, J. (2018). JAMA 2018; October 17:[Epub ahead of print].
31. Zoet, G.A., Benschop, L., Boersma, E. (2018). Circulation, 137:877-9.
32. Stuart, J.J., Tanz, L.J., Missmer, S.A. (2018). Ann Intern Med, 169:224-32.
33. Riise, H.K., Sulo, G., Tell, G.S. (2017). J Am Heart Assoc, 6.
34. Vaught, A.J., Kovell, L.C., Szymanski, L.M. (2018). J Am Coll Cardiol, 72:1-11.
35. American College of Obstetricians and Gynecologists. (2013). Task Force on Hypertension in Pregnancy. Obstet Gynecol, 122:1122-31.
36. Ghanem, F.A., Movahed, A. (2008). Cardiovasc Ther, 26:38-49.
37. Behrens, I., Basit, S., Melbye, M. (2017). BMJ, 358:j3078