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A Cross-Sectional Study on Current Practice of the Management of Pregnancy Induced Hypertension and Its Maternal and Foetal Complications and Outcome in the Western Part of the State of Libya

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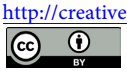

Abstract

Background: Maternal and perinatal mortality and morbidity are mainly affected by hypertension during pregnancy (pre-eclampsia). Haemorrhage and cerebral bleeding are the primary and significant complications of pre-eclampsia. Abruption of the placenta and renal failure are among the major complications caused by this disease. The perinatal complications include Intrauterine Growth Restriction (IUGR), Intrauterine Foetal Death (IUFD), neonatal death, and prematurity. **Objective:** The purpose of the present study was to examine the incidence of Pregnancy-Induced Hypertension (PIH), pre-eclampsia, the management of PIH, including the early diagnosis, the pharmacological drug management used, and the prevalence of maternal and perinatal complications. **Methods and Patients:** This cross-sectional study took place in Aljalaa maternity hospital, which represented the west of the state of Libya in the period from 1st January 2012 to 31st December 2012, with patients who were diagnosed, managed, and terminated according to protocol management of the hospital. **Results:** In the present study, from the total deliveries in Aljalaa Maternity hospital, the incidence of PIH is 8.4% and pre-eclampsia is 5.1%. No maternal mortality, CNS haemorrhaged, hepatic failure, or renal failure. The reported maternal complications included: Eclampsia, HELLP syndrome (Haemolysis, Elevated Liver enzymes and Low platelets), abruption placenta, and pulmonary oedema see in eighteen percent, six percent, nine percent, four percent and less than one percent, respectively. The foetal complications were preterm babies and Intrauterine Growth Restriction (IUGR) in nineteen percent, Low Birth Weight (LBW) in twenty-five percent, Intrauterine Foetal Death (IUFD) in five

percent, neonatal death in two percent and, perinatal death in six percent. In the PIH patients, the presence of associated symptoms such as headache, blurred vision, irritability, and the presence of signs such as severe hypertension, generalized oedema, exaggerated reflexes, and abnormal investigations (protein urea, high uric acid, abnormal LFT, haemoconcentration, and low pits) increased the risk of maternal and foetal complications. **Conclusion:** To decrease mother and newborn mortality and morbidity in PIH patients, a rigorous management protocol is required to implement evidence-based guidelines.

Keywords

Preeclampsia, Proteinuric, Pregnancy Induced Hypertension

1. Introduction

Hypertension experienced by pregnant women is a leading concern for obstetricians. In some instances, this critical condition becomes associated with proteinuria and, ultimately, pre-eclampsia. Pre-eclampsia affects the morbidity and death rate of both mothers and babies. The risk of severe complications such as abruption placenta, cerebrovascular accident, organ failure, and disseminated intravascular coagulation increases in pregnant women with hypertension and the fetus is at risk for intrauterine growth retardation, prematurity, and intrauterine death [1]. Hypertension During Pregnancy (HDP) results from complicated factors more than increased blood pressure rates. The persistent diastolic pressure of 95mmHg and above is the defining level for hypertension during pregnancy. The systolic pressure is usually not considered, although ongoing systolic pressure exceeding 160 mmHg causes concern (World Health Organization). Severe hypertension is present when diastolic levels of over 110 mmHg are reached [2].

Pre-eclampsia is a disease of theories since the leading causes that produce complications in this disease have not been determined yet. However, increasing research in this area makes researchers understand the underlying events. Preeclampsia is related to a disturbance in the myometrium due to trophoblast invasions [3]. Pre-eclampsia may be associated with other factors such as invasions in the trophoblast. Moreover, it is also found that pregnancies get affected by growth restriction of the fetus even when there is no presence of disease in mothers. The second half of pregnancy gets affected by poor perfusion and ischemia due to ruptured blood flow. This condition may lead to the creation of reactive oxygen in species. A state of oxidative stress exists once the normal endogenous antioxidants are overwhelmed [4].

Incidence: Hypertension during pregnancy can be categorized into three types; non-proteinuric hypertension, chronic hypertension, and pre-eclampsia. Preeclampsia has been estimated to be responsible for the death of one in every ten women each year in the United Kingdom. The World Health Organization (WHO) report has estimated the death of around 60,000 women across the globe

each year [5]. After 20 weeks of gestation, pregnancy-induced hypertension commonly occurs in one in five women. However, only a slim percentage of the population gets this disease, leading to high morbidity. Contrarily, severe and adverse effects create concern for intense protection and management for many women [6].

Delivery is the primary cure and definite management for pre-eclampsia. However, it is associated with 15% of premature births and ultimately related to prematurely born babies' future health and well-being [7]. The U.K. figures of pre-eclampsia revealed its occurrence in one of every twenty women as an average. Its incidence was around 2% in the primigravida population in Ireland [8]. Some forms of the disorder complicate 7% to 10% of all pregnancies [8]. Pre-eclampsia is more common in multifetal gestations.

Moreover, its incidence was up to 30% of pregnancies in diabetic patients and 20% of gestations in chronic hypertensive. However, two-thirds of all cases occur in otherwise healthy, nulliparous patients. It is a prominent factor behind maternal and perinatal morbidity and is associated with higher mortality rates [8]. Copious studies conducted in the United States have shown that the prevalence of pre-eclampsia is nearer 10%, possibly related to the high-risk population studied [8].

Clinical features of pre-eclampsia: Usually it includes some, but not all of the clinical features and pathological discussed below:

Hypertension is the main feature of pre-eclampsia, and pre-eclampsia is a raised resistance in systemic as the colour area and a reduced cardiac output antenatally but an increased cardiac output during labour [9]. The World Health Organization recommends that the diastolic blood pressure should be recorded at the level of the fourth Korotkoff sound (muffling) and not the fifth (disappearance) (World Health Organization, 1987). Yet, in a survey of 91 obstetricians and midwives in the U.K., 53 percent used the fifth sound [10]. Furthermore, a variety of cuff sizes should be available so that the length of the inflation bladder is at least 80 percent of the circumference around the arm. Errors are generally more significant when using too small a cuff than one too large [11].

Proteinuria has been referred to as the accumulation of urinary protein on two or more occasions at least four hours apart in concentrations greater than 0.3 g/l in a 24-hour urine collection or a concentration of greater than 1 g/l in a random urine collection [2]. The proteinuria is essentially albuminuria and reflects the severity of the disease in the absence of pre-existing renal pathology. In 1973 Naeye and Friedman reported that the fetal mortality rate was 17.2 per 1000 for normotensive pregnancies and 37.9 per 1000 for pregnancies complicated by proteinuric hypertension [12]. Seventy percent of the deaths were related to placental infarction, severe IUGR, or placental abruption [10]. The finding of proteinuria during pregnancy may be due to unsuspected renal disease and not due to pre-eclampsia, such as coexistent chronic glomerulonephritis and polycystic kidneys. If renal impairment persists beyond the pregnancy, renal biopsy may be indi-

cated. When pre-eclampsia manifests as hypertension, no long-term detectable renal impairment seems to occur, providing that no co-existing renal pathology is present, except if renal tubular necrosis of acute renal injury has occurred [10] [11].

Oedema is a common non-specific clinical sign during pregnancy. The only reduction in serum albumin levels during normal pregnancy relates to haemodilution, while the total circulating albumin levels remain unchanged. As per Starling's law, tissue fluids would increase, allowing oedema to occur. Oedema usually reflects a reduction in protein rather than fluid volume [2]. Studd *et al.* reported a mean decrease of 34 percent decrease in circulating levels of albumin in 10 patients with severe pre-eclampsia compared with non-pregnant levels [13].

Placental pathology: Impaired trophoblastic infiltration of the uterine arterial walls during placentation is now considered to be pivotal to the onset of the syndrome [10] [14]. The conversion of spiral arteries into uteroplacental arteries appears to depend upon trophoblastic invasion of the vascular walls. Should the process be impaired, the pathological process that follows may be pre-eclampsia, intrauterine growth restriction or both [15] [16]. As seen on the histological examination of placental bed biopsies, these changes may include fibrin deposition, arteriosclerosis, endothelial vacuolation, and thrombosis [15] [17]. Since these changes will have become apparent by the mid-trimester, examining the placental bed using Doppler ultrasound to assess the uterine (arcuate) vessels may predict those women who may develop the syndrome of pre-eclampsia before any clinical features become apparent [18].

Clothing system: The changes within the clotting system during early pregnancy are well documented. Bonnar *et al.* (1969) reported that the haemostatic mechanism changes towards an increased capacity to form fibrin and a diminished ability to lyse it, as demonstrated by a 200 percent increase in plasminogen and fibrinogen levels, allowing for plasma expansion during pregnancy. It is not clear why these changes occur [19]. Still, Pritchard *et al.* (1976) [20] believe that at sites of vascular endothelial damage they relate to platelet adherence because of vasospasm rather than thromboplastin release from the placenta, whilst Redman *et al.* (1989) [15], suggests that they may be related to disorders of increased platelets consumption in pre-eclampsia. However, there is no good evidence which explains these changes. Haemolysis (microangiopathic haemolytic anaemia) may also occur in pre-eclampsia, and the syndrome of haemolysis, elevated liver enzymes and low platelets count (HLEPP syndrome) is now well recognized, but rare, and cholecystitis is considered to be an indication for inducing delivery [21] [22].

Liver pathology: Jaundice may occur, especially concerning eclampsia due to acute fatty liver, intravesical coagulopathy, Gram-negative septicaemia, or coincidental viral hepatitis [21] [22]. The characteristic hepatic lesion in pre-eclampsia is haemorrhage and ischemia around the portal tract and under the liver capsule, perhaps related to the fibrin deposits in the liver vasculature [23].

Central nervous system: The incidence of eclampsia would appear to lie be-

tween 1390 pregnancies and 1 in 4250 pregnancies [10] [24]. The perinatal mortality rate in eclampsia seems to lie between 86 and 213 per 1000 [26]. There is also appreciable maternal mortality. This has been reported to be in the region of 2 percent [26] [27]. It is essential to observe that not all eclampsia occurs before delivery. Some 46 per cent of the patients had their first convulsion antenatally, 17 percent during labour, and 37 percent postnatally [25]. Eclampsia has been likened to hypertensive encephalopathy [10] but with superimposed features, such as cerebral oedema, haemorrhage, and infarction. The cause of the cerebral dysfunction is unclear. Still, it is associated with vasoconstriction, which some believe to be a cause [28] and others believe to be a protective mechanism against bleeding [10]. Traditionally, the eye is considered part of the central nervous system and ocular complications of eclampsia occur. Narrowing of the retinal arterioles due to vasoconstriction occurs to some degree in up to 70 percent of hypertensive pregnant women, and this vasoconstriction reverses when the blood pressure subsides [29]. However, "cottonwool" exudates and retinal oedema may occur in 1 percent of women with pre-eclampsia, and rarely these may lead to retinal detachment in 1 in 700 such women [30] [31]. Spontaneous retinal re-attachment follows, but some degree of visual impairment may persist. Transient cortical blindness has also been reported in up to 5 percent of women who have had eclamptic fits [25].

The foetus: Acute and/or chronic changes in the uteroplacental circulation can affect the foetus. The most typical cause of perinatal death is abruption placenta [25]. Preeclampsia reduces birth weight, as illustrated in **Table 1** [32]. Moor and Redman (1983) [33] matched 24 women with pre-eclampsia, with 48 controls matched for age and parity who were randomly selected. Some 8 per cent of rules gave birth to a newborn whose birth height was below the 10^{th} centile compared with 82 percent of the patients with pre-eclampsia. Intrauterine growth restriction occurred in 14% of the babies born to mothers with pre-eclampsia. Still, the figures rose to 33% percent of the babies born to mothers with pre-eclampsia superimposed chronic hypertension. Other authors confirm this association [34] [35].

Table 1. The influence of parity.

Pregnancy	No. of pregnancies	% Preeclampsia hypertension	% With mild pre-eclampsia
First	11,534	6.1	27.5
Second	9730	1.9	17.7
Third	5002	1.2	15.7
Fourth	2155	0.6	15.9
5 - 15	1430	1.1	18.0

From Campbell *et al.* 1985 [23].

Predisposing factors to preeclampsia: These appear to affect the incidence of pre-eclampsia in each pregnancy; this includes **parity** as the propensity of both mild and severe pre-eclampsia is high in subsequent pregnancies [36]. The incidences of proteinuric hypertension and mild pre-eclampsia in the higher parity group are demonstrated in (Table 1) [18]. Should the first pregnancy have ended in spontaneous abortion, the incidence of proteinuric hypertension (4.8 percent) was more similar to that in the first pregnancy (6.1 percent) than a second pregnancy (1.9 percent) [18].

Maternal age: A graph of the incidence of pre-eclampsia against maternal age-eclampsia is generally considered a J-shaped curve. It is uncertain whether or not the incidence opf pre-eclampsia is the very young mothers [37] [38]. The World Health Organization Study Group (1987) suggested that the apparent increased incidence in teenage girls may relate to the increased incidence of concealed pregnancies in this group. However, a recent survey of over one and a half million pregnancies in the USA did confirm the traditional J-shaped curve with a relative risk of 1.9 for the development of pre-eclampsia in women aged 17 years or less compared with those aged 20 - 24 years [39]. However, The British Perinatal Mortality Survey [40] demonstrated a notable increase in the incidence of severe pre-eclampsia in women aged more than 35 years, regardless of their parity (Table 2).

Previous pre-eclampsia also has been found to affect subsequent pregnancies. It has been stated that proteinuric hypertension in the first pregnancy increased in the following pregnancy [18].

Family history of pre-eclampsia is associated with increased incidence of both mild and severe pre-eclampsia in the female relative of parents who have had this disease. Chelsey and cooper (1986) [41] reported that among the women who were suffering from pre-eclampsia during the first viable pregnancy, 25 percent of their daughters had pre-eclampsia, as did 20 percent of their granddaughters, but only 6 percent of daughters-in-law [42]. It may be that maternal genetic susceptibility is influenced by antigen sharing between maternal and genotype [42].

Table 2. The influence of maternal age.

Parity	Singleton pregnancies				
	Maternal age	Number of deliveries	Mild pre-eclampsia (%)	Moderate pre-eclampsia (%)	Severe pre-eclampsia (%)
All	All	16981	17.4	4.0	6.1
Primigravidae	<25	3619	18.4	4.4	8.5
	25 - 34	2412	21.4	5.2	7.9
	>35	249	30.9	6.8	15.3
Multi-gravidae	<25	2288	12.4	2.0	3.5
	25 - 24	6459	15.9	3.7	3.7
	>35	1954	19.4	4.7	6.5

Maternal weight: There is conflicting evidence concerning the influence of maternal obesity on the prevalence of pre-eclampsia an Edward *et al.* (1978) [43] reported that 23 percent of obese women had some form of hypertension during pregnancy compared with 10 percent of non-obese controls matched for age and parity. However, Chesley [44] reported 242 women with eclampsia and found that the condition has a slight preference for underweight women; but was unable to find any association between pre-eclampsia and obesity. Waters [45] reported the weight gain of 21 - 45 lb was correlated with the occurrence of pre-eclampsia 3.5 percent weight gain of 26 - 30 lb with an occurrence of pre-eclampsia of 5. Pre-eclampsia 31 - 35 lb with an incidence of pre-eclampsia whilst the mean weight gain for the group (23.2 lb) was associated with an overall incidence of pre-eclampsia. Therefore, almost 90 percent of those who gained eight "excessively" did not develop pre-eclampsia.

Other obstetric conditions: There is a relation between a hydatidiform mole and pre-eclampsia [46]. Brudnell [47], reporting the United Kingdom Diabetic Pregnancy Survey, revealed that pre-eclampsia and established diabetics' overall incidence rate was 14.4 percent. Twinning is correlated with a more significant occurrence of pre-eclampsia [48], combined the data from five research studies on the association between the incidence of pre-eclampsia and multiple pregnancies are 26 percent. It can also be seen that the prevalence of pre-eclampsia in twin pregnancies especially in twins of the opposite sex than the same sex, and higher in twin pregnancies which were presumed to be dizygous (29.6 percent) compared with those that were assumed to be monogynous (15.6 percent).

In twin pregnancies, the rate of pre-eclampsia is higher than singleton and the overall rate is around 9.5%, about two- to three-fold increased risk compared to singleton [2]. Furthermore, pre-eclampsia in twins has been reported to occur at earlier gestational age and has a more severe form [3]. The reasons why twins developed pre-eclampsia more than singleton are still inconclusive. However, it has been proposed that the pathogenesis of pre-eclampsia among twin pregnancies may be due to a higher immunologic response and placental mass [4].

2. The Aim of Present Study

The study emphasised examining the protocol regime of the hospital aimed at the early detection, making the diagnosis and effective management of Pregnancy-Induced Hypertension (PIH) and pre-eclampsia. It also aimed to determine the incidence and prevalence of PIH patients in Aljalaa maternity hospital, which serves the west of the country and examine the prevalence of PIH maternal and foetal complications.

3. Patients and Methods

The current research was carried out through a cross-sectional research design between 1st January 2012 to the end of December 2012 on 688 patients diagnosed, managed. It terminated their pregnancy at Aljalaa maternity hospital using the management protocol in the hospital. The doctor performed a physical

exam and ordered laboratory tests. Data collection was done through a recorded review of the antenatal card. A specified questionnaire was done to collect the needed information from the antenatal and labour ward through the files of 660 PIH patients; this questionnaire was supported with an obstetric history related (signs, symptoms, clinical investigations, biophysical profile, drugs used, termination of pregnancy and outcome for mother and baby). A separate 28 cases of pregnant ladies who developed PIH with a gestational age starting from 32 weeks and received fractionated heparin (Daltaparin 7500 IU) twice a day for two weeks then once a day until termination of pregnancy, with the use of coagulation profile every third day. Then the biostatistics were done by using Excel and SPSS software to clarify the relationship between variables and explain the extent of the correlation between different signs, symptoms, and abnormal clinical investigations with maternal and perinatal outcomes. In the present study the protocol of management of termination of pregnancies in the PIH patients depended on the severity of the condition of those admitted to the hospital; mild PIH, severe PIH, eclampsia, or/and associated maternal, or/and foetal complications.

The gestational age of pregnant women at the time of the presentation was as follows:

1) If the patients were in mild PIH, without any maternal or foetal complication before 34 weeks gestation, expectant management, immediate antenatal care, investigation, anti-hypertensive drugs, dexamethasone, until 38weeks gestation;

2) If the patients were in severe PIH, without any maternal or foetal complication before 34 weeks gestation, and all the investigations are routine, the expectant management with close observational care in the hospital, with the research, anti-hypertensive drugs, and dexamethasone until 38 weeks gestation;

3) Suppose the patient in imminent eclampsia or/and severe PIH developed abnormal investigation or/and maternal or/and foetal complication. In that case, she needs to be managed in the ICU with prophylactic Mg. Sulphate, I/V anti-hypertensive drugs, dexamethasone and termination of the pregnancy at any gestational age within 24 - 48 hours;

4) If the patient were in an eclamptic fit, she would be managed in the ICU with the I/V Mg sulphate, I/V anti-hypertensive drugs, dexamethasone, and termination of the pregnancy at any gestational age within 4 - 8 hours.

4. Results

From 1^{s} January to 31st December 2012, 688 patients with pregnancy-induced hypertension (PIH) gave birth at Aljalaa maternity hospital; this is out of a total of 7906 deliveries during this period, with an incidence of (8.4%) with PIH.

Relationship between parity and severity of PIH: Table 3 and Figure 1 showed the relationship between the parity and severity of PIH in the cohort of patients delivered at Aljalaa Maternity hospital during the present study. The

Table 3. Relation between parity and severity of PIH.

Parity	Mild	Sever	TOTAL
Primagravida	96 (43.2%)	126 (56.8%)	222 (33.6%)
P1 - P4	216 (52.2%)	198 (47.8%)	414 (62.6%)
≥P5	12 (50%)	12 (50%)	24 (3.6%)

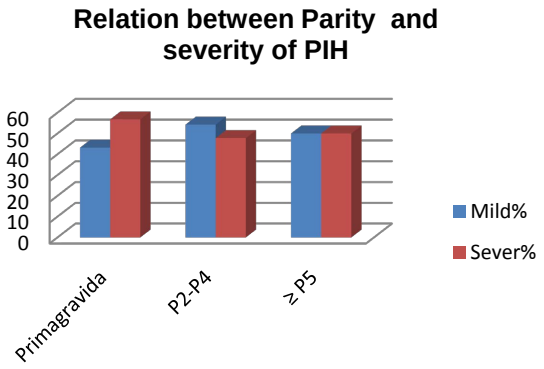

Figure 1. The relationship between parity and the severity of PIH.

study showed that PIH is common in P.G. an PGG2 (56.3%). The vast majority (96.3%) of the patient with PIH are those gravid 1 to gravida 4. The figures showed that the severe PIH is more prevalent in (P.G.) 5PG 8% than grand multipara (50%) than multiparty. 47.8%. However, the P-value for this study is equal to 0.09, which reflects that the tests are not significant.

Relationship between gestational age and severity of PIH: Table 4, Figure 2 showed the relationship between periods of gestational age and severity of PIH in the 660 patients with PIH, there were 28 patients with GA of 25 - 30 weeks, all of them (100%) had severe PIH. Another 95 patients out of 126 patients (75.4%) with GA of 30 - 35 weeks had severe PIH. Also, 125 patients out of 186 patients (67.2%) with GA of 35 - 37 weeks had severe PIH. There were 88 out of the rest of 320 patients had severe PIH. The P-value is equal to 0.00, which is highly significant.

There was no maternal mortality, CNS haemorrhage, hepatic failure, haemorrhages or renal failure. In the present study, the maternal complications that occurred were eclampsia (6.4%), HELLP syndrome (9.3%), abruption placenta (4.5%), and pulmonary oedema (0.5%). In addition, the fetal complications that occurred were: Preterm babies (19.1%), IUGR (19.1%), LBW (24.6%), IUFD (4.6%), and neonatal death (1.8%), *i.e.*, perinatal mortality is equal to (6.8%), (P value = 0.00). Therefore, the test is highly significant. The relationship between the maternal complications and the severity of clinical presentation of the patients with PIH indicated that Abruptio placenta occurred in severe eclampsia

Table 4. The relationship between gestational age and the severity of PIH.

GA	Mild PE	Sever PE	Total
25 - 30	0 (0%)	28 (100%)	28 (4.2%)
30 - 35	31 (24.6%)	95 (75.4%)	126 (19.1%)
35 - 37	61 (32.8%)	125 (67.2%)	186 (28.2%)
≥37 WK	232 (72.5%)	88 (27.5%)	320 (48.5%)

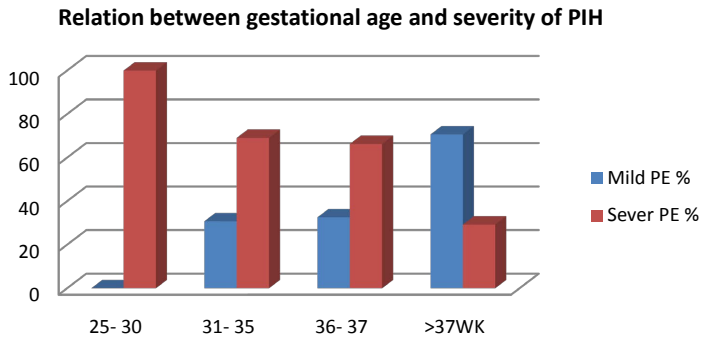

Figure 2. The relationship between gestational age and the severity of PIH.

(66.7% and 20%), respectively, with a total of 86.7%. HELLP syndrome occurred in severe hypertension (10%) with 91.7%. Pulmonary oedema reported in severe blood pressure was 100%. The maternal complications may occur even with mild hypertensive eclampsia (71.4%), abruption placenta (33.3%), and HELLP syndrome (8.3%). (P value = 0.00) making results highly significant.

The relationship between the foetal complications and severity of clinical presentation of the patients with PIH showed that IUGR occurred in severe hypertensive eclampsia (19.1%) with a total of 76.2%. Preterm babies occurred similarly in severe hypertension (14.3%) with a total of 61.9%. IUFD occurred in severe hypertension cases (20%), with a total of 88.7%. Neonatal death occurred in severe hypertension (41 eminent eclampsia (58.3%) with 100%. The foetal complication can occur even with mild blood pressure: IUGR (23.8%), preterm babies (38.1%), and IUFD (13.3%). The IUFD and neonatal death mostly occurred in those with severe hypertension. The incidence of proteinuric hypertension was (5.1%). When there was no protein urea (<+2), it was mostly associated with mild hypertensive patients (87.7%) and only (12.3%) of patients associated with severe hypertension. Contrarily, when there was proteinuric hypertension, indicating an increase in the risk of maternal complications, especially: Eminent eclampsia (93.3%), eclamptic fits (83.3%), HELLP syndrome (30%) and abruption placenta (100%). Proteinuric hypertension, leads to an increase the risk of foetal complications, especially: IUGR (91.3%), IUFD (60%), premature babies (65.1%), neo-

natal death (66.7%), and LBW (80.9%). Pathological CTG increases the risk of foetal complications, especially IUGR (100%), IUFD (100%), premature babies (42.9%), and neonatal death (0%). P value = 0.00 which reflects that the test was highly significant.

4.1. Maternal Complications

The prevalence of **mild PIH** in the study was 55.6%. In mild PIH, most of the associated symptoms and signs, investigation, foetal well-being was expected. If there were any abnormality in one or more of these variables, then that would indicate a change to severe PIH.

The prevalence of **severe PIH** in the t study was 20%. The severe PIH could have resulted in maternal complications, HELLP syndrome, and abruption placenta were 36.4%, 9.1%, 14.4%, and 10.6%. The severe PIH associated with foetal complications, especially: IUGR, IUFD, neonatal death, and preterm babies, were: 31.8%, 15.2%, 3.8% and 22.8%.

The prevalence of **eclampsia** in the study was 18%. Eclampsia can be complicated and associated with maternal complications and HELLP syndrome, IUGR, and neonatal deaths (5%, 25%, 1%, and 25%).

The prevalence of **HELLP syndrome** in the present study was 9.1%. HELLP syndrome was complicated and associated with foetal complications, especially: IUGR, IUFD, neonatal death, and preterm babies with a per cent of (10%, 10%, 10% and 30%).

The prevalence of **abruption placenta** in the present study was 4.5%. The abruption of the placenta associated with maternal complications was 20%. The abrupt placenta associated with foetal complications especially; IUGR, neonatal death, and preterm babies were (40%, 20%, and 60%).

The prevalence of **pulmonary oedema** in the present study was 0.5%. The presence of associated symptoms with pulmonary oedema increased the risk of complications especially, headache (100%). The presence of the related signs within pulmonary oedema increased the risk of complications, primarily; severe hypertension, generalised oedema, and exaggerated reflexes were 100%, 66.7%, and 33.7% respectively. Abnormal clinical investigations within pulmonary oedema increased the risk of complications exceptionally; high protein urea, high uric acid, haemoconcentration (Hb > 12 g/dl), abnormal Liver Function Test (LFT), and low platelets were 100%, 66.7%, 33.3%%, 33.3% and 33.3% respectively.

4.2. Foetal Complications

The prevalence of **preterm deliveries** in the study was 19.1%. Prematurity was found to be associated with maternal complication were 23.8%, 14.3% respectively. Birth of Premature babies associated with foetal complications such as IUGR, IUFD, and neonatal death (23.8%, 4.8%, and 4.8%). 76.2% of the preterm babies were low birth weight, and 59.3% of those born with low birth weight were premature.

The prevalence of **Intrauterine Growth Restriction (IUGR)** was 19.1%. The presence of IUGR is also associated with maternal complications such as eclampsia, abruption placenta and HELLP syndrome (23.8%, 19.1%, 4.8% and 4.8%, respectively). IUGR has been associated with foetal complications especially Intra uterine foetal death, neonatal death, and preterm babies (4.8%, 4,8%, and 19.1%). 4.8% of IUGR deliveries associated were Abruptio placenta and Neonatal death. 52.4% of the IUGR were LBW, and 40.4% of the LBW, were IUGR.

The prevalence of **Low Birthweight (LBW)** was 24.6%, and when associated with maternal symptoms primarily, headache, blurred vision, and epigastric pain, it increased the risk of complication (83.3%, 37%, and 29.6%). On the other hand, the presence of associated signs with (LBW) has increased the risk of complications especially severe hypertension, generalised oedema, and exaggerated reflexes (62.6%, 37%, and 14.8%).

The prevalence of **Intrauterine Foetal Death (IUFD)** was 4.6%. The IUFD showed links with foetal complications especially; IUGR and preterm babies (20% and 20%). 20% of IUFD were IUGR and preterm babies.

The prevalence of **neonatal death** was 1.8% = 18/1000. The neonatal death resulted or/and was associated with maternal problems, mainly eclampsia, abruption placenta and HELLP syndrome 58.3%, 50% and 50%).

The percentages of the drugs used for the management of mild PIH were aspirin (42.8%), aldomet (85.7%), hydralazine (5%), labetalol (5%), nifedipine (85%), Mg. Sulphate (1%), diazepam (28.5%) and dexamethasone (75%).

The percentages of the drugs used for the management of severe PIH were aspirin (13%), Aldomet (85.7%), hydralazine (78%), labetalol (20%), nifedipine (85.7%), Mg. Sulphate (0%), diazepam (45.5%) and dexamethasone (75%).

The percentages of the drugs used for the management of eminent eclampsia were Aspirin (10%), aldomet (75%), hydralazine (80%), labetalol (18%), nifedipine (50%), Mg. Sulphate (95%), diazepam (10%) and dexamethasone (50%).

The percentages of the drugs used to treat eclamptic PIH were aspirin (15%), aldomet (85.7%), hydralazine (65%), labetalol (14.2%), nifedipine (85.7%), Mg. Sulphate (87.4%) and dexamethasone (71.1%).

The use of Heparin in the management of PIH associated with improvements in the growth of the foetus even in those with IUGR (63.6%). Heparin therapy improved nutrition and respiratory function and improved the development of the foetus.

Termination of pregnancies: In severe and complicated PIH, pregnancies were terminated by spontaneous vaginal delivery (7%), Induced vaginal delivery (29.1%), elective caesarean section (54.2%) and emergency section (64.4%). In proteinuric hypertension, the mode of termination of pregnancy was through spontaneous vaginal delivery (8%), Induced delivery (45.2%), elective section (68.8%), and emergency section (81.1%). The total percentage of pregnant women with proteinuria (≥2+) who underwent termination by caesarean section was 61.5%, which is 2.3 times the general incidence of caesarean section S in the hospital and higher than

the incidence of caesarean section in non-proteinuric hypertension (16.2 %).

5. Discussion

Hypertension disorder during pregnancy is related to maternal illness and death rates [49]. The maternal stigmas of the disease are hypertension, protein urea, and failure of placental development can lead to growth restriction [50]. James W. [51], even though there has been a reduction in maternal mortality from pre-eclampsia, pre-eclampsia remains the most significant reason women encounter morbidity during their pregnancy. The symptoms of this disease start with the placental trigger along with the systemic response. Since the systemic response to the condition remain inconsistent, it gets difficult to identify the clear clinical representation which could affect other organs of the female body. There has been an increased understanding of the disease process and it is bringing advancements in management with the introduction of several therapeutic interventions. The current study also focused on the treatment and management protocol of pre-eclampsia and eclampsia and measured its occurrence and incidence.

Manisto T. *et al.* [52] suggested that a rise in blood pressure during pregnancy could lead to cardiovascular and chronic kidney diseases even without the presence of known risk factors. Ghulmiyyah *et al.* [53] concluded that the maternal complications, and maternal mortality from the disease are higher in developing countries in comparison to developed countries.

Among the expected effects of pre-eclampsia, Eclampsia and (HELLP) syndrome are the commonly occurring conditions. Luo B. [54] found that risk factors of pre-eclampsia include gestational age, increased body mass index, fewer antenatal visits, and cold seasons. Akerman N. *et al.* [50] pre-eclampsia affects about 3% of pregnant women. On the other hand, other hypertensive disorders complicate pregnancies up to 5% to 10% in the USA. The rates of gestational hypertension, PET, and chronic hypertension have increased in industrialised countries. Still, it is lower than the present study, which looks at pre-eclampsia and its incidence of hypertensive disorders.

Barton J.R. *et al.* [55] pregnant women affected by pre-eclampsia management process that may include assessment and stabilisation of maternal condition along with evaluation of foetal well-being this is line with the findings of the present study. Vajira H.W. *et al.* [56] found the maternal complication which occurs in the PET involves severe blood pressure in 83% of patients proteinuria > 2 in 87%, renal failure requiring dialysis in (2%), low platelets in 15%, Abruption placenta 4%, eclampsia 9%, and one maternal death. The finding indicated that the maternal complication suggestive of the severity of the disease apart from severe hypertension had no significant difference in early or late-onset PET, which agreed with the present study.

Akerman N. *et al.* [50] addressed that PET is the prominent 3rd direct cause of the death of pregnant women in the U.K. Eclampsia incidence in Sri Lanka found to be 9% which exceeds the figure of our study. Abd El *et al.* [57], In Assiut uni-

versity hospital, estimated the figure of 3.9% of eclampsia death. Sibai B.M. [58], women < 34 weeks' gestation with pre-eclampsia/eclampsia suffered from pulmonary oedema, disseminated intravascular coagulation, renal insufficiency, abruption placenta, abnormal foetal testing, HELLP syndrome, and persistent severe PET, which was following our study. Akerman eclampsia [50], eclampsia can occur with mild disease and not necessarily associated with severe PET; the same observed in our study. ODU L.A. *et al.* [59] found that maternal haemoglobin levels are significantly elevated before giving birth in that suffering pre-eclampsia. Maternal haemoglobin concentration has an inverse relation to birth weight percentile.

Vajira *et al.* [56], Low platelet was 13% which is higher than the present study, the incidence of Abruptio placenta in Sri Lanka was (4%), which is in line with the present study. Vajira *et al.* [56], with late-onset PET foetal outcome was better, but PET in Sri Lanka was associated with severe morbidity in pregnant women and foetal morbidity and mortality, which agrees with the present study. Abd El al *et al.* [57], In Assiut university hospital, after eclamptic fit, the perinatal mortality occurred in 7.9% of cases, which is greater than the current study. Voskamp *et al.* [60] women who had SGA in their first pregnancy were likely to have SGA in the subsequent pregnancy. Vajira *et al.* [56], in Ser lanka 48% of babies born were small for gestational age, which is greater than the present study.

Tuffnell D.J. *et al.* [61], in PIH, 15% of pregnant women required no anti-hypertensive therapy and 53% of the remainder were subjected to oral treatment only. And (18.5%) required more than one drug. which is in line with the present study. Lo Jamie [49], the early detection of hypertension and its acute management help reduce the mortality in women, which agrees with the present study [62]. Studies have found that bromocriptine is a suitable inhibitor of lactation. Serious accidents have occurred during the postpartum period. However, aspirin, when given in low doses, did not help in reducing the incidence of pre-eclampsia. Annapre *et al.* [62], treatment with low-dose Acetylsalicylic Acid (ASA), started early in pregnancy, could prevent PET, IUGR and possible premature birth among nulliparous women with early pregnancy indication of placental dysfunction, which is agree with the present study. Egarter C. *et al.* [63] demonstrated that the low-dose aspirin starting at less than 16 weeks was the significant predictor of decreased perinatal death. Contrarily, the aspirin effect was not significant when initiated after 16 weeks. On the other hand, Roberge S. *et al.* [64] found that the use of low-dose aspirin is significantly helpful in decreasing perinatal death regardless of the condition if the treatment is applied on or before 16 weeks of gestation [65]. Duley L. [66], antiplatelet drugs reduced the risk of premature birth without increasing the risks of bleeding, similar to what we observed in this study. However, concerning the mixed findings the search for valuable tools to identify pregnant women at risk continues [67]. Thus, it is not yet conclusive whether low-dose aspirin is beneficial for maternal safety (Lo, Jamie *et al.* [50]). Study has indicated the positive effect of good antenatal care and magnesium sulphate use with the better outcome,

following the present study (Abbassi *et al.* [68]). Several neonatal complications are also significantly related to the increased magnesium levels in the maternal circulation. Tukur *et al.* [69] looked at the incidence of fetal mortality in eclampsia and pre-eclampsia, which was up to 2.3%. Moreover, the death rate dropped in centers using diazepam with the same has been noticed in our study. Akerman N. [50] $MgSO_4$ is given by I/V infusion following an eclamptic convulsion. It may also be given as a prophylactic measure to women being treated for PET in whom a decision to deliver has been made, which is agree with the present study.

Dodd J.M. *et al.* [70] Antithrombotic therapy that involves the use of heparin treatment improves the maternal and foetal health outcomes. Previous studies showed that pregnant women who received Heparin were more likely to give birth to healthy babies and had a lower incidence of pre-eclampsia.

The current study results suggested that maternal complications of PIH could be both acute and long-term. There is a dire need for proper protocols for early detection and management of hypertension in pregnant women for better maternal and perinatal health. This is an essential requirement in any efficient health care system.

6. Conclusions

Preeclampsia and eclampsia are significant factors related to death rates in mothers and babies. However, the condition completely resolves postpartum. The current study concluded that in order to reduce the morbidity and mortality rates in both mother and foetus in such cases of PIH, it's essential to have protocols to ensure efficient care and regular antenatal monitoring and follow-ups. The use of low-dose aspirin as prophylactic, especially in vulnerable patients, along with early detection and diagnosis of PIH, early detection of maternal or foetal complication, anticipating treatment provided the patients of PIH, by the use of anti-hypertensive drugs with oral, methyldopa, for mild hypertension, and I/V hydralazine and labetalol for severe high hypertension, early referral of severe cases to intensive care units, the wide use of magnesium sulphate as prophylactic in the treatment of pre-eclampsia and eclamptic fits, which would help to decrease maternal and foetal deaths and illness rates and reduce morbidities. Dexamethasone should be used for all patients with PIH diagnosed before 34 weeks gestation to increase the lung maturity of the foetus.

The definitive management of PIH is the termination of the pregnancy and delivery of the placenta. We terminate pregnancy for the patients with severe PIH, eclampsia, eclamptic fits, HELLP syndrome, abruption placenta, pulmonary oedema, cerebral haemorrhage, liver and renal failure, or foetal complication, at any gestational age.

Heparin trial as an anti-coagulant to improve maternal and foetal outcomes has been used in 28 cases. Though we don't have much experience with its use, results and data from the literature seem promising. It decreases blood pressure and proteinuri, improves the Platelet count and placental function, and improves foe-

tal nutrition, respirations, and the general outcome.

Pre-eclampsia is a multisystem disorder of pregnancy that and still an underestimated risk factor for future cardiovascular, cerebrovascular, and kidney diseases. There is a need for early pre-eclampsia screening and effective prophylaxis to prevent the most severe complications related to increased risk of maternal death and disability. On the other hand, women with pregnancy complicated by pre-eclampsia should also be covered by very watchful follow-ups, which can extend for many years after pregnancy, because of rising cardiovascular disease-related mortality in adult women, especially those aged 35 - 45 years [1].

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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