1. Introduction

1.1. Overview

Hypertension is recently termed ***"The Silent Killer";*** it contributes the highest percentage of cardiovascular diseases burden which is now more commonly seen in developing countries with low to medium income and poor health care systems. Its late diagnosis is mainly because patients are asymptomatic at the early stage while others go undiagnosed, receive delayed treatment or even not at all. Early and sufficient control of hypertension reduces economic and health burden which ultimately improves manpower and productivity of a nation. Hypertension is defined by The World Health Organization (WHO) as; "a systolic blood pressure equal to or above 140 mmHg and/or diastolic blood pressure equal to or above 90 mmHg (WHD/2013.2). Pregnancy-Induced Hypertension is a form of newly diagnosed hypertension in pregnancy after 20 weeks of gestation, devoid of proteinuria and other signs of Pre-eclampsia.

1.2. Study Objectives

Objectives of this study are: To educate patients on the disease, lifestyle modification and adherence to medications; evaluate the impact of patient education on Blood Pressure control and lastly justify the ways in which Pharmacist's intervention will reduce morbidities and mortalities from Pregnancy-Induced Hypertension.

1.3. Prevalence

Hypertension has a global prevalence rate of 20%-30% in adult population and more than 5%-8% of pregnancies worldwide (DeCherney, 2012). In a systematic review study carried out in Nigeria in 2015, the results showed a higher prevalence in the male population of 6.2% to 48.9% as compared to that of the female population of about 10% to 47.3% (Akinlua et al, 2015).

1.4. Regional Disparity

Though it is still unclear why there are world-wide regional differences in the prevalence of hypertension, it may probably be due to difference in its awareness and control. For example, the European countries have higher prevalence than countries in Northern America (Lacrus et al,

2015). Several studies carried out in Nigeria reported that late onset pregnancy induced hypertension accounts for most cases of hypertension in pregnancy, with the early onset ones relatively rare compared to that seen in Caucasian populations (Lacruz et al. 2015).

1.5. Location of the Study

Nigeria is a tropical Sub Saharan country in the West African region that covers a land mass area of 923, 768.00 square kilometers. It has a maximum and minimum temperatures of 45^0C and 6^0C respectively. Katsina is a North Western State in Nigeria having a Muslim-Hausa predominant population. It occupies an area of 24, 192 square kilometers and has a population of over 6.5 million (NDHS, 2008). This Study was carried out in a tertiary medical institution (Federal Medical Center) in Katsina State of Nigeria.

1.6. Background

One of the most common medical complications of pregnancy is Hypertensive disorders and pregnancy-induced hypertension (PIH). Pregnancy Induced Hypertension is defined as BP≥140mmHg/90mmHg, taken on two occasions after rest or ≥160mmHg/110mmHg taken once in a previously normotensive woman (Sibai, 2003). The American College of Obstetricians and Gynecologists (ACOG) classified Hypertensive disorders in pregnancy into four groups:
(1) Gestational hypertension; here the resting blood pressure (BP) is 140/90 mmHg or higher after 20th week of gestation;

(2) Chronic hypertension; this occurs before pregnancy or begins in the first week 20 weeks of gestation;

(3) Pre-eclampsia, defined by hypertension and/or edema;

(4) Pre-eclampsia superimposed on chronic hypertension (ACOG, 2000).

1.7. Preeclampsia

Based on severity of pre-eclampsia, The American Congress of Obstetricians and Gynecologists (ACOG) used blood pressure and systemic involvement parameters to categorize preeclampsia into two classes: i. mild to moderate; and ii. Severe preeclampsia.

1.7.1. Classification

Mild to Moderate preeclampsia: the patient's blood pressure here ranges from 140 to 159 mmHg systolic and 90 to 109 mmHg diastolic.

Severe preeclampsia: Here, any or a combination of the following conditions are seen;

- Blood pressure of ≥160/110mmHg in two or more readings taken at least 6 hours apart in a patient on bed rest,
- Intrauterine growth restriction and/or retardation,
- Presence of ≥5g or triple positive value of protein in urine within 24 hours in at least in two random urine samples collected at least 4 hours apart,
- Low urine output of less than 500ml in 24 hours (Bosio PM et al, 1999).

However, an epidemiological study carried out in the USA from 1995 to 2004 showed that pregnancy-induced hypertension (gestational hypertension/preeclampsia) was the most commonly diagnosed hypertensive condition in pregnancy when compared to pre-existing hypertension (Savitz et al, 2013).

In 2011, WHO reported pregnancy induced hypertension as one of the main causes of maternal, fetal and neonatal mortality and morbidity in developing countries (WHO, 2011) including Nigeria (Salako et al,) and the most common cause of maternal death in Europe (Onuh et al, 2004). With the current population of Nigeria which is estimated to be over 170 million, it is most likely that the prevalence of hypertension in Nigeria may form a substantial proportion of the total burden in Africa (Adeloye et al, 2015).

1.8. Clinical Pharmacist's Intervention; a form of Pharmaceutical Care.

Pharmaceutical Care is a patient-centered form of practice which utilizes the professional knowledge and skills of the pharmacist to achieve best drug therapy outcomes (Hepler, 1990).The role a Clinical Pharmacist plays here; is to optimize pharmaceutical care and proper medication use for the attainment of best outcomes. The target duties in this study includes; setting goals, providing patient counselling and evaluating treatment outcomes.

2. Literature Review

2.1.Hypertensive Disorders of Pregnancy

Hypertensive disorders of pregnancy continue to be one of the largest causes of maternal and fetal mortality and morbidity and affects 3-10% of all pregnancies worldwide (Granger et al., 2001). Hypertension is a common clinical complication during pregnancy. It is usually defined as systolic blood pressure of at least 140 mmHg and/or diastolic blood pressure of at least 90 mmHg (Cnossen et al., 2006). Hypertension is reported to account for 15% of all antenatal hospitalizations for pregnancy complications in the United States (Scott et al., 1997). Depending on the region, between 9.1% (Africa, Asia), 16.1% (developed countries), and 25.7% (Latin America) of maternal deaths may be attributed to pregnancy associated hypertension (Khan et al., 2006). About 18% of fetal deaths are associated with hypertensive disorders (Cnossen et al., 2006).

The diagnostic criteria for disorders of hypertension in pregnancy are not presently consistent and there are a number of different systems made known by major working groups and international societies. The National High Blood Pressure Education Program Working Group (2000) has classified hypertensive disorders of pregnancy as chronic hypertension, preeclampsia superimposed on chronic hypertension, preeclampsia-eclampsia, gestational hypertension (pregnancy-induced hypertension).

2.2.Pregnancy-Induced Hypertension

Pregnancy-Induced Hypertension (PIH) is defined as the occurrence of hypertension after 20 weeks of gestation in a woman without prior hypertension (National High Blood Pressure Education Group, 2000). It is usually defined as systolic blood pressure of at least 140 mmHg and/or diastolic blood pressure at introduction of least 90 mmHg (Cnossen et al., 2006). When accompanied by proteinuria, the disorder is termed preeclampsia and when it is without significant proteinuria it is termed; gestational or transient hypertension (National High Blood Pressure Education Group, 2000).

Table 1: Classifications of Hypertensive Disorders during Pregnancy (McCoy and Baldwin, 2009; Vest and Cho, 2012; Steegers et al., 2010; Brown et al., 2001).

American College of Obstetricians and Gynecologists (ACOG)	International Society for the Study of Hypertension in Pregnancy (ISSHP)	Working Group on High Blood Pressure in Pregnancy	Royal College of Obstetricians and Gynecologists (RCOG)	
Chronic Hypertension	BP ≥ 140/90 mm Hg Present before 20 weeks	BP ≥ 140/90 mm Hg Present before 20 weeks	BP ≥ 140/90 mm Hg Present before 20 weeks	***
Gestational Hypertension	BP ≥ 140/90 mm Hg Onset after 20 weeks	BP ≥ 140/90 mm Hg Present after 20 weeks	BP ≥ 140/90 mm Hg Onset after 20 weeks	***
Pre-eclampsia	BP ≥140/90 mm Hg Onset after 20 weeks Proteinuria*	BP ≥ 140/90 mm Hg Onset after 20 weeks Proteinuria*	BP ≥140/90 mm Hg Onset after 20 weeks Proteinuria*	Gestational hypertension Proteinuria*
Severe Preeclampsia	BP > 160/110 mm Hg Excessive proteinuria**	BP ≥ 160/110 mm Hg	DBP ≥ 110 mm Hg Severe symptoms^	BP ≥ 170/110 mm Hg (severe hypertension)
Preeclampsia Superimposed on Chronic Hypertension	BP ≥ 140/90 mm Hg Present before 20 weeks New onset proteinuria	BP ≥ 140/90 mm Hg Present before 20 weeks New onset proteinuria	New onset proteinuria during pregnancy in chronic hypertension	

*Proteinuria is defined as > 300 mg on 24 hour urine collection or > 30 mg on a urine spot test

**Excessive proteinuria is defined as > 5 grams on 24 hour urine collection

***RCOG follows NICE guidelines

2.2.1.Pathophysiology of Pregnancy-Induced Hypertension

The pathogenesis of hypertensive pregnancy still remains uncertain; it has been said to be multifaceted and includes immune, genetic, placental abnormalities and environmental factors. Kopcow & Karumanchi, (2007) documented the immunological basis of preeclampsia. Chelbi and Vaiman, (2008) hypothesized that genetic, epigenetic and environmental factors are involved in the pathogenesis of preeclampsia. Additionally, it has been reported that there is decreased

formation of vasodilators such as nitric oxide (Granger et al., 2001; Mitchell et al., 2007) and prostacyclin (Granger et al., 2001) in women who present with gestational hypertension and preeclampsia. Mitchell et al, (2007) has also reported that increased reactive oxygen species (superoxide and peroxynitrite) production and decreased bioavailability of the endothelial nitric oxide (NO) synthase (eNOS), cofactor tetrahydrobiopterin (BH_4) could add to maternal endothelial dysfunction in rats with pregnancy-induced hypertension and the numerous characteristics of preeclampsia.

It has also been suggested that preeclampsia is a two-stage disease (Roberts, 2000); the first stage is asymptomatic and characterized by abnormal placental development during the first trimester leading to placental insufficiency and the release of disproportionate amounts of placental materials into the maternal circulation. This in turn leads to the second, symptomatic stage, where the pregnant woman develops characteristic hypertension, renal impairment, and proteinuria and also at increased risk for the HELLP syndrome (haemolysis, elevated liver function enzymes and low platelets), eclampsia, and other end organ damage (Hladunewich et al., 2007). All these may contribute to endothelial dysfunction typical of gestational hypertension and preeclampsia and this endothelial dysfunction may in turn, trigger several critical features of preeclampsia, including vasoconstriction, hypertension, loss of the usual pregnancy-associated refractoriness to pressor effects of angiotensin II, increased platelet aggregation and proteinuria (National High Blood Pressure Education Group, 2000).

2.2.2.Incidence and Prevalence of Pregnancy-Induced Hypertension

There are varying reports on the incidence of hypertensive disorders of pregnancy worldwide and these incidence reports show great disparity. This may be attributable to differences in definition, population composition, demographic and obstetric characteristics, actual disease incidence, or access to and availability of antenatal care services (WHO, 1988). A population based international collaborative study designed to control for these factors found that clinically recognized hypertension during pregnancy varied by a factor of 25 (incidence range 1.2% to 31.0%) between countries. Even using a strict definition of proteinuric hypertension, the incidence varied by a factor of 5 (incidence range 1.5% to 8.3%) (WHO, 1988).

Hypertension complicating pregnancy (approximately 9% worldwide) has been reported to be associated with substantial maternal and perinatal morbidity and death, mostly because of

preeclampsia (pure or superimposed on chronic hypertension) (Villar et al., 2006). Pregnancy-Induced Hypertension complicates 5-10 % pregnancies in the United States and is a major cause of maternal, fetal and neonatal morbidity and mortality (Seely & Solomon, 2003). Gestational hypertension has been reported to complicate between 4.4 and 17.5% of pregnancies, with a weighted mean of 14.6% (Hauth et al., 1993; North et al, 1999; Stone et al, 1995).

The reported incidence of preeclampsia varies between 3-10% (Mittendorf et al., 1996; Redman & Jefferies, 1988; WHO, 1988) and some of this variation may be attributable to differences between study populations. In Tehran, an incidence of 3% for preeclampsia has been reported (Pyri et al., 2001). In Sri Lanka, studies on hypertensive disorders of pregnancy have been reported to occur in 4.9% of pregnant women delivering in a tertiary care hospital (Jayawardana & Fernando, 1995). This study and another from the same hospital (Jayawardana & Lekamge, 1994), report the proportion of hypertensive women having preeclampsia (43.4% and 46.5% respectively) as being not much less than that of gestational hypertension (51.1% and 53.4% respectively). In Ghana, incidence of preeclampsia amongst pregnant women has been reported to be about 7.03% (Obed & Aniteye, 2006).

2.2.3.Risk Factors for Pregnancy-Induced Hypertension

Research into PIH has been unlimited as a result of its growing prevalence, but to date the etiology remains unknown.However, a number of risk factors have been identified (Roberts & Lain, 2002; Zhang et al., 1997). These risk factors for hypertensive pregnancy (preeclampsia and gestational hypertension) includes maternal, paternal, genetic, environmental and/or obstetric factors. Reportedly, primiparas are known to be at markedly greater risk of preeclampsia than multiparas (Chesley, 1984). Preeclampsia is reported to complicate 25-30% of nulliparous pregnancies, it is more common in nulliparous women than in multiparous women and as such the first pregnancy is understood to be a risk factor for preeclampsia (Serhal et al., 2003).

Lack of leisure-time physical activity early in pregnancy (Marcoux et al., 1989), the use of barrier contraceptives (Klonoff-Cohen et al., 1989), young maternal age (Saftlas et al., 1990), and partner change (Duckitt & Harrington, 2005; Sibai et al., 1997; Trupin et al., 1996); have all been reported to amplify the risk of PIH or preeclampsia. Women with hypertensive pregnancy

are also reported to present with pregnancy overweight and metabolic derangement and are thought to present with a syndrome similar to the Metabolic Syndrome.

2.2.3.1.Pregnancy-Induced Hypertension and Metabolic Syndrome

It is a common knowledge that women who develop Pregnancy-Induced Hypertension also develop a syndrome similar to metabolic Syndrome. Such women, exhibit exaggerated insulin resistance and metabolic changes (Seely & Solomon, 2003). There is uncertainty as to the pathogenesis of these factors in hypertensive pregnancy but many suggested that it may play a role in either disease evolution or markers underlying a disease process (Seely & Solomon, 2003).

Women in whom PIH eventually develop are more likely to present with pregnancy overweight and demonstrate during pregnancy, some of the risk factors characterizing atherosclerosis, such as dyslipidemia (hypertriglyceridemia, low levels of high-density lipoprotein (HDL) cholesterol and low-density lipoprotein (LDL) cholesterol (Belo et al., 2002; Sattar et al., 1997) insulin resistance (Kaaja et al., 1999; Seely & Solomon, 2003) and endothelial dysfunction (Roberts, 1998). Indeed, these metabolic irregularities (increased adiposity, hyperlipidemia, hyperglycemia, and elevated blood pressure) are indicative of the metabolic syndrome (Forest et al,2005). However geographical, social, economic and racial differences are thought to be responsible for incidence rates up to 3 times higher in some populations (Lopez-Jaramillo et al., 2001; WHO, 1988). In some countries such as Columbia it is the main cause of maternal mortality; up to 42% of maternal deaths are attributed to this disorder in Colombia (Lopez-Jaramillo et al, 2001). Because of the increased risk for morbidity and mortality associated with the Metabolic Syndrome, an understanding of the dimensions of this syndrome is critical both for allocating health care and research resources and for other purposes (Ford & Giles, 2003).

2.2.3.2.Dietary Salt and Pregnancy-Induced Hypertension

There is evidence in support of the hypothesis which proposes that prolonged increases in salt intake or the habitual high intake of salt is related to rise in arterial blood pressure (Chobanian & Hill, 2000) however, the mechanisms by which salt intake raises the blood pressure still remains

unclear (de Wardener et al., 2004). Cappuccio et al, (2006) showed a significant and positive relationship between the level of salt intake and both systolic and diastolic blood pressure.

Hypertension involves abnormal and persistent changes in the blood pressure control mechanisms. In industrialized societies, the individual's average salt consumption is 10 g/day, and the incidence of hypertension is greater than in rural societies. A large body of evidence points to a link between dietary salt, kidney function, and hypertension (Guyton, 1991; Hall et al., 1980; Meneton et al., 2005). Various mechanisms by which salt increases blood pressure have been put forward. A decrease in the capacity of kidneys to excrete salt would cause salt and water retention, increased extracellular and plasma volume, and increased blood pressure.

The kidneys' ability to excrete sodium declines gradually with age, and smaller increases in salt intake induce a rise in blood pressure. Also, with increasing age, the glomerular filtration rate is reduced, accompanied by a decline in functioning nephrons and progressive glomerulosclerosis. If with age salt consumption is not reduced, sodium balance is maintained by raising fractional sodium excretion, which requires elevation of blood pressure (Corman & Michel, 1987; Khalil, 2006). High salt diet may also increase renal medullary osmolality and decrease Nitric Oxide (NO) synthase expression (Herrera et al., 2006) and reduced renal medullary NO synthase activity is associated with salt-sensitive hypertension (Tian et al., 2003).

2.2.3.3.Age; a risk factor for Pregnancy-Induced Hypertension

Several epidemiological studies have considered the association of maternal age and the risk of Pregnancy-Induced Hypertension (gestational hypertension and preeclampsia). Inspite of this, there have been inconsistent reports on the effects of maternal age on preeclampsia. While some studies did not find age a significant risk factor (Anorlu et al., 2005; Conde-Agudelo & Belizan, 2000; Eskenazi et al., 1991), some studies have reported increased risk of preeclampsia in younger women who are ≤ 21 years (Sibai, 1990) and other studies have reported an association of increased risk of preeclampsia with women who are 35 years or older (Conde-Agudelo & Belizan, 2000; Sibai, 1990).

Several studies have reported that increasing age is associated with an increased risk of gestational hypertension (Brown & de Swiet, 1999; Eras et al., 2000; Hartikainen et al., 1998). Teenagers and women aged 35 years and over, generally have been shown to have a greater risk of adverse perinatal outcomes, including low birth weight, (Fraser et al., 1995; Reichman &

Pagnini, 1997) small-for-gestational age, (Cnattingius et al., 1992; Fraser et al., 1995), preterm birth (PTB), (Cnattingius et al., 1992; Fraser et al., 1995; Jacobsson et al., 2004) and perinatal or infant mortality (Cnattingius et al., 1992; Jacobsson et al., 2004; Olausson et al., 1999) and these are largely associated with Pregnancy-Induced Hypertension.

2.2.3.4.Anthropometric Indices and Pregnancy-Induced Hypertension

Anthropometric measurement is the science of measuring the human body parts for height, weight, and size of component parts including skinfold thickness, to study and compare the relative proportions under normal and abnormal conditions. Anthropometric indices includes;Weight(Wt), Height (Ht), Ponderal Index (PI), Body Mass Index (BMI), Thigh Circumference/Head Circumference Ratio (THR), Waist-to-Hip Ratio (WHR),Mid-Arm Circumference to Occipito-Frontal Circumference(MAC/OFC), Weight to Occipito-Frontal Circumference (W/OFC), and Weight/Length (W/L).

Anthropometric measurements are among the most frequently applied methods for assessing nutritional status in pregnant women and are recognized as an effective tool for the prevention of perinatal, morbidity and mortality, the prognosis of child health, and the promotion of women's health (Oliveira et al., 2004; Padilha & Nelson, 2009).

2.2.4.Stress, Exercise and Hypertensive Disorders of Pregnancy

The occurrence of preeclampsia as well as gestational hypertension has been associated with stress at work and reduced participation in exercise during pregnancy. Some studies have reported that high job stress have a positive relationship with gestational hypertension (Landsbergis & Hatch, 1996; Marcoux et al., 1989). Preeclampsia has been hypothesized as a stress-related disease and indeed epidemiologic studies show that the relative risk for preeclampsia is increased in many stressful situations (Takiuti et al., 2003).

Reduced levels of physical exercise have also been associated with the development of gestational hypertension (Marcoux et al., 1999; Marcoux et al., 1989). Similarly, moderate/high physical activity is reported to be associated with a two times increase in the risk of severe preeclampsia compared to mild activity (Spinillo et al., 1995). Marcoux et al., (1989) also

reported that the lack of leisure-time physical activity early in pregnancy had the tendency to increase the risk of developing gestational hypertension.

2.3.Parity andPregnancy-Induced Hypertension

Parity which refers to the number of times a woman has given birth, for long have been associated with hypertensive pregnancy. The strong relationship between parity and the clinical condition was documented over 300 years ago by Mauriceau, who indicated that ‘‘primigravidas are at far greater risk of convulsions than multiparas.’’ Several studies have corroborated this observation and others have revealed that the association does not hold for nulliparity and nonproteinuric hypertension (Campbell et al., 1985; Misra & Kiely, 1997). Indeed, gestational hypertension has been reported to be more common in nulliparous than multiparous women (1.6- to 2-fold), but the association is less remarkable than that seen in preeclampsia (Campbell & MacGillivray, 1999; Hartikainen et al., 1998; Trupin et al., 1996).

In a Scottish study of over 130,000 pregnancies, the relative risk (RR) of gestational hypertension in nulliparous women compared to multiparas was 1.98 (95% CI 1.94–2.03) in singleton pregnancies and 1.85 (95% CI 1.55–2.21) in twin pregnancies (Campbell & MacGillivray, 1999). Among nulliparous women, gestational hypertension was more common in the first pregnancy compared to subsequent pregnancies, [odds ratio (OR) 2.29 (95% CI 1.65-3.20)] (Eras et al., 2000). Indeed, preeclampsia is frequently considered as being a clinical condition of first pregnancies (Roberts & Redman, 1993). Serhal and Craft, (1987) also reported that first pregnancy is a risk factor for preeclampsia and its occurrence is more common in nulliparous than multiparous women. Suggesting that after a previous normal pregnancy, there is a markedly lower incidence of preeclampsia in subsequent pregnancies. But the protective effect of multiparity, is however lost with change of partner (Dekker, 2002). This has led to widespread epidemiological studies, and has given rise to data to implicate immunological factors in the etiology of preeclampsia.

2.4.Primipaternity andPregnancy-Induced Hypertension

It has been suggested that primipaternity rather than primiparity is an appropriate risk factor (Robillard et al., 1999; Robillard et al., 1993) for hypertensive pregnancy since some studies

have shown that the protective effect of multiparity, is lost with change of partner (Belfort et al., 2002). This implies that, not only are primiparas at high risk, multiparas having a child with a new father (Li & Wi, 2000; Lie et al., 1998; Need et al., 1983; Trupin et al., 1996) are equally at a high risk for hypertensive pregnancy. Trupin et al., (1996) reported that multiparous women who change their partners have a slightly higher risk of developing gestational hypertension than multiparous women with the same partner [OR 1.3 (95% CI 1.1–1.6)].

In a cohort study based on 140,147 women with two successive births during 1989–1991, Li and Wi (2000), established that among women without preeclampsia in the first birth, changing partners resulted in a 30% increase in the risk of preeclampsia in the subsequent pregnancy as compared with women who did not change partners. However, among women with preeclampsia in the first birth, changing partners resulted in a 30% reduction in the risk of preeclampsia in the subsequent pregnancy. Li and Wi's findings show that preeclampsia is rather a disease of primipaternity rather than primigravidity and are consistent with the hypothesis that normal pregnancy reflects a state of tolerance to the foreign paternally derived antigens of the foetus, whereas in women with preeclampsia, this immunological tolerance is impaired. Similarly, an increased risk has been noticed among women who had artificial insemination by an unknown donor (Belfort et al., 2002; Smith et al., 1997).

2.5.Contraceptive Use andPregnancy-Induced Hypertension

Pregnancy-Induced Hypertension has long been considered to have an immunological basis, as its frequency is largely increased with primigravidae and rarely affects multigravid women unless there is a change in paternity (Robillard et al., 1993). This concept has been supported by the results of several studies suggesting that repeated exposure to father's spermatozoa prior to conception may reduce the risk of pregnancy-induced hypertension in the first pregnancy (Marti & Herrmann, 1977). A prospective study of 1011 pregnant women reported a strong inverse association between the length of sexual cohabitation with the father and the risk of pregnancy-induced hypertension, (Robillard et al., 1994).

The risk of developing gestational hypertension or preeclampsia was increased 12-fold if the duration of sexual cohabitation before conception was less than 4 months compared to more than 12 months (Robillard et al., 1994). The very high incidence (24.0%) of pregnancy-induced hypertension among new-paternity multiparous women was shown to be related to a remarkably

short period of sperm exposure preceding conception, suggesting that extended duration of sexual intercourse might reduce this risk. It is therefore assumed that this may be related to the contact of spermatozoa with the female genital tract. However, it is yet to be established whether the risk of developing pregnancy-induced hypertension is dependent on the type of contraception used (Gratacos et al., 1996).

2.6.Pharmacologic Treatments

Pharmacologic treatment of Pregnancy-Induced Hypertension and preeclampsia does not lead to resolution. The only known resolution is delivery of the fetus and placenta (McCoy and Baldwin, 2009; Dattel et al., 2005; Borgelt et al., 2010). The focus of pharmacologic treatment is management of the maternal signs and symptoms so gestation may be prolonged and fetal outcomes improved (Steegers et al., 2010). Treatment often requires balancing maternal safety and fetal safety. An increased gestation leads to decreased morbidity and mortality for the fetus, but this should be weighed against maternal condition, as preeclampsia may quickly progress to eclampsia, HELLP syndrome, or other morbidities (McCoy and Baldwin, 2009).

Numerous medications are available to treat hypertension and caution should be used when selecting an agent for use during pregnancy. Treating pregnancy-induced hypertension and preeclampsia requires knowledge of the mechanism of action and the safety and efficacy profiles of the medications. Commonly used antihypertensive pharmacologic agents include labetalol, hydralazine, methyldopa, nicardipine, or nifedipine (McCoy and Baldwin, 2009; Dattel et al., 2005; Barss et al., 2012).

Certain classes of antihypertensive medications should not be used during pregnancy, including angiotensin converting enzyme inhibitors (ACEIs), angiotensin II receptor blockers (ARBs), some beta-blockers, and diuretics (Vest and Cho, 2012). ACEIs and ARBs have been associated with detrimental effects on fetal growth and development, including renal failure and death of the fetus (LexiComp, 2010).

Additionally, some beta-blockers such as atenolol and metoprolol have been associated with an increased risk of intrauterine growth restriction and are therefore avoided (Vest and Cho, 2012; Giannubilo et al., 2012). Diuretics, although often helpful in treating hypertension, do not have

much of a role in treating pregnancy-induced hypertension and preeclampsia since women with these conditions may already be in a state of decreased volume (Vest and Cho, 2012). Use of diuretics may further deplete circulating volume, potentially leading to hypovolemia and decreased placental perfusion (Vest and Cho, 2012).

2.6.1.Hydralazine

Hydralazine lowers blood pressure by decreasing systemic vascular resistance through direct vasodilation of arterioles (LexiComp, 2010). Acute maternal hypertensive emergency is the most common use of parenteral hydralazine during pregnancy. Common side effects of hydralazine may present as nausea, vomiting, and headache in up to 50 percent of patients with preeclampsia (Nij et al., 2010). Hydralazine may cause maternal hypotension, reflex tachycardia, and flushing (McCoy and Baldwin, 2009). Maternal use of hydralazine has also been associated with thrombocytopenia in neonates (Vest and Cho, 2012).

2.6.2.Labetalol

Labetalol is a non-selective antagonist at alpha1, beta1, and beta2 adrenergic receptors, and is FDA approved for the treatment of hypertension and hypertensive emergencies (LexiComp, 2010; Giannubilo et al., 2012); Pregnancy induced hypertension and preeclampsia are off-label uses. Labetalol may be preferred over other beta blockers as it dilates arterioles and decreases vascular resistance without significantly lowering cardiac output (Molvi et al., 2012). Labetalol may be administered as an oral or intravenous product. Hypotension, bradycardia, and hypoglycemia are common adverse effects of beta blockers.

2.6.3.Methyldopa

Methyldopa, or α-methyldopa, is an alpha2 adrenergic agonist which causes a reduction in blood pressure by decreasing the effects of the sympathetic nervous system (LexiComp, 2010). Methyldopa is frequently used to treat hypertension during pregnancy (Vest and Cho, 2012). This medication may be administered by either intravenous or oral routes. Methyldopa has the most long-term safety data to support its use during pregnancy (Cockburn et al., 1982). No adverse effects on growth and development were seen in a 7.5 year follow-up in children exposedto methyldopa in utero.

2.6.4.Nicardipine/Nifedipine

Calcium channel blockers inhibit the L-type calcium channels in the cardiac and vascular smooth muscle cells, which exerts negative inotropic effects on the heart and causes vasodilation, leadingto decreased systemic vascular resistance. Both Nicardipine and Nifedipine have been studied for use during pregnancy (Vest and Cho, 2012; McCoy and Baldwin, 2009). Nicardipine has been found to be highly selective for vascular smooth muscle compared to cardiac muscle. Nicardipine has also been found to have more selective effects thanNifedipine, resulting in less reflex tachycardia and less pronounced negative inotropic effects. Nicardipine is available in both oral and intravenous dosage forms, while Nifedipine is only available in oral forms (LexiComp, 2010). Both Nicardipine and Nifedipine have been shown to be effective at lowering blood pressure in pregnant women (Aya et al., 1999).

2.7.Medication Therapy versus Induction of Labor

Currently, there is no clear consensus on when to treat hypertensive disorders in pregnancy. Several organizations recommend treatment thresholds varying from 140/90 mm Hg to 170/110 mm Hg based on differing criteria (Vest and Cho, 2012). The American College of Obstetricians and Gynecologists recommends treating pregnancy-induced hypertension when blood pressure increases to 150-160 mm Hg systolic or 100-110 mm Hg diastolic (Vest and Cho, 2012). Table 2 lists the treatment thresholds of several organizations and guidelines. A lack of consensus exists on target blood pressure during treatment as well (Vest and Cho, 2012). In pregnancies complicated by pregnancy-induced hypertension and preeclampsia between 20 weeks gestation and the viabilityage, maternal risk seems to be substantially increased with low survival rates for the fetus (Steegers et al., 2008). This is further complicated by the fact that various institutions define the age of viability differently; ranging between 23 weeks, 0 days to 24 weeks, 6 months depending on local definitions (Steegers et al., 2008).

Table 2: Treatment Thresholds for Hypertension in Pregnancy (Vest and Cho, 2012; ACOG, 2002).

Treatment Threshold (mmHg)	
National High Blood Pressure Education Program (US) Working Group Report 2000	150-160 Systolic 100-110 Diastolic
American College of Obstetricians and Gynecologists Practice Bulletin 2002	>105-110 Diastolic
European Society of Cardiology 2011	150/95 or 140/90 if high risk
Society of Obstetricians and Gynecologists of Canada 2008	160/110
Society of Obstetric Medicine of Australia and New Zealand 2008	170/110 or 160/100 in chronic hypertension 140-160/90-100 treatment is reasonable
National Institute for Health and Care Excellence (UK) 2010	150-159/100-109 140/9 if end-organ damage in chronic hypertension

2.8.Non-Pharmacological Management of Pregnancy-Induced Hypertension

- Use salt as needed for taste.
- Drink at least 8 glasses of water a day.
- Increase the amount of protein you take in, and decrease the amount of fried foods and junk food you eat.
- Get enough rest.
- Exercise regularly.
- Elevate your feet several times during the day.
- Avoid drinking alcohol.

- Avoid beverages containing caffeine.

2.9.Role of Pharmacist in Management of Pregnancy-Induced Hypertension

2.9.1.Detection and Diagnosis

- Taking every appropriate opportunity to assess the blood pressure of pregnant women in order to facilitate early detection of hypertension.
- Knowledgeable regarding the process involved in the diagnosis of hypertension.
- Educating the patients about regular and self/home blood pressure monitoring techniques and appropriate equipment to assist in potential diagnosis and the monitoring of hypertension.
- Educating the patients on their target blood pressure and the importance of achieving and maintaining this target.

2.9.2.Assessment and Development of a Treatment Plan

2.9.2.1.Lifestyle Interventions

Working with patients to identify lifestyle factors that may influence hypertension management, recognize potential areas for change and create a collaborative management plan to assist in reaching goals, which may prevent secondary complications.

2.9.2.2.Diet

Assessing for and educate patients about dietary risk factors as part of management of hypertension, in collaboration with dietitians and other members of the healthcare team.

Counselling clients with hypertension to consume the DASH Diet (Dietary Approaches to Stop Hypertension), in collaboration with dietitians and other members of the healthcare team.

Counselling the patients on hypertension to limit their dietary intake of sodium to the recommended quantity of 65-100 mmol/day, in collaboration with dietitians and other members of the healthcare team.

2.9.2.3.Healthy Weight

Assessing patients' weight, Body Mass Index (BMI) and waist circumference.

Advocate that patients with a BMI greater than or equal to 25 and a waist circumference over 102 cm (men) and 88 cm (women) consider weight reduction strategies.

2.9.2.4.Exercise

Assessing patients' current physical activity level.

Counselling, in collaboration with the healthcare team, to engage in moderate intensity dynamic exercise to be carried out for 30-60 minutes, 4 to 7 times a week.

2.9.2.5.Alcohol

Assessing patients' use of alcohol, including quantity and frequency, using a validated tool.

Routinely discuss alcohol consumption with patients and recommend limiting alcohol use, as appropriate to a maximum of: Two standard drinks per day or 14 drinks per week for men; one standard drink per day or 9 drinks per week for women and lighter weight men.

2.9.2.6.Smoking

Knowledgeable about the relationship between smoking and the risk of cardiovascular diseases

Establish patients' tobacco use status and implement Brief Tobacco Interventions at each appropriate visit, in order to facilitate smoking cessation.

2.9.2.7.Stress

Assist clients diagnosed with hypertension to understand how they react to stressful events and to learn how to cope with and manage stress effectively.

2.9.2.8.Medications

Obtain medication history of patients, which will include prescribed, over-the-counter, herbal and illicit drug use.

Knowledgeable about the classes of medications that may be prescribed for clients diagnosed with hypertension.

Provide education regarding the pharmacological management of hypertension, in collaboration with physicians.

2.9.3.Assessment of Adherence

- Endeavour to establish therapeutic relationships with patient
- Explore patients' expectations and beliefs regarding their hypertension management.
- Assess patients' adherence to the treatment plan at each appropriate visit.

2.9.4.Promotion of Adherence

- Provision of the information needed for patients to make educated choices related to their treatment plan.
- Work with prescribers to simplify dosing regimens.
- Encourage routine and reminders to facilitate adherence.
- Ensure that patients who miss appointments receive follow-up telephone calls in order to keep them in care.
-

2.9.5.Monitoring and Follow-up

Advocate that clients who are on antihypertensive treatment receive appropriate follow-up, in collaboration with the healthcare team.

2.9.6.Documentation

Document and share comprehensive information regarding hypertension management with the client and healthcare team

3. Materials and Methods

3.1. Materials

Laboratory Coat

Name Tag

Sphygmomanometer

Stethoscope

Weighing Balance

Height Measuring Rod

Checklist

Educational Material

Hospital Medical Record

JNC 8 Guideline

3.2. Study Setting

A Prospective Cohort Study approved by the Medical Centre's Ethics and Research Committee was carried out over a period of three months; February, March and April in 2017 at the

Outpatient Unit of the Obstetrics and Gynecology Department of Federal Medical Centre Katsina in Nigeria.

Federal Medical Centre Katsina is a tertiary health institution situated in the State Capital; it is one of the 22 Federal Medical Centers in Nigeria (www.health.gov.ng/index.php/parastatals/federal-medical-centres).

3.3. Study Population

Amongst 774 pregnant women on antenatal visit to the Outpatient Unit of the Obstetrics and Gynecology Department of Federal Medical Centre Katsina, 31 were qualified to be recruited to the study while 7 were excluded having one or more of the exclusion criteria; 24 were followed up to conclusion. Patients were asked for consent to participate in the study and recruitment started using the following Inclusion and Exclusion criteria.

3.4. Patient Education and Interventions

3.4.1. Knowledge on Pregnancy-Induced Hypertension: It is seen after 20 weeks of gestation with an elevated blood pressure above 140/90 mmHg without proteinuria and edema at baseline. Symptoms may include; swelling, sudden weight gain, blurred or double vision, abdominal pain, oliguria and altered liver and kidney functions. The condition can progress to preeclampsia which may further worsen to eclampsia; these in turn can harm the placenta, brain, liver and kidneys. Preeclampsia is the leading cause of maternal mortality, fetal growth retardation, premature delivery and death. Complications seen in eclampsia among others are seizures, coma and death.

3.4.2. Lifestyle Modification: These includes; smoking cessation, moderate alcohol consumption, getting enough sleep, avoiding stressful situations/conditions, controlling blood glucose and lipids, reduce sodium intake to no more than 2.4g/day, engaging in moderate to vigorous physical activity in at least 3-4 days of the week averaging 40 minutes per session, implementing the Dietary Approach to Stop Hypertension (DASH) diet. DASH diet should be; rich in proteins, fiber, calcium, potassium and magnesium elements; it also should be low in

sodium, saturated and trans fats. It has benefits of lowering sodium levels, blood pressure and also Low Density Lipoprotein (LDL).

3.4.3. Medication Adherence: sticking to drug regimen as advised which comprises of taking the exact medication, dose, strength, at the right frequency, time and duration.

3.4.4. Monitoring and Follow-up: All Interventions instituted should be monitored by further interviews, blood pressure measurements and phone calls.

3.4.5. Documentation: Each and every step is documented, data recorded and kept safely. Such documents are useful for continued patient follow-up and collaboration with other members of the health care team.

3.5. Inclusion Criteria

- $\geq$ 20 weeks gestation.
- $\geq$140/90 mmHg in at least two successive BP measurements at baseline.
- Devoid of proteinuria at baseline.

3.6. Exclusion Criteria

- Presence of proteinuria at baseline.
- Pedal Edema.
- Complications of Pre-eclampsia.

3.7. Data Collection

The Clinical Pharmacist wore a white laboratory coat with an attached name tag for easy identification for the interviews with participants at baseline and again on every routine follow-up visits. A Checklist containing questions related to demographic data (Age, Weight and Height), Parity, literacy level of patients, physical exercise, medication use, contraceptive use, knowledge about Pregnancy-Induced Hypertension, prior history of PIH amongst others was used and documented as well. Participants were then educated on; the disease and it's

complications, lifestyle modification, salt restriction, stress management, adherence to medications, and Blood Pressure control according to JNC 8 guidelines. Blood pressure measurement was repeated and updated at follow up visits and results were recorded on Microsoft Excel Sheet.

3.8. Statistical Analysis

Results of baseline and follow up Blood Pressures were compared and analyzed statistically using the SPSS 2.0 (Statistical Package for the Social Sciences). The results were expressed as mean $\pm$ standard deviations, percentage, frequencies with 95% Confidence Interval (CI), descriptive forms and tables. Blood Pressure results at baseline and follow-up after intervention were compared to see the significance of patient education on BP control using the Paired Student t-test and a p-value <0.05 was considered significant.

4. Results

724 pregnant women visited the research study health facility on participant recruitment days; 31 of which were found to have Pregnancy-Induced Hypertension (PIH) while 7 were excluded giving a total of 24 study participants within a period 3 months (February, March and April, 2017). This gives an incidence of 3.31% in the study period.

4.1. Demographics: Table (3) shows the demographic characteristics of pregnant women with PIH at baseline showed a good number of them were; above 30 years of age, 78kg weight, 160cm height, had more than 5 births and are at around the 34^{th} week of gestation.

Table 3. Demographics

	Mean	**S.D±**
AGE (years)	30.95	6.72
WEIGHT (kg)	78.66	20.29
HEIGHT (cm)	160.73	6.53
PARITY	5.62	3.62
GESTATIONAL AGE (Weeks)	34.66	2.76

S.D; Standard Deviation and N = 24

4.2. Contraceptive Use: In Table (4), some women could not explain the type of contraceptive they used while some started on a certain one and later switched to another. It was observed that half of the participants do not use contraceptives while the other half varied by use of various contraception methods.

Table.4. Contraceptive Use

	NUMBER	PERCENTAGE (%)
NO	12	50.0
YES/PILLS	3	12.5
YES/INJ	4	16.7
YES/INJ&PILLS	1	4.2
YES/IMPLANT	3	12.5
YES/LOOP	1	4.2
TOTAL	**24**	**100**

4.3. Physical Exercise: In Table 5, a good number of women assume their daily household chores are enough for daily physical exercise requirement. The highest duration was between 20-30 minutes per day physical exercise in the form of walk.

Table.5. Physical Exercise

TIME (mins)	NUMBER	PERCENTAGE (%)
NO	5	20.8

10 – 15	1	4.2
15 – 20	5	20.8
20 – 30	8	33.3
>30	5	20.8
TOTAL	**24**	**100**

4.4. Delivery Outcomes: The table below describes the different delivery outcomes and their percentages.

Table.6. Delivery Outcomes

	NUMBER	**PERCENTAGE (%)**
CS/PRETERM	2	8.3
CS/FULLTERM	2	8.3
ND/FULLTERM	17	70.9
INDUCTION/STILLBIRTH	1	4.2
INDUCTION/FULLTERM	2	8.3
TOTAL	**24**	**100**

CS; Caesarean- Section, ND; Normal Delivery

4.5. Pre and Post Intervention Blood Pressure: Shows the mean Systolic and Diastolic Blood Pressures ±Standard Deviation Pre and Post Intervention respectively.

Table.7. Pre and Post Intervention Blood Pressure

	Mean (mmHg)	**S.D±**

Pre-intervention		
Systolic	150.00	16.68
Diastolic	100.83	8.29
Post-intervention		
Systolic	142.70	17.75
Diastolic	93.95	8.72

S.D; Standard Deviation and N = 24

4.6.Paired T-test: Table (8) shows the association between Clinical Pharmacist's Intervention and Blood Pressure Control.

Table.8.Paired T-test

	Paired differences					
	Mean	Standard Deviation	Standard Error Mean	95% CI		Sig. (2-tailed)
				Lower	Upper	
Pair 1 Systole 1 – Systole 2	7.29	9.44	1.93	3.31	11.28	0.01
Pair 2 Diastole 1 – Diastole 2	6.88	10.20	2.08	2.57	11.18	0.03

CI; Confidence Interval

4.7. Results of other parameters

1. The Mean Arterial Pressures (MAP) calculated for pre and post intervention were 117.22 mmHg and 110.20 mmHg respectively.
2. Salt restriction; 50% had reduced use of salt, 25% do not use at all and further 25% used moderately.
3. Previous history of Pregnancy-Induced Hypertension; 50% had a prior history while the other 50% never had.
4. Relative with PIH; 67% had no relative with PIH while the remaining 33% had at least a relative with PIH.

5. Knowledge on PIH; 67% do not have any knowledge of PIH while 33% had some knowledge even though not adequate (probably because they had a previous history of the condition).
6. Drugs Used: The most commonly used medications were Methyldopa 250mg and Low dose Aspirin 75mg which was seen in 87.5% of patients while others were not on any medication.
7. Patients with the highest Blood Pressure at baseline mostly were those with Chronic Hypertension and/or a history of previous Pregnancy-Induced Hypertension.

5. Discussion

Pregnancy-Induced Hypertension (PIH) is a common cause of morbidity and mortality amongst pregnant women; it affects 5-8% of pregnant women globally (Arshad A et al, 2011). Insufficient resources and poor knowledge of the management of PIH poses a huge hazard towards adequate control of this burden (Muti M et al, 2015). Although Incidence of PIH 3.31% was low; improved PIH knowledge amongst participants was achieved; they knew the basic symptoms of the condition, disease progression and its complications. A similar studies carried out in India showed a comparable Incidence of 3.8% for PIH (Hiralal K et al, 2006). In some works higher incidence of PIH was seen in younger mothers of less than 22 years (Goonewardene et al, 2005 and Manjusha S et al, 2015) while in the contrary, an incidence of 9.73% was seen in a six months study carried out in a multi-specialty hospital in Chennai, India which also observed an improved blood pressure control as a positive impact of Pharmacist's intervention (Lavanya et al, 2015). They also learnt the lifestyle changes which help in lowering blood pressure amongst which are; smoking cessation, moderate alcohol intake, daily salt intake of between 1,500mg – 2,300mg, regulating fats and sugar consumption, good sleep and stress management engaging in moderate to vigorous physical activity of about 40 minutes in 3-4 days of the week and implementing the DASH (Dietary Approach to Stop Hypertension) diet. DASH diet is; food low in saturated and Trans fats, rich in potassium, calcium, magnesium, fiber and protein and last low in sodium this contributes immensely in lowering of sodium, Low Density Lipoproteins (LDL) and ultimately the blood

pressure (JNC 8 Guidelines). Participants in this study were all non-smokers and do not consume alcohol as such that gave us a good control in that aspect. As for other dietary recommendations; it was difficult to get good control due to their low economic income. With all the limitations encountered a decrease in systolic as well as diastolic blood pressure was observed post intervention. It is also worth mentioning that majority of the participants were happy with their interaction with the Clinical Pharmacist's follow up with phone calls to monitor and remind them of the education given at baseline before their next follow up visits, some called back to ask questions on their medications. Indeed it was a good established Patient-Pharmacist relationship and acceptance from patients. The sole aim of pharmaceutical care is the Pharmacist partnering with the individual patient to customize his/her treatment or management plan and thereafter monitoring therapy; this was maximally achieved in this study especially because of the low number of participants. Great emphasis on medication adherence was given to patients; the time and frequencies of their medications, to take them before or after meals, discouraging use of herbal and over the counter medications and to refill their medications before the run out of them.Though the JNC 8 guidelines recommends the use of Labetalol as a first line drug for managing hypertension in pregnancy, it was observed in the study setting thatthe conditionwas being managed with Methyldopa and low dose Aspirin probably because of the maternal and fetal safety seen in evidence based medicine (Redman et al, 1977) of the former and the protective property in High risk of preeclampsia patients of the latter (Henderson JT et al, 2014).

6. Study limitations

- **Duration of the Study;** the three months carried out was not adequate to get a representation of the true population.
- **Study Setting and Population size;** Federal Medical Centre Katsina is a tertiary institution which ideally is supposed to be a referral facility. More patients and especially those with PIH will probably be found in a primary health care facility which is the first point of call of the general population and is more accessible.

- **Large Patient Load against medical personnel;** as the Clinical Pharmacist relied on the hospital personnel to single-out patients with high Blood Pressure reading from the whole lot of pregnant women before further screening them. The number of patients seen are too many as against the number of nurses as such; some could be missed due to high work load.

7. Conclusion and Recommendations

From the strong positive association between Intervention and reduction in Blood Pressure, it can be inferred that; "the Clinical Pharmacist played a positive role in the management of the Pregnancy-Induced Hypertensive patients mainly from the Patient education and monitoring. It is recommended that the study setting and others alike should engage and partner with Clinical Pharmacists in the management of Pregnancy-Induced Hypertensive cases. Also, it is suggested that Doctors in the study setting should explore manage their patients according to the JNC 8 Guidelines of using Labetalol as a first line treatment for pregnant women with hypertension rather than the conventional Methyldopa together with Low dose Aspirin. The hospital should also improve on measuring and documenting important parameters to allow for availability of data for retrospective research work which could be compared with retrospective ones. To form a base for the recommendations stated above, it is suggested that same study be carried out in a larger population particularly in a Primary Health Care Centre.

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Appendix I; Figure 1. Age Variation

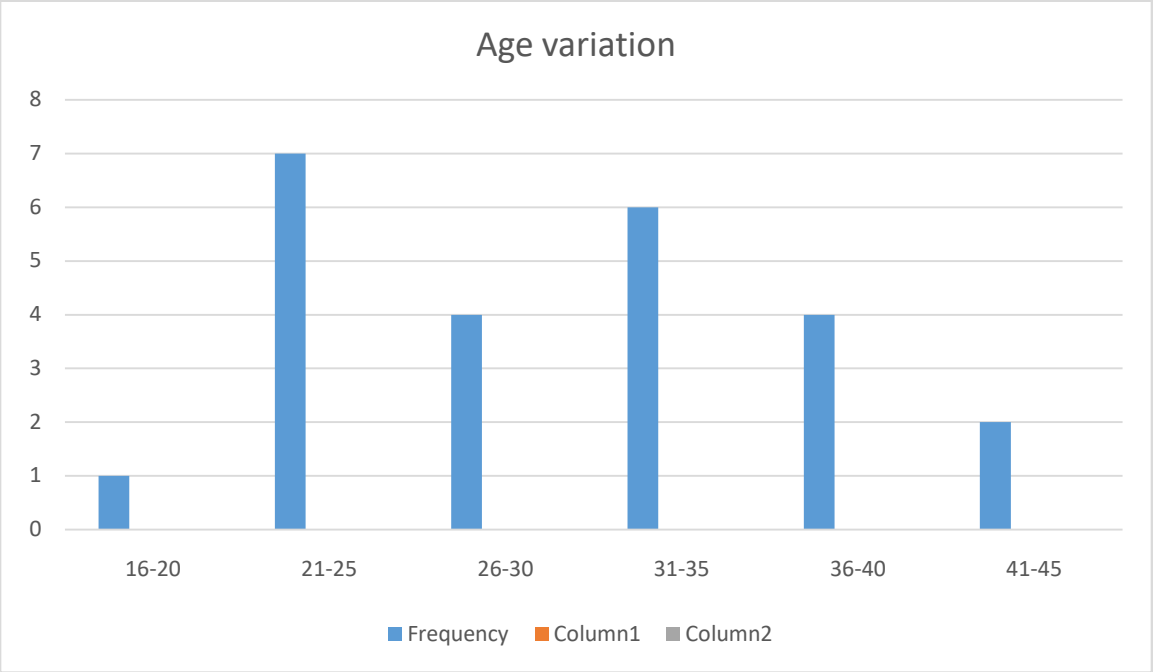

Figure 1. Illustrates the age distribution of the study participants.

Figure 2. Literacy Level

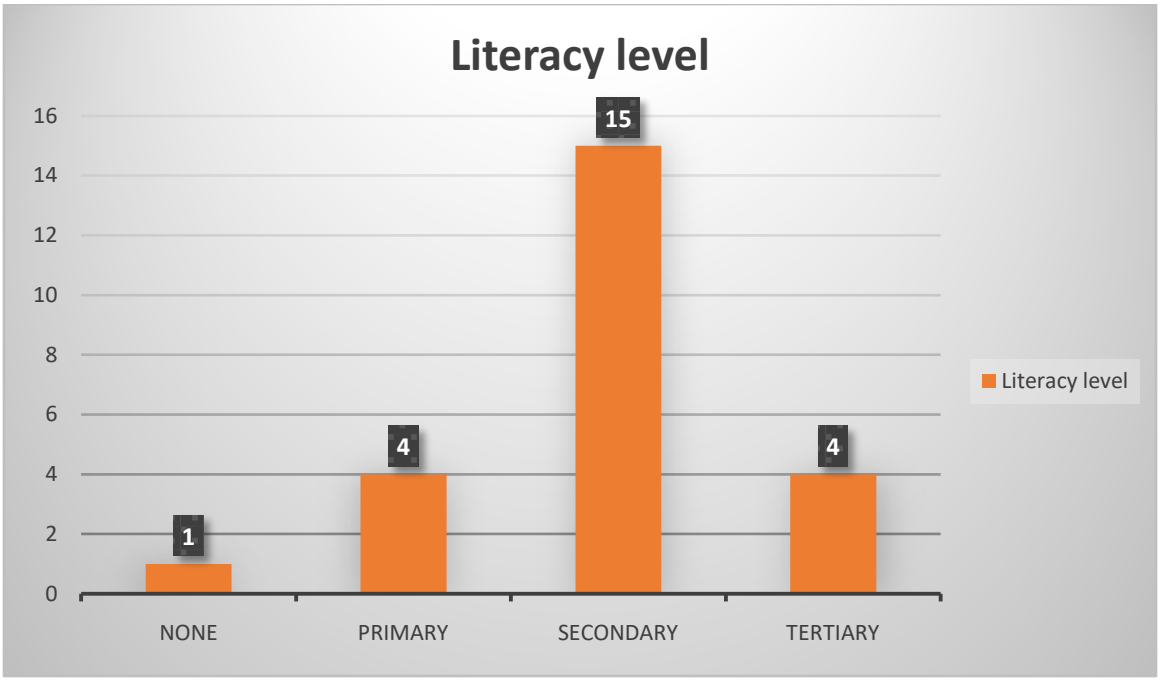

Figure 2. A Bar Chart describing distribution of literacy level.

Appendıx II.

CHECK LIST.

I. DEMOGRAPHICS

Age

Weight

Height

Level of literacy

Gestational age and type

Comorbidities

Baseline BP

Others; Name and phone number

II. KNOWLEDGE ON PREGNANCY-INDUCED HYPERTENSION (PIH)

1. Do you have a prior knowledge about Pregnancy Induced Hypertension, symptomsand its complications?
2. Do you have any cultural view on PIH?
    a. If yes, does it have any effect on your present perception about PIH?

3. Are you on any diet and salt restriction?
    a. If yes, how well do you comply?
4. How much rest and exercise do you do?
5. Are you on any medication(s) for PIH, Prescriptions, OTCs and Herbal medications?
6. How regular do you take your medications (drug specific and frequency)?
7. Do you experience any side-effect(s) from taking these medications?
    a. If yes, have you discussed this with the Physician?
8. How regular are you on your scheduled antenatal visits?
9. Which one of the following risk factors relates to you?
    - Age less than 20 years,
    - Age more than 40 years,
    - History of Chronic Hypertension,
    - History of previous PIH,
    - Have a female relative with a history of PIH,
    - Underweight or Overweight,
    - Diabetes prior to pregnancy,
    - Have an immune system disorder e.g. lupus or rheumatoid arthritis,
    - Kidney disease,
    - History of alcohol, drug or tobacco use,
    - Expecting twins or triplets.
    - Have you used any contraceptive method?
    - If yes, what type of contraceptive method?

T.R.N.C

NEAR EAST UNIVERSITY
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Master of Science in Clinical Pharmacy

Advisor

Assoc. Prof. Dr. Bilgen BASGUT

NICOSIA 2017

Dedication

This work is dedicated to my parents, husband, siblings, twodaughters;and toall women who were/are predisposed, suffered, survived and died from Pregnancy-Induced Hypertension (PIH). I pray for an improved global approach to preventing and managing PIH.

Approval

Thesis submitted to the Institute of Health Sciences of Near East University in partial fulfillment of the requirements for the degree of **Master of Science in Clinical Pharmacy.**

Thesis Committee:

Chair of the committee:**Prof. Dr. NurettinAbacıoğlu**

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Director of Health Sciences Institute

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List of Abbreviations

S/No.	Abbreviations	Meaning
1.	PIH	Pregnancy-Induced Hypertension
2.	WHO	World Health Organization
3.	FMC	Federal Medical Centre
4.	ACOG	American College Of Obstetrics and Gynecology
5.	BP	Blood Pressure
6.	JNC	Joint National Committee
7.	SPSS	Statistical Program for Social Science
8.	CI	Confidence Interval
9.	CIU	Cyprus International University
10.	ACOG	American College of Obstetricians and Gynecologists
11.	ISSHP	International Society for the Study of Hypertension in Pregnancy
12.	RCOG	Royal College of Obstetricians and Gynecologists
13.	NICE	National Institute for Health Care Excellence
14.	eNOs	Endothelial Nitric Oxide Synthase
15.	BH4	Cofactor Tetrahydrobiopterin
16.	HELLP	Hemolysis Elevated Liver function enzymes and Low Platelets
17.	LDL	Low-Density Lipoprotein
18.	PTB	Pre-Term Birth
19.	Wt	Weight
20.	Ht	Height
21.	PI	Ponderal Index
22.	BMI	Body Mass Index
23.	THR	Thigh circumference/ Head circumference Ratio
24.	WHR	Waist-to-Hip Ratio
25.	MAC/OFC	Mid-Arm Circumference to Occipito-Frontal Circumference
26.	W/OFC	Weight to Occipito-Frontal Circumference
27.	W/L	Weight/Length
28.	OR	Odds Ratio

29.	**ACEI**	**Angiotensin Converting Enzyme Inhibitor**
30.	**ARB**	**Angiotensin II Receptor Blocker**
31.	**DASH**	**Dietary approaches to Stop Hypertension**
32.	**CS**	**Caesarean Section**
33.	**ND**	**Normal Delivery**

Abstract

The study evaluated prospectively for a period of 3 months (February to April, 2017)the effects of Clinical Pharmacist's Intervention on Blood Pressure control in a cohort of Pregnancy-Induced Hypertensive women visiting the outpatient unit of the Obstetrics and Gynecology Department of Federal Medical Centre Katsina. The aim of the study was to educate patients on the disease, lifestyle modification and adherence to medications; evaluate the impact of patient education on Blood Pressure control and recommend ways in which Pharmacist's intervention will reduce morbidities and mortalities from Pregnancy-Induced Hypertension.Pregnancy-Induced Hypertension otherwise referred to as Gestational Hypertension is a newly diagnosed hypertension after 20 weeks of gestation, devoid of proteinuria and other signs of Pre-eclampsia.This shows the need to involve the Clinical Pharmacist in managing Pregnancy-Induced Hypertensive patients in such a clinical setting. Mean $\pm$ SD results for demographics were found to be 30.95years $\pm$ 6.72, 78.66kg $\pm$ 20.29, 160.73cm $\pm$6.53, 5.62 $\pm$ 3.62, 34.66 weeks $\pm$ 2.76 for Age, Weight, Height, Parity and Gestational Age respectively. Patients in 21-25 years age bracket, Secondary School level of Education, and between 20-30 minutes daily physical activity showed highest percentages of 29.17%, 62.5% and 33.3% respectively. The Mean Pre-Intervention Systolic and Diastolic Blood Pressures were 150.00 mmHg $\pm$ 16.68 and 100.83 mmHg $\pm$ 8.29 respectively, while Post-Intervention the Systolic and Diastolic values were 142.70 mmHg $\pm$ 17.75 and 93.95 mmHg $\pm$ 8.72 respectively. The Paired T-test for Pre Systolic – Post

Systolic BP was 7.29 mmHg ± 9.44 while that of Pre Diastolic – Post Diastolic BP was 6.88 mmHg ± 10.20; showing a positive association between the Intervention and Blood Pressure control.

Keywords – Clinical Pharmacist's Intervention, Pregnancy-Induced Hypertension, Federal Medical Centre Katsina, Blood Pressure Control, Delivery Outcome.

OZET

Table of Contents

List of tables

Tables **Page**

List of figures