Persistent Hypertension After Puerperirrum Among Women With Pre-Eclampsia in Wad Medani Obstetrics and Gynecology Teaching Hospital, Gezira State, Sudan (2014)

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A thesis submitted to University of Gezira in Partial Fulfillment for the Requirement of the Degree of Clinical Doctorate in Obstetrics and Gynecology

Department of Obstetrics and Gynecology

Faculty of Medicine

University of Gezira

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Persistent Hypertension After Puerperium Among Women
With Pre-Eclampsia in Wad Medani Obstetrics and Gynecology
Teaching Hospital, Gezira State, Sudan (2014)

Dr. Zubaida Moftah El Kheir Fadalallah

Supervisor Committee:

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Date of examination: 5/9/2016
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Date of examination: 5/9/2016
قال تعالى:

(اقرأ باسم ربك الذي خلق).

صدق الله العظيم

سورة العلق الآية (1)
Dedication

This work is dedicated to

The spirit of my dear mother

My father….

my family who offered me financial and moral support.

This thesis is dedicated to all those who supported me in this study.
ACKNOWLEDGEMENT

Thanks to Allah for giving me this opportunity and strength to complete this thesis.

I would like to express my thanks and gratitude to professor Elhassan Mohamed Elhassan and professor Ishag Adam.

I would like to thank the considerable help of my colleagues, the hospital staff, the library staff and my family.
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<td>ALT</td>
<td>Alanine aminotransferase.</td>
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<td>ANC</td>
<td>Antenatal care.</td>
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<td>AST</td>
<td>Aspartate aminotransferase.</td>
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<tr>
<td>BP</td>
<td>Blood Pressure.</td>
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<td>BM</td>
<td>Body mass index.</td>
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<td>BUN</td>
<td>Blood urea nitrogen.</td>
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<td>CBC</td>
<td>Complete blood count.</td>
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<td>C/S</td>
<td>Caesarean section.</td>
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<td>DM</td>
<td>Diabetes mellitus.</td>
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<td>GFR</td>
<td>Glomerular filtration rate.</td>
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<td>GH</td>
<td>Gestational hypertension.</td>
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<td>HCG</td>
<td>Human chorionic gonadotrophin.</td>
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<tr>
<td>HELLP</td>
<td>Hemolysis, elevated liver enzymes, low platelet.</td>
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<tr>
<td>IDDM</td>
<td>Insulin dependent diabetes mellitus.</td>
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<tr>
<td>IM</td>
<td>Intramuscular.</td>
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<tr>
<td>IUGR</td>
<td>Intrauterine growth restriction.</td>
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<td>IV</td>
<td>Intra-venous.</td>
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<td>LDH</td>
<td>Lactate dehydrogenase.</td>
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<td>PCR</td>
<td>Polymerase chain reaction.</td>
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<td>PIGF</td>
<td>Placental insulin growth factor.</td>
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<td>sFLt1</td>
<td>Like tyrosine kinase-1.</td>
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<tr>
<td>SGA</td>
<td>Small for gestational age.</td>
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Abstract

Chronic hypertension accounts for approximately 20% of the cases of high blood pressure seen in pregnancy. To determine the occurrence and significance of persistent hypertension after puerperirum, and to determine the factors that provoke persistent hypertension, to correlate between antipartum, postpartum hypertension and persistent hypertension, and to determine the relation between the gestational age, the mode of delivery and the severity of the persistent hypertension. This study is a prospective, cross-sectional descriptive hospital based to assess Persistent hypertension after puerperirum among women with pre-eclampsia at Wad Madani obstetrics and gynaecology teaching hospital from (Jan 2014 - Dec 2014). The study population was all women came to deliver in Wad Medani Teaching hospital and diagnosed as pre-eclampsia. Data was collected in questionnaire containing all relevant information and analyzed by computer program (Excel). The rate of persistent hypertension in this study is 28.3%. The risk factors are age, parity, obesity history of previous pre-eclampsia, family history of pre-eclampsia, body mass index (BMI), Blood pressure and Proteinuria. Regarding the parity [78.8%] were multiparae, The incidence increases among multiparous women the explanation not known. Gestational age distribution in this study was [69.1%] at gestational age 36wks to 39wks, most of women had no ANC [53.9%], the percentage of women had ANC is [46.1%]. It’s important to follow women at preconception period. Most of women had no antenatal care, so this why most of them diagnosed in the late gestational age. Obesity as a risk factor for pre-eclampsia, this is quite clear in our study as [60.6%] were overweight. The occupation in this study [81.2%] was house wife, the rate of patient to developed persistent hypertension is increased in primary school [79.4%]. Women with severe pre-eclampsia/eclampsia are at risk of developing chronic hypertension in future. Regarding serum uric acid level most of patients had high level more than 7 gm\dl [40.6%]. The study assessed the incidence of persistent
hypertension and factors associated with persistent hypertension in patients with pre-eclampsia/eclampsia. About the mode of delivery, most of women delivered by vaginaly [55.2%]. IUGR is diagnosed when a fetus fails to achieve its growth potential in utero. It is usually associated with a small for gestational age (SGA) fetus, in this study [67.9%] of the babies weight equal or less than 2500 gram.

There is a need to coordinate and introduce antenatal care programme, health education and organized training on diagnosis, management and care of patients antipartum and postpartum and by improving knowledge and skills of providers.
فرط ضغط الدم المستمر لما بعد النفاس في حالات مقدمة الارتعاج في مستشفى النساء والتوليد ومدني، ولاية الجزيرة، السودان (2014)

زيادة مفتاح الخير فضل الله

ملخص الأطروحة

هذه الدراسة دراسة وصفية قطعية بالمستشفى تهدف لتقييم خدمات الرعاية والكشف المبكر للنساء الحوامل في مستشفى ومدني لأمراض النساء والتوليد (السودان) في الفترة من يناير إلى ديسمبر 2014، وتحديد معدل الإصابة بارتفاع ضغط الدم والطرق المستعملة في العلاج. تقديم النصح والمشورة ومعرفة مدى استدامة ارتفاع ضغط الدم بعد انقضاء فترة النفاس. أجريت علي المريضات اللائي حضرن إلى الحوادث لمتابعة الحمل بعد موافقتهن. وتم جمع المعلومات باللقاءات المباشرة عن طريق استبيان معد مسبقا وقد تم تحليل المعلومات عن طريق برامج التحليل الإحصائي. نسبة ارتفاع ضغط الدم بعد انقضاء فترة النفاس في هذه الدراسة 28.3% أهم العوامل المؤثرة هي عمر الأم والعمر الحملي وزنها وعدد مرات الحمل. هناك حاجة لتنسيق وإدخال برامج متابعة الحمل وتنظيم دورات تدريبية حول الرعاية بعد الولادة وتحسين معرفة ومهارة مقدمي الخدمات.
Introduction and literature review

Hypertensive disorders of pregnancy are a major cause of maternal mortality and morbidity, especially in developing countries [1]. Hypertension may be present before or during pregnancy or postpartum [2]. Postpartum hypertension can be related to persistence of gestational hypertension (GH), pre-eclampsia, or preexisting chronic hypertension or it could develop de novo secondary to other causes [3]. During the past decades, there has been extensive research regarding the incidence, risk factors, pathogenesis, prediction, prevention, and management of pre-eclampsia[4]. However, patients who were readmitted with postpartum hypertension-pre-eclampsia were not considered in reported studies [2, 4]. In addition, the available data in the medical literature have primarily focused on antenatal and peripartum management of such patients [4,5], even though some patients can develop de novo eclampsia and hemolysis, elevated liver enzymes, and low platelet (HELLP) syndrome in the late postpartum period [6,7, 8, 9]. Thus, there are few data regarding the evaluation, management, and complications in women who are rehospitalized with diagnosis of postpartum hypertension [3, 10,11]. Therefore, this report will focus on the prevalence, etiology, evaluation and management of women who have de novo or persistent postpartum hypertension. Postpartum hypertension can be related to persistence of gestational hypertension, pre-eclampsia, or preexisting chronic hypertension or it could develop de novo postpartum secondary to other causes.

There are limited data describing the etiology, differential diagnosis and management of postpartum hypertension. The differential diagnosis is extensive, and varies from benign (mild gestational or essential hypertension) to life-threatening such as severe pre-eclampsia, eclampsia, pheochromocytoma, and cerebrovascular accidents. Therefore, medical providers caring for postpartum women should be educated about continued monitoring of signs and symptoms and prompt management of these women in a timely fashion. Evaluation and management should be performed in a stepwise fashion and may require a multidisciplinary approach that considers predelivery risk factors, time of onset, associated signs/symptoms, and results of selective laboratory and imaging findings. The objective of this review is to increase awareness and to provide a stepwise approach toward the diagnosis and management of women with persistent and/or new-onset hypertension, pre-eclampsia postpartum period.
The exact incidence of postpartum hypertension is difficult to ascertain. In clinical practice, most women will not have their blood pressure (BP) checked until the 6 weeks' postpartum visit in physician's offices or in postpartum clinics. As a result, women with mild hypertension who are asymptomatic are usually not reported. In addition, postpartum women who have hypertension in association with symptoms such as headaches or blurred vision are often seen and managed in the emergency department and will not be coded as hypertensive unless they are hospitalized.

Risk factors that can be assessed at booking are age, parity, history of previous pre-eclampsia, family history of pre-eclampsia, Multiple pregnancy, pre-existing medical conditions: insulin dependent diabetes (IDDM), chronic hypertension, renal disease, autoimmune disease, antiphospholipid syndrome, time between pregnancies, body mass index (BMI), Blood pressure and Proteinuria.

**Definition of hypertension in pregnancy:**

Normal pregnancy is characterized by a fall in blood pressure, detectable in the first trimester and usually reaching a nadir in the second trimester. Blood pressure rises towards pre-conception levels by term.

Hypertension in pregnancy is defined as:

- Systolic blood pressure greater than or equal to 140 mmHg and/or Diastolic blood pressure greater than or equal to 90 mmHg (Korotkoff 5).

These measurements should be confirmed by repeated readings over several hours. Elevations of both systolic and diastolic blood pressures have been associated with adverse maternal and fetal outcome and therefore both are important [21]

**Classification of hypertensive disorders in pregnancy:**

This classification of the hypertensive disorders in pregnancy reflects the pathophysiology of the constituent conditions as well as the risks and potential outcomes for both mother and baby. The classification is as follows:

1- Chronic hypertension.
2- Gestational hypertension.
3- Preeclampsia – eclampsia.
Chronic Hypertension

Chronic hypertension is defined as a blood pressure measurement of 140/90 mm Hg or more on two occasions before 20 weeks of gestation or persisting beyond 12 weeks postpartum. Treatment of mild to moderate chronic hypertension neither benefits the fetus nor prevents pre-eclampsia. Excessively lowering blood pressure may result in decreased placental perfusion and adverse perinatal outcomes. When a patient's blood pressure is persistently greater than 150 to 180/100 to 110 mm Hg, pharmacologic treatment is needed to prevent maternal end-organ damage [12]. Important secondary causes of chronic hypertension in pregnancy include:
- Chronic kidney disease e.g. glomerulonephritis, reflux nephropathy and adult polycystic kidney disease.
- Renal artery stenosis.
- Systemic disease with renal involvement e.g. diabetes mellitus and systemic lupus erythematosus.
- Endocrine disorders e.g. phaeochromocytoma, Cushing’s syndrome and primary hyperaldosteronism.
- Coarctation of the aorta.

In the absence of any of the above conditions it is likely that a woman with high blood pressure in the first half of pregnancy has essential hypertension. Women with chronic hypertension require careful monitoring during pregnancy as they have an increased risk of adverse events, including superimposed pre-eclampsia, placental abruption, fetal growth restriction, premature delivery and stillbirth [13].

Pre-pregnancy counseling and management of chronic hypertension is essential. Some commonly prescribed antihypertensive drugs are contraindicated or best avoided before conception and during pregnancy. These include ACE inhibitors and angiotensin receptor antagonists cause fetal renal dysfunction and oligohydramnios, diuretics cause fetal electrolyte disturbances, reduction in maternal blood volume [13,14]. Where indicated, it’s advisable to look for secondary causes of hypertension before conception, as normal physiological changes in pregnancy can make many of these screening tests difficult to interpret. In all cases, preconception assessment for proteinuria [with urine protein: creatinine ratio] is recommended as a base line measurement.
Pre-eclampsia superimposed on chronic hypertension

Pre-existing hypertension is a strong risk factor for the development of pre-eclampsia [15,16]. Superimposed pre-eclampsia is diagnosed when a woman with chronic hypertension develops one or more of the systemic features of pre-eclampsia after 20 weeks gestation. Similarly, SGA occurs more frequently in women with chronic hypertension and evidence of fetal effect other than SGA eg. Oligohydramnios or abnormal uterine artery Doppler flows is required to diagnose superimposed pre-eclampsia [14]

Investigation of new onset hypertension after 20 weeks gestation

Any woman presenting with new hypertension after 20 weeks gestation should be assessed for signs and symptoms of pre-eclampsia. If features of pre-eclampsia are detected, admission to hospital is indicated. The presence of severe hypertension, headache, epigastric pain, oliguria or nausea and vomiting are ominous signs which should lead to urgent admission and management, as should any concern about fetal wellbeing [17,18].The following investigations should be performed in all women with new onset hypertension after 20 weeks gestation:
- Full blood count
- Creatinine, electrolytes, urate.
- Urinalysis for protein and urine microscopy on a carefully collected mid-stream urine sample.
- If there is thrombocytopenia or a falling haemoglobin, investigations for disseminated intravascular coagulation and/or haemolysis (coagulation studies, blood film, LDH, fibrinogen) are indicated.
- Patients with severe, early onset preeclampsia warrant investigation for associated conditions e.g. systemic lupus erythematosus, underlying renal disease or antiphospholipid syndrome. The timing of these investigations will be guided by the clinical features.

Subsequent investigation and management will be based on the results of ongoing blood pressure measurement and these investigations [19, 20].

Ongoing investigation of women with hypertension in pregnancy

At each assessment following the detection of hypertension in pregnancy, the clinician should systematically review the woman’s symptoms, examination, laboratory investigations and fetal wellbeing.
Treatment of mild to moderate chronic hypertension neither benefits the fetus nor prevents pre-eclampsia [22, 24]. Excessively lowering blood pressure may result in decreased placental perfusion and adverse perinatal outcomes [25]. When a patient's blood pressure is persistently greater than 150 to 180/100 to 110 mm Hg, pharmacologic treatment is needed to prevent maternal end-organ damage [12, 22, 24, 26].

Methyldopa, labetalol, and nifedipine (Procardia) are oral agents commonly used to treat chronic hypertension in Pregnancy. Angiotensin-converting enzyme inhibitors and angiotensin-II receptor antagonists are not used because of teratogenicity, intrauterine growth restriction (IUGR), and neonatal renal failure [24]. The beta blocker atenolol (Tenormin) has been associated with IUGR [23], and thiazide diuretics can exacerbate intravascular fluid depletion if superimposed pre-eclampsia develops. Women in active labor with uncontrolled severe chronic hypertension require treatment with intravenous labetalol or hydralazine [27].

Morbidity occurs primarily from superimposed pre-eclampsia or IUGR [24]. A sudden increase in blood pressure, new proteinuria, or signs and symptoms of severe pre-eclampsia indicate superimposed pre-eclampsia. Fetal growth may be assessed by serial fundal height measurements supplemented by ultrasonography every four weeks starting at 28 weeks of gestation [24].

**Gestational Hypertension**

Gestational hypertension is usually defined as having a blood pressure higher than 140/90 measured on two separate occasions, more than 6 hours apart, without the presence of protein in the urine and diagnosed after 20 weeks of gestation [22]. Gestational hypertension has replaced the term pregnancy-induced hypertension to describe women who develop hypertension without proteinuria after 20 weeks of gestation [28].

The underlying cause of gestational hypertension uncommonly believed to be an improperly implanted placenta. Humans have evolved to have a very invasive placenta to facilitate better oxygen transfer from the mother to the fetus, to support the growth of its large brain [30].

The origins of gestational hypertension may lie with the development of humans’ hemochorial placenta. A hemochorial placenta optimizes the amount of oxygen and nutrients that can be absorbed into the fetal blood supply, while at the same time ensuring rapid diffusion of wastes away from the fetus. This hemochorial placenta differs from lower primates’
epitheliochorial placenta in the way that it allows the fetal tissues to interact directly with the mother’s blood. The hemochorial placenta thereby promotes more rapid diffusion to and from the fetal blood supply [31]. Gestational hypertension is a provisional diagnosis that includes women eventually diagnosed with pre-eclampsia or chronic hypertension, as well as women retrospectively diagnosed with transient hypertension of pregnancy [46].

Expectant management of mild gestational hypertension can reduce the increased rate of cesarean delivery associated with the induction of nulliparous women who have an unripe cervix [47]. Women who progress to severe gestational hypertension based on the degree of blood pressure elevation have worse prenatal outcomes than do women with mild pre-eclampsia, and require management similar to those with severe pre-eclampsia[48].

The fetal cells that implant into the uterine wall are known as the trophoblast. The hemochorial placenta bathes the fetal trophoblast in maternal blood by forming lacunae, or lakes, of the mother’s blood that surround fetal tissue. The lacunae are filled by the spiral arteries, which mean that the mother’s blood pressure is the driving force behind the introduction of new blood, which contains both oxygen and food for the fetus to the system [33].

Despite these risks for gestational hypertension, the hemochorial placenta has been favored because of its advantages in the way that it aids in diffusion from mother to fetus later in pregnancy. The bipedal posture that has allowed humans to walk upright has also led to a reduced cardiac output, and it has been suggested that this is what necessitated humans’ aggressive early placental structures [42]. Increased maternal blood pressure can attempt to make up for lower cardiac output, ensuring that the fetus’s growing brain receives enough oxygen and nutrients [41]. The benefits of being able to walk upright and run on land have outweighed the disadvantages that come from bipedalism, including the placental diseases of pregnancy, such as gestational hypertension. Similarly, the advantages of having a large brain size have outweighed the deleterious effects of having a placenta that does not always convert the spiral arteries effectively, leaving humans vulnerable to contracting gestational hypertension.

Gestational hypertension in the early stages of pregnancy has been shown to improve the health of the child both in its first year of life, and its later life [44]. However, when the disease develops later in the pregnancy, or turns into pre-eclampsia, there begin to be detrimental health effects for the fetus, including low birth-weight [29].
It has been proposed that fetal genes designed to increase the mother’s blood pressure are so beneficial that they outweigh the potential negative effects that can come from pre-eclampsia [44]. It has also been suggested that gestational hypertension and pre-eclampsia have remained active traits due to the cultural capacity of humans, and the tendency for midwives or helpers to aid in delivering babies [45]. Expectant management of mild gestational hypertension can reduce the increased rate of cesarean delivery associated with the induction of nulliparous women who have an unripe cervix [47]. Women who progress to severe gestational hypertension based on the degree of blood pressure elevation have worse prenatal outcomes than do women with mild pre-eclampsia, and require management similar to those with severe pre-eclampsia.

**Pre-eclampsia**

Is a multi-system disorder unique to human pregnancy characterized by hypertension and involvement of one or more other organ systems and/or the fetus. Pre-eclampsia is gestational hypertension plus proteinuria (>300 mg of protein in a 24-hour urine sample). Raised blood pressure is commonly but not always the first manifestation. Proteinuria is the most commonly recognized additional feature after hypertension but should not be considered mandatory to make the clinical diagnosis. As this classification is based on clinical data, it is possible that women with another condition will sometimes be classified incorrectly as having pre-eclampsia during pregnancy. This is not usually a clinical problem as the diagnosis of pre-eclampsia should lead to increased observation and vigilance which is appropriate for conditions which may mimic pre-eclampsia.

A diagnosis of pre-eclampsia can be made when hypertension arises after 20 weeks gestation and is accompanied by one or more of the following signs of organ involvement:

**Renal involvement**
- Oliguria:<80mL/4hr.
- Significant proteinuria –a spot urine protein/creatinine ratio ≥ 30mg/mmol.
- Serum or plasma creatinine > 90 μmol/L.
- hyperuricemia.

**Haematological involvement**
- Thrombocytopenia <100,000 /μL.
- Haemolysis: schistocytes or red cell fragments on blood film, raised bilirubin, raised lactate dehydrogenase >600mIU/L, decreased haptoglobin.
- Disseminated intravascular coagulation.

**Liver involvement**
epigastric and/or right upper quadrant pain.

**Neurological involvement**
- convulsions (eclampsia).
- Hyporeflexia with sustained clonus.
- Persistent, new headache.
- Persistent visual disturbances (photopsia, scotomata, cortical blindness, posterior reversible encephalopathy syndrome, retinal vasospasm).
- Stroke.
- Pulmonary oedema.
- Intrauterine growth restriction (IUGR).

**Controversies in classifying the severity of pre-eclampsia**

A number of features of pre-eclampsia are recognized to significantly increase the risk of adverse maternal and fetal outcomes and are sometimes used to classify severe pre-eclampsia [48,49,51, 52]. The natural history of pre-eclampsia is to progress at an unpredictable rate, at least until delivery. Therefore all women with pre-eclampsia should be closely monitored. The classification of severe pre-eclampsia would ideally allow the identification of those women and babies at increased risk of adverse maternal/fetal outcomes and/or requiring more intensive monitoring and/or treatment. A number of classification systems and surveys have attempted to identify which features are predictive. One study reported that features that had previously been recommended as indicators of severe disease were neither sensitive nor specific in identifying women and/or babies at particular risk [53]. The recent ISSHP statement suggested there was general consensus that factors determining severity include difficulty in controlling blood pressure and deteriorating clinical condition including HELLP syndrome, impending eclampsia, worsening thrombocytopenia or worsening fetal growth restriction while there is less concern regarding increasing proteinuria [48]. Alterations in circulating angiogenic factors are pathophysiologically important in the development of preeclampsia and may have a potential role in diagnosis [54]. Changes in these angiogenic factors are detectable both prior to and at the time of onset of hypertension in women with pre-eclampsia. Other rare disorders may present with some of the features of pre-eclampsia [50]. Disorders such as acute fatty liver of pregnancy,
haemolytic uremic syndrome, and thrombotic thrombocytopenic purpura, exacerbation of systemic lupus erythematosus or cholecystitis may need to be excluded.

Rarely pre-eclampsia presents before 20 weeks gestation; usually in the presence of a predisposing factor such as hydatidiform mole, multiple pregnancy, fetal triploidy, severe renal disease or antiphospholipid antibody syndrome.

The measurement and interpretation of proteinuria in hypertensive disorders of pregnancy has been recently reviewed [57,58]. Dipstick testing is not accurate to confirm or exclude significant proteinuria (≥300mg/24 hours): sensitivities of 22-82% have been reported [59-62]. This is improved slightly with automated dipstick testing but even this will miss more than half the patients with significant proteinuria [63,64]. The presence of 2+ or 3+ proteinuria or repeated +1 dipstick testing increases both sensitivity and specificity and, therefore, should be assumed to represent significant proteinuria until proven otherwise by confirmatory tests.

Twenty four hour urine protein has been the historic gold standard for quantifying proteinuria in pregnancy although its accuracy is affected by numerous factors such as adequacy and accuracy of collection and variations in protein excretion.

A spot urine protein/creatinine cut-off level of 30 mg/mmol equates to a 24-h urine protein >300 mg per day and at this level has adequate sensitivity and specificity to be used as a ‘rule out’ value below which true proteinuria is unlikely to be present [65]. This is the recommended method and cut-off for diagnosing proteinuria in pregnancy.

In practise, dipstick testing is simple, cheap and an appropriate screening test but spot urine PCR is recommended for confirmation or exclusion of proteinuria when pre-eclampsia is suspected [65].

In women with underlying renal disease, particularly with pre-existing hypertension, the interpretation of proteinuria is difficult and pre-eclampsia should not be diagnosed until other features are present.

Serum/plasma creatinine usually falls in normal pregnancy and levels even at the upper end of the normal range (70-100 umol/L) may indicate impaired renal function. Other guidelines have used cut-offs up to >100-110umol/L to indicate renal impairment in pre-eclampsia [49, 55, 56, 57]. Serum/plasma creatinine (along with other parameters) is an indicator of adverse maternal outcome in pre-eclampsia particularly in the presence of proteinuria [52].
Oliguria is generally defined as urine output <500mL/24 hrs. By the time the pregnant pre-eclamptic women has been observed for 24 hrs, significant renal impairment may have occurred; hence this guideline recommends observation of urinary output over 4 hrs. Intrapartum and in the immediate postpartum period, oliguria is common and physiological and does not require fluid therapy unless the serum/plasma creatinine is rising.

Hyperuricemia is a common but not diagnostic feature of pre-eclampsia. The literature regarding uric acid as a predictor of maternal and/or fetal complications in pre-eclampsia is conflicting although a recent meta-analysis did suggest its usefulness in the management of pre-eclampsia [66]. It is important to use gestational corrected normal ranges which may correlate better with adverse events [67].

Upper limits for uric acid based on mean+2SD differ according to gestational age. At 32 weeks is 0.32 mmol/l, at 336 weeks is 0.34 mmol/l and at 38 weeks is 0.38 mmol/l. The platelet count normally decreases in pregnancy. A platelet count of <100,000/μL is seen in 4.5% of women with proteinuric pre-eclampsia and 9.9% with non-proteinuric pre-eclampsia and only 1% of normal pregnant women [68,69]. Coagulation studies are not indicated if the platelet count is normal [70].

HELLP syndrome represents a subset of women with severe pre-eclampsia characterized by haemolysis, raised liver enzymes (transaminases) and low platelets with or without other pre-eclamptic features. Often only two of the three components are recognizable [71].

IUGR is diagnosed when a fetus fails to achieve its growth potential in utero. It is usually (although not always) associated with a small for gestational age (SGA) fetus and is often associated with features suggestive of placental disease including abnormal umbilical artery Dopplers or oligohydramnios (in the absence of alternate diagnoses for such changes).

Pre-eclampsia remains a leading cause of maternal and fetal morbidity and mortality. Because the primary target of injury in pre-eclampsia is the glomerular endothelial cell, affected patients invariably present with depression of the glomerular filtration rate (GFR), proteinuria, and hypertension. Pre-eclampsia is a multiorgan disease process of unknown etiology [72], characterized by the development of hypertension and proteinuria after 20 weeks of gestation. Prevention through routine supplementation with calcium, magnesium, omega-3 fatty acids, or antioxidant vitamins is ineffective [75,76]. Calcium supplementation reduces the risk of developing pre-eclampsia in high-risk women and those with low dietary calcium intakes [112].
Pre-eclampsia: Etiology and Risk Factors

Abnormal placental implantation (defects in trophoblasts and spiral arterioles)[78-79]. It is thought that “failings” in normal hemochorial placental structure lead to pre-eclampsia and gestational hypertension [35]. The human placenta implants “earlier, deeper, and more extensively” into the uterine wall, which can potentially lead to many problems that are found in human pregnancies. Miscarriage and pre-eclampsia are both very rare in other species, but are two of the most common pregnancy-related diseases in humans [36]. The genetic roots of gestational hypertension and pre-eclampsia are certain, as women with a family history of the condition are three times more likely to suffer from it when they are pregnant [37]. One of the potential causes of gestational hypertension and pre-eclampsia is when the trophoblast does not invade far enough into the uterine lining [38]. When the fetus’ trophoblast does not fully extend into the uterine wall, the spiral arteries do not become fully converted into low-resistance channels [36]. It has been found that this incomplete conversion of spiral arteries increases the resistance to uterine blood flow during pregnancy, and that this occurrence was associated with gestational hypertension [39]. One potential cause of this incomplete breach of the spiral arteries that leads to gestational hypertension is a mistaken immune response by the maternal tissue, reaction to the alien fetal tissue [40]. Therefore, it is clear that the complication of gestational hypertension has roots in the early implantation of the fetus in the uterine wall, an implantation technique that is unique to humans. The highly invasive placenta that is found in humans is thought to be linked to humans’ high circulating levels of the hormones CG and HCG. It has been shown that the higher the levels of these hormones, the deeper the trophoblast’s invasion into the uterine wall. Instances of gestational hypertension and pre-eclampsia have been shown to occur when the invasion of the uterine wall is not deep enough, because of lower CG and HCG levels in the mother [41]. And theories of pathogenesis are:

-Genetic predisposition (maternal, paternal, thrombophilias) [81–82].

-Immunologic intolerance between fetoplacental and maternal tissue [27].

-Platelet activation.

-Vascular endothelial damage or dysfunction [27].
Maternal risk factors are obesity, age 35 years or more, past history of D.M, Hypertension and Renal diseases, adolescent pregnancy, new paternity, thrombophilias (anti-phospholipid syndrome, protein C/S deficiency, factor V Leiden), having donated a kidney and history of pre-eclampsia in previous pregnancy (particularly if severe pre-eclampsia before 32 weeks gestation). Pregnancy factors are multiple gestation (twins or triplets, etc.), Placental abnormalities: Hyperplacentosis, excessive exposure to chorionic villi and placental ischemia, and another factor is family history of pre-eclampsia [27, 73].

**Diagnosis**

Blood pressure should be measured at each prenatal visit with an appropriately sized cuff and the patient in a seated position [83-84]. Diagnostic criteria for pre-eclampsia are systolic Blood pressure of 140 mm Hg or more or a diastolic blood pressure of 90 mm Hg or more on two occasions at least six hours apart [73,83 ,84]. An increase of 30 mm Hg systolic or 15 mm Hg diastolic from baseline is no longer diagnostic for pre-eclampsia [73] because similar increases are common in uncomplicated pregnancies. The diagnostic threshold for proteinuria is 300 mg in a 24-hour urine specimen. A 24-hour determination is most accurate because urine dipsticks can be affected by variable excretion, maternal dehydration, and bacteriuria [27]. A random urine protein/creatinine ratio of less than 0.21 indicates that significant proteinuria is unlikely with a negative predictive value of 83 percent; however, confirmatory 24-hour urine protein determination is recommended [85]. Generalized edema (affecting the face and hands) is often present in patients with preeclampsia but is not a diagnostic criterion [21]. Pre-eclampsia is characterized as mild or severe based on the degree of hypertension and proteinuria, and the presence of symptoms resulting from involvement of the kidneys, brain, liver, and cardiovascular system [73]. Severe headache, visual disturbances, and hyperreflexia may signal impending eclampsia. Increased peripheral vascular resistance and pulmonary edema may occur. A decreased glomerular filtration rate may progress to oliguria and acute renal failure. The increased glomerular filtration rate of pregnancy lowers serum creatinine, and levels greater than 0.9 mg per dL (80 μmol per L) are abnormal in pregnancy. Liver manifestations include elevated transaminase levels, subcapsular hemorrhage with right upper quadrant pain, and capsular rupture with life-threatening intraabdominal bleeding. Obstetric complications include IUGR, placental abruption, and fetal demise [73].
Diagnostic Criteria for Severe Pre-eclampsia:

- Blood pressure ≥ 160 mm Hg systolic or 110 mm Hg diastolic on two occasions at least six hours apart during bed rest.

- Proteinuria ≥ 5 g in a 24-hour urine specimen or 3+.

Any of the following associated signs and symptoms:

- Cerebral or visual disturbances.

- Epigastric or right upper quadrant pain.

- Intrauterine growth restriction.

- Impaired liver function.

- Oliguria < 500 mL in 24 hours.

- Pulmonary edema.

- Thrombocytopenia [73].

HELLP Syndrome:

The acronym HELLP describes a variant of severe pre-eclampsia characterized by hemolysis, elevated liver enzymes, and low platelet count [86]. HELLP syndrome occurs in up to 20 % of pregnancies complicated by severe pre-eclampsia [87]. The clinical presentation of HELLP syndrome is variable; 12 to 18 % of affected women are normotensive and 13 % do not have proteinuria [88]. At diagnosis, 30 % of women are postpartum, 18 % are term, and 52 % are preterm [87].

Management of mild pre-eclampsia and gestational hypertension

Expectant management refers to prolongation of the pregnancy beyond these 48 hours with maternal and fetal monitoring.
Management of mild preeclampsia and gestational hypertension is commonly managed expectantly as follows:

- Maternal monitoring.

- Measure blood pressure twice weekly.

- Obtain laboratory tests weekly:

  CBC, platelet count, ALT, AST, LDH, uric acid and creatinine.

- Assess for proteinuria: screen with dipstick or spot protein/creatinine ratio and obtain periodic 24-hour urine collections.

- Fetal monitoring.

- Obtain nonstress test twice weekly.

- Measure amniotic fluid index once or twice weekly

Biophysical profile may be done weekly in place of one of the twice-weekly nonstress tests and amniotic fluid index [21, 27]. Perform ultrasonography for fetal growth every three to four weeks. Nonstress tests, amniotic fluid index measurements, and biophysical profiles are used to monitor patients for uteroplacental insufficiency [21, 27].

Umbilical artery systolic/diastolic ratios measured by Doppler ultrasonography may detect early uteroplacental insufficiency [91, 92]. The decision to deliver involves balancing the risks of worsening pre-eclampsia against those of prematurity. Delivery is generally not indicated for women with mild pre-eclampsia until 37 to 38 weeks of gestation and should occur by 40 weeks [21,27]. Patients with severe pre-eclampsia are admitted to the hospital, placed on bed rest, and carefully monitored. The goals of treatment are to prevent seizures, lower blood pressure to avoid maternal end-organ damage, and expedite delivery.

Pre-eclampsia is a progressive disorder that will inevitably worsen if pregnancy continues. Current therapy does not ameliorate the placental pathology nor alter the
pathophysiology or natural history of pre-eclampsia. Delivery is the definitive management and is followed by resolution, generally over a few days but sometimes much longer. Obstetric consultation is mandatory in all women with life-threatening complications (HELLP syndrome and eclampsia).

**Timing of delivery:**

Immediate management refers to delivery planned within 48 hours, usually after blood pressure stabilization and corticosteroid administration to accelerate fetal pulmonary maturity. Timing of delivery is dependent upon the severity of the maternal disease and the gestation at which the pre-eclampsia or gestational hypertension presents.

**Delivery plan:**

**24-31 week-**
Consult and transfer to Tertiary institution: likely to need preterm delivery. Aim to prolong pregnancy where possible.

**32-36 =** Aim to prolong pregnancy where possible, deliver in institution with appropriate Pediatric care.

**37+0 onwards** Plan delivery on best day in best way.

Fetal mortality and morbidity is strongly associated with gestational age at delivery. In many cases, the timing of delivery will be based upon a number of factors, maternal and/or fetal rather than single absolute indication for delivery [70, 72].

**Indications for delivery in women with preeclampsia or gestational hypertension:**

**Maternal:**
- Gestational age ≥ 37 weeks. -Inability to control hypertension.
- Deteriorating platelet count.
- Intravascular haemolysis
- Deteriorating liver function.
-Deteriorating renal function.

-Persistent neurological symptoms.

-Persistent epigastric pain, nausea or vomiting with abnormal LFTs

-Pulmonary edema.

**Fetal:**

-Placental abruption.

-Severe IUGR.

- Fetal wellbeing [76].

In cases of preterm pre-eclampsia before 34 weeks, delivery should be delayed for at least 24-48 hours, if maternal and fetal status permit, to allow fetal benefit from antenatal corticosteroids administered for lung maturation [76].

Antihypertensive treatment should be commenced in all women with a systolic blood pressure of greater than or equal to 160mm /Hg or a diastolic blood pressure greater than or equal to 110 mm/Hg because of the risk of intracerebral hemorrhage and eclampsia [6, 7, 11, 13].

There is controversy regarding the need to treat mild to moderate hypertension in women with pre-eclampsia. Antihypertensive therapy does not prevent pre-eclampsia (RR 0.99; 95% CI 0.84–1.18) or the associated adverse prenatal outcomes, but it decreases by half the incidence of development of severe hypertension among women with mild hypertension (RR 0.52; 95% CI 0.41–0.64) (24 trials, 2815 women) (93).

Approximately 10 women need to be treated with an antihypertensive drug to prevent an episode of severe hypertension (93). Uncontrolled hypertension is a frequent trigger for delivery and control of hypertension may allow prolongation of pregnancy.

The use of magnesium sulfate helps prevent seizures in women with pre-eclampsia[38-40]; women with mild pre-eclampsia would need to be treated to prevent one seizure [41]. Magnesium sulfate has the additional benefit of reducing the incidence of placental abruption [42]. Magnesium sulfate slows neuromuscular conduction and depresses central nervous system irritability without significant effects on blood pressure. Serum magnesium levels should be monitored in women with elevated serum creatinine levels, decreased urine output, or absent deep tendon reflexes [43].
Magnesium toxicity can lead to respiratory paralysis, central nervous system depression and cardiac arrest. The antidote is calcium gluconate, 1 g infused intravenously over two minutes [44].
Management of Mild Gestational hypertension or Preeclampsia[2]
Management Severe Preeclampsia[2]
Labor and Delivery Sample Admission Orders for Severe Pre-eclampsia

- Bed rest with seizure precaution.

- Vital signs (blood pressure, pulse, respiration).

- deep tendon reflexes; and mental status every 15 to 60 minutes until stable, then every 60 minutes while on magnesium sulfate.

- Accurate intake and output.

- Foley catheter if needed.

- Administer lactated Ringer's solution at 75 mL per hour IV to maintain urine output of 30 to 40mL per hour; total intake (IV and oral) should not exceed 125 mL per hour or 3,000 mL per day.

- Continuous fetal heart rate monitoring [27].

Laboratory tests;

- Dipstick urine collection for protein level on admission.

- 24-hour urine collection for total protein level.

- CBC with platelets, peripheral blood smears BUN, creatinine, uric acid, AST, ALT, and LDH.

- Fetal evaluation.

- nonstress test on admission.

- Obstetric ultrasonography for estimated fetal weight, amniotic fluid volume, and umbilical artery Doppler measurements.

Medications

Magnesium sulfate Loading dose of 4 to 6 g diluted in 100 mL of normal saline, given IV over 15 to 20 minutes, followed by a continuous infusion of 2 g per hour [73]. Assess serum magnesium level if urine output is < 30 mL per hour or there is a loss of deep tendon reflexes, decreased respiratory rate, or altered mental status Therapeutic range for serum magnesium is 4 to 7 mg per dL.
Corticosteroids (if between 24 and 34 weeks of gestation and not previously administered). Betamethasone (Celestone), 12 mg IM initially, then repeat in 24 hours or Dexamethasone, 6 mg IM initially, and then repeat every 12 hours for three additional doses. For systolic blood pressure > 160 mm Hg or diastolic > 110 mm Hg, one of the following should be given to achieve a systolic measurement of 140 to 155 mm Hg and/or a diastolic measurement of 90 to 105 mm Hg: Hydralazine, 5 to 10 mg IV every 15 to 30 minutes (maximal dose: 30 mg) or Labetalol, 20 mg IV initially; if the initial dose is not effective, double the dose to 40 mg and then 80 mg at 10-minute intervals until target blood pressure is reached or a total of 220 mg has been administered [21,27]; the maximal dose of IV labetalol is 220 mg in a 24-hour period [27,73]. Calcium gluconate, 1 g IV; keep at bedside in case of respiratory depression from magnesium sulfate use. Intravenous labetalol and hydralazine are commonly used for the acute management of preeclampsia [21,98]. Fluid Management. Excessive fluid administration can result in pulmonary edema, ascites, and cardiopulmonary overload, whereas too little fluid exacerbates an already constricted intravascular volume and leads to further end-organ ischemia. Urine output should be greater than 30 mL per hour and intravenous fluids limited to 100 mL per hour [89, 97].

Delivery Decisions in Severe Pre-eclampsia:

Delivery is the only cure for pre-eclampsia. Decisions regarding the timing and mode of delivery are based on a combination of maternal and fetal factors. Fetal factors include gestational age, evidence of lung maturity, and signs of fetal compromise on antenatal assessment. Patients with treatment-resistant severe hypertension or other signs of maternal or fetal deterioration should be delivered within 24 hours, irrespective of gestational age or fetal lung maturity. Fetuses older than 34 weeks, or those with documented lung maturity, are also delivered without delay [27]. For patients with severe pre-eclampsia between 24 and 34 weeks of gestation, the data are insufficient to recommend “interventionist” versus expectant management [99]. Subspecialty consultation is indicated [100,101]. Corticosteroids are administered to accelerate fetal lung maturity [27]. Expectant management, with close monitoring of the mother and fetus, delays delivery when possible and reduces neonatal complications and length of stay in the newborn intensive care nursery [99,101]. Contraindications to expectant management include persistent severe symptoms, multiorgan dysfunction, severe IUGR (i.e., estimated fetal weight below the 5th percentile), suspected placental abruption, or nonreassuring fetal testing [101]. Vaginal delivery is
recommended for women with severe preeclampsia if there is no evidence of maternal or fetal compromise or other obstetric contraindication [21].

Some experts recommend cesarean delivery for fetuses younger than 30 weeks when the cervix is not ripe, but a trial of induction may be considered. In patients with HELLP syndrome, cesarean delivery carries special risks, such as bleeding from thrombocytopenia and difficulty controlling blood pressure because of depleted intravascular volume [87, 88]. Most patients with preeclampsia respond promptly to delivery with decreased blood pressure, diuresis, and clinical improvement. Eclampsia may occur postpartum; the greatest risk of postpartum eclampsia is within the first 48 hours [97]. Magnesium sulfate is continued for 12 to 24 hours, or occasionally longer if the clinical situation warrants. There are no reliable data on postpartum hypertensive management [102]; however, oral nifedipine is commonly used [27].

**ECLAMPSIA**

An eclamptic seizure may be preceded by increasingly severe pre-eclampsia, or it may appear unexpectedly in a patient with minimally elevated blood pressure and no proteinuria. The timing of an eclamptic seizure can be antepartum 53%, intrapartum 19% or postpartum 28% [103].

**Management of eclampsia**

Initial management of an eclamptic seizure includes protecting the airway and minimizing the risk of aspiration by placing the woman on her left side, suctioning her mouth, and administering oxygen. A medical professional skilled in performing intubations should be immediately available [53]. Close observation, soft padding, and use of side rails on the bed may help prevent trauma from falls or violent seizure activity. After the convulsion has ended and the patient is stabilized, plans should be made for prompt delivery. In rural or remote areas, physicians need to consider the risk of transfer versus the benefits of tertiary maternal and neonatal care.

It is important to avoid unnecessary interventions and iatrogenic complications [43, 53]. Magnesium sulfate is the drug of choice because it is more effective in preventing
Persistent hypertension

Pre-eclampsia may be the commonest clinical variety of hypertensive disorders that precedes to persistent hypertension after puerperium.

Incidence

The exact incidence of postpartum hypertension is difficult to ascertain. In clinical practice, most women will not have their blood pressure (BP) checked until the 6 weeks’ postpartum visit in physician’s offices or in postpartum clinics.

Etiology and differential diagnosis:

The etiology and differential diagnosis of postpartum hypertension is extensive, but it can be focused based on clinical and laboratory findings as well as response to treatment of BP. Pre-eclampsia is the most common cause, however, other life-threatening conditions such as pheochromocytoma and cerebrovascular accidents should also be considered.

Differential diagnosis of postpartum hypertension

- New-onset hypertension-pre-eclampsia (Onset 3-6 d postpartum without headache).
- Persistence of pre-eclampsia.
- Late-onset eclampsia.
- HELLP syndrome.
- Preexisting/undiagnosed hypertension.
- Cerebral vasoconstriction syndrome.
- Cerebral venous thrombosis/stroke (Onset 3-7 d).
- TTP/hemolytic uremic syndrome [107,108].

In addition, postpartum women who have hypertension in association with symptoms such as headaches or blurred vision are often seen and managed in the emergency department and will not be coded as hypertensive unless they are hospitalized. Suggested first line antihypertensive drugs
that are safe in breast feeding mothers include labetalol, nifedipine and enalapril. Hypertension in the postpartum period affects several groups of women, including those with previous chronic hypertension, gestational hypertension, pre-eclampsia, and eclampsia. In addition, pre-eclampsia may present for the first time in the postnatal period. Although the underlying causes and clinical presentation of these types of hypertension vary, patients can be investigated and treated in a similar manner.

The most common cause of postpartum hypertension is high blood pressure (from gestational hypertension or pre-eclampsia) that persists after delivery preexisting Postpartum hypertension can be related to persistence of gestational hypertension (GH), pre-eclampsia, or chronic hypertension, or it could develop de novo secondary to other causes. Women with severe pre-eclampsia are at risk of developing chronic hypertension in future. Chronic hypertension may manifest initially as persistent hypertension at the end of the puerperium.

The objective was to determine the incidence and maternal biochemical, hematological and socio-demographic risk factors for persistent hypertension in patients with pre-eclampsia, mothers with pre-eclampsia are at risk of persistent hypertension after the puerperium. Factors that predict chronic hypertension include maternal age and gestation age of onset of pre-eclampsia [3]. A higher maternal age [4] and lower gestation age at onset increase risk of chronic hypertension. During the postnatal period, the hypertension and proteinuria due to pre-eclampsia resolve within six weeks, and women having persistent hypertension and proteinuria thereafter may have an underlying cause. Postnatal review at 6-12 weeks after delivery provides an opportunity to ensure that manifestations of pre-eclampsia and any associated systemic complications have resolved. Early identification of chronic hypertension among these women during follow-up might prevent or reduce its long-term complications if relevant interventions to decrease or avert associated renal or cardiovascular damage are instituted in time. There is a decrease in BP within 48hours, but BP increases again between 3-6 days postpartum [111].

In summary, there are several causes for postpartum hypertension; some may be benign such as mild GH or mild chronic hypertension whereas others can be life threatening such eclampsia or stroke. Therefore, a high index of suspicion for secondary dangerous causes of hypertension should be considered when evaluating such women. By directing efforts and educating health care Providers about the continued monitoring, reporting, and prompt evaluation of symptoms in the postpartum period, it is expected that some of the maternal complications will be avoided.
Evaluation and management of women with postpartum hypertension should be guided by obtaining a detailed history, careful physical examination, selective laboratory and imaging studies, and response to initial treatment [109,110].
Justification:

Hypertensive disorders of pregnancy are a major cause of maternal mortality and morbidity, especially in developing countries [1]. Hypertension may be present before or during pregnancy or postpartum [2]. Postpartum hypertension can be related to persistence of gestational hypertension (GH), pre-eclampsia, or preexisting chronic hypertension or it could develop de novo secondary to other causes [3]. In addition, the available data in the medical literature have primarily focused on antenatal and peripartum management of such patients [4,5], even though some patients can develop de novo eclampsia and hemolysis, elevated liver enzymes, and low platelet (HELLP) syndrome in the late postpartum period [6,7,8,9]. Diagnostic criteria for pre-eclampsia are systolic Blood pressure of 140 mm Hg or more or a diastolic blood pressure of 90 mm Hg or more on two occasions at least six hours apart. The diagnostic threshold for proteinuria is 300 mg in a 24-hour urine specimen. Maternal risk factors are obesity, age 35 years or more, past history of D.M, Hypertension and Renal diseases, adolescent pregnancy, new paternity, thrombophilias (anti-phospholipid syndrome, protein C/S deficiency, factor V Leiden), having donated a kidney and history of pre-eclampsia in previous pregnancy (particularly if severe pre-eclampsia before 32 weeks gestation). Pregnancy factors are multiple gestation (twins or triplets, etc.), Placental abnormalities: Hyperplacentosis, excessive exposure to chorionic villi and placental ischemia, and another factor is family history of pre-eclampsia [27, 73]. Delivery is the only cure for pre-eclampsia. Decisions regarding the timing and mode of delivery are based on a combination of maternal and fetal factors. Fetal factors include gestational age, evidence of lung maturity, and signs of fetal compromise on antenatal assessment. Patients with treatment-resistant severe hypertension or other signs of maternal or fetal deterioration should be delivered within 24 hours, irrespective of gestational age or fetal lung maturity. Fetuses older than 34 weeks, or those with documented lung maturity, are also delivered without delay. The exact incidence of postpartum hypertension is difficult to ascertain. Hypertension in the postpartum period affects several groups of women, including those with previous chronic hypertension, gestational hypertension, pre-eclampsia, and eclampsia. pre-eclampsia may present for the first time in the postnatal period. Although the underlying causes and clinical presentation of these types of hypertension vary, patients can be investigated and treated in a similar manner. pre-eclampsia is the most common cause of persistent hypertension. So the management of persistent hypertension depends on the history, examination and investigations. Persistent hypertension after puerperium has few litreture,
there is no publish data except very few.
The Justifications of doing this study are:
- persistent hypertension may have severe complications if not diagnosed and managed properly.
- Determination of risk factors and causes of delay of the diagnosis may prevent the occurrence of the condition.
- The improvements of the condition and the outcome need clear recommendation for follow up postpartum, during puerperirum and after.
Objectives of the study

General

- To determine the occurrence and significance of persistent hypertension after puerperium in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014.

Specific:

- To determine the factors that provoke persistent hypertension in postpartum period in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014.

- To correlate between antipartum and postpartum hypertension and persistent hypertension, to determine the relation between the gestational age, mode of delivery and the severity of the persistent hypertension in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014.
Methodology

Study Design:

Descriptive observational study in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014.

Study Area

Wad Medani Teaching Hospital of Obstetrics and Gynaecology is the main tertiary hospital in Wad Medani city, the capital of Gezira state, it is located almost in the centre of the Sudan, it is the first and largest specialized Obstetrics and Gynaecology hospital in Gezira state (which is the 2nd state in population size in Sudan after Khartoum state). The hospital serves a large catch up area in central Sudan, it consists of big 8 localities, has population about 6 millions live in Wad Medani the capital and 10 towns as well as in small villages and camps. The health system mainly based on health center and rural hospitals. Have 70 rural hospital, 311 health center, 23 blood banks and 8 dialysis center.

Wad Medani hospital receives women booked with the hospital's antenatal clinic, self referrals and referrals from other health units. The hospital conducts about 35 deliveries daily, over 21,000 deliveries per year.

Wad Medani Teaching Hospital of Obstetrics and Gynaecology has 330 beds, 20 consultant, 30 registrar, 5 theater rooms, 2 vaginal delivery and rooms and nursery units of 20 beds and cover 24 hours by 2 consultant and registrar of pediatrics and trained sisters and midwives.

Study populations:

The study carried out all women admitted with pre-eclampsia and delivered in Wad Medani Teaching hospital and agreed to be included in the study. These were followed up to the end of the puerperium. Hypertension in pregnancy was defined as any of the following: a blood pressure measurement of higher than or equal to 140 mmHg systolic or 90 mmHg diastolic, measured at rest on at least 2 occasions in a patient of more than 20 weeks of gestation; i a rise in systolic pressure of 30 mmHg or higher and diastolic pressure of 15 mmHg or higher.
Pre-eclampsia was defined as presence of hypertension (as defined above) and albuminuria (on urinary dipstick examination). A diagnosis of mild pre-eclampsia was made when a blood pressure of less than 160/110 mmHg was associated with proteinuria (albuminuria) of 2+ or less on urinary dipstick examination, at any time during the management.

The diagnosis of persistent hypertension was made when hypertension (as defined above) was found at any time six weeks or later postpartum.

**Data Collection:**

Data was collected through interviews using an interviewer-administered questionnaire containing all relevant information. Other data was collected by participant examination, biomedical investigations and review of participants' medical records.

This data included social-demographic data (age, educational level, occupation); obstetric history (such as parity, gestational age at presentation, past history of pre-eclampsia, past history of miscarriage). Biochemical data included dipstick urine collection for protein level on admission, 24-hour urine collection for total protein level, CBC with platelets, peripheral blood smears, uric acid, AST and ALT. Examination: general look of the woman, weight, height, blood pressure was measured using the manual sphygmomanometer machine in a sitting position, pulse rate, respiratory rate, examination of the chest, abdomen and lower limb.

Fetal evaluation and obstetric ultrasonography for estimated fetal weight, amniotic fluid volume, and umbilical artery Doppler measurements.

The primary outcome was persistent hypertension at six weeks postpartum. The number of women in this study was 600 participants with pre-eclampsia, 17 participants failed to turn up for postpartum review in the postnatal clinic, 12 participants were found to have normal blood pressure at five weeks postpartum. Out of 583 participants, we analyzed data for 165 participants had persistent hypertension.

**Data Analysis:**

The data entered in computer and double checked before analysis by SPSS 19 for windows. The means, percentages calculated & presented in tables, graphs & chart.
Ethical considerations:
Scientific and ethical approval obtained from Sudanese Medical Specialization Board Ethical Committee. The participant Patients counseled and consent obtained.
Result

This study showed the rate of persistent hypertension at Wad Madani obstetrics and gynaecology teaching hospital from the 1st of Jun 2014 to 31 December 2014. During the study period, 600 participants with pre-eclampsia were recruited into the study, 17 participants failed to turn up for postpartum review in the postnatal clinic, 12 participants were found to have normal blood pressure at five weeks postpartum.

Out of 583 participants, we analyzed data for 165 participants (28.3%) had persistent hypertension. **Table (1)** shows distribution of the study population according to age in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: <20 years 9 cases (5.5%), 31 cases (18.8%) ranging between 20-30 Years, 91 cases (55.2%) ranging between 30-40 years and 34 cases (20.6%) >40 years old.

**Table (2)** shows distribution of the study population according to the gestational age in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: 23 cases (14%) ranging between 30---33 weeks, 24 cases (14.5%) ranging between 33---36 weeks, 114 cases (69.1%) ranging between 36---39 weeks and 4 cases (2.4%) >39 weeks.

**Table (3)** shows distribution of the study population according to ANC in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: most of cases has no ANC 89 cases represent (53.9%), 76 cases has ANC (46.1%).

**Table (4)** shows distribution of the study population according to the past history of pre-eclampsia in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: 129 cases had history of pre-eclampsia (78.2%) and 36 cases (21.8%) had no history.

**Table (5)** shows distribution of the study population according to the past history of miscarriage in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: 86 cases (52.1%) had history of miscarriage and 79 cases (47.9%) had no history.

**Table (6)** shows distribution of the study population according to the body mass index in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: 65 cases (39.4%) were normal weight and 100 cases (60.6%) were overweight.
Table (7) shows distribution of the study population according to the platelet count in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: 98 cases (59.3%) platelet count > 150*10^3/mm, 32 cases (19.4%) platelet count ranging between 100*10^3/mm ----150*10^3/mm, 25 cases (15.2%) platelet count ranging between 50*10^3/mm ----100 *10^3/mm and 10 cases (6.1%) platelet count <50*10^3/mm.

Table (8) shows distribution of the study population according to the serum uric acid level in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: 98 cases (59.4%) serum uric acid level 3---7mg/dl and 67 cases (40.6%) serum uric acid level >7mg/dl.

Table (9) shows distribution of the study population according to the birth weight in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: 112 cases (67.9%) birth weight <2500 gm and 53 cases (32.1%) birth weight >2500 gm.

Figure (1) shows the distribution of the study population according to the occupation in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: Patients, housewives were 134 Patients (81.2%), laborers were 17 Patients (10.3%), and employers were 14 Patients (8.5%).

Figure (2) shows the distribution of the Patients according to level of education, primary school were 109 Patients (66.1%), secondary school 41 Patients (24.8%), university were 15 Patients (9.1%).

Figure (3) shows distribution of the study population according to parity in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: multipara represent 130 cases (78.8%) while primigravida represent 32 cases (19.4%) and grandmultipara 3 cases (1.8%).

Figure (4) shows distribution of the study population according to the mode of delivery in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014: 91 cases (55.2%) were delivered vaginally and 74 cases (44.8%) were delivered by c/s.
**PERSISTENT HYPERTENSION AFTER Puerperirurn Among Women with Pre-Eclampsia (Jan 2014 - Dec 2014)**

Table (1)

Distribution of the study population by age in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014:

<table>
<thead>
<tr>
<th>Age</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;20 Yrs</td>
<td>9</td>
<td>5.5%</td>
</tr>
<tr>
<td>20-30 Yrs</td>
<td>31</td>
<td>18.8%</td>
</tr>
<tr>
<td>30-40 Yrs</td>
<td>91</td>
<td>55.2%</td>
</tr>
<tr>
<td>&gt;40 Yrs</td>
<td>34</td>
<td>20.6%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>165</td>
<td><strong>100%</strong></td>
</tr>
</tbody>
</table>
PERSISTENT HYPERTENSION AFTER PUERPERIRUM AMONG WOMEN WITH PRE-ECLAMPSIA  (Jan 2014 - Dec 2014)

Table (2)

Distribution of the study population by gestational age in Wad Medani Teaching Hospital of Obstetrics and Gynecology from January 2014 to December 2014:

<table>
<thead>
<tr>
<th>gestational age</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>30----33wks</td>
<td>23</td>
<td>14%</td>
</tr>
<tr>
<td>33---36 wks</td>
<td>24</td>
<td>14.5%</td>
</tr>
<tr>
<td>36----39 wks</td>
<td>114</td>
<td>69.1%</td>
</tr>
<tr>
<td>&gt;39 wks</td>
<td>4</td>
<td>2.4%</td>
</tr>
<tr>
<td>Total</td>
<td>165</td>
<td>100%</td>
</tr>
</tbody>
</table>
PERSISTENT HYPERTENSION AFTER PUERPERIRUM AMONG WOMEN WITH PRE-ECLAMPSIA  (Jan 2014 - Dec 2014)

Table (3)

Study population by ANC in Wad Medani Teaching Hospital of Obstetrics and Gynecology from January 2014 to December 2014:

<table>
<thead>
<tr>
<th>ANC</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>76</td>
<td>46.1%</td>
</tr>
<tr>
<td>No</td>
<td>89</td>
<td>53.9%</td>
</tr>
<tr>
<td>Total</td>
<td>165</td>
<td>100%</td>
</tr>
</tbody>
</table>
Table (4)

Distribution of the study population by Past history of pre-eclampsia in Wad Medani Teaching Hospital of Obstetrics and Gynecology from January 2014 to December 2014:

<table>
<thead>
<tr>
<th>Past history of pre-eclampsia</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>129</td>
<td>78.2%</td>
</tr>
<tr>
<td>No</td>
<td>36</td>
<td>21.8%</td>
</tr>
<tr>
<td>Total</td>
<td>165</td>
<td>100%</td>
</tr>
</tbody>
</table>
PERSISTENT HYPERTENSION AFTER PUERPERAL AMONG WOMEN WITH PRE-ECLAMPSIA  (Jan 2014 - Dec 2014)

Table (5)

Distribution of the study population according to history of miscarriage in Wad Medani Teaching Hospital of Obstetrics and Gynecology from January 2014 to December 2014:

<table>
<thead>
<tr>
<th>Past history of miscarriage</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>86</td>
<td>52.1%</td>
</tr>
<tr>
<td>No</td>
<td>79</td>
<td>47.9%</td>
</tr>
<tr>
<td>Total</td>
<td>165</td>
<td>100%</td>
</tr>
</tbody>
</table>
PERSISTENT HYPERTENSION AFTER Puerperirum AMONG WOMEN WITH PRE-ECLAMPSIA (Jan 2014 - Dec 2014)

Table (6)

Distribution of the study population by BMI in Wad Medani Teaching Hospital of Obstetrics and Gynecology from January 2014 to December 2014:

<table>
<thead>
<tr>
<th>BMI</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;25</td>
<td>65</td>
<td>39.4%</td>
</tr>
<tr>
<td>&gt;25</td>
<td>100</td>
<td>60.6%</td>
</tr>
<tr>
<td>Total</td>
<td>165</td>
<td>100%</td>
</tr>
</tbody>
</table>
Table (7)

Distribution of the study population by Platelet count in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014:

<table>
<thead>
<tr>
<th>PLT</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 150*10^3/mm</td>
<td>98</td>
<td>59.3 %</td>
</tr>
<tr>
<td>100<em>10^3/mm ~ 150</em>10^3/mm</td>
<td>32</td>
<td>19.4%</td>
</tr>
<tr>
<td>50<em>10^3/mm ~ 100</em>10^3/mm</td>
<td>25</td>
<td>15.2%</td>
</tr>
<tr>
<td>&lt;50*10^3/mm</td>
<td>10</td>
<td>6.1 %</td>
</tr>
<tr>
<td>Total</td>
<td>165</td>
<td>100%</td>
</tr>
</tbody>
</table>
PERSISTENT HYPERTENSION AFTER PUERPERIRUM AMONG WOMEN WITH PRE-ECLAMPSIA (Jan 2014 - Dec 2014)

Table (8)

Distribution of the study population by serum uric acid in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014:

<table>
<thead>
<tr>
<th>Serum uric acid</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>3-------7mg/dl</td>
<td>98</td>
<td>59.4%</td>
</tr>
<tr>
<td>&gt;7 mg/dl</td>
<td>67</td>
<td>40.6%</td>
</tr>
<tr>
<td>Total</td>
<td>165</td>
<td>100%</td>
</tr>
</tbody>
</table>
PERSISTENT HYPERTENSION AFTER PUERPERIRUM AMONG WOMEN WITH PRE-ECLAMPSIA  (Jan 2014 - Dec 2014)

Table (9)

Distribution of the study population by Birth Wight in Wad Medani Teaching Hospital of Obstetrics and Gynecology from January 2014 to December 2014:

<table>
<thead>
<tr>
<th>Birth Wight</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2500 gm</td>
<td>112</td>
<td>67.9%</td>
</tr>
<tr>
<td>&gt;2500g</td>
<td>53</td>
<td>32.1%</td>
</tr>
<tr>
<td>Total</td>
<td>165</td>
<td>100%</td>
</tr>
</tbody>
</table>
Figure (1) Distribution of the study population by Occupation in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014.
PERSISTENT HYPERTENSION AFTER PUERPERIRUM AMONG WOMEN WITH PRE-ECLAMPSIA  
(Jan 2014 - Dec 2014)

Figure (2)

Distribution of the study population by Educational Level in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014.
**Figure (3)**

Distribution of the study population by Parity in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014
Figure (4) Distribution of the study population by mode of delivery in Wad Medani Teaching Hospital of Obstetrics and Gynaecology from January 2014 to December 2014.
DISCUSSION

Hypertensive disorders of pregnancy are a major cause of maternal mortality and morbidity, especially in developing countries [1]. Hypertension may be present before or during pregnancy or postpartum [2]. Postpartum hypertension can be related to persistence of gestational hypertension (GH), pre-eclampsia, or preexisting chronic hypertension, or it could develop de novo secondary to other causes [3]. During the past decades, there has been extensive research regarding the incidence, risk factors, pathogenesis, prediction, prevention, and management of GH-pre-eclampsia[4]. In addition, the available data in the medical literature have primarily focused on antenatal and peripartum management of such patients [4, 5], even though some patients can develop de novo eclampsia and hemolysis, elevated liver enzymes, and low platelet (HELLP) syndrome in the late postpartum period [6, 7,8, 9]. Thus, there are few data regarding the evaluation, management, and complications in women who are rehospitalized with diagnosis of postpartum hypertension [3, 10,11]. The differential diagnosis is extensive, and varies from benign mild gestational or essential hypertension to life-threatening such as severe pre-eclampsia-eclampsia and cerebrovascular accidents. The exact incidence of postpartum hypertension is difficult to ascertain. In clinical practice, most women will not have their blood pressure (BP) checked until the 6 weeks' postpartum visit in physician's offices or in postpartum clinics. This classification of the hypertensive disorders in pregnancy is as follows: Chronic hypertension, gestational hypertension and pre-eclampsia–eclampsia reflects the pathophysiology of the constituent conditions as well as the risks and potential outcomes for both mother and baby. Pre-existing hypertension is a strong risk factor for the development of pre-eclampsia (15, 16). Investigations of new onset hypertension after 20 weeks include: Full blood count, creatinine, electrolytes, urate, Urinalysis for protein and urine microscopy on a carefully collected mid-stream urine sample. Gestational hypertension is a provisional diagnosis that includes women eventually diagnosed with pre-eclampsia or chronic hypertension, as well as women retrospectively diagnosed with transient hypertension of pregnancy[46].

Pre-eclampsia is a multi-system disorder unique to human pregnancy characterized by hypertension and involvement of one or more other organ systems and/or the fetus. Raised blood pressure is commonly but not always the first manifestation. Proteinuria is the most commonly recognized additional feature after hypertension but should not be considered mandatory to make
the clinical diagnosis. A diagnosis of pre-eclampsia can be made when hypertension arises after 20 weeks gestation and is accompanied by one or more of the following signs of organ involvement. Twenty four hour urine protein has been the historic gold standard for quantifying proteinuria in pregnancy although its accuracy is affected by numerous factors such as adequacy and accuracy of collection and variations in protein excretion Oliguria is generally defined as urine output <500mL/24 hrs. Pre-eclampsia remains a leading cause of maternal and fetal morbidity and mortality. Diagnostic criteria for pre-eclampsia are systolic blood pressure of 140 mm Hg or more or a diastolic blood pressure of 90 mm Hg or more on two occasions at least six hours apart [73,83,84]. The diagnostic threshold for proteinuria is 300 mg in a 24-hour urine specimen. A 24-hour determination is most accurate because urine dipsticks can be affected by variable excretion, maternal dehydration, and bacteriuria [27]. An eclamptic seizure may be preceded by increasingly severe pre-eclampsia, or it may appear unexpectedly in a patient with minimally elevated blood pressure and no proteinuria. The timing of an eclamptic seizure can be antepartum 53 %, intrapartum 19 %, or postpartum 28 % [103].

The most common cause of postpartum hypertension is pre-eclampsia that persists after delivery. The etiology and different diagnosis of postpartum hypertension is can be focused based on clinical and laboratory findings as well as response to treatment of BP, pre-eclampsia (new onset or preexisting prior to delivery) is the most common cause. Chronic hypertension may manifest initially as persistent hypertension at the end of the puerperium. Mothers with pre-eclampsia/eclampsia are at risk of persistent hypertension after the puerperium. Because they are at risk of developing chronic hypertension, all women with pre-eclampsia /eclampsia require follow up after delivery. Postnatal review at 6-12 weeks after delivery provides an opportunity to ensure that manifestations of pre-eclampsia and any associated systemic complications have resolved. Early identification of chronic hypertension among these women during follow-up might prevent or reduce its long-term complications if relevant interventions to decrease or avert associated renal or cardiovascular damage are instituted in time.

The persistent hypertension rate in this study is (165/583X 100) =28.3%, that is consistent and comparable with the study which stated that, nearly every one in four mothers with pre-eclampsia/eclampsia are at risk of persistent hypertension after the puerperium done in UGANDA from November 2008 to May 2009 at Mulago hospital labor ward and postnatal
Participants were 200 women managed for pre-eclampsia/eclampsia and followed up to the end of the puerperium. The result fifty four (27.7%) out of the total 195 women had persistent hypertension after puerperium. Serum creatinine and the age of the patient were the only factors associated with persistence of hypertension after puerperium. Factors that predict chronic hypertension include maternal age and gestation age of onset of pre-eclampsia 64 (34%) out of the 188 women analyzed had persistent hypertension three months after delivery. Maternal age, gestational age at delivery and parity were predictors of persistent hypertension, the age distribution in this study, most of the women were in the age group of 30-40 yrs [55.2%]; a consistent higher maternal age [Layla F, Badria ZA: Pre-eclampsia: 2005] The study assessed the incidence of persistent hypertension and factors associated with persistent hypertension in patients with pre-eclampsia/eclampsia. The study shows that the incidence of persistent hypertension is 27.7% (54/195). Participants' age (especially among women aged 30-34 years), serum creatinine at admission, serum uric acid six weeks postpartum and urine protein at six weeks postpartum were the factors independently associated with persistent hypertension. Risk factors that can be assessed at booking time as age 35 years or more, Parity, obesity, history of Previous pre-eclampsia, family history of pre-eclampsia and interpregnancy interval, body mass index (BMI), blood pressure and proteinuria, past history of D.M, Hypertension and Renal diseases, adolescent pregnancy, new paternity, thrombophilias (anti-phospholipid syndrome, protein C/S deficiency, factor V Leiden), having donated a kidney, history of pre-eclampsia in previous pregnancy (particularly if severe pre-eclampsia before 32 weeks gestation) and family history [27-73].

Regarding the parity in this study [78.8%] were multiparae, [19.4%] were primigravidae, [1.8%] were grandmultiparae. The incidence increases among multiparous women the explanation not known. Gestational age distribution in this study was [69.1%] at gestational age 36wks to 39wks.

Antenatal Care [ANC] is very important to discovered high blood pressure and its complications, most of women had no ANC [53.9%], the percentage of women had ANC is [46.1%]. It’s important to follow women at preconception period. Most of women had no antenatal care, so this why most of them diagnosed in the late gestational age.
Regarding body mass index, obesity as a risk factor for pre-eclampsia, this is quite clear in our study as [60.6%] were overweight and [39.4%] were obese. Regarding the occupation in this study [81.2%] were house wife,[10.8%] were laborer and [8.5%] were employer.

Regarding the educational level, the rate of patient to developed persistent hypertension is increased in primary school [66.1%], the secondary [24.8%] and university [9.1%]. That means the educated women had low rate to developed preeclampsia and so persistent hypertension. Women with severe pre-eclampsia/eclampsia are at risk of developing chronic hypertension in future. Chronic hypertension may manifest initially as persistent hypertension at the end of the puerperium. The objective was to determine the incidence and maternal biochemical, hematological and socio-demographic risk factors for persistent hypertension in patients with pre-eclampsia/eclampsia.

Regarding platelet count [PTL], most of patient had PLT more than >150/mm [59.3%]. Regarding serum uric acid level most of patients had high level more than 7 gm/dl [40.6%].

The study assessed the incidence of persistent hypertension and factors associated with persistent hypertension in patients with pre-eclampsia/eclampsia. Most of women had past history of miscarriage the rate were [52.1%]; past history of Pre-eclampsia this was quite clear in our study as [78.2%] as risk factor of persistent hypertension but had no significance in this study. Family history of hypertension, history of diabetes and history of pre-eclampsia among siblings were not significantly associated with persistent hypertension.

Regarding the mode of delivery, most of women delivered by vaginal delivery [55.2%]. IUGR is diagnosed when a fetus fails to achieve its growth potential in utero. Umbilical artery systolic/diastolic ratios measured by Doppler ultrasonography may detect early uteroplacental insufficiency [91, 92]. The decision to deliver involves balancing the risks of worsening pre-eclampsia against those of prematurity. Delivery is generally not indicated for women with mild pre-eclampsia until 37 to 38 weeks of gestation and should occur by 40 weeks [21, 27]. It is usually associated with a small for gestational age (SGA) fetus and is often associated with features suggestive of placental disease including abnormal umbilical artery
Doppler’s or oligohydramnios in this study [67.9%] of the babies weight equal or less than 2500 gram. In addition, postpartum women who have hypertension in association with symptoms such as headaches or blurred vision are often seen and managed in the emergency department and will not be coded as hypertensive unless they are hospitalized. Suggested first line antihypertensive drugs that are safe in breast feeding mothers include labetalol, nifedipine, and enalapril. Refer women with persistent hypertension or proteinuria six weeks after delivery to a specialist.

Inform women with recent hypertensive disorders of pregnancy of the risk of recurrence in a future pregnancy. Hypertension in the postpartum period affects several groups of women, including those with previous chronic hypertension, gestational hypertension, pre-eclampsia, and eclampsia. In addition, pre-eclampsia may present for the first time in the postnatal period. Although the underlying causes and clinical presentation of these types of hypertension vary, patients can be investigated and treated in a similar manner. Management of pre-eclampsia is a common regimen for expectant management of mild pre-eclampsia [21,27]. Pre-eclampsia is a progressive disorder that will inevitably worsen if pregnancy continues. Current therapy does not ameliorate the placental pathology nor alter the pathophysiology or natural history of pre-eclampsia. Delivery is the definitive management and is followed by resolution, generally over a few days but sometimes much longer.
Conclusion

Pre-eclampsia complicate about 6-8% all pregnancies worldwide and are associated with increased maternal morbidity and mortality. They are also associated with development of persistent hypertension as well as cardiovascular and renal complications later in life.

Maternal age, gestational age, obesity and serum uric acid levels are predictors of persistent hypertension after the puerperium in women with pre-eclampsia.

Because they are at risk of developing persistent hypertension, all women with pre-eclampsia require follow up after delivery. Factors that predict persistent hypertension include maternal age and gestation age of onset of pre-eclampsia. A higher maternal age and lower gestation age at onset increase risk of persistent hypertension. Early identification of persistent hypertension among these women during follow-up might prevent or reduce its long-term complications.
**Recommendation**

1- There is need to coordinate and introduce regular follow up programmers in all postpartum care centres to enabling Patients to have access to quality information, counseling and services & also organize training on postpartum care at all levels.

2- Enhance the people to do a lot of thesis about persistent hypertension.

3- Health education and early antenatal care visit among pregnant women and women at risk.

4- Improve knowledge and skills of the providers & there is an urgent need to recognize and diagnosis of the problem.

5- Health care provider should be inform their pts, antipartum and postpartum about pre-eclampsia, its signs and symptoms and important of timely reporting of symptoms to health care provider, and the information should be re-emphasized at subsequent visits. Pregnancies affected by hypertensive disorders require careful monitoring due to the increased risks of adverse pregnancy outcomes. New onset hypertension in pregnancy warrants consideration of pre-eclampsia. Antihypertensive drugs for all forms of hypertensive disorder of pregnancy tend to be reserved for persistent or severe hypertension.
References
18. Martin JN, Jr., May WL, Magann EF, Terrone DA, Rinehart BK, Blake PG. Early risk assessment of severe preeclampsia: admission battery of symptoms and laboratory tests to predict likelihood of subsequent significant maternal morbidity. American journal of obstetrics and gynecology. 1999;180(6 Pt 1):1407-


50- Chung Y, de Greeff A, Shennan A. Validation and compliance of a home monitoring device in pregnancy: microlife WatchBP home. Hypertension in pregnancy : official journal of the


Flow chart of preeclampsia

1. Name:

2. Age

3. Parity

4. GA:

5. Education Level:
   a. None
   b. Primary
   c. Secondary
   d. University
   e. Post graduate

6. Occupation:
   a. H.W
   b. Laborer
   c. Employee

7. Weight

8. Height

9. BP

10. Family history of hypertension in first degree only:
   a. Yes
   b. No

11. History of miscarriage
   a. No
   b. Yes

12. Birth weight in Gm is IUGR
   a. Yes
   b. No
13. Gender: a. male ☐ b. female ☐

14. Basic lab investigation:

a. thrombocytes (platelet) ........ neutrophils

b. leukocytes (WBC) ................. Eosinophils

c. Monocytes ........ Basophils

15. HB ........ Lymphocyte ........ RBC ............... RDW ............

SGOT ........ SGPT ......... Urea ........ creatinine ....... Uric acid ............

16. Drugs received including antihypertensive ............

17. Mode of delivery: a. Vaginal ☐ b. EMCS ☐ c. LCS ☐

18. Maternal serum ........ cord serum ............

We need singleton viable pregnancy you can attach hemogram ....