Management Of Woman With Hypertensive Disorders In Pregnancy In Samawa City

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ABSTRACT:

Hypertensive disorders of pregnancy (HDP) remain among the most significant and intriguing unsolved problems in obstetrics. HDP are common and complicate obstetric practice in Iraq. The incidence of pre-eclampsia in hospital practice in Iraq varies from 7% to 13% and that of eclampsia about 1.1%.

Hypertensive disorders of pregnancy are a major issue of concern for the mother and her infant. Pregnancy with its enormous influence on the CV physiology is like a stress test in a woman's life. Hypertensive disorders in pregnancy can be considered as a failed CV stress test identifying the woman susceptible to CVD in later life. Pregnancy can be considered as a channel to identify women who are at risk for future CVD. As clinicians, we should utilize this channel to implement lifestyle modification in women to reduce their future burden of CVD. Further research is required to identify the mechanisms in pregnancy and HDP that contribute to CVD in later life so as to initiate appropriate preventive measures.

Key words: Hypertensive, pregnancy and disorders**.**

INTRODUCTION:

The hypertensive disorders of pregnancy (HDP) are no longer seen as "transitory diseases cured by delivery", but as windows into the future that allow us to predict a woman's future cardiovascular and kidney health. This paradigm shift has led to an increased need for integrated care for women who experience a hypertensive disorder during pregnancy, involving specialists that can oversee and coordinate treatment, and, whenever possible, work to identify risk factors and correct them [1,2].

Definitions of the hypertensive disorders of pregnancy have changed greatly in recent years. The objective of this article is to briefly review their main definitions and epidemiology as a support for nephrologists, who are increasingly involved in the management of HDP in the short and long term. In order to accomplish our task, we reviewed the available guidelines and the relevant literature.

The definition of HDP encompasses a spectrum of conditions that extends from gestational hypertension to preeclampsia (PE), eclampsia, and to hemolysis, elevated liver enzymes, and low platelet-count syndrome (HELLP). Other anomalies are non-uniformly aggregated in this family of diseases; this is the case for isolated or pregnancy-induced proteinuria (in normotensive pregnancies) and fetal growth restriction (FGR), which have, however, been demonstrated to predict the risk of developing PE during gestation as well as the risk of adverse short- and long-term maternal-fetal outcomes, including the development of chronic kidney disease (CKD).

The incidence of hypertensive disorders during pregnancy has increased in the last three decades, reaching 18,080,000 cases/year (a 10.92% increase), with a higher rate in East Asia[3].

Despite the growing number of cases, the number of deaths, estimated at 27,830 per year, has decreased significantly (a 30.05% reduction over 30 years, from 1990 to 2019); however, HDP-related mortality and morbidity remain unacceptably high [3].

There are two peaks in the age distribution of HDP, corresponding to the extremes of reproductive age [3]. The highest risk of all adverse pregnancy outcomes (including low birth weight, preterm delivery, and PE) recorded in younger age groups is influenced by several factors associated with teenage pregnancy, including low educational level, low income, malnutrition, precarious health before gestation, and marital status, factors that also reflect a continued lack of concern for young women's health [4,5].

The other extreme of the spectrum is represented by advanced maternal age; the increase that is observed can largely be explained by hormonal changes, together with an increased prevalence of obesity, chronic diseases (mainly diabetes and hypertension), and, at least in Western countries, with recourse to medically assisted reproduction techniques [6].

Classification:

Another distinction in the classification of pregnant women with hypertension compared with nonpregnant adults is that the category of HDP depends on how far along in pregnancy the woman is when first diagnosed. Twenty weeks' gestation is the cut point used, reflecting the return to an approximate baseline blood pressure after the first-trimester decline.(7)

1- chronic Hypertension

Chronic hypertension is defined as systolic blood pressure greater than or equal to 140 mm Hg and/or diastolic blood pressure greater than or equal to 90 mm Hg before pregnancy or before 20 weeks of gestation, the use of antihypertensives before pregnancy, or the persistence of hypertension more than 12 weeks after delivery.[8] Around 3% to 5% of pregnancies are estimated to be afflicted with chronic hypertension.

2- Gestational Hypertension

Gestational hypertension is defined as systolic blood pressure greater than or equal to 140 mm Hg and/or diastolic blood pressure greater than or equal to 90 mm Hg after 20 weeks of gestation in a woman who was at baseline normotensive.[9] If a woman diagnosed with gestational hypertension has persistent postpartum increases in blood pressure, she should be reclassified as having chronic hypertension.

3- Preeclampsia with and Without Severe Features

Preeclampsia is an HDP that is typically associated with new-onset hypertension with proteinuria, which occurs most often after 20 weeks of gestation.[9] Proteinuria is defined by ACOG as (1) 300 mg or more per 24-hour urine collection; (2) protein to creatinine ratio greater than or equal to 0.3 mg/dL; or (3) a dipstick reading of 2+ if quantitative methods are not available. However, preeclampsia can also manifest in the absence of proteinuria, and additional diagnostic criteria include (1) thrombocytopenia, defined as a platelet count less than 100,000 × 10^9/L; (2) impaired hepatic function, defined as transaminase level greater than 2 times the upper limit of normal; 6) severe right upper right quadrant or epigastric pain that is not associated with other diagnoses; (7) renal insufficiency, defined as serum creatinine greater than 1.1 mg/dL or a doubling of serum creatinine level in the absence of other renal disease; (5) pulmonary edema; (9) new-onset headache unresponsive to acetaminophen and not associated with other diagnosis or visual symptoms.

4- chronic Hypertension with Superimposed Preeclampsia

Chronic hypertension occurs in 1% to 5% of pregnant women, and 20% to 50% of these women go on to develop superimposed preeclampsia.[10] The risk of superimposed preeclampsia in women with chronic hypertension is increased in women who are black, obese, smoke, have a diastolic blood pressure greater than 100 mm Hg, have had chronic hypertension for more than 4 years, and have a history of preeclampsia during a prior pregnancy.[10,11] The incidence of superimposed preeclampsia is even higher in women with end-organ failure or secondary hypertension and approaches 75%.[12] However, in women with chronic hypertension and baseline proteinuria, superimposed preeclampsia can be difficult to distinguish from worsening chronic hypertension, and a high index of suspicion is required. The presence of new-onset thrombocytopenia or a sudden increase of liver enzyme levels is often the first sign of superimposed preeclampsia in this group.[12,13]

Pathophysiology of hypertension

Any hypertensive disorder of pregnancy can result in preeclampsia. It occurs in up to 35% of women with gestational hypertension[13] and up to 25% of those with chronic hypertension. The underlying pathophysiology that upholds this transition to, or superposition of, preeclampsia is not well understood; however, it is thought to be related to a mechanism of reduced placental perfusion inducing systemic vascular endothelial dysfunction. This arises due to a less effective

cytotrophoblastic invasion of the uterine spiral arteries. The resultant placental hypoxia induces a cascade of inflammatory events, disrupting the balance of angiogenic factors, and inducing platelet aggregation, all of which result in endothelial dysfunction manifested clinically as the preeclampsia syndrome. Angiogenic imbalances associated with the development of preeclampsia include decreased concentrations of angiogenic factors such as the vascular endothelial growth factor (VEGF) and placental growth factor (PIGF) and increased concentration of their antagonist, the placental soluble fms-like tyrosine kinase 1 (sFlt-1).[14] Impeding the binding of VEGF and PIGF to their receptors is a factor in the reduction of nitric oxide synthesis, a crucial factor in vascular remodeling and vasodilation, which may otherwise be able to ameliorate placental ischemia.[15] Early-onset preeclampsia (EOPE), occurring before 34 weeks of gestation, is thought to be primarily caused by the syncytiotrophoblast stress leading to poor placentation, whereas late-onset preeclampsia (LOPE), occurring at or after 34 weeks, is understood to be secondary to the placenta outgrowing its own circulation.

It is worth mentioning that EOPE is more frequently associated with fetal growth restriction than LOPE, due to a longer duration of placental dysfunction.[16]

During the postpartum period, up to 27.5% of the women may develop de novo hypertension. This is due to several factors, including mobilization of fluid from the interstitial to intravascular space, administration of fluids and vasoactive agents. The shift of fluids increases the stroke volume and cardiac output up to 80%, followed by a compensatory mechanism of diuresis and vasodilation, which softens the rise in blood pressure.[17,18]

The pathophysiology of hypertension in pregnancy becomes particularly relevant when reviewing the current state of adjunct therapies to antihypertensives that may help prevent preeclampsia.

Management:

The basic management objectives should be facilitating the birth of an infant who subsequently thrives and complete restoration of health to the mother, or the termination of pregnancy with the least possible trauma to mother and foetus in severe pre-eclampsia.19

The most important information to salvage the foetus is the precise knowledge of the age of the foetus. Early prenatal detection of HDP is usually by new onset rise in diastolic BP (≥81–89 mmHg) and a sudden abnormal weight gain (more than about 900–1000 g/week during the third trimester). (20)

Once a HDP is detected, outpatient surveillance is continued unless supervened by overt hypertension, proteinuria, visual disturbances or epigastric discomfort. Hospital isation is considered if there is persistent or worsening hypertension or development of proteinuria. In mild pre-eclampsia, reduced physical activity throughout much of the day is beneficial. Absolute bed rest is not necessary. Sedatives and tranquilisers are not prescribed. Ample, but not excessive,

protein and calories should be included in the diet. Sodium and fluid intakes should not be limited or forced. Delivery or termination of pregnancy is the cure for severe pre-eclampsia or eclampsia. The prime objectives in this situation are to forestall convulsion, prevent intracranial haemorrhage and serious damage to vital organs, ultimately to deliver a healthy infant if possible.(21-24)

Treatment

The goal of treating chronic hypertension in pregnancy is to avoid its acute complications and reduce the fetal risk posed by hypertension itself and the medications used for it. (25)

The aim is to ensure a healthy and safe pregnancy if possible till term. American College of Obstetricians and Gynecologists (ACOG) task force recommendations1 for hypertension management in pregnancy are:

• Home BP monitoring to ensure better control in patients with poorly controlled hypertension. (26)

• Ambulatory BP monitoring to rule out suspected white coat hypertension to avoid over treating hypertension.

• Patients with chronic hypertension on medications can be

◦ Off their medications and closely monitored as BP normally drops during pregnancy .(27)

◦ Continue their medications if warranted and safe

◦ Shift to alternate group of medications if their antihypertensive medication is not safe in pregnancy. (28)

◦ Closely monitored for symptoms of worsening hypertension and/or preeclampsia. Most patients with mild to moderate hypertension can be managed without medications. Antihypertensive drugs are mandatory if SBP exceeds 160 or if DBP exceeds 110 mm Hg. However, lower thresholds for initiating antihypertensive drugs are recommended in special circumstances.(29)

CONCLUSION:

HDP are common in Iraq; various risk factors increase the occurrence. The basic management objectives should be facilitating the birth of an infant who subsequently thrives and complete restoration of health to the mother. This comprises obstetric management, adequate foetal surveillance, antihypertensive management, anticonvulsant therapy, the anaesthetic management of labour and safe analgesia for labour and anaesthesia for delivery. Antihypertensive drugs and magnesium therapy are used to control hypertension and prevent seizures. Invasive haemodynamic monitoring is required to prevent the serious complication of fluid overload, pulmonary oedema. Anaesthetic problems in HDP may be due to the systemic effects. Careful pre-anaesthetic assessment, optimising physiology and expedited delivery is the goal. Risks and

benefits of techniques should be weighed – there is no conclusive evidence towards the advantage of regional or general anaesthesia.

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