

## HYPERTENSION IN PREGNANCY

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Hypertension (HTN) related complications occur in about 8% to 10% of all pregnancies and contribute significantly to maternal and neonatal morbidity and mortality worldwide. The National High Blood Pressure Education Program Working Group Report on High Blood pressure in Pregnancy classifies Pregnancy-related hypertension into four categories<sup>1</sup>. (a) HTN occurring prior to the onset of 20<sup>th</sup> week is defined as “chronic hypertension” (b) if Preeclampsia occurred later in pregnancy superimposed on chronic hypertension, it is labeled as “chronic hypertension with superimposed preeclampsia.” HTN arising after the 20<sup>th</sup> week is either (c) gestational hypertension or (d) preeclampsia, the difference between the two being that “preeclampsia” is a pregnancy related syndrome that includes proteinuria; whereas “gestational hypertension” occurs without proteinuria but may progress to preeclampsia and if it resolves before the 12<sup>th</sup> postpartum week, a retrospective diagnosis of “transient hypertension of pregnancy” is made<sup>1</sup>. Out of the above it is the preeclampsia and eclampsia that are responsible for most of the adverse effects attributed to HTN in pregnancy.

### CHRONIC HYPERTENSION IN PREGNANCY

“Chronic hypertension” is defined as hypertension that is present before pregnancy or that is diagnosed before the 20<sup>th</sup> week of gestation. It is defined as a blood pressure of 140 mm Hg systolic or 90 mm Hg diastolic. Hypertension that is diagnosed for the first time during pregnancy and that persists beyond the 12<sup>th</sup> week post partum is also classified as chronic hypertension. It is categorized into mild-to-moderate chronic HTN (systolic blood pressure [SBP] < 180 mm Hg systolic and diastolic BP [DBP] < 110 mm Hg) and severe (SBP>180 and DBP>110). Mild-to-moderate chronic HTN is associated with 33% risk for preterm delivery and 11% risk for small-for-gestational-age (SGA) infants whereas severe chronic HTN results in 62%-70% of preterm deliveries and 40% of SGA<sup>2</sup>. Women with chronic HTN are also at increased risk for preeclampsia (10%–25% of patients). Of late, a prediction model for preeclampsia using elevated systolic blood pressure, elevated uric acid, and low plasma renin activity has been devised for patients with chronic HTN<sup>3</sup>. The results showed that women with three risk factors (BP > 140 mm Hg, uric acid > 3.6 mg/dL, and plasma renin activity < 4 ng/mL/hr) had an 86% probability of superimposed preeclampsia. Women with one and two risk factors had a 40% and 62% probability, respectively. This prediction model awaits validation in other studies.

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### PREECLAMPSIA

A pregnant woman with a blood pressure of > 140 mm Hg systolic, > 90 mm Hg diastolic recorded at least on two occasions in a previously normotensive woman, and new onset proteinuria of > 300 mg/24 hr has preeclampsia. Presence of edema and BP rise of 30 mm Hg systolic and 15 mm Hg diastolic are no longer part of the criteria for diagnosis of preeclampsia. Quantitative 24-hour urine protein is the most accurate measure of proteinuria. If 24-hour urine testing is unavailable, at least two random urine samples of at least 1+ (30 mg/dL) on dipstick, collected 6 hours apart, will suffice<sup>1</sup>. Severe proteinuria is at least 5g of protein excretion on quantitative urine protein estimation. Both severe proteinuria and severe HTN (e” 180/110 mm Hg) are features of severe preeclampsia. Presence of pulmonary edema, seizures, oliguria, thrombocytopenia, and abnormal liver enzymes may also suggest severe preeclampsia. Two severe complications of preeclampsia are (1) the HELLP syndrome, characterized by the constellation of hemolysis, elevated liver enzymes, and low platelets, and (2) eclampsia, characterized by the development of seizures. Both conditions are rare, but are associated with a relatively poor prognosis.

**Pathophysiology :** Despite decades of research, the cause of preeclampsia remains uncertain. Several abnormalities (placental ischemia, increased pressor responsiveness, activation of the coagulation cascade, endothelial dysfunction, oxidative stress, leukocyte activation, cytokine activation, and hyperinsulinemia) have been observed in cross sectional studies in preeclampsia, but the exact sequence of events and whether these abnormalities are a result or a cause of disease remains unknown<sup>4</sup>. Pathophysiology of preeclampsia can broadly be summarized in two stages: (1) abnormal placental perfusion and (2) the maternal syndrome. Poor placental perfusion is thought to initiate the cascade of events resulting from abnormalities in vascular remodeling within the placenta. Recent studies have shown that there are abnormalities in circulating angiogenic factors as early as in the first trimester of pregnancy<sup>5</sup>. The second stage of the syndrome is the hypoperfusion of major organs and subsequent multiorgan failure. This is thought to be mediated by endothelial dysfunction—hence, its diffuse nature<sup>6</sup>. Elevated levels of plasma endothelial microparticles are evident in women with preeclampsia when compared with patients with gestational HTN<sup>7</sup>. Another classic finding is glomeruloendotheliosis, characterized by glomerular enlargement due to endothelial cell proliferation. This adds to the evidence implicating endothelial dysfunction. The mechanism linking the abnormal placental perfusion and the maternal multi-organ failure is still unknown. Oxidative stress has been proposed as a potential