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Editorial

Sympathovagal Imbalance in Early Part of Pregnancy Could be a Biological Marker of Gestational Hypertension

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Gestational hypertension (GH) is defined as a systolic blood pressure (BP) of at least 140 mm Hg and/or a diastolic blood pressure of at least 90 mm Hg recorded on at least two occasions at least 6 hr apart after the 20th week of gestation in women known to be normotensive before pregnancy.[1] It is one among the various categories of Hypertensive Disorders of Pregnancy (HDP) which also includes chronic hypertension, pre-eclampsia and eclampsia. Hypertension is one of the most common medical problem encountered in 15% of pregnancies and it contributes to 12% of maternal morbidity and mortality especially in developing countries of South-East Asia.[1] A study in India reported the prevalence of hypertensive disorders in which GH (47.4%), was the most common disorder followed by pre-eclampsia (32.6%), pre-eclampsia superimposed on chronic hypertension (11.8%) and chronic hypertension (8.2%).[2]

During normal pregnancy, there is a marked initial drop in mean arterial pressure, with an eventual rise to pre-pregnancy levels later, but blood pressure does not rise abnormally in GH.[3] However, BP in GH does not show initial drop and increases to an abnormally high level after 20th week of gestation.[2] The development of HDP involves a number of factors that result in volume and hemodynamic alterations that fail to adapt to the changes associated with pregnancy. Several epidemiological studies have proven the connection between Hypertensive Disorders in Pregnancy and increased Cardiovascular Diseases (CVD) later in life. Although the associations between pre-eclampsia and future CVD have long been known, newer evidence suggests that there are also long-term CVD risks related to gestational hypertension.[4]

Sympathovagal imbalance was reported in gestational hypertension and Heart Rate Variability (HRV) and Baroreflex Sensitivity (BRS) were used as the early markers of sympathovagal imbalance and autonomic dysregulation in various clinical disorders.[5] Recently, Pal et al. have reported that decrease in BRS is linked to inflammation and oxidative stress in pregnancy-induced hypertension.[6,7] Also, retrograde inflammation and oxidative stress in various conditions are reported to be associated with increased sympathovagal imbalance and CV risks in GH.[8]

Though the mechanisms responsible for the pathogenesis of Pregnancy Induced Hypertension (PIH) are unclear, it has been established that the disease is characterized by low circulating volume and high vascular resistance.[9] The increased peripheral vascular resistance is caused due to sympathetic overactivity which in turn causes the increase in blood pressure.[10] The sympathetic activity is reported to be high in the early part of pregnancy in women having risks of GH and it is presumed that this subtler increased sympathetic discharge could be the cause of increased peripheral resistance and absence of initial fall of BP in these high risk women. Pregnant women at high risk of GH are advised to take 75 mg of aspirin daily from 12 weeks until the birth of the baby (NICE 2010 Guidelines). The effects of low dose aspirin (60-150 mg/day) for preventing and treating pre-eclampsia were small but there was considerable reduction in the risk of pre-eclampsia.[11] It is believed that aspirin might be causing in decrease in sympathetic discharge in addition to its other metabolic effects to reduce BP in GH.

Studies reported that either pre-existing hypertension/PIH or pregnancy changes could be responsible for the occurrence of pre-eclampsia. About 15-25% of women who are initially diagnosed with GH eventually progressed to Pre-eclampsia.[10] WHO systematic analysis (2014) which included 417 datasets from 115 countries reported that hypertensive disorders of pregnancy account for about 14% (11.1-17.4) maternal death (n=341,000) in developing countries, whereas in Asia, 111,590 maternal deaths occur annually due to pregnancy related hypertensive disorders. Specifically, in Southern Asia, PIH affects health 10.3% (5.8-16.6) of maternal life.[12] Fetal adverse events include intrauterine growth retardation, prematurity, low birth weight and death.[11] Therefore, there is a need of detection of a marker which can predict development of GH and also the genesis CV risks in the early part of pregnancy, especially in women having risk of GH so that intervention can be initiated early and they do not develop GH in the later part of pregnancy. HRV and BRS have been established as marker of sympathovagal imbalance and CV risks, respectively. Studies should be conducted in larger cohort to assess if these physiological markers could be used for prediction of development of GH in women in the early part of pregnancy.

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