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Editorial

Preeclampsia

Conception about Preeclampsia (PE) has been significantly advanced in recent times. With better understanding of this condition, we can optimise prevention, diagnosis and timely management of this complex disorder.

Preeclampsia was formerly defined as a multisystemic disorder characterized by new onset of hypertension (i.e. systolic blood pressure (SBP) e" 140 mmHg and/or diastolic blood pressure (DBP) e" 90 mmHg) and significant proteinuria (> 300 mg/ 24 h) after 20 weeks of gestation in a previously normotensive, nonprotienuric woman. In recent time the American College of Obstetricians and Gynecologists (ACOG) has stated that even in absence of proteinuria , hypertension along with any one of the features e.g. renal impairment, thrombocytopenia, epigastric pain, liver dysfunction, visual disturbances, headache is sufficient for diagnosis of pre-eclampsia. Severe hypertension (SBP e" 160 mm Hg and/or DBP e" 110 mmHg) and/ or anyone of these above mentioned features will be designated as severe PE.[1]

Pathogenesis of PE begins to evolve in early pregnancy though clinical presentation appears in the second half of pregnancy justifying the two- stage model and supported by animal and human studies.[2] In first stage, cytotrophoblast fails to remodel spiral arteries, leading to hypoperfusion or ischemia of the placenta with consequent fetal growth restriction. In second stage ischemic placenta releases antiangiogenic factors into the maternal circulation provoking generalized maternal endothelial dysfunction. The endothelial dysfunction is in turn responsible for the symptoms and complications of PE.

Despite this better understanding of pathophysiology and maternal hemodynamic alterations in preeclampsia, still the only curative treatment of pre-eclampsia is delivery of placenta and fetus. However recent studies showing promising role of low dose aspirin and Calcium in preventing PE. In 2013, ACOG Task Force recommended low-dose aspirin (60-80 mg) from the late first trimester specially for women with history of prior early onset PE with preterm delivery at <34 weeks' gestation, or PE in more than one prior pregnancy for prevention of PE in present pregnancy. This recommendation was based on several meta-analyses that demonstrated a 10% to 17% reduction in risk with no increase in bleeding, placental abruption, or other adverse events.[3,4,5] In 2014 September the U.S. Preventive Services Task Force (USPSTF) supplemented the ACOG recommendation guideline on basis of systematic review by defining the high risk group for development of PE.[6] According to the recommendation low-dose aspirin should be initiated after 12 weeks of gestation for women who are considered high-risk for development of PE e.g. having history of preeclampsia, especially when accompanied by an adverse outcome, multifetal gestation, chronic hypertension, Diabetes (Type 1 or Type 2), renal disease and autoimmune diseases (such as systematic lupus erythematosus, antiphospholipid syndrome). This view is also supported by WHO and recommended to initiate low-dose Ecosprin(75 mg) before 20 weeks of pregnancy for the prevention of PE and its related complications specially for the women who are at high risk of developing the condition.[7]

In addition WHO strongly recommends calcium supplementation during pregnancy (at doses of 1.5–2.0 g elemental calcium/day) for all women, especially those who are high risk for developing pre-eclampsia and in areas where calcium intake is low.[7] This recommendation was based on Cochrane systematic review (13 trials, 15730 women; RR 0.45 95% CI 0.31–0.65) which showed that all women, irrespective of the baseline risk of developing pre-eclampsia and calcium intake status, calcium supplementation more than halved the risk of pre-eclampsia when compared with placebo. This risk reduction was 41% for low risk and 78% for high risk group for developing pre-eclampsia.[8]

At the time of diagnosis, initial assessment should focus on categorization of severity of the disease. Medical treatment i.e. antihypertensive and magnesium sulfate though cannot alter the course of the disease but to be initiated in preeclampsia with

severe features aiming to prevent catastrophic maternal complications e.g. intracranial hemorrhages and seizures. The decision of terminating pregnancy is based on gestational age and severity of preeclampsia. ACOG recommended delivery after 37 weeks in cases of preeclampsia without severe and after 34 weeks with severe features. Between 24 and 34 weeks of gestation, conservative management may be considered in selected patients. Antenatal corticosteroids should be administered to less than 34 wks to promote fetal lung maturity. Maternal end organ dysfunction and non-reassuring tests of fetal well-being are indications for delivery at any gestational age.[1]

Preeclampsia has been called the "great masquerader" and the "disease of exceptions" because of its complex, varied, and often insidious presentation. As because of the complexity and variability of the disorder, which is so daunting that every specific statement about preeclampsia seems to have exceptions - it is truly a disease of exceptions.

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