PREECLAMPSIA AND ECLAMPSIA

Preeclampsia is a multisystem disorder unique to human pregnancy.This syndrome is characterized by new onset hypertension and proteinuria after 20 weeks of gestation.It effects about 7-10% of all pregnancies and together with eclampsia(seizure due to preeclampsia) is a leading cause of fetomaternal mortality and morbidity especially in underresourced settings and is recognized as a contributer of future cardiovascular and metabolic dysfunction. Preeclampsia(PE) and eclampsia is accounting for more than 75000 deaths per year.[1]

It is a systemic disorder of endothelial dysfunction which is considered to be central to the multiple organ pathophysiology of PE/eclampsia,but the pathogenesis involved in the initiation and progression of the disease process is still unknown. The definitive cure of PE/eclampsia is delivery of placenta. As patients with molar pregnancy can develop PE rather at early gestation so it is reasonable to ascertain that placenta play a major role in the pathogenesis of this disorder. Risk factors for PE/Eclampsia are first pregnancy, teenage pregnancy, age >35 years, low socioeconomic status, family history of PE/Eclampsia, past history of PE/Eclampsia, multifetal gestation, poor outcome in previous pregnancy including IUGR, abruption, fetal death, preexisting obesity, chronic hypertension, renal disease, gestational diabetes, vascular and connective tissue disorders, various inflammatory diseases like urinary tract and periodental infections.

Numerous genetic, immunologic and environmental factors interact complex mechanism involving lipid, protein oxidation, altered nitric oxide production and placental glycoprotein playing role in the trophoblastic dysfunction. The main pathology at placental level is inadequate trophoblastic invasion in the spiral arteriols resulting in the failure of conversion of spiral arteriols into low resistance vessels. What cause this defective placentation and ultimately this multisystem disorder remain unknown.

The poorly perfused placenta releases increased amount of vasoactive factors in maternal circulation. Among the most well characterized factors in this disease are anti angiogenic protein soluble Fms like tyrosine kinase-I, inflammatory cytokines and agonistic angiotensin-II type I receptor autoantibodies[2]. Elevation in these factors are proposed to result in endothelial dysfunction by decrease in bioavailable nitric oxide and increase in reactive oxygen and endothelion. These endothelial abnormalities in turn leads to increase in blood pressure by impairing normal pressure natriuresis and increase in total peripheral resistance and other manifestation of the disease.

Larger multicenter trials are undergoing to find out the exact etiology of this dreadful condition. Determining the underlying pathogenesis of PE/Eclampsia will be significant medical advance. Better understanding of this condition will allow us to screen high risk group which will ultimately help in reducing the adverse fetomaternal outcome.

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