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RESEARCH ARTICLE

Assessment of Inflammatory Markers in Preeclampsia

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ABSTRACT

Aim: Pregnancies, including preeclampsia, are low-grade systemic inflammation which has been associated with complications. Endothelial dysfunction in preeclampsia is accompanied by elevated levels of inflammatory markers and cytokines such as high sensitivity C-reactive protein (hs-CRP), tumor necrosis factor-α (TNF-α) and interleukin-10 (IL-10). Our aim in this study is to assess the levels of these above markers in preeclampsia and to predict its severity.

Materials and methods: A case-control study was performed on 100 pregnant women with preeclampsia as cases (50 mild and 50 severe) as per clinical guidelines and 50 healthy pregnant women as controls. They were all age and parity-matched primigravidas at the third trimester of pregnancy.

Results: Significantly increased ($p < 0.001$) serum levels of hs-CRP, TNF-α and IL-10 were found in cases of mild and severe preeclamptic when compared to controls. In severe preeclamptics, all these parameters were also found highly significant in comparison to mild.

Conclusion and clinical significance: The observed high levels of hs-CRP, TNF-α, and IL-10 in preeclamptic women indicate that these markers can be used in identifying the severity of preeclampsia and it can help the clinician in their diagnosis, treatment, and management.

Keywords: High sensitivity C-reactive protein (hs-CRP), Interleukin-10 (IL-10), Preeclampsia, Tumor necrosis factor-α (TNF-α).

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INTRODUCTION

Preeclampsia is one of the hypertensive disorders of pregnancy. It is a major cause of maternal and perinatal mortality and morbidity and is a pregnancy-specific hypertensive disease with multisystem involvement. It is a disorder of widespread vascular endothelial malfunction and vasospasm that occurs after 20 weeks of gestation and can present as late as 4 to 6 weeks postpartum.[1]

Hypertension is one of the most common medical problem encountered during pregnancy, complicating up to 10% of pregnancies.[2] Preeclampsia occurs in 3 to 6% of all pregnancies and the incidence is 1.5 to 2 times higher in first-time pregnancies.[3] The incidence of preeclampsia has been increased by 25% in the United States during the past two decades. In India, the incidence of preeclampsia is reported to be 8 to 10% among pregnant women. According to Dubey L, the prevalence of hypertensive disorders of pregnancy was 7.8% with preeclampsia in 5.4% of the study population in India. Preeclampsia is a leading cause of maternal and perinatal morbidity and mortality, with an estimated 50000-60000 preeclampsia related deaths per year worldwide.[4,5]

Preeclampsia syndrome, in many cases, is thought to cause by a shallowly implanted placenta which becomes hypoxic, leading to an immune reaction characterized by secretion of upregulated inflammatory mediators from the placenta and acting on the vascular endothelium.[6] It is characterized by widespread endothelial dysfunction throughout the maternal circulation resulting in hypertension attributable to vasoconstriction, proteinuria due to glomerular damage and edema as a result of increased vascular permeability.[7] Inflammatory cells are activated in preeclampsia and localized to the site of vascular injury and associated with higher levels of pro-inflammatory molecules, cytokines, and adhesion molecules.

- One of the most important objectives and sensitive indexes of overall inflammatory activity in the body is hs-CRP.[8] The hepatic synthesis of hs-CRP increases in response to inflammatory cytokines such as IL-10 and IL-6 which are responsible for inflammatory reactions and maternal endothelial activation in pre-eclampsia.[9,10]
- The IL-10 is a potent pleiotropic cytokine, which has the dual ability of immune-suppression or immune-stimulation via the production of pro-inflammatory cytokines by the inhibition of T-helper 1 (Th1) lymphocytes and stimulation of B and Th2 lymphocytes and thus downregulates the inflammatory response.[11]

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