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HAEMOGLOBIN AND LEUCOCYTES IN PRECLAMPSIA: A REVIEW

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Abstract

Isolated mononuclear leukocytes, when incubated with purified haemoglobin Ao (HbAo), release the pro-inflammatory cytokines interleukin-8 (IL-8) and tumor necrosis factor-α (TNF-α). Thus, inflammation is believed to play a role in preeclampsia. Leukocytes increase due to inflammatory response. There isalsoincreased hemoglobin production in the PE placenta. The hemoglobin may be released into the placenta blood vessel lumen. In addition, Gene and protein profilingstudies have shown increased expression and accumulation of free fetal hemoglobinin the preeclamptic placenta. Predominantly due to oxidative damage to the placentalbarrier, fetal hemoglobin leaks over to the maternal circulation. Oxidative stress in general, and more specifically fetal hemoglobin-inducedoxidative stress, could play a key role in the pathology of preeclampsia seen both in theplacenta and ultimately in the maternal endothelium.

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Introduction:-

Preeclampsia (PE) complicating 6-8% of all pregnancies [1-5] and according to statistics preeclampsia occurs in up to 5% of all pregnancies, in 10% of first pregnancies, and 20–25% of women with a history of chronic hypertension [6]. PE is a multisystem disorder that leads to end-organ damage and/or hypoperfusion such as renal dysfunction, hematological dysfunction, hepatic dysfunction (raised transaminases), and neurological dysfunction.

Known for centuries, it remains a leading cause of maternal mortality and morbidity. Clinical manifestations of PE (hypertension and proteinuria) appear after the 20th week of gestation, and treatment of these problems is symptomatic and less effective than one would wish it to be. Delivery of the fetus and removal of the placenta is the only curative treatment, a fact that has lead to the generally accepted theory that placental pathology is central to the development of PE [7]. In addition, the disorder is also a major cause of perinatal morbidity and mortality globally, especially from preterm delivery [1]

Globally, the incidence of preeclampsia, was 2.16% [8] and according to the World Health Organization, the prevalence of preeclampsia is estimated to be seven times higher in developing countries (2.8% of live births) than in developed countries (0.4%) [6].

Inflammation is believed to play a role in preeclampsia. Leukocytes increase due to inflammatory response. However, there is an ongoing debate about whether inflammatory system hyperactivity indeed exists during PE, and if available data are sufficient for justification of broad anti-immune system treatment strategies [10].

Heomoglobin and Leucocytes in Preeclampsia

One of the theories states thatpreeclampsia is caused by the failure ofthe trophoblast invasion process, whichleads to hypoperfusion and ischemic of theplacenta. This, in turn, will create destructionof endothelial cells and inducevasospasm, thrombocytes aggregation,and thrombocytes usage [9] Angiogenesis imbalance and systemic inflammationare believed to play a role in theetiopathogenesis of preeclampsia. Theinflammatory response in preeclampsiainvolves leukocyte activation, andleukocytes are found to be increased dueto the inflammatory response.

A Prospective study conducted to create a library enriched in cDNAs from preeclamptic placentas to print onto microarrays for placental profiling of preeclampsia (PE) and high-risk pregnancies showed that Thirty genes were significantly altered in at least one group comparison. Differences in two candidate genes were confirmed using quantitative real-time PCR. Hemoglobin a2 and g transcripts were significantly over-expressed in the PE placenta. Scattered cells in the placenta and placental blood vessels were shown to express genes encoding these hemoglobin chains [7].

A study done in Brazil found lower whole blood and plasma nitrite concentrations in preeclamptic patients compared with healthy pregnant women. Plasma samples from preeclamptic women consumed 63% more nitric oxide (NO) and had 53% higher plasma hemoglobin (pHb) and 10% higher ceruloplasmin levels than those found in healthy pregnant women. The study also found significant positive correlations between plasma hemoglobin (pHb) and plasma NO consumption (pNOc), negative correlations between pNOc and whole blood or plasma nitrite concentrations, and negative correlations between pHb and whole blood or plasma nitrite concentrations [11].

A study that recruited 93 women with PE and 94 normal pregnant women matched for both maternal age and gestational age as controls showed that there were no statistically significant differences between patients with PE and normal pregnancy with regard to the maternal age and gestation at delivery; however, patients with PE exhibited significantly higher blood pressure, proteinuria levels and parity. Maternal age, multiparity status and the complete blood count parameters including hemoglobin (Hg), white blood cell (WBC), platelets, neutrophil/lymphocyte counts, mean platelet volume (MPV), and mean corpuscular volume (MCV) were not statistically different between the groups. Platelet/lymphocyte ratio (PLR) and neutrophil/lymphocyte ratio (NLR) levels were comparable between PE and normal pregnancies. Moreover, in subgroup analysis, patients with severe PE had similar NLR but lower PLR levels compared to women with mild PE [10].

A an observational analyticstudy with a cross-sectional approach done at Dr. Kariadi General Hospital in Indonesia showed that there was a significant difference in platelet count and leukocyte count between severepreeclampsia and normotensive pregnancy. Therefore, a differencein platelet-white blood cell ratio between severepreeclampsia and normotensive pregnancy was observed [9].

Conclusion:-

Heomoglobin and Leucocytes could be involved in inflammatory and thrombotic processes that known cause preeclampsia. The relationship between hemoglobin and Leucocytesto preeclampsia is critical and should be studied.

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