

بسم الله الرحمن الرحيم





شبكة المعلومات الجامعية التوثيق الالكتروني والميكرو فيلم



جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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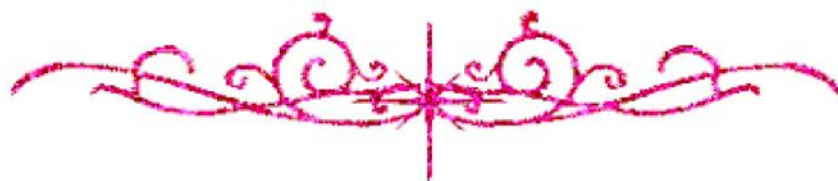


بعض الوثائق الأصلية تالفة





بالرسالة صفحات
لم ترد بالأصل





The Accuracy of Neutrophil/Lymphocyte Ratio in Prediction of Preeclampsia in Low Risk Population

Thesis

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List of Abbreviations

| Abb. | Full term |
|-----------------------|---|
| ACOG | American College of Obstetricians and Gynecologists |
| ALT | Alanine transaminase |
| AST | Aspartate aminotransferase |
| BMI | Body mass index |
| CBC | Complete blood counts |
| CEMACH | Confidential Enquiry into Maternal and Child Health in UK |
| CRP | C-reactive protein |
| DBP | Diastolic blood pressure |
| EDTA | ethylenediaminetetraacetate |
| eNOS | Endothelial nitric oxide synthase |
| HELLP | Hemolysis, Elevated Liver enzymes and Low Platelets |
| IL-8 | Interleukin-8 |
| ILT | Immunoglobulin-like transcript receptor |
| IUGR | Intrauterine growth restriction |
| mg | Milligram |
| mg/dL | Milligram per deciliter |
| mm Hg | Millimeter of mercury |
| mm³ | Millimeter of mercury |
| NK | Natural killer |

| Abb. | Full term |
|--------------------------------|---------------------------------------|
| NLR | Neutrophil lymphocyte ratio |
| N/Th1 | Neutrophil helper T1 lymphocyte ratio |
| N/Th2 | Neutrophil helper T2 lymphocyte ratio |
| PE | Preeclampsia |
| PET | Preeclampsia toxemia |
| PGF | Placental growth factor |
| PLR | Platelet lymphocyte ratio |
| Pr/Cr | Protein creatinine ratio |
| P/Th1 | Platelet helper T1 lymphocyte ratio |
| P/Th2 | Platelet helper T2 lymphocyte ratio |
| ROC | Receiver Operating Characteristic |
| SBP | Systolic blood pressure |
| sEng | Soluble endoglin |
| sFlt-1 | Soluble fms-like tyrosine kinase 1 |
| SLE | Systemic lupus erythematosus |
| SNPs | Single-nucleotide polymorphisms |
| TNF-α | Tumor necrosis factor- α |

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Introduction

Hypertensive disorders are the most common medical problem encountered in pregnancy, affecting up to 15% of pregnancies and accounting for approximately 25% of antenatal admissions. They are classified into four categories: Preeclampsia-Eclampsia, chronic hypertension, chronic hypertension with superimposed preeclampsia and gestational hypertension (**Butalia et al., 2018; ACOG, 2013**).

preeclampsia (PET) is a major cause of maternal and fetal or neonatal mortality and morbidity (**Sibai et al., 2005**). This disorder complicates 5%-7% of all pregnancies (**Wagner, 2004**).

Preeclampsia is defined as a new onset of hypertension associated with proteinuria and fluid retention detected for the first time after the 20th week of gestation (**Salam et al., 2015**).

Clinical symptoms are hypertension $\geq 140/90$ mm Hg and proteinuria ≥ 0.30 g/d. Severe conditions are often associated with intrauterine growth restriction (IUGR), acute renal failure, HELLP syndrome (Hemolysis, Elevated Liver enzymes and Low Platelets) or other systemic manifestations (**Cornelius and Wallace, 2019**). It is

associated with high risks of preterm delivery, intrauterine growth restriction, placental abruption, renal failure, sub-capsular hepatic hematoma and perinatal mortality, along with maternal morbidity and mortality **(ACOG, 2013; McClure et al., 2011)**. The pathophysiology of PET is not clear, there are many theories highlighting its multifactorial basis. It may be related to both placental and maternal factors including abnormal placentation, systemic endothelial dysfunction or cell activation, and an angiogenic imbalance favoring anti-angiogenic factors **(Fisher, 2015)**.

Preeclampsia is associated with a more maternal systemic inflammation than occurs in normal gestation. The features of PET arise from the sum of the circulatory disturbances caused by systemic maternal endothelial cell dysfunction or activation. so leukocytes activates endothelium and vice versa **(Mihu et al., 2015; Tannetta et al., 2017)**.

Hence, all the markers of inflammation that already are changed in normal pregnancy are affected more severely in PET. The inflammatory changes may progress to the point of decompensation, which can account for the different crisis of the condition such as eclampsia, HELLP syndrome and so on **(Cintesun et al., 2018)**.

Although the exact etiology has not been clearly defined, studies have shown that leukocyte activation plays a significant role during the disease process in PET. Significant findings of leukocyte activation have been made, including increased superoxide generation and enhanced integrin CD11b and CD64 expressions in monocytes and in neutrophils in women with PET **(Cintesun et al., 2018)**.

Activated leukocytes also release a variety of substances such as cytokine interleukin-8 (IL-8) and tumor necrosis factor- α (TNF- α), which are capable of mediating endothelial function. Interactions between activated leukocytes, platelets, and vascular endothelium are believed to contribute to the vascular injury in this pregnancy disorder. Furthermore; neutrophil activation is believed to be a major component of exaggerated inflammatory responses in the maternal vascular system during PET **(Kurtoglu et al., 2015; Yıldız et al., 2016)**.

Clearly, cytokines such as interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α) are elevated in PET **(LaMarca et al., 2016)**. These cytokines support the expression of the acute-phase protein C-reactive protein (CRP). CRP is produced in the liver and has been found in amniotic fluid **(Parchim et al., 2015)**.