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Correlation Between Maternal TNF-α Levels and Neonatal Outcomes in Obese Pregnant Women with Term Pregnancy

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Background: Maternal obesity causes chronic low-grade inflammation with elevated TNF-α, potentially disrupting the intrauterine environment and fetal development. Previous studies have primarily focused on preterm inflammation or cytokine levels earlier in pregnancy. However, data on the relationship between maternal TNF-α levels at term pregnancy and immediate neonatal outcomes in obese women remains limited, especially in the Indonesian population. This study evaluates their correlation in term obese pregnancies.

Materials and methods: Maternal serum TNF-α levels were assessed in obese pregnant women with gestational age ≥37 weeks. Blood samples were collected during the third trimester of pregnancy and analyzed using ELISA. Neonatal outcome data were extracted from medical records and evaluated within the first 24 hours of life. Pearson correlation was applied to parametric variables, including maternal TNF-α levels and birth length, while Spearman correlation was used for non-parametric neonatal outcome variables. This cross-sectional study analyzed secondary data from a parent cohort.

Results: Maternal TNF-α levels were not significantly associated with neonatal outcomes (all $p>0.05$). Pearson correlation analysis showed a weak association with birth length ($r = 0.214$). In contrast, Spearman correlation analyses demonstrated weak or negligible associations with birth weight ($\rho = 0.227$), head circumference ($\rho = 0.043$), APGAR score at 1 minute ($\rho = -0.100$), and at 5 minutes ($\rho = -0.014$).

Conclusion: Maternal TNF-α levels were not significantly associated with neonatal outcomes among obese women with term pregnancies. Assessment of inflammatory status limited to the late third trimester may be inadequate to fully evaluate this relationship, indicating the need for future studies with inflammatory measurements initiated earlier in pregnancy.

Keywords: *TNF-α, maternal obesity, neonatal outcomes, APGAR score, term pregnancy*

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Introduction

Maternal obesity is an increasing global health concern and is associated during pregnancy with metabolic disturbances and chronic low-grade inflammation that may affect maternal and neonatal outcomes.[1–5] This study selected maternal tumor necrosis factor-alpha (TNF-α) as an inflammatory marker due to its sensitivity in reflecting obesity-related chronic inflammation, as TNF-α plays a key role in immune regulation, insulin resistance, and endothelial dysfunction. Indonesia was chosen as the study setting because of the rising prevalence of maternal obesity and the limited availability of regional data on inflammatory biomarkers in obese pregnancies.[6,7]

During normal pregnancy, TNF-α plays a physiological role in implantation, placentation, and vascular remodeling. However, excessive maternal TNF-α levels may disrupt placental homeostasis and uteroplacental circulation.[8–10] Elevated TNF-α levels have been reported in obese pregnant women and are associated with placental inflammation and impaired placental function, which may influence fetal growth and development.[11,12]

The placenta functions as a key regulator of fetal development by integrating maternal signals and controlling nutrient transfer as well as inflammatory pathways. While direct transplacental passage of cytokines is limited, persistent maternal inflammation can modify placental structure and function, thereby indirectly influencing fetal growth.[12] Such placental alterations have been associated with adverse neonatal anthropometric outcomes, including variations in birth weight, birth length, and head circumference.[13–16]

Neonatal anthropometric parameters and APGAR scores are widely used indicators of intrauterine growth and early neonatal adaptation.[17,18] Although term pregnancy is generally associated with favorable outcomes, maternal obesity-related inflammation may still affect neonatal health despite delivery at term.[19,20] Evidence regarding the potential correlation between maternal TNF-α levels and neonatal outcomes among obese women with term pregnancy remains relatively scarce, highlighting a significant gap in the current literature and underscoring the need for further comprehensive and well-designed studies to better elucidate this relationship. Therefore, this study aims to evaluate the correlation between maternal TNF-α levels and neonatal outcomes in obese pregnant women with term pregnancy.

Materials and methods

Study Population

Data were collected by obtaining blood samples (n = 44) from obese pregnant women in the third trimester with a gestational age of more than 37 weeks. The number of samples was acquired by the analytic correlation equation. This research was conducted as a secondary analysis of existing data, and all participant information was anonymized prior to analysis. Informed consent had been obtained from all participants in the parent study. The study was carried out in accordance with the principles of the Declaration of Helsinki.

The study population consisted of obese pregnant women with term pregnancies. Data were obtained from the medical records of a parent study conducted between 2024 and early 2025 that examined the association between maternal obesity and placental function. The data on placental function were obtained from examinations, including the TNF-α. Subjects were selected using consecutive sampling based on the parent study. Inclusion criteria were obese pregnant women according to the World Health Organization (WHO) Asia-Pacific Guideline (body mass index ≥27 kg/m²) with singleton term pregnancy (gestational age ≥37 weeks). Exclusion criteria included incomplete medical records, multiple pregnancies, and maternal conditions that could affect inflammatory status, such as chronic inflammatory diseases, autoimmune disorders, acute infections, preeclampsia, and gestational diabetes mellitus.

Data Collection Procedures

Secondary data were obtained from medical records and laboratory reports of a previously conducted study. No additional clinical procedures were performed. Only records containing complete data for all study variables were included in the analysis. Maternal characteristics, including age, parity, body mass index, and gestational age, as well as maternal TNF-α levels and neonatal outcomes (birth weight, birth length, head circumference, and APGAR scores at 1 and 5 minutes), were extracted from medical records, laboratory reports, and delivery room records.

The maternal TNF-α was assessed using an ELISA kit (Human TNF- α ELISA Kit 96T, Cat. E0082Hu, Bt. Lab, Shanghai, China). Maternal TNF-α concentrations were expressed in nanograms per liter (ng/L). All collected data were compiled and analyzed using the SPSS software.

Statistical Analysis

Data analysis was conducted using the Statistical Package for the Social Sciences (SPSS). The normality of data distribution was evaluated using the Shapiro–Wilk test. Normally distributed variables were summarized as mean ± standard deviation, whereas non-normally distributed variables were expressed as median with interquartile range. Associations between maternal TNF-α levels and neonatal outcomes were examined using Pearson's correlation for normally distributed variables and Spearman's rank correlation for variables with non-normal distribution. Statistical significance was defined as a p-value of less than 0.05.

Results

Samples Characteristics

A total of 44 obese pregnant women who met the inclusion and exclusion criteria were included in this study. Most participants were within the non–high-risk maternal age group (20–35 years), 29 (65.9%), while 15 (34.1%) were classified as high-risk (>35 years). Based on parity, 24 (54.5%) were primiparous, and 20 (45.5%) were multiparous. The majority of pregnancies were categorized as early term (38; 86.4%), followed by full term (4; 9.1%) and late term (2; 4.5%). APGAR Score in 1 minute after birth's median was 7, while the 5-minute APGAR score was increased to 9. Anthropometric outcome in this study showed a median of birth weight in this study (3228 g), a mean of birth length (48.1 cm), and a median of head circumference (34 cm) (Table 1).

Maternal Clinical and Laboratory Characteristics

All participants were classified as obese based on the mean pre-pregnancy BMI. Maternal TNF-α levels showed consistently elevated values with moderate variability, accompanied by relatively high leukocyte counts, indicating an inflammatory profile. Blood pressure measurements were generally within the normal range, although some subjects exhibited markedly higher values. Hematological parameters, including platelet count, hemoglobin, and hematocrit, were mostly within acceptable limits despite noticeable inter-individual variation. Random blood glucose levels were largely within the normal range but demonstrated a heterogeneous metabolic pattern among the participants (Table 2). Overall, these findings indicate that the study population consisted of obese pregnant women with variable inflammatory, hematological, hemodynamic, and metabolic profiles at term.

Correlation Analysis of Maternal TNF-α Levels and Neonatal Outcomes

Correlation between maternal TNF-α levels and neonatal outcomes was evaluated. Overall, maternal TNF-α levels showed weak correlations with birth weight, birth length, head circumference, and APGAR scores at 1 and 5 minutes. None of these associations were statistically significant (all $p > 0.05$) (Table 3).

Discussion

Most participants were within the non–high-risk maternal age group (20–35 years), and the majority of pregnancies reached 37–38 weeks of gestation (Table 1). These characteristics suggest that the study population largely consisted of clinically stable obese pregnant women without extreme maternal age risk. Previous studies have demonstrated that maternal obesity, excessive gestational weight gain, and other maternal health conditions are associated with adverse neonatal outcomes, including preterm birth, low birth weight, and increased neonatal risk. However, the magnitude of these effects varies depending on pregnancy characteristics and underlying maternal conditions.[21–23] Maternal obesity has been reported as an independent risk factor for inflammatory dysregulation during pregnancy; however, maternal age and gestational maturity may influence the extent of inflammatory impact on fetal outcomes.[3,5,24]

Previous studies reported that maternal obesity and related metabolic conditions are associated with alterations in maternal physiological and inflammatory profiles, which may influence neonatal outcomes such as birth weight and early neonatal condition. Several studies conducted in Indonesia have examined maternal inflammatory markers and their association with neonatal outcomes. A previous study measured maternal TNF-α and IL-6 levels and reported a weak positive association between TNF-α and birth weight, whereas IL-6 showed no significant correlation with neonatal outcomes. Another study evaluated CRP levels in pregnant women and found that elevated CRP was associated with lower APGAR scores at 1 minute. These findings suggest that maternal inflammatory status may influence neonatal outcomes, but evidence specifically

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Table 1. Sample characteristics based on maternal age, parity, and gestational age.

Variable	Frequency (n)	Percentage (%)
Maternal Age		
Low-risk age	29	65.9
High-risk age		
<20 years	0	0
>35 years	15	34.1
Parity		
Primiparous	24	54.5
Multiparous	20	45.5
Gestational age		
Early term	38	86.4
Full term	4	9.1
Late term	2	4.5
Postterm	0	0

Table 2. Sample characteristics based on APGAR, birth weight, birth length, and head circumference.

Variable	Score Analysis
APGAR Score in 1 minute (Median Q1-Q3)	7 (7—8)
APGAR Score in 5 minutes (Median Q1-Q3)	9 (8—9)
Birth Weight (Median Q1-Q3)	3228 (3000-3486) g
Birth length (Mean±SD)	48.1±2.30 cm
Head circumference (Median (Q1-Q3))	34 (33—34) cm

regarding TNF-α in obese term pregnancies remains limited, highlighting the need for the current study.[25–27]

Maternal health parameters presented in Table 2 demonstrated elevated pre-pregnancy body mass index and increased maternal TNF-α levels, consistent with the inflammatory profile commonly observed in obese pregnancies.[6,11,28] Previous studies have shown that obesity during pregnancy is associated with increased circulating pro-inflammatory cytokines, including TNF-α, which contribute to a chronic low-grade inflammatory state.[29,30] Despite this condition, other maternal laboratory parameters in the present study remained within acceptable clinical ranges, indicating the absence of overt inflammatory or hematological complications.

The correlation analysis presented in Table 3 showed weak and non-significant correlations between maternal TNF-α levels and neonatal anthropometric parameters, including birth weight, birth length, and head

Table 3. Sample characteristics based on APGAR, birth weight, birth length, and head circumference.

Maternal Health Parameters	Min	Max	Mean ± SD
Pre-pregnancy BMI (kg/m²)	26.30	46.10	30.20 ± 4.62
Maternal TNF-α level (ng/L)	69.33	178.90	134.28 ± 24.03
Leukocyte count (/mm³)	6280	20750	11580.45 ± 3.650
Systolic blood pressure (mmHg)	90	216	127.05 ± 22.26
Diastolic blood pressure (mmHg)	58	144	78.64 ± 13.76
Platelet count (/mm³)	50000	380000	247113 ± 65.923
Hemoglobin (g/dL)	9.40	14.40	11.96 ± 1.20
Hematocrit (%)	27.00	41.80	34.58 ± 3.48
Random blood glucose (mg/dL)	68	156	99.18 ± 17.77

circumference. These findings suggest that maternal TNF-α levels were not significantly associated with neonatal anthropometric outcomes in this cohort of obese women with term pregnancies. The placenta plays an important role in regulating fetal exposure to maternal inflammatory factors and supporting fetal growth. At the same time, TNF-α has a prominent role in implantation during the first trimester. Although the reported data of TNF-α increased levels among obese pregnant women are inconsistent, the study revealed that preterm labor risk was increasing due to excessive apoptosis induced by elevated TNF-α levels. Previous studies, highlight the critical role of TNF-α by showing how anti-TNF-α drugs can increase the risk of adverse fetal effects like intrauterine growth restriction. This point is emphasized to underscore the importance of TNF-α homeostasis, as all subjects in our current study were excluded if they were taking such immunomodulating medications.31 Thus, one of the possible explanations for the non-significant findings is the absence of placental function analysis in this secondary study. At the same time, treatments were done since the first trimester, and may interfere with the possibility of adverse fetal outcome among patients with pre-pregnancy obesity.[8,9,12] Besides, the cross-sectional study design failed to observe the trend in TNF-α levels and reduced the significance of the results.

In addition, maternal TNF-α levels demonstrated very weak negative correlations with APGAR scores at 1 and 5 minutes. Although higher TNF-α levels tended to correspond with slightly lower APGAR scores, these associations were not statistically significant. APGAR scores primarily reflect immediate neonatal adaptation influenced by intrapartum and perinatal factors, which may outweigh the effects of maternal inflammatory markers alone.[18,32]

The lack of significant correlations in this study may also be attributed to the inclusion of only term pregnancies. Adverse inflammatory effects on neonatal outcomes are more frequently observed in pathological conditions such as preterm birth, placental insufficiency, or hypertensive disorders of pregnancy.[16,33,34] Maternal obesity is generally associated with adverse neonatal outcomes; however, the magnitude of these effects varies according to pregnancy complications and gestational age at delivery.[23] In term pregnancies, compensatory maternal and placental mechanisms may mitigate the impact of inflammatory cytokines on neonatal outcomes. In addition, the small sample size is a notable limitation in this study, suggesting a bigger sample size and broader assessment in future research.

Overall, this study found no statistically significant correlations between maternal TNF-α levels and neonatal anthropometric outcomes or APGAR scores among obese women with term pregnancies. These findings underscore

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Table 4. Correlation of maternal TNF-α levels with neonatal outcomes.

Neonatal Outcome	r	p
Birth weight	0.227	0.139
Birth length	0.214	0.163
Head circumference	0.043	0.782
APGAR score (1 min)	–0.100	0.518
APGAR score (5 min)	–0.014	0.927

*r = correlation coefficient. Based on normality testing, Pearson correlation was applied for birth length, whereas Spearman's rank correlation was used for birth weight, head circumference, and APGAR scores.

the complexity of maternal fetal inflammatory interactions and suggest that neonatal outcomes may be influenced by multiple factors beyond a single inflammatory marker.[35,36]

Conclusion

This study found weak and non-significant correlations between maternal TNF-α levels and neonatal anthropometric parameters and APGAR scores among obese women with term pregnancies. In this cohort of obese women with term pregnancies, maternal TNF-α levels were not significantly associated with birth weight, birth length, head circumference, or APGAR scores at 1 and 5 minutes. These findings suggest that, in term pregnancies, neonatal outcomes are likely influenced by multifactorial mechanisms, and elevated maternal TNF-α may not independently determine neonatal outcomes.

Authors' Contributions

AZ conceived the study, collected and curated the data, performed the statistical analysis, and drafted the manuscript. YW and HK contributed to the study conception and design, supervised the research process, and provided critical revisions of the manuscript for important intellectual content. AL and AM contributed to refining the research questions, interpreting the findings in the clinical context, and reviewing the final version of the manuscript. All authors read and approved the final manuscript and agreed to be accountable for all aspects of the work.

Ethical Statement

Ethical approval for this study was obtained from the Ethics Committee of the Faculty of Medicine, Universitas Andalas, Padang, Indonesia (Approval No.530/UN.16.2/KEP-FK/2024).

Conflict of Interest

The authors declare that there are no conflicts of interest related to this study.

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