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ANALISIS POLIMORFISME BDNF rs6265 VAL66MET, KADAR BDNF MATERNAL DAN TALI PUSAT DENGAN NEUROBEHAVIORAL NEONATAL PADA IBU HAMIL OBESITAS ETNIK MINANGKABAU

DISERTASI

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ABSTRACT

Introduction

Maternal obesity has the potential to disrupt fetal nervous system development through chronic inflammation, oxidative stress, and intrauterine metabolic changes. Brain-Derived Neurotrophic Factor (BDNF) plays an important role in neurogenesis and synaptic plasticity, while BDNF Val66Met (rs6265) polymorphism can reduce BDNF secretion dependent on neuronal activity.

Methods

An observational analytical study with a cross-sectional design was conducted on 72 Minangkabau ethnic obese full-term pregnant women at Hermina Hospital in Padang. The BDNF Val66Met polymorphism was analyzed using PCR, maternal and umbilical cord blood BDNF levels were measured using ELISA, and neonatal neurobehavioral outcomes were assessed using the Neonatal Behavioral Assessment Scale (NBAS).

Results

Subject characteristics were generally within normal limits, with BMI slightly above normal; leukocyte levels differed significantly between the mutant and wildtype groups ($p=0.034$). The Val66Met polymorphism was dominated by the heterozygous mutant (CT) variant at 54.2%, followed by homozygous mutant (TT) at 26.4%, and wildtype (CC) at 19.4%. The mean umbilical cord blood BDNF level was higher than the maternal level (2.36±0.53 ng/mL vs. 2.04±0.44 ng/mL). All NBAS domains were dominated by the suboptimal category, with the highest suboptimal proportion in the Orientation domain (91.7%) and the lowest in the Autonomic domain (66.7%). No significant differences in maternal or cord blood BDNF levels were found across the three genotype groups ($p=0.217$ and $p=0.947$, respectively). Similarly, no significant correlation was found between maternal BDNF ($p=0.838$) or umbilical cord BDNF ($p=0.119$) levels and NBAS scores.

Discussion

Higher cord blood BDNF levels reflect the placenta's neurotrophic compensatory mechanism in response to obesity. The dominance of suboptimal NBAS outcomes indicates neurobehavioral impairment in neonates. The absence of significant associations is attributable to the multifactorial nature of neonatal neural development and the limitations of a single BDNF measurement in reflecting neurobehavioral outcomes.

Conclusion

The BDNF Val66Met polymorphism in obese Minangkabau pregnant women is dominated by the heterozygous mutant variant. Umbilical cord BDNF levels were higher than maternal levels, while NBAS outcomes were predominantly suboptimal across all domains. No significant associations were found between the Val66Met polymorphism, maternal and cord blood BDNF levels, and NBAS outcomes.

Keywords: BDNF Val66Met; umbilical cord blood BDNF; neonatal neurobehavior; maternal obesity; Minangkabau ethnicity.