Секция «Генетика»

The relationship between clinic-laboratory blood parameters and the association of renin-angiotensin system gene polymorphisms in pregnancies complicated by pre-eclampsia

Научный руководитель – Шкурат Татьяна Павловна

Алаяса Надим Н И

Аспирант

Южный федеральный университет, Академия биологии и биотехнологии им. Дмитрия Иосифовича Ивановского, Кафедра генетики, Ростов-на-Дону, Россия

E-mail: alayasa.nadeim@gmail.com

Pre-eclampsia is a significant issue in modern obstetrics, affecting 2-10% of pregnancies and leading to high maternal and infant morbidity and mortality rates [1]. Despite extensive research, the exact causes of pre-eclampsia remain unclear, emphasizing the importance of biochemistry and molecular genetic studies to understand its pathogenesis and develop early diagnostic and preventive measures.

Liver and renal insufficiency are critical factors in the outcome of pre-eclampsia pregnancies [2], with impaired liver function indicated by elevated levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) [3]. Additionally, alkaline phosphatase (ALP) activity increases in pre-eclampsia due to placental damage, while low ALP levels suggest insufficient placental development [4]. Monitoring serum urea and creatinine levels is crucial for assessing kidney function in pregnant women with pre-eclampsia. The renin-angiotensin system (RAS) also plays a role in pre-eclampsia pathogenesis, with dysfunctional circulating RAS contributing to salt-water balance and placental perfusion [5]. Moreover, genetic factors, including polymorphisms in RAS-related genes, such as *AGT* (C521T and T704C), *AGTR1* (A1166C), and *AGTR2* (G1675A), may influence pre-eclampsia susceptibility, highlighting the need for further research in this area.

Our study aimed to investigate the impact of pre-eclampsia on biochemical blood parameters and explore the association between RAS gene polymorphisms and pre-eclampsia susceptibility in Russian pregnant women. The research involved measuring ALT, AST, ALP, urea, creatinine, and total protein levels in pregnant women with varying degrees of pre-eclampsia severity. Additionally, genotyping for RAS gene polymorphisms was conducted in pre-eclamptic and normotensive pregnant women. The results showed significant increases in ALT, AST, ALP, urea, and creatinine levels, as well as decreased total protein levels in pre-eclampsia pregnancies compared to normal pregnancies. The study also identified an association between the *AGT* (C521T) polymorphism and pre-eclampsia risk, with the T allele and TT genotype being significantly linked to pre-eclampsia ($p=0.005$ and $p=0.034$, respectively). In addition, the multifactor dimensionality reduction (MDR) analysis showed a significant interaction among the four RAS gene polymorphisms and their association with pre-eclampsia risk ($p<0.0001$).

The findings suggest that biochemical blood parameters can serve as diagnostic markers for pre-eclampsia severity, while genetic factors such as RAS gene polymorphisms may contribute to pre-eclampsia susceptibility. Overall, the study underscores the importance of understanding the biochemical and genetic factors involved in pre-eclampsia to improve diagnosis, prevention, and treatment strategies for this serious pregnancy complication.

Источники и литература

1) Mendoza M, Tur H, Garcia-Manau P, Hurtado I, Serrano B, Lopez-Martinez RM, Castellote L, Martinez L, Bonacina E, Carreras E. Cut-off values for Gaussian first-

trimester screening for early-onset preeclampsia with maternal history, biochemical markers and uterine artery Doppler. // Journal of Gynecology Obstetrics and Human Reproduction. 2021, V.50, N1. – P.101827.

2) Wisner K. Gestational hypertension and preeclampsia. // MCN: The American Journal of Maternal/Child Nursing. 2019, V.44, N3. – P.170.
3) Alese MO, Moodley J, Naicker T. Preeclampsia and HELLP syndrome, the role of the liver. // The Journal of Maternal-Fetal & Neonatal Medicine. 2021, V.34, N1. – P.117-23.
4) Delforce SJ, Lumbers ER, Morosin SK, Wang Y, Pringle KG. The Angiotensin II type 1 receptor mediates the effects of low oxygen on early placental angiogenesis. // Placenta. 2019, V.75, P.54-61.