

Genetic aspects of fetoplacental blood flow regulation: the role of the ACE gene in pregnancies lasting more than 40 weeks



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Introduction

The fetoplacental complex is a key link in ensuring adequate exchange between the mother and fetus, and its hemodynamic stability determines the favorable course of pregnancy and childbirth. In prolonged pregnancies (over 40 weeks), signs of placental dysfunction are often observed, which can lead to fetal hypoxia, blood flow disorders, and complications during childbirth. One of the genetic factors that influence the regulation of vascular tone and placental perfusion is the polymorphism of the angiotensin-converting enzyme (ACE) gene, which is involved in the regulation of the renin-angiotensin system. The study of ACE gene variants (I/I, I/D, D/D) provides a better understanding of the mechanisms of individual reactivity of the fetoplacental complex in late pregnancy.

The aim of the study

To evaluate the role of ACE gene polymorphism in regulating the hemodynamics of the fetoplacental complex during pregnancies lasting more than 40 weeks and to determine possible associations between genetic variants and placental blood flow parameters.

Discussion

The ACE gene encodes angiotensin-converting enzyme, which catalyzes the conversion of angiotensin I to angiotensin II, a potent vasoconstrictor that affects vascular tone, plasma volume, and blood pressure.

The I/D polymorphism in the 16th intron of the ACE gene causes different levels of enzyme expression: carriers of the D allele have increased ACE activity, which can lead to vasoconstriction, decreased placental blood flow, and an increased risk of placental insufficiency. In turn, the I allele is associated with lower enzyme activity and more stable hemodynamics. According to recent studies, the D/D genotype is more common among women with prolonged pregnancy, which correlates with signs of fetal hypoxia and decreased fetal-placental blood flow according to Doppler measurements. Thus, the ACE genetic variant may be a marker of risk for placental adaptation disorders in late pregnancy.

Conclusions

ACE gene polymorphism plays a significant role in the formation of individual variability in the hemodynamics of the fetoplacental complex. The identification of risk genotypes may be a promising direction for a personalized approach to the management of women with prolonged pregnancy. Further research into the relationship between genetic factors and placental blood flow is important for the prevention of complications and optimization of obstetric management.