

Symptoms of Preeclampsia and its Prevention by At1-B2 Receptor Heteromers

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DESCRIPTION

Preeclampsia is seen in 7% of pregnant women worldwide. It is a pregnancy complication without any cure. It is the major cause for morbidity in both mother and fetus. It includes several symptoms like

- High blood pressure after 20 weeks pregnancy.
- Signs of damage to another organ.
- Kidney dysfunction which leads to proteinuria.

Among different symptoms, vascular and systemic angiotensin II hypersensitivity is one of the best-established features of preeclampsia. It is also a disease which predisposes mother and infant to develop cardiovascular diseases in future. An increased plasma level of the anti-angiogenic Soluble Fms-Like Tyrosine kinase-1 (sFLT1) is being developed as a diagnostic marker for preeclampsia. However studies are still going on for the pathomechanisms, which trigger this process. The increased plasma level of sFLT1 is due to enhanced angiotensin II-stimulated signalling; another symptom of preeclampsia, the decrease in vascular RGS5 is related to this.

Angiotensin II hypersensitivity

It is a consequence or bystander of preeclampsia because the increased protein aggregation between the angiotensin II AT1 receptor and the bradykinin B2 receptor is the root cause of preeclampsia. This situation has been addressed in transgenic pregnant mice with increased expression of AT1-B2 under SM22-alpha promoter which is specific to smooth muscle. It is observed that the given amount of AT1-B2 was sufficient to develop the symptoms of preeclampsia in that mouse. Not only to angiotensin II stimulation but also to pathomechanical

stimulation AT1-B2 caused vascular hypersensitivity. The role of the angiotensin II hypersensitivity in the treatment of preeclampsia is not known but it contributes to the long-term cardiovascular morbidity of women who have had preeclampsia. Hence a transgenic pregnant mice is taken and in vivo an increased vascular AT1-B2 heteromerization contributes to the angiotensin II hypersensitivity of pregnancies complicated by preeclampsia by an increased angiotensin II-stimulated signalling vascular angiotensin II hypersensitivity was documented ARR1-S412A prevents symptoms of preeclampsia in pregnant Tg-AT1-B2 mice Placental biopsies from pregnancies complicated by preeclampsia show an increased p-S412-ARR1 content, increased FOS and decreased RGS5 expression.

Pregnant mice with smooth muscle-specific AT1-B2 expression develop symptoms of preeclampsia characterization of pregnancies complicated by preeclampsia is performed by increased vascular AT1-B2 heteromerization. The impact of increased vascular AT1-B2 heteromerization on pregnancy outcome in laboratory conditions is also analysed. The target pregnant Tg-AT1-B2 mice developed symptoms of preeclampsia as evidenced by hypertension on beginning of third week approximately on 18th day of pregnancy, low platelet count, and increased levels of circulating sFlt1.

CONCLUSION

Amlodipine could be used for reducing risk of preeclampsia hypertension. It indicates Amlodipine show no side effects on pregnancy. It can also be used to reduce risk in patients in severe conditions of preeclampsia. Long term action of amlodipine reduces blood pressure and also targets AT1-B2 heteromer-mediated signalling as a characteristic feature of preeclampsia.

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