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Cardiovascular and cerebrovascular disease associated microRNAs are dysregulated in placental tissues affected with preeclampsia and intrauterine growth restriction

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Aim: To explore placental tissue expression profile of microRNAs involved in pathogenesis of cardiovascular and cerebrovascular diseases (miR-1, miR-16, miR-17, miR-20a, miR-20b, miR-21, miR-23a, miR-24, miR-26a, miR- 29a, miR-33a, miR-92a, miR-100, miR-103, miR-122, miR-125b, miR-126, miR-130b, miR-133a, miR-143, miR-145, miR-146a, miR-155, miR-181a, miR-195, miR-199a, miR-208, miR-210, miR-221, miR-342-3p, miR-499 and miR- 574-3p) in patients with gestational hypertension (n=35), preeclampsia (n=80) and fetal growth restriction (n=35) by real-time qRT-PCR.

Methods: Cardiovascular microRNA expression profile was correlated with the severity of the disease with respect to clinical signs, requirements for the delivery and Doppler ultrasound parameters.

Results: The down-regulation of 3/32 (miR-26a, miR-103, miR-145) microRNAs was found in preeclampsia before 34 weeks. Mir-1 was up-regulated in preeclamsia after 34 weeks. Mir-499 was dysregulated in the group of gestational hypertension and preeclampsia irrespective of the severity of the disease. Mir-499 up-regulation was detected in mild and severe forms of the disease occurring after 34 weeks. Additionally, the up-regulation of miR-499 was observed in IUGR patients. The difference within the IUGR group with normal and abnormal values of flow rate in the umbilical artery was observed for miR-1, abnormal blood flow velocity waveforms showed increased expression of miR-1.

Conclusion: MicroRNAs playing a role in pathogenesis of dyslipidemia (miR-1), insulin resistance and diabetes (miR-26a, miR-103), atherosclerosis (miR-145), coronary artery disease (miR-1, miR-145), myocardial infarction and heart failure (miR-1, miR-26a and miR-499) were shown for the first time to be dysregulated in preeclampsia. Gestational hypertension and IUGR were associated with the dysregulation of miR-499, involved in pathogenesis of myocardial infarction and heart failure.

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