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Biochemical markers of cardiac dysfunction in pregnancies with placental dysfunction

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Objective

The exact mechanism responsible for fetal smallness in pregnancy is still unclear. Recent studies have revealed that except for pathological placentation, dysfunction of maternal cardiovascular system also plays a role. The aim of this study is to analyse the laboratory markers of cardiac dysfunction in pregnant women with fetal smallness and/or preeclampsia.

Methods

Prospective cohort control study in 211 patients with singleton pregnancies with pregnancy pathology – fetal smallness (EFW < 10. pct (Intergrowth – 21)) or preeclampsia. The control group represents a cohort of 309 physiologic singleton pregnancies. As markers of cardiac dysfunction, we evaluated: NT-proBNP, troponin-T and copeptin (measured on the BRAHMS KRYPTOR Compact analyser).

Results

The average gestational age of symptomatic women was 33. gestational week, in controls it was 34. gestational week. There were no significant differences in age and BMI in both groups. Symptomatic women showed statistically higher blood pressure values (syst. BP: 131mmHg vs. 121 mmHg (p 0,00); diast. BP: 85 mmHg vs. 75 mmHg (p 0,00)). Laboratory markers of cardiac dysfunction NT-proBNP (p 0,00) and troponin-T (p 0,00) were significantly higher in the symptomatic group. After dividing the FGR group to late-onset and early-onset FGR we described significant elevation of NT-proBNP (p 0,00) and troponin T (p 0,029) in women with early-onset FGR but not in patients with late-onset FGR. We observed a significant elevation of copeptin in patients with preeclampsia (p 0,029), but not in patients with FGR.

Conclusion

Patients with FGR showed higher diastolic blood pressure than women with healthy pregancies. According to our data, the laboratory markers of cardiac dysfunction were elevated in comparison with the controls. After diving to early-onset and late-onset FGR, we can assume that in women with early-onset FGR, a damage of cardiovascular system of the mother is present.