Abstract

Heat shock proteins provide a universal stress response to cells, for example, exposed to high temperature, heavy metals, extreme pH, infection, inflammation, oxygen radicals, or in case of lack of nutrients and oxygen. We assumed that stress conditions associated with pregnancy-related complications will result in a change in gene expression profile of heat shock proteins we selected for the study. Heat shock protein mRNA levels (Hsp27, Hsp60, Hsp70, Hsp90α and HspBP1) were tested in placental tissue, whole peripheral venous blood and maternal plasma in women with the following pregnancy-related complications - preeclampsia (PE), fetal growth restriction (FGR), gestational hypertension (GH), spontaneous preterm birth (PTB), preterm premature rupture of membranes (PPROM) and normal pregnancies. We also investigated whether the severity of the disease had any impact on hsp gene expression in particular biological samples.

In placental tissue, overexpression of Hsp27, Hsp90α and HspBP1 was found in patients with mild preeclampsia (that does not require immediate termination of pregnancy if properly treated), and in women with late onset of preeclampsia with clinical manifestation after 34th week of gestation. Concerning preterm birth, overexpression of Hsp27 and Hsp60 was observed in both groups (PTB and PPROM). In addition, downregulation of Hsp70 and HspBP1 was observed in patients with PPROM compared to normal pregnancies. Moreover, HspBP1 showed a change in gene expression between the groups of patients with PTB and PPROM. Strong positive correlation between gene expression of Hsp60 and Hsp70 in placental tissue and CRP levels in maternal sera was observed in the PTB group. Some of the examined hsp mRNA displayed increased levels with advancing gestational age at delivery in placental tissue of patients with PPROM (Hsp27, Hsp70, Hsp90α) and PTB (Hsp27). Maternal circulation reflects pathological conditions of both mother and placenta. In whole peripheral venous blood, Hsp70 was upregulated and Hsp90α was downregulated in PE, FGR and GH compared to normal pregnancy. Upregulation of Hsp60 in whole peripheral venous blood was observed in patients with PE and FGR. Subsequent analysis performed on maternal plasma samples confirmed increased Hsp70 gene expression in PE and GH groups.