Are Placental Cell-Derived Exosomes a Predictive Biomarker of Preeclampsia?

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Abstract

Placenta is endocrine organ that regulate biological function of several maternal tissues and the majority of fetal organs across gestation through releasing of extracellular vesicles incorporated into secretom [1]. Indeed, circulating number of exosomes derived from placenta cells in healthy pregnancies increases in 50-100 fold times to healthy volunteers and they may be detected in several biological fluids across all gestation age starting with 6 weeks of gestation. Interestingly, asymptomatic pregnant woman at risk of preeclampsia may demonstrate extremely increased levels of total exosomes and placental cell-derived (CD63+) exosomes (PCDE) in blood when compared with woman at risk free of preeclampsia. In has been suggested that the risk of preeclampsia could associate with an altered relation between various phenotypes of PCDE. The circulating exosomes regulating fetal vascular response play a pivotal role in motility and proliferation of placenta, endothelial dysfunction, cell proliferation, inflammation, angiogenisis and neangiogenisis [4,5]. They are key factors contributing in endogenous vascular reparative processes and adaptation to hypoxia / ischemia-induced injury of cytotrophoblast [5]. Depending on their origin (activated or apoptotic fetal cells) exosomes appear to be distinguished in morphology, immune phenotypes, abilities to cargo some molecules (active proteins, pro-coagulants, growth factors, lipids, enzymes, micro-RNAs) and induce autocrine / paracrine effects on vasculature and trophoblast. Interestingly, circulating number of placental cell-derived-exosomes could be an individual probe for personalization of a risk of life-threatening event across pregnancy. The short communication is depicted the role of placental cell-derived-exosomes as biomarker of preeclampsia in asymptomatic pregnancies.

Keywords: Pregnancy; Preeclampsia; Exosomes; Vascular complications; Prediction, Biomarkers

Recent clinical studies have shown that placental cells may regulate biological function of maternal tissues and the majority of fetal organs across gestation through releasing of extracellular vesicles incorporated into secretom [1]. Indeed, circulating number of exosomes derived from placenta cells in healthy pregnancies increases in 50-100 fold times to healthy volunteers and they may be detected in several biological fluids across all gestation age starting with 6 weeks of gestation. Interestingly, asymptomatic pregnant woman at risk of preeclampsia may demonstrate extremely increased levels of total exosomes and placental cell-derived (CD63+) exosomes (PCDE) in blood when compared with woman at risk free of preeclampsia. In has been suggested that the risk of preeclampsia could associate with an altered relation between various phenotypes of PCDE. The circulating exosomes regulating fetal vascular response play a pivotal role in motility and proliferation of placenta, endothelial dysfunction, cell proliferation, inflammation, angiogenisis and neangiogenisis [4,5]. They are key factors contributing in endogenous vascular reparative processes and adaptation to hypoxia / ischemia-induced injury of cytotrophoblast [5]. Depending on their origin (activated or apoptotic fetal cells) exosomes appear to be distinguished in morphology, immune phenotypes, abilities to cargo some molecules (active proteins, pro-coagulants, growth factors, lipids, enzymes, micro-RNAs) and induce autocrine / paracrine effects on vasculature and trophoblast [6,7]. Apart from hypoxia /ischemia, insulin resistance, impaired fasting glucose, lipid toxicity and inflammation are recognized main regulators of cell secretom including exosomes [8]. On this occasion, a final effect of PCDE on target cells could be related to their structure, which is modified by several triggers. It has been suggested that apoptosis of fetal cells may lead to secretion of exosomes that could directly worse endothelial function contributing in preeclampsia, while PCDE secreted by activated fetal cells may attenuate repARATION and angiogenesis. Interestingly, there was no found increased activity of some anti-apoptotic factors, i.e., tissue matrix metalloproteinase inhibitor and survivin (a member of apoptosis proteins family inhibitors), which are actively transferred by exosomes, in physiological pregnancy. These anti-apoptotic molecules suppress caspases, influence on expression of vascular endothelial growth factor(s) and promote proliferative capability of endothelial cells and their precursors depending on gestation age [9], while their role in preeclampsia are still uncertain. Consequently, the results received recently [2] maintain the hypothesis that impaired immune phenotype of PCDE may be a personal biomarker of preeclampsia as life-threatening vascular complication in pregnancy.

References


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