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Cellular Stress in Women with Chronic Venous Disease during Pregnancy

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Abstract

The time of a woman's life during pregnancy during which the circulatory system experiences hemodynamic and biochemical changes is known as pregnancy. There is a higher risk of developing chronic venous disease (CVD) at this time due to the remodelling of blood arteries and the exchange of maternal-fetal products. CVD may have long-term effects on both the mother and the baby after delivery. Previously, we looked into how pregnancy-associated CVD differs from healthy controls (HC) who have no history of the disease in terms of placental architecture at angiogenesis, lymphangiogenesis and villi morphology. Through the use of multiple markers, we wanted to more thoroughly explore the oxidative stress response in the placenta from women with CVD versus HC. A prospective, analytical and observational cohort study including 114 pregnant women was carried out (32 weeks). 62 patients in all had a clinical diagnosis of CVD. 52 healthy controls (HC) who had no prior history of CVD were also investigated. Real-time polymerase chain reaction and immunohistochemistry were used to examine the gene and protein expressions of NRF2, KEAP1, CUL3 and GSK-3. While Keap1, CUL-3 and GSK-3 gene and protein expressions were significantly reduced in the placental villi of women with CVD, Nrf2 gene and protein expressions were significantly higher. In the placenta of women with CVD, our findings identified abnormal gene and protein expression of Nrf2 and some of its primary regulators, Keap1, CUL-3 and GSK-3, which may be a sign of the oxidative environment seen in this tissue.

Keywords: Chronic venous disease • Placenta • Oxidative stress

Introduction

During her pregnancy, a woman's circulatory system experiences changes in both its hemodynamics and its biochemistry. There is an increased risk of developing chronic venous disease (CVD) during this time period due to the reorganization of blood vessels and the exchange of maternal and fetal products. CVD may have an impact on life after childbirth for both the mother and the child. In comparison to healthy controls (HC) without a history of cardiovascular disease, we previously investigated whether pregnancy-associated CVD involves changes in placental architecture at angiogenesis, lymphangiogenesis and villi morphology. Using a number of markers (NRF2, KEAP1, CUL3 and GSK-3) we wanted to investigate the oxidative stress response in placentas of women with CVD versus HC in greater depth. 114 pregnant women in their third trimester (32 weeks) were the subjects of a prospective cohort study that combined observation, analysis and observational methods. CVD was clinically diagnosed with 62 participants. 52 controls (HC) with no CVD history were studied concurrently. Real-time polymerase chain reaction (RT-qPCR) and immunohistochemistry were used to examine the gene and protein expressions of NRF2, KEAP1, CUL3 and GSK-3.Keap1, CUL-3 and GSK-3 gene and protein expressions were significantly lower in placental villi of women with CVD than Nrf2 gene and protein expressions. The abnormal gene and protein expression of Nrf2 and some of their main regulators-Keap1, CUL-3 and GSK-3-in the placenta of women with CVD was defined by our findings. This could be a sign that the tissue has an oxidative environment [1-3].

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A woman's circulatory system is subjected to hemodynamical, mechanical and biochemical changes during her pregnancy. During this period, while rebuilding veins and trading maternal-fetal items, there is an expanded gamble of creating constant venous illness (CVD). This could be because the diameter of veins tends to grow during pregnancy. However, despite a significant decrease in diameter following childbirth, veins do not return to their original size, particularly in pregnant women undergoing CVD. The risk of developing cardiovascular disease (CVD) during pregnancy can increase with the number of pregnancies; approximately 40% of pregnant women suffer from this condition. As ambulatory venous hypertension progresses over time in patients with CVD, venous return from the lower extremities becomes challenging. Based on the clinical manifestations, cardiovascular disease (CVD) can be categorized using the Clinical-Etiology-Anatomy-Pathophysiology (CEAP) criteria. CVD is thought to result from systemic stress on the placenta during pregnancy, according to previous research. Pregnancy-related cardiovascular disease, for instance, has been linked to changes in placental villous architecture and increased apoptosis. Similar to healthy controls (HC) without a history of cardiovascular disease (CVD), pregnancy-associated CVD involves alterations in lymphangiogenesis, angiogenesis and the extracellular matrix in multiple ways. Overall, these factors may have an adverse effect not only on the mother's life during and after childbirth but also on the development of the fetus [4].

Description

We, first and foremost, identified an improved Nrf2 articulation in the placenta of ladies with CVD in examination with their sound controls. Numerous genes involved in the cellular stress response are controlled by Nrf2, a major transcription factor. As a result, the body's antioxidant response depends on Nrf2, which also has an impact on detoxification, metabolism and inflammation. Thus, it is broadly acknowledged that unsettling influences in the Nrf2 framework address a significant driver of a few pathologies,

including provocative, metabolic and cardiovascular problems. In a similar vein, trophoblast behavior and function have been adversely affected by Nrf2 overexpression or downregulation in a number of pregnancy-related complications, including gestational diabetes mellitus, intrauterine growth restriction, reproductive toxicity, preeclampsia and preterm birth. According to our research, oxidative stress, which causes cellular damage to nucleic acids, lipids and proteins, is a major cause of many obstetric complications. This could result in significant changes in fetal programming through epigenetic mechanisms, which could have potential pathogenic implications not only for the mother but also for the fetus. In contrast, previous research supports the dual role that oxidative stress may play in pregnancy complications by demonstrating that reduced Nrf2 activity appears to increase angiogenesis in placental tissue and improve maternal and fetal outcomes in animal models of preeclampsia. The obstetric complication may have different causes of oxidative stress. For instance, the placenta is the primary source of free radicals and oxidative stress in preeclampsia. Patients with cardiovascular disease (CVD) frequently exhibit signs of both local and systemic oxidative stress. This appears to have negative effects on the placenta and umbilical cord, as well as increased detection of lipid peroxidation in the blood of the mother and a decrease in the pH of the fetus. Based on these findings, we hypothesize that cardiovascular disease is linked to systemic oxidative stress, which may have negative effects on the health of the mother and child. As a result, Nrf2 may be a sign that the placenta tissue has been exposed to significant stressors related to CVD; however, the effects of Nrf2 dysregulation on complications during pregnancy are still poorly understood [5,6].

Conclusion

There is an auto regulatory loop between Nrf2 and the Keap1/CUL3 complex in physiological conditions. However, electrophiles and free radicals appear to disturb this homeostatic equilibrium. The exposure to electrophilic stimuli led to an increase in Nrf2 without affecting the levels of Keap1 and CUL-3, starting from a basal point in which Nrf2 levels were lower than those of Keap1 and CUL-3.As a result, patients with cardiovascular disease may have higher levels of Nrf2, but the mechanisms underlying the homeostatic loss between this component and the Keap1/CUL-3 complex are still unclear. Non-coding RNAs, such as microRNAs (miRNAs) and long non-coding RNAs (lncRNAs), have been shown to play a role in the regulation of oxidative stress and have potential translational applications. Here, we propose a possible implication for future research. Keap1 and CUL3 levels have been found to be altered in preeclampsia and other pregnancy complications in previous studies, suggesting that they may play a role in the pathophysiology of obstetric diseases. This could have the same pathophysiological effects that CVD women's placentas show. For instance, as has been observed in the placenta of women with CVD, it appears that reduced expression of CUL-3 can be associated with matrix remodeling in the placental tissue. However, a lipidome-significant effect of Keap1 deficiency is associated with a fasted metabolic state. In this sense, we have previously reported that the lipidomic profile of the placenta of women with cardiovascular disease is significantly different from that of women without disease. To better comprehend the pathophysiological function of these components in placental tissue, additional research is required.

Acknowledgement

None.

Conflict of Interest

None.

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