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Study of Oxidative Stress during Pregnancy

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Abstract

Oxidative stress is defined as an imbalance in the balance between antioxidants and pro-oxidants in favor of antioxidants. The increase in lipid peroxidation and the significant decrease in GSH and total antioxidant power ORAC in the serum and erythrocytes of pregnant women especially during the third trimester of pregnancy clearly show the evolution of the state of stress oxidative therapy associated with pregnancy in pregnant women. So pregnancy is a physiological state characterized by oxidative disturbance that contributes to the initiation and progression of complications associated with pregnancy.

Keywords : Pregnancy; Diagnosis; Fetus; Oxidative stress

Introduction

Pregnancy is a period of intense physical and physiological changes, which is accompanied by certain changes in the maternal organism from fertilization to childbirth and during which the embryo and then the fetus develops in the maternal uterus [1,2]. It is characterized by physiological changes related to the development and growth of the fetus, maternal adaptation to the pregnancy, preparation of the mother at childbirth, maintenance of maternal homeostasis and preparation for breastfeeding [3]. This event is accompanied by serious health risks, even for women with no previous health problems [4]. Oxidative stress is defined as an imbalance in the balance between antioxidants and pro-oxidants in favor of antioxidants [5]. Antioxidants play a major role in protecting against molecular oxidative damage [6]. Indeed pregnancy exposes too many complications that can be related to an alteration of oxidative stress that is also associated with the appearance of several pathologies during pregnancy. Oxidative stress is considered a risk factor during pregnancy [7].

Oxidative Stress and Pregnancy Complications

Radical phenomena play an important role in the reproduction, the nesting of the fertilized egg and the development of the embryo. But an imbalance between their production, intense during gestation, and their elimination can generate oxidative stress [8]. On the other hand, iron supplementation during pregnancy contributes to the increase of oxidative stress in pregnant women who take it, in particular an increase in maternal and placental plasma MDA [9]. Oxidative stress is closely related to the clinical severity of pregnancy-induced nausea and vomiting and suggests that the evaluation of markers of total oxidant/antioxidant status would be effective as an additional diagnostic of this symptomatology [10]. Gestational diabetes and fetal macrosomia are associated with a decline in antioxidant status regulation and macrosomia is associated with altered lipid metabolism [11]. Increased oxidative stress is a widely accepted participant in the development and progression of diabetic tissue damage and induced changes in the activities of antioxidant enzymes in various tissues [12]. Pre-eclampsia and pregnancy hypertension are a major cause of maternal mortality and morbidity, and are often the cause of premature childbirth. Oxidative stress is considered to be one of the factors inducing this pathology. The increase in the level of lipid peroxidation, particularly MDA in pre-eclampsia patients, is due to the decline in antioxidant defenses (SOD, catalase and glutathione peroxidase and glutathione reductase) [13].

Oxidative Stress and Fetal Development

Disruption of maternal antioxidant vitamin status during pregnancy can affect fetal development [14]. Reactive oxygen species (EOR) act as primary or secondary messengers on both growth and cell death. Several studies have been able to demonstrate the important and direct role of ROS on development because the redox status acts on the regulation of certain transcription factors that influence pathological cellular signaling due to proliferation and erroneous differentiation evolving towards apoptosis. However, oxidative stress could alter several reactions that affect embryonic development [15]. Oxidative stress may even exist during intrauterine life in the fetus [16]. Pregnancy is also associated with a significant increase in oxidative stress [17]. In addition, oxidative stress could alter several reactions that affect embryonic development [18].

Oxidative Stress and Placenta

The placenta, a hormone-rich tissue, is an important source of pro-oxidizing agents, but also of antioxidant enzymes, but it is capable of maintaining lipid peroxidation under control which increases during normal pregnancy [19,20]. In addition, disturbances in the maternal compartment can affect the placental gene methylation state and increase placental oxidative stress, resulting in changes in placental function [21].

Oxidative stress and childbirth

During childbirth, the woman is confronted with a major oxidative stress, where pro-oxidants come from the placenta [22]. However, caesarean section does not induce more oxidative stress than low childbirth [23]. Oxidative stress in Utero can be a determining factor in the mortality and morbidity of premature newborns. Disruption of maternal and placental redox status predisposes to premature childbirth. We also note that spontaneous abortion is multifactorial and among its causes, oxidative stress plays a role during pregnancy [24].

Discussion

Pregnancy is a physiological condition in women that is accompanied by physiological and organic changes from fertilization to childbirth. Oxidative stress is a damage caused by an increase in free radicals and oxidants such as reactive oxygen species [25]. MDA elevation during pregnancy is based on the products of lipid peroxidation produced in the placenta during pregnancy could pass into maternal blood and act as agents triggering damage in other tissues because; the human placenta produces lipid peroxides which are secreted mainly on the maternal side of the placenta and remain in the maternal circulation for some time and increased lipid peroxidation markers are observed during normal pregnancy [26-28].

In addition, the concentration of lipid peroxidation markers is decreased maternal blood after the placenta delivered during childbirth [29]. On the other hand, during hypoxia, the decrease of oxygen at the last mitochondrial complex (Complex IV-cytochrome oxidase) would imply a decrease in the activity of this complex and an increased production of ERO at the level of the complex III mitochondrial with an abrupt rise in the amount of superoxide anion (O2-) that escapes from the mitochondria [30,31]. This decrease in cytochrome oxidase activity is accompanied by a decrease in cellular metabolism [32]. This may explain the increased concentration of serum and erythrocyte MDA in the third trimester of pregnancy. Study of Walsh & Wang [27] reported a deficiency of glutathione peroxidase (GPx) activity during pregnancy due to decreased concentration of its cofactor glutathione to convert lipid peroxidation [33].

NADPH is required to maintain a normal redox status of GSSG / GSH which is considered indicative of oxidative stress [34,35]. Glutathione (GSH) participate in the cellular defense system against oxidative stress by scavenge free radicals and reactive oxygen intermediates as a co-substrate for glutathione peroxide (GPx), which explained decreased GSH concentration with increased oxidative stress [36]. Therefore the intracellular concentration of GSH probably decreases due to increased cellular demand for NADPH. In addition, the increase in MDA associated with the increase in the source of the free radicals of which the NADPH oxidize which is a membrane enzymatic complex in the vascular wall catalyses the oxidation reaction of NADPH by the oxygen (O2), producing NADP+, H+ and O2- there is also activation of NADP-dependent thioredoxin system, where they are associated with NADPH and an enzyme called NADP-thioredoxinreductase [37,38]. The activation of these enzymes may be accompanied by a decrease in NADPH resources to the detriment of other reactions which also require this cofactor, among which glutathione reductase [39]. Because the amount of NADPH available to regenerate GSSG in GSH is largely decreased significantly reducing the non-enzymatic antioxidant defense system (GSH) [40].

Conclusion

The pregnant woman is exposed to significant risks of complications that can affect her health and that of her newborn. On the other hand, an imbalance of the oxidative/antioxidant balance in pregnant women. This is marked by variations of the antioxidant defense system. Pregnancy is a physiological case characterized by a fragility of defense against prooxidizing agents which causes a disturbance of oxidizing/antioxidant status. These disturbances can be responsible for important materno-fetal complications during pregnancy (pre-eclampsia, miscarriage, abortion, gestational diabetes...), and justify the interest of special surveillance.

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