

An Institutional Investigation of oxidative and antioxidant State in Pregnant Women

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Received: 10-04-2022 / Revised: 16-05-2022 / Accepted: 26-05-2022

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Conflict of interest: Nil

Abstract

Background: The imbalance between reactive oxygen species formation and antioxidant defense capabilities is known as oxidative stress. The pathophysiology of oxidative stress in pregnancy has been connected to a variety of diseases.

Aims and Objectives: The goal of this research was to learn more about the role of antioxidant capacity in pregnant women and how oxidative stress causes free radicals.

Material and methods: The current research included 150 individuals with normal pregnancies who were admitted to the Department of Obstetrics and Gynecology. Pregnant women's antioxidant capacity and oxidative state were assessed in serum and whole blood.

Results: According to the findings, the mean age was 22.65 years old, with a gestational age of 36.75 weeks. NO RNI, a reactive species, was found in higher concentrations in the serum of the complete patient pregnant female. When compared to the control category, there was no significant difference in the antioxidants examined (GST, GSH, SOD, GPx). When compared to the control category, pregnant women's TBARS levels were significantly higher.

Conclusion: Changes in free radical and antioxidant defenses occur as a result of body and circulation changes that occur during pregnancy, according to this research.

Keywords: Antioxidants; free radicals, pregnancy; Malonyldialdehyde.

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Introduction

The physiological and metabolic processes of a female's life are known to change during pregnancy. Pregnancy is well-known for exacerbating oxidative stress, a process caused by a typical systemic inflammatory response that results in elevated levels of circulating reactive oxygen species (ROS), which can cause tissue damage. When the

balance between the creation of reactive species in living organisms and the antioxidant power regulated by both antioxidant enzymes and antioxidants involved is disrupted, oxidative stress develops. Antioxidants are chemicals that inhibit the initiation or spread of harm caused by free radicals. Endogenous

enzymes such as Super-oxide Dismutase (SOD), Catalase (CAT), and Glutathione Peroxidase (GPX) are enzymatic antioxidants [1,2]. Non-enzymatic antioxidants such as vitamin C and vitamin E are derived through external food sources. Pregnancy may induce an oxidative imbalance due to the raised metabolic demand. Eclampsia, miscarriages, and other pregnancy problems can be caused by oxidant im-balance. Because pregnancy needs a high level of energy expenditure, the maternal antioxidant system requires special attention in order to combat any potential issues. Previous research has found that during normal, uncomplicated pregnancies, changes in antioxidant defense mechanisms and raised metabolic demand lead to an increase in reactive oxygen species production. Although free radical production is a normal physiological process, an excess of them can react with lipids and cause lipid peroxidation. Antioxidants work by inhibiting the creation of scavenge radicals or increasing their breakdown to reduce tissue damage caused by free radicals. Antioxidant enzymes such as SOD, glutathione peroxidase (GPx), and catalase (CAT) are the first line of defense against ROS, metabolizing reactive oxygen species to harmless by-products. Direct detection of ROS levels; indirect measurement of protein, lipid, and DNA damage instead of assessing oxidative stress; and assessment of antioxidant status, which can be an indirect approach for detecting oxidative stress [3-5].

Aims and Objectives

The goal of this research was to look at oxidative stress and antioxidant systems in pregnant women.

Material and Methods

The participants in this research were 150 patients hospitalized to the Department of Obstetrics and Gynecology. Before

beginning the trial, all patients provided written informed permission and the Ethical Committee approved it. A thorough history was taken, as well as a general physical examination and an obstetric examination. Women who were pregnant and had one of the following conditions were excluded from the research: Bleeding disorders, women taking nonsteroidal anti-inflammatory medicines like aspirin, splenomegaly, connective tissue illness like SLE, hypertension, HIV, and hepatitis B infection are all factors to consider. The clinical records were used to extract information such as medication history, splenomegaly, and HIV/hepatitis B status.

Specimen: A blood sample (at random) was taken from each pregnant lady, and a serum sample was prepared by centrifuging blood samples at 2000rpm for 10 minutes and storing it at 20 degrees until analysis. The Griess method was improved by Fiddler to measure serum nitric oxide in terms of its products, nitrite and nitrate. 8 GSH in plasma was determined using the Thomas and Skrinska method, which is based on GSH reacting with 5,5', dithiobisnitrobenzoic acid to generate a compound that absorbs at 412 nm. The results were given in milligrams per deciliter of plasma. Draper and Hadley's approach was used to calculate MDA in plasma. Colorimetrically, the color produced by the interaction of thiobarbituric acid with MDA was detected at 533 nm. The results were expressed as nmoles/ml plasma.

Statistical Analysis

Statistical analysis was carried out using the SPSS Package version. To determine the relationship between two categories, simple proportions, mean, standard deviation, Student "t" test, and Chi-square test were utilized. Statistical significance is defined as a P value of less than 0.05.

Results

Table 1: Comparison of characteristics and clinical features in between normal healthy individuals and patients categorys.

Parameters	Mean \pm SD
age (years)	22.32 \pm 2.24
Parity	2.14 \pm 0.26
Gestational age (weeks)	36.38 \pm 2.27

Table 1 lists the general characteristics of the pregnant women who took part in the research. The mean age of the participants in this research was 22.32 \pm 2.24 years. The mean parity of the research subjects was 2.07. The female's gestational age was 36.38 \pm 2.27 weeks. (See Table 1)

Table 2: Estimation of Nitric Oxide (NO) levels ($\mu\text{mol/dl}$) and Reactive Nitrogen Intermediates (RNI) levels (nmol/ml) in the control and patient categorys.

Table 2

Biochemical parameters	Control	1st trimester	2nd trimester	3rd trimester
Nitric oxide ($\mu\text{mol/dl}$)	0.50 \pm 0.08	0.38 \pm 0.02	1.25 \pm 0.04*	1.38 \pm 0.52*
RNI	324.92 \pm 21.42	339.24 \pm 24.19*	348.90 \pm 29.39	407.06 \pm 42.36**

The total NO concentration in the serum of the entire sick pregnant female was greater. Between the patients and the control category, there were statistically significant variations in total NO concentrations in the serum. Similarly, the levels of Reactive Nitrogen Intermediates (RNI) in the patients category raised significantly when compared to the control category (Table 2). In comparison to the control category, the increase in the patients category was much higher. Data is presented as Mean SE, with *p 0.05 denoting significance and **p 0.01, denoting extreme significance.

Table 3: Estimation of antioxidant levels in the control and patient categorys

Biochemical parameters	Control	1st trimester	2nd trimester	3rd trimester
GSH (mg/dl)	11.42 \pm 0.42	12.04 \pm 0.24	12.22 \pm 0.26	13.42 \pm 0.30
SOD (units/100 mg protein)	23.24 \pm 1.8	23.4 \pm 1.40	24.70 \pm 1.26	24.26 \pm 2.14
GST (mg/dl)	10.46 \pm 0.36	10.22 \pm 1.08	11.27 \pm 1.42	11.42 \pm 1.41
GPx (units/mg protein)	15.40 \pm 2.26	15.40 \pm 1.44	16.76 \pm 1.38	16.30 \pm 1.45

All biochemical indicators evaluated (GST, GSH, SOD, GPx) showed no significant differences from baseline levels of antioxidants. When compared to the relevant control categorys, the patients in all trimesters did not demonstrate any significant decline in any of the examined parameters (Table 3). Lipid peroxidation was not observed in the control category. When compared to the control category, pregnant women had a substantial increase in lipid peroxidation products (TBARS).

The data is presented as Mean SE, with a significance level of *p 0.05.

Discussion

In this work, the oxidative stress and antioxidant mechanisms in pregnant women are demonstrated. Pregnancy is characterized by a high metabolic load and raised tissue oxygen requirements, resulting in an increase in reactive oxygen species production. Monitoring oxidative stress in pregnant women is critical for gaining a

better understanding of the link between oxidative stress and pregnancy outcomes [6-8]. Free radicals produced from oxygen participate in redox processes that lead to oxidative changes in biomolecules, with proteins and lipids being the most common targets.

Pregnancy is a normal part of life. In a normal pregnancy, the developing foetus develops in a low oxygen environment called physiological hypoxia of the early gestational sac, which is beneficial because it protects the developing foetus from the harmful and teratogenic effects of excessive ROS stimulation throughout all phases of pregnancy can result in hyper-glycemia, IUGR, miscarriage, and spontaneous abortion [9,10]. Placental oxidative stress is caused by a number of causes, including maternal history, genetics, and environmental variables, and it can lead to poor pregnancy outcomes. The concentrations of total NO and Reactive Nitrogen Intermediates were greater in the serum of all pregnant women in the current investigation, which was statistically significant. The negative effects of reactive species during pregnancy can have an impact on the pregnancy's progress, the development of the foetus, and the infant's health after birth. As a result, antioxidants, which play a vital role in scavenging activity, are always needed as a defense mechanism [11-13].

Antioxidants are substances or molecules that can reduce or prevent the harmful effects of oxidants. Micronutrients such as vitamins and trace elements, as well as metallo-enzymes such as catalase, super-oxide dismutase, and glutathione peroxidase, are among these compounds. Super-oxide dismutase levels have been reported to rise during normal pregnancy [14,15]. The most well-known natural antioxidant enzymes that can remove ROS are super-oxide dismutase and glutathione peroxidase.

During the initial months of pregnancy, SOD activity in the blood begins to rise. SOD protects embryos against lipid peroxidation, which is particularly crucial early in pregnancy. Serum SOD activity is lower in preeclampsia patients than in healthy pregnant women. The SOD level in placental homogenization is low in the early stages of pregnancy, but it can grow by 2-3 times as the pregnancy progresses. The shift in placental SOD levels during pregnancy is attributed to the change in placental oxygen requirements. Placental SOD activity was lower in placenta-related diseases than in healthy pregnant women. Jenkins discovered that serum SOD activity in preeclampsia categories was statistically considerably higher than in the control category in his research [16].

In preeclampsia, however, SOD activity was higher, lower, and no significant differences were seen in comparison to the control category, according to the literature. The most well-known natural antioxidant enzymes that can remove ROS are super-oxide dismutase (SOD) and glutathione peroxidase (GPx).

When compared to control patients, antioxidant enzyme (SOD, GST, GPx) activities were higher in this research. The current research's findings clearly demonstrated that circulating levels of thiobarbituric acid-reactive compounds are much lower in women who are pregnant normally, and that these levels grow as the gestation period progresses. Earlier investigations had made similar observations [17,18].

Conclusion

To summarize, oxidative stress in the form of reactive oxygen species (ROS) is a major contributor to a wide range of pregnancy problems. Despite the fact that the antioxidant defense is designed to restrict the production of ROS, the raised amount of

ROS cannot be controlled, resulting in oxidative stress. Future research should focus on improving the breakdown of intracellular ROS and enhancing antioxidant bioavailability to lower the occurrence of reproductive diseases.

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