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Evaluation Of Oxidative Stress in Postmenopausal Iraqi Women

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Abstract

Oxidative stress is a physiological imbalance caused by reactive oxygen species (ROS) generation and the body's capacity to neutralize or detoxify them. Postmenopausal women are one of the many demographic groups impacted by oxidative stress. This study aimed to evaluate oxidative stress in postmenopausal women and compare it with fertile women. A cross-sectional comparative study was conducted on 50 Iraqi women from the city of Shatrah, Iraq, of which 25 were postmenopausal women with an age range of 46-51 years and 25 fertile women with an age range of 40-45 years. The results indicated a highly significant increase ($p < 0.01$) in MDA levels and body fat percentage and a highly significant decrease ($p < 0.01$) in SOD and CAT levels in postmenopausal women compared to fertile women, while there was a non-significant difference ($p > 0.05$) in GPX levels. Regarding the correlation between oxidative stress parameters and body fat percentage, the results showed a positive significant correlation with MDA ($r = 0.5$, $p = 0.012$) and a negative significant correlation with SOD ($r = -0.444$, $p = 0.026$) and CAT ($r = -0.575$, $p = 0.002$), while there was a non-significant correlation with GPX ($r = -0.257$, $p = 0.214$). The study concluded that postmenopausal women suffer from an increased level of oxidative stress, which makes them more susceptible to several diseases such as osteoporosis, cardiovascular disease, and metabolic disorders.

Keywords

Oxidative stress, Oxidative status, Postmenopausal, Fertile, Antioxidants

Introduction

Oxidative stress is a physiological state caused by an imbalance in the creation of reactive oxygen species (ROS) and the body's ability to neutralize or repair their negative consequences [1]. ROS, which comprise chemicals like superoxide radicals, hydrogen peroxide, and hydroxyl radicals, are naturally occurring by products of cellular metabolism [2, 3]. While these molecules are necessary for cellular signaling and pathogen defense, an excess of ROS can cause oxidative stress, which can damage lipids, proteins, and DNA [4].



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Maintaining cellular health requires a precise balance between the body's antioxidant defense mechanisms and the creation of reactive oxygen species (ROS) [5]. Numerous diseases, including cancer, heart disease, neurological problems, aging, and other ailments, have been linked to oxidative stress in their pathogenesis [6].

The postmenopausal phase is a pivotal period in a woman's life, characterized by considerable physiological changes [7]. Oxidative stress is a complicated process that influences postmenopausal health [8]. As women enter menopause, changes in hormonal profiles, particularly a drop in estrogen levels, can increase sensitivity to oxidative stress [9].

Determining the effects of oxidative stress on general health and well-being in postmenopausal women requires an understanding of its dynamics [10]. Numerous chronic age-related illnesses, such as osteoporosis, neurological disorders, and cardiovascular diseases, have been linked to oxidative stress [11]. Examining the particular pathways and indicators linked to oxidative stress in women who have gone through menopause might offer a significant understanding of the underlying pathophysiology and suggest future directions for focused therapies [8, 12].

This study aims to investigate and assess oxidative stress in Iraqi postmenopausal women using a thorough examination of oxidative stress parameters, physiological parameters, and lifestyle factors. by clarifying the complex relationship between oxidative stress and hormonal changes. Oral and written informed consents were obtained from all women participating in this study after informing them of the objectives and methods of the study. The study was also conducted according to ethics guidelines stipulated in the Helsinki Declaration in 1964 and its subsequent amendments.

Methods

Participants

Twenty-five postmenopausal women aged 46-51 years from Shatrah City, Iraq, participated in this study, and twenty-five fertile women aged 40-45 years were selected as a control group. All participants in this study did not use hormonal or antioxidant treatments before three months of the study, and participants who suffered from diabetes, high blood pressure, heart disease, and other chronic diseases were excluded.

Specimens:

Following an 8-hour fast, blood samples were taken from each participant in the study between 8:00 AM and 9:00 AM. A gel tube was filled with about 5 ml of blood from a forearm vein, which was then left to coagulate at room temperature. The serum was then extracted after being



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centrifuged for five minutes at 4000 rpm to using for determine the oxidative stress parameters used during this investigation.

Estimation of biochemical parameters

The HumaReader HS system (Human®-Germany) was utilized to assess serum MDA, SOD, CAT, and GPX in both study groups through laboratory tests using a human ELISA kit (Elabscience®-USA).

In addition, the body fat percentage was calculated using the formula [13]:
$$\text{Body Fat\%} = (1.2 \times \text{BMI}) + (0.23 \times \text{age}) - 5.4 - (10.8 \times 1) \quad (1)$$

Also, the Body Mass Index was computed using the formula [14]:

$$\text{BMI (kg/m}^2\text{)} = \text{Weight (kg)} / [\text{Height(m)}]^2 \quad (2)$$

Statistical analysis:

The data were assessed using the SPSS statistical program, version 23.0, developed by the IBM Group. The Student's T-test was used to assess the significance of any baseline differences that existed between each group. The data was displayed as the mean $\pm$ standard deviation (SD). The correlation between the body fat percentage and oxidative stress parameters of the postmenopausal group was also calculated. In statistical analysis, all p values were two-tailed, and $p < 0.05$ was considered significant.

Results and discussion

Table 1 shows the descriptive data for the studied groups, which showed a highly significant difference ($p < 0.01$) in age, body mass index, and body fat percentage in the postmenopausal group compared to the fertile group. This is due to choosing a different age range between the studied groups.



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Table 1 - The Descriptive data of the present study.

The traits	postmenopausal group	Fertile group	p-value
Number (N)	25	25	
Age (year) Mean $\pm$ SD	48.6 $\pm$ 1.64	42.16 $\pm$ 1.56* *	p<0.01
BMI (kg/m²) Mean $\pm$ SD	32.03 $\pm$ 5.73	27.29 $\pm$ 4.61**	p<0.01
Body fat percentage (%) Mean $\pm$ SD	33.41 $\pm$ 7.02	26.24 $\pm$ 5.63**	p<0.01

BMI: body mass index, SD: Standard deviation, $p>0.05$: non-significant, $*p\leq 0.05$: significant, $**p\leq 0.01$: highly significant.

Table 2 shows the oxidative stress parameters for the studied groups, which showed a highly significant increase ($p<0.01$) in the level of malondialdehyde in the postmenopausal group compared to the fertile group (1.86 $\pm$ 0.46, and 1.10 $\pm$ 0.41, respectively).

Also, the results showed a highly significant decrease ($p<0.01$) in the levels of superoxide dismutase (46.98 $\pm$ 12.51, and 59.82 $\pm$ 6.02, respectively), and Catalase (48.55 $\pm$ 12.10, and 63.11 $\pm$ 9.21, respectively). while there was a non-significant difference ($p>0.05$) in the level of Glutathione peroxidase (63.62 $\pm$ 13.6, and 70.43 $\pm$ 11.86, respectively) in the postmenopausal group compared to the fertile group.

Table 2 - Oxidative stress parameters in studied groups.

Parameters	postmenopausal group	Fertile group	p-value
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	Mean $\pm$ SD	Mean $\pm$ SD	
MDA (nmol/dl)	1.86 $\pm$ 0.46	1.10 $\pm$ 0.41**	p<0.01
SOD (U/ml)	46.98 $\pm$ 12.51	59.82 $\pm$ 6.02**	p<0.01
CAT (U/ml)	48.55 $\pm$ 12.10	63.11 $\pm$ 9.21**	p<0.01
GPX (U/ml)	63.62 $\pm$ 13.6	70.43 $\pm$ 11.86 ⁿ	p>0.05

MDA: Malondialdehyde, SOD: Superoxide dismutase, CAT: Catalase, GPX: Glutathione peroxidase, SD: Standard deviation, **p $\leq$ 0.01: highly significant, n: non- significant.

Regarding the correlation between body fat percentage and oxidative stress parameters in the postmenopausal group, the results showed a positive significant correlation with malondialdehyde ($r = 0.5$, $p = 0.012$), as in Fig.1. There was also a negative significant correlation with Superoxide dismutase ($r = -0.444$, $p = 0.026$) and catalase ($r = -0.575$, $p = 0.002$), as shown in Figures 2 and 3, respectively. While there was no significant correlation ($r = -0.257$, $p = 0.214$) with Glutathione peroxidase, as in Fig.4.

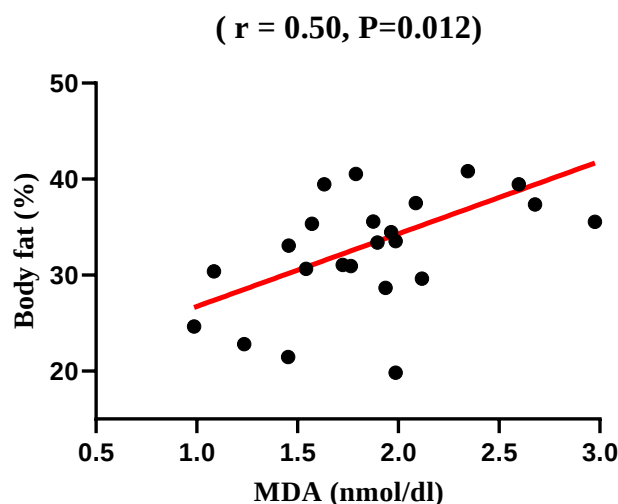


Fig.1- Correlation between body fat percentage and malondialdehyde (MDA) in postmenopausal group.



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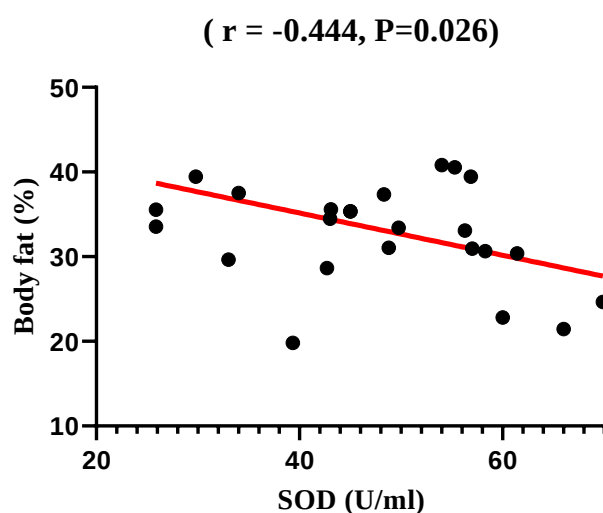
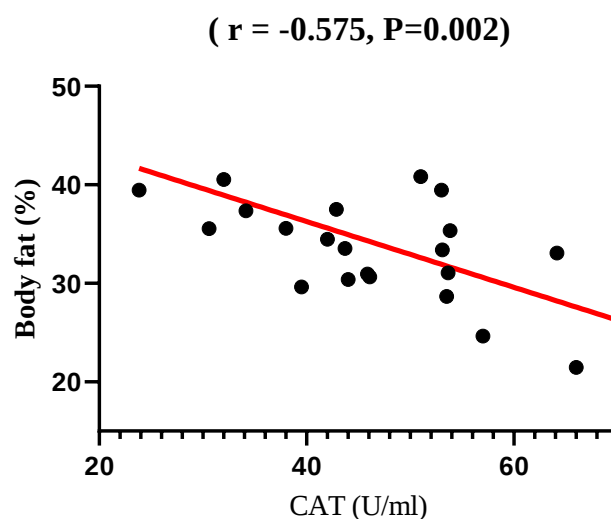


Fig.2- Correlation between body fat percentage and Superoxide dismutase (SOD) in postmenopausal group.





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Fig.3- Correlation between body fat percentage and Catalase (CAT) in postmenopausal group.

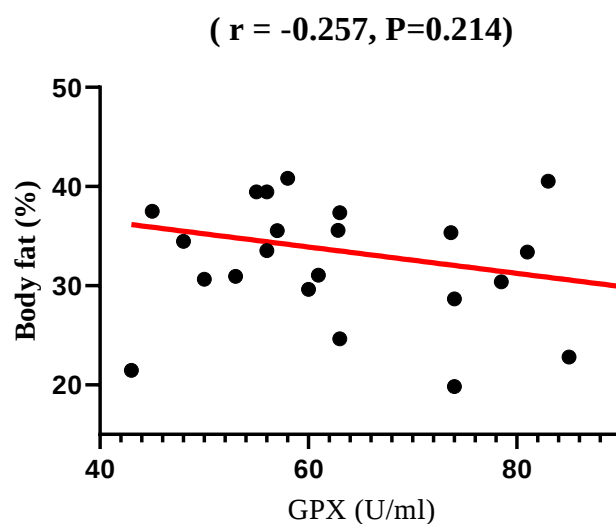


Fig.4- Correlation between body fat percentage and Glutathione peroxidase (GPX) in postmenopausal group.

The phase of a woman's life that comes after menopause, or the permanent end of her menstrual cycle, is known as postmenopausal. Menopause usually happens between the ages of 45 and 50, but Perimenopause, which precedes it, can begin several years earlier. Women who have reached postmenopausal have finished this transition and are no longer having menstrual cycles [15, 16].

A notable reduction in estrogen and progesterone levels is one of the signs and symptoms of menopause [17]. Numerous physical and psychological problems, such as mood swings, dry vagina, oxidative stress, changes in bone density, sleep disruptions, weight gain, and an elevated risk of cardiovascular disease, might result from these hormonal alterations [18, 19]. The results of this study indicated an increased incidence of oxidative stress in postmenopausal women through increased levels of MDA and decreased levels of antioxidants. This study was consistent with many previous studies [20, 21].



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One indicator of oxidative stress and lipid peroxidation in the body is malondialdehyde [22]. Reactive lipid peroxides, including malondialdehyde, are created when polyunsaturated fatty acids in cell membranes react with free radicals. This process is known as lipid peroxidation [23].

Because of its well-known antioxidant qualities, estrogen can aid in preventing oxidative stress on cells [24]. Estrogen levels significantly drop when ovarian function declines throughout menopause. Higher MDA levels are a result of increased oxidative stress, which is linked to estrogen reduction [25, 26].

High malondialdehyde levels in postmenopausal women are also attributed to several reasons, including age-related changes, increased aging, endocrine-related hormonal changes, decreased antioxidant production, increased chronic inflammation, and lifestyle changes such as changes in dietary habits, physical activity, and stress levels [27].

Bodily fat, or adipose tissue, is more than just a storehouse of stored energy; It is also capable of producing reactive oxygen species and is metabolically active. Body fat and adipose tissue mass rise together, which may lead to increased ROS generation, increased MDA levels, and decreased antioxidants [28]. Postmenopausal women suffer from an increased level of body fat resulting from increased insulin resistance. For this reason, body fat levels showed a positive correlation with MDA and a negative correlation with antioxidant levels in this study [29].

Enzymes that are essential to the body's antioxidant defense system include glutathione peroxidase (GPX), catalase (CAT), and superoxide dismutase (SOD). They are in charge of preventing oxidative damage to cells and neutralizing reactive oxygen species (ROS) [30]. Lower levels of these antioxidant enzymes in postmenopausal women than in fertile women are caused by several factors including a decline in estrogen levels that are known to have antioxidant properties and can help protect cells from oxidative stress, Chronic Inflammation where menopause is often associated with an increase in chronic inflammation, Aging that associated with a decline in the activity of antioxidant enzymes [31]. As women age and go through menopause, the combined effects of aging and hormonal changes contribute to reduced synthesis and activity of SOD, CAT, and GPX [32].

Also, changes in mitochondrial activity may be experienced by postmenopausal women, and this may lead to an increase in ROS generation. One of the main sources of ROS is mitochondria, and when these organelles malfunction, the antioxidant defense mechanism is overpowered and SOD, CAT, and GPX levels drop [33, 34].



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Additionally, women who have gone through menopause may be more vulnerable to lifestyle choices that affect levels of antioxidant enzymes. These elements include an unhealthy diet, inactivity, and elevated stress levels, all of which can lead to oxidative stress and a decline in the levels of antioxidant enzymes [35, 36].

Postmenopausal women suffer from an increased level of oxidative stress, making them more vulnerable to several diseases such as osteoporosis, cardiovascular disease, and metabolic disorders. This recommends a modified lifestyle, including a diet rich in antioxidants, regular exercise, stress management, and use more effective preventive treatment protocols by health care providers in health institutions [37, 38].

Conclusion

In conclusion, the results presented in this study indicate an increase in oxidative stress in postmenopausal Iraqi women by increased levels of MDA and decreased levels of antioxidants resulting from decreased levels of estrogenic, aging, and increased fat content in the body, which makes them more vulnerable to osteoporosis, cardiovascular disorders, metabolic disorders, and other diseases. This portends recommended modifications of lifestyle, including a diet rich in antioxidants, regular exercise, stress management, and the use of more effective preventive treatment protocols by health care providers in health institutions.

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