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OXIDANT/ANTIOXIDANT STATUS IN PRE AND POSTOPERATIVE BREAST CANCER PATIENTS

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ABSTRACT

Breast cancer, the third most common cancer worldwide, accounts for the highest morbidity and mortality. Oxidative stress has been implicated in playing a crucial role in the pathogeneses of a number of diseases, including breast cancer. Oxidative stress occurs due to an imbalance in prooxidant and antioxidant levels. The aim of this study is to investigate oxidant/antioxidant status in pre and postoperative breast cancer patients. The level of malondialdehyde (MDA), as an index of lipid peroxidation, was measured. Also, the activities of superoxide dismutase (SOD), glutathione peroxidase (GPx) and catalase (CAT) enzymes, as well as the level of reduced glutathione (GSH) were measured in groups of preoperative breast cancer patients (n=40), postoperative (n=40) and healthy female controls (n=35). The level of MDA was significantly higher in both two groups of breast cancer patients as compared with healthy controls ($p < 0.05$). Similarly, the activities of antioxidant enzymes SOD and GPx were significantly increased in both preoperative and postoperative patients groups as compared with healthy controls ($p < 0.05$). However, the level of GSH and CAT activity were found significantly decreased in both two groups of breast cancer patients as compared with their control subjects ($p < 0.05$). There was no significant difference between preoperative and postoperative patients. In Conclusions, a poor antioxidant status and high oxidative stress are associated with breast cancer risk. There is no difference in the oxidant/antioxidant status in pre and postoperative breast cancer patients. Prospective studies in a larger population should be carried out to demonstrate our present findings.

Key Words: Breast cancer; preoperative; postoperative; oxidative stress; Lipid peroxidation; antioxidants; catalase enzyme (CAT) ; malondialdehyde (MDA); reduced glutathione (GSH); superoxide dismutase enzyme (SOD); glutathione peroxidase enzyme (GPx) .

INTRODUCTION

During last two decades, a considerable attention has been focused on the involvement of free radicals in various diseases. Reactive oxygen species (ROS) such as superoxide anion radical ($O_2^{\cdot}$), hydroxyl ($OH^{\cdot}$), and hydrogen peroxide (H_2O_2) are produced in aerobic metabolism and highly reactive and toxic [**Aebi H , *et al* (1984) , Aymelek G , *et al* (2006)**]. Exposure to free radicals from a variety of sources has led organisms to develop a series of defence mechanisms including antioxidant defences [**Aebi H , *et al* (1984) **]. Antioxidant is a substance that protects the biological tissue from damage due to free radicals, and can be recycled or regenerated by biological reducers [**Aymelek G , *et al* (2006)**]. The system includes numerous enzyme and non-enzyme type of antioxidant groups that are located in the cell and in the extracellular fluid [**Aymelek G , *et al* (2006)**]. Antioxidant system is an integrated defense network with many different mechanisms for protection and repair caused by oxidative damage [**Aebi H , *et al* (1984) , Aymelek G , *et al* (2006) , Aymelek G , *et al* (2006) , Beuge JA, *et al* (1978)**]. Enzymes have the key role in this defense from oxidative stress. Enzymatic antioxidant defences include superoxide dismutase (SOD), glutathione peroxidase (GPx), catalase (CAT) and glutathione reductase (GRs). Superoxide dismutase (SOD) catalyses the dismutation of the superoxide anion into H_2O_2 [**Beuge JA, *et al* (1978) , Das UN. , *et al* (1959)**]. However, catalase and glutathione peroxidase (GPx) metabolize H_2O_2 into water and molecular oxygen, while oxidizing glutathione which is further reduced back to its reduced state by glutathione reductase in presence of NADPH [**Ellman GL., *et al* (1959) , Gago-Dominguez M , *et al* (2007) , Gonenc A , *et al* (2006)**]. However, non-enzymatic antioxidants are represented by reduced glutathione (GSH), Vitamin C, Vitamin E, carotenoids, flavonoids, and other antioxidants [**Das UN. , *et al* (1959)**]. Under normal conditions, there is a balance between both the activities and the intracellular levels of these antioxidants. This balance is essential for the survival of organisms and their health [**Aymelek G , *et al* (2006), Ellman GL., *et al* (1959)**].

Malondialdehyde (MDA), a natural product of lipid peroxidation and prostaglandin biosynthesis, is known to induce carcinogenesis [**Aebi H , *et al* (1984), Aymelek G , *et al* (2006)**]. Plasma MDA levels have been studied as an indicator of lipid peroxidation in humans and animals.

Changes in levels of GSH and the activities of antioxidant enzymes in pre and postoperative breast cancer patients remain need to know, must indeed to known. To address this issue, we measured the levels of MDA, GSH, SOD, GPx and CAT in series of breast cancer patients and examined the effect of tumor removal on their preoperative levels.

MATERIALS AND METHODS

Subjects:
The present study has been based on eighty female breast cancer patients admitted to new oncology hospital, Azerbaijan Medical University; from December 2007 to May 2009, classified into two groups and thirty five healthy volunteers. In the first group, forty preoperative breast cancer patients are ranging in age from 31 to 65 years with the mean age of 48.6±7.2 years. In the second group, forty postoperative breast cancer patients, three weeks after mastectomy are ranging in age from 38 to 69 years with the mean age of 49.9±8.1 years. The 35 healthy volunteers (age- and sex-matched), serving as the control subjects, are ranging in age from 28 to 69 years with the mean age of 48.3±9.2 years. The patients and control subjects were non-smokers and they used no hormones or oral contraceptives, the classified due to the menopausal status and clinical stage disease **Table (1).**

Sample collection and preparation: Fasting blood samples after a 12 hours fast period were collected from the patients and the controls into tubes without additive and allowed to coagulate for 30 min and serum was obtained by centrifugation at (1500 x g) for 15 min at room temperature, and stored at --20°C.

Measurements of MDA: MDA was determined by the thiobarbituric acid (TBA) method [**Gago-Dominguez M , *et al* (2007)**]. MDA is formed as an end product of lipid peroxidation which reacts with TBA reagent under acidic conditions to generate a pink-colored product. Values of MDA are assigned in (µmol/ml).

Measurement of GSH: GSH was determined by the method of Ellman [**Gonenc A , *et al* (2006)**] based on the development of a yellow color when 5,5-dithiobis (2-nitrobenzoic acid) (DTNB) is added to compounds containing sulfhydryl groups. Values of GSH are assigned in (µmol/ml).

Measurements of SOD and GPx activities: The SOD and GPx activities were determined by using a commercial Ransod kit (Randox Laboratories, UK). Values of SOD and GPx are assigned in (U/g Hb) [**Gonenc A , *et al* (2006), Jose E , *et al* (2006)**].

Measurement of CAT activity: CAT activity was assayed by monitoring the rate of H_2O_2 decomposition spectrophotometrically at 240 nm following the procedure of Aebi [**Khanzode SS , *et al* (2004)**]. Values of CAT are assigned in (U/g Hb).

Table(1) : *General characteristics of healthy controls and the breast cancer patients*

Variables	Controls	Preoperative	Postoperative
1.Total number of subjects	35	40	40
2.Age in years (mean±SD)	48.3±9.2	48.6±7.2	49.9±8.1
3.Age at menarche	12-15	12-15	12-15
4.Menopausal status			
Premenopausal	23 (65.7%)	24 (60.0%)	18 (60.0%)
Postmenopausal	12 (34.3%)	16 (40.0%)	12 (40.0%)
5.Clinical stages	healthy		
Stage I($T_1N_0M_0$)		17 (42.5%)	18 (45.0%)
Stage II($T_2N_1M_0$)		20 (50.0%)	20 (50.0%)
Stage III($T_3N_0M_0$)		3 (07.5%)	2 (05.0%)

T: Tumour size; $T_1 \leq 2$cm; $T_2 \leq 2$–4 cm; $T_3 \geq 4$cm
N: Nodal metastasis; N_0 = No regional lymph node metastasis; N_1 -Metastasis in a single ipsilateral node of ≤ 3cm diameter; N_2 -Metastasis in a single ipsilateral node of ≥ 3cm diameter
M: Distant metastasis; M_0 = No distant metastasis

Statistical Analysis: Statistical analysis was determined using STATGRAPHICS plus 5.1 statistical package software (**STATPOINT TECHNOLOGIES, INC., United States**) and Microsoft Excel. The experimental data were expressed as mean±standard deviation (SD). A *P* value of $p < 0.05$ was considered significant.

RESULTS

The results of the plasma levels of MDA as an index of lipid peroxidation of healthy control (n=35) and 80 patriots with breast cancer are collected in **table (2)** .

Table (2) : Plasma levels of MDA μ mol/ml in the examined blood samples in pre and postoperative breast cancer patients and healthy controls

Parameter	Controls (n =35)	Preoperative (n =40)	postoperative (n =40)
MDA (μmol/ml)	8.06±2.33	18.69±1.87*	16.01±1.22*†
Minimum	6.13	14.22	11.57
Maximum	9.45	22.62	20.14

Values are expressed as mean±SD,
* P <0.05; as compared with control, † P >0.05; as compared with Preoperative

The results illustrated that the levels of Plasma MDA were found significantly higher in both two investigated groups of the breast cancer patients compared with their corresponding control (p <0.05). While, There was decrease in the levels of Plasma MDA in postoperative than their preoperative level but not reached statistically significant (p >0.05).

The Plasma MDA level for preoperative group is 18.69±1.87 μ mol/ml *vs* 8.06±2.33 μ mol/ml for the control, with increasing percentage by 131.89 %; e.g. 2.13 fold (p <0.05) and for postoperative group is 16.01±1.22 μ mol/ml *vs* 8.06±2.33 μ mol/ml for the control with increasing percentage by98.34 %; e.g. 1.98 fold (p <0.05), see **fig. (1)** .

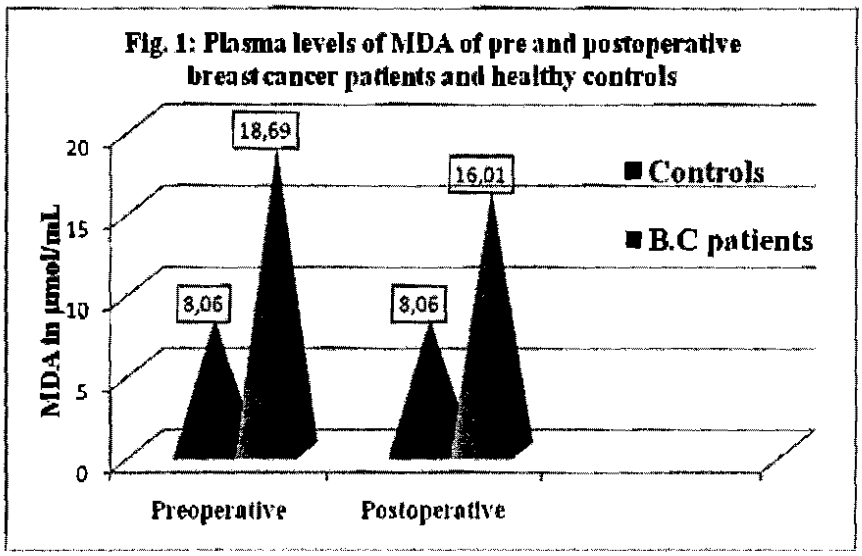


Fig. (1) : Levels of plasma MDA µ mol/ml in the blood samples for controls and breast cancer patients. Values are expressed as mean±SD.

A summary of descriptive statistics for the results of the levels of GSH as a nonenzymatic antioxidant are collected in **table (3)**.

Table (3) : Levels of GSH µ mol/ml in the examined blood samples in pre and postoperative breast cancer patients and healthy controls [a]

Parameter	Controls (n =35)	Preoperative (n =40)	postoperative (n =40)
GSH (µmol/ml)	1.89±0.12	1.29±0.19*	1.44±0.21*†
Minimum	1.53	1.11	1.23
Maximum	1.98	1.64	1.71

[a]Values are expressed as mean±SD,
* P <0.05; as compared with controls, † P >0.05; as compared with preoperative

The results illustrated that the plasma levels of reduced glutathione (GSH) were found significantly lower in both two investigated groups of the breast cancer patients as compared with their corresponding control (p <0.05). However, there was no significant difference between the plasma levels of reduced glutathione (GSH) in postoperative patients groups than their preoperative patients group (p >0.05).

The plasma levels of reduced glutathione (GSH) in breast cancer patients groups as eompared with their corresponding controls for preoperative group is 1.29±0.19 µmol/ml vs 1.89±0.12 µmol/ml with decreasing percentage by 31.75 %; e.g. -0.32 fold ($p < 0.05$) and for postoperative group is 1.44±0.21 µmol/ml vs 1.89±0.12 µmol/ml reduced with decreasing percentage by 23.81 %; e.g.-0.24 fold ($p < 0.05$). The plasma levels of reduced glutathione (GSH) in postoperative patients group as compared with the preoperative group is 1.44±0.21 µmol/ml vs 1.29±0.19 µmol/ml with increasing percentage by 11.63 %;e.g. 1.12 fold ($p > 0.05$), see **fig. (2)**.

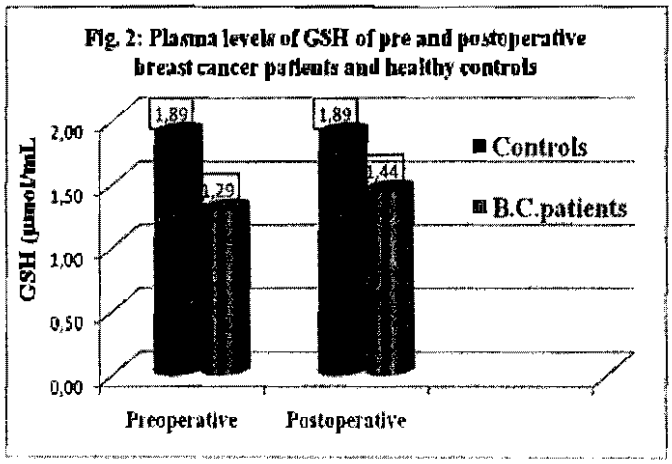


Fig. (2) : Plasma levels of GSH µmol/ml for pre and postoperative breast cancer patients and healthy controls. Values are expressed as mean±SD.

A summary of descriptive statistics for the results of the activities of SOD, GPx and CAT as an enzymatic antioxidant are collected in **table(4)**.

The results illustrated that the enzyme activities of superoxide dismutase (SOD) and glutathione peroxidase (GPx) were found significantly higher in both two investigated groups of the breast cancer patients as compared with their corresponding controls ($p < 0.05$). However, the activity of catalase (CAT) was found significantly lower in both two investigated groups of the breast cancer patients as compared with the corresponding control ($p < 0.05$).

Table (4) : SOD, GPx and CAT activities of healthy controls, pre and postoperative breast cancer patients [a]

Parameter	Controls (n =35)	Preoperative (n =40)	postoperative (n =40)
SOD (U/g Hb)	806±129	1195±166[*]	998±122[*,†]
Range	713-961	901-1316	897-1186
GPx (U/g Hb)	46.8±10.00	65.78±9.36[*]	62.69±8.63[*,†]
Range	39-56	49-71	45-71
CAT (U/g Hb)	72.98±9.26	49.16±8.41[*,†]	51.64±9.81[*,†]
Range	66-89	43-62	45-61

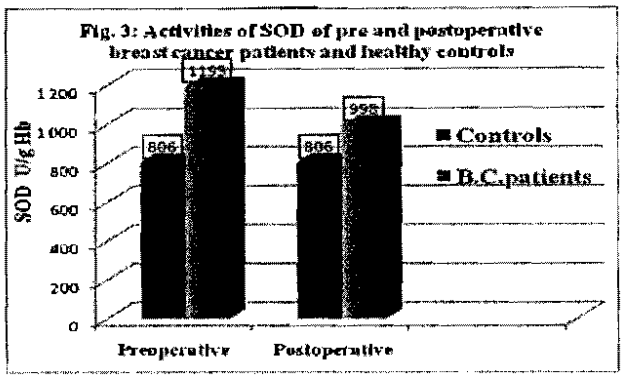
[a]Values are expressed as mean±SD,
* P <0.05; as compared with controls, † P >0.05; as compared with Preoperative

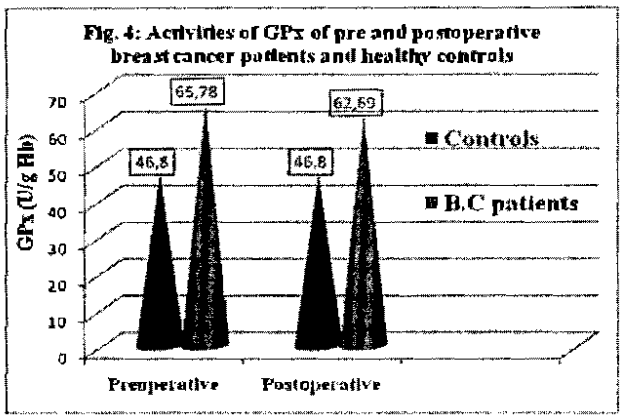
Furthermore, there were decreases in both activities of SOD and GPx postoperative groups and increases in the activities of CAT than their preoperative activities but not reached statistically significant so there were no significant difference between the activities of SOD, GPx and CAT among both investigated groups (p >0.05).

The activity of SOD for breast cancer patients as compared with the corresponding control for preoperative group is 1195±166μ/ghb vs 806±129 μ/ghb with increasing percentage by 48.26 %;e.g. 1.48 fold (p <0.05) and for postoperative group is 998±122 μ/ghb vs 806±129 μ/ghb with increasing percentage by 23.82 %; e.g. 1.24 fold (p <0.05).

The activity of GPx for preoperative group compared with the corresponding control is 65.78±9.36 μ/ghb vs 46.8±10.00 μ/ghb with increasing percentage by 40.75 %;e.g. 1.41 fold (p <0.05) and for postoperative group compared with their corresponding controls is 62.69±8.63 vs 46.8±10.00 with increasing percentage 33.95 %; 1.34 fold (p <0.05.

The activity of catalase (CAT) in breast cancer patients groups as compared with their corresponding control for preoperative group is 49.16±8.41 μ/ghb *vs* 72.98±9.26 μ/ghb with decreasing percentage by 34.64 %; e.g. -0.35 fold (p <0.05) and for postoperative group is 51.64±9.81 μ/ghb *vs* 72.98±9.26 μ/ghb with decreasing percentage by 29.24 %; e.g.-0.29 fold (p <0.05), see **fig (3-5)**.





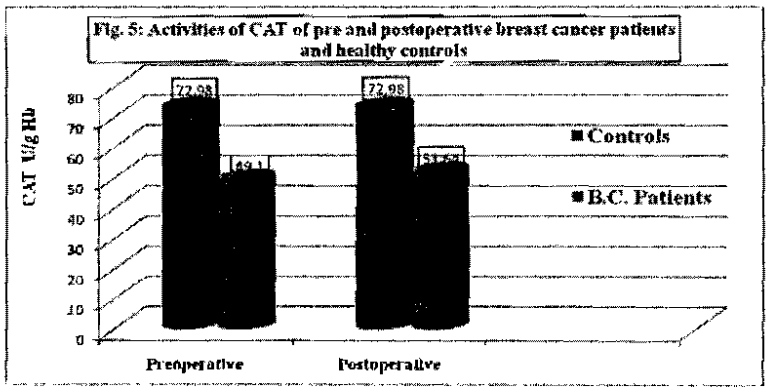


Fig. (3-5) : The activities of antioxidant enzymes µ/ghb of SOD (3), GPx(4) and CAT (5)for pre and postoperative breast cancer patients and healthy control.

DISCUSSION

Increased oxidative stress and lipid peroxidation are implicated in carcinogenic processes. The magnitude of this damage (called oxidative stress) depends not only on ROS levels but also on the body's defense mechanisms against them which are mediated by various cellular antioxidants [**Aebi H , *et al* (1984) , Aymelek G , *et al* (2006)**].

The natural antioxidant system consists of a series of antioxidant enzymes and numerous endogenous and dietary antioxidant compounds that react with and inactivate ROS. The primary antioxidant enzymes include, but are not limited to, superoxide dismutases (SOD), catalase (CAT) and glutathione peroxidase (GPX). Meanwhile, the nonenzymatic antioxidants include reduced glutathione (GSH). Cells must maintain their levels of antioxidants, often defined as their antioxidant potential, through dietary intake and/or de novo synthesis [**Aymelek G , *et al* (2006)**].

ROS have been found to modulate signaling events in the cell and play a functional role in the pathogenesis of malignancy, including breast cancer [**Beuge JA, *et al* (1978) , Das UN. , *et al* (1959) , Ellman GL., *et al* (1959)**]. Damage to the mammary epithelium by ROS can lead to fibroblast proliferation, epithelial hyperplasia, cellular atypia, and breast cancer [**Kumaraguruparan R , *et al* (2002)**]. In particular, peroxidation of lipids induced by ROS generates products such as MDA that can react with DNA and leads to mutations in proto-oncogenes and tumor-suppressor genes and eventually transformation of normal epithelium to a malignant phenotype [**Beuge JA, *et al* (1978) , Ellman GL., *et al* (1959) , Nazarewicz RR , *et al* (2007)**].

Many studies have examined the possibility of a connection between lipid peroxidation and cancer [**Obrador E, *et al* (1997) , Paglia D , *et al* (1967)**]. Higher plasma MDA levels have been reported in cancer patients than those in the controls [**Paglia D , *et al* (1967) , Portakal O, *et al* (2000)**]. However, lower plasma MDA has also been reported in the breast cancer group compared with controls [**Rajneesh C P, *et al* (2008)**]. In the present study, our results are in agreement with most of the earlier studies which suggested that there was a possibility of the accumulation of ROS which might result in significantly higher lipid peroxidation cellular and molecular levels [**Paglia D , *et al* (1967) , Portakal O, *et al* (2000) , Rajneesh C P, *et al* (2008)**]. In addition, the increase in lipid peroxidation observed in both pre- and postoperative groups was accompanied by enhanced antioxidant status.

Antioxidant enzymes such as SOD, CAT and GPx are the first line of defense against ROS and to the oxidant assault on cells. The increased activity of these enzymes is consistent with reports of antioxidant enzyme overexpression in tumors [**Nazarewicz RR , *et al* (2007), Ripple MO, *et al* (1997) , Sun Y , *et al* (1998)**]. Although GPx shares the substrate H_2O_2 with CAT and both are able to destroy H_2O_2; GPx alone can react effectively with lipid and other organic hydroperoxides [**Aymelek G , *et al* (2006) , Paglia D , *et al* (1967)**], and has a much higher affinity for H_2O_2 than does CAT

[**Tabassum H , *et al* (2001)**] suggesting that H_2O_2 is mainly degraded by GPx under normal conditions.

The significant increase in SOD activity indicates the formation of more superoxide radicals and their removal as SOD metabolizes superoxide radicals [**Tas F, *et al* (2005)**]. However, the significant decrease in CAT activity indicates the decomposition of H_2O_2 (forming H_2O and O_2) [**Tiwari AK., *et al* (2004)**]. Antioxidant depletion in circulation may be due to increased scavenging of lipid peroxides as well as sequestration by tumor cells. Reduced glutathione is a physiologically important nonproteinthiol with multiple functions ranging from antioxidant defense to modulation of cell proliferation. In particular, intracellular GSH maintains the reduced status of thioredoxin that activates ribonucleotide reductase, a key enzyme essential for DNA synthesis. A positive correlation has been observed between enhanced synthesis of GSH and high rates of cell proliferation in tumors [**Kumaraguruparan R , *et al* (2002) , Tas F, *et al* (2005)**]. GSH in conjunction with GPx protects cells against a wide variety of xenobiotics, including ROS and other toxic compounds [**Portakal O, *et al* (2000), Tiwari AK., *et al* (2004)**]. In addition, the observed decrease in the non-enzymatic antioxidant GSH was in line with several published reports [1, 3] and diseases including maliznannanus[**Li JJ , *et al* (1998) , Vaca CE , *et al* (1988)**].

Our data showed that there were significant increases in SOD and GPx activities and significant decreases in GSH levels and CAT activity in both pre- and postoperative breast cancer patients than those of the controls, which might be due to the free radical scavenging activity. In addition, these significant increases in the circulating enzymatic antioxidants SOD and GPx were in line with a number of published reports [**Aymelek G , *et al* (2006) , Beuge JA, *et al* (1978) , Tiwari AK., *et al* (2004) , Vaca CE , *et al* (1988) Yeh CC , *et al* (2005)**]. However, contrary to our findings, decreased levels of SOD and GPx have also been reported [**Zieba M , *et al* (2001)**] . Our data also showed that, there was no difference in the oxidant/antioxidant status between pre- and postoperative breast cancer patients.

CONCLUSIONS:

Breast cancer complication is related to increase of lipid peroxidation in serum with concomitant decrease of antioxidant defense capacity. In addition, there is no difference in oxidant/antioxidant status between pre- and postoperative patients. Prospective studies in a larger population should be carried out to demonstrate our present findings.

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الملخص العربي

حالات الأكسدة / مضادات الأكسدة قبل وبعد الجراحة لمرضى سرطان الثدي

د./ محمد الحفنى

قسم السرطان والبيولوجيا الجزيئية المعهد القومي للسرطان – جامعة القاهرة

يعتبر سرطان الثدى من أشهر ثلاثة أنواع من السرطان فى العالم والذى تحدث منه وفيات بنسبة عالية . يلعب الضغط التأكسدى دور رئيسي فى هؤلاء المرضى متضمنة سرطان الثدى .

يحدث الشد التأكسدى نتيجة عدم إتزان بين مستويات الأكسدة ومضادات الأكسدة . تهدف هذه الدراسة إلى بيان حالات الأكسدة ومضادات الأكسدة قبل وبعد إجراء جراحة لمرضى سرطان الثدى .

تم قياس مستوى نواتج أكسدة الدهون (MDA) كمؤشر لأكسدة الدهون وكذلك نشاط إنزيم (SOD) و (GPx) و (CAT) إضافة إلى مستوى (GSH) فى مجموعات قبل الجراحة وعددهم ٤٠ وبعد الجراحة وعددهم ٤٠ أيضاً إضافة إلي ٣٥ من الإناث الأصحاء .

إرتفع مستوى (MDA) إرتفاعاً ملموساً فى كلا المجموعتين مقارنة بالأصحاء ، وكذلك فإن نشاط إنزيمى – SOD – GPx زاد فى كلا المجموعتين من المرضى مقارنة بالمجموعة الظابطة . أما معدل GSH ونشاط إنزيم CAT فقد إنخفض فى كلا المجموعتين بالمقارنة بالمجموعة الظابطة ولم يكن هناك اختلافات بين قبل أو بعد الجراحة .

يستنتج من ذلك أن إحتمال الإصابة بسرطان الثدى يصاحب بقدر ضئيل من الخلل فى مضادات الأكسدة وزيادة الضغط التأكسدى . ولا يوجد إختلاف بين حالات الأكسدة ومضادات الأكسدة قبل وبعد الجراحة وننصح بدراسة عدد أكبر من الحالات لتأكيد هذه النتيجة .