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Role of Balance between Some Free Radicals and Co-enzyme Q10 in Progression of Breast Cancer in Iraqi Women

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Abstract

Background: According to reports from World Health Organization, approximately 2.3 million new cases of breast cancer were diagnosed globally. Reactive oxygen species are closely associated with chronic inflammation, as evidenced by altered lipid peroxide concentrations in breast cancer.

Objective: to evaluate the role of oxidative stress as well as Co-enzyme Q10 in aggressiveness of breast cancer in Iraqi women.

Patients and Methods: This cross-sectional study was conducted from the first of December 2025 to the end of March 2026.at the Kirkuk Oncology Center. involved 90 patients with positive breast cancer and 45 healthy control subjects. We were evaluated the level of the following parameters SOD, GPX, CoQ10, MDA, Vit. D and Vit. C in patents and control groups.

Results: demonstrate a significant reduction in CoQ10 levels in patients than that of healthy control group (0.61 ±0.18, 0.74 ±0.18) respectively, reduction in CoQ10 levels across patient groups from stage I to stage IV. it was confirmed there was a strong negative correlation between the levels of CoQ10 and MDA in patient group.

Conclusion: lower plasma CoQ10 was associated with higher breast cancer risk. Also, this study revealed that a strong negative correlation was found between CoQ10 and MDA suggesting that CoQ10 supplementation could be reduces lipid peroxidation.

دور التوازن بين مؤشرات الإجهاد التأكسدي والإنزيم المساعد في تطور سرطان الثدي لدى النساء العراقيات

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الخلاصة:
عُد سرطان الثدي من أكثر أنواع السرطان شيوعًا على مستوى العالم، حيث يُسجل ما يقارب ٢,٣ مليون حالة جديدة عالميًا. وترتبط أنواع الأكسجين التفاعلية ارتباطًا وثيقًا بحدوث الالتهاب المزمن، ويتجلى ذلك من خلال التغيرات في تراكيز بيروكسيدات الدهون، والتي تُعد من المؤشرات الحيوية للإجهاد التأكسدي لدى مريضات سرطان الثدي. هدفت هذه الدراسة إلى تقييم دور الإجهاد التأكسدي في شدة وتطور سرطان الثدي لدى النساء العراقيات. أُجريت هذه الدراسة المقطعية في مركز كركوك للأورام خلال المدة من الأول من كانون الأول/ديسمبر ٢٠٢٥ إلى نهاية آذار/مارس ٢٠٢٦. وشملت الدراسة ٩٠ مريضة مشخصة بسرطان الثدي و٤٥ امرأة سليمة ظاهرياً كمجموعة سيطرة .أظهرت النتائج انخفاضاً معنوياً في مستويات الإنزيم المساعد كيو لدى مريضات سرطان الثدي مقارنةً بمجموعة السيطرة. كما لوحظ انخفاض تدريجي في مستويات الانزيم المساعد كيو ١٠ عبر المراحل السريرية للمرض من المرحلة الأولى إلى المرحلة الرابعة. كذلك تبين وجود ارتباط عكسي معنوي بين مستويات الانزيم المساعد كيو ١٠ومستويات المالونداياالديهايد لدى مجموعة المرضى .ارتبط انخفاض مستويات الانزيم المساعد كيو ١٠ في البلازما بزيادة خطر الإصابة بسرطان الثدي. كما أظهرت الدراسة وجود ارتباط عكسي قوي بين الانزيم المساعد كيو ١٠ والمالون داي الديهايد ، مما يشير إلى احتمال مساهمة الانزيم المساعد كيو ١٠ في الحد من بيروكسدة الدهون والإجهاد التأكسدي.

الكلمات المفتاحية: الإنزيم المساعدQ10 ، المالونداياالديهايد ، سرطان الثدي، الإجهاد التأكسدي

INTRODUCTION

According to World Health Organization report, approximately 2.3 million new cases of breast cancer were diagnosed globally in 2022 representing roughly 1 in 4 of all new cancer cases in women, as well as breast cancer is the leading cause of cancer-associated deaths in women [(1)]. Despite rising incidence, many cases in Iraq are diagnosed at advanced stages. A study from Baghdad revealed that nearly 47% of women presented with stage III or IV disease, indicating late detection and significant barriers to timely screening. The majority of patients are diagnosed in their 40s, which is relatively younger than the median age of diagnosis in Western countries. These findings suggest that lifestyle changes, population aging, and improved diagnostic capacity may all contribute to the observed increase in incidence, while late diagnosis continues to worsen outcomes [(2)].

The normal cells have ability to divide many times as needed and stop when they attach to other cells and stay in place in tissues. Cancer Cells lost their ability to stop dividing when they attach to other cells so they still dividing forming new tissues [(3)]. The pathophysiology of breast cancer tumorigenesis is influenced by a variety of factors, Genetic predisposition is the first and the most noticeable part, some individuals inherit defects in the DNA and genes like the BRCA1, BRCA2 and P53 among other cytochrome P450 genes, glutathione S-transferase family, tumor stemness, intra-tumoral microbiota, DNA repair genes and genes encoding cell signaling molecules and estrogen receptor (ER) [(4)]. Inactivation of the second allele of tumor suppressor genes would be an early event in this oncogenic pathway.

Oxidative stress plays a dual role in breast cancer: sustained, moderately elevated reactive oxygen species (ROS) promote carcinogenesis and tumor progression, while very high ROS levels can trigger cancer cell death and are exploited by some therapies. Breast cancer cells and their microenvironment typically show increased oxidative damage to DNA, lipids, and proteins together with adaptive upregulation of antioxidant defenses, creating a fragile redox balance that can be both a driver of disease and a therapeutic vulnerability [(5)].

Clinical and biochemical studies consistently show higher levels of oxidative damage markers such as malondialdehyde (MDA), 8-oxo-2′-deoxyguanosine (8-oxodG), and protein oxidation products, together with reduced systemic antioxidant capacity, in women with breast cancer compared with healthy controls [(6)]. Serum measurements often reveal increased total oxidant status or oxidative stress index and

decreased total antioxidant status, glutathione (GSH), and related ratios (for example GSH/GSSG), and these changes may correlate with tumour stage, adiposity, or risk, although they are not specific enough for standalone diagnosis [7]. Oxidative stress is another top player that is involved in metabolic dysregulation, as ROS are contributed mainly by increased pro-oxidant activities including oxidative phosphorylation, O2 • generation, and protein kinase C activation. Emerging evidence points to the adipotropic effects of heavy metals and metalloids and their potential role in pathophysiological changes in obesity, diabetes, or metabolic syndrome [8].

Energy metabolic balance is often necessary to maintain the sustained proliferation of cancer cells. Cancer cells usually increase their demand to energy and metabolism by increase transporter and enzyme blood levels, mediated by oncogene activation and divert metabolites towards biosynthetic pathways for nucleoside and amino acid production to sustain cellular proliferation [9]. Oxidative stress can cause oxidative damage to DNA, including mutation, deletions, chromosomal deformities, mutated tumor suppressor genes or proto-oncogenes, and also contribute to the induction of the Akt/PI3K/mTOR signaling pathway, which is a characteristic of cancer development and progression [10]. Disturbances in the redox balance affect the development of many malignant tumors, including breast cancer, it is a multistep process, beginning with hyperplasia and progressing through atypical hyperplasia to in situ and invasive cancer [11].

There are many factors attribute by way or another to increase incidence and development and progression of cancer and play a vital role in the pathophysiology and microenvironment of cancer lesion. Genetic, environmental, age, stress and behavioral factors all play a role in the development of breast cancer.

Antioxidants are substances that may protect cells from the damage caused by unstable molecules known as oxygen reactive species which may cause different type of disease include breast cancer. Antioxidants interact with these molecules free radicals and may stop and prevent their damage effect [12]. Antioxidants work to slow tumor development in certain types of cancer, but in others they may accelerate tumor growth, spread, and blood vessel formation. Additionally, some antioxidants also affect the immune system in ways that make treatments more effective [13]. Co-enzyme Q10 is a lipid soluble isoprenoid quinone consists of a 1,4-benzoquinone head group attached to a polyisoprenoid tail of ten isoprene units in humans, which anchors it in membranes and makes it highly hydrophobic.

Another important role of CoQ10 is as an antioxidant. CoQ10 is the sole lipid-soluble antioxidant that is endogenously synthesized. Reduced CoQ10 inhibits both the initiation and the propagation of lipid peroxidation [14]. Moreover, reduced CoQ10 also regenerates the other antioxidants—α-tocopherol and ascorbate into an active reduced form. CoQ10 is also involved in the β-oxidation of fatty acids, de novo pyrimidine biosynthesis, sulfide oxidation [15] an essential cofactor for uncoupling proteins (UCPs), and modulation of the mitochondrial permeability transition pore. Thus, the importance of the distinct state of CoQ10 is gaining growing attention [16].

The role of coenzyme Q10 in the pathogenesis of breast cancer involves multiple factors: clearance of reactive oxygen species, membrane mechanics and extracellular matrix signaling, mitochondrial bioenergetics ‹regulation of iron-induced cell death, inflammation, and its impact effect related to estrogen signaling mechanism. Besides its direct antioxidant effect, CoQ10 is related to the restoration of other antioxidants like vitamin C and vitamin E [17].

Breast tumors typically exhibit high levels of oxidative damage along with an adaptive upregulation of antioxidant systems, creating a critical state of high but viable reactive oxygen species (ROS) that supports proliferation, mutation, and treatment resistance [18]. Coenzyme Q10 (COQ10) contributes to this balance by neutralizing lipid peroxidation radicals and modulating mitochondrial ROS production; decreased COQ10 levels in tissues or plasma have been associated with elevated lipid peroxidation [19]. This work seeks to evaluate the role of oxidative stress as well as Co-enzyme Q10 in aggressiveness of breast cancer in Iraqi women.

PATIENTS AND METHODS

Study Design and Participants

This cross-sectional study involving diagnostic evaluation of serum biomarkers in breast cancer patients compared to healthy controls. The study was conducted from the first of December 2025,

to the end of March 2026 at the Kirkuk Oncology Center. The study protocol was approved by the scientific committee of Tikrit University College of Medicine, and all participants provided written informed consent.

This study involved 90 patients with positive breast cancer (BC) diagnosed by the oncology center according to the who criteria [20]. Forty-five apparently healthy women in age with between (30-65) years old, were taken as a control group.

Inclusion criteria: Breast cancer patients diagnosed at varying stages (Stage I, II, III, IV).

Exclusion criteria: metastatic disease, concurrent malignancy, renal or hepatic dysfunction and coronary diseases

Five ml of Fasting venous blood samples were taken during the patients' routine follow-up visits to the oncology center. The following parameters were evaluated SOD, GPX, CoQ10, MDA, Vit. D and Vit. C in patents and control groups.

A questionnaire has been designed by the investigator including age, menopausal status, family history, height and weight. Body mass index (BMI) has been obtained from all subjects involved in this study. The histopathological diagnosis of breast cancer, grade, stage of the tumour, and hormone receptor status was recorded from the pathology reports of breast cancer patients.

Statistical analysis

Data were analyzed using SPSS version 26. Quantitative variables were expressed as mean ± standard deviation, whereas qualitative variables were presented as number and percentage. Data distribution was assessed before statistical analysis. Comparisons between breast cancer patients and healthy controls were performed using the independent samples t-test. Differences among breast cancer stages were evaluated using one-way analysis of variance (ANOVA). The association between study variables was assessed using Pearson's correlation coefficient. Categorical variables were compared using the Chi-square test. A p-value < 0.05 was considered statistically significant.

RESULTS

The mean age of the breast cancer group was 47.5 ± 9.09 years compared with 45.2 ± 6.69 years in the control group, with no statistically significant difference between the two groups (P= 0.77) This study showed that the peak age of women with breast cancer was between 40 – 60 years old and their percentage was 43.3% while the lowest group was between 30-40 years old with 8.9% as percentage see table (1).

The results demonstrate that patients in the stage IV cohorts are markedly older than those in the other groups. Despite the ANOVA not demonstrating overall significance, these particular comparisons suggest a possible age-related trend in illness progression, indicating that older age may correlate with more advanced stages as show in the table (2).

The table 3 includes a description comparing the characteristics and risk factors of the breast cancer between the patient and control group indicating (24%) of patients were employed against 58% in control group, also in patients (31%) were unmarried related to (33%) in healthy group as well as, seventy four percent of the patients and 64% of the controls belonged to upper lower socio-economic status. Majority of the patients (79%) belonged to urban area of residence while (78%) for control.

The table (4) shows the comparison between patients and control group for the parameters under investigation, demonstrate there is a significant difference in many of these parameters in patients than that of healthy control group.

Coenzyme Q10 is a compound made in the human body that has been linked with numerous benefits. Figure (1) show analysing the CoQ10 level and their statistically significant across the study groups ranging from healthy controls to patients at various clinical stages. These data show that CoQ10 levels are significantly lower in breast cancer patients than in healthy controls (0.613± 0.181 and 0.743 ± 0.180) respectively and tend to reduce with advancing clinical stage, with a bottom at stage II (table 4) The one-way ANOVA gave no significant difference in CoQ10 level between group stage in breast cancer patients p-value (< 0.069), indicating that the CoQ10 level was not association with breast cancer progressing.

Regarding to the levels of MDA as shown in table (4) and figure (3) the present study demonstrates there was a significant higher level of MDA in patients than that of control group.

There is a negative correlation between MDA and Co-Q10 level in patients group. r = 0.367 and P = 0.004

Table (1): the distribution of the breast cancer with age group.

		Frequency	Percent
Age groups	30 -40	12	13.3%
	40- 50	25	27.8%
	50 - 60	39	43.3%
	60 - 70	14	15.6%
	Total	90	100.0%

Table (2): show the age differences among patients in relation to cancer stages.

Age	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	90.176	3	30.059	.374	.772
Within Groups	6914.724	86	80.404		
Total	7004.900	89			

Table (3): Socio-Demographic Risk Factors among Women in patients and control and Case Groups.

Variables	Group	Patients		Control		P value
		No.	%	No.	%	
Age (years)	< 40 years	55	61%	31	68%	0.376
	≥ 40 years	35	39%	14	32%	
Work	Employed	22	24%	26	58%	<0.001
	House wife	68	76%	19	42%	
Residence	Rural Area	19	21%	10	22%	0.882
	Urban Area	71	79%	35	78%	
Social status	Married	62	69%	30	67%	0.794
	unmarried	28	31%	15	33%	
Economic status	High	67	74%	29	64%	0.227
	Low	23	36%	16	36%	

Table (4): The levels of parameters in patients and control groups.

	group	N	Mean	Std. Deviation	P value
Age (years)	Patients	90	47.53	9.09	0.114
	control	45	45.26	6.99	
MDA (ng/ml)	patients	90	26.27	6.34	<0.001
	control	45	18.20	5.72	
CoQ10 (µmol/L)	patients	90	.61	.18	<0.001
	control	45	.74	.18	
SOD ng/ml	patients	90	151.8	18.82	<0.01
	control	45	160.04	20.84	
GPx U/ml	patients	90	.80	.27	<0.001
	control	45	1.06	.25	

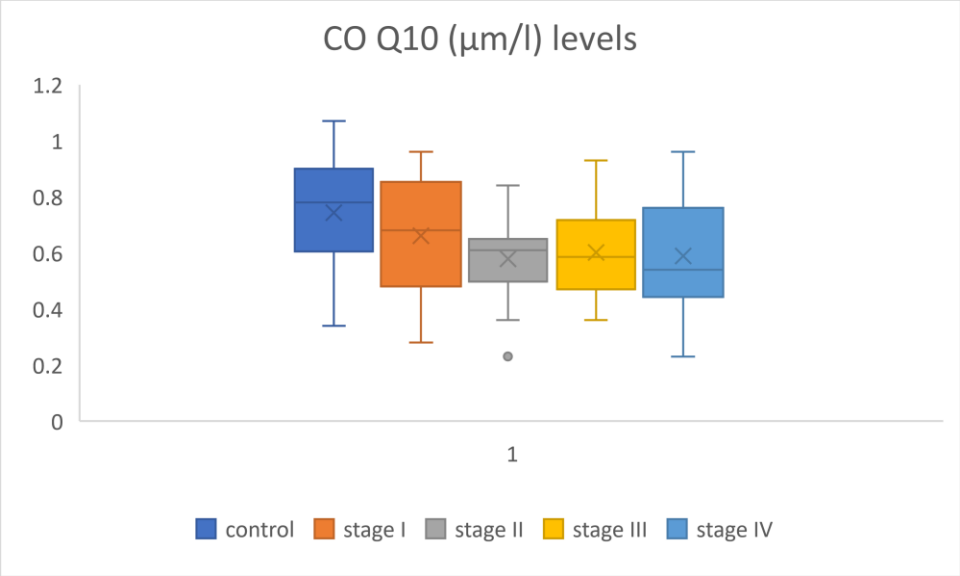

Figure (1). The level of CO-Q10 in different patient group stages comparing with control group

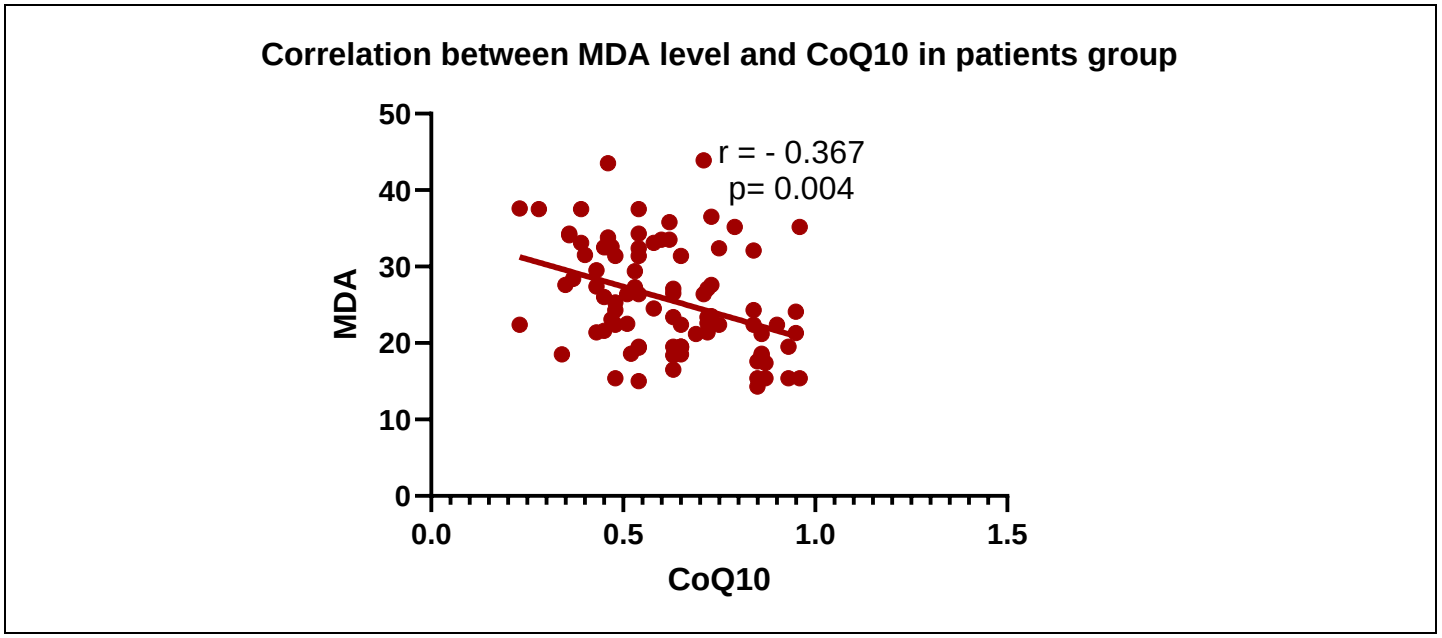

Figure (2): show the correlation between MDA and Co-Q10.

Table (5): Biochemical parameter levels across different breast cancer stages.

		Sum of Squares	df	F	P value
MDA	Between Groups	870.	3	6.827	.000
	Within Groups	3657	86		
	Total	4528	89		
Co-enzyme Q10	Between Groups	.106	3	1.065	.368
	Within Groups	2.842	86		
	Total	2.947	89		
SOD	Between Groups	1424	3	1.356	.262
	Within Groups	30131	86		
	Total	31556	89		
GPX	Between Groups	.899	3	4.426	.006
	Within Groups	5.823	86		
	Total	6.722	89		

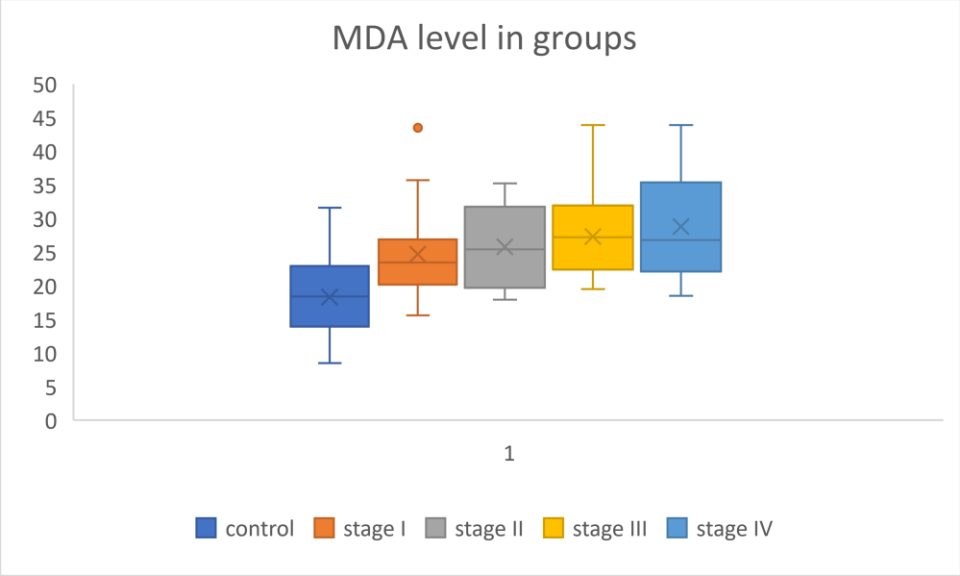

Figure (3): The level of MDA (ng/ml) in different patient group stages comparing with control group

Table 6: the correlations between parameters under investigation.

AGE	1						
BMI	-0.056	1					
Vit. D	0.039	0.247*	1				
MDA	- 0.027	-0.103	-0.45**	1			
Co. Q10	-0.041	0.021	0.27**	-0.36**	1		
SOD	0.046	0.122	0.054	-0.38**	0.011	1	
GPx	0.006	-0.033	0.150	-0.071	0.027	0.085	1
	AGE	**BMI**	**Vit. D**	**MDA**	**Co. Q10**	**SOD**	**GPx**

*Correlation is significant at the 0.05 level.
** Correlation is significant at the 0.01 level.

DISCUSSION

This study showed that the peak age of women with breast cancer was between 40 – 60 years. Age is strongly related to breast cancer occurrence: the risk rises with increasing age, and most cases are diagnosed after age 50, with the highest incidence typically seen in older women. The result come in agreement with others who reported that a non-linear relationship between age and BCSS, with the highest risk of breast cancer mortality observed in the youngest patients (less than 40 years), followed by an increase after 50-60 years old hazard at a rate of 5% per year with increasing age [21-22]. Lifetime oestrogen exposure matters most: early menarche, late menopause, or HRT elevate risk. Post-menopause, adipose tissue aromatizes androgens to oestrogens, Progesterone also promotes proliferation via PR signalling in hormone-receptor-positive subtypes [23]. lifestyle factors associated with aging may also have a contribution; dietary choices or physical activity and environmental exposures such as radiation exposure and certain chemicals are also contributors to the associations of breast cancer and age [24].

Breast cancer develops due to a combination of genetic, hormonal, lifestyle, and environmental factors. All these factors contribute in one way or another to the occurrence and development of breast cancer. Although this study is not concerned with studying these factors, we found it important to mention them to determine their role in influencing the study results.

Regarding the serum level of Coenzyme Q10 this study revealed that CoQ10 levels are significantly lower in breast cancer patients than in healthy controls and tend to reduce with advancing clinical stage, with a bottom at stage II, indicating that the CoQ10 level was not association with breast cancer progressing. Cooney et al. found an inverse association between plasma CoQ10 and subsequent breast cancer risk: the lower the CoQ10, the higher the risk [25]. In the same manner case–control study in Chinese women found that lower plasma CoQ10 was associated with higher breast cancer risk, with about 90% greater risk in the lowest quintile vs. mid-range after excluding early cases, suggesting that suboptimal CoQ10 may predispose to breast cancer [26].

Co enzyme Q10 can influence breast cancer risk mainly through its roles in mitochondrial bioenergetics, redox balance, membrane integrity, and inflammation, but the relationship is complex. It is considered to be a key electron carrier in the mitochondrial ETC (complexes I/II → III), maintaining ATP production and limiting electron leak that generates ROS. Low CoQ10 impairs oxidative phosphorylation and increases ROS, promoting DNA damage, lipid peroxidation, and oncogenic mutations [27]. As a lipid-soluble antioxidant, CoQ10 prevents lipid peroxidation in membranes and can regenerate other antioxidants (vitamin E, vitamin C), which are themselves linked to breast cancer risk modulation [17], this explains the strong negative correlation between Coq10 and MDA that revealed in this study figure (2). As well as other study was showed that CoQ10 supplementation reduces MDA (lipid peroxidation) and inflammatory cytokines (TNF-α, IL-6), parameters known to promote carcinogenesis and hormone driven tumour progression [19], suggesting an important role of CoQ10 therapy in improving treatment response.

Regarding to the levels of MDA the present study demonstrated that there was a significant higher level of MDA in patients than that of control group. The descriptive statistics of MDA serum levels across different groups ranging from healthy controls to patients at various pathological stages demonstrated a considerable and statistically significant rise in patients compared to controls (Table 4). There are Several studies show significantly elevated serum or plasma MDA in breast cancer [28,29]. Excessive oxidative stress occurs when free radical production exceeds the capacity of antioxidant defences (enzymatic and non-enzymatic) to neutralize them, leading to cellular damage. Overproduction of ROS usually comes from mitochondrial ETC leakage, chronic inflammation, radiation, xenobiotics, and carcinogens or due to impaired antioxidant defences, when this imbalance persists, it drives lipid peroxidation (↑MDA), DNA damage, and protein oxidation which consider to be a key mechanism in carcinogenesis, including breast cancer [30]. Tumors generate elevated ROS and upregulated oxidative metabolism that increases oxidative damage; this is a recognized feature of many cancers including breast cancer. Tissue measurements show decreased CoQ10 (mitochondrial Q) in breast-tumor tissue versus adjacent normal tissue, which investigators attribute to consumption of CoQ10 during antioxidant defence. Antioxidant enzyme activities (SOD, GPx) are often altered in tumors and may also be reduced during antioxidant defence as indicating by this study.

CONCLUSIONS

The stage specific comparisons suggest a possible age related pattern in disease severity; whereby older age may be associated with more advanced stages. lower plasma CoQ10 was associated with higher breast cancer risk. Also, this study revealed that a strong negative correlation was found between Coq10 and MDA suggesting that CoQ10 supplementation could be reduces lipid peroxidation.

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