Evaluation Some Biochemical Test and Antioxidant in Iraqi Women Patients with Cervical Cancer

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Abstract

Background.Reactive oxygen species causes DNA damage and cellular membrane deterioration; Triglycerides have been linked to an increased risk of Cervical cancer (CC). Material and methods: One hundred samples (serum) were taken from patients aged between 20−60 years; Whole blood was allowed to coagulate for one hour at room temperature in order to acquire the serum sample. As well as divided into six groups to achieve the tests which TAC, TC, TG, HDL, LDL, GPX Results: Some important findings were observed regarding the clinical significance from lipid profiles in patients with CC. First, a detailed analysis of the blood lipid differences between patients and healthy women using a relatively large number of CC patients showed that those with the disease had lower levels of HDL cholesterol and higher levels of TC, TG, and LDL cholesterol.Conclusion: In conclusion, a disordered lipid profile with lower HDL levels and greater TG, TC, and LDL levels may be linked to cervical cancer. CC exhibits oxidative stress, which is reflected by altered antioxidative enzyme activity, increased lipid peroxidation, this stress is accentuated in later stages by the increasing tumor burden.

Keywords: ROS, lipid profile, cervical cancer

1. Introduction

The cells of the cervix, the part of the uterus at the base that connects to the vagina, are where cervical cancer begins. Cervical cancer is caused by DNA changes (point mutation) that occur in normal cervix-based cells. DNA carries the instructions that tell a cell how to act (1). Reactive oxygen species (ROS), which are produced by a number of different processes, trigger the lipid peroxidation pathway, increase the creation of MDA, change cellular activity, and encourage the growth of cancer (2). the increased oxidative damage resulting from uterine cancer patients' increased ROS production. As the situation becomes worse, more oxygen radicals are produced, which leads to oxidative damage. DNA damage and cellular membrane deterioration. Serum glutathione peroxidase concentrations rise as a result of excessive lipid peroxidation brought on by the increased production of free radicals. Free radicals have the potential to significantly alter the structure of DNA and the functionality of cell membranes, that can also result in mutations. To calculate how many antioxidants are present in a biological sample, use the total antioxidant capacity (TAC) formula (4). It is a reliable biomarker that may be used to diagnose, predict, and prevent a variety of diseases. Among the finest detoxifiers is glutathione (GSH). It is well established that a cell's GSH concentration affects both how poisonous the cell is and how sensitive the cell is to anticancer therapies (a decrease in GSH content increases toxic side effects) (5). It is critical to assess the GSH level to establish if malignant cells will be vulnerable to a drug's effects or if normal cells will be unaffected (6). It is known that patients with cervical cancer who experience a complete response to treatment as contrast to a partial response have considerably lower levels of GSH in their blood and tumors. The start of cancer and lipid abnormalities are linked, based on a large body of studies. Lipids play a critical role in cell proliferation and cancer formation by influencing cellular signaling, cell membranes, and cell-cell interconnections. Preliminary studies suggest that adipocytes may promote the fast onset and spread of cancer (3). Serum triglycerides (TG) have been discovered to have a significant correlation with the danger from endometrial cancer (7). With normal levels from total cholesterol (TC) and low-density lipoprotein (LDL), the risk of colorectal cancer increased (LDL). Hypertriglyceridemia increased the risk from prostate cancer, and that these tumors tended to be more malignant. Ailment. According to a research, triglycerides do in fact increase the chance of developing cervical cancer (8).

2. 2.Material and methods

doi.org/10.31838/hiv23.02.5 Received: 18.10.22, Revised: 11.11.22, Accepted: 04.12.22

2.1 Serum collection

One hundred serum samples from individuals (patients =50, and control=50) between the ages of 20 and 60 were obtained were obtained collected from the Iraqi National Centre for Cancer Research . To obtain a specimen of serum, whole blood was allowed to coagulate at room temperature for 60 minutes. For eliminate the clot, the blood was centrifuged for five minutes at 2,000 rpm. A Pasteur pipette was used to deposit the liquid (serum) into a clean polypropylene tube samples were centrifuged. During treatment, the serum samples were kept at (2–8 °C).

The Glutathione Peroxidase 1 was measured from serum samples by ELISA using Human GPX1 (Elabscience, chain, Cat No. E-EL-H5410)

The TAC ELISA kit uses a monoclonal anti-TAC antibody, a TAC-HRP conjugate, and a selective enzyme immunoassay method. The assay sample, buffer, and TAC-HRP conjugate are incubated for an hour in a pre-coated plate. That after incubation period, the wells are cleaned five times and poured. Subsequently, the wells are used to incubate the HRP enzyme substrate. The combination formed as a result of the enzyme-substrate reaction has a blue color. By adding a stop solution, which turns the solution yellow, the activity is eventually terminated. At 450 nm, the color intensity is spectrophotometrically quantified in a microplate reader. The intensity of the color is inversely linked to the amount of TAC because TAC-HRP conjugate and TAC from specimens compete for the anti-TAC antibody binding site. As even more spots are occupied by TAC from the collection, less locations are left which may engage TAC-HRP conjugate since there are only a finite number of sites. The relationship between the color's intensity (O.D.) and standard concentration is shown by a standard curve. The TAC quantity for each sample is calculated using interpolation from this standard curve ,and lipid profiles by lipid profiles.

3. Results and Discussion

Several important conclusions came from this work into the predictive use of lipid profiles in cervical cancer patients. The disease was linked to lower levels of HDL cholesterol and greater levels of TC, TG, and LDL cholesterol, according to a detailed analysis of the blood lipid differences between cervical cancer patients and healthy women using a sizeable sample of patients.

Table (1): Comparison in parameters that show the mean of values between study groups.

Parameters	Mean ±S. E		P. value
	Control N. O=50	Patients N. O=50	
TAC(U/ml)	10.14±0.30	1.99±0.89	0.0001
TC(mg/dl)	154.43±0.52	238.30±5.64	0.0001
TG(mg/dl)	96.24±13.98	259.60±4.69	0.0001
LDL(mg/dl)	83.54±1.70	160.81±1.51	0.0001
HDL(mg/dl)	91.34±2.46	28.08±0.66	0.0001
GPX (pg/ml)	1236.43±28.73	488.10±23.57	0.0001

Lipids are crucial components of cell membranes, and the way they are processed has a significant impact on the bioenergetics and biomass generation of tumor cells. The two primary lipids found in plasma are triglycerides and cholesterol. LDL and HDL lipoproteins carry cholesterol throughout our organs. While the HDL facilitates the elimination of excess cholesterol, the LDL promotes the supply of cholesterol to the cells and the accumulation of adipose in the artery (9). Although contentious, epidemiological research showed a relationship among plasma triglyceride levels and the risk from developing cancer. A higher risk of gynaecological, thyroid, renal, lung, and prostate cancer has been linked to elevated levels of triglycerides (10).

Table (2): Comparison in correlation of parameters between healthy women and patients with cervical cancer

		TAC	TC	TG	LDL	HDL	GPX
TAC	r	1	.211	-.025	.191	.024	.048
	P-value		.142	.861	.184	.870	.739
TC	r	.211	1	-.202	-.033	-.002	.288*
	P-value	.142		.160	.822	.988	.042
TG	r	-.025	-.202	1	-.157	.467**	-.152
	P-value	.861	.160		.277	.001	.292
LDL	r	.191	-.033	-.157	1	.080	-.185
	P-value	.184	.822	.277		.581	.198
HDL	r	.024	-.002	.467**	.080	1	-.150
	P-value	.870	.988	.001	.581		.298
GPX	r	.048	.288*	-.152	-.185	-.150	1
	P-value	.739	.042	.292	.198	.298	

.*Correlation is significant at the 0.05 level (2-tailed).
.**Correlation is significant at the 0.01 level (2-tailed)., r= Pearson correlation

Triglycerides were discovered to be a major risk factor for cervix cancer in a prior cohort research among Icelanders, which would have been comparable to our discovery of enhanced lipid prolife in cervical cancer. India provides more proof that triglycerides are involved. When compared to healthy controls, cervical cancer patients' TG levels were higher (11). In light of this, a disordered lipid profile with increased TG, TC, and LDL and decreased HDL is associated with cervical cancer. Dyslipidemia and cervical cancer may be related (12). The majority of biological processes are governed by free radicals, and when they are active, they typically indicate oxidative stress in a cell. The body is protected against oxidative stress-related harm by the antioxidant system. Triglycerides were discovered to be a major risk factor for cervix cancer in a prior cohort research among Icelanders, which would have been comparable to our discovery of enhanced lipid prolife in cervical cancer. India provides more proof that triglycerides are involved. When compared to healthy controls, cervical cancer patients' TG levels were higher (11). In light of this, a disordered lipid profile with increased TG, TC, and LDL and decreased HDL is associated with cervical cancer. Dyslipidemia and cervical cancer may be

related (12). The majority of biological processes are governed by free radicals, and when they are active, they typically indicate oxidative stress in a cell. The body is protected against oxidative stress-related harm by the antioxidant system. This serves as the molecular basis for the beginning and development of cancer (14). While GPx contributed in the transformation of glutathione (GSH) to glutathione disulphide (GSSH), glutathione reductase catalyzes the reduction from GSSH to glutathione (GSH). They serve as secondary antioxidants and protect the cell against a number of cytotoxic and cancer-causing factors by eliminating ROS (2). A high GSH quality is necessary to replenish the necessary amount of antioxidants and to activate the scavenger enzymes needed to halt free radical damage. GSH concentrations in CC patients were significantly lower than those in a group of healthy control women, according to an analysis of the antioxidant's levels between the two groups of patients (15).

4. Conclusion

In conclusion, a disordered lipid profile with lower HDL levels and greater TG, TC, and LDL levels may be linked to cervical cancer. When compared to healthy controls, patients in all four categories of cases had an imbalance between their oxidant-antioxidant state. Even if the participation of these factors in oxidative stress changes, this imbalance is crucial to the etiology and development of cervical cancer. cervical cancer exhibits oxidative stress, which is reflected by altered antioxidative enzyme activity, increased lipid peroxidation. This stress is accentuated in later stages by the increasing tumor burden.

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