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ANALYSIS OF LIPID PEROXIDATION STATUS IN CERVICAL CANCER FEMALE PATIENTS AFTER RECEIVING CHEMOTHERAPY

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Abstract:

Introduction: *Cervical cancer is the second most common cancer in women, and the seventh overall. Risk factors for cervical cancer include: early age at first sexual intercourse, number of sexual partners, malnutrition, smoking and longtime use of oral contraceptives.*

Objectives of the study: *This study is aim to analyze lipid peroxidation status in cervical cancer female patients after receiving chemotherapy.*

Methodology of the study: *This cross-sectional study was conducted in Rawalpindi Medical College, Rawalpindi during June 2018 to January 2019. The data was collected from 50 cervical cancer patients. The data was collected through non probability sampling technique. 5.0 ml blood sample was taken from vein. Blood was further processed for the estimation of lipid peroxidation. Commercially available enzymatic kits of Randox were used. Blood was centrifuged at 4000 rpm for 10 minutes and serum was separated.*

Results: *The data was collected from 50 patients. The statistical analysis shows that levels of MDA become increasing in prostate cancer patients who received adjuvant radiotherapy or simple radiotherapy. The level of MDA before radiotherapy is 3.48±0.65and it become increases in post radiotherapy. As the value of MDA post radiotherapy is 5.66±0.95. But in case of adjuvant radiotherapy it becomes 3.27±0.16 (pre-treatment) and 6.79±0.40 (post-treatment). The levels of MDA become increased because cell membrane is damaged due to therapies.*

Conclusion: *It is concluded that MDA is one of the important marker of body for protecting the body against the diverse effects of radiotherapy.*

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INTRODUCTION:

Cervical cancer is the second most common cancer in women, and the seventh overall. Risk factors for cervical cancer include: early age at first sexual intercourse, number of sexual partners, malnutrition, smoking, long time use of oral contraceptives and, most importantly, Human Papilloma Virus infection [1]. Recent data revealed the role of oxidative stress in cervical cancer. The development of cervical cancer at first begins with precancerous lesions, either low squamous intraepithelial lesions (L-SIL) or the more advanced high squamous intraepithelial lesion (H-SIL). These lesions can develop further into a neoplasia [2].

Reactive oxygen species (ROS) tend to pair with adjacent molecules, which can be lipids, proteins and DNA. On the other hand, antioxidants serve as scavengers of ROS. In a healthy system, ROS and antioxidants are in a state of balance [3]. However, when the quantity of ROS exceeds that of antioxidants, the system enters a state of oxidative stress. Antioxidative enzymes, such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione reductase (GR) and glutathione-S-transferase (GST), maintain redox balance. Their activity can be used to estimate the oxidative defense mechanism strength [4]. Antioxidative enzymes inhibit both the initiation and promotion of carcinogenesis. Cervical cancer is the most prevalent genital tract cancer in the world, including India. It is a multifactorial disease process and several risk factors include, early age intercourse, multiple sex partners, low socioeconomic status, and Human papillomavirus (HPV) infection. Chronic inflammation and infection over a prolonged period of time have been recognized as major risk factor for disease initiation [5].

Carcinoma in situ is but a phase leading to frank cancer. Evidence has indicated that reactive oxygen species (ROS) are involved in the initiation and progression of carcinogenesis [6]. This may be due to the damage caused to the tumor suppressor genes or immunological defenses in our body. Superoxide and hydroxyl radicals are oxygen-free radicals, involved in producing oxidative stress. This oxidative stress can be associated with other factors which may lead to various neoplastic transformations [7].

OBJECTIVES OF THE STUDY:

This study is aim to analyze lipid peroxidation status in cervical cancer female patients after receiving chemotherapy.

METHODOLOGY OF THE STUDY:

This cross-sectional study was conducted in Rawalpindi Medical College, Rawalpindi during June 2018 to January 2019. The data was collected from 50 cervical cancer patients. The data was collected through non probability sampling technique. 5.0 ml blood sample was taken from vein. Blood was further processed for the estimation of lipid peroxidation. Commercially available enzymatic kits of Randox were used. Blood was centrifuged at 4000 rpm for 10 minutes and serum was separated. Blood samples will be collected into EDTA tubes from fasting proteins. The blood will be centrifuged and indomethacin and butylated hydroxytoluene will be added into the plasma samples before they will be stored at -80°C until analysis.

STATISTICAL ANALYSIS:

Student's t-test was performed to evaluate the differences in roughness between group P and S. Two-way ANOVA was performed to study the contributions. A chi-square test was used to examine the difference in the distribution of the fracture modes (SPSS 19.0 for Windows, SPSS Inc., USA).

RESULTS:

The data was collected from 50 patients. The statistical analysis shows that levels of MDA become increasing in prostate cancer patients who received adjuvant radiotherapy or simple radiotherapy. The level of MDA before radiotherapy is 3.48±0.65and it become increases in post radiotherapy. As the value of MDA post radiotherapy is 5.66±0.95. But in case of adjuvant radiotherapy it becomes 3.27±0.16 (pre-treatment) and 6.79±0.40 (post-treatment). The levels of MDA become increased because cell membrane is damaged due to therapies.

Table 01: MDA levels in prostate cancer patients

PROSTATE	CONTROL	MDA(moles/ml)			
		MALES (n=13)		FEMALES (n=00)	
		BEFORE	AFTER	BEFORE	AFTER
	2.35moles/ml				
R1	0.00	3.5±0.74	5.22±0.85	0.00	0.00
R2	0.00	3.6±0.82	5.42±0.80	0.00	0.00
R1+C	0.00	0.00±0.00	0.00±0.00	0.00	0.00
R2+C	0.00	3.27±0.16	6.79±0.40	0.00	0.00
C	0.00	0.00±0.00	0.00±0.00	0.00	0.00
Total	2.35	3.48±0.65	5.66±0.95	0.00	0.00

Means SD: R1=Received Radio Therapy Single Time, **R2**=Received Radio Therapy Two Times, **R1+C**=Received Radio Therapy Single Time + Chemotherapy, **R2**=Received Radio Therapy Two Times + Chemotherapy, **C**=Only Received Chemotherapy

DISCUSSION:

Cancer therapy, such as chemotherapy, can result in the generation of excess ROS/RNS. Thus cancer therapy and the resulting production of excess oxidative stress can damage biological systems other than tumors. Thus, in the present study we have demonstrated the status of lipid peroxides and antioxidants in plasma and erythrocytes of prostate cancer patients in comparison with normal subjects [6]. During chemotherapy the highest known levels of oxidative stress are generated by anthracycline antibiotics, followed in no particular order by alkylating agents, platinum-coordination complexes, epipodophyllotoxins, and camptothecins. The primary site of ROS/RNS generation during cancer chemotherapy is the cytochrome P450 monooxygenase system within liver microsomes [8]. Enzyme systems, such as the xanthine-xanthine oxidase system, and non-enzymatic mechanisms also play a role in creating excess oxidative stress during chemotherapy.

Chemotherapeutic agents used to treat cancer cause oxidative stress, which produces side effects, and among the most common side effects is chronic fatigue. Chronic fatigue caused by cancer therapy can reduce therapeutic efficacy [7]. They must also have intact apoptotic pathways. Thus oxidative stress interferes with cell cycle progression by inhibiting the transition of cells from the G0 to G1 phase, slowing progression through S phase by inhibition of DNA synthesis. This results in inhibition of cell cycle progression of the G1 to S phase, and it also results in inhibition by checkpoint arrest [9].

Lipid Peroxidation is the oxidative conversion of polyunsaturated fatty acids to MDA (malondialdehyde); which is cytotoxic and acts as a tumor promoter and a co-carcinogenic agent. Damage caused by LPO impairs the functioning of the biological membrane and the continued damage leads to loss of membrane integrity [10]. Beevi *et al* observed increased plasma as well as erythrocyte MDA in patients with cervical cancer, although there was no significant variation in lipid peroxides according to the stage of the tumor in their study.

Manju *et al*. and Kim *et al.* have similarly demonstrated the involvement of rising LPO and compromised antioxidant levels [11]. Ahmed *et al* in their study demonstrated an overall progressive impaired status of antioxidants in Ca Cx. Manoharan *et al* have demonstrated enhanced erythrocyte LPO and impaired antioxidant enzyme activities, suggesting avenues for exploring damage to the red cell membrane structure and function in cervical cancer [12].

CONCLUSION:

It is concluded that MDA is one of the important marker of body for protecting the body against the diverse effects of radiotherapy. Although many anti-neoplastic agents have clearly established mechanisms of action that are not dependent upon the generation of ROS/RNS, these drugs can only mediate their anticancer effects on cancer cells that are exhibiting unrestricted progression through the cell cycle.

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