



CODEN [USA]: IAJPBB

ISSN: 2349-7750

**INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES**<http://doi.org/10.5281/zenodo.3550945>Available online at: <http://www.iajps.com>

Research Article

**ANALYSIS OF LIPID PEROXIDATION STATUS IN
PROSTATE CANCER PATIENTS RECEIVING
RADIOTHERAPY IN PAKISTAN**Zainab Ejaz¹, Shoaib Jamil², Muhammad Faisal Shahzad²¹Sheikh Zaid Hospital Rahim Yar Khan²Bahawalpur Victoria Hospital**Abstract:**

Introduction: Prostate cancer is the fourth most common male malignancy worldwide and is the commonest malignancy of older age. The prevalence of prostate cancer increases with rise in age. It is the major cause of morbidity and mortality in male older than 65 years of age worldwide. **Objectives:** This study is aim to analyze the lipid peroxidation status in prostate cancer patients who are receiving radiotherapy in Pakistani hospitals. **Methodology of the study:** This descriptive study was conducted in Sheikh Zaid Hospital Rahim Yar Khan during January 2019 to July 2019. Those prostate cancer patients who receiving radiotherapy were selected to study the lipid peroxidation status in the diseased condition. **Results:** The data pertaining in the table explained that radiotherapy and chemotherapy both effect on the MDA levels of cancer 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 ± 0.65 and 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. 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.

Corresponding author:

Zainab Ejaz,

Sheikh Zaid Hospital Rahim Yar Khan

QR code



Please cite this article in press Zainab Ejaz et al., *Analysis Of Lipid Peroxidation Status In Prostate Cancer Patients Receiving Radiotherapy In Pakistan.*, Indo Am. J. P. Sci, 2019; 06(11).

INTRODUCTION:

Prostate cancer is the fourth most common male malignancy worldwide and is the commonest malignancy of older age¹. The prevalence of prostate cancer increases with rise in age. It is the major cause of morbidity and mortality in male older than 65 years of age worldwide². Autopsy studies have shown that every man at age of 90 almost have prostate cancer. Prostate cancer has the lowest number of life year's loss of all major cancers in men and women. It is the leading cancer diagnosed and is the second most common causes of cancer related death in men in United States³. African-American men have the highest incidence of prostate cancer in the United States and also Asian-American men have lower prostate cancer incidence than white or African-American men⁴.

Prostate Cancer is the most ubiquitous form of cancer found in men above the age of fifty years. Prostate cancer is the second most frequently diagnosed cancer and the sixth leading cause of cancer death in males, accounting for 14% (903,500) of the total new cancer cases and 6% (258,400) of the total cancer deaths in males in 2008. Incidence rates vary by more than 25-fold worldwide, with the highest rates recorded primarily in the developed countries of Oceania, Europe, and North America, largely because of the wide utilization of prostate-specific antigen (PSA) testing that detects clinically important tumors as well as other slow-growing cancers that might otherwise escape diagnosis⁵.

Prostate cancer incidence rates are strongly affected by diagnostic practices and therefore difficult to interpret, but mortality rates show that death from prostate cancer is about 10 times more common in North America and Europe than in Asia⁶. Oxidative stress is caused by an unfavorable balance between reactive oxygen species (ROS) and antioxidant defenses. ROS are generated during normal cellular metabolism, as a result of the influence of various environmental factors, as well as during pathological processes⁷.

Objectives of the study

This study is aim to analyze the lipid peroxidation status in prostate cancer patients who are receiving radiotherapy in Pakistani hospitals.

METHODOLOGY OF THE STUDY:

This descriptive study was conducted in Sheikh Zaid Hospital Rahim Yar Khan during January 2019 to July 2019. Those prostate cancer patients who receiving radiotherapy were selected to study the lipid peroxidation status in the diseased condition.

Blood collection

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 pertaining in the table explained that radiotherapy and chemotherapy both effect on the MDA levels of cancer 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 ± 0.65 and 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)	
	2.35moles/ml	BEFORE	AFTER	BEFORE	AFTER
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+C**=Received Radio Therapy Two Times + Chemotherapy**C**=Only Received Chemotherapy**DISCUSSION:**

Cancer therapy, such as chemotherapy, can result in the generation of excess ROS/RNS⁷. Thus cancer therapy and the resulting production of excess oxidative stress can damage biological systems other than tumors⁸. 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. 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 camptothecins⁹. The primary site of ROS/RNS generation during cancer chemotherapy is the cytochrome P450 monooxygenase system within liver microsomes. Enzyme systems, such as the xanthine-xanthine oxidase system, and non-enzymatic mechanisms also play a role in creating excess oxidative stress during chemotherapy¹⁰. The very high levels of oxidative stress caused by anthracyclines is also related to their ability to displace coenzyme Q10 (CoQ10) from the electron transport system of cardiac mitochondria, resulting in diversion of electrons directly to molecular oxygen with the formation of superoxide radicals¹⁰.

Anthracyclines and other chemotherapeutic agents cause generation of high levels of ROS/RNS, but not all chemotherapeutic agents generate excess oxidative stress. Some agents generate only modest amounts of ROS/RNS. Examples of this are: platinum-coordination complexes and camptothecins, taxanes, vinca alkaloids, anti-metabolites, such as the antifolates, and nucleoside and nucleotide analogues¹¹.

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.

REFERENCES:

1. Di Mascio P, Kaiser S and Sies H (1989) Lycopene as the most efficient biological carotenoid singlet oxygen quenches. Arch. Biochem. Biophys. 274, 530-532.
2. Eaton JW (1991) Catalases and peroxidases and glutathione and hydrogen peroxide: mysteries of the bestiary Lab. Clin. Med. 18,3-5.
3. Wilson MJ, Kaye D, Smith WE, Sinha AA, Quach HT and Vatassery GT (2003) Effect of vitamin E deficiency on the growth and secretory function of the rat prostatic complex. Exp Mol.Pathol. 74, 267-275.
4. Panieri, E, Gogvadze, V, Norberg, E, Venkatesh, R, Orrenius, S, Zhivotovsky, B. Reactive oxygen species generated in different compartments induce cell death, survival, or senescence. Free Radic Biol Med. 2013;57:176-187
5. Sena, LA, Chandel, NS. Physiological roles of mitochondrial reactive oxygen species. Mol Cell. 2012;48:158-167
6. Bernardes, SS, de Souza-Neto, FP, Ramalho, LN. Systemic oxidative profile after tumor removal and the tumor microenvironment in

- melanoma patients. *Cancer Lett.* 2015;361:226-232
7. Vences-Catalán, F, Rajapaksa, R, Srivastava, MK. Tetraspanin CD81 promotes tumor growth and metastasis by modulating the functions of T regulatory and myeloid-derived suppressor cells. *Cancer Res.* 2015;75:4517-4526.
 8. Jones, LM, Broz, ML, Ranger, JJ. STAT3 establishes an immunosuppressive microenvironment during the early stages of breast carcinogenesis to promote tumor growth and metastasis. *Cancer Res.* 2016;76:1416-1428.
 9. Koebel, CM, Vermi, W, Swann, JB. Adaptive immunity maintains occult cancer in an equilibrium state. *Nature.* 2007;450:903-907.
 10. Lee-Chang, C, Bodogai, M, Martin-Montalvo, A. Inhibition of breast cancer metastasis by resveratrol-mediated inactivation of tumor-evoked regulatory B cells. *J Immunol.* 2013;191:4141-4151.
 11. Hiten RH Patel. (2014) Foreword. *Expert Review of Anticancer Therapy* 14:11, pages 1251-1252.