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ANALYSIS OF LEVEL OF SIALIC ACID IN THE TREATMENT OF PROSTATE CANCER IN MALES AFTER RECEIVING RADIOTHERAPY

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Abstract:
Objectives: *The main objective of the study is to the role of sialic acid in the treatment of prostate cancer in males after receiving radiotherapy.* ***Methodology:*** *5.0 ml blood sample was taken from vein. Blood was further processed for the estimation of Sialic acid. Commercially available enzymatic kits of Randox were used. Blood was centrifuged at 4000 rpm for 10 minutes and serum was separated.* ***Results:*** *Neuraminidase is used for the hydrolytic breakage of sialic acid in the body. The data present in the table shows that there is a random change in the neuraminidase levels of prostate cancer. As it increases in those patients who received radiotherapy only one time. The value is 0.59±0.15 (pre) and 1.15±0.53 (post). But in those patients who recived radiotherapy twice the level become slightly decreases as 1.02±0.60 to 0.99±0.32. But the trend become changed in adjuvant radiotherapy. As the levels of neuraminidase become highly increases in adjuvant radiotherapy.* ***Conclusion:*** *It is concluded that sialic acid is one of the important marker of body for protecting the body against the effects of radiotherapy.*

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INTRODUCTION:

Cancer chemoprevention, which targets normal and high risk populations, involves the natural compounds or other chemical agents to inhibit, delay, or reverse cancer development. Using approaches, several thousand agents have been reported to have chemo preventive activity[1].Various types of bioactive compounds including alkaloids, coumarins, cucurbitacins, diarylheptanoids, fatty acids, flavonoids, iridoids, lignans, limonoids, naphthoquinones, oligorhamnosides, physalins, phenanthrene derivatives, polyacetylenes, stilbenoids, sesquiterpenoids, and triterpenoids have been isolated from different natural compounds and they all are involved in the cancer chemoprevention using various pathways. Many of these dietary compounds appear to act on multiple target signaling pathways.

A total of 989,600 new stomach cancer cases and 738,000 deaths are estimated to have occurred in 2008, accounting for 8% of the total cases and 10% of total deaths. Over 70% of new cases and deaths occur in developing countries. Generally, stomach cancer rates are about twice as high in males as in females Stomach cancer rates have decreased substantially in most parts of the world[2].

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 diagnosis[3]. 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 Asia[4].

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 processes. Reactive oxygen species play an important role in the pathogenesis of cancer. Oxidative stress caused by increased free radical generation and/or decreased antioxidant level in the target cells and tissues has been suggested to play an important role in carcinogenesis[4]. Free radicals are capable of altering all major classes of biomolecules, such as lipids, nucleic acids and proteins, with changes in their structure and function[5].

Objectives

The main objective of the study is to the role of sialic acid in the treatment of prostate cancer in males after receiving radiotherapy.

METHODOLOGY OF THE STUDY:

This cross sectional study was conducted in Bahawalpur Victoria Hospital during January 2019 t July 2019. Those prostate cancer patients who receiving radiotherapy were selected to study the sialic acid status in the diseased condition. 5.0 ml blood sample was taken from vein. Blood was further processed for the estimation of Sialic acid. 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:

Neuraminidase is used for the hydrolytic breakage of sialic acid in the body. The data present in the table shows that there is a random change in the neuraminidase levels of prostate cancer. As it increases in those patients who received radiotherapy only one time. The value is 0.59±0.15 (pre) and 1.15±0.53 (post). But in those patients who recived radiotherapy twice the level become slightly decreases as 1.02±0.60 to 0.99±0.32. But the trend become changed in adjuvant radiotherapy. As the levels of neuraminidase become highly increases in adjuvant radiotherapy.

Table 01: Effect of cancer therapy on sialic acid (µg/dl) in prostrate cancer patients

PROSTATE	CONTROL	NEU (µg/dl)			
		MALES (n=13)		FEMALES (n=00)	
		BEFORE	AFTER	BEFORE	AFTER
	1.41				
R1	0.00	0.59±0.15	1.15±0.53	0.00	0.00
R2	0.00	1.02±0.60	0.99±0.32	0.00	0.00
R1+C	0.00	0.00±0.00	0.00±0.00	0.00	0.00
R2+C	0.00	0.38±0.13	1.17±0.46	0.00	0.00
C	0.00	0.00±0.00	0.00±0.00	0.00	0.00
Total	1.41	0.71±0.45	1.10±0.41	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[6]. Thus cancer therapy and the resulting production of excess oxidative stress can damage biological systems other than tumors. 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[7]. 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 chemotherapy[8].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 radicals[9].

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 anti-folates, and nucleoside and nucleotide analogues[10]. However, most chemotherapeutic agents generate some oxidative stress, as do all anti-neoplastic agents when they induce apoptosis in cancer cells. Drug-induced apoptosis is usually triggered by the release of cytochrome c from the mitochondrial electron transport chain. When this occurs, electrons are diverted from NADH dehydrogenase and reduced CoQ10 to oxygen, resulting in the formation of superoxide radicals[11].

CONCLUSION:

It is concluded that sialic acid is one of the important marker of body for protecting the body against the effects of radiotherapy.

REFERENCES:

1. Cui, J, Chen, Y, Wang, HY, Wang, RF. Mechanisms and pathways of innate immune activation and regulation in health and cancer. Hum Vaccin Immunother. 2014; 10:3270-3285.
2. 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
3. Sena, LA, Chandel, NS. Physiological roles of mitochondrial reactive oxygen species. Mol Cell. 2012;48:158-167
4. 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
5. 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.

6. 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.
7. Koebel, CM, Vermi, W, Swann, JB. Adaptive immunity maintains occult cancer in an equilibrium state. Nature. 2007;450:903-907.
8. 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.
9. Hiten RH Patel. (2014) Foreword. *Expert Review of Anticancer Therapy* 14:11, pages 1251-1252.
10. Ford ME, Vernon SW, Havstad SL, Thomas SA, Davis SD. Factors influencing behavioral intention regarding prostate cancer screening among older African-American men. J Natl Med Assoc. 2006;98(4):505–14. pmid:16623062; PubMed Central PMCID: PMCPMC2569259.
11. Dube CE, Fuller BK, Rosen RK, Fagan M, O'Donnell J. Men's experiences of physical exams and cancer screening tests: a qualitative study. Preventive medicine. 2005;40(6):628–35. pmid:15850858