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Research Article

Assessment of Lipid Peroxidation and Biochemical Response in Prostate Cancer Patients

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Abstract

Background: Prostate Cancer is a prominent health risk for men all over the world. Prostate cancer is also general class of cancer in men and the second major cause of cancer death in men after lung cancer. The prostate is a male gland situated beneath the bladder that starts to develop earlier birth because of androgens. The role of the prostate is to reserve an alkaline fluid, which compose almost one-third of the volume of semen. Methodology: Blood samples (5.0 ml) of 30 diagnosed Prostate cancer patients and 30 healthy individuals were taken from vein in clotted gel vials from oncology department of Mayo hospital and Jinnah Hospital. **Results:** The spectrophotometric reading of samples portrays that MDA level in prostate cancer patients is remarkably inflated than diseased person (6.36 ± 0.43) whereas there level in healthy individual is extremely low (1.75 ± 0.24). The value of GSH demonstrates that in cancer patients the level of GSH reduced (2.12 ± 0.02) as compared to normal individual (5.32 ± 0.15). The CAT level is moderately decreased in patients (2.74 ± 0.19) than normal person (4.11 ± 1.05). Results parade the amount of SOD that is slackening in prostate cancer patients (1.14 ± 0.21) though it is high in normal people (3.12 ± 0.25). PSA values elevated in effected person (17.31 ± 18.38) than normal values (4.45 ± 1.51). This data shows that data is statistically significant (0.002). The nitric oxide (NO) score prevailed from prostate cancer patients greater (10.30 ± 0.19) than normal person (2.01 ± 0.37). This indicates that data is statistically significant (P=0.000).

Conclusion: Reactive oxygen species play an important role in carcinogenesis. It has been reported that changes in MDA level and glutathione were associated with the pathogenesis of cancer whereas increase in MDA level. The GSH level is moderately decreased in patients than normal person. Reduction in vitamin A has more chances of prostate cancer. The value of Vitamin C is extremely low in prostate cancer patients and its level is extremely high in healthy patients. PSA level elevated in effected person carried out prostate cancer.

Keywords: Prostate Cancer; MDA; Lipid Peroxidation; GSH; Vitamins

Introduction

Prostate cancer is also general class of cancer, 2nd major malignancy of cancer death [1]. In males the gland situated beneath the bladder is prostate because of androgens that develop before time to birth. An alkaline fluid is reserved by prostate gland almost volume of semen is developed. Fluid plays a significant role during the working and movement of sperm in the process of zygote formation. If six men are examined than one is suffering from and from 36, 1 is died from this disease. While prostate cancer is almost general, the progression of prostate cancer extended by the environmental prospects and genetic factor. Probably with the aging Prostate cancer is progressed significantly expresses thee more chances in aged person.

Rather than ethnicity and aging, prostate cancer could be affected by using cigarette, characteristics of hormone, food which is full of fat, and inherited` susceptibility [2,3]. Hence behavior, culture identify the danger and demonstrating the influence of prostate cancer. Although cancer of the prostate is almost devastating class of disorder. More than 40 percent of men investigated

showing the action of tumor metastasis, which is proliferation of cancer to the areas affected like cartilage, lymph gland, rectal and urinary track. The prostate diagnosis methods consist of prostate specific antigen (PSA), blood test, a digital rectal exam, tissue biopsy. Prostate manufactured protein named PSA, blood of healthy individual. The probability of cancer exceeded by 50 percent when PSA level expand then 10ng/ml and If PSA level is in between 4 and 10ng/ml, 25 percent identification of prostate cancer. Age and prostatitis could be elevating the PSA level not associated with the cancer of the prostate. Biopsy is an exact process; although involved by chance however, its limitations include its invasive carcinoma and errors in handling sample that could reveal imperfect recognition.

Major contribution in the progression of cancer recognized as Oxidative stress along with diverse common carotenoids and most powerful antioxidant is lycopene [4]. In the Ames test mutagenesis can be lessened by trapping single oxygen compound through lycopene. The formation of reactive substance of thiobarbituric acid inhibited in multilamellar liposomes show antioxidant action of carotenoids [5]. Globally prostate cancer is significantly dangerous issue all around the world at vast circumference. Early stage of cancer detected by screening procedure having minute volume, showing lessened grade. Fundamental role in the evolution of most prostate cancers carried out by androgens. Before puberty Prostate cancer rarely develops in men castrated [6]. Because most prostate cells depend on androgen for growth, androgen ablation has been the first line treatment for patients with prostatic carcinoma. Androgen ablation induces the cessation of cell proliferation and the activation of apoptosis in androgen-dependent prostatic cancer cells.

Hypoxia and its impact on prostate cancer proliferation is the indication of environment having low oxygen.

Oxygen and nutrients are permanently supplied tumor cell grow strongly hence, when mass of the tumor gain a crucial size, the blood vessels show impotent behavior to carry on over extension. Embryonic progress is initiated by the development of embryo needed to system of the arrangement of the body that have the ability for the prognosis of the cancer [7]. In the cancerous cell level of ROS exceed from the normal range during Hypoxic state, showing malignancy phenotypically of prostate cancer initiating and its therapy of rays is hindered [8]. According to Harman in 1956 proposed "theory of aging related to free radical" in which he proposed that accumulation of damage to bimolecular cells by the accumulation of free radicals and people are being old by the excess of these radicals [9,10].

Material and Methods

The whole experimental work was done in the Biochemistry Lab, School of medical Lab Technology Minhaj University Lahore after the approval of ethical and Research committee, Minhaj University Lahore.

Blood/data collection

5.0 ml blood sample of 30 prostate cancer patients and 30 Samples of healthy individuals was taken from vein in clotted gel vials from oncology department of Mayo hospital and Jinnah Hospital. Blood was further processed for the estimation of Reduce Glutathione (GSH) [11], Catalase (CAT) [12], Superoxide Dismutase (SOD) [13], Malondialdehyde (MDA) [13], Estimation of Nitric oxide (NO) [14], Estimation of micronutrients (Vitamin A, Vitamin C and Vitamin E) [15], and Electrolytes concentration by flame photometer (Na^+ and K^+). Blood sample was centrifuged at 4000 rpm for 10 minutes and serum was separated. Blood sample was collected into EDTA tubes.

Results

Table 1: Comparison of Oxidative Stress Biomarkers between Prostate Cancer Patients and Healthy Individuals.

Variables	Control (n = 30)	Patients (n = 30)	P < 0.05
MDA	1.75 ± 0.24	6.36 ± 0.43	0.000
GSH	5.32 ± 0.15	2.12 ± 0.02	0.000
Catalase (CAT)	4.11 ± 1.05	2.74 ± 0.19	0.000
SOD	3.12 ± 0.25	1.14 ± 0.21	0.000

Data presented in table 1 showed the comparison of oxidative stress biomarkers among patients suffering from prostate cancer and healthy individuals. Table revealed that lipid peroxidation occurred more in terms of malondialdehyde (MDA) in Patients (6.36) as compared to healthy subjects (1.75). Glutathione (GSH) which is also considered as universal antioxidant, the level of GSH also declined in patients (1.14) as compared to healthy persons (5.32). Catalytic activity was determined in diseased and healthy persons in terms of Catalase (CAT) which showed that serum catalase level was also decreased in prostate cancer patients (2.74) while in healthy individuals it was recorded as (4.11) which is raised. Serum superoxide dismutase (SOD) level was also dropped in cancer patients (1.14) as compared to normal individuals (3.12). Statistical evaluation shows that all oxidative stress biomarkers was significant statistically ($P = 0.000 < 0.05$).

Table 2: Comparison of Micronutrients between Prostate Cancer Patients and Healthy Individuals.

Variables	Control (n = 20)	Patients (n = 30)	P < 0.05
Vitamin A (retinol)	4.14 ± 0.44	1.37 ± 0.28	0.000
Vitamin C (Ascorbic acid)	6.35 ± 1.05	0.13 ± 0.24	0.000
Vitamin E (Alpha tocopherol)	3.39 ± 0.97	0.43 ± 0.32	0.000

Table 2 represents the serum micronutrients (Vitamin A, C and E) in prostate cancer patients as well as healthy persons. Data revealed that serum retinol level in cancer patients decreased remarkably (1.37) as compared to the normal individuals (4.14). When serum ascorbic acid (vitamin C) level was measured, it noticed that ascorbic acid level was also declined (0.13) as compared to healthy persons (6.35). Data presented in table 2 also shown that Alpha tocopherol (Vitamin E) level was dropped significantly in prostate cancer patients (0.43) as compared to healthy individuals (3.39) while data also the status of statistically significant (P = 0.000 < 0.05).

Table 3: Comparison of Prostate Specific Antigen (PSA) and biochemical markers between Prostate Cancer Patients and Healthy Individuals.

Variables	Control (n = 20)	Patients (n = 30)	P < 0.05
Prostate Specific Antigen (PSA)	4.45 ± 1.51	17.31 ± 18.38	0.002
AGE's	3.33 ± 0.47	0.27 ± 0.10	0.000
Nitric Oxide (NO)	2.01 ± 0.37	10.30 ± 0.19	0.000

Data presented in table 3 represent the level of PSA and biochemical markers estimated in prostate cancer patients and healthy persons. Data revealed that PSA level was inclined significantly (17.31) in prostate cancer patients as compared to normal persons (4.45). While Advanced glycation end products (AGE's) level was dropped remarkably (0.27) in cancer patients when compared with healthy subjects (3.33) and statistically significant (P = 0.000 < 0.05). When serum nitric oxide (NO) level was measured in cancer patients it was noticed that serum NO increased remarkably in patients (10.30) as compared to healthy persons (2.01) and data also shows significant statistically (P = 0.000 < 0.05).

Table 4: Comparison of Serum Electrolytes between Prostate Cancer Patients and Healthy Individuals.

Variables	Control (n = 20)	Patients (n = 30)	P < 0.05
Sodium (Na^+)	138.29 ± 11.27	152.22 ± 0.09	0.000
Potassium (K^+)	5.27 ± 0.13	9.11 ± 2.32	0.000

Data presented in table 4 revealed the level of serum electrolytes in cancer and healthy persons. When serum sodium level was measured it was noticed that sodium was increased in cancer patients (152.22) as compared to normal individuals (138.29) while serum potassium level also elevated in patients (9.11) as compared to healthy subjects (5.27) and data also significant statistically (P = 0.000 < 0.05).

Discussion

Free radicals destroy the cell membrane and cell, are the result of free radicals produced by the destruction of cell membrane. Mammalian tissues are able to produce free radicals in normal as well as disease state. Generally prostate cancer is malignant around the world at 2nd level [16,17]. The reason for the metastasis of the disease is the elevated level of lipids in effected individuals. Healthy individuals have low level of GSH as compared to the disease patients. Antioxidants have greater involvement for the production of free radicals. Compounds which show the reaction are formed the enzyme involving process and which don't involve it, gain or loss the electron lead to the formation of free radicals [18-21]. Chain reactions could be stopped by the addition of God gifted antioxidants like glutathione, CAT, Vit D, Vit E., free radicals elevate with their specific antibody as a result of loss, when body's defense system tired or sow no response [22].

Free radicals are the cause of loss are brought by the oxidative stress and is considered those specific individuals are affected by the reaction of therapy. The pattern of antioxidants are commenced by the defense system of the body and worked to fulfill the loss and accommodate according the requirements of the body. Free radicals are destroyed, accommodate and have not the ability to enter or action are taken by the antioxidants. The body meet with different ways to defend instead of tat are formed by the body or taking through diet. Antioxidants facilitating food sow the prevention of

the cancer of the bladder indication of various analyses. There are the chances of lessening the cancer of the prostate by absorbing food which have high concentration of antioxidants. 95 percent cases sow the early symptom of the elevation of the disease [23].

In the cancerous tissues SOD show scarcity might be linked with the lipid per oxides in advance form. In By the consumption an enzymes CAT could be slacken as the result of accession per oxidation. In the outer layer of the cell lipid as well as antioxidants that prevent the chain and soluble [24]. Antioxidants which dissolve water and have specific antigen capacity be the part of the cell and ability to reduce. By the counter balance vitamin E and Vit C radical reducing self-changed to a free state (non-reactive) although rejuvenating vitamin E [25,26].

Conclusion

Free radicals are developed in both physiological and pathological states in mammalian tissues. Reactive oxygen species play an important role in carcinogenesis. It has been reported that changes in MDA level and glutathione were associated with the pathogenesis of cancer whereas increase in MDA level was linked to metastasis. A reduce in the activity of CAT could be due to elevate in the lipid per oxidation product, malondialdehyde which can form cross links, thereby breaking up numerous membrane. Vitamin C is extremely low in prostate cancer patients and its level is extremely high in healthy patients. PSA level elevated in effected person carried out prostate cancer.

Bibliography

1. Cheung E., *et al*. "Diet and prostate cancer risk reduction". *Expert Review of Anticancer Therapy* 8 (2008): 43-50.

2. Borgermann C., *et al*. "Early detection of prostate cancer: is serum PSA testing alone sufficient?" *Urologe A*.49 (2010): 1351-1355.

3. Di Mascio P. "Lycopene as the most efficient biological carotenoid singlet oxygen quencher". *Archives of Biochemistry and Biophysics* 274 (1989): 532-538.

4. Stahl W., *et al*. "Carotenoid mixtures protect multilamellar liposomes against oxidative damage: synergistic effects of lycopene and lutein". *FEBS Letter* 427 (1998): 305-308.

5. Pienta KJ., *et al*. "Risk factors for prostate cancer". *Annals of Internal Medicine* 118 (1993): 793-803.

6. Pilat M J., *et al*. "Hormone resistance in prostate cancer". *Cancer Metastasis Reiew* 17 (1998): 373-381.

7. Cheema U., *et al*. "Switching off angiogenic signalling: creating channelled constructs for adequate oxygen delivery in tissue engeneered constructs". *European Cells Materials* 20 (2010): 274-281.

8. Anastasiadis AG., *et al*. "Tumor cell hypoxia and the hypoxia-response signaling system as a target for prostate cancer therapy". *Current Drug Targets* 4 (2003): 191-196.

9. Harman D. "Aging: a theory based on free radical and radiation chemistry". *Journal of Gerontology* 11 (1956): 298-300.

10. Harman D. "The aging process". *Proceedings of the National Academy of Sciences of the United States of America (USA)* 78 (1981): 7124-7128.

11. Moron MS., *et al*. "Levels of glutathione, glutathione reductase and glutathione-S-transferase activities in rat lung and liver". *Biochimica et Biophysica Acta* 582 (1979): 67-78.

12. Arsalan HM., *et al*. "Prospective biochemical analysis of chronic myeloid leukemia patients in response to tyrosine kinase inhibitors". *Pakistan Journal of Zoology* 55.5 (2023): 2103-2111.

13. Haider B., *et al*. "Prospective Study of Biochemical and Antioxidants Status in Oral Squamous Cell Carcinoma Patients Receiving Paclitaxel". *African Journal of Biological Sciences* 6.5 (2024): 5841-5855.

14. Moshage H., *et al*. "Nitrite and nitrate determinations in plasma: a critical evaluation". *Clinical Chemistry* 41.6 (1995): 892-896.

15. Mehmood F., *et al*. "Pre and Post treatment effect of Imatinib and Anti-oxidant status in Chronic Myeloid Leukemia Patients". *Journal of Population therapeutics and Clinical Pharmacology* 31.5 (2024): 1391-1401.

16. Zheng QS., *et al*. "Re-differentiation of human gastric cancer cells induced by ascorbic acid and sodium selenite". *Biomedical and Environmental Sciences* 15 (2002): 223-232.

17. Olusi SO. "Obesity is an independent risk factor plasma lipid per oxidation and depletion of erythrocyte cytoprotective enzymes in humans". *International Journal of Obesity and Related Metabolic Disorders* 26 (2002): 1159-1164.

18. Zima T., *et al*. "Antioxidant enzymes and lipid peroxidation in patients with multiple myeloma". *Neoplasma* 43 (1996): 69-73.

19. Sabitha KE and Shyamaladevi CS. "Oxidant and antioxidant activity changes in patients with oral cancer and treated with radiotherapy". *Oral Oncology* 35 (1999): 273-277.

20. Ghalia AA., *et al*. "Estimation of glutathione S-transferase and its Pi isoenzyme in tumor tissues and sera of patients with ovarian cancer". *Anticancer Research* 20 (2000): 1229-1235.

21. Kehrer JP. "Free radicals as mediators of tissue injury and disease". *Critical Reviews in Toxicology* 23 (1993): 21-48.

22. Cotgreave I., *et al*. "Host biochemical defense mechanisms against prooxidants". *Annual Review of Pharmacology and Toxicology* 28 (1988): 189-212.

23. Pfafferott C., *et al*. "The effect of MDA on erthrocyte deformability". *Blood* 59 (1982): 62.

24. Wagner BA., *et al*. "Slows the rate of free radical mediated lipid peroxidation in cells". *Archives of Biochemistry and Biophysics* 334 (1996): 261-267.

25. Fairfield KM., *et al*. "Human cervical carcinoma". *Disease Markers* 15 (2001): 283-291.

26. "Risk of ovarian carcinoma and consumption of vitamin A, C and E and specific carotenoids; a prospective analysis". *Cancer* 92: 2318-2326.