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Some Oxidative Stress Biomarkers among Patients with Prostate Cancer in Sokoto, North Western Nigeria

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Abstract

Globally, prostate cancer (PCa) is the most common malignancy and the second leading cause of cancer-related death in men. It is a significant contributor to the burden of diseases and affects over a million men. This study investigated the levels of malondialdehyde and plasma total antioxidant capacity among patients with prostate cancer in Sokoto. This case-control study was conducted among 28 confirmed prostate cancer patients attending the Urology clinics in Usmanu Danfodiyo University Teaching Hospital and Sokoto Specialist Hospital in North Western Nigeria. Twenty-eight age-matched healthy males were monitored as controls. Determination of Total Antioxidant Capacity (TAC) was determined using Ferric Reducing Antioxidant Power (FRAP) reagent while the Malondialdehyde in serum was determined as a conjugate with Thiobarbituric acid (TBA) acid. Data were collected using a semi-structured interviewer-administered questionnaire. Data were processed using SPSS version 20 and results were reported as Mean ± Standard deviation. The malondialdehyde level was significantly increased ($p < 0.0001$) among subjects with prostate cancer (0.215 ± 0.06) compared to controls (0.073 ± 0.04). The plasma total antioxidant capacity decreased significantly among the subjects (247.9 ± 63.3) compared to controls (743.3 ± 104.40) ($p < 0.0001$). The findings from this indicated a high Malondialdehyde (lipid peroxidation indicator) and low levels of Total Antioxidant Capacity among prostate cancer patients as evidence of redox imbalance. Subjects in monogamous relationships compared to polygamous, rural dwellers, farmers, individuals of Hausa ethnicity and subjects who reported no family history of the disease were more predisposed to prostate cancer. Further epidemiological

studies are needed to determine the predisposing factors and the potential role of these markers in the diagnosis, prognosis and management of prostate cancer patients in Sokoto in particular and Nigeria in general. We recommend that Malondialdehyde and Total Antioxidant Capacity be routinely monitored among patients with prostate cancer patients in the area.

Keywords

Some Oxidative Stress Biomarkers, Prostate Cancer, Sokoto, North Western Nigeria

1. Introduction

Globally, prostate cancer (PCa) is the most common malignancy and the second leading cause of cancer-related death in men. It is a significant contributor to the global burden of disease and the second most frequent malignancy after lung cancer in men worldwide accounting for 1,276,106 new cases and causing 358,989 deaths which amount to 3.8% of all deaths caused by cancer in men [1]. Annual incidence of PCa stands at approximately more than 1.1 million [2]. It is a major public health problem in developing countries where the incidence continues to increase and the mortality is still high [3]. Prostate cancer (PCa) is an adenocarcinoma or glandular carcinoma. It starts when the semen secreting epithelial cells mutate and become cancerous resulting in deregulation of prostate growth [4]. Despite its high incidence, little is known about the causes of the disease. The incidence rate of prostate cancer varies across the regions and populations. In 2018, 1,276,106 new cases of prostate cancer were registered worldwide, representing 7.1% of all cancers in men [1]. An estimated 1.1 million men worldwide were diagnosed with prostate cancer in 2012, accounting for 15% of the cancers diagnosed in men, with almost 70% of the cases (759,000) occurring in more developed regions. With an estimated 307,000 deaths in 2012, prostate cancer is the fifth leading cause of death from cancer in men (6.6% of the total men deaths). Prostate cancer incidence rates are highly variable worldwide. The age-standardized rate (ASR) was highest in Oceania (79.1 per 100,000 people) and North America (73.7), followed by Europe (62.1). Conversely, Africa and Asia have incidence rates that are lower than those of developed countries (26.6 and 11.5, respectively) [2]. The definitive risk factors for cancer of the prostrate are ageing, the presence of testes and dihydrotestosterone and estrogen testosterone imbalance [5]. Dietary fat, hormones, vasectomy, cadmium, vitamin A, vitamin D deficiency and sexual behaviour are probable and potential risk factors. It is characterized by clinical manifestations of locally advanced or metastatic disease such as weight loss, bone pain, lethargy, lower urinary tract symptoms of bladder outlet obstruction or irritable symptoms [5]. Although the causes of the high incidence of prostate cancer are poorly understood, epidemiological, experimental and clinical studies, suggest that oxidative stress (OS) plays a major role

in explaining prostate cancer development and progression [6] [7].

The redox equilibrium is important in preserving the correct functionality of cellular vital functions [8]. Oxidative stress is defined as the imbalance in the redox characteristics of some cellular environment which can be the result of either biochemical processes leading to the production of reactive species, exposure to damaging agents (environmental pollutants and radiations), or limited capabilities of endogenous antioxidant systems [9] [10]. Reactive oxygen and nitrogen species (ROS/RNS) produced under oxidative stress are known to damage all cellular biomolecules (lipids, sugars, proteins, and polynucleotides) [11] [12]. Thus, several defense systems have been involved within the cells to prevent uncontrolled ROS increase. These systems include non-enzymatic molecules (glutathione, vitamins A, C, and E, and several antioxidants present in foods) as well as enzymatic scavengers of ROS, with superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX) being the best-known defense systems (8). Mitochondria are the predominant source of ROS in all cell types [13]. Superoxide ($O2^{\bullet -}$) is mainly generated at the level of the mitochondrial electron transport chain and can be converted to hydrogen peroxide (H_2O_2) by SOD or undergo spontaneous dismutation [8]. In the presence of transition metal ions, for example, iron and copper ions, H_2O_2 can generate via Fenton reaction the highly reactive hydroxyl radical (HO•). Reactive species may also be enzymatically produced by xanthine oxidase (XO), uncoupled nitric oxide synthases (NOS), and NADPH oxidase (NOX). ROS production is related not only to cell damage or death, but physiological and signalling roles for ROS have also been ascertained.

Production of malondialdehyde (MDA), a well-known end product of lipid peroxidation, is up-regulated in response to an increased number of free radicals. Studies have shown that increased levels of MDA contribute to the pathogenesis of several metabolic diseases including diabetes, and cancer [14]. Malondialdehyde (MDA) is an extensively utilized biomarker to predict the pattern of various diseases such as diabetes, hypertension, cancer, heart failure and atherosclerosis. MDA has been used as a potent biomarker in both in vivo as well as in-vitro studies [15]. In patients suffering from osteoarthritis, MDA can be detected in the sections of joint tissue. In both patients suffering from lung cancer as well as glaucoma, the concentration of MDA is high; thereby validating the reliability of MDA assay to find out oxidative stress in relation to pathology of various diseases [16] [17]. A Study in a tertiary hospital in Nigeria reported lipid peroxidation with a decrease in antioxidant activity [18] among breast cancer patients.

Among the various cellular and tissue systems, red blood cells (RBCs) are uniquely vulnerable to oxidative stress due to the lack of nucleus and mitochondria, inability to synthesize fresh protein along with degradation of detoxifying enzymes, etc. So, they are among the first cells to be affected by alterations in the redox status of the body and can be explored for the early detection of pathophysiological alterations of the body in early stages [19]. A number of studies

have shown that systemic inflammation plays an important role in the development and progression of various cancers [20].

In Nigeria, with a population of nearly 180 million people, complex diseases such as cancer are currently emerging as important health care priority for the future. The subsequent attendant increase in life expectancy is likely to lead to an increase in the incidence of all types of cancers, as a higher proportion of the population reaches the complex disease-bearing age [21]. In a descriptive 10 (2006-2015) years analysis of all diagnosed cancers in the department of histopathology, Usmanu Danfodiyo University Sokoto [22], the most frequent cancers in male was prostate 267(16.00%), having a higher incidence than bladder cancer which was the most common cancer in this hospital between (1999-2004) [23]. Despite the increasing incidence of prostate cancer in Nigeria and Sokoto, and the role of oxidative stress in the pathogenesis of malignant diseases, to our knowledge, we have not seen baseline data for the level of oxidative stress in a patient with prostate cancer in Sokoto to serve as a guide for health intervention measures and future researches. Although various studies have evaluated the role of oxidative stress among patients with prostate cancer, there is limited literature available in Nigeria. The aim of this study was to assess the level of some oxidative stress biomarkers (Malondialdehyde and Total antioxidant capacity) among patients with prostate cancer attending Sokoto Specialist Hospital and the Urology Clinic in Usmanu Danfodiyo University Teaching Hospital (UDUTH) in Sokoto, North Western Nigeria.

2. Materials and Method

2.1. Background of the Study Area

The study was carried out in collaboration with the Urology Centre of Usmanu Danfodiyo University Teaching Hospital Sokoto Nigeria, the only teaching hospital serving people of Sokoto, Kebbi, Zamfara States and some neighbouring Niger and Benin Republic and Sokoto State Specialist Hospital. Sokoto State is one of the 36 states in Nigeria, located to the extreme north western part of Nigeria between longitude 4Â˚8'E and 6Â˚54'E and latitudes 12Â˚N and 13Â˚58'N. It shares common border with Niger Republic to the North, Kebbi State to the Southwest and Zamfara state to the East. The total land area is about 32,000 sq.km. In terms of vegetation, the state falls within the savannah zone. Rainfall starts late and ends early with mean annual falls ranging between 500 mm to 1300 mm. The dry season starts from October, lasts up to April in some part and may extend to May or June in other parts. The wet season on the other hands begins in most parts of the state in May and last up to September or October.

Sokoto State had a population of 3,696,999 based on the 2006 general census with estimated population of 5,297,612 projected for 2018 [24]. The inhabitants of the area are predominately Muslims and of the Hausa and Fulani ethnic groups. Other minority groups include the Zabarmawa and Tuareg. All these groups speak Hausa as a common language. The Fulani speaks Fulfulde. Other ethnic groups resident in the area Igbo, Yoruba, Nupe, Ebira, Igala, etc. It has 23

local government areas (LGAs), Five (5) of which are urban and eighteen (18) rural LGAs. The classification of urban rural areas in the state is by the National Population Commission based on a location of 16km radius from the centre of the state. The population of the area, availability of modern facilities, utilities, access road networks, banks, secondary health facilities, the state leadership and schools are available in the region. The major industrial and social infrastructure and facilities are located in the urban areas in addition to modern business and commercial ventures.

The state is divided into four health zones with 586 functional health facilities (2 tertiary, 19 secondary and 565 primary health facilities. The main economic activities in the area are farming, business, and cattle rearing. Agriculture is the backbone of the economy and riverine food plains provide cash crops such as rice, onion, groundnut, while upland areas are planted with sorghum, millet, beans and cassava. There is a generally low literacy level in western education not only of the dependent population but also the adult population with more affected. Literacy rate for women is 9% as compared with 45% for men [25]. Most women in the area are financially dependent on their husbands and most decisions on how to run the family, health issues and even social events are made by the husband and his parents [25]. Other cultural practices include "Purdah" where married women are restricted from going out except with their husband's permission and when going out have to cover their bodies fully including the face.

2.2. Study Population

The study population for this cross-sectional study comprised of prostate cancer subjects attending the Urology clinics of Usmanu Danfodiyo University Sokoto and Specialist Hospital Sokoto with apparently healthy male monitored as controls.

2.3. Inclusion Criteria

Patients with confirmed cases of prostate cancer attending Urology clinics in UDUTH and SSH, who gave a written informed consent to participate in the study were consecutively recruited until the sample size was attained.

2.4. Exclusion Criteria

Patients with confirmed cases of prostate cancer attending the Urology clinics in UDUTH and SSH, who refuse to offer a written informed consent to participate in the study, and those with prostate cancer and other comorbidities were excluded from participating in the study.

2.5. Sample Size Determination

The minimum sample size was determined using this formula [26]:

$$n = \frac{Z^2 pq}{d^2}$$

where n = minimum sample size

z = two-sided percentage of point of the normal distribution corresponding to the required significant Level (=0.05) = 1.96

p = prevalence of prostate cancer in a previous study = 2.5% [27] = 0.025

q = complimentary probability of $p = 1 - p$

d = tolerable alpha error or level of precision = 5% = 0.05

$n = 1.96 \times 0.025 \times (1 - 0.025/0.052) = 3.8416 \times 0.025 \times 0.932/0.0025$

$n = 38$

2.6. Blood Sample Collection

Three millilitres (3 mls) of blood samples were collected by venepuncture into the plain tubes. The blood sample was allowed to clot and centrifuged at 3000 rpm for 5 minutes. The serum was harvested and sample will be stored at −20˚C until used for assay.

2.7. Laboratory Analysis

Determination of Total Antioxidant Capacity (TAC) was determined using Ferric Reducing Antioxidant Power (FRAP) reagent as previously described [28]. The principle is based on the fact that at low pH, reduction of 2,4,6-tripyridyl-s-triazine (TPTZ)-ferric complex to Ferrous form (which has an intense blue colour). The colour change can be monitored by measuring the change in absorbance at 593 nm by spectrophotometry. The reaction is non-specific, any half reaction that has a lower redox potential under reaction conditions than that of ferric-ferrous half reaction will drive the ferrous ion formation. The change in absorbance is therefore directly related to the combined or total reducing power of the electron donating antioxidants present in the reaction mixture. Malondialdehyde in serum was determined as a conjugate with Thiobarbituric acid (TBA) acid. Serum proteins were precipitated by Trichloroacetic acid (TCA) and then removed by centrifugation. The MDA TBA complex was measured by spectrophotometry at 534 nm [29].

2.8. Data Collection and Management

An interviewer-administered questionnaire was used as the data collection instrument. The interviews took place within the wards and clinic at the Urology centre of UDUTH and SSH. Data processing and statistical analysis was done using a Statistical Package for Social Sciences (SPSS) version 2.3. Results were expressed as mean ± Standard Deviation. Group comparisons was made using one-way analysis of variance (ANOVA), paired comparisons were carried out using the Student's t-test. A p-value of equal to or less than 0.05 ($p \leq 0.05$) was considered as significant.

2.9. Ethical Considerations

Ethical approval was obtained from the Ethics and Research Committee of UDUTH and SSH Sokoto. Written informed consent was obtained from all study participants before enrolment.

3. Results

This study assessed some oxidative stress biomarkers among patients with prostate cancer in Sokoto. The peak age of incidence of prostate cancer was 50 - 60 years (6th decade) as shown in Figure 1. The majority of the patients in this study were of the Hausa ethnic groups representing 78.6%) (Figure 2). The distribution of the subjects based on their occupation indicated that majority were farmers (85.7%) while 14.3% were civil servants as shown in Figure 3. With regards to residence, rural dwellers (85.7%) accounted for the majority of the prostate cancer subjects compared to urban dwellers (14.3%) (Figure 4). Subjects in monogamous relationship constituted a significant number (71.4%) compared to those who practice polygamy (28.6%) as shown in Figure 5. A large percentage of the prostate cancer subjects (96.4%) reported no family history of the disease compared to 3.6% that reported history of family history of the disease (Figure 6). A total of 96.4% of the subjects were on therapy (chemotherapy, radiotherapy or combined therapy) while 3.6% were treatment naïve.

Biochemical Parameters of Study Participants

The values of MDA of test subjects were significantly increased compared with controls ($p < 0.0001$) as depicted in Table 1. The mean values of TAC of test participants decreased significantly when observed in comparison with controls ($p < 0.0001$).

Table 1. Mean Comparison of Biochemical parameters test and control subjects.

Parameters	Control (n = 28)	Test (n = 28)	t-value	p-value
MDA (µm/ml)	0.073 ± 0.04	0.215 ± 0.06	9.64	0.0001 (s)
TAC (µm/L)	743.3 ± 104.40	247. 9 ± 63.3	−21.46	0.0001 (s)

Values are presented as Mean ± SD MDA = Malondialdehyde, TAC = Total antioxidant capacity, (s) = significant, (ns) = not significant.

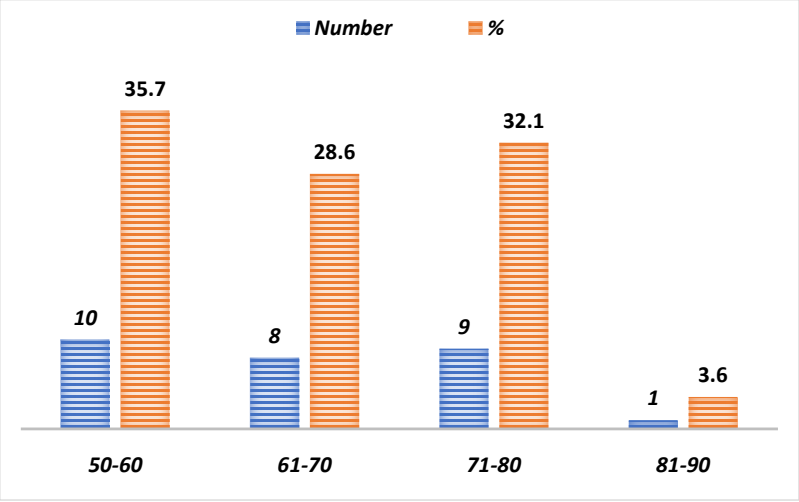

Figure 1. Age distribution of the prostate cancer subjects.

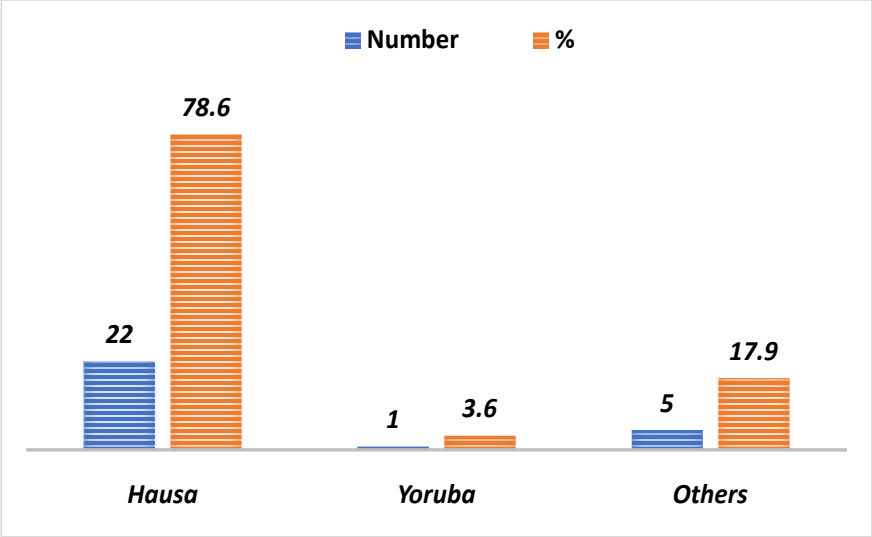

Figure 2. Distribution of prostate cancer subjects based on ethnicity.

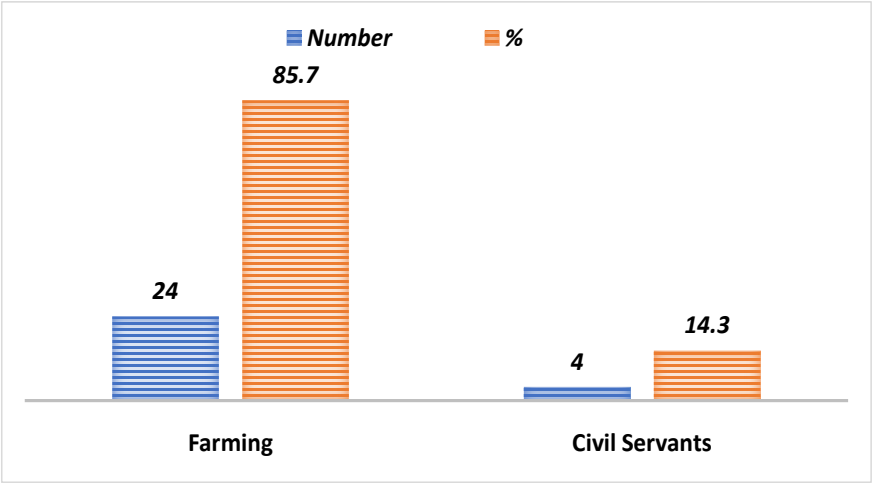

Figure 3. Distribution of prostate cancer subjects based on their occupational group.

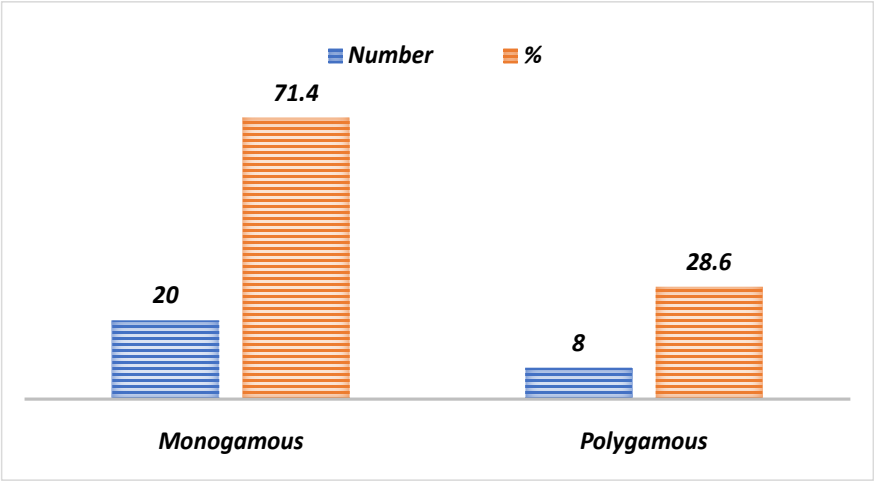

Figure 4. Distribution of the prostate cancer subjects based on their sexual orientation.

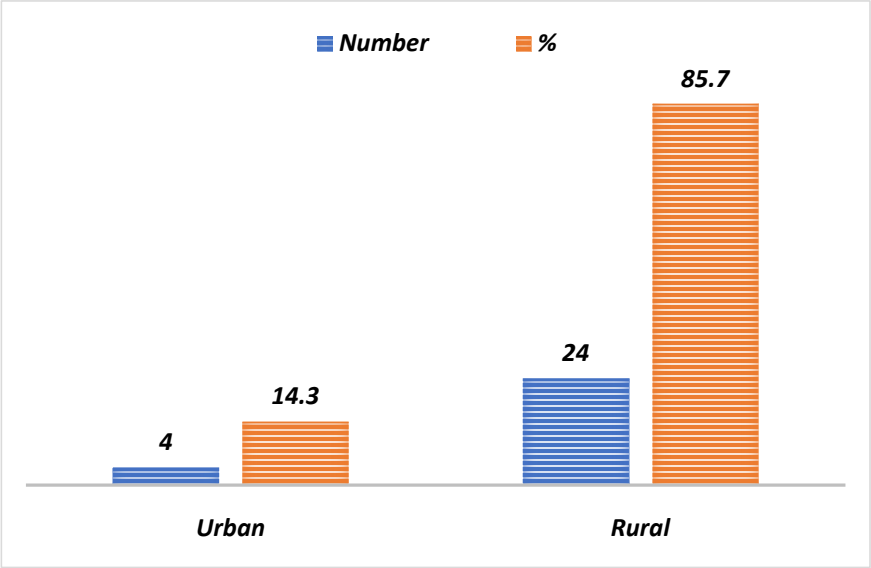

Figure 5. Distribution of prostate cancer subjects based on residence.

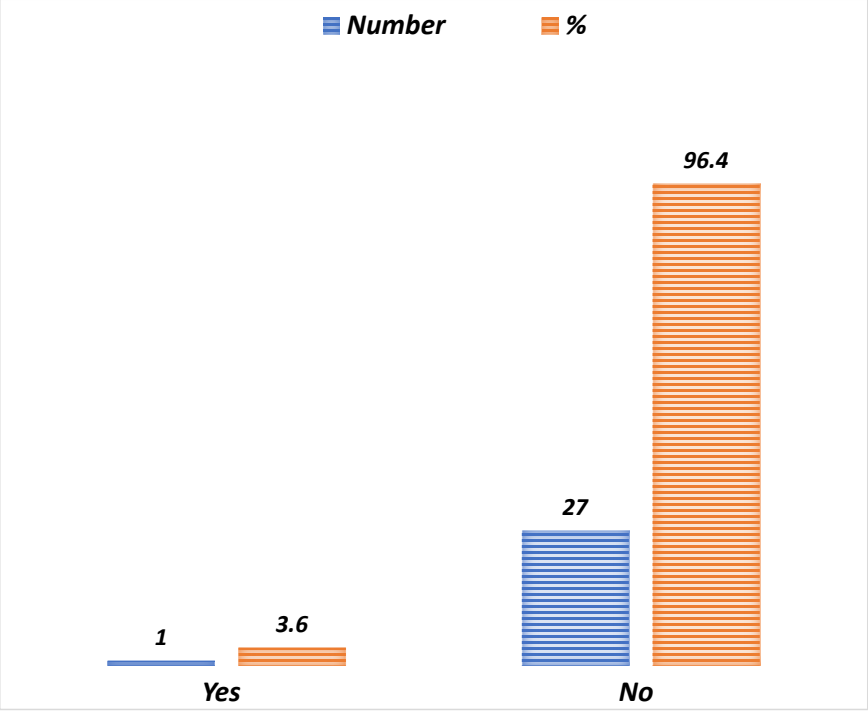

Figure 6. Distribution of the prostate cancer subjects based on family history of the disease.

4. Discussion

Prostate cancer is the second most common malignancy and the leading cause of death in men worldwide [2]. Excessive generation of oxygen-derived radicals with compromised antioxidant defense systems can cause oxidative stress. The pathogenesis of malignant processes has not been clarified yet, but substantial evidence suggests that free radicals, particularly oxygen radicals, play an impor-

tant role in the complex course of multistep carcinogenesis. Free radical generation is controlled by a large number of antioxidant systems that act as protection against free radicals. The disturbance of the pro-oxidant-antioxidant balance —resulting from increased free radical production—antioxidant enzyme inactivation or excessive antioxidant consumption is a causative factor in oxidative damage [30] [31] [32]. This study measured malondialdehyde and total antioxidant capacity of prostate cancer patients and compare with that of apparently healthy men.

In this study, the MDA value was significantly increased among the patients with prostate cancer when compared with the control groups. This is an indication of increased lipid peroxidation among the patients. The Oxidation of lipid or lipid peroxidation is one of the most commonly reported indices of oxidative stress which is recognized as a pathological factor contributing to chronic disease including cancer and aging [33] [34]. Our finding is consistent with a previous reported that oxidative stress may be involved in prostate cancer as evidenced by the higher MDA levels and lower GSH levels [35]. Our finding is consistent with previous studies that measured lipid peroxidation status in adenocarcinoma of breast and colorectal cancer [36] [37] [38] [39]. Similarly, correlation of OS and the risk of cancer in various tumours groups reported a significantly increased lipid peroxidation and DNA damage in lung, liver, head, and neck cancers and squamous cell carcinoma [40] [41] [42] [43]. These studies found higher reactive oxygen species production and enhanced lipid peroxidation in malignancies which support the oxidative stress hypothesis in carcinogenesis. In a study conducted in a tertiary hospital in Nigeria [44] to assess the levels of malondialdehyde and total oxidant capacity among prostate cancer patients undergoing androgen deprivation therapy. Results indicated that, the ADT-treated patient had higher malondialdehyde (MDA) than the controls. There was a significantly positive correlation between MDA and duration of treatment ($r = 0.280$, $p = 0.018$) in ADT-treated patients with CaP. The study demonstrated that patients with CaP have higher levels of MDA compared with men without CaP.

The study showed that oxidative stress is increased and antioxidant status decreased in patients with CaP irrespective of treatment status and that MDA levels increased with duration of treatment. This is consistent with our findings. Our finding is consistent with a previous report [45] who found high lipid peroxidation among patients with prostate cancer compared with lower levels in the control groups ($p < 0.001$). Antioxidant systems are capable of removing free radicals, thereby protecting from free radical attack from such destructive molecules as hydrogen peroxide (H_2O_2) the alkoxyl radicals ($RO^{\bullet}$), peroxyl radicals ($ROO^{\bullet}$) and superoxide dimutase ($O2^{\bullet -}$) radicals. The main groups of antioxidants make up the antioxidant defense system. These include primary, secondary and tertiary defense. Primary antioxidants prevent the formation of new free radical species. These include SOD, GPx, and metal-binding proteins (e.g. ferri-

tin or ceruloplasmin). Secondary antioxidants trap radicals thereby preventing chain reactions. These include vitamin E, vitamin C, beta-carotene, uric acid, bilirubin, and albumin. Tertiary antioxidants repair biomolecules damaged by free radicals. These include DNA repair enzymes. The concentrations of this antioxidant can be measured individually, but it is time-consuming and expensive. The total antioxidant system by FRAP assay measures the total antioxidant effect of these three defense systems in circulation. TAC measurements provide a tool for establishing links between antioxidant capacity and the risk of disease, as well as for the monitoring of antioxidant therapy [46].

The age distribution of the prostate cancer subjects indicated the incidence was highest in the 50 - 60 years' age group followed by the 71 - 80 years' age group. Our finding is consistent with a previous report which indicated that prostate cancer incidence increases with age [2]. Similarly, 1 in 350 men under the age of 50 years will be diagnosed with prostate cancer [47]. The incidence rate increases up to 1 in every 52 men for ages 50 to 59 years. The incidence rate is nearly 60% in men over the age of 65 years [48]. For African-American men, the incidence rates are higher when compared to the White men, with 158.3 new cases diagnosed per 100,000 men and their mortality is approximately twice as White men [49]. Reasons for this disparity have been hypothesized to be due to differences in social, environment al and genetic factors [47] [48] [49].

In this study, the mean plasma total antioxidant value was significantly lower in prostate cancer patients compared to that of healthy control. The findings of decreased antioxidant status are in agreement with the findings in Nigeria [47], Turkey [48] and the USA [49]. Similarly, a previous report [50] reported significantly decreased antioxidant enzymes (glutathione peroxidase and superoxide dismutase) and vitamins (vitamin C and vitamin E) in the patients with benign prostate hyperplasia and prostate cancer when compared with the control group ($p < 0.005$). In another study [18], an increased lipid peroxidation with decreased antioxidant status was observed among breast cancer patients of African Descent in Sokoto, Nigeria. Similarly, a previous report [51] proved that the antioxidant capacity of plasma in chronic obstructive pulmonary disease patients increased about 2 folds as compared with normal subjects measured using the ferric reducing ability of plasma assay. Our finding is in agreement with a previous report which indicated that there is alteration in the in the antioxidant defence system in prostate cancer patients compared to Benign Prostatic Hyperplasia (BPH) patients [50] [52]. Imbalance between the antioxidants and oxidative stress may play a role in the development of prostate cancer [53]. Our finding is however at variance with a previous report [54] who did not find any significant change in lipid peroxidation or antioxidant system parameters in the plasma of patients with BPH and prostate cancer.

The majority of the patients in this study were of the Hausa ethnic groups representing (78.6%) compared to other ethnic groups. People of Hausa/Fulani ethnicity constitute the predominant ethnic group in the study area. Previous

report in the USA indicate that African Americans are twice and three to four times as likely to develop or die from prostate cancer compared to individuals of European and Asian Americans respectively [55]. Also, prostate cancers diagnosed in African Americans tend to be of a more aggressive in nature and tend to be advanced or metastatic disease at diagnosis compared to those of European Americans [56]. Similar observations of high incidence and increased mortality have been seen among men of African descent in areas outside of USA in Jamaica and Ghana [57] [58]. The reason for this disparity in prostate cancer disposition and mortality among African men is unknown. However, environmental, genetic factors and socioeconomic factors are thought to play a role [59]. The age of attainment of puberty is also hypothesize to play a role in the increased susceptibility to prostate carcinogenesis. African American boys initiate genital development a 1 year earlier and go through longer periods of pubertal maturation compared with European American boys. Age of attainment of puberty is believed to be a potential factor in the increased susceptibility among African American men [60].

In this study, we observed that occupation seems to play a role in prostate cancer disposition. Majority of the prostrate can subjects were involved in farming (85.7%) as an occupation compared to 14.3% who were civil servants. Farming in the study area is associated with the use of fertilizers and pesticides. Exposure of these farmers to fertilizers and pesticides may be responsible for this occupational-related prostate cancer disposition. Our finding is consistent with a previous retrospective study which indicated that a range of occupations (farming, metal working, and the rubber industry) has been associated with prostate cancer [61]. Similarly, a previous report in Canada observed that persons in white collar, construction, transportation, and protective services occupations were more predisposed to prostate cancer and recommended the need for regular assessment of job-specific exposures, sedentary behaviour, psychological stress and shift work [62]. The International Agency for Research on Cancer (IARC) has reported that there is limited evidence of occupational risk factors for prostate cancer including jobs associated with exposure to arsenic, cadmium compounds, the insecticide malathion, radiation, and the rubber production industry [63]. Other occupation predisposition of prostate cancer including agriculture occupations, firefighting occupations, shift work, and whole-body vibrations has been reported [64] [65] [66] [67].

We observed a variation in the distribution of subjects with prostate cancer based on their residence. Rural dwellers (85.7%) accounted for the majority of the prostate cancer patients compared to those living in urban areas (14.3%). Our finding is consistent with a previous report which indicated that there are urban–rural variations in cancer incidence [68]. Our finding however at variance with several studies which suggest that cancer rates are higher in urban than rural areas [69] [70] [71] [72].

Subjects in monogamous relationship were 2.5 times more at risk of prostate

cancer compared to those who practice polygamy (71.4% compared to those 28.6%) respectively. The people in the study area are predominantly Muslims and Polygamy associated with marriage to a maximum of 4 wives is permissive. Our finding is at variance with previous reports which indicated that there may be an association between the number of sexual partners and prostate cancer [73] [74]. Similarly, a previous report that various dimension of sexual activity including the; age of first sexual debut, number of sexual partners, gender of the sexual partners, frequency of ejaculation and presence of sexually transmitted disease may play a role in the aetiology of prostate cancer [75] [76] [77].

A large percentage of the prostate cancer subjects (96.4%) reported no family history of the disease compared to 3.6% that reported history of family history of the disease. The question then arises whether prostate cancer does run in families and whether any genetic or hereditary factor in the predisposition to prostate cancer. A previous review provided an overview of the genetic basis underlying hereditary predisposition to prostate cancer and recommended that cost-efficient genetic testing of patients and families who may be at an increased risk (based on clinical features, family history, and ethnicity) of developing prostate cancer. There are epidemiological studies that reported that first-degree relatives of a prostate cancer patient have a two- to three-fold increased risk of developing the disease compared to the general population and the risk increases even further depending on the number of affected relatives [78]. Similarly, evidence of familial aggregation of fatal prostate cancer have been with the first-degree relatives of a patient who died of disease having a two-fold increased risk of death from prostate cancer compared to men without a family history of the disease [79].

5. Conclusion

The findings from this indicated a high Malondialdehyde (lipid peroxidation indicator) and low levels of Total Antioxidant Capacity among prostate cancer patients as evidence of redox imbalance. Subjects in monogamous relationships compared to those in polygamous relationships, rural dwellers, farmers, individuals of Hausa ethnicity and subjects who reported no family history of the disease were more predisposed to prostate cancer.

6. Recommendations

Further epidemiological studies are needed to determine the predisposing factors and the potential role of these markers in the diagnosis, prognosis and management of prostate cancer patients in Sokoto in particular and Nigeria in general. We recommend that Malondialdehyde and Total Antioxidant Capacity be routinely monitored among patients with prostate cancer patients in the area. There is need to improve the economic status of men in the study area. Enlightenment program is needed to change food custom in the region to encourage men to eat balanced diet and refrain from unhealthy food that may increase their

predisposition to prostate cancer. Access to quality healthcare should be enhanced to potentially reduce the poor prognosis associated with late diagnosis of the disease. Working habit should be improved along with the provision of protective and safety gadgets for men in the region who are occupationally predisposed to prostate cancer.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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