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EVALUATION OF LEVELS OF PROSTATE SPECIFIC ANTIGEN AND LIPID LEVELS IN PROSTATE AND NON PROSTATE CANCER PATIENTS IN PORT HARCOURT, NIGERIA

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ABSTRACT

Aim: In order to determine possible linkages between the onset of prostate cancer and some lipid characteristics, the levels of Prostate Specific Antigen (PSA) and Lipid characteristics of prostate and non -prostate cancer subjects were evaluated.

Methods: Blood samples were collected from 100 subjects; 50 of whom had newly been diagnosed for prostate cancer but had not yet been on medication and the other 50 as non -prostate cancer subjects. Fasting Blood samples were collected for Lipid Profile and PSA.

Results: The age range of the subjects was from 40-89 years. 84% of diseased subjects were within the age range of 50-79 years while 34% fell within the 70-79 years age range. Mean levels of PSA, Total Cholesterol, Triglycerides, HDL and LDL in the Prostate cancer subjects were respectively as follows: 51.50 ± 39.05µg/l; 6.37 ± 1.63mmol/l; 1.90± 0.59mmol/l; 0.91 ± 0.18 mmol/l and 4.44 ± 1.46 mmol/l. On the other hand, the Mean PSA, Total Cholesterol, Triglycerides, HDL and LDL in the Control subjects were 2.12± 1.40µg/l; 4.08± 1.03mmol/l; 1.23±0.48mmol/l and 2.53±0.88mmol/l respectively. The correlation matrix showed a moderately positive correlation between PSA and Cholesterol(r=0.43) and between PSA and LDL(r=0.38).

Conclusion: Advanced age, high PSA, Total cholesterol, Triglycerides and LDL levels may be predisposing factors to the incidence of prostate cancer, while high HDL may inhibit the onset of the disease.

Key words: Prostate Specific Antigen, Lipid profile, Cancer

INTRODUCTION

Prostate Cancer, a form of cancer that develops in the prostate gland of the male reproductive system, has become one of the major causes of death amongst males. Although its aetiology is obscure, the disease has been linked to a number of risk factors. These include advanced age, dietary habits, extravagant lifestyles and occupational hazards (Waalkes *et al*., 2001). Signs and symptoms of the disease have been linked to frequent and painful urination, which is usually bloody, erectile dysfunction as well as painful sexual intercourse and waist pain (American Society for Cancer, 2015). Studies by Ogunbiyi & Shittu (1999) indicated that prostate cancer constitutes about 11% of all male cancer cases in Nigeria. In Port Harcourt, a study conducted by Obiorah & Nwosu, (2011) on 528 histological specimens of cancer revealed that 34.7% of the cases confirmed prostate carcinoma – possibly indicating an increased incidence of prostate cancer in Nigerian males. Recent records (American Society for cancer, 2015) have reported an incidence of 35% -50% among African Americans in the United States of America. The apparent increased incidence of prostate cancer has necessitated the scientific investigation of the scourge. In this study; the relationship between some risk factors and the onset of prostate cancer in patients in Port Harcourt,

Nigeria has been investigated. Port Harcourt is one of the highly industrialized cities in Nigeria. It is the hub of oil and gas industry, accommodates an airport, two seaports and serves as the seat of government of Rivers state. As a result of these facilities in the city, micro, small and large scale industries have sprung up with accompanying population increase in recent years. The growth of Port Harcourt in terms of population and space, has been rapid since its creation in 1913. Census figures for the city have increased through the years; from 71,634 in 1953 to 645,883 in 1991. The 2006 census estimates the population of Port Harcourt at 1,382,592 (Arizona-Ogwu, 2009). Concomitant to this population explosion in the city are the associated varied lifestyles and social vices engaged by the inhabitants in the different strata of the economy. In an earlier study of about 22,895 Norwegian men of 40years and above (Nilsen *et al*., 2000), it was observed that there was a high association between high socioeconomic status and prostate cancer. With the advent of eateries, fast food centres and restaurants in Nigeria, the consumption of saturated and unsaturated fats as well as other chemicals (meant for attractiveness, palatability and preservation of foods), has increased in most urban centres (e.g. in Port Harcourt) (Mathias & David, 2015) and this trend has resulted in the change of lifestyle of urban dwellers especially, among those in the high socioeconomic cadre. Fats and chemicals, generally, have been considered responsible for various types of diseases, including cardiovascular diseases (Moyad, 2003). Such reports have prompted the investigation, in this study, of the relationship between Prostate cancer and lipid profile of male patients in Port Harcourt.

MATERIALS AND METHODS

A total of one hundred (100) subjects were selected; 50 baseline subjects diagnosed for prostate Cancer using Direct Rectal Examination (DRE) and PSA. Inclusion criteria to this group were those who had a PSA >10ug/l and had a high prostate volume but who were not yet on medication. The 50 other subjects served as control i.e. non-prostate cancer patients having a PSA level < 4 µg/l. Fasting Blood samples were collected by venepuncture into sterile, plain vacutainer bottles from the subjects. Each blood sample was centrifuged at 3000 rpm for 3 minutes and serum aliquots were put into two sets of micro- vial tubes. One set was used for Lipid Profile analysis and the second set for Prostate Specific Antigen (PSA) analysis. The micro-vials were stored frozen at -20^{o}C and then allowed to thaw to room temperature for analysis. PSA was analysed using the PSA Enzyme linked Immunoassay test which was a solid phase two-site immunoassay. Rabbit anti-PSA was coated on the surface of the microtitre wells and another anti-PSA monoclonal antibody labelled with horseradish peroxidise was used as the tracer. The PSA molecules present in the standard solution or serum were "sandwiched" between the two antibodies. Following the formation of the coated antibody-antigen-antibody enzyme complex, the unbound antibody-enzyme tracers were removed by washing. The horseradish peroxidase activity bound in the wells was then assayed by a colorimetric reaction. The intensity of the colour formed was proportional to the concentrations of PSA present in the samples (Diagnostic Automation Inc. 2003). The lipid characteristics analysed were Total Cholesterol (TC), Triglycerides (TG) as well as High and Low Density Lipoproteins (HDL and LDL). Total Cholesterol was determined after enzymatic hydrolysis and oxidation, (Trinder, 1969). The indicator, quinoneimine, was formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase. The colour formed was read against a reagent blank in a colorimeter -WPA 705. Serum triglyceride (TG) was analysed using enzymatic hydrolysis with lipases. The indicator was quinoneimine formed from hydrogen-peroxidase, 4-aminophenazone and 4- chlorophenol under the catalytic influence of peroxidase. The colour formed was read against a reagent blank at 550nm wavelength from a WPA-705 colorimeter. High Density Lipoprotein (HDL) was determined using dextrin sulphate-magnesium acetate method, in this method, Low density lipoprotein, very low density lipoprotein and chilomicrons fractions were precipitated quantitatively by the addition of phosphotungstic acid in the presence of magnesium ions. After centrifugation, the cholesterol concentration in the HDL-C fraction, which remains in the supernatant, was determined, using the method for total cholesterol (Trinder, 1969). Low density Lipoprotein (LDL) was determined by difference using the Friedwald equation LDL = (TC-(TG/2.2) + HDL) (Friedwald *et al*., 1972). The commercial kits containing the reagents

were products of Randox Laboratories Limited, United Kingdom.

RESULTS

Fig 1 depicts the frequency polygon of the age ranges of the control and diseased subjects in the study. 84% of diseased subjects were within the age range of 50-79 years while 34% fell within the 70-79 years age range.

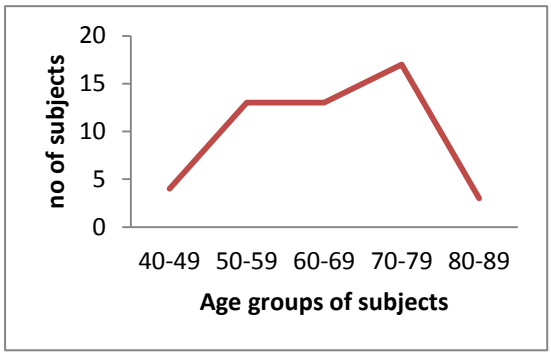

Fig 1: Distribution of prostate cancer subjects in age groups.

A summary of the levels of PSA and lipid profile is presented in Table 1.The mean PSA level in the diseased (51.50 ± 39.05µg/l) was significantly higher ($p<0.05$) than in the control (2.12 ± 1.40 µg/l). Similarly, the mean TC (6.37 ± 1.63mmol/l), TG (1.90 ± 0.59mmol/l) and LDL (4.44mmol/l ± 1.46) levels in the diseased patients were significantly higher than in the control (4.08mmol/l ± 1.0), (1.23mmol/l ± 0.48) and (2.53mmol/l ± 0.88) respectively. On the other hand, mean HDL level in the diseased (0.91mmol/l ± 0.18) was significantly lower than in the control (1.01mmol/l ± 0.19). A correlation matrix (Table 2) to establish a relationship between PSA and the lipid profile indicated that PSA correlated moderately positively with TC (r=0.43),TG (r= 0.32) and LDL (r=0.38).

Table 1: PSA and lipid characteristics of subjects

Parameter		Diseased subjects	Control subjects
PSA (µg/l)	Range	10.3-143.2	0.3-6.9
	Mean	51.50± 39.05	2.12 ± 1.40
TC (mmol/l)	Range	4.2 – 10.7	2.5 -7.2
	Mean	6.37 ± 1.63	4.08 ± 1.0
TG (mmol/l)	Range	1.0 – 3.0	0.6 – 2.5
	Mean	1.90 ± 0.59	1.23 ± 0.48
HDL (mmol/l)	Range	0.6 – 1.3	0.7 - 1.3
	Mean	0.91 ± 0.18	1.01 ± 0.19
LDL (mmol/l)	Range	1.8 – 8.4	1.2 -5.5
	Mean	4.44 ± 1.46	2.53 ± 0.88

Table 2: Correlation matrix of PSA and the Lipid characteristics

	r value
PSA: TC	r = 0.43
PSA: TG	r = 0.32
PSA: HDL	r = -0.13
PSA: LDL	r = 0.38

DISCUSSION

The results indicate that 84% of diseased subjects were within the age range of 50-79 years while 34% fell within the 70-79 years age range. This trend is in agreement with studies by Ogunbiyi *et al*., (1999), who noted that advancement in age was a risk factor in the development of prostate cancer. The Mean PSA, Total Cholesterol and LDL levels in the prostate cancer subjects were significantly higher than in the Control subjects. On the other hand, mean HDL level in the diseased was significantly lower than in the control. This observation is indicative of the significance of PSA level in the development of prostate cancer, since the healthy subjects (control) recorded very low PSA levels. Also these trends may imply that high levels of TC, TG and LDL are contributory to the onset of prostate cancer whereas a high level of HDL may inhibit its possible onset. An earlier study (Crowe *et al*., 2008) had found that up to 30% of the rate of growth of prostate cancer cells could be inhibited by having a low-fat, high fibre diet combined with regular exercise. The correlation matrix (Table 2) to establish a relationship between PSA and the lipid profile indicated that PSA correlated moderately positively with TC (r=0.43),TG (r= 0.32) and LDL (r=0.38). This trend was in agreement with the study by Moyad *et al*., (2003) which noted that most prostate cancer patients had high cholesterol levels.

CONCLUSION

The results of the study indicate that advanced age, high PSA, Total cholesterol, Triglycerides and LDL levels may be predisposing factors to the incidence of prostate cancer. On the other hand, high HDL may inhibit the onset of the disease.

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