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The Role of Lipoproteins in Possible Development of Benign Prostatic Hyperplasia and Prostate Cancer Patients in Owo, Ondo State

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Abstract: *Objectives*: To determine the roles of all classes of lipoproteins in the existence of Prostate Cancer and Benign Prostatic Hyperplasia. *Materials and Methods*: The subjects of this study were men attending urology clinic at Federal Medical Centre, Owo who are already diagnosed with prostate cancer or benign prostatic hyperplasia. 50 men already diagnosed with Prostate cancer and 50 men already diagnosed with Benign prostatic hyperplasia were recruited for this study. Lipid profiles (High Density Lipoprotein, Total Cholesterol, Triglycerides and Low Density Lipoprotein) were determined spectrophotometrically while Prostate Specific Antigen was determined using ELISA method. Data obtained were statistically analyzed using one-way analysis of variance and $P < 0.05$ was considered significant. *Results*: This study shows the mean values of Triglycerides (3.94 mmol/L ± 0.27) in Prostrate Cancer patients to be significantly higher than the mean values of Triglycerides (1.08 mmol/L ± 0.46) in Benign Prostatic Hyperplasia patients. While the mean values of High Density Lipoproteins (0.73 mmol/L ± 0.03) and Low Density Lipoproteins Levels (3.67 mmol/L ± 0.13) were lower in Prostrate Cancer patients than the mean values of High Density Lipoproteins (0.83 mmol/L ± 0.04) and Low Density Lipoproteins Levels (3.69 mmol/L ± 0.12) in Benign Prostatic Hyperplasia patients, these significant differences may be accounted for by the age, race or diets of the subjects. *Conclusion*: There are significant increase in the levels of Total Cholesterol and Low Density Lipoprotein-Cholesterol in both Prostate cancer Patients and Benign Prostatic Hyperplasia patients which constitutes a risk factor for both prostate diseases.

Key words: Benign Prostatic Hyperplasia · Prostate Cancer · Lipid Profiles · Prostate Specific Antigen

INTRODUCTION

Lipoproteins square measure advanced particles that have a central hydrophobic core of non-polar lipids, primarily sterol esters and triglycerides. This hydrophobic core is enclosed by a hydrophilic membrane consisting of phospholipids, free sterol and apolipoproteins [1]. Plasma lipoproteins square measure divided into categories supported size, lipid composition and apolipoproteins [1]. Lipids square measure comparatively insoluble in binary compound media, they're transported in body fluids as typically spherical, soluble macromolecule complexes referred to as lipoproteins [2]. Lipids derived from food (exogenous) or synthesized within the body (endogenous). The soluble (polar) teams of proteins, phospholipids associated free sterol face outward and surround an inner insoluble (non polar) core of glyceride and sterol esters. Lipids transported in association with proteins (lipoproteins) within the circulation. These lipoproteins play a key role within the absorption and transport of dietary lipids from the liver to peripheral tissues and vice versa [2].

Lipoproteins square measure classified by their buoyant density, that reciprocally reflects their size. Lipoproteins classified into 5 main teams; As posited by Martin [3], the primary 3 are triglyceride rich, due to their massive size; they scatter light-weight, which might provide plasma a cloudy look (lipaemic) if present in high concentrations:

- Chylomicrons square measure the biggest and least dense lipoproteins and transport exogenous lipid from the internal organ to any cells.
- Very low-density lipoproteins (VLDLs) transport endogenous lipoid from the liver to cells.

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- Intermediate-density lipoproteins (IDLs), that square measure transient and fashioned throughout the conversion of very low density lipoprotein to low density lipoproteins (LDL), don't seem to be commonly present in plasma.

The alternative 2 compound protein categories contain primarily sterol and square measure smaller in size:

- Low-density lipoproteins square measure fashioned from VLDLs and carry sterol to the cells.
- High-density lipoproteins (HDLs) square measure the densest lipoproteins and square measure concerned within the transport of sterol from cells back to the liver (reverse sterol transport) [4].

These lipoproteins will be more divided by density into HDL2 and HDL3. Additionally, the prevalence of elevated blood total cholesterol (TC) levels was highest in the World Health Organization of Europe (54% for each sexes) followed by the World Health Organization Region of the Americas (48% for each sexes); the prevalence of high TC levels was rock bottom in the World Health Organization Region of African (22.6%) and in South East Asian Region (29.0%) [4].

Benign prostate Hyperplasia (BPH) and Prostate Cancer (PCa) square measure among serious health issues rising with maturity within the increasing male population worldwide [5]. Benign prostatic hyperplasia (BPH) and Prostate Cancer (PCa) square measure each epidemiologically and histopathologically endocrine dependent diseases and prostatic inflammation associated chronic diseases requiring long time each for development and progression [5]. Benign prostatic hyperplasia have an effect on adults of all ages, however it will co-exist with prostate cancer and its mechanistic relationship to prostate cancer and to alternative pre-malignant conditions like prostate inflammatory atrophy (PIA) is unsure [5].

Prostate Cancer: In each malign and benign prostate diseases, there's associate imbalance between prostate cell growth and cell death as a result of intrinsic and extraneous factors having a direct or indirect impact on prostate tissue growth and differentiation [5].

Prostate cancer will be a serious unwellness, however most men diagnosed with prostate cancer don't die as a result of it. It's the second commonest reason behind cancer and also the sixth leading reason behind cancer death among men worldwide indeed, quite 2.5 million men within the U.S World Health Organization are diagnosed with Prostate cancer at some purpose square measure still alive these days. In Asian country, prostate cancer was fifth commonest tumor occurring in 7.3% of all men [6].

Geographically, prostate cancer incorporates a high incidence in Western Europe and North America, however a low incidence in Asia [7], that suggests that the Western mode or atmosphere might need a causative role within the incidence of prostate cancer in these areas [8].

Prostate cancer is common in most Western countries like U.S, it's the foremost usually diagnosed cancer and ranks second in terms of cancer mortality [9]. A study administered by Barnard [10] has shown a awfully high prevalence rate of cancer of the prostate (30.8%) in Owo, Ondo State. additionally to age, race and case history of prostate cancer, the documented risk factors for prostate cancer embrace environmental and lifestyle-related factors like fleshiness, physical inactivity and a high-fat diet [11]. Many markers, as well as the Prostate Specific Antigen (PSA) take a look at and established the fact about patients with prostate cancer.

Benign Prostate Hyperplasia: Benign prostate hyperplasia (BPH) is one amongst the foremost common conditions moving the old males, as the old represent the major proportion of the population [12]. The enlargement of the prostate will manufacture expelling symptoms, that will lead to pathological changes in urinary bladder and the urinary organ [12].

Worldwide, diseases of Prostate gland square measure accountable for vital morbidity and mortality among adult males [13]. it's calculable that range of males within the U.S World Health Organization can expertise inflammation throughout their lifetimes vary up to five hundredth. BPH affects most men as they develop. It will result in urinary issues like those with inflammation. hyperplasia seldom causes symptoms before age forty, however additional than 0.5 of men in their 60s and most men in their 70s and 80s can have signs of hyperplasia [13].

The prostate is about the size of a walnut once a person is in his 20s. By the time he's forty, it's going to have full-grown slightly larger, to the scale of associate apricot. By age sixty, it could be the size of a lemon. The enlarged prostate will press against the bladder and also the channel. This may slow down or block piddle flow [13]. Some men would possibly notice it laborious to begin a piddle stream, although they feel the necessity to go. Once the piddle stream has started, it's going to be laborious to prevent. Alternative men could desire they have to pass piddle all the time or they're been woken up with the sudden urge to pass piddle [13].

However, lipoprotein sterol (HDL-C) has recently received a lot of attention as a attainable risk marker of prostate cancer development and prognosis. Identification of risk factors and screening tools are important in preventing and treating prostate cancer. Recently, the chance that lipoprotein sterol (HDL-C) and HDL particles square measure related to cancer has generated considerable interest [14]. Lipoproteins, significantly HDL particles, are essential parts within the reverse sterol transport pathway whereby lipid homeostasis in peripheral tissues is controlled by removal of excess cellular sterol by HDL and delivery to the liver for excretion. Blood levels of HDL-C square measure stricken by chronic inferior inflammatory diseases like cancer, however HDL may additionally have an immediate pathophysiological role in prostate cancer development and/or progression [14].

The hypothesis that there is a significant difference in the levels of lipoproteins in the development of Benign Prostatic Hyperplasia (BPH) and Prostate Cancer was supported by several findings as stated above. On the basis of these suggestions, the aim of this study was to evaluate the association between serum total cholesterol, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides in the development of Benign Prostatic Hyperplasia (BPH) and Prostate Cancer. For this purpose, we measured in serum samples withdrawn locally through venous puncture the Total Cholesterol, Triglycerides, High Density Lipoproteins, Low Density Lipoproteins and Prostate Specific Antigen levels as specific markers of the Prostate gland.

MATERIALS AND METHODS

The study is a hospital based cross-sectional study among men with already diagnosed prostate cancer and Benign Prostatic Hyperplasia (BPH). The subjects were selected using a well structured questionnaire who were age and sex matched. Informed consent was obtained from subjects, hospital authorities, laboratory scientists and clinicians involved in the management of the patients. A total of 100 consecutive patients from the Urology clinic of the Federal Medical Centre, Owo. 50 affected by BPH and 50 affected by PCa were selected for the current study. The duration of this study was about two months.

Experimental Materials used include Spectrophotometer, sample bottles (lithium heparin and plain bottles), needle/syringe, cotton wool, PSA ELISA kit from Inteco Diagnostics Uk limited with Catalog Number: 10109, Randox reagents for lipid profiles, test tubes, micro pipettes, test tube rack, methylated spirit, hand gloves, incubator, ELISA machine DR-200Bc.

In Serum Samples, We Measured: Total Cholesterol, Triglycerides and High-Density Lipoproteins levels Spectrophotometrically While Low Density Lipoproteins levels was calculated using (TC- $^{TG}/_{2.2}$- HDL) formula. Serum samples were stored appropriately until analysis was done. PSA levels were measured in serum samples using Elisa Kits.

Parameters to Be Analyzed:

Determination of Total Cholesterol

Test Principle: Determination of cholesterol after enzymatic hydrolysis and oxidation. The colorimetric indicator is quinoneimine which is generated from 4-aminoantipyrine and phenol by hydrogen peroxide under the catalytic action of peroxidase (Trinder's reaction).

Cholesterol ester + H_2O → CHE Cholesterol + fatty acid
Cholesterol + O_2 → CHO Cholesteol-3-one + H_2O_2
2 H_2O_2 + Phenol + 4 Aminoantipyrine → POD Quinonimine + 4 H_2O

The intensity of the pink/red colour is proportional to the Cholesterol concentration in the sample.

Procedure:

Bring reagents and samples to room temperature

Pipette into test tubes	Blank	Standard	Sample
Reagent	1000 µl	1000 µl	1000 µl
Sample	-	-	10 µl
Standard	-	10 µl	-
Distilled water	10 µl	-	-

Mix and Incubate for 20 minutes at 37°C. Read absorbance at 546nm of sample and standard within 60 minutes against the reagent blank

Reference Range:

Normal level ---- Less than 200mg/dL (5.2 mmol/L)
Borderline high-risk ---- Between 200 to 239 mg/dL (5.2-6.2 mmol/L)
High-risk ----- More than 240 mg/dL (6.2mmol/L)

Determination of Triglycerides:

Principle: Determination of triglycerides after enzymatic splitting with lipoprotein lipase. Indicator is quinoneimine which is generated from 4-aminoantipyrine and 4-chlorophenol by hydrogen peroxide under the catalytic action of peroxidase.

Triglycerides → LPL Glycerol + fatty acid
Glycerol + ATP → GK Glycerol-3-phosphate + ADP
Glycerol-3-phosphate + 0_2 → GPO Dihydroxyaceton phosphate + H_2O_2
2 H_2O_2 + Aminoantipyrine + 4-Chlorophenol → POD Quinoneimine + HCl + 4 H_2O

Procedure:

Bring reagents and samples to room temperature.

Pipette into test tubes	Blank	Standard	Sample
Reagent	1000 µl	1000 µl	1000 µl
Sample	-	-	10 µl
Standard	-	10 µl	-
Distilled water	10 µl	-	-

Mix and Incubate 20 minutes at 37°C. Read absorbance at 546nm of sample and standard within 60 minutes against the reagent blank.

Reference Range:

The normal range of triglycerides: Between 100 to 150mg/dL (2.6-3.9 mmol/L).
Borderline High-Risk: Between 150 to 199mg/dL (3.9- 5.2 mmol/L)
High-Risk: Between 200 to 499mg/dL (5.2-12.9 mmol/L)
Very High-Risk:500 mg/dL (13 mmol/L)

High Density Lipoproteins-cholesterol:

Principle: Phosphotungstic acid and magnesium ions selectively precipitating all lipoproteins except the HDL fraction – cholesterol present in the supernatant can be determined by the same method used for total cholesterol.

Procedure:

Precipitation

Sample	0.25ml
Precipitating reagent	0.5ml

Vortex, let stand for 10 minutes, centrifuge for 15 minutes at 3000 rpm. Measure HDL –Cholesterol in the supernatant using the same method for total Cholesterol.

Determination of HDL-Cholesterol:

	Blank (ml)	Standard (ml)	Sample (ml)
Distilled water	0.1	-	-
Standard(R2)	-	0.1	-
Supernatant	-	-	0.1
Working reagent	1.0	1.0	1.0

Mix and incubate at 37°C for 20 minutes. Read absorbance at 546nm of sample and standard against the blank.

Reference Range:

Men: 30 – 70 mg/dL (0.78- 1.82 mmol/L)
Women: 35 – 85 mg/dL (0.91- 2.21 mmol/L)
For men- lower than 40mg/dL (1.04 mmol/L) indicates high risk.
For women- lower than 50mg/dL (1.30 mmol/L) indicates high risk.

Low Density Lipoprotein

Principle: This direct method for quantifying cholesterol in low-density lipoproteins (LDL) is a homogeneous enzymatic test in which the differential precipitation and further sedimentation of the rest of lipoproteins and chylomicrons is avoided. The procedure comprises two steps. In the first step cholesterol in lipoproteins other than LDL in the test sample are decomposed by the simultaneous action of cholesterol esterase (CE) and cholesterol oxidase (CO) at pH 7.0, giving as end products cholesterol and hydrogen peroxide, the latter being decomposed to water and oxygen by catalase. In the second step a surfactant which specifically acts on LDL is added to the reaction product of the first step being the remaining cholesterol quantified by a Trinder´s type reaction in which the aniline derivate, HDAOS{ N-(2-hydroxy-3-sulfopropyl)-3, 5-dimethoxyaniline} and 4-aminoantipyrine (4-AA) as a coupling reagent are condensed by the H_2O_2 in presence of peroxidase (POD) to form a red quinonimine dye proportional to the concentration of LDL-cholesterol present in the sample.

Reference Range:

LDL-Cholesterol levels
< 100 mg/dL (2.59 mmol/L) -- Optimal
100 - 129 mg/dL (2.59-3.34 mmol/L) -- Near optimal
130 - 159 mg/dL (3.37-4.12 mmol/L) -- Borderline
160 - 189 mg/dL (4.14-4.89 mmol/L) -- High
≥ 190 mg/dL (4.92 mmol/L) -- Very high

Prostate Specific Antigen (Psa) Test: Measurement of PSA was done using ELISA technique.

Principle of the Test: The PSA ELISA test is based on the principle of a solid phase enzyme-linked immunosorbent assay. The assay system utilizes a rabbit anti-PSA antibody directed against intact PSA for solid phase immobilization (on the microtiter wells). A monoclonal anti-PSA antibody conjugated to horseradish peroxidase (HRP) is in the antibody-enzyme conjugate solution. The test sample is allowed to react first with the immobilized rabbit antibody at room temperature for 60 minutes. The wells are washed to remove any unbound antigen.

The monoclonal anti-PSA-HRP conjugate is then reacted with the immobilized antigen for 60 minutes at room temperature resulting in the PSA molecules being

sandwiched between the solid phase and enzyme-linked antibodies. The wells are washed with water to remove unbound labeled antibodies. A solution of TMB Reagent is added and incubated at room temperature for 20 minutes, resulting in the development of a blue color. The color development is stopped with the addition of Stop Solution changing the color to yellow. The concentration of PSA is directly proportional to the color intensity of the test sample. Absorbance is measured spectrophotometrically at 450 nm.

Procedure:

- The desired number of coated wells was secured in the holder.
- 50 µl of standards, specimens and controls was dispensed into appropriate wells.
- 50 µl of Zero Buffer was dispensed into each well.
- It was thoroughly mixed for 30 seconds as it is very important to have a complete mixing in this setup.
- It was incubated at room temperature (18-25°C) for 60 minutes.
- The incubation mixture was removed by emptying plate contents into a waste container.
- The microtiter wells was rinsed and emptied 5 times with distilled or deionized water. (Tap water was not used).
- The wells was stroked sharply onto absorbent paper or paper towels to remove all residual water droplets.
- 100 µl of Enzyme Conjugate Reagent was dispensed into each well and was gently mixed for 10 seconds.
- It was incubated at room temperature (18-25°C) for 60 minutes.
- The incubation mixture was removed by emptying plate contents into a waste container.
- The microtiter wells was rinsed and emptied 5 times with distilled or deionized water. (Tap water was not used).
- The wells were stroked sharply onto absorbent paper to remove residual water droplets.
- 100 µl of TMB Reagent was dispensed into each well and was gently mixed for 10 seconds.
- It was incubated at room temperature for 20 minutes.
- The reaction was stopped by adding 100 µl of Stop Solution to each well.
- It was gently mixed for 30 seconds. It is important to make sure that all the blue color changes to yellow color completely.
- Using a microtiter plate reader, the optical density was read at 450nm within 15 minutes.

Reference Range: 4.0 nanograms per milliliter (ng/mL) and lower is normal.

Methods of Data Analysis: The data was presented in tables as mean ±, standard error and p-value analyzed using statistical packages for social sciences (SPSS, Version 20.0) and level of significance set as $p \leq 0.05$.

Ethical Clearance: Ethical approval was obtained from the ethics committee of FMC Owo, after submitting a detailed project proposal, questionnaires, informed consent forms which were in English language. Confidentiality was assured to subjects, participation in the study was voluntary and any subject not willing to continue was free to withdraw at any stage.

RESULTS

Ages were been compared between BPH and PCa patients at a significance level of $p<0.05$

In the tested serum samples, we found an increased levels in triglycerides in prostate cancered patients (3.94 ± 0.27) than in benign prostatic hyperplasia (1.08 ± 0.46) patients. This raise was statistically insignificant in patients with PCa and in those affected with BPH. Also, TC, LDL-C and HDL-C levels were non statistically different in BPH and PCa patients.

In the above Table 4, it was shown that for both BPH and PCa patients, there were increase in the levels of Total Cholesterol to High Density lipoprotein which shows statistical significance. Also, the levels of Total Cholesterol and Low Density Lipoprotein as there are increased in both levels. The levels of Triglycerides to PSA levels are also statistically significant at 0.01 level. The other parameters do not show any form of statistical significances.

This table shows that for PCa- patients, increase in Total Cholesterol levels causes an increase in Triglycerides levels which is a positive correlation between both levels. Also between Total Cholesterol and Low Density Lipoprotein, statistical significance can be seen.

This table shows that for BPH patients, increase in Total Cholesterol levels causes an increase in Triglycerides levels just as it was seen in Table-5 which is a positive correlation between both levels. Also between Total Cholesterol to Low Density Lipoprotein and High Density Lipoprotein statistically significant can be seen as increase in TC triggers, an increase in LDL-C and HDL-C. The same positive correlation can also be seen in levels of TG to HDL-C and LDL-C.

Table 1: Subject Demographic Characteristics

	BPH (n=50)	PCa (n=50)	P-Value
AGE	63.12± 1.047	81.94± 1.185	0.001*
RELIGION			
Christian	36 (72%)	36 (72%)	
Muslim	14 (28%)	14 (28%)	
OCCUPATION			
Civil Servant	27 (54%)	5 (10%)	
Business	16 (32%)	6 (12%)	
Retired	7 (14%)	39 (78%)	

Values were expressed as mean ± standard error of mean. BPH = Benign prostatic hyperplasia, PCa = Prostate Cancer. Significance level at $p<0.05$

Table 2: Comparison of Lipoprotein Levels Between BPH and PCa Subjects

	BPH (n=50)	PCa (n=50)	P-Value
TC(mmol/l)	5.09 ± 0.14	4.93 ± 0.14	0.423
TG (mmol/l)	1.08 ± 0.46	3.94 ± 0.27	0.001*
LDL-C (mmol/l)	3.69 ± 0.12	3.67 ± 0.13	0.914
HDL-C (mmol/l)	0.83 ± 0.04	0.73 ± 0.03	0.069

BPH = Benign prostatic hyperplasia, PCa = Prostate Cancer. n=number of subjects.TC=Total cholesterol, TG=Triglyceride, HDL-C= High density lipoprotein cholesterol, LDL-C=Low density lipoprotein cholesterol, PSA=Prostate specific antigen. Significance level at $p<0.05$

Table 3: Level of Prostate Specific Antigen (Psa)

Parameter	BPH (n=50)	PCa (n=50)	P-Value
PSA (mg/ml)	18.12 ± 3.88	46.01 ± 5.76	0.001*

BPH = Benign prostatic hyperplasia, PCa= Prostate Cancer. n=number of subjects Mean+SE. Significance level at $p<0.05$

Table 4: Pearson's Correlation Coefficients of Biochemical Parameters Within BPH and PCa Subjects

Parameter	TC (mmol/l)	TG (mmol/l)	HDL-C (mmol/l)	LDL-C (mmol/l)	PSA (ng/ml)
TC(mmol/l)	1	0.194	0.282**	0.905**	0.139
TG (mmol/l)		1	-0.039	0.218*	0.357**
HDL-C (mmol/l)			1	0.051	-0.048
LDL-C (mmol/l)				1	0.108
PSA (ng/ml)					1

TC=Total cholesterol, TG=Triglyceride, HDL-C= High density lipoprotein cholesterol, LDL-C=Low density lipoprotein cholesterol, PSA=Prostate specific antigen

**Correlation is significant at the 0.01 level (2-tailed).

*Correlation is significant at the 0.05 level (2-tailed).

Table 5: Pearson's Correlation Coefficients of Biochemical Parameters Within the PCa Subjects

Parameter	TC (mmol/l)	TG (mmol/l)	HDL-C (mmol/l)	LDL-C (mmol/l)	PSA (ng/ml)
TC(mmol/l)	1	0.393**	0.131	0.965**	0.324*
TG (mmol/l)		1	0.142	0.360*	0.163
HDL-C (mmol/l)			1	-0.023	0.094
LDL-C (mmol/l)				1	0.242
PSA (ng/ml)					1

TC=Total cholesterol, TG=Triglyceride, HDL-C= High density lipoprotein cholesterol, LDL-C=Low density lipoprotein cholesterol, PSA=Prostate specific antigen

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Table 6: Pearson's Correlation Coefficients of Biochemical Parameters Within the BPH Subjects

Parameter	TC (mmol/l)	TG (mmol/l)	HDL-C (mmol/l)	LDL-C (mmol/l)	PSA (ng/ml)
TC(mmol/l)	1	0.787**	0.387**	0.848**	-0.015
TG (mmol/l)		1	0.381**	0.613**	0.005
HDL-C (mmol/l)			1	0.108	-0.058
LDL-C (mmol/l)				1	-0.061
PSA (ng/ml)					1

TC=Total cholesterol, TG=Triglyceride, HDL-C= High density lipoprotein cholesterol, LDL-C=Low density lipoprotein cholesterol, PSA=Prostate specific antigen

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

DISCUSSION

As reported in Table 2, the mean values of Triglycerides (3.94 ± 0.27mmol/l) in Prostrate Cancer patients was observed to be significantly higher than the mean values of Triglycerides (1.08 ± 0.46mmol/l) in Benign Prostatic Hyperplasia patients. While the mean values of High Density Lipoproteins (0.73 ± 0.03mmol/l) and Low Density Lipoproteins Levels (3.67 ± 0.13mmol/l) were lower in Prostrate Cancer patients than the mean values of High Density Lipoproteins (0.83 ± 0.04mmol/l) and Low Density Lipoproteins Levels (3.69 ± 0.12mmol/l)in Benign Prostatic Hyperplasia patients which may be due to age, ethnicity or western diets.

This study (Table 5) shows that in Prostrate Cancer patient, there was a positive relationship between levels of Total Cholesterol and Triglycerides as they are statistically significant. This implies that as the levels of Total Cholesterol keeps increasing Triglycerides level also increases in such patients. Also, it was observed that there was a significant relationship between the levels of Low Density Lipoproteins and Total Cholesterol which may be due to risk of total aggressiveness of the cancer in which increased Total Cholesterol can be assumed to be a major risk of Prostate cancer [18]. Also, modern Western diets which contains substantial levels of cholesterol and other fatty substances greatly promotes prostate cancer progression which may be suggested according to this present study to bring about the association between lipids and prostate cancer [19].

In a prospective, population-based study carried out by Krycer and Brown [15] in which the association between blood lipid profiles and prostate cancer risk was evaluated. It revealed that high levels of both Total cholesterol and LDL cholesterol were associated with an increased risk of total and aggressive prostate cancer. In contrast, high levels of HDL cholesterol were associated with an increased risk of non-aggressive prostate cancer [20]. Hence, prostate cancer risk has been suggested to be related to the high levels of circulating lipoproteins (cholesterol) concentrations. In this present study, there are no evidences supporting the aggressiveness or non- aggressiveness of prostate cancer as regards levels of HDL cholesterol but there are positive correlation from the results obtained that connotes that Total cholesterol is assumed to be a risk for Prostate cancer.

Epidemiological evidences; Jemal *et al.* [9] suggests that the modern Western diets which contains substantial levels of cholesterol and other fatty substances greatly promotes prostate cancer progression. Hence, it has previously been suggested that associations between blood lipid profiles and prostate cancer risk depend on the aggressiveness of the disease.

In this present study, there are no significant correlations between HDL cholesterol in Prostrate cancer patients. However, Liu *et al.* [16] shows that detailed analyses with quartiles of HDL cholesterol did show statistically significant associations with high-grade prostate cancer risk. Although, a definite conclusion has not been reached for the non-significant association between HDL and prostate cancer risk, based on the currently available evidences. Therefore, more relevant studies are needed to assess the potential effect of HDL on prostate cancer risk.

In the present study (Table-6), in Benign Prostatic Hyperplasia patients, it was clearly shown that there was a significant positive correlation between levels of Total Cholesterol and Triglycerides, also between Low Density Lipoproteins and Total Cholesterol and Triglycerides. This implies that as levels of Total Cholesterol are increasing, the Triglycerides levels also increases alongside the Low density Lipoprotein levels. This can be due to Hypercholesteremia (increase in cholesterol levels) in the body of the patient which leads to cardiovascular disease in men which is also a risk factor for BPH [17].

Mitropoulos and Ploumidou [17] study showed that Hypercholesterolemia, (a major risk factor for Cardiovascular Disease) is also a risk factor for BPH. Abnormal concentrations of lipids and lipoproteins are well-described risk factors for cardiovascular disease which include elevated serum low density lipoprotein (LDL) cholesterol (usually defined as ≥= 130–140 mg/dL), decreased serum high density lipoprotein (HDL) cholesterol (< 40 mg/dL) and increased serum triglycerides (≥ 150 mg/dL). These factors are components of the metabolic syndrome and frequently occur in association with other cardiovascular risk factors, including diabetes. This observation raises the possibility that abnormal lipids and lipoproteins may also be connected to BPH pathogenesis [17].

This research was limited to only Owo locality hence more researches are needed to give us a clearer and much more precise view of the effects of lipoproteins in the existence of benign prostatic hyperplasia and prostate cancer patients.

CONCLUSION

From this study, it can be concluded that there was a significant increase in the levels of Total Cholesterol and Low Density Lipoprotein-Cholesterol in both Prostate

cancer Patients and Benign Prostatic Hyperplasia patients which constitutes a risk factor for both prostate diseases and may be helpful in differential diagnosis. It is recommended that Prostate cancer and Benign Prostatic Hyperplasia patients should endeavor to visit clinicians and undergo lipid profiles test as often as possible so as to regulate their lipids levels in order to reduce risks and complications of this prostate diseases.

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