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Red Blood Cell Lipids, Serum Trace Elements and Immunological Markers in Prostatic Disease Patients: An Investigative Study

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ABSTRACT

Aim: In this study, the red cell lipids, serum trace elements and immunological markers in prostatic disease patients attending the Nephrology Department of Abia state University Teaching Hospital Aba were evaluated.

METHODOLOGY: A total of one hundred and ten (110) adult males (aged 40-80years) comprising of (60) prostatic disease patients and fifty (50) normal subjects were recruited. The prostatic disease patients comprised of thirty (30) prostatitis, twenty (20) benign prostatic hyperplasia (BPH) and ten (10) prostate cancer patients. Red cell lipids, trace elements and immunological markers were determined on blood samples collected from the subjects between January 2017 and December 2020.

RESULTS: Results obtained following analysis indicated a significant increase in red cell total cholesterol, red cell LDL-cholesterol, red cell Triglyceride and red cell phospholipids ($P<0.05$) compared to control subjects whereas red cell HDL-cholesterol were significantly decreased compared to control subjects ($P<0.05$) serum trace elements: copper were significantly decreased in prostatitis and BPH compared to control subjects ($P<0.05$) but no significant decrease in prostate cancer patients compared to control subjects ($P>0.05$). Serum iron and lead showed no significant difference in prostatic patients compared to control subjects. Serum manganese levels showed significant increase in BPH patients compared to control subjects ($P<0.05$) whereas prostatitis and prostate cancer patients showed no significant decrease ($P>0.05$) compared to control subjects. Globulin concentrations of prostatatic patients did not show significant increase in prostatic disease patients compared to control subjects ($P<0.05$).

CONCLUSION: Lipids serves as a promoter of peroxidation, oxidative stress and oxidative damage which has been implicated in many diseases and immunological markers is an evidence of inflammation which is evidence and risk factors in development of prostatic diseases.

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Keyword= BPH-Benign Prostatic Hyperplasia, HDLC-High Density Lipoprotein Cholesterol LDLC-Low Density Lipoprotein Cholesterol, TAG- Triglyceride, Total Chol- Total Cholesterol

1. INTRODUCTION

Globally there is upsurge in men suffering from prostatic diseases and has continued to be a major source of mobidity and mortality among men (1). Benign prostatic hyperplasia and prostate cancer are the most common forms of prostatic diseases (2). BPH accounts for up to 48% of all cases. Prostate cancer is the leading cancer seen in older men (3). Clinical signs associated with each of these diseases also appear to be overlapped and deaths due to prostatic diseases have been linked to complications such as urinary retention, urinary tract infections, bladder stones, bladder damage and kidney failure

(4). Studies have also shown that chronic inflammation, life style/diets and family history are all risk factors in the development of prostatic diseases (5). Pathogenesis of prostatic diseases remain hazy, oxidative stress from endogenous and exogenous sources have been fingered as possible pathway for their development (6) Aberrant lipid metabolism has been shown to be the hallmark of malignant phenotype and increased lipid accumulation secondary to changes in the levels of variety of lipid metabolic enzymes in prostate diseases (7). Trace elements (Mn, Selenuim and Cu) are integral part of endogenous antioxidant enzymes that are involved in defense line of the body, changes in trace element in prostatic disease, further exposes the relationship between oxidative stress which results when the antioxidant capacity is overwhelmed and trace elements which are components of these antioxidant enzymes, therefore serum trace elements may be of value in the diagnosis of prostatic diseases. Immunologic parameters (globulin, alpha macro globulin and antitrypsin) human serum proteins (globulins) play significant roles in inflammation (8) which may regulate benign and malignancy in prostatic tissues. Hence the levels of globulin in prostatic patient maybe a yardstick for accessing the severity of the disorder and may therefore be a parameter to be considered in the diagnosis of prostatic diseases. In this study, red cell lipids, trace elements and serum globulins alpha macro globulins as well as antitrypsin of prostatic disease patients who attended the Nephrology Department of the Abia state University Teaching Hospital were accessed and presented to add to the body of currently available data needed for prostatic disease diagnosis.

2. MATERIALS AND METHODS

2.1. Area of study/ Recruitment of subjects

The study was conducted in Aba, Abia State. A total of one hundred and ten (110) samples were collected from 2017- 2020. Sixty (60) were patients certified to be prostatic patients by clinicians in the Nephrology department of Abia State University Teaching Hospital Aba whose prostate specific antigen (PSA) were above four nano grams per ml (4ng/ml) Fifty (50) were control subjects.

2.2. Collection of Blood Samples

Ten milliliters of venous blood was collected from each of the recruited subjects and dispensed into heparinized bottles for red cell lipids. Another portion of blood was dispensed into plain bottles, allowed to clot and then centrifuged to collect serum which was used for trace elements and serum globulins, α-macro-globulin and antitrypsin analysis.

2.3. Determination of red cell lipids concentration

2.3.1. Extraction of lipids from red cell

Extraction of lipids from the red cell was done using (Rose and Oklander 1965 method). One hundred microliter of washed red cell was pipetted into test tubes followed by addition of nine microliter chloroform-isopropanol mixture (7:11 v/v) vortexed and left to stand for one hour for complete extraction. The suspension was centrifuged at 4000 rpm for 10 minutes and supernatant containing the lipid was removed using pasteur's pipette and was used for the analysis. The rbc-cholesterol, the lipid extract, one hundred microliter of chloroform was pipetted into tubes labelled sample, standard and blank. They were evaporated to dryness over boiling water. The dried extracts were redissolved in 20µl of triton X / chloroform (1:1) mixture and evaporated again and were used in determination of cholesterol using the method of Allain et al; 1974.

Procedure

Determination of RBC cholesterol

Lipid extract one hundred microliter (100µl) of RBC, one hundred microliter (100µl) of standard cholesterol and one hundred microliter (100µl) of chloroform were pipetted into different test-tubes labeled sample, standard and blank. They were evaporated to dryness over boiling water. The dried extracts were redissolved in 20µl of triton x/chloroform (1:1) mixture and evaporated again and were used in determination of cholesterol

Principle

Cholesterol concentration was determined spectrophotometrically. The method involved the use of three enzymes; cholesterol esterase, cholesterol oxidase and peroxidase. In the presence of the enzymes (hydrolysis and oxidation) the following product were formed hydrogen peroxide (H_2O_2) and cholestrene – 3- one and in the presence of phenol and 4-amino antipyrine a quinonenimine dye was formed which was proportional to the concentration of cholesterol present in the sample.

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{esterase}} \text{cholesterol} + \text{fatty acid}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{cholesterol Oxidase}} \text{cholestene -3- one} + H_2O_2$$

$$2H_2O_2 + \text{phenol} + \text{4-aminoantipyrine} \xrightarrow{\text{peroxidase}} 4H_2O + \text{quinonimine}$$

Test procedure.

Three (3) test-tubes marked blank (B), test (T) and standard (S) was arranged in a test tube rack. To each test tube was added one milliliter (1ml) of the reagent (piper buffer 80 mmo/L pH 6.8, 4-aminoantopyrine 0.25mmol/L, phenol 6mmoL/L, peroxidase ≥ 0.5u/mL)

Cholesterol calculation:

$$\frac{\triangle \text{A sample}}{\triangle \text{A standard}} \times \frac{5.12\text{mmo/L}}{1}$$

Determination of RBC Triglyceride

RBC lipid extract one hundred milliliter (100ml), one hundred milliliter (100ml) of standard triglyceride extract and one hundred milliliter (100ml) of chloroform were pipette into different tubes and labeled sample, standard and blank. These were evaporated to dryness and then redissolved in 20µl triton X/chloroform (1:1) and in triglyceride determination using Buccolo and David (1973).

Principle:

These methods were based on the enzymatic hydrolysis of triglyceride to glycerol and free fatty acids by lipoprotein lipase. The glycerol was phosphorylated by adenosine triphosphate in the presence of glycerokinase to form glycerol – 3- phosphate and adenosine diphosphate. Glycerol -3-phosphate was oxidized by glycerol phosphate oxidase to dihydroxyacetone phosphate and hydrogen peroxide. A red chromogen is produced by the peroxidase catalysed coupling of 4-aminoantipyrine and phenol with hydrogen peroxide. The colour intensity is proportional to the concentration of triacylglycerol in the sample.

Triglyceride + H_2O $\xrightarrow{\text{lipases}}$ glycerol + fatty acids

Glycerol + ATP $\xrightarrow{\text{GK}}$ glycerol – 3- phosphate + ADP

Glycerol – 3- phosphate + O_2 $\xrightarrow{\text{GPO}}$ dihydroxyacetone – phosphate + H_2O_2

$2H_2O_2$ + 4-amino phenazone + 4 chlorophenol $\xrightarrow{\text{POD}}$ quinonamine + HCL +$4H_2O$

Procedure

Three (3) test-tubes marked blank (B), test (T) and standard (S) was arranged in test tube rack. To each of the test tube was added one milliliter (1ml of the reagent (Buffer – Pipes 40mmol/L PH 7.6 buffer, 4-chloro 5.5mmol/L – phenol, magnesium 17.5 mmol/L – 10ns, 4 – amino phenazone 0.5mmol/L, ATP 1.0mmol/L lipase ≥ 150µ/mL, glycerol – kinase ≥ 0.4µ/mL, glycerol – 3 – phosphate oxidase ≥ 1.5µ/mL peroxidase ≥ 0.5µ/mL).

The ten microliter (10µl) of the extract was in the test-tube marked test, ten microliter (10µl) of standard triglyceride with equivalent of 2.25mmol/L put in the tube marked standard. The mixture was thoroughly mixed and incubated for 15 minutes at 25°c.

The absorbance of the sample and standard was read at 505nm against the reagent blank.

Triacyglycerol concentration (mmol/L) in the extract was calculated as;

$$\text{Mmol/L triacylglycerol} = \frac{\text{absorbance of sample}}{\text{Absorbance of standard 1}} \times \frac{2.25}{}$$

Determination of RBC HDL

(Folch *et al*; 1956)

HDL extract one hundred microliter (100µl), standard one hundred microliters (100µl) and one hundred microliter (100µl) chloroform were put into test-tubes labeled test, standard, and blank respectively. They were evaporated to dryness over boiling water. The dried extracts were re-dissolved by addition of 20µl of triton x/chloroform mixture (1:1) and again evaporated and used to determine HDL (folch *et al*; 1956) method.

Princple: The apoB containing lipoproteins in the specimen are reacted with a blocking reagent that renders them non- reactive with the enzymatic cholesterol reagent under conditions of the assay. The apoBcontaining lipoproteins are thus effectively excluded from the assay and only HDL- cholesterol is detected under the assay conditions.

Test procedure

One hundred microliter (100µl) of supernatant of samples and standard was pipetted into their corresponding tubes, while one hundred microliter (100µl) of distilled water was pipette into the reagent blanked test tube. After, one milliliter (1ml) of the HDL reagent was added into all the tubes, mixed and incubated for 5 minutes at 37^{o}c.

Absorbance was read at 546nm and HDL concentration calculated.

$$\text{HDL concentration in mg/dl: } \frac{\text{Absorbance of sample x Concentration of standard (mg/dl)}}{\text{Absorbance of standard}}$$

Determination of LDL (low density lipoprotein) was by calculation by modification of Friedwald's formular (Sandkapm *et al*; 1990)

$$\text{LDL- cholesterol mgldl} = \text{total cholesterol} - \left(\text{HDL - Cholesterol} + \frac{\text{Triglycerides}}{5}\right)$$

Determination of RBC phospholipids (Stewart, 1997)

Principle:

The method was based on complex formation between ammonium ferrothiocyanate and phospholipids. It allowed for phospholipid measurement in the range of 0.01 -10mg (15– 150nmol).

Procedure

One hundred microliter (100µl) of RBC chloroform extract was evaporated to dryness over hot water bath. The dried extract was dissolved in 20µl of chloroform and then 2ml ammonium ferrothiocynate was added and vortexed.

The chloroform layer was removed after 10 minutes of allowing the mixture to separate. Absorbance was read at 488nm against chloroform blank mixture of 2ml chloroform and 2ml ammonium ferrothiocynate.

$$\text{Calculation} = \frac{\text{OD of test}}{\text{OD of standard}} \times \frac{\text{conc. Of standard}}{1}$$

2.4. Determination of trace element

Trace elements were measured using atomic absorptiometric sphectrophotometer (AAS) model 210VGP, Buck Scientific inconporated USA.

PRINCIPLE

Principle of the operation of the equipment: The machine operates at a wavelength of 190- 900nm using the principles of absorption spectrophotometry which accesses the concentration of analyte in a sample. A light source (hollow cathode lamp) operating in the ultra- violet- visible spectral region was used to cause electronic excitation of the metals assayed. The absorbance was measured with a uv-visible dispersive spectrometer with photomultiplier detector. The concentration of each sample in parts per million was then extrapolated from the calibration curve.

PROCEDURE:

One mililiter (1) of serum sample was aspirated into acetylene flame after the equipment was zeroed with de-ionized water as the blank. This caused evaporation of the solution and atomization of the free trace element evaluated respectively (iron, copper, lead, zinc, manganese, selenium).

2.5. Determination of serum proteins (globulins, α-macro-globulin and antitrypsin)

Alpha macroglobulin by ELISA in serum (Helen M Atkinson et al; 2012)

Principle:

The alpha macroglobulin in the sample is bound to polyclonal rabbit antibodies (in excess) which are imoblised to the microtiter wells. To remove all unbound substances, washing steps is carried out. In a second incubation step, a peroxidase- labeled anti α- macroglobulin antibody (POD-antibody) is added after another washing step to remove all unbound substance the solid phase is incubated with the substrate, tetramethylbenzidine (TMB). An acidic stop solution is then added to stop the reaction. The color converts from blue to yellow. The intensity of the yellow color is directly proportional to the concentration of α- macroglobulin in the sample. A dose response curve of the absorbance unit verses concentration is generated using the result obtained from the calibrators. Alpha macroglobulin present in the patient sample is determined directly from the curve.

Reagents: Microtiter plate, pre- coated, wash buffer concentrate, conjugate concentrate, rabbit anti – α macroglobulin (peroxidase labeled) standards.

PROCEDURE:
The wells were washed five times with two hundred and fifty microliter (250µl) wash buffer residual wash buffer was removed by tapping the plate on absorbent paper.

One hundred microliter (100µl) each of the standard /control /sample were put into the respective wells.

It was incubated for one hour at room temperature (15-30) degrees centigrade. After incubation each of the content was washed for five times with two hundred and fifty microliter (250µl) wash buffer. After washing, the buffer was removed by tapping the plate on the absorbent paper. One hundred microliter (100µl) conjugate was added into each well. It was covered and incubated for one hour at room temperature. The content of each well was discarded and washed five times with two hundred and fifty microliter (250µl) wash buffer. One hundred microliter substrate was added into each tube and it was incubated for 20 minutes at room temperature in the dark. One hundred microliter stop solution was added to each well and mixed well. Absorption was read immediately with the ELISA reader at 450nm. Plasma concentration was gotten by extrapolation from standard curve from the calibrators used, Plasma = g/l.

Determination of Antitrypsin by ELISA Assay (by Schoo et al; 1991)
Principle: The alpha 1 antitrypsin Elisa assay determines human alpha-1- antitrypsin according to sandwich principle. Alpha- 1 antitrypsin in sample, standard and controls binds to antibodies which are coated to the microtiter plate. After washing step, a peroxidase labeled detection antibody is added. A second washing step is followed by the addition of the substrate which is converted to a product by peroxidase. The reaction is terminated by the addition of an acid stop solution. The optical densities are read at 450nm (against the reference wavelength 620) in a microtiter plate reader. The alpha – 1 antitrypsin concentration can be calculated from the standard curve.

Reagents: microtiter plate coated, Elisa wash buffer solution, standard (0, 3.3, 10, 30 ,90ng/ml), control 1and 2 (1ml), conjugate perosidase labeled antibody, TMB substrate (tetramethylbenzidine), stop solution.Sample collection: 3.0mls of venous blood was collected in an EDTA container centrifuged at 3000rpm for 10 minutes at 2-8^{o}c within 60 minutes of collection of venous blood.

PROCEDURE: All reagents and samples were stablised at room temperature before use. The microtiter plates were washed with two hundred and fifty microliter (250µl) wash buffer and tapped and dried with absorbent paper after the washing step. One hundred microliter (100µl) of sample | standard | control was put in microtiter plate respectively in duplicates. The stripes were covered and incubated after shaking for 60 minutes at room temperature. After incubation, the content was discarded and washed for 5 times with two hundred and fifty microliter (250µl) diluted wash solution. It was incubated after one hundred microliter (100µl) conjugate was added to each micro well and covered for 60minutes at room temperature. It was washed again for five times with two hundred and fifty microliter (250µl) diluted wash buffer and after it was discarded completely by tapping the plate on absorbent paper after the last washing step. One hundred microliter (100µl) substrate was added to each micro well. It was incubated for 10- 15minutes at room temperature. Fifty microliter (50µl) of stop solution was added to each and mixed well. The absorbance was read at 450nm in a micro plate reader.

Calculation: α- antitrypsin concentration in ng /ml was gotten from standard curve generated from the calibrators.

Estimation of globulin was done by determination of total protein minus albumin.

Determination of total protein
(Folin-Ciocalteau methoid by Lowry et al; 1951)

Principle: Cupric ions in an alkaline solution react with the peptide bonds of proteins and polypeptides containing at least two peptide bonds to produce a violet colored complex. The absorbance of the complex at 546nm is directly proportional to the concentration of the protein in the sample.

C^{++} + protein __oH^{-}____ Cu-protein complex

Reagents: potassium iodide – 30mmol/l, sodium tartrate – 100mmol/l, copper sulphate – 30mmol/l, sodium hydroxide – 3.8mmol/l, total protein standard.

Procedure:

One thousand microliter (1000µl) of the reagent was put into tubes labeled sample |standard| blank respectively. Ten microliter (10µl) of serum sample was put in the tube labeled sample and ten microliter (10µl) of standard was also put in the tube labeled standard, ten microliter (10µl) of distilled water was put into tube labeled blank. The tubes were mixed and allowed to incubate for 10 minutes at room temperature and the absorbance of the test| standard| blank were read in a colorimeter at 546nm.

Calculation: $\frac{A_{sample}}{A_{Standard}} \times \frac{\text{concentration of standard}}{1}$ = concentration of test sample in g/l

Determination of Albumin by colormetric bromocresol green method (Doumas 1971)

Principle: At pH 4.2, albumin binds with bromocresol green to produce a blue – green complex. The change in absorbance at 628nm correlates with the concentration of albumin.

Reagents: bromocresol green 0.15 mmo|/l, succinate buffer pH4.2, 75mmol/l, Brij 35, standard 7mmol/l.

Procedure:

0ne milliliter (1ml) of the reagent was pippetted into tubes labeled blank| standard |serum sample respectively. Ten microliter (10µl) of distilled water |standard| serum sample was also pippetted into respective labelled tubes. The tubes were mixed and incubated for 30 minutes at room temperature and read in a colorimeter at 650nm wave length.

Calculation: $\frac{A_{sample}}{A_{standard}} \times \frac{\text{concentration of standard}}{1}$ = concentration of albumin g/l.

2.6. Statistical analysis

All values were expressed as a mean SD (n= 30 Group1, n= 20 Group2, n=10 and Group3, n= 50 Group4.

Data were analyzed using one-way ANOVA followed by the post –hoc- Ducan multiple range test for analysis of biochemical data using SPSS version11. And also student – test values were statistically significant at ($P < 0.05$)

3. RESULTS

Table 1: Lipid Profile of patients with prostatic conditions

Study Groups	Total Chol. Conc.(mg/dl)	HDL Conc. (mg/dl)	LDL Conc. (mg/dl)	TAG Conc. (mg/dl)	Phospholipid Conc.(mg/dl)
Prostatitis	96.01±19.31^{b}	28.88±7.47^{b}	55.51±15.34^{a}	50.48±10.75^{b}	326.77±91.60^{b}
BPH	97.92±18.54^{b}	27.31±7.42^{b}	54.93±18.37^{b}	53.53±11.74^{b}	357.63±99.68^{bc}
Prostatic cancer	113.80±11.45^{c}	22.96±5.29^{a}	54.92±21.17^{b}	62.17±18.93^{c}	390.74±107.49^{c}
Control subjects	62.92±21.38^{a}	38.71±3.36^{c}	33.76±10.26^{c}	42.22±6.96^{a}	265.09±53.24^{a}

3.1. Effects of prostatic disease on rbc-lipids out of a total of 60 prostatic diseases patients(40-80years) who were diagnosed at the Nephrology Department of Abia State University Teaching Hospital Aba. 30 prostatitis (Group 1), 20 had benign prostate hyperplasia (Group2) while 10 had prostate cancer (Group 3). All categories of prostatic patients studied had significantly increased rbc-cholesterol, rbc-LDC-C, Triglycerides and phospholipids ($P<0.05$) compared to control subjects whereas rbc -HDL-C was significantly reduced compared to control subjects (Table 1)

Table 2: Trace Elements

Study Groups	Copper (ppm)	Iron (ppm)	Lead (ppm)	Manganese (ppm)	Selenium (ppm)	Zinc (ppm)
Prostatitis	0.573±0.43^{ab}	0.983±0.83^{a}	0.245±0.35^{a}	0.760±0.88^{ab}	0.295±0.36^{a}	0.744±0.50^{a}
BPH	0.505±0.31^{a}	1.116±0.53^{a}	0.412±0.54^{a}	1.088±0.97^{c}	0.420±0.53^{a}	0.688±0.42^{a}
Prostatic cancer	0.753±0.50^{bc}	1.272±0.48^{a}	0.439±0.62^{a}	0.611±0.39^{a}	0.328±0.55^{a}	0.757±0.40^{a}
Control subjects	0.902±0.27^{c}	1.230±0.39^{a}	0.232±0.37^{a}	0.920±0.40^{ab}	0.903±0.34^{b}	1.113±0.32^{b}

Values are presented as mean ± standard deviation and values with different superscripts are significantly ($P<0.05$) different from any paired mean within each column.

3.2. Effects of prostatic diseases on trace elements

Serum selenium and zinc of prostatic disease patients were significantly reduced compared to control subjects ($p< 0.05$)while serum copper of prostatitis and benign prostatic hyperplasia were significantly decreased

compared to control subjects ($p<0.05$) but serum copper of prostate cancer patients was not significantly decreased compared to control ($p>0.05$). The serum manganese of prostatitis and prostate cancer patients were not significantly decreased compared to control subjects ($p>0.05$) whereas benign prostatic patients had their serum manganese significantly increased compared to control subjects ($p<0.05$) (Table 2)

Table 3: Immunological paramters

Study Groups	Globulin (mg/dl)	Alpha macro globulin (mg/dl)	Antitrypsin (mg/dl)
Prostatitis	263.79±22.47[a]	301.98±15.78[b]	120.31±7.11[b]
BPH	249.26±21.22[a]	300.39±20.55[b]	118.75±10.04[b]
Prostatic cancer	260.39±21.74[a]	298.71±19.25[b]	123.13±9.30[b]
Control subjects	246.01±19.90[a]	195.59±21.06[a]	93.87±8.41[a]

Values are presented as mean ± standard deviation and values with different superscripts are significantly ($P<0.05$) different from any paired mean within each column.

3.3. Effects of prostatic diseases on serum protein values. The serum alpha macroglobulin and antitrypsin levels of all prostatic patients were significantly increased compared to control subjects ($p<0.05$) whereas the serum globulin of prostatic disease patients were not significantly increased ($p>0.05$) compared to control subjects (Table 3).

4. Discussion

Physiological and biochemical changes do accompany the onset of prostatic diseases such as prostatitis, benign prostatic hyperplasia and prostate cancer is well supported by this study. Increase in the lipids except HDL-C shows that lipid metabolism is altered. There is accumulation and also changes in the levels of lipid's metabolic enzymes. Alterations in prostate lipid metabolism including up- regulation of several lipogenic enzymes as well as enzymes that function to oxidized fatty acids as an energy source do occur (8). Furthermore phospholipids and metabolites which are altered may lead to up-regulation of various functions through receptor systems (9) and oxidative stress which comes from peripheral blood depends on many factors especially changes in conformation of hemoglobin and it's affinity to oxygen (10). This may worsen the disease, the low levels of HDL-C observed shows that HDL-C signaling will be altered and this agreed strongly with the findings of Theodore et al; 2013. The changes in trace elements observed shows important role played by these elements will be altered, such as enzyme's reactions where they attract and facilitate conversion of substrate molecules to specific end products. Some trace elements play structural roles and are responsible for stability of important biological molecules (12). Prostatic disease patients may suffer deficiency symptoms due to these altered trace elements. Increased levels of serum proteins observed corroborated with Victor et al; 2005 (8) and this shows that serum proteins in this report may play a role in regulating benign and malignancy in the prostatic tissue.

5. Conclusion:

We therefore, conclude that dyslipidemia, altered trace element and inflammation may all be factors that may lead to prostatic disease and these parameters may be of value in the diagnosis of prostatic conditions in men.

CONSENT AND ETHICAL APPROVAL

Informed consent was obtained from all subjects recruited for the study while ethical approval for the study was obtained from ethical committee of the Abia State University Teaching hospital Aba Abia State with Ref number ABSUTH/MAC/1117/Vol/1/10

COMPETING INTERESTS

Authors have declared that no competing interest exist.

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