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Research Article:

Studies On Some Oxidative Stress Markers and Antioxidant Status in Subjects with Benign Prostate Hyperplasia on Therapy in Imo State University Teaching Hospital, Orlu

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ABSTRACT:
Benign prostatic hyperplasia (BPH) is a highly prevalent age-related prostatic condition with lower urinary tract symptoms. There is increasing evidence that oxidative stress and defective antioxidant defence may play a role in the pathogenesis of BPH. Information on oxidative stress and antioxidant status in Nigerian men with BPH, particularly during medication, is however scarce. This study examined certain oxidative stress markers and antioxidant levels of men with BPH on therapy at Imo State University Teaching Hospital, Orlu, Nigeria. Material and methods: A prospective comparative study was done among 150 males in the age group of 40-65 years with 75 therapy naïve BPH patients and 75 apparently healthy controls. The patients with BPH were followed-up at 3 and 6 months of therapy. Serum malondialdehyde (MDA), reduced glutathione (GSH), glutathione peroxidase (GPx), superoxide dismutase (SOD) and vitamin C were evaluated by standard spectrophotometric and ELISA based procedures. Data were examined using SPSS version 23.0. Comparisons between groups were carried out using Student's t-test and Pearson's correlation was utilised to analyse the correlations between biochemical markers. Statistical significance was defined as $p<0.05$. Therapy-naïve BPH patients had significantly higher serum MDA than controls (1.93±0.14 vs 1.35±0.03 mmol/L, p=0.001) and significantly lower GSH (3.28±0.41 vs 4.88±0.31 mmol/ml), GPx (199.59±31.89 vs 317.86±58.71), SOD (135.35±14.51 vs 174.03±14.75) and vitamin C (0.74±0.15 vs 1.41±0.31 µg/ml) (p=0.001 for all). After three months of therapy, MDA fell dramatically whereas GSH, GPx, SOD and vitamin C increased significantly (p=0.001). After 6 months, MDA further reduced to 1.47±0.11 mmol/L while GSH, GPx, SOD and vitamin C increased to 4.50±0.34 mmol/ml, 313.30±29.54, 167.96±26.12 and 1.50±0.23 µg/ml, respectively. Increased lipid peroxidation and decreased antioxidant defence have been demonstrated in therapy-naïve BPH patients. The gradual decrease in MDA and the increase in antioxidant markers during the therapy indicate that the oxidative-antioxidant balance improves over six months. Biomarkers of oxidative stress may provide important supplementary information in understanding the biochemical alterations associated with BPH and its therapy.

Keyword: Benign Prostatic Hyperplasia; Oxidative Stress; Malondialdehyde; Glutathione; Glutathione Peroxidase; Superoxide Dismutase; Vitamin C; Antioxidant Status.

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INTRODUCTION

Benign prostatic hyperplasia (BPH) is a very prevalent urological condition in ageing men and an important cause of lower urinary tract symptoms (LUTS). It is characterised by non-malignant proliferation of stromal and epithelial cells within the prostate, especially in the transition zone leading to progressive enlargement of the gland and varying degrees of urethral obstruction [1]. Clinically, BPH can manifest with urinary frequency, urgency, nocturia, weak urinary stream, hesitancy, intermittency and incomplete bladder emptying with symptoms that may significantly affect quality of life. Histological BPH is increasingly common with age but the severity of symptoms varies greatly between individuals. The prevalence of clinically significant BPH

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rises sharply from middle age and may involve a significant proportion of men by the seventh and eighth decades of life. Therefore, the ageing population has made BPH an important issue in both public health and clinical management [2].

The prostate is an androgen-dependent male reproductive gland located below the urinary bladder and surrounding the proximal urethra. Its secretions are a major component of seminal fluid and help in sperm viability and motility. Changes in androgen signalling, cellular growth, stromal-epithelial interactions and local inflammatory processes with age may all contribute to prostatic enlargement. Crucially, BPH and prostate cancer are separate pathological entities. Both conditions occur predominantly in older men, are influenced by hormonal and inflammatory pathways and may present with similar LUTS. BPH is not considered a direct precursor of prostate cancer. Consequently, accurate clinical and laboratory assessment is essential for distinguishing benign prostatic enlargement from malignant disease [3].

Increasing evidence suggests that oxidative stress may contribute to the development and progression of BPH. Oxidative stress occurs when the generation of reactive oxygen species (ROS) and other reactive intermediates exceeds the capacity of biological antioxidant systems to neutralize them effectively. ROS, including superoxide anion, hydrogen peroxide and hydroxyl radicals, are generated as normal products of cellular metabolism, particularly through mitochondrial oxidative phosphorylation. Under physiological conditions, their production is tightly regulated by enzymatic and non-enzymatic antioxidant mechanisms. However, excessive ROS generation or inadequate antioxidant protection can result in oxidative modification of lipids, proteins and nucleic acids, leading to cellular dysfunction and tissue injury [4].

The prostate may be particularly susceptible to oxidative injury because ageing is associated with progressive changes in mitochondrial function, antioxidant capacity and inflammatory activity. Chronic inflammation within prostatic tissue can further enhance ROS generation, particularly through the activation of inflammatory cells such as neutrophils and macrophages. ROS generated during inflammatory responses may promote cellular signalling pathways involved in proliferation, apoptosis, tissue remodelling and fibrosis. Experimental evidence has demonstrated that exposure of prostatic cells to relatively low concentrations of hydrogen peroxide can stimulate cellular proliferation, whereas antioxidant supplementation may attenuate this response. These observations support the possibility that oxidative stress is not merely a consequence of prostatic enlargement but may participate in the biological mechanisms underlying BPH[5].

Oxidative stress is commonly evaluated through the measurement of products of oxidative damage and the body's antioxidant capacity. Malondialdehyde (MDA), a product of lipid peroxidation, is one of the frequently investigated biomarkers of oxidative stress [6]. Polyunsaturated fatty acids in cellular membranes are particularly vulnerable to attack by ROS, resulting in lipid peroxidation and formation of secondary products, including MDA. Increased MDA concentrations therefore provide an indirect indication of enhanced oxidative damage to cellular membranes. Other oxidative stress-related markers may similarly provide information about the balance between oxidant generation and antioxidant protection [7].

The antioxidant system consists of enzymatic and non-enzymatic components that act together to maintain redox homeostasis. Major endogenous antioxidant enzymes include superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPX). SOD catalyses the conversion of superoxide radicals to hydrogen peroxide, while CAT and GPX subsequently participate in the removal of hydrogen peroxide and other peroxides. Glutathione (GSH), an important intracellular non-enzymatic antioxidant, also contributes to the detoxification of reactive intermediates and maintenance of cellular redox balance. Other non-enzymatic antioxidants include vitamins C and E, carotenoids, bilirubin, uric acid, albumin, coenzyme Q10 and other dietary antioxidant compounds. The coordinated activity of these systems limits oxidative damage and helps preserve normal cellular function [7].

Total antioxidant status (TAS) provides an integrated estimate of the collective antioxidant capacity of biological fluids and may be useful when investigating disorders associated with oxidative imbalance. Rather than evaluating a single antioxidant in isolation, assessment of total antioxidant capacity can provide a broader indication of the ability of the biological system to counteract oxidative challenges. A reduction in antioxidant defence, particularly when accompanied by increased lipid peroxidation, may indicate a shift towards an oxidative environment. Such an imbalance may promote persistent cellular injury and inflammatory

signalling and potentially contribute to the structural and functional changes observed in BPH [8].

The relationship between oxidative stress and inflammation is particularly relevant to prostatic hyperplasia. Inflammatory cells can generate ROS, while oxidative damage can activate inflammatory pathways, creating a potentially self-perpetuating cycle of inflammation and oxidative injury. Chronic prostatic inflammation has been associated with cellular proliferation and tissue remodelling, processes that may contribute to prostate enlargement and LUTS. In addition, inflammatory mediators and oxidative stress may interact with hormonal and growth-factor pathways involved in prostate biology. This interaction provides a biological basis for investigating oxidative stress and antioxidant status in men with BPH [9].

Oxidative stress is also relevant to the broader spectrum of prostate disorders, including prostate cancer. Although BPH does not transform into prostate cancer, oxidative stress, chronic inflammation and ageing-related cellular damage have been implicated in mechanisms associated with carcinogenesis. Studies comparing men with BPH, prostate cancer and apparently healthy controls have generally suggested differences in oxidative stress and antioxidant profiles among these groups[10]. In some reports, prostate cancer has been associated with greater oxidative stress and reduced antioxidant defence compared with benign prostatic conditions. This reinforces the potential value of oxidative stress-related biomarkers as complementary indicators of biological alterations occurring in prostate disease, although such biomarkers should not be regarded as substitutes for established diagnostic procedures [11].

Therapeutic intervention may also influence oxidative stress and antioxidant status in individuals with BPH. Pharmacological treatment can improve urinary symptoms and reduce the functional consequences of prostatic enlargement, but the effects of therapy on systemic oxidative balance may vary according to the drug, duration of treatment, disease severity and individual patient characteristics. Evaluation of oxidative stress markers and antioxidant status among patients receiving therapy may therefore provide additional information about the biological environment accompanying treated BPH. Such information could be useful in understanding whether oxidative imbalance persists despite clinical treatment and whether antioxidant status differs according to treatment response or other clinical characteristics [12].

Several factors may influence oxidative stress in individuals with BPH, including age, obesity, dietary patterns, smoking, alcohol consumption, physical inactivity, environmental exposures and coexisting diseases. These factors are particularly important in interpreting oxidative stress biomarkers because they may independently influence MDA concentrations and antioxidant activity. Consequently, assessment of oxidative stress in a defined clinical population provides an opportunity to characterize the local biochemical profile of patients and generate evidence that may inform future clinical and epidemiological investigations [13].

Despite the growing body of evidence linking oxidative stress, inflammation and prostatic disorders, published information on oxidative stress markers and antioxidant status among Nigerian men with BPH remains limited. In particular, there is a paucity of locally generated data describing the biochemical profile of patients with BPH receiving therapy in South-Eastern Nigeria. Differences in dietary practices, environmental exposures, healthcare-seeking behaviour, treatment patterns and population characteristics may influence oxidative stress and antioxidant status, making locally relevant evidence important. Data from Imo State may therefore contribute to a better understanding of the biochemical characteristics of treated BPH within the Nigerian population.

The present study was therefore undertaken to evaluate selected oxidative stress markers and antioxidant status among subjects with benign prostatic hyperplasia receiving therapy at Imo State University Teaching Hospital, Orlu. By assessing indicators of oxidative damage alongside antioxidant defence, the study seeks to provide evidence on the redox status of men with treated BPH and contribute to the growing understanding of the possible role of oxidative imbalance in the pathophysiology and management of benign prostatic hyperplasia.

MATERIALS AND METHODS

Study Design and Setting

This study was a prospective comparative study designed to evaluate selected oxidative stress markers and antioxidant status among men with benign prostatic hyperplasia (BPH) receiving therapy at the Urology Clinic of Imo State University Teaching Hospital (IMSUTH), Orlu, Imo State, Nigeria. The study involved newly diagnosed, treatment-naïve patients with BPH who were followed up during treatment for six months.

Apparently healthy men without clinical evidence of BPH or other major chronic diseases were recruited as controls.

The study participants were recruited and followed up at baseline, after three months of therapy and after six months of therapy. The study population comprised men aged 40–65 years.

Study Population

A total of 150 participants were enrolled in the study. The study population comprised 75 patients with newly diagnosed, treatment-naïve BPH and 75 apparently healthy controls. The BPH patients were followed prospectively during therapy, with assessment of selected oxidative stress and antioxidant biomarkers at baseline and after three and six months of treatment.

The treatment regimen consisted of bicalutamide (Casodex®) 150 mg once daily, finasteride (Proscar®) 10 mg once daily and tamsulosin 0.4 mg once daily, as prescribed by the attending urologist. Treatment was administered under clinical supervision, and participants were assessed at the scheduled follow-up periods.

Participant Recruitment and Grouping

Participants were recruited with the assistance of the consultant urologist and medical staff of the Urology Department of IMSUTH, Orlu.

The BPH participants were categorized according to treatment status:

- Treatment-naïve BPH group: newly diagnosed patients before commencement of therapy.
- Three-month therapy group: BPH patients who had completed three months of prescribed therapy.
- Six-month therapy group: BPH patients who had completed six months of prescribed therapy.
- Apparently healthy control group: men without a documented history or clinical evidence of BPH or other major chronic illness.

Where the same participants were followed longitudinally from baseline through three and six months, the repeated measurements were treated as longitudinal observations rather than as independent participants.

Inclusion Criteria

Participants were eligible for inclusion if they met the following criteria:

1. Men aged 40–65 years.
2. Newly diagnosed, treatment-naïve patients with BPH attending the Urology Clinic of IMSUTH, Orlu.
3. Patients who commenced the prescribed BPH treatment and were available for follow-up at three and/or six months.
4. Apparently healthy men within the same age range who served as controls.
5. Participants who provided written informed consent.
6. Participants who fulfilled the clinical and laboratory requirements of the study.

Exclusion Criteria

Participants were excluded if they:

1. Were younger than 40 years or older than 65 years.
2. Had a known diagnosis or clinical evidence of prostate cancer.
3. Had diabetes mellitus or other major metabolic, renal, hepatic, cardiovascular or systemic disorders that could significantly influence oxidative stress.
4. Were current smokers.
5. Were regular alcohol consumers.
6. Were receiving antioxidant supplements or other medications known to substantially influence oxidative stress biomarkers, except medications prescribed as part of the study participants' BPH treatment regimen.
7. Had received previous treatment for BPH before enrolment in the treatment-naïve group.
8. Were unable or unwilling to provide informed consent.
9. Failed to attend the scheduled follow-up assessment where longitudinal follow-up was required.

Ethical Considerations

Ethical approval for the study was obtained from the Health Research Ethics Committee of Imo State University Teaching Hospital, Orlu, before commencement of the study. Permission was also obtained from the relevant authorities of the institution and the Urology Department.

The study procedures were explained to each participant, and written informed consent was obtained before enrolment. Participants were informed of their right to withdraw from the study at any stage without prejudice to their medical care. Confidentiality of participants' information was maintained throughout the study, and

data were coded to prevent direct identification of participants.

Blood Sample Collection

Five millilitres (5 mL) of venous blood was collected aseptically from each participant using a sterile syringe and needle. Blood samples were collected from the treatment-naïve BPH participants before commencement of therapy and subsequently at the three- and six-month follow-up visits. A corresponding blood sample was collected from each apparently healthy control at enrolment.

The blood was transferred into appropriate plain sample containers and allowed to clot at room temperature. Samples were subsequently centrifuged, and the serum was separated into appropriately labelled containers. Serum samples were stored under suitable conditions until analysis.

Determination of Oxidative Stress Markers and Antioxidant Status

Selected oxidative stress and antioxidant biomarkers were determined in the serum samples. The biomarkers assessed included malondialdehyde (MDA), glutathione peroxidase (GPx), superoxide dismutase (SOD), reduced glutathione (GSH) and vitamin C.

Malondialdehyde was determined as an indicator of lipid peroxidation and oxidative damage, while GPx, SOD and GSH were assessed as components of the antioxidant defence system. Vitamin C was determined as an important non-enzymatic antioxidant.

The biochemical parameters were determined using validated spectrophotometric and enzyme-linked immunosorbent assay (ELISA) methods, according to the manufacturers' instructions and/or established laboratory protocols. Appropriate quality-control procedures were applied during sample analysis.

Sample Size Determination

The minimum sample size was initially estimated as shown;

$$N = \frac{Z^2P(1-P)}{d^2}$$

Where:

- **N** = minimum required sample size;
- **Z** = standard normal deviate corresponding to the 95% confidence level = 1.96;
- **P** = estimated prevalence of BPH = 8.8% (0.088);
- **d** = desired precision = 5% (0.05).

Thus:

$$N = \frac{(1.96)^2(0.088)(1-0.088)}{(0.05)^2}$$

$$N = \frac{3.8416 \times 0.088 \times 0.912}{0.0025}$$

$$N \approx 123$$

The calculated minimum sample size was therefore 123 participants. However, to improve the adequacy of the study population and allow for possible attrition during follow-up, a total of 150 participants were recruited, comprising 75 BPH patients and 75 apparently healthy controls.

Data Management and Statistical Analysis

Data obtained from the study were coded, entered and analyzed using the Statistical Package for the Social Sciences (SPSS), version 23.0. Continuous variables were expressed as mean ± standard deviation (mean ± SD).

For comparisons involving two independent groups, the independent-samples Student's t-test was used where the assumptions of parametric analysis were satisfied. Comparisons of oxidative stress and antioxidant parameters across treatment time points were performed using appropriate comparative statistical procedures. Where the same participants were assessed repeatedly at baseline, three months and six months, a repeated-measures approach was used to account for within-participant correlation.

The Pearson correlation coefficient was used to assess relationships between selected oxidative stress markers and antioxidant parameters. The strength and direction of associations were reported using correlation coefficients (r).

A two-tailed P-value <0.05 was considered statistically significant. Where appropriate, 95% confidence intervals were reported.

RESULTS:

Table 1: Mean+SD serum values of MDA, GPx, SOD, GHS and Vit C in therapy naive benign prostate hyperplasia patients verses controls

Variables (mean + SD)	BPH (N) (n=75)	Control (n = 75)	t- value	P-value
MDA(mmol/l)	1.93±0.14	1.35±0.03	33.12	0.001
Lower	1.89	1.34		
Upper	1.97	1.35		

GSH (mmol/ml)	3.28±0.41	4.88±0.31	-23.49	0.001
Lower	3.18	4.80		
Upper	4.80	4.96		
GPx(mmol/I)	199.59±31.89	317.86±58.71	-14.97	0.001
Lower	191.35	302.69		
Upper	207.83	333.02		
SOD(mmol/l)	135.35±14.51	174.03±14.75	-12.94	0.001
Lower	131.60	170.22		
Upper	139.09	177.84		
Vit C (ug/ml)	0.74±0.15	1.41±0.31	-14.66	0.001
Lower	0.70	1.33		
Upper	0.78	1.48		

KEYS: PCa - prostate cancer, BPH -benign prostate hyperplasia, GPx- glutathione peroxidase, MDA; Malondialdehyde, SOD: superoxide Dismutase, GSH: glutathione, Fe-Iron, F- ferritin, TIBC - Total iron binding capacity.

There were significantly higher serum levels of MDA (1.93±0.14mmol/l verses 1.35±0.03mmol/l ,p=0.001) in therapy naïve benign prostate hyperplasia patients compared to controls. While there were significant lower Serum levels of GSH(3.28±0.41mmol/ml verse 4.88±0.31mmol/ml,p=0.001),GPx(199.59±31.89mmol/ml verses 317.86±58.71mmol/ml ,p=0.001),SOD (135.35±14.51mmol/l verses 174.03±14.75mmol/l, p=0.001) Vit C(0.74±0.15ug/ml verses 1.41±0.31ug/ml,p=0.001) in therapy naive benign prostate hyperplasia patients compared to controls respectively.

Table 2: Mean + SD serum values of MDA, GPx, SOD, GHS, Vit C in therapy naive benign prostate hyperplasia patients verses benign prostate hyperplasia patients after 3 months therapy

Variables (mean + SD)	BPH (N) (n=75)	BPH (3mth) (n = 75)	t-value	P-value
MDA(mmol/l)	1.93±0.14	1.54±0.09	21.67	0.001
Lower	1.89	1.52		
Upper	1.97	1.57		
GSH (mmol/ml)	3.28±0.41	3.95±0.37	-10.55	0.001
Lower	3.18	3.85		
Upper	3.39	4.04		
GPx(mmol/	199.59±31.89	267.80±36.94	-8.70	0.001
Lower	191.35	258.25		
Upper	207.83	277.34		
SOD(mmol/l)	135.35±14.51	153.53±27.01	-5.14	0.001
Lower	131.60	146.55		
Upper	139.09	160.50		
Vit C (ug/ml)	0.74±0.15	1.13±0.27	-7.28	0.001
Lower	0.70	1.05		
Upper	0.78	1.20		

There were significantly higher serum levels of MDA (1.93±0.14mmol/l verses 1.54±0.09mmol/l ,p=0.001) in therapy naive benign prostate hyperplasia patients compared to benign prostate hyperplasia patients after 3 months therapy respectively. While there were significant lower serum levels of GSH (3.28±0.41mmol/l verses 3.95±0.37mmol/ml, p=0.001) ,GPx(199.59±31.89mmol/ml verses 267.80±36.94mmol/ml,p=0.001) , SOD (135.35±14.51mmol/l verses 153.53±27.01mmol/l,p=0.001),Vit C(0.74±0.15ug/ml verses 1.13±0.27ug/ml,p=0.001)in therapy naive benign prostate hyperplasia patients compared to benign prostate hyperplasia patients after 3 months therapy respectively.

Table 3: serum values of MDA,GPx, SOD, GHS, Vit C on benign prostate hyperplasia patients after 3 months therapy verses benign prostate hyperplasia patients after 6 months therapy

Variables (mean + SD)	BPH (3mt) (n=75)	BPH (6mt) (n = 75)	t-value	P-value
MDA (mmol/l)	1.54±0.09	1.47±0.11	5.24	0.001
Lower	1.52	1.44		
Upper	1.57	1.49		
GSH (mmol/ml)	3.95±0.37	4.50±0.34	-9.42	0.001
Lower	3.85	4.41		
Upper	4.04	4.59		
GPx(mmol/	267.80±36.94	313.30±29.54	-8.02	0.001
Lower	258.25	305.66		
Upper	277.34	320.93		
SOD(mmol/l)	153.53±27.01	167.96±26.12	-2.94	0.005
Lower	146.55	161.21		
Upper	160.50	174.71		
Vit C (ug/ml)	1.13±0.27	1.50±0.23	-7.49	0.001
Lower	1.05	1.44		
Upper	1.20	1.55		

There were significantly higher serum levels of MDA (1.54±0.09mmol/ verses 1.47±0.11mmol/l, p=0.001) in benign prostate hyperplasia patients after 3 months therapy compared to benign prostate hyperplasia patients after 6 months therapy respectively. There were significant lower serum levels of GSH (3.95±0.37mmol/ml verses 4.50±0.34mmol/ml, p=0.001), GPx (267.80±36.94 verses 313.30±29.54 mmol/ml, p=0.001), SOD (153.53±27.01 mmol/l verses 167.96±26.12 mmol/l, p=0.005), Vit C (1.13±0.27ug/ml verses 1.50±0.23 ug/ml, p=0.001) in benign prostate hyperplasia patients after 3 months therapy compared to benign prostate hyperplasia patients after 6 months therapy respectively.

DISCUSSION

The present investigation indicated considerably increased serum concentration of malondialdehyde (MDA) in therapy-naive individuals with benign prostatic hyperplasia (BPH) in comparison with seemingly healthy controls. MDA is a commonly utilised marker of lipid peroxidation and an indirect sign of oxidative damage to cellular membranes [14]. Therefore, the high level of MDA in the untreated BPH group can be related to the imbalance between the production of reactive oxygen species (ROS) and the neutralisation capacity of endogenous antioxidant systems. Increased oxidative stress can cause cellular damage, inflammatory signalling and prostatic cells growth, all of which are involved in the pathogenesis of BPH. This conclusion is similar with the observations published by [15] who showed changes in markers associated to oxidative stress in patients with prostatic problems. This study further supports the hypothesis that oxidative imbalance may be involved in the biochemical processes linked with BPH .

Conversely, the antioxidant indices glutathione peroxidase (GPx), reduced glutathione (GSH), superoxide dismutase (SOD) and vitamin C were considerably lower in therapy-naïve BPH patients compared to the control group. The concomitant rise in MDA and fall in enzymatic and non-enzymatic antioxidants indicates a defective antioxidant defence mechanism in people with untreated BPH. SOD is a crucial first-line enzyme defence against superoxide radicals, while GPx has a role in the reduction of hydrogen peroxide and lipid hydroperoxides. GSH is a major reducing agent and cofactor in several antioxidant processes.

Vitamin C is part of the non-enzymatic antioxidant defence mechanism. A decrease in these antioxidant components can reduce the capacity of the cells to properly scavenge ROS, hence promoting oxidative damage and lipid peroxidation[16]. The lower levels of vitamin C found in untreated patients with BPH may be of special interest. Vitamin C helps to protect cellular components from oxidative damage and helps with the regeneration of other antioxidants such as vitamin E. The interaction between enzymatic and non-enzymatic antioxidants is necessary for redox homeostasis. Therefore, the observed decline in the levels of SOD, GPx, GSH and vitamin C with concurrent increase in MDA imply that untreated BPH can be related with a systemic change to a pro-oxidant state. However, the study was observational in terms of baseline illness condition, and therefore the data show a connection, but not a direct causal role of oxidative stress in BPH[17]. Another interesting observation was that there was considerably higher concentration of MDA in therapy-naïve BPH patients than those who were treated with 3 months of medication. The reduced level of MDA after 3 months of treatment may be due to reduction of lipid peroxidation and oxidative load during treatment. This observation may be explained by improvement of the underlying prostatic state, modifications of inflammatory activity, adjustments in cellular metabolism or an indirect effect of treatment on pathways linked with oxidative stress. This finding is mostly in agreement with earlier observations indicating that treatment interventions in prostatic diseases may be associated with beneficial modifications of the biomarkers related to oxidative stress . It also showed changes in oxidative stress indicators after intervention in patients with prostatic disease[18]. The significantly increased levels of GPx, GSH, SOD and vitamin C detected after three months of therapy in comparison to the therapy-naïve group, give further evidence for an improvement in antioxidant status after treatment. The improvement in these antioxidant measures could be a reflection of a restoration or an increase in the endogenous antioxidant capacity with better clinical control of the disease. The increased antioxidant activity may enhance the ability of the biological system to scavenge ROS and to decrease lipid peroxidation. However, the reported improvement should be regarded with caution, as the study could not independently establish whether the improvements were directly due to the pharmacological treatments, clinical improvement, lifestyle changes or other factors occurring during the follow-up period.[19]

The study also indicated significantly increased MDA concentrations in BPH patients after 3 months of medication compared with those evaluated after 6 months of therapy. The increasing decrease in MDA from 3 to 6 months of treatment could imply a sustained reduction in lipid peroxidation and oxidative burden with longer treatment. The persistent oxidative stress present in the early therapy period may be indicative of persistent disease-related inflammation and cell proliferation or of insufficient recovery of antioxidant capacity. The decreased MDA concentration after six months could be related to a better clinical control of BPH and potentially to an attenuation of inflammatory and oxidative processes with a longer period of therapy. This is in agreement with [20] who showed a relationship between treatment and the modifications in oxidative stress-related markers in patients with prostatic illness.

The overall pattern observed in this study, high MDA and lower antioxidant parameters in therapy-naïve patients and subsequent improvement in antioxidant status and decrease in MDA following therapy, suggests that oxidative imbalance may be associated with untreated BPH and that the redox environment may be altered during therapy. The findings are biologically realistic as excess ROS can induce lipid peroxidation and damage cellular macromolecules and activate inflammatory pathways, whereas a sufficient antioxidant system can restrict these consequences. The results therefore encourage additional research on oxidative stress biomarkers as potential

supplementary indicators of biochemical changes happening during the therapy of BPH[21].

However, at present oxidative stress biomarkers should not be taken as single indications of diagnosis or response to therapy in BPH. The MDA can be regulated by a variety of physiologic and pathologic circumstances, while the concentrations of antioxidants can vary according to age, Nutritional status, lifestyle, comorbidities and usage of medication [22]. The study eligibility criteria accounted for significant potential confounders, although some residual confounding cannot be ignored. Also, the investigation was carried out in one tertiary health institution which may restrict the generalisability of the findings to the larger community of males with BPH in Imo State or Nigeria[23].
The findings may have clinical implications since knowledge of oxidative stress and antioxidant status may provide further insight into the biochemical processes associated with BPH and changes that occur during treatment. However, larger and more heterogeneous prospective investigations are needed to determine if these biomarkers have predictive value for severity, treatment response or long-term clinical outcomes. Other oxidative stress and inflammatory biomarkers, dietary antioxidant intake, treatment adherence, prostate volume, symptom scores and established clinical parameters should also be included in future studies to provide a more comprehensive assessment of the relationship between oxidative stress and BPH[24].

CONCLUSIONS

The present investigation indicates an altered oxidative-antioxidant balance in therapy-naïve patients with benign prostatic hyperplasia with significantly elevated levels of MDA and decreased levels of SOD, GPx, GSH and vitamin C compared to seemingly healthy controls. Results indicate increased lipid peroxidation and decreased antioxidant defence system in BPH untreated. MDA levels dropped post medication and selected antioxidant parameters rose, additional reduction in MDA was observed post six months of treatment. The results of the present study indicate that oxidative stress may be involved in BPH and that oxidative-antioxidant balance may be altered during the treatment. The results indicate that oxidative stress and antioxidant biomarkers may be useful in understanding the biochemical profile of BPH but further longitudinal and multicentre studies are needed to determine their clinical significance and possible role in monitoring treatment response.

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