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RESEARCH ARTICLE

The role of oxidative stress and inflammation biomarkers in pre- and postoperative monitoring of prostate cancer patients

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ABSTRACT

Introduction: Prostate Cancer (PC) is a global health concern affecting men worldwide. Oxidative stress is believed to contribute to the initiation of early-stage PC lesions. Additionally, inflammation has long been acknowledged as a factor in the development of PC. We aimed to examine the biomarkers of oxidative stress and inflammation in PC patients before and after surgery.

Patients and methods: A cross-sectional study was conducted at the Urology Outpatient Clinic of Bezmialem Vakif University Hospital. A total of 150 individuals were included in the study, divided into five groups: 50 Healthy controls, 25 patients with Benign Prostatic Hyperplasia (BPH), 25 patients with Low-Risk Prostate Cancer (LRPC), 25 patients with Medium-Risk Prostate Cancer (MRPC), and 25 patients with High-Risk Prostate Cancer (HRPC). Measurements of Total Oxidant Status (TOS), Total Antioxidant Status (TAS), Total Thiol (TT), and Native Thiol (NT) were performed using photometric methods. Oxidative Stress Index (OSI) and Disulfide (DIS) levels were calculated mathematically. Levels of Interleukin-10 (IL-10), Interleukin-1beta (IL-1β), Tumor Necrosis Factor-alpha (TNF-α), Interleukin-6 (IL-6), and Presepsin were determined using commercially available enzyme-linked immunosorbent assay (ELISA) kits.

Results: Compared to the healthy control group, the results indicated a statistically significant increase in both oxidative stress and inflammation levels. In the groups receiving both pharmaceutical therapy and surgical treatment (PC), a significant decrease in oxidative stress and inflammation levels was observed.

Conclusion: Consequently, it is suggested that the assessment of oxidative stress and inflammatory biomarkers should be incorporated in the pre- and postoperative monitoring of patients with PC.

HIGHLIGHTS

- Total Antioxidant Status (TAS) levels are found to be statistically lower in all PC groups, indicating a correlation between oxidative stress and the progression of PC.
- Levels of inflammatory biomarkers (IL-1β, IL-6, IL-10, TNF-α) were found to be higher before and after surgery in PC groups, and their variation correlated with tumor grade and size.
- Post-surgery, a decrease in presepsin levels is associated with a reduced likelihood of sepsis in PC patients.
- Reductions in oxidative stress and inflammation levels postoperatively suggest the effectiveness of surgical intervention in mitigating these factors.
- The potential for personalized medicine to decrease PC mortality is highlighted by better understanding the functional relationship coordinating inflammatory signatures in the tumor microenvironment.

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Inflammation; oxidative stress; prostate cancer; surgery; thiol-disulfide

Introduction

Cancer is the world's leading cause of mortality, and a lethal disease with a fast-growing prevalence. Cancers are categorized based on the organ or tissue from when they originated. However, molecular pathways are increasingly taken into account when determining the classification of cancer [1]. The health of men around the world is greatly affected by Prostate Cancer (PC). Men are more likely to develop it than any other

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type of cancer. Global Cancer Statistics (GLOBOCAN) estimates that 19 million individuals will receive a cancer diagnosis in 2020, with 10 million of those cases ending in death. Among these rates, PC ranked fourth among other cancer types in terms of diagnosis. High-grade and low-grade PC, early-onset PC, and indolent PC are several subtypes of PC, which is a complicated and diverse malignancy. Age, ethnicity, and inheritance are now the only three known risk factors for PC, despite published results suggesting that exogenous variables (such as food, physical activity, sexual behavior, and occupation) influence PC disease development (i.e. first-line relatives or relatives with early-onset PC) [2–4]. Prostate biopsy is still the go-to method for confirming PC diagnosis. The ability to diagnose PC is made possible by improvements in risk stratification and biomarkers. Prostate-specific antigen (PSA), a glycoprotein isolated by Wang [5] in 1979, is a special multifunctional biomarker utilized for screening, diagnosis, and staging as well as tracking the course and therapy effectiveness of PC.

Digital Rectal Examination (DRE), PSA analysis, imaging using a Transrectal Ultrasound-Guided Scan (TRUS) with at least 12 prostate biopsies, and multiparametric magnetic resonance imaging (mpMRI) scan are some of the current clinical approaches and techniques used to diagnose PC [6, 7]. Currently, the Gleason score is the best predictor of clinical prognosis and therapy responsiveness. The scoring method is based on the Modified Gleason System developed by the International Society of Urological Pathology in 2005. PSA, DRE, and TRUS are used to determine clinical TNM staging, which is complemented by bone scintigraphy, CT scan/PET-CT scan, and mpMRI [8]. In addition, in the American Joint Cancer Committee (AJCC) staging system, urological cancers are evaluated by the universally accepted Tumor Node-Metastasis (TNM) system, including clinical and pathological changes. Reactive oxygen species (ROS) are produced and accumulate in cells and tissues, and a biological system's capacity to detoxify these reactive products is what leads to the phenomenon known as oxidative stress [9]. Infections, damaged cells, and toxic materials are just a few of the factors that can trigger inflammation, the immune system's biological response. Acute and/or chronic inflammatory reactions may occur in the heart, pancreas, liver, kidney, lung, brain, digestive system, and reproductive system; these reactions may cause tissue damage or cancer [10].

Considering that oxidative stress and inflammation are associated with cancer, in this clinical study, we aimed to examine the biomarkers of oxidative stress and inflammation in PC patients before and after surgery.

Patients and methods

Study design

A cross-sectional study was carried out at the Urology Outpatient Clinic of Bezmialem Vakif University Hospital in Istanbul, Turkey. The study sample comprised 150 participants, who were divided into five groups: a control group of 50 healthy individuals, and 4 patient groups consisting of 25 individuals each, with diagnoses of Benign Prostatic Hyperplasia (BPH), Low-Risk Prostate Cancer (LRPC), Medium-Risk Prostate Cancer (MRPC), and High-Risk Prostate Cancer (HRPC). European Association of Urology risk group classification, based on D'Amico's classification system, was used for PCa risk classification. After signing the voluntary consent form from the volunteers, approximately 4–5mL of venous blood was collected from each patient in gel Biochemistry tubes before and after surgical treatment. Samples were centrifuged at 3000 × g for 10min, serums were transferred to eppendorf tubes, and stored at −80°C until analysis. Ethical approval for our study was approved by the local Ethics Committee. Demographic characteristics of healthy volunteers and patients with prostate cancer groups are listed in Table 1.

Tumor stage procedure

Clinical data such as the biopsy Gleason score, clinical T classification, and pretreatment serum PSA and C-reactive protein levels were taken from the medical files. A pathologist determined the Gleason score and stage following prostatectomy. The TNM Staging System was employed by the pathologist to describe the extent of the prostate cancer's spread. Details of the stages are shown in Table 2.

Investigation of oxidative stress parameters

Total Oxidant Status (TOS), Total Antioxidant Status (TAS), Total Thiol (TT), and Native Thiol (NT) levels were measured by photometric methods. Oxidative Stress Index (OSI) and Disulfide (DIS) levels were calculated using mathematical methods. Absorbances were measured using a microplate reader (Synergy-HTX, Biotek, ABD).

Briefly, OSI was calculated by dividing TOS levels by TAS levels, and DIS was calculated by taking half of the difference between TT levels and NT Levels.

$$OSI = \frac{TOS}{TAS}, and\ DIS = \frac{\left(TT - NT\right)}{2}$$

Examining inflammation parameters

Interleukin-1beta (IL-1β, BTLAB E0143Hu), Interleukin-6 (IL-6, BTLAB E0090Hu), Tumor Necrosis Factor-alpha

Table 1. Demographic characteristics of healthy volunteers and patients with prostate cancer groups.

	Healthy ($n=50$)	BPH ($n=25$)	LRPC ($n=25$)	MRPC ($n=25$)	HRPC ($n=25$)	p-value
Age (year)	62.64 ± 6.91	63.88 ± 7.79	63.24 ± 7.17	64.12 ± 6.67	63.48 ± 8.05	>.05
Smoking						
Yes	0 (0%)	11 (44%)	19 (76%)	20 (80%)	21 (84%)	**<.01****
No	50 (100%)	14 (56%)	6 (24%)	5 (20%)	4 (16%)	
Alcohol						
Yes	0 (0%)	6 (24%)	6 (24%)	6 (24%)	10 (40%)	>.05
No	50 (100%)	19 (76%)	19 (76%)	19 (76%)	15 (60%)	
Marital Status						
Married	37 (74%)	18 (72%)	21 (84%)	18 (72%)	22 (88%)	>.05
Single	13 (26%)	7 (28%)	4 (16%)	7 (28%)	3 (12%)	
Education						
Elementary School	9 (18%)	9 (36%)	6 (24%)	9 (36%)	8 (32%)	
High School	20 (40%)	7 (28%)	6 (24%)	4 (16%)	10 (40%)	>.05
University	18 (36%)	8 (32%)	10 (40%)	9 (36%)	5 (20%)	
Graduated	3 (6%)	1 (4%)	3 (12%)	3 (12%)	2 (8%)	
Chronic Disease						
Yes	0 (0%)	12 (48%)	13 (52%)	16 (64%)	18 (72%)	>.05
No	50 (100%)	13 (52%)	12 (48%)	9 (36%)	7 (28%)	

BPH: Benign Prostatic Hyperplasia, LRPC: Low-Risk Prostate Cancer, MRPC: Medium-Risk Prostate Cancer, HRPC: High-Risk Prostate Cancer. The ages of the volunteers are given as mean + SD, while other demographic data are given as percentages.
* $p<.05$ ** $p<.01$ *** $p<.001$.

Table 2. Gleason scores, TNM stages, PSA values, and CRP values of healthy, and patients with prostate cancer groups.

	Healthy ($n=50$)	BPH ($n=25$)	LRPC ($n=25$)	MRPC ($n=25$)	HRPC ($n=25$)	p-value
Gleason Score	–	–				
6			25 (100%)			
7				25 (100%)	7 (28%)	**<.001*****
8					17 (68%)	
9					1 (4%)	
TNM Staging	–	–				
T1c-T2a			25 (100%)			
T2b				25 (100%)		**<.001*****
T2c					25 (100%)	
PSA (ng/mL)	0.24 ± 0.12	2.20 ± 0.82	5.52 ± 1.69	12.08 ± 2.23	20.32 ± 2.66	**<.001*****
CRP (mg/L)	1.42 ± 0.93	7.64 ± 3.30	18.08 ± 5.23	17.96 ± 5.81	18.56 ± 7.19	**<.001*****

Gleason score and TNM staging data are given as percentiles. PSA and CRP levels data are given as mean + standard deviation(SD). Statistically significant was assumed at the $p<.05$ point. BPH: Benign Prostatic Hyperplasia, LRPC: Low-Risk Prostate Cancer, MRPC: Medium-Risk Prostate Cancer, HRPC: High-Risk Prostate Cancer, TNM: Tumor-Node-Metastasis, PSA: Prostate-Specific Antigen, CRP: C-Reactive Protein. *$p<.05$; **$p<.01$; ***$p<.001$.

(TNF-α, BTLAB E0082Hu), and Interleukin-10 (IL-10, BTLAB E0102Hu) levels were determined with commercially purchased enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturer's instructions. Absorbances were measured using a microplate reader (Synergy-HTX, Biotek, ABD).

Inflammation/sepsis status

Presepsin levels were measured with commercially purchased ELISA kits (BTLAB E3754Hu) according to the manufacturer's instructions. Absorbances were measured using a microplate reader (Synergy-HTX, Biotek, ABD).

Statistical analysis

The packaged software Statistical Package for the Social Sciences (SPSS) version 26 was used for the statistical analysis. Non-parametric data were reported as the median and interquartile range (IQR), whereas parametric data were expressed as mean ± standard deviation (SD). Mann–Whitney U test and Student's t-test were used to compare two independent groups, whereas the Kruskal–Wallis test was used to compare more than two independent parameters.

Results

Subjects with active neurological bladder dysfunction, diabetes, urinary infection, cardiovascular diseases, hypogonadism, endocrine dysfunction, psychiatric disorders, and taking anti-depressants were among the exclusion criteria. The age range was 55–70 years for the healthy control group, 56–70 years for the BPH group, 56–71 years for the LRPC group, 57–71 years for the MRPC group, and 55–71 years for the HRPC group.

The healthy control group was excluded from treatment. Pharmacological treatment was performed in the BPH group, the tumor part of the patients in the LRPC group was removed, and radical prostatectomy, which is the main type of surgery for prostate cancer, was performed in the MRPC and HRPC groups.

Oxidative stress parameters of the healthy and PC groups are summarized in Figures 1 and 2. TAS levels were found to be significantly lower than the healthy control group. Postoperative TAS levels were higher in PC groups. TOS and OSI levels increased compared to healthy control. Post-operative, the TOS and OSI levels in the PC groups decreased ($p < .001$).

TT and NT values were found to be significantly lower than the healthy control group. Postoperative TT and NT levels were higher among PC groups ($p < .001$). DIS levels were higher than healthy control. There was no significant difference in DIS levels before or after surgery between the PC groups.

Inflammation/sepsis status and inflammatory parameters of healthy and PC groups are summarized in Figure 3. IL-1β, IL-6, IL-10, TNF-α, and presepsin levels were found to be significantly higher than the healthy control group. Among the PC groups, IL-1β, IL-6, IL-10, and TNF-α levels were significantly lower after surgery ($p < .001$). Presepsin levels (in LRPC, MRPC, and HRPC groups) were significantly higher than the healthy control. Presepsin levels among PC groups (LRPC, MRPC, and HRPC groups) decreased significantly after surgery ($p < .001$).

Evaluating the correlative relationships within pretreatment data demonstrates a significant negative correlation between the antioxidant parameters TAS, TT, and NT levels, and the inflammatory parameters ($p < .01$). Additionally, a positive correlation is evident between TOS, OSI, and DIS – representing oxidative stress parameters – and the inflammatory parameters, as outlined in Table 3 ($p < .01$).

Nevertheless, significant positive correlations were found between TOS, OSI and DIS levels with inflammatory parameters after treatment ($p < .01$). Conversely, significant inverse correlations were found between TT, NT, and TAS levels with inflammatory parameters. Detailed information ($p < .01$) is provided in Table 4.

Discussion

PC, the second most prevalent type of cancer in males, is a serious condition that has an impact on men's health all over the world [11]. One of the most successful methods for treating metastatic PC is testosterone suppression. The risk of PC has been linked to

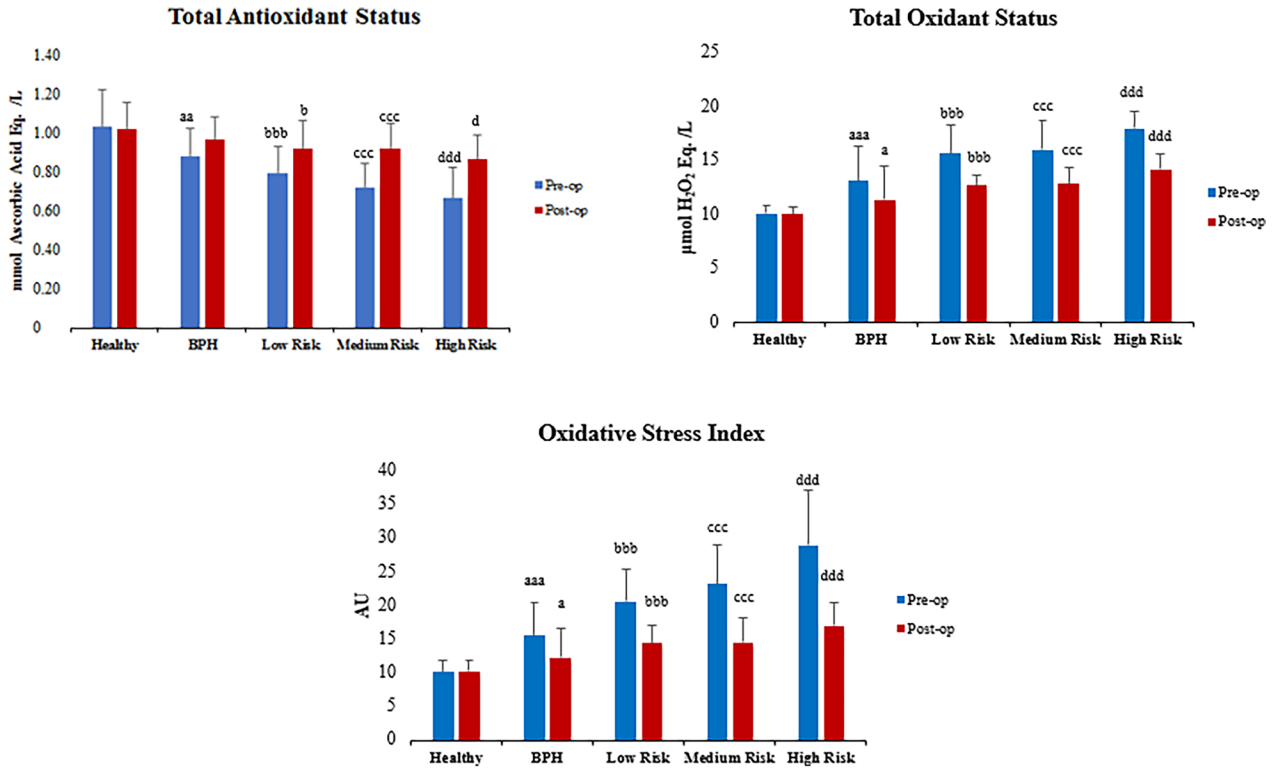

Figure 1. Total Antioxidant Status, Total Oxidant Status, and Oxidative Stress Index levels of healthy and prostate cancer patient groups. BPH: Benign Prostatic Hyperplasia, Pre-op: Preoperative, Post-op: Postoperative. Statistically significant was assumed at the $p < .05$ point. $^{a}p < .05$, $^{aa}p < .01$, $^{aaa}p < .001$; $^{b}p < .05$, $^{bb}p < .01$, $^{bbb}p < .001$; $^{c}p < .05$, $^{cc}p < .01$, $^{ccc}p < .001$; $^{d}p < .05$, $^{dd}p < .01$, $^{ddd}p < .001$.

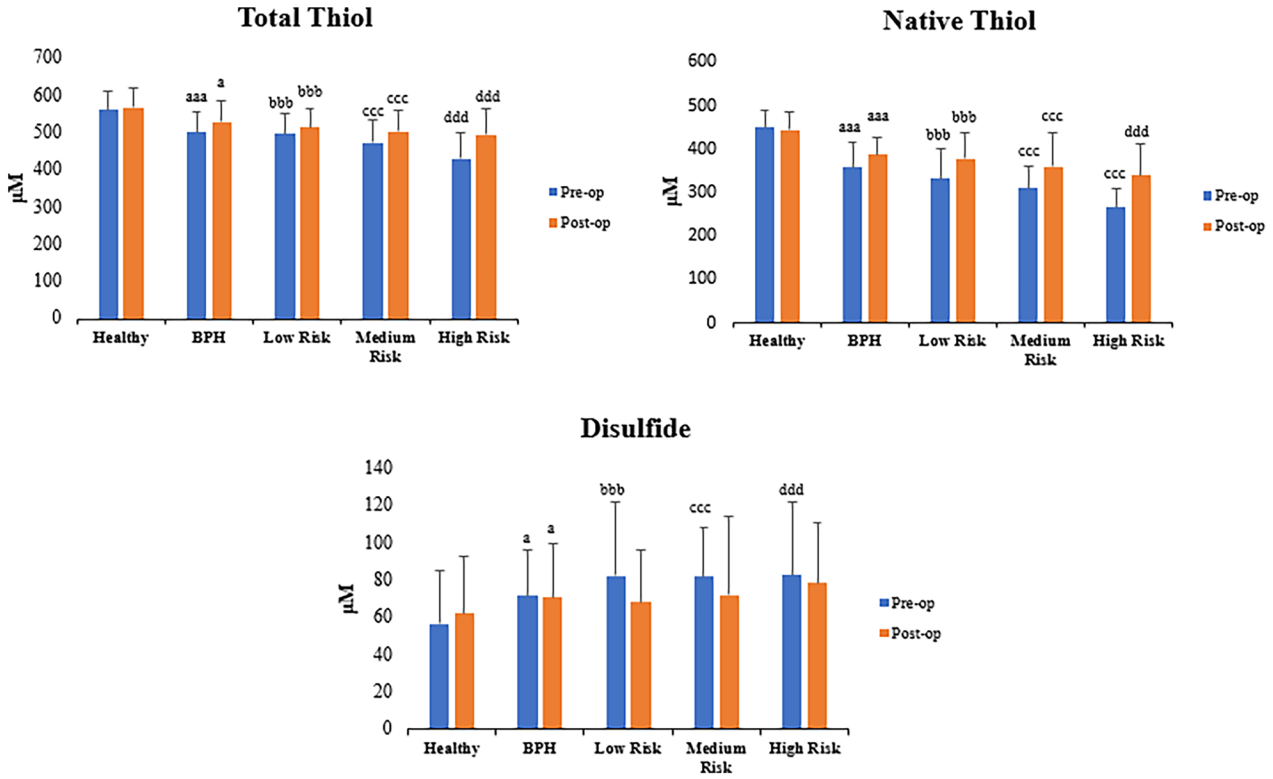

Figure 2. Total Thiol, Native Thiol, and Disulfide levels of healthy and prostate cancer patient groups BPH: Benign Prostatic Hyperplasia, Pre-op: Preoperative, Post-op: Postoperative. Statistically significant was assumed at the $p < .05$ point. $^{a}p < .05$, $^{aa}p < .01$, $^{aaa}p < .001$; $^{b}p < .05$, $^{bb}p < .01$, $^{bbb}p < .001$; $^{c}p < .05$, $^{cc}p < .01$, $^{ccc}p < .001$; $^{d}p < .05$, $^{dd}p < .01$, $^{ddd}p < .001$.

elevated cellular reactive oxygen species (ROS) and weakened defense mechanisms. The development of PC is significantly influenced by ROS. A small increase in ROS can stimulate proliferation, but a significant increase in ROS levels will cause apoptosis. ROS affects both cell proliferation and apoptosis [12]. Oxidative stress is thought to be one of the mechanisms that cause the early stages of PC lesions, specifically benign prostatic hyperplasia (BPH) [13]. Here, we aimed to investigate the relationship between oxidative stress and inflammation levels in different stages of PC' patient groups.

Through the immunological activity, oxidative metabolism, and mitochondrial bioenergetics, the body continuously produces ROS. ROS are typically created during cell division, growth, death, and differentiation in the forms of hydroxyl radical (•OH), hydrogen peroxide (H_2O_2), superoxide anion ($O\cdot^-_2$), singlet oxygen ($^1O_2$), and lipid peroxides. They can bind to enzymes, nucleic acids, membrane lipids, proteins, and other biomolecules. A major risk factor for the development and spread of PC has been identified as increased oxidative stress. Ahmed Amar, Safa Ali, et al. reported that the Superoxide Dismutase (SOD), Glutathione (GSH), and Catalase (CAT) activities of the patient groups in PC patients were significantly lower than the healthy control group [14, 15]. To minimize tissue damage brought on by oxidative stress, Battisti et al. [16] hypothesized that the elevated levels might comprise a compensating mechanism. In a case-control study by Byeongsang Oh et al. oxidative stress markers were found to be significantly higher in patients with PC than in controls [17]. Few studies have assessed total antioxidant status, which encompasses the combined effects of various antioxidant defenses in plasma and offers a more comprehensive assessment of antioxidant defenses [18]. According to the findings of our study, TAS levels were found to be statistically lower in all PC groups (BPH, Low risk, Medium risk, High risk) before and after surgery compared to the healthy control group. Before and after surgery, TOS and OSI levels were found to be statistically significant and higher in all PC groups (BPH, Low risk, Medium risk, High risk) compared to the healthy control group. When we compared the groups among themselves, as the cancer risk increased, TAS levels decreased significantly, and TOS and OSI levels increased. TT and NT levels were found to be statistically lower before and after surgery in all PC groups compared to the healthy control group. DIS levels were found to be significantly lower in all PC groups compared to the healthy control group. While reactive oxygen and nitrogen species were produced

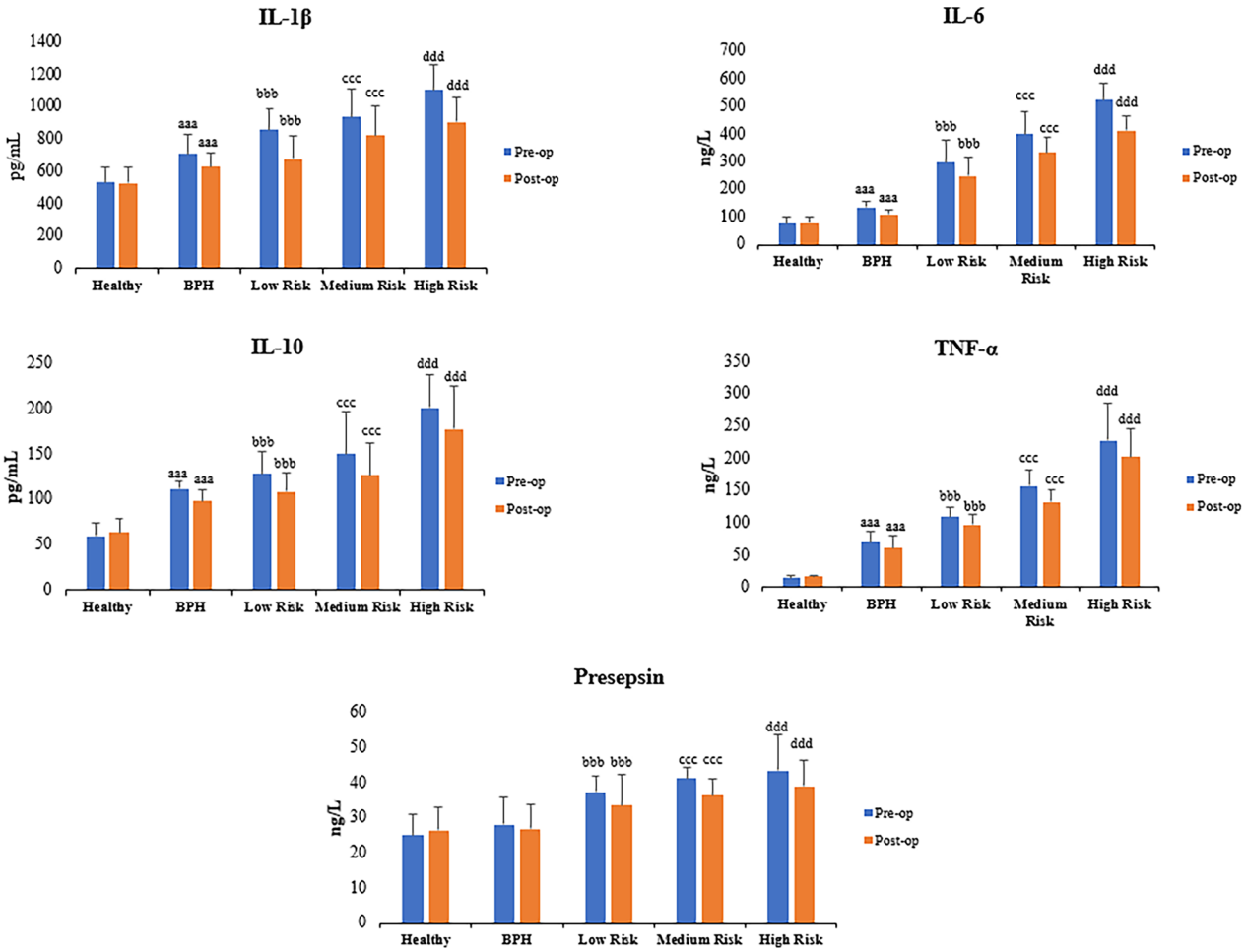

Figure 3. Interleukin-1beta (IL-1β), Interleukin-6 (IL-6), Interleukin-10 (IL-10), Tumor Necrosis Factor-alpha (TNF-α), and Presepsin levels of healthy and prostate cancer patient groups. BPH: Benign Prostatic Hyperplasia, Pre-op: Preoperative, Post-op: Postoperative. Statistically significant was assumed at the $p < .05$ point. $^{a}p < .05$, $^{aa}p < .01$, $^{aaa}p < .001$; $^{b}p < .05$, $^{bb}p < .01$, $^{bbb}p < .001$; $^{c}p < .05$, $^{cc}p < .01$, $^{ccc}p < .001$; $^{d}p < .05$, $^{dd}p < .01$, $^{ddd}p < .001$.

Table 3. Association between serum oxidative stress levels and inflammatory parameters for pretreatment.

	IL-1β	IL-6	IL-10	TNF-α	Presepsin
TOS	.702**	.764**	.675**	.747**	.564**
	0.000	0.000	0.000	0.000	0.000
TAS	−.686**	−.590**	−.599**	−.594**	−.409**
	0.000	0.000	0.000	0.000	0.000
OSI	.807**	.749**	.710**	.721**	.500**
	0.000	0.000	0.000	0.000	0.000
TT	−.646**	−.563**	−.568**	−.597**	−.376**
	0.000	0.000	0.000	0.000	0.000
NT	−.782**	−.690**	−.686**	−.701**	−.547**
	0.000	0.000	0.000	0.000	0.000
DIS	.290**	.263**	.253**	.240**	.286**
	0.000	0.001	0.002	0.003	0.000

TOS: total oxidant status, TAS: total antioxidant status, OSI: oxidative stress index, TT: total thiol, NT: native thiol, DIS: disulfide, IL: Interleukin, TNF-α: tumor necrosis factor-alpha.
* $p < .05$, ** $p < .01$, *** $p < .001$.

by all aerobic cells in general, an increase in ROS is linked to aging and its beginning. Although the creation of ROS may not be a major factor in the aging process, oxidative damage and interactions with mitochondria are more likely to be the driving forces behind aging [19]. Different forms of cancer have been linked to inflammation, with persistent inflammation and chronic infections thought to be responsible for 20% of cancer-related mortality [20]. It has long been recognized that inflammation plays a part in PC.

Table 4. Correlative relation between post-treatment serum oxidative stress and inflammatory parameters.

	IL-1β	IL-6	IL-10	TNF-α	Presepsin
TOS	.522**	.640**	.573**	.613**	.356**
	0.000	0.000	0.000	0.000	0.000
TAS	−.367**	−.376**	−.306**	−.350**	−.240**
	0.000	0.000	0.000	0.000	0.003
OSI	.544**	.608**	.525**	.571**	.361**
	0.000	0.000	0.000	0.000	0.000
TT	−.349**	−.404**	−.331**	−.438**	−.216**
	0.000	0.000	0.000	0.000	0.008
NT	−.483**	−.491**	−.474**	−.543**	−.244**
	0.000	0.000	0.000	0.000	0.003
DIS	.174*	0.129	.182*	0.152	0.049
	0.033	0.115	0.026	0.064	0.553

TOS: total oxidant status, TAS: total antioxidant status, OSI: oxidative stress index, TT: total thiol, NT: native thiol, DIS: disulfide, IL: Interleukin, TNF-α: tumor necrosis factor-alpha.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Inflammation is traditionally defined as a normal reaction to tissue injury or infection based on the physical appearance of immune invading cells. The significance of immune cell infiltration (coexistence with prostate tumor) and correlative connection (protective or tumor-promoting) remain ambiguous, despite the detection of infiltrating immune cells in PC specimens. According to multiple research that examined the histopathology of prostate tumor biopsies, inflammatory infiltrates are not positively correlated with PC. Studies have shown that inflammation in PC-negative biopsies reduces the risk of PC and the likelihood of a subsequent PC diagnosis [21]. The function of NF-κB in the development of inflammation and prostate cancer has been recognized. Tumor-promoting cytokines like IL-6 and TNF-α are upregulated in response to the nuclear factor-kappa B (NF-κB). Numerous studies have suggested that inflammatory cytokines and chemokines play a function in PC to identify a possible target of inflammation [22, 23]. Clinically, the levels of IL-6, IL-18, and IL-1β are correlated with the possibility of developing carcinoma and the prognosis for cancer that has already spread. Although research on serum IL-18's diagnostic value in cancer is scarce, the significance of IL-6 in prostate cancer is widely known. According to a study by Dwivedi et al. of 149 patients, locally advanced PC had BPH and significantly higher serum IL-18 levels than healthy controls. In contrast, they reported that IL-10 showed a significant direct association with carcinoma progression [24]. Urine reflux can cause a chemical irritant to the prostate epithelium that can lead to the release of pro-inflammatory cytokines and the development of chronic inflammation. Studies on rats have shown that urine reflux increases the production of the inflammatory cytokines TNF-α, IL-6, and IL-1β in the prostate, as well as the infiltration of inflammatory cells [25]. We found that the levels of inflammation decreased after surgical treatment. IL-1β, IL-6, IL-10, and TNF-α levels were statistically higher before and after surgery compared to the healthy control group. According to the tumor grade, we observed a substantial difference in the levels of inflammatory biomarkers between the groups. The reason for the high oxidative stress levels before surgery supports the idea that ROS accumulated during tumor formation and the overproduction of inflammatory cytokines that support the formation of inflammation. The size of the tumor or the stage of the cancer was found to correlate with an increase in inflammation. Prostate biopsy samples frequently show signs of acute or chronic inflammation, which is regarded to be one of the main reasons for increased serum PSA. PSA is a marker specific to an organ rather than a malignancy, although it is employed in the early diagnosis of PC, preoperative staging, and follow-up after therapy [26]. Histopathologic inflammation in prostate biopsies among men with originally elevated serum PSA levels was not associated with an elevated risk of PC in a PC screening study. Furthermore, PC detection in follow-up biopsy was adversely correlated with inflammatory histological findings during the initial prostate biopsy [27, 28]. CRP and lactate dehydrogenase (LDH) are two additional circulating inflammatory variables that have been investigated about PC prognosis in distinct malignancies. A common circulating marker called CRP is used to identify both acute and chronic inflammation in the body. The risk dimension of cancer was correlated with CRP and PSA levels and was statistically significant. Presepsin is used as a predictive biomarker during the evaluation of sepsis status, and it is predicted that it may provide better insight into laboratory studies of sepsis, especially when used together with other biomarkers and clinical rating scores [29].

While evaluating the sepsis/inflammation status, the tumor size is important in patients with PC. When the results were examined, it was revealed that the sepsis status was not significant in the healthy control and BPH groups. However, the presepsin levels of the LRPC, MRPC,

and HRPC groups were significantly higher than the healthy control group, both before and after surgery. We found that presepsin levels were decreased in the LRPC, MRPC, and HRPC groups after surgery, and we observed that the likelihood of sepsis in patients was reduced. Nearly all men diagnosed with PC will deal with the repercussions of their diagnosis and treatment because the 5-year cancer survival rate is close to 100%. Guidelines for prostate survivorship have been developed by the American Cancer Society (ACS) to assist patients, carers, and medical professionals with this part of care (i.e. the quality of life and health of males after treatment) [7, 30]. The molecular role of inflammation as a causal event at the beginning of PC, however, is still being investigated. It may be possible to predict PC progression and response to therapeutic approaches in advanced disease by better understanding the functional relationship coordinating the inflammatory signatures in the tumor microenvironment, enabling personalized medicine (targeted treatment approaches) to decrease PC mortality. It is clear that the interactions and complicated mechanisms underlying the relationship between the risk of PC and ROS-mediated oxidative stress demand additional discussion. With future studies, oxidative stress and inflammation parameters in PC can be used with other differential diagnostic criteria and may be an option to differentiate prognosis in PC patient groups.

Conclusion

In the present study, we aimed to evaluate the changes in oxidative stress and inflammation parameters in individuals diagnosed with Prostate Cancer (PC) before and after surgical intervention. Our findings revealed a reduction in both oxidative stress and inflammation levels postoperatively in these patients. It is well established that inflammation induced by oxidative stress plays a significant role in the development of PC. Given this, our results highlight the importance of monitoring oxidative stress and inflammation biomarkers in the pre- and postoperative treatment follow-up of PC patients. Furthermore, our study proposes the use of a combination of oxidative stress and inflammation biomarkers, along with clinical and laboratory data, to predict the prognosis of PC. To the best of our knowledge, this is the first study to evaluate the changes in oxidative stress and inflammation biomarkers in individuals diagnosed with PC before and after surgical intervention.

Acknowledgments

Our current study was presented as an oral presentation at the Multidisciplinary Cancer Research Association (MOKAD) congress on 21–24 July 2022. The authors dedicated this publication to the 100th anniversary of the Republic of Türkiye. As scientists raised by Türkiye, they are proud to be citizens and scientist of this country. Long live the Republic of Türkiye!

Author contributions

HB: Analysis and Arrangement, Data curation, Data editing, Formal analysis, Writing - original draft. **CE**: Project administration, Data curation, Formal analysis, Writing – original draft. **BNO**: Data improvement, Formal analysis. **IO**: Conceptualization, Analysis and Arrangement. **HSP**: Review and editing. **AID**: Data optimization, Formal analysis. **EC**: Resources, Review and regulation. **EMG**: Conceptualization, Writing - review and editing, Supervision, Methodology.

Disclosure statement

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Ethical approval

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