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CORRELATION BETWEEN PROSTATE SPECIFIC ANTIGEN (PSA) LEVEL AND C-REACTIVE PROTEIN (CRP) LEVEL IN PATIENTS WITH BENIGN PROSTATE HYPERPLASIA

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Abstract

Background: *Benign prostatic hyperplasia (BPH) is a common condition in aging men, and inflammation may contribute to its development and progression. Prostate-specific antigen (PSA) can be elevated in BPH, while C-reactive protein (CRP) may reflect systemic inflammatory activity. This study evaluated the relationship between PSA and CRP in patients with BPH.*

Objective: *To determine the correlation between serum PSA and CRP levels in patients with BPH.*

Methods: *This cross-sectional study was conducted at the Department of Medicine, Khyber Teaching Hospital, Peshawar, from 20 November 2024 to 25 March 2025. A total of 347 male patients aged 40–80 years with BPH diagnosed by ultrasonography were enrolled through consecutive sampling. Serum PSA and CRP levels were measured, and their correlation was assessed using Pearson's correlation coefficient. Stratified analyses were performed according to age, BMI, comorbidities, and prostate size.*

Results: *The mean age was 62.4 ± 8.7 years and mean prostate size was 48.6 ± 16.2 g. Mean PSA was 5.21 ± 3.84 ng/mL, while median CRP was 0.72 (IQR: 0.38–1.34) mg/dL. A significant positive correlation was observed between PSA and CRP ($r = 0.28$, $p < 0.001$).*

Conclusion: *PSA and CRP demonstrated a weak but significant positive correlation in patients with BPH, suggesting a possible association between systemic inflammation and PSA elevation.*

INTRODUCTION

Benign prostatic hyperplasia (BPH) is one of the most common benign conditions affecting aging men and is characterized by nonmalignant proliferation of prostatic stromal and epithelial components, predominantly in the transition zone of the prostate. The prevalence of BPH increases substantially with advancing age and is frequently associated with lower urinary tract symptoms (LUTS), including urinary frequency, urgency, weak urinary stream, hesitancy, and incomplete bladder emptying. Although BPH is a benign condition, its clinical presentation and laboratory findings may overlap with other prostatic disorders, particularly prostatitis and prostate cancer, making accurate assessment important in older men [1,2].

Prostate-specific antigen (PSA) is a serine protease produced predominantly by prostatic epithelial cells and is widely used as a biomarker in the

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evaluation of prostate disorders. PSA normally enters the circulation in small amounts; however, disruption of the normal architecture and epithelial integrity of the prostate can increase its passage into the bloodstream. Although PSA is extensively used for prostate cancer detection and risk assessment, its elevation is not specific to malignancy. Benign conditions such as BPH and prostatitis, as well as prostatic manipulation including catheterization, cystoscopy, prostate biopsy, and prostate massage, may also increase serum PSA concentrations [1,3].

The interpretation of an elevated PSA level in a patient with BPH therefore remains clinically challenging. PSA concentrations may increase with increasing prostate volume because a greater amount of benign prostatic tissue contributes to PSA production. In addition, variations in PSA may occur because of concomitant inflammatory processes within the prostate. Consequently, an elevated PSA level does not necessarily indicate prostate cancer, and additional clinical and laboratory information may be required to identify factors contributing to PSA elevation in men with apparently benign prostatic disease [2,4].

Inflammation has increasingly been recognized as an important component of the pathophysiology of BPH. Chronic or subclinical inflammatory activity within prostatic tissue may contribute to cellular proliferation, tissue remodeling, and progression of prostate enlargement. Inflammatory mediators and immune cells may influence the local prostatic microenvironment and potentially contribute to the development and progression of LUTS. Several studies have therefore proposed that inflammation may represent an important therapeutic and biological target in patients with BPH [5].

C-reactive protein (CRP) is an acute-phase protein synthesized predominantly by hepatocytes in response to inflammatory cytokines, particularly interleukin-6 (IL-6). It is a commonly available and relatively sensitive systemic marker of inflammation, although it is not specific for any particular inflammatory disease. Increased CRP concentrations may occur in infections, inflammatory disorders, tissue injury, malignancies, and other systemic conditions. Because prostatic inflammation may induce systemic inflammatory responses, circulating CRP has been investigated as a potential indirect marker of inflammatory activity associated with prostate disease [3,6].

The possible relationship between CRP and PSA is of particular interest in patients with BPH because inflammation may provide a biological explanation for PSA elevation in the absence of malignancy. Previous investigations have examined inflammatory biomarkers in prostate diseases and have suggested an association between inflammatory activity and various clinical characteristics of prostatic disorders. However, systemic CRP may be influenced by numerous factors unrelated to the prostate, and its ability to reflect localized prostatic inflammation remains uncertain. Therefore, the relationship between serum PSA and CRP in men with BPH requires further evaluation [4,7].

Previous evidence has also highlighted the potential role of inflammatory pathways in the development and progression of BPH. Prostatic inflammatory biomarkers may provide additional information regarding disease activity and could potentially contribute to understanding why some patients with benign prostatic enlargement have higher PSA concentrations than others. However, the clinical significance of circulating CRP in relation to PSA among patients with BPH has not been adequately established, particularly in routine clinical populations. Determining whether PSA and CRP demonstrate a significant correlation may provide further insight into the possible contribution of systemic inflammatory activity to PSA elevation in BPH [5,7].

Despite the widespread use of PSA in the evaluation of men with prostate disorders, there remains uncertainty regarding the contribution of inflammation to PSA elevation among patients diagnosed with BPH. Establishing a correlation between PSA and CRP may help clarify the potential association between inflammatory activity and PSA levels in these patients. Such information may contribute to a better understanding of the biological relationship between BPH, inflammation, and PSA and may provide a basis for future studies evaluating

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inflammatory markers and anti-inflammatory approaches in BPH [1,6,7].

OBJECTIVE

To determine the correlation between serum prostate-specific antigen (PSA) level and serum C-reactive protein (CRP) level in patients with benign prostatic hyperplasia.

METHODOLOGY

This cross-sectional study was conducted in the Department of Medicine, Khyber Teaching Hospital (KTH), Peshawar, from 20 November 2024 to 25 March 2025. The study included male patients aged 40–80 years who presented to the outpatient department and were diagnosed with benign prostatic hyperplasia (BPH) according to the predefined operational criteria. BPH was defined on the basis of abdominal ultrasonography, with a prostate volume greater than 30 g considered consistent with BPH. Patients were recruited using a non-probability consecutive sampling technique. The study was initiated after obtaining approval from the institutional research review board, and written informed consent was obtained from all participants before enrollment.

Male patients aged 40–80 years with ultrasonographically documented BPH were eligible for inclusion. Patients with active infection, a previous history of malignancy, use of steroids or anti-inflammatory medications during the preceding three months, urethral stricture, previous urinary bladder surgery, or previous prostate surgery were excluded. These exclusion criteria were applied to minimize factors that could independently influence circulating PSA or CRP levels and thereby affect the assessment of their correlation.

After enrollment, demographic and clinical information was recorded using a structured proforma. Variables included age, body mass index (BMI), residence, educational status, profession, socioeconomic status, and relevant comorbidities. Weight and height were recorded for calculation of BMI as weight in kilograms divided by height in meters squared. Diabetes mellitus was documented according to the predefined study criterion of fasting blood sugar greater than 130 mg/dL, while hypertension was documented when blood pressure was greater than 140/90 mmHg. Prostate size was determined by abdominal ultrasonography and recorded in grams.

For biochemical assessment, approximately 10 mL of venous blood was obtained from a peripheral vein of the non-dominant upper limb under aseptic conditions. The blood sample was transported to the hospital laboratory for measurement of serum PSA and plasma CRP levels according to the laboratory's standard procedures. Laboratory analysis was performed by an experienced biochemist who was blinded to the participants' clinical and demographic information. Serum PSA concentration was recorded in ng/mL, while CRP concentration was recorded in mg/dL. A PSA level greater than 4 ng/mL was categorized as raised PSA, and a CRP level greater than 1 mg/dL was categorized as raised CRP according to the operational definitions of the study.

All collected information was entered into a structured database and checked for completeness and consistency before statistical analysis. Continuous variables, including age, BMI, prostate size, PSA level, and CRP level, were assessed for distribution using the Shapiro–Wilk test. Normally distributed continuous variables were presented as mean ± standard deviation, whereas non-normally distributed variables were presented as median with interquartile range. Categorical variables, including residence, education, profession, socioeconomic status, diabetes mellitus, hypertension, and categorized PSA and CRP levels, were presented as frequencies and percentages.

The primary analysis evaluated the relationship between serum PSA and CRP levels using the Pearson correlation coefficient (r) when the data fulfilled the assumptions for parametric correlation. The magnitude of correlation was interpreted as weak when r was less than 0.40, moderate when r was 0.40–0.60, and strong when r was greater than 0.60. A positive correlation coefficient indicated that increasing PSA levels were associated with increasing CRP levels,

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whereas a negative coefficient indicated an inverse relationship. The statistical significance of the correlation was assessed using a two-tailed test, with a p-value ≤0.05 considered statistically significant.

To assess potential effect modification, the correlation between PSA and CRP was further examined after stratification according to age, BMI, presence of comorbidities, and prostate size. Where appropriate, post-stratification comparisons were performed using the independent-samples t-test for normally distributed continuous variables and the Mann–Whitney U test for non-normally distributed continuous variables. All statistical analyses were performed using IBM SPSS Statistics version 25. Confidentiality of participant information was maintained throughout the study, and data were used exclusively for research purposes.

RESULTS

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DISCUSSION

Benign prostatic hyperplasia (BPH) is a common age-related condition in men and is characterized by proliferation of stromal and epithelial components of the prostate. Although the pathogenesis of BPH is multifactorial, increasing evidence indicates that chronic inflammation may contribute to the development and progression of prostatic enlargement and lower urinary tract symptoms (LUTS). Inflammation may promote tissue remodeling, cellular proliferation, and changes in the local prostatic microenvironment through the release of cytokines and other inflammatory mediators [9,10]. The present study was conducted to determine whether serum prostate-specific antigen (PSA) levels are correlated with C-reactive protein (CRP), a commonly available marker of systemic inflammation, among patients with BPH.

In the present study, the mean age of participants was 62.4 ± 8.7 years, indicating that the study population predominantly represented older men in whom BPH is commonly encountered. The mean prostate size was 48.6 ± 16.2 g, while the mean PSA concentration was 5.21 ± 3.84 ng/mL. Approximately 38.3% of participants had PSA levels above 4 ng/mL, despite all participants being enrolled on the basis of benign prostatic enlargement. This finding illustrates an important clinical issue: PSA elevation is not specific to prostate cancer and may also occur in men with BPH because of prostate volume and other benign prostatic processes. Contemporary evidence emphasizes that PSA should not be interpreted as a binary marker of malignancy and can also provide information regarding benign prostatic disease and its progression [11].

The principal finding of the present study was a statistically significant positive correlation between serum PSA and CRP levels, with a Pearson correlation coefficient of **$r = 0.28$ ($p < 0.001$)**, as shown in Table 3. According to the predefined interpretation used in this study, this represents a weak positive correlation. Thus, participants with higher PSA levels tended, on average, to have higher CRP concentrations. Importantly, the relatively modest correlation coefficient indicates that CRP explains only a limited proportion of the variation in PSA levels. Therefore, although the finding supports an association between inflammatory activity and PSA concentration, it should not be interpreted as evidence that CRP is an independent determinant of PSA or that CRP can be used to distinguish benign from malignant prostatic disease.

The observed positive relationship is biologically plausible because inflammation may disrupt the normal epithelial-stromal architecture of the prostate and increase the leakage of PSA into the circulation. Previous pathological studies have demonstrated an association between the intensity or aggressiveness of prostatic inflammation and serum PSA levels in men with BPH. In particular, glandular inflammatory changes that involve

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disruption of the prostatic epithelium may be more relevant to PSA elevation than the mere presence of inflammatory cells [12]. Similarly, earlier work in men with elevated PSA and negative evaluations for clinically detectable prostate cancer demonstrated that prostate volume and subclinical inflammation contributed to serum PSA variation [13]. These observations provide a possible biological explanation for the positive association identified in the current study. Our findings are also consistent with clinical evidence suggesting that systemic inflammatory markers may be associated with PSA and symptoms in men with BPH. In a large study of men with BPH and LUTS, serum CRP was significantly associated with PSA as well as with storage urinary symptoms, although the relationship between CRP and prostate volume was not significant [14]. This is relevant to the current findings because it suggests that CRP may reflect inflammatory processes related to the clinical phenotype of BPH rather than simply reflecting prostate size. Another investigation found that increased serum CRP was associated with urgency and other storage symptoms in men with BPH, supporting the possibility that inflammatory activity may contribute to the clinical manifestations of the disease [14].

The present study also found that participants with PSA levels greater than 4 ng/mL had a higher median CRP concentration than participants with PSA levels ≤4 ng/mL. Similarly, participants with CRP >1 mg/dL had higher mean PSA levels than those with CRP ≤1 mg/dL (Table 5). These findings are compatible with the hypothesis that inflammatory activity may contribute to increased PSA concentrations in some men with BPH. However, the relationship should be interpreted cautiously because PSA is affected by several factors, including prostate volume, age, benign prostatic disease, infection, instrumentation, and malignancy. Likewise, CRP is affected by systemic inflammatory conditions and is therefore not specific to inflammation within the prostate [11,15].

The relationship between systemic CRP and actual prostatic inflammation remains an important consideration. Although CRP is a useful marker of systemic inflammation, it does not necessarily reflect the intensity of inflammation within prostate tissue. A study involving patients undergoing surgery for BPH found that serum CRP was associated with overactive bladder symptoms but was not correlated with the histological magnitude of prostatic inflammation [16]. This finding indicates that serum CRP should not be regarded as a direct surrogate for histologically confirmed prostatic inflammation. Therefore, the positive PSA–CRP correlation observed in the present study may reflect systemic inflammatory activity associated with BPH or related metabolic and clinical factors rather than direct measurement of inflammation within the prostate.

The stratified analysis in the present study demonstrated that the positive association between PSA and CRP remained present across several clinical subgroups (Table 4). The correlation was somewhat stronger among participants aged ≥60 years (**r = 0.31**) than among those younger than 60 years (**r = 0.23**). This observation may be related to the increasing prevalence of both BPH and chronic inflammatory changes with advancing age. Persistent prostatic inflammation has been reported more frequently in older men and in those with larger prostate volumes or more severe storage symptoms [9,10]. Nevertheless, the differences in correlation coefficients between age groups should be interpreted as exploratory because the present study was not specifically powered to compare correlation coefficients between all subgroups.

A similar pattern was observed according to prostate size. Participants with prostate size >50 g demonstrated a stronger correlation between PSA and CRP ($r = 0.34$, $p < 0.001$) compared with those with prostate size ≤50 g ($r = 0.21$, $p = 0.006$) (Table 4). This finding may have clinical relevance because larger prostates contain greater quantities of epithelial tissue capable of producing PSA and may also exhibit a greater burden of chronic inflammatory changes. Previous reviews have reported that persistent prostatic inflammation is particularly common in men with larger prostate volumes and may contribute to progression of BPH and LUTS [9,10]. However, prostate volume

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itself is an important determinant of PSA, and the present findings do not establish that inflammation independently causes the greater PSA concentration observed in men with larger prostates.

The correlation was also observed among participants with and without diabetes mellitus and hypertension. The coefficient was slightly higher among hypertensive participants than among those without hypertension, while a positive relationship was present in both diabetic and nondiabetic subgroups. Systemic metabolic and vascular conditions may influence inflammatory markers and could therefore contribute to variation in CRP. Previous epidemiological evidence has suggested associations between systemic inflammatory markers and symptomatic BPH, although some relationships are attenuated after adjustment for body mass index and other potential confounding variables [17]. This emphasizes the importance of considering comorbidities and metabolic factors when interpreting the relationship between CRP and PSA.

The current findings are further supported by recent evidence highlighting the biological importance of chronic inflammation in BPH. Chronic inflammatory processes may arise through multiple mechanisms, including immune responses, tissue injury, urinary reflux into prostatic ducts, hypoxia, hormonal influences, and infection-related pathways. These processes can stimulate inflammatory cytokines and growth factors that promote stromal and epithelial proliferation [10]. Consequently, inflammation may be involved not only in PSA elevation but also in the progression of BPH and associated LUTS. The present finding of a positive PSA–CRP correlation therefore fits within the broader concept that inflammation may be an important component of BPH pathophysiology.

At the same time, evidence regarding CRP as a marker of prostatic inflammation is inconsistent. In a study of BPH patients undergoing surgical treatment, serum CRP was not associated with histologically assessed prostatic inflammation, suggesting that systemic CRP may not accurately quantify local inflammatory activity [16]. Similarly, an earlier population-based cohort study found that CRP was associated with irritative LUTS and decline in peak urinary flow but was not significantly associated with subsequent increases in PSA or prostate volume [17]. These findings indicate that inflammation may influence particular clinical manifestations of BPH without producing a uniform increase in PSA or prostate size. Therefore, the relatively weak correlation observed in the present study is plausible and may reflect the complex and multifactorial determinants of PSA.

Studies evaluating inflammatory biomarkers in BPH have also produced variable findings regarding their relationship with PSA. In one study of patients with BPH and prostate cancer, CRP and other inflammatory biomarkers were elevated in BPH compared with healthy individuals, but some inflammatory markers did not correlate with PSA [18]. Conversely, research among BPH patients with smoking exposure demonstrated positive associations between PSA and high-sensitivity CRP together with other inflammatory and oxidative stress markers [19]. Differences in study populations, smoking status, comorbidities, biomarker assays, PSA distributions, and methods of defining BPH may account for some of the variation between studies. The current study therefore adds evidence from a clinical BPH population but does not establish a universal relationship between CRP and PSA.

The findings may have potential implications for understanding PSA elevation in men with BPH. A proportion of patients with BPH may have elevated PSA in the absence of malignancy, and inflammation represents one possible contributing mechanism. Identification of inflammatory associations may therefore improve understanding of PSA variability in benign prostate disease. However, CRP should not currently be considered a substitute for established methods of prostate cancer evaluation. Because CRP is nonspecific and may be elevated in many infectious, inflammatory, metabolic, and malignant conditions, an elevated CRP level cannot be used to attribute an elevated PSA specifically to BPH-related inflammation [15,18].

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The potential therapeutic relevance of these findings is also noteworthy. Increasing evidence suggests that persistent inflammation may represent a therapeutic target in BPH and LUTS. Reviews have described several possible anti-inflammatory approaches, including pharmacological agents and phytotherapeutic interventions, although the strength of evidence varies considerably among treatments [20]. A pilot study evaluating urinary and serum CRP in men with BPH also demonstrated that urinary CRP could be detected and that its level changed following treatment, supporting further investigation of inflammatory biomarkers in BPH. Nevertheless, the present study was observational and did not evaluate treatment response; therefore, the observed PSA–CRP correlation should not be interpreted as evidence that anti-inflammatory treatment will reduce PSA or modify the progression of BPH.

The present study has several strengths. First, a relatively large sample of 347 patients was included, providing adequate observations for assessment of the primary correlation. Second, participants with active infection, previous malignancy, recent steroid or anti-inflammatory drug use, urethral stricture, and previous prostate or bladder surgery were excluded to reduce some important sources of variation in PSA and CRP. Third, PSA and CRP measurements were performed using laboratory-based biochemical assessment, and laboratory personnel were blinded to the clinical information. Finally, the relationship was additionally examined across clinically relevant subgroups, including age, BMI, comorbidities, and prostate size.

Several limitations should also be considered when interpreting the findings. The cross-sectional design permits identification of an association but does not establish a temporal or causal relationship between CRP and PSA. It cannot be determined whether inflammation contributes to PSA elevation, whether both markers are influenced by another underlying factor, or whether higher PSA is associated with inflammatory changes through another mechanism. Furthermore, serum CRP is a nonspecific systemic inflammatory marker and does not directly demonstrate inflammation within the prostate. Histological confirmation of prostatic inflammation was not performed because the participants were managed as patients with clinically diagnosed BPH. In addition, prostate size was assessed by abdominal ultrasonography rather than by more precise volumetric methods, and factors such as smoking, dietary factors, physical activity, obesity-related inflammation, and other systemic inflammatory conditions may influence CRP levels.

Another important limitation is that the study was conducted at a single hospital and used consecutive non-probability sampling. Consequently, the findings may not be directly generalizable to all men with BPH in other populations or healthcare settings. The use of a PSA threshold of >4 ng/mL also provides a convenient categorical classification but does not account for age-specific PSA interpretation or other contemporary approaches to PSA assessment. Similarly, CRP was analyzed both as a continuous variable and using a predefined cutoff; different CRP thresholds may produce different categorical results. Future studies should consider larger multicenter populations, standardized assessment of prostate volume, detailed assessment of LUTS, and direct histological or molecular measures of prostatic inflammation.

CONCLUSION

This study demonstrated a statistically significant but weak positive correlation between serum PSA and CRP levels among patients with benign prostatic hyperplasia ($r = 0.28$, $p < 0.001$), suggesting that increased systemic inflammatory activity may be associated with higher PSA concentrations in men with BPH. The positive relationship persisted across different age groups, BMI categories, comorbidities, and prostate sizes, with relatively stronger correlations observed among older patients and those with larger prostates. However, because CRP is a nonspecific marker of systemic inflammation, the observed association does not establish a causal relationship or confirm localized prostatic inflammation. Further prospective multicenter studies using prostate-specific inflammatory biomarkers and

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histopathological assessment are recommended to clarify the role of inflammation in PSA elevation and BPH progression.

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