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ORIGINAL ARTICLE

Prostatic Inflammation's Effects on Individuals with Benign Prostate Hyperplasia's Clinical Consequences

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ABSTRACT

Objective: In order to ascertain the impact of silent inflammatory prostatitis on the clinical results of individuals receiving trans urethral prostate resection owing to benign prostatic hyperplasia.
Study Design: Retrospective study
Place and Duration: This retrospective study was conducted at Liaquat National Hospital Karachi in the period from April, 2022 to September, 2022.
Methods: Total 330 patients were presented in this study. In this study, individuals over 45 years without a history of urologic surgery who presented to a urology clinic with lower urinary tract symptoms related to benign prostatic hyperplasia were included. Pathological outcomes and clinical indicators were evaluated before and one year after surgery. Mean Standard deviation was used to present data and categorical variables were assessed by frequencies and percentages.
Results: Mean age of the patients was 61.3±10.42 years. Among 330 cases, 200 (60.6%) patients were had benign prostatic hyperplasia and 130 (39.4%) cases had both prostatic inflammation (category-IV) and benign prostatic hyperplasia. Before TURP, mean prostate volume in BP patients was 55.7±8.11 m^3 and in BPH/PI patients was 55.3±10.6 m^3 and after TURP mean prostate volume in BP patients was 23.2±1.20 m^3 and in BPH/PI group 23.4±8.17 m^3. We found that people with prostate inflammation had lower Qmax values and a higher preoperative prostate score compared to those who had no signs of inflammation before to prostate transurethral resection.
Conclusion: In this study, we came to the conclusion that in individuals with benign prostatic hyperplasia, prostate inflammation without symptoms might worsen lower urinary tract symptoms and the rate of urine flow.
Keywords: Benign Prostatic Hyperplasia (BPH), Prostate Pathology, Prostatic Inflammation

INTRODUCTION

The most frequent condition treated in urology is BPH. According to an examination of postmortem cases, BPH is more common as people become older. It is histologically present in around 40% of patients in their 50s, >70% of patients in their 60s, and over 90% of patients over the age of 80. [1] Some individuals, however, may not have LUTS while having histological proof of BPH. According to 11 cross-sectional population studies, males in their 50s and 70s are more likely than women to experience moderate-to-severe LUTS at a rate of 29% and 56%, respectively. [2] According to these findings, patients with histological BPH are not always consistent with LUTS symptoms.

In adult men, CP is also a frequent condition. CP is a syndrome that is often caused by a number of underlying reasons and is not a single, isolated ailment. Although prostatitis was traditionally believed to be an illness that only affected young men, a recent study found that the incidence of prostatitis in men over the age of 50 (8%) is almost as high as that in younger men under the age of 50. [3]

Despite the fact that the pathophysiology of benign prostatic hyperplasia (BPH) is still not fully understood, there is some evidence to suggest that prostatic inflammation may play a significant role in prostate enlargement and the development of BPH. On biopsy of surgical specimens from patients with or without lower urinary tract symptoms (LUTS) or prostatitis, the presence of persistent histologic inflammation in the prostatic tissue is a frequent observation. [4] Although the timing and causes of persistent inflammation are still unknown, it has been proposed that BPH is an immune-mediated inflammatory illness [4]. It has been demonstrated that a number of growth factors and cytokines, including T and B lymphoid cells and macrophages, are engaged in the inflammatory process as well as the interactions between epithelial and stromal prostatic cells. [5]

Numerous research have looked at the connection between LUTS-related BPH and histologic prostatic inflammation. [6] When compared to individuals who did not have inflammatory infiltrates in the prostate at baseline, those with chronic infiltrates had a greater risk of BPH development and acute urine retention. Depending on how prostatic inflammation and LUTS are connected, anti-inflammatory drugs may be a unique therapy option for the management of LUTS caused by BPH.

Lower urinary tract symptoms (LUTSs) in males are most frequently brought on by BPH. Urination and storage symptoms are LUTSs that have a significant impact on quality of life [7]. Additionally, BPH raises the chance of urinary tract infections (UTI) and severe urine retention (AUR). Changes in androgen levels and tissue remodelling brought on by ageing have been regarded as significant contributors to homeostasis disturbances in the prostate, despite the fact that the cellular and molecular causes of BPH remain unknown [8]. Additionally, some data suggests that the development and progression of BPH/LUTS may potentially be influenced by metabolic syndrome and chronic inflammation [9].

The pathogenesis of BPH is still poorly known, despite its high incidence and socioeconomic effect. For instance, it is still entirely unclear why some men produce a 40-g prostate while others get a 200-g prostate. Here, we go through some of the pathophysiology (with a focus on the androgen route) and clinically significant features of BPH and BPE. In addition to BPH/BPE, there are several other pathomechanisms that contribute to LUTS, including receptor status of the anticholinergic system, pelvic ischaemia, alterations in the urothelium and bladder ultrastructure, and many more [10].

The purpose of the current study was to mimic the impact of prostatic inflammation on the clinical parameters and outcomes of patients who have prostate TURP due to benign prostatic hyperplasia.

MATERIAL AND METHODS

This retrospective study was conducted at Liaquat National Hospital Karachi in the period from April, 2022 to September, 2022

and comprised of 330 patients. Patients with positive urine tests, persistent pelvic pain complaints that met the criteria set out by the NIH, bladder stones, neurological conditions, prostate cancer, and urethral stenosis were excluded.

After scanning patient data, patients, together with histopathological findings and clinical information, were included in the research. Patients over fifty without a history of urologic surgery who have lower urinary tract symptoms related to benign prostatic hyperplasia are admitted to the urology clinic. The diagnosis of benign prostatic hyperplasia was made using a digital rectal examination, transrectal guided ultrasonography, and symptoms related to micturition. Except for those who had urethral catheters, all patients had at least three months of alpha blocker medication prior to transurethral resection. All physical examinations and the patient's full medical history were reviewed. Transrectal ultrasound can be used to determine the prostate's volume. International prostate symptoms score (IPSS) questionnaires were administered before to TURP and after transurethral resection for a period of one year. evaluated transurethral resection sample data to identify prostate inflammation. According to the histological categorization system of prostate inflammation described by Nickel et al., infiltration of inflammatory cells inside benign prostatic hyperplasia tissue was characterised.

The normality test was carried out utilizing the Kolmogorov-Smirnov test. All analysis was done using SPSS 22.0. the mean and standard deviation for demographic variables. All tests were 2talied and p-valued for statistical significance.

RESULTS

Among 330 cases, 200 (60.6%) patients were had benign prostatic hyperplasia and 130 (39.4%) cases had both prostatic inflammation (category-IV) and benign prostatic hyperplasia.(figure-1)

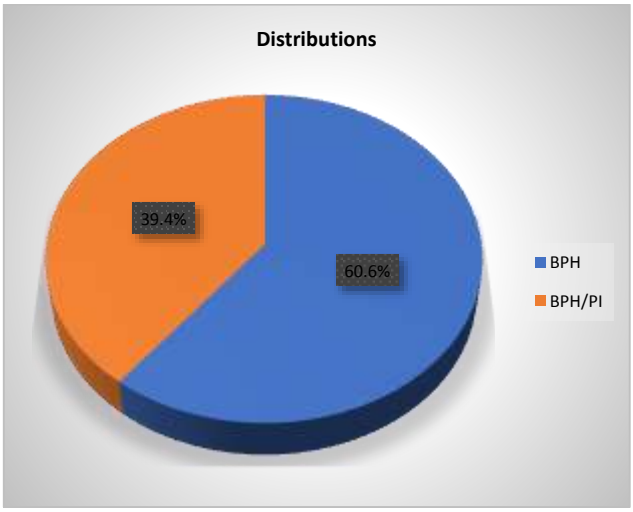

Figure-1: Distribution of cases with and without prostatic inflammation

Table-1: Characteristics of the enrolled cases

Variables	BPH (200)	BPH/PI (130)
Before Operation		
Mean Prostate voiding residue (ml)	64.3±13.10	70.2±11.23
Mean Prostate volume (m3)	55.7±8.11	55.3±10.6
After Operation		
Mean Prostate voiding residue (ml)	14.9±5.13	16.3±8.23
Mean Prostate volume (m3)	23.2±1.20	23.4±8.17
Catheterization		
Yes	20 (10%)	23 (25.4%)
No	180 (90%)	107 (74.6%)

Mean age of the patients was 61.3±10.42 years. Before TURP, mean prostate volume in BP patients was 55.7±8.11 m^3 and in BPH/PI patients was 55.3±10.6 m^3 and after TURP mean prostate volume in BP patients was 23.2±1.20 m^3 and in BPH/PI group 23.4±8.17 m^3. (table 1)

We found that people with prostate inflammation had lower Qmax values and a higher preoperative prostate score compared to those who had no signs of inflammation before to prostate transurethral resection.(table 2)

Table-2: Scores from IPSS and Qmax are compared.

Variables	BPH (200)	BPH/PI (130)
Early before TURP		
Qmax values (ml/s)	8.5±1.3	6.9±4.37
IPSS	21±2.9	29±6.8
After 1-years		
Qmax values	13±6.13	10±2.6
IPSS	16±0.7	15±5.18

We found adverse outcomes in 22 (6.7%) cases after TURP.(table 3)

Table-3: After-surgery adverse outcomes

Variables	Frequency	Percentage
Adverse Events		
Yes	22	6.7
No	308	93.3
Types of Complications		
Urinary tract infection	12	3.6
Urinary retention	6	1.8
Transient hematuria	4	1.2

DISCUSSION

The connection between BPH and prostate inflammation has been the subject of various investigations in recent years. There is a clear correlation between the levels of inflammation and LUTS, according to epidemiological and histological investigations. Prostate inflammation is not only a common observation in BPH but also a major contributor to the expansion of prostatic cells. [11,12]

Although men with chronic prostatitis frequently get anti-inflammatory and antibacterial medication, researchers discovered that leukocyte and bacterial counts, as they defined them, did not correspond with the severity of symptoms in a prior study. [13] It explains why counting white blood cells cannot be used to identify prostate inflammation. However, in another research, BPH specimens that had been surgically removed had histology that showed the presence of inflammatory cells and proinflammatory cytokines such tumour necrosis factor-alpha (TNF-a), interferon-mRNA, and interleukins (IL-2, IL-4, IL-6, IL-7, IL-8). [14] NIH category IV asymptomatic inflammatory prostatitis, a high prevalence of prostatic calcification, and considerably higher TNF-a expression in patients with obstructive BPH were all shown to be directly related by Engelhardt et al. [15] In this study, inflammation was identified by examining inflammatory cells in BPH specimens that had been surgically removed.

In current study 330 patients were presented. Mean age of the patients was 61.3±10.42 years. Among 330 cases, 200 (60.6%) patients were had benign prostatic hyperplasia and 130 (39.4%) cases had both prostatic inflammation (category-IV) and benign prostatic hyperplasia. These results were comparable to the previous study.[16] The prostate volume among patients in the current study did not reach statistical significance according to Nickel's study. Chronic prostatic inflammation was found in a sizable patient group in the Nickel research at a rate of 77.6%. [17] Our study has shown that there is a significant difference in the total IPSS and Qmax scores at baseline and one year after surgery between individuals with and without prostate inflammation. Prostatic inflammation is a common finding in patients with benign prostatic hyperplasia. [18] Our study demonstrates that patients with benign prostatic hyperplasia/prostate inflammation had a

higher rate of pre-operative urine catheterization than patients in the benign prostatic hyperplasia alone group. Cytokines released by inflammatory cells may be to blame.

Prostate tissue obtained from transurethral resection of the prostate was evaluated for extent, anatomical position, and histological categorization system. According to other research, the majority of prostatic inflammation was stromal, localised, and moderate. According to Nickel et al.[18], periglandular inflammation was the most prevalent pattern. In 93 patients who underwent transurethral resection of the prostate without showing signs of clinical prostatitis, it made up 0.5% of all glandular volume in their surgical specimen. [19] According to Okada et alstudy[20], .'s individuals with acute histological prostatitis had high blood levels of prostate-specific antigen, whereas those with chronic histological prostatitis had levels that were normal with a preponderance of mononuclear cells. Our findings supported the finding that the prostate specific antigen level was greater in the group of patients with histological prostatic inflammation than in the group of individuals without inflammation. Statistics showed that even the slight variations mattered. We found adverse outcomes in 22 (6.7%) cases after TURP.

CONCLUSION

In this study, we came to the conclusion that in individuals with benign prostatic hyperplasia, prostate inflammation without symptoms might worsen lower urinary tract symptoms and the rate of urine flow.

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