ORIGINAL ARTICLE

The Prostatic Inflammation Effect on Clinical Examination of patients Whom UndergoneProstate Transurethral Resection (TURP) due to benign Prostatic Hyperplasia

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ABSTRACT

Aim: To examine asymptomatic inflammatory prostatitis affects on patients clinical consequences experiencing transurethral resection of the prostate because of prostatic hyperplasia (BPH).

Method: This retrospective study was carried out during 1st July 2016 to 31st December 2017 at Bolan Medical Complex Hospital, Quetta and included 950 patients during this period. Before and one year after surgery, pathological results and clinical parameters were compared. The patients with lower urinary tract due to benign prostatic hyperplasia admit to urology clinic, over the fifty years of age, without past urologic surgery was included in this study. The patients having +ve urine results, chronic pelvic pain symptoms resulted in accordance with NIH rating, bladder stone, neurological disease, prostate cancer and urethral stenosis were excluded.

Results: Six hundred and five patients were identified with only benign prostatic hyperplasia and remaining 345 patients were indentified with both benign prostatic hyperplasia (BPH) and prostatic inflammation (category-IV). Between two groups there was found no statistical significance among limitation/variables comprising prostate volume, post evacuate remnant (p value = >0.05) and age. Prostate inflammation's patients presenting lower Qmax values and higher pre-operative prostate score as compared to patients had not found inflammation before prostate transurethral resection.

Conclusion: In benign prostatic hyperplasia patients, prostate inflammation with no symptoms can cause to deteriorate lower urinary tract symptoms and rate of urinary flow

Keywords: Prostatic Inflammation, Prostate Pathology, Benign Prostatic Hyperplasia (BPH),

INTRODUCTION

Hyperplasia in glands and stroma is explained as benign prostatic hyperplasia (BPH), and it is mostly found in male population having age above than fifty years.[1] BPH is distinguished by lower urinary tract symptoms (LUTS). Lower urinary tract symptoms resulted due to numerous disorders but the major reason found is benign prostatic enlargement and about fifty percent of male population having ages >50 years, believe to experience lower urinary tract symptoms[2-4].

Benign prostatic hyperplasia/lower urinary tract symptoms are probably causes to heavy load on health care systems, this condition pathogenesis are still unknown largely. There are several factors probably involved in development as well as prostate enlargement progression. There are many studies histopathological and epidemiological have indicate the role of prostate inflammation in benign prostatic hyperplasia and lower urinary tract symptoms pathogenesis.[5] Prostate inflammation histological evidences examined in patients having benign prostatic hyperplasia which did not have prostatitis symptoms. Without symptoms prostate is confirmed in NIH (national institute of health) prostatitis grouping, type-IV, and has been noticed in forty three% to 98% of surgically extracted prostatic tissues detached for benign prostatic hyperplasia.[6] Nickel et al[7] proposed a standard classification system of chronic pelvic pain syndrome and chronic prostatitis can be used in prostate biopsies for examination of prostatic inflammation,prostatectomy specimens or transurethral extracted prostatic tissues. The objective of current research was to emulate the prostatic inflammation effect on clinical parameters of outcomes of patients who undergo prostate TURP because of benign prostatic hyperplasia.

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MATERIAL AND METHODS

The patients were examined who undergo transurethral resection of prostate due to benign prostatic hyperplasia retrospectively during the period from 1st July 2016 to 31st December 2017 at Bolan Medical Complex Hospital, Quetta. A total 950 patients along with histopathological results and clinical information were included in the study after scanning data of patients. Lower urinary tract patients due to benign prostatic hyperplasia admit to urology clinic, over the fifty years of age, without past urologic surgery were included in this study and those patients were excluded from study whose urine culture was positive, chronic pelvic pain syndrome diagnosed in accordance with National Institute of Health classifications group/type-III), bladder stone, neurological disease, prostate cancer. According to these criteria after screening medical data, 605 patients defined as benign prostatic hyperplasia. Among these patients, 161(16.95%) patients undergo catheterization because of severe urinary retention, and 345(36.36%) patients had prostatic inflammation (prostatitis category-IV). With the help of digital rectal examination transrectal guided ultrasound and micturition symptoms the benign prostatic hyperplasia was diagnosed. Before transurethral resection all patients received at least three months alpha blocker therapy except those patients with

urethral catheter. Patients detailed history and all the physical tests were examined. Calculate the volume of prostate through transrectalultrasonography. Before TURP and one year after transurethral resection, international prostate symptoms score (IPSS) was surveyed through questionnaire. Determined prostate inflammation through samples evaluations obtained from transurethral resection. Infiltration of inflammatory cells within benign prostatic hyperplasia tissue was defined in accordance with histopathological classification system of prostate inflammation as reported by Nickel t al.

By using Kolmogorov Smirnov test, normality test was performed. SPSS used to perform all analysis. Demographic values presented as mean with SD. For statistical significance all tests were 2talied and p value <0.5 was considered significant.

RESULTS

In this study 950 patients were included of both genders. All patients undergo transurethral resection of prostate (TURP). Out of 905 participants, 605 patients were identified only had benign prostatic hyperplasia and the remaining 345 hadfound with benign prostatic hyperplasia and prostatic inflammation, category-IV, both. In 68 (7.16%) patients with only benign prostatic hyperplasia inserted a urethral catheter and 93 (9.79%) patients with benign prostatic hyperplasia/prostatic inflammation respectively. The patients were between the age range of >49 years to 80 years, 58ml (32-91) was the average prostate volume. 905individualshad undergone transurethral resection of prostate (TURP) procedure. 63.4±23.6 minutes were the mean operation time and 3.6±1.2 days was the average catheterization time. There were postoperative complications in 72 patients (30 patients transient hematuria, 24 patients had urinary retention resolved by recatheterization and urinary tract infection found in 18 patients).

Patients with prostate inflammation presented worse international prostate symptom score (IPSS) preoperatively & lower Qmax scores as compared to those with no inflammation before transurethral resection of the prostate (TURP), 30.8±10.4 versus 22±4.2 and 7.1±2.8 versus 9.3±2.8 respectively, p value < 0.05. In total international prostate symptom scores and Qmax scores statistically significant differences were found one year after transurethral resection of prostate, in the same way, p value < 0.05.

Whether depending on patients were catheterized pre-operatively or not, they were categorized into 2 groups. After that these two groups further catagorized into two groups according to having prostatic inflammation or not. There was no statistical difference was found between patients according toPV, age and post voiding residue (p value >0.05). High pre-operative urethral catheterization rate was presented by patients with prostate inflammation when compared to those with no inflammation. 93 patients among 345 patients with benign prostate hyperplasia/prostate inflammation versus 68 of 605 patients with only benign prostate hyperplasia, 26.96%, 11.24% respectively (p value < 0.05) as shown in table-I. Overall, 605 (63.68%) of 950 patients showing no sign of prostate inflammation at histology report, while 345 (36.36%) of 950 patients presented inflammatory infiltrate. Mild inflammation in 170 (49.28%) of 345 patients, in 140 (40.58%) of 345 patients it was moderate and severe in 35 (10.14%) of 345 patients according to histopathological classification system. Prostate inflammations' anatomic location within stromal 60, 90 glandular and 95 patients periglandular. Regarding nflammation infiltration extent, focal region infiltration occur in 180 (52.17%) patients, in 80 (23.18%) patients multifocal zone infiltration take place and in 85 (24.64%) patients there was diffused are infiltration occurred as shown in Table 4.

Table 1: Patients baseline characteristics before and one year after transurethral resection of the prostate

Variable	Benign prostate Hyperplasia **(N=605)**	Benign Prostate hyperplasia/Prostate inflammation (Category IV) **[N=345]**	P value
Before Transurethral Resection of Prostate (TURP)			
Age (years)	64.8±7.8	66±8.5	0.81
Serum prostate specific antigen (ng/dl)	2.4±1.7	3.7±4.5	0.001
Prostate volume (m^3)	56.4±12.4	56±7.9	0.75
Prostate voiding residue (ml)	65.3 (35.1-92.4)	71.9 (360-100.3)	0.48
TURP Findings after 1 years			
Prostate volume (m^3)	24.7±8.4	24.3±7.2	0.64
Prostate voiding residue (ml)	13.8±3.2	15.0±4.1	0.67
Catheterization/Severe Urinary Retention	68 (11.24%**)**	93 (26.96%)	0.0002

DISCUSSION

The relationship between BPH(benign prostatic hyperplasia) and prostatic inflammation have examined in many studies from the last few years. In benign prostatic hyperplasia, prostate inflammation is not only a common finding but also play a key role in overgrowth of prostatic cells and there was direct association exists between inflammation degrees and lower urinary tract symptoms (LUTS) as indicated by histopathological and epidemiological studies[8-10].

Although men received routinely anti-inflammatory and anti-microbial therapy who have chronic prostatitis, leukocytes and bacterial count do not correlate with symptoms severity as they defined them.[11]Proinflammatory cytokines such as interleukins (IL2, IL4, IL6, IL7, IL8, IL15, IL17), TNF-a (tumor necrosis factor-alpha) and inflammatory cells have been diagnosed on histopathology of benign prostatic hyperplasia resected portion as shown

in another study.[12]Engelhardt et al[13] reported that there is a direct relationship between asymptomatic inflammatory prostatitis National institute of health category-IV, prostatic calcification of high incidence and significantly superior TNFa expression in patients with obstructive benign prostatic hyperplasia. Through investigating inflammatory cells in benign prostatic hyperplasia resected specimens, we diagnosed inflammation in this present study.

Table 2:Comparison of IPSS and Qmax Scores

Scores(N=848)	Benign prostate hyperplasia (n=538)	Chronicprostatitis /Benign prostatichyperplasia (n=310)	P value
Before TURP			
IPSS total score	22±4.2	30.8±10.4	0.0001
Q max (ml/s)	9.3±2.8	7.1±2.8	0.0001
TURP findings after 1 year			
IPSS value decrease	17.1±3.8	14.3±3.7	0.001
Q max value increase	12.7±4.8	9.9±3.6	0.001

Table.3:Post-operative complications found in patients

Complications	No.	%
Transient hematuria	30	3.15
Urinary retention	24	2.53
Urinary tract infection	18	1.89

Table-4: Prostatic Inflammation Histopathological Characteristics

Characteristics	Benign Prostatic Hyperplasia (Category IV)
Anatomical location	
Glandular	85 (24.64%)
Periglandular	120 (34.78%)
Stromal	140 (40.58%)
Inflammation infiltration extent	
Focal	180 (52.17%)
Multi-focal	80 (23.18%)
Diffuse	85 (24.64%)
Grade	
Mild	170 (4.28%)
Moderate	140 (40.58%)
Severe	35 (10.14%)

In present study, among patients there was no statistical significance found in prostate volume as go through the Nickel's study. In study of Nickel, chronic prostatic inflammation at a rate of 77.6% was detected in a large cohort of patients.[14]Hu et al[1] reported in his study that patients had typically larger prostate volumes who are in the group of benign prostatic hyperplasia/asymptomatic prostate than the group of only benign prostatic hyperplasia. Huet al[1] also concluded that neither the rate of Qmax nor density of serum PSA demonstrate a difference significantly among two groups. Total scores of international prostate symptoms scores (IPSS) demonstrate a significant difference among benign prostatic hyperplasia/prostate inflammation and separate groups of benign prostatic hyperplasia shown in present study.

It is confirmed from our research that prostatic inflammation commonly found in benign prostatic hyperplasia patients significant difference pertaining to baseline and one year after post-operative total IPSS and Qmax score examined among patients with prostate inflammation and without prostate inflammation. Our research shows that benign prostatic hyperplasia/prostate inflammation patients had high rate of pre-operative urinary catheterization as compared to patients with only benign prostatic hyperplasia group. It may because of cytokines secretion from inflammatory cells.

Evaluation of extent, anatomical location of histopathological classification system and prostate tissue obtain from transurethral resection of prostate was done. Other study resulted that stromal, focal and mild prostatic inflammation had accounted for majority. Periglandular inflammation was the most general pattern as demonstrated by Nickel et al[14], and in 93 patients' surgical specimen, it constitute 0.5% of all glandular volume, who undergo to transurethral resection of the prostate without clinical prostatitis.[1]

The level of prostate specific antigen increases in symptomatic prostatitis' patients.16 However, effects on serum prostate specific antigen level by inflammatory prostatitis is under discussion. There was no association between prostate volume, serum prostate specific antigen level and inflammation pattern as reported by Nickel et al.15 Moreover, Morote et al17 did not found any significant difference statistically in serum prostate specific antigen levels among patients with prostate inflammation and without prostate inflammation.17 Similarly, Okada et al18 reported in his study that in patients with acute histological prostatitis the levels of serum prostate specific antigen were high and in patients with chronic histological prostatitis the levels of serum prostate specific antigen were normal with mononuclear cell predominance.Our results confirmed that prostate specific antigen level was higher in histological prostatic inflammation patients group as compared with those without inflammation. The small differences were significant statistically.

CONCLUSION

The outcomes of our study recommended that in patients with benign prostatic hyperplasia the prostatic inflammation may lead to adverse Qmax and IPSS scores. Further, it can also cause higher levels of prostatic specific antigen in patients having benign prostatic hyperplasia/prostate inflammation. However, no significant differences were observed among two groups pertaining to prostate volume. We feel that this condition can helpful to recognized the effects of prostatic inflammation on adverse Qmax and IPSS scores independent of prostate volume. But it is still not clear that how inflammation causes signs/symptoms of urinary tract such as high IPSS score, lower flow rate and acute urinary retention. Moreover, we have to do more work regarding this malignant disorder to overcome the complication, mortality and morbidity rate.

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