JANUARY - MARCH 2015

MEDICAL CHANNEL

Original Article

URINARY RETENTION INCIDENCE IN PATIENTS OF BENIGN PROSTATIC HYPERPLASIA WITH UNDERLYING PROSTATITIS

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ABSTRACT

Objective: To compare, the clinical features of the patients having urinary retention and benign prostatic hyperplasia (BPH) with those having histological prostatitis.

Methods: The clinical data and histopathology reports of patients with BPH for TURP presented with or without urinary retention admitted to Armed Forces Institute of Urology Rawalpindi between MAY 2013 and NOV 2013 were evaluated. Statistical analysis was performed with Student's t-test, Mann–Whitney test and Fisher's exact test where appropriate and $P < 0.05$ was considered to indicate statistical significance.

Results: Prostatic histology was available in 186 patients, 94 with AUR and 92 without AUR, 38(24 with AUR and 12 without AUR) prostatitis.

Comparing those with BPH+AUR and those with BPH and no AUR showed statistically significant differences in mean age (69.5 vs 71.9 years), serum prostate-specific antigen (PSA) level (18.6 vs 899.5 ng/mL) and histological prostatitis (48 % vs 25%) but not duration of catheterization, prostate volume or urinary tract infection (UTI).

Comparing those with BPH only and those with BPH plus prostatitis showed significant differences in mean age (71.9 vs 67.1 year) and PSA level (14.6 vs 22.8 ng/mL) but not prostate volume, UTI or duration of catheterization.

Conclusion: Histological prostatitis was almost half as common in patients with urinary retention associated with underlying BPH than in patients with only BPH and presented with AUR, but there was no significant difference in the duration of catheterization, prostatic volume or presence of UTI, suggesting that histological prostatitis contributes to the development of retention in patients with underlying BPH than in those without prostatitis. In patients with BPH, histological prostatitis was associated with urinary retention at a significantly younger age. The association of prostatitis and AUR should be considered in secondary prevention for BPH patients.

Keywords: AUR, Acute urinary retention, CUR, Chronic urinary retention

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INTRODUCTION

BPH is a disease of ageing men[1]. It is reported that an estimated 70% of men

aged 61-70 years and 90% of those aged 81-90 have pathological BPH.[2.] The prevalence of LUTS associated with BPH parallels that of pathological BPH; > 50% of men over 50 are believed to experience LUTS secondary to an enlarged prostate gland.

Prostatitis, a histological diagnosis, has evolved over the years to describe a clinical syndrome that was believed to be associated with prostatic inflammation. It is well-recognized by both urologists and pathologists that BPH and prostatitis can coexist[3,4] .Twenty percent of patients with LUTS associated with BPH might also have symptoms suggestive of prostatitis. [5,6.] Little attention has been given to the association between BPH and prostatitis, despite the high prevalence of both conditions in ageing men.

BPH would present with LUTS. The progression of LUTS can result in AUR[5].Other common causes of retention include BPH, adenocarcinoma of the prostate (ACP), urethral stricture, gross Hamaturia with clot formation, neuropathic bladder dysfunction, constipation and idiopathic [7]. Prostatic infection as well as infarction has been suggested as etiological events [8] .Histological evidence of inflammation has been reported in ?40% of BPH cases and is associated with a significantly higher risk for acute urinary retention (AUR) [9] .A sub analysis of the medical therapy of prostatic symptoms (MTOPS) study suggested that inflammation detected in baseline prostatic biopsies was highly predictive of progression of BPH to AUR and the need for more invasive therapy[10]. Results from the MTOPS study. An examination of baseline prostate biopsies in a subgroup of 1,197 patients in the Medical Therapies of Prostatic Symptoms (MTOPS) study found that there was a chronic inflammatory infiltrate in 43% of the men[11.] Prostatitis has traditionally been considered a condition which inflicts younger men, but it is apparent that it is as common in older men [12].. Compared to men aged 51 and higher the odds of a documented prostatitis diagnosis is only 2-fold greater in younger men[13]. Little attention has been given to the association between BPH and prostatitis, despite the high prevalence of both conditions in ageing men.

Many physicians have trouble clinically distinguishing prostatitis from BPH in the older male population [14] .It was hypothesized that the presence of histological inflammation may be a predictor of progression. There was a very clinically significant difference in the progression rate based on the presence or absence of inflammation. Patients in all groups (placebo, finasteride, doxazosin and combination finasteride and doxazosin) with inflammation were more likely to progress clinically in terms of symptoms, AUR or BPH related surgery. For those with no inflammation, there was overall clinical progression in 13.2% of patients, while 3.9% had BPH-related surgery and none had AUR, corresponding values for those who had chronic inflammation were 21% (P=0.08 vs. no inflammation), 7.3% (not significant) and 5.6% (P=0.003). Chronic inflammation accounted for every AUR event in this subgroup of patients with prostate biopsies, while in patients with no inflammation there was no AUR.[11] If inflammation and BPH had a causal relationship, by derivation the incidence of inflammation would be higher in AUR, the most severe symptom in BPH than in those patients with BPH who do not present with AUR.[5]

The aims of the present study were to compare the clinical features of the patients with BPH who were scheduled to undergo TURP or open prostatectomy having urinary retention with those having histological prostatitis and to determine whether the incidence of prostatic inflammation was significantly different in patients presented with AUR than in those presented with LUTS.

METHODOLOGY

The clinical data of patients with a diagnosis of urinary retention admitted to the, Armed Forces Institute urology Rawalpindi period from May 2013 to Nov 2013 were analyzed. The results of special investigations were obtained from the chemical pathology, microbiology and radiology departments, and histopathology reports on prostatic tissue specimens obtained by transurethral or open prostatectomy were provided by the department of anatomical pathology Armed Forces Institute Of Urology Rawalpindi.

The study included 186 patients (from a cohort of 200 consecutive potentially eligible subjects) with a diagnosis of BPH, but no history or symptoms of prostatitis. Histological sections were obtained from specimens collected prospectively at transurethral resection of the prostate (TURP), immunostained for leukocyte common antigen and scanned using a computerized image-analysis system. Histopathologic prostatic samples from both groups were fixed in formalin and examined by two consultant histopathologists. Sections were stained with hematoxilin and eosin, and examined using light microscopy. Prostatitis was diagnosed by the presence of edema, vascular congestion and numerous neutrophils within prostatic acini infiltrating the lining epithelium and extending to a lesser extent into the surrounding stroma. The studied sample was consisting of 200 patients, of whom complete data was available for 186 patients. The patients who had bladder stones or prostate cancer or complained of prostatitis manifestations or any other pathology of prostate were excluded. Independent variables included age and presence of prostatitis.

The data were entered on an Excel database for statistical analysis with Student's t-test, Mann–Whitney test and Fisher's exact test, where appropriate, using Graph Pad InStat® software. A two-tailed P value < 0.05 was considered to indicate statistical significance. All values are expressed as means or medians (range). The study was approved by the local ethics committee as part of the postgraduate research project of the first author.

RESULTS

There was a total of 186 patients, mean (median, range) age 66.4 (69.1, 12.8–94.7) years. 94(90.7%) patients presented with AUR. The probable cause of retention was BPH.

Prostatic tissue was examined histologically in 186 patients: it was obtained by TURP . Histological evidence of prostatitis was recorded by the pathologist in 38/186 (20%) patients with BPH on histology ($P < 0.001$).

Compared with the group of patients with BPH+AUR, patients with BPH+AUR & prostatitis were statistically significantly younger, had a higher rate of transurethral rather than suprapubic catheterization, a higher mean haemoglobin and a lower proportion of anaemia, a lower mean serum PSA and a lower proportion with PSA level >4 ng/mL, and a higher proportion with histological evidence of prostatitis (Table 1).

The patients with histological BPH only were significantly older than the group with BPH plus histological prostatitis (Table 1). There was no statistically significant difference in prostate volume, the proportion with positive urine culture or duration of catheterization between the two groups.

DISCUSSION

In the present study of patients with urinary retention, the probable cause was BPH in 36.6% and prostatitis in 36.0%. The group with prostatitis was, on average, about 2 years older than the group with BPH only, had significantly lower mean hemoglobin and a higher serum PSA level, with no significant difference in prostate volume. These findings are consistent with the expected association of prostatitis with age, anemia and increased serum PSA level.

In these patients with urinary retention, histological evidence of prostatitis was almost twice as common in specimens with BPH (48%), although there was no significant difference in the duration of catheterization, prostatic volume or presence of UTI. This suggests that prostatic inflammation was not caused by prostatic infection due to catheterization or UTI. It also suggests that concomitant prostatitis more often contributes to the development of retention in patients with underlying BPH.

The cause of prostatic inflammation, previously reported in ?40% of patients with BPH, is unclear [15]. Potential causes include direct infection, urine reflux, dietary factors, estrogens or combinations of these[16]. Patients with intraprostatic inflammation have a significantly higher risk for developing AUR[15] .In the Medical Therapy of Prostatic Symptoms (MTOPS) study, patients with acute or chronic inflammation of the prostate had larger prostates and higher PSA levels and were more prone to progress clinically in terms of symptoms and crossing over to invasive therapy. The risk of AUR in patients with acute or chronic inflammation in the placebo arm was 5.6 vs 0% in those with no evidence of inflammation on biopsy [17].

Tuncel et al[18] found only prostatic inflammation to be a

TABLE 1
COMPARISON OF PATIENTS WITH HISTOLOGICAL BPH ONLY AND THOSE WITH BPH PLUS PROSTATITIS

	BPH only **Mean (range), mean (median, range) or n (%) unless noted otherwise**	**BPH + prostatitis** **Mean (range), mean (median, range) or n (%) unless noted otherwise**	**P value**
Number	148	38	
Age, years	71.9 (45.3–94.7)	67.1 (45.4–87.1)	<0.004
DRE: prostate clinically malignant	4/98 (4.1)	10/87 (11.5)	NS
Prostate volume, Ml	58.2 (20–150)	58.1 (20–220)	NS
Urine culture positive	20/62 (32.2)	22/57 (38.6)	NS
PSA level, ng/mL	14.6 (6.3, 0.1–206)	22.8 (10.8, 1–588)	<0.001
>4	60/93 (64.5)	78/89 (87.6)	0.001
>50	6/93 (6.5)	4/89 (4.5)	NS
Anaemia (Hb <10 g/dL)	5/79 (6.3)	8/68 (11.8)	NS
Renal failure			
(serum creatinine >240 µmol/L)	17/98 (17.3)	10/86 (11.6)	NS
Median (range) duration of			
catheterization, days	51 (1–345)	62.5 (1–421)	NS

contributory risk factor for AUR (3.03 times relative risk). Increased post void residual volumes could induce symptomatic or non-symptomatic UTI, and consecutive UTIs could lead to prostatic inflammation and PSA levels were not influenced by prostatic inflammation or AUR time. On the contrary Mishra et al.[5.] reported that intraprostatic inflammation appeared to play an important role in the pathogenesis and progression of BPH. The risk of AUR due to BPH was significantly greater in patients with prostatic inflammation, and the association of TURP with inflammation was stronger than that with prostate weight. Acute and chronic inflammation of the prostate can be associated with a marked increase in the serum PSA concentration. Nadler et al.[19.] showed that prostatic inflammation plays an important role in serum PSA elevation in patients without clinically detectable cancer. They found prostate volume and inflammation to be the most important factors contributing to serum PSA elevation, with 23 and 7% contributions, respectively, to the serum PSA variance on simultaneous regression analysis.

The pathophysiology of increased PSA in prostatitis has not been established, but disruption of the natural anatomic and physiological barriers between the prostatic milieu and the bloodstream is likely an important factor. Hasui et al.[20.] described a positive correlation between serum PSA levels and prostatitis (acute and chronic-active) in patients with BPH but no clinical evidence of prostatitis, indicating that the effects of prostatic inflammation on the PSA level might occur in the absence of clinical signs and symptoms of acute prostatitis and found that the extension of the inflammatory process as evaluated by the number of specimens involved was directly related to the elevations of serum PSA levels in asymptomatic patients.

In the present study of patients with retention, those with BPH plus prostatitis were significantly younger and had higher mean serum PSA than those with BPH only, although there was no significant difference in prostate volume, presence of UTI and duration of catheterization. This indicates that in patients with BPH, concomitant prostatitis precipitates retention at a younger age and causes higher serum PSA levels.

The present study has certain limitations. Only patients admitted to the urology wards were included, thus excluding patients with retention who passed a trial without catheter, and those with urethral strictures who were treated with dilatation or urethrotomy as outpatients. Thus, the prevalence of BPH and urethral stricture in our referral population could have been under-estimated. Histological examination of prostatic tissue was not performed by a single pathologist, and histological data analysis was based on the written reports of various pathologists. Thus, there could be under-reporting of inflammatory changes as the extent or grade of inflammation was not quantified.

In conclusion, patients with BPH plus prostatitis were younger and had a higher mean PSA level than those with BPH only, but there were no significant differences in prostate volume, duration of catheterization or presence of UTI. This indicates that in patients with BPH, concomitant prostatitis precipitates retention at a younger age and causes higher serum PSA levels.

CONCLUSION

Histological prostatitis was almost half as common in patients with urinary retention associated with underlying BPH than in patients with only BPH and presented with AUR, but there was no significant difference in the duration of catheterization, prostatic volume or presence of UTI, suggesting that histological prostatitis contributes to the development of retention in patients with underlying BPH than in those without prostatitis. In patients with BPH, histological prostatitis was associated with urinary retention at a significantly younger age. The association of prostatitis and AUR should be considered in secondary prevention for BPH patients.

ACKNOWLEDGEMENT

We would like to thank all staff of AFIU who helped us in every field .

CONFLICT OF INTEREST

None declared.

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