150

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FACTORS DETERMINING THE AMOUNT OF RESIDUAL URINE IN MEN WITH BLADDER OUTLET OBSTRUCTION: COULD IT BE A PREDICTOR FOR BLADDER CONTRACTILITY?

Hypothesis / aims of study

The natural history of untreated bladder outlet obstruction (BOO) has been explained by the concept of detrusor compensation to outlet resistance followed by eventual
Decompensation [1]. Post-voiding residual urine (PVR) starts to build up in BOO. Whether the etiology of the progressive build up of post-voiding residual urine is related to bladder failure or increased outlet resistance or both; still a debate among investigators[2]. The aim of our study is to find a urodynamic explanation for the increasing residual urine in males with bladder outlet obstruction. The second aim to know whether we can predict bladder contractility from the amount of residual urine or not

Study design, materials and methods

A retrospective analysis of the pressure flow (P/F) urodynamic studies of 90 consecutive male patients with bladder outlet obstruction was done. All patients should score > 7 in the IPSS (International Prostate Symptom Score) to be diagnosed as having BOO. Patients with neurogenic diseases or diabetes mellitus were excluded from the study to rule out detrusor underactivity.
In all patients, we studied bladder capacity, compliance, detrusor overactivity, voided volume, Q-max, opening pressure, Pdet Qmax, duration of contraction, bladder contractility index (BCI) and urethral resistance factor (URA).
ROC curve and scattered blots showed that using 450 ml as a cutoff value carried the best sensitivity and specificity to detect start of decline of bladder contractility (98% and 68.9% respectively) with a positive predictive value of 85% and a negative predictive value of 92.2% and overall accuracy of 87.7% [Fig 1 & 2]. Nine patients could not void in the presence of the urodynamic catheter and the remaining 81 patients were divided into 3 groups according to the amount of residual urine. Group A (30patients) included patients with residual urine < 100 ml, Group B (30 patients) included those with residual from 100 - 450 ml, while Group C (21 patients) included those with residual urine > 450 ml.

Results

Figure 1: The ROC curve for PVR and BCI

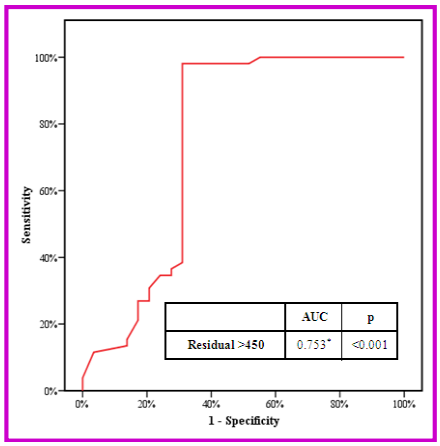

Figure 2: Scattered plots for BCI and PVR

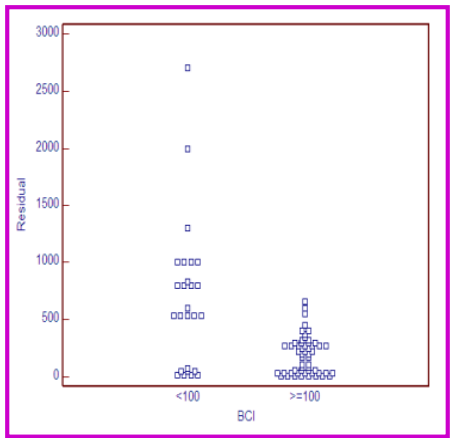

Table 1: Results of different urodynamic parameters in the 3 study groups

	Group A PVR<100	Group B PVR 100-450	Group C PVR>450
Pdet Qmax (cmH2O)	78.3 ± 42.1	104.5 ± 55.3	69.3 ± 16.3
p_1		**0.028***	0.348
p_2			**0.018***
Opening Pressure (cmH2O)	81.7 ± 55.4	116.2 ± 61.9	83.9 ± 47.5
p_1		**0.010***	0.114
p_2			**0.041***
Duration of Cont. (sec)	51.2 ± 26.7	74.4 ± 45.8	68 ± 53.4
p_1		**0.020***	0.455
p_2			0.466
BCI	113.8 ± 26.5	153.5 ± 43.6	82.8 ± 16.6
p_1		**<0.001***	**<0.001***
p_2			**<0.001***
URF	30.6 ± 5.7	67.2 ± 35.3	77.5 ± 26.1
p_1		**<0.001***	**<0.001***
p_2			**0.094**

Interpretation of results

Mean patient age was 61.6 ± 11.9 and the cause of infravesical obstruction was benign prostatic hyperplasia in 76 patients and bladder neck obstruction disease in 14 patients. Analysis of the results of the filling phase of urodynamic studies showed that bladder capacity is increasing with the increase in the amount of residual urine among the 3 groups. We also found that detrusor compliance was low in all groups but with no statistically significant difference. Regarding detrusor overactivity, it was significantly lower in Group C than groups A &B (p=0.048) [Table 1].

Results of the voiding phase showed that voided volume and Qmax decrease significantly as the residual urine increase (p< 0.001 & p=0.015 respectively). Urethral resistance was increasing from group A to B to C as showed by the significant increase in URA in group B above that in group A (p= 0.005) and the further increase in group C above both group A&B (p< 0.001 & p=0.096 respectively). Pdet@Qmax was significantly high in group B followed by group A then group C (p=0.028 & p=0.018 respectively). A similar observation was detected in the opening pressure; where it was significantly higher in group B than group A and group C (p=0.01 & p=0.04 respectively). A very interesting observation was that group C had the lowest bladder contractility index (BCI) with a statistically significant difference with both group A and B (p <0.001). On the other hand, group B had the highest BCI above both group A and C with a statistically significant difference (p<0.001). Additionally, group B had the longest duration of contraction with statistically significant difference with group A (p<0.02) but not significant with group C (p= 0.4) [Table 1].

Concluding message

In men with BOO, PVR results from increasing outlet resistance at the start and till a PVR of 450 ml where the bladder reaches its maximum compensation and power of contractility. In volumes > 450 ml, both outlet resistance and bladder failure are working together leading to detrusor decompensation. Amount of residual urine can predict the status of bladder contractility.

References

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2. Abarbnel J, Marcus E (2007) Impaired detrusor contractility in community-dwelling elderly presenting with lower urinary tract symptoms. Urology 69: 436-439.

Disclosures

Funding: None **Clinical Trial:** No **Subjects:** HUMAN **Ethics Committee:** Alexandria Faculty of Medicine Research Ethical Committee **Helsinki:** Yes **Informed Consent:** Yes