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THE EVOLUTION OF DETRUSOR OVERACTIVITY AFTER WATCHFUL WAITING, MEDICAL THERAPY AND SURGERY IN PATIENTS WITH BLADDER OUTLET OBSTRUCTION

COSIMO DE NUNZIO, GIORGIO FRANCO, ANDREA ROCCHEGIANI, FRANCESCO IORI, COSTANTINO LEONARDO AND CESARE LAURENTI

From the "U. Bracci" Department of Urology, "La Sapienza" University of Rome, Rome, Italy

ABSTRACT

Purpose: We analyzed the evolution of detrusor overactivity in patients with bladder outlet obstruction treated with either medical or surgical therapy or watchful waiting.

Materials and Methods: Of 255 patients with symptomatic benign prostatic enlargement who completed the International Prostate Symptom Score and underwent full urodynamic investigation 161 presented with bladder outlet obstruction. Of the 161 men 101 were reevaluated with a second clinical evaluation and urodynamics 1 to 5 years (mean 2) after watchful waiting in 20, medical treatment (alfuzosin 20 and finasteride 16) in 36 and surgery (transurethral incision of the prostate 13 and prostatectomy 32) in 45. For statistical analysis Wilcoxon matched paired data and Kruskal Wallis tests were used as appropriate.

Results: Overall detrusor overactivity was present in 53 patients (52%) at baseline and 41 (40%) at followup. Detrusor overactivity was present in 9 patients (45%) at baseline and 11 (55%) at followup in the watchful waiting group ($p = 0.17$); 7 (35%) at baseline and 6 (30%) at followup in the alfuzosin group ($p = 0.37$); 10 (62.5%) at baseline and 10 at followup in the finasteride group ($p = 1$); 6 (46%) at baseline and 4 (30%) at followup in the transurethral prostate incision group ($p = 0.48$); and 21 (68%) at baseline and 10 (31%) at followup in the prostatectomy group ($p = 0.02$).

Conclusions: Detrusor overactivity is highly prevalent (52%) in patients with bladder outlet obstruction, and appears to persist for long periods when obstruction is left untreated or treated only with medical therapy. However, surgical treatment of bladder outlet obstruction, prostatectomy in particular, significantly reduces the incidence of detrusor overactivity and lessens the chance of its de novo appearance for up to 5 years.

KEY WORDS: prostatic hyperplasia, urodynamics, evaluation studies, follow-up studies, treatment outcome

Benign prostatic enlargement is one of the most common diseases in older men and is caused by benign prostatic hyperplasia, which usually develops after age 40 years. Clinical manifestations of benign prostatic hyperplasia may include voiding and/or storage symptoms frequently due to detrusor overactivity. In fact this condition is an important cause of lower urinary tract symptoms in men with benign prostatic enlargement, and overactivity may occur without any underlying neurological disease.[1] According to the International Continence Society, detrusor overactivity is defined as a urodynamic observation characterized by involuntary detrusor contractions during the filling phase which may be spontaneous or provoked.[2]

Although many theories have been proposed to explain the development of detrusor overactivity, its pathogenesis remains unclear. Since 1786, when Hunter first described an "increased irritability" of the bladder associated with bladder outlet obstruction, many studies, most using animal models, have suggested a relationship between detrusor overactivity and bladder outlet obstruction.[3,4] Moreover, scarce data exist on the natural evolution of detrusor overactivity in patients with bladder outlet obstruction or on its changes in response to different treatments. We analyze the clinical and urodynamic long-term evolution of detrusor overactivity in a group of patients with bladder outlet obstruction who were treated with watchful waiting, or medical or surgical therapy.

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Editor's Note: **This article is the fourth of 5 published in this issue for which category 1 CME credits can be earned. Instructions for obtaining credits are given with the questions on pages 670 and 671.**

MATERIAL AND METHODS

A total of 255 consecutive patients presented with lower urinary tract symptoms due to benign prostatic enlargement from 1992 to 1997. These patients completed the International Prostate Symptom Score (I-PSS), and underwent digital rectal examination, prostatic transrectal ultrasound and invasive urodynamic investigation with pressure flow study (1 examination). A total of 161 patients (63%) were found to be obstructed based on the Schafer nomogram (obstruction class 2 to 6) while 94 (37%) were unobstructed. Of the 161 patients 101 were contacted by telephone, and consented to a second clinical (I-PSS, digital rectal examination and transrectal ultrasound) and urodynamic evaluation with pressure flow study (1 examination) 1 to 5 years after followup (mean 24 months, median 20).

Of these 101 patients 81 (80%) had received either medical (36) or surgical (45) treatment according to patient or physician preference, while 20 (20%) who had refused treatment were placed on watchful waiting. In the medical treatment group 16 patients received 5 mg. finasteride daily and 20 received 5 mg. alfuzosin daily (at bedtime) during week 1, followed by 5 mg. alfuzosin twice daily (before breakfast and at bedtime). In the surgical group 13 patients underwent transurethral incision of the prostate and 32 patients underwent prostatectomy (transurethral resection of the prostate

TABLE 1. *Baseline clinical and urodynamic characteristics of patients with (group 1) and without (group 2) detrusor overactivity*

	Median (mean ± SD) (range)		p Value
	Group 1 (53 pts.)	Group 2 (48 pts.)	
Age	60 (60.1 ± 8.9)	63 (64.5 ± 7.6)	0.445
Prostate vol. (cc)	40 (37 ± 24) (28–80)	45 (42 ± 12) (30–85)	0.345
I-PSS	15 (17.58 ± 6.4) (2–34)	17 (15 ± 5.7) (5–29)	0.328
Quality of life	3 (3.3 ± 1.5) (1–6)	3 (2.6 ± 1.2) (0–5)	0.574
Free voided vol. (ml.)	308 (255 ± 121)	250 (346 ± 192)	0.435
Free max. flow rate (ml./sec.)	12 (10.6 ± 4)	10 (11.9 ± 5.1)	0.610
Free residual vol. (ml.)	45 (90 ± 110)	40 (102 ± 148)	0.530
Bladder capacity (ml.)	315 (287 ± 110)	264 (342 ± 146)	0.467
Max. flow at urodynamic study (ml./sec.)	8 (8.8 ± 3.9)	8 (8.3 ± 4.3)	0.3
Residual vol. after urodynamic study (ml.)	50 (42.5 ± 58.1)	40 (85 ± 119)	0.321
Detrusor pressure at max. flow (cm. water)	60 (72 ± 24)	66 (62.6 ± 16.2)	0.502
Min. urethral opening pressure (cm. water)	40 (42.2 ± 18.2)	42 (39.7 ± 12.2)	0.303
Urethral resistance factor (cm. water)	37 (38.4 ± 11.7)	37 (36.5 ± 12.2)	0.862
Schafer obstruction class (range)	3 (3.1 ± 1) (2–6)	3 (2.8 ± 0.7) (2–5)	0.340
Projected isometric pressure (cm. water)	100 (109 ± 26.3)	100 (100.8 ± 23.7)	0.239
Detrusor adjusted mean passive urethral resistance relation factor (cm. water)	50 (64.5 ± 15.5)	54 (59 ± 13.2)	0.349

TABLE 2. *Baseline clinical and urodynamic results stratified by treatment*

	Prostatectomy	Transurethral Incision of Prostate	Watchful Waiting	Alfuzosin	Finasteride	p Value
No. pts.	32	13	20	20	16	
Median age (mean ± SD)	62 (62 ± 6.2)	62 (63 ± 7.6)	57 (60 ± 8.6)	64 (63 ± 10)	63 (64 ± 9)	0.44
Median mos. followup (mean ± SD)	24 (25 ± 12)	16 (24 ± 15)	19 (25 ± 14)	15 (23 ± 14)	16 (24 ± 15)	0.63
Median I-PSS (mean ± SD)	17 (17 ± 5.5)	18 (17.6 ± 5)	14 (15.5 ± 6.9)	14 (14.9 ± 6.8)	16 (17.3 ± 6.7)	0.34
Median ml. prostate vol. (mean ± SD)	55 (50 ± 25)	28 (25 ± 15)	38 (37 ± 24)	45 (44 ± 25)	50 (45 ± 18)	0.15
Median ml./sec. free max. flow rate (mean ± SD)	12 (12 ± 4.3)	9 (9.3 ± 3.4)	12 (11.5 ± 4.1)	10 (12.4 ± 5.9)	9 (9.7 ± 3.9)	0.88
Median ml. bladder capacity (mean ± SD)	300 (316 ± 130)	350 (361 ± 131)	253 (302 ± 164)	284 (308 ± 126)	300 (290 ± 62)	0.33
Median cm. water urethral resistance factor (mean ± SD)	38 (38.9 ± 125)	38 (38.4 ± 13.2)	35 (37.2 ± 14)	36 (38 ± 8)	33 (34.7 ± 11)	0.44
Median Schafer obstruction class	3 (3.1 ± 1)	3 (3 ± 0.9)	3 (2.9 ± 0.9)	3 (2.7 ± 0.7)	3 (3.1 ± 0.7)	0.25
No. detrusor instability	21	6	9	7	10	0.22

22 and open prostatectomy 10). Surgical procedures were performed using standard techniques by a single experienced surgeon (G. F.).

Transrectal ultrasound was performed with a 5 MHz. biplanar linear transrectal probe and residual urine volume was assessed with a suprapubic 3.5 MHz. convex probe. For urodynamic equipment we used a multichannel system (Urodesk 300, Siem, Milan, Italy). Investigations were performed using fluid filled lines and an external transducer. For bladder pressure measurement and bladder filling we used a 5Fr and 8Fr transurethral single lumen catheter, respectively. An 8Fr rectal balloon catheter was used for abdominal pressure recording to obtain detrusor pressure. Before cystometry the bladder was emptied through the lumen of the transurethral catheter, and transducers were balanced to atmospheric pressure. The bladder was then filled with sterile water at 20C with a filling speed of 50 ml. per minute. Cystometrograms were obtained recording first desire, maximal cystometric capacity, detrusor compliance and presence of detrusor overactivity. According to the International Continence Society detrusor overactivity is defined as any phasic detrusor contraction during the filling phase or terminal detrusor contraction occurring at cystometric capacity.[2] The pressure flow study was performed with only the 5Fr catheter in situ.

Urodynamic variables analyzed included free maximum flow rate, free voided volume, residual volume after free flowmetry, functional bladder capacity at cystometry, maximum flow during pressure flow study, detrusor pressure at maximum flow, minimal urethral opening pressure (manually measured) and residual volume after urodynamic pres-

TABLE 3. *Baseline and followup characteristics of patients stratified by treatment*

	Median (mean ± SD) (p value)			
	Watchful Waiting		Alfuzosin	
	Baseline	Followup	Baseline	Followup
I-PSS (range)	14 (15.5 ± 6.9)	14 (14.3 ± 7.5) (0.37)	14 (14.9 ± 6.8)	12 (13.3 ± 5.2) (0.15)
Quality of life	2 (2.5 ± 1.6)	2 (2.6 ± 1.5) (0.82)	2 (2.6 ± 1.5)	2 (2.2 ± 1.2) (0.18)
Prostate vol. (cc)	38 (37 ± 24)	43 (42 ± 12) (0.79)	45 (44 ± 25)	49 (50 ± 11) (0.25)
Free voided vol.	232 (278 ± 169)	250 (253 ± 156.7) (0.36)	315 (345.2 ± 193)	241 (297 ± 199) (0.3)
Free residual vol.	30 (85.9 ± 151.9)	50 (112.4 ± 155.4) (0.65)	30 (103.5 ± 132)	15 (33.5 ± 38.4) (0.02)
Free max. flow rate (ml./sec.)	12 (11.5 ± 4.1)	12 (12 ± 4.2) (0.6)	10 (12.4 ± 5.9)	13 (14 ± 5.9) (0.13)
Bladder capacity (ml.)	253 (302 ± 164)	320 (322.2 ± 81.4) (0.36)	286 (308 ± 126)	320 (335 ± 178) (0.11)
Max. flow at urodynamic study	9 (9 ± 3.8)	8 (8.9 ± 3.6) (0.41)	9 (9.1 ± 4.1)	10 (10.7 ± 4.4) (0.01)
Residual vol. after urodynamic study	15 (67.3 ± 111.4)	0 (55.7 ± 124.1) (0.48)	0 (64 ± 105.8)	0 (28.8 ± 43.7) (0.1)
Detrusor pressure at max. flow	65 (70.2 ± 22.3)	58 (67.5 ± 23.7) (0.33)	56 (64.4 ± 22.9)	50 (53.5 ± 12.3) (0.04)
Minimal urethral opening pressure	40 (43.4 ± 18)	35 (46.9 ± 27.4) (0.35)	36 (36.5 ± 9.3)	31 (31.4 ± 9) (0.02)
Projected isometric pressure (cm. water)	120 (114 ± 25.9)	105 (112 ± 21.4) (0.62)	95 (105.7 ± 36)	98 (105 ± 26.4) (0.91)
Urethral resistance algorithm (cm. water)	35 (37.2 ± 14)	3 (37 ± 16) (0.43)	35 (38 ± 8)	28 (27.7 ± 7.1) (0.02)
Schafer obstruction class	3 (2.9 ± 0.9)	2 (2.8 ± 1.2) (0.86)	3 (2.7 ± 0.7)	2 (2.2 ± 0.8) (0.04)
Detrusor adjusted mean passive urethral resistance relation factor (cm. water)	63 (64.7 ± 16)	58 (64.3 ± 20.3) (0.33)	51 (57.1 ± 10.4)	50 (50.4 ± 7.8) (0.03)

sure flow study. Maximum flow, detrusor pressure at maximum flow and minimal urethral opening pressure were manually plotted on the Schafer nomogram[5] to obtain the linear passive urethral resistance relation, Schafer obstruction class, detrusor adjusted mean passive urethral resistance relation factor and projected isometric pressure. Values of the urethral resistance algorithm were also obtained with the urethral resistance algorithm curves superimposed on the Schafer linear passive urethral resistance relation nomogram. Urinary sediment and culture were negative at urodynamic evaluation.

Patient characteristics at baseline. Mean patient age at first evaluation was 62.5 years (median 63, range 45 to 80). Mean total prostatic volume measured by transrectal ultrasound was 50 ml. (median 48, range 20 to 80). Mean I-PSS was 16.4 (median 16, range 2 to 34). Mean maximum flow at free uroflowmetry was 11.2 ml. (median 11, range 3 to 25), mean residual urine was 96 ml. (median 40, 0 to 500), mean bladder capacity was 196 ml. (median 300, 80 to 800), and mean and median Schafer obstruction class was 3 (range 2 to 6). A total of 53 patients (52%) presented with detrusor overactivity. No patient was treated with anticholinergic drugs, and no one presented with neurological disease.

Statistical analysis. Statistical analysis was performed using the nonparametric Wilcoxon matched pairs signed rank test and was applied to the change in I-PSS and urodynamic parameters, comparing end point results with baseline for each treatment group. Differences in baseline clinical and urodynamic characteristics among the treatment groups were also compared using the nonparametric Kruskal-Wallis test. Data are presented as median and mean ± SD with a $p < 0.05$ considered statistically significant.

RESULTS

Baseline characteristics. There were no clinical or urodynamic differences between baseline characteristics of the total group of 161 patients with obstruction and the reevaluated group of 101 patients (detrusor overactivity 54% and 52%, respectively). The 101 patients were separated into 2 groups according to baseline cystometrogram findings of stable (48) and overactive (53). No statistically significant differences were noted between the 2 groups in regard to clinical and urodynamic parameters (table 1). Also no statistical significant differences were noted between baseline clinical and urodynamic characteristics of patients in the different treatment groups (table 2).

Evolution of symptoms and bladder outlet obstruction at baseline and followup. In the watchful waiting group no statistically significant differences were noted in the I-PSS and obstruction urodynamic parameters between baseline and followup. Alfuzosin treatment produced statistically significant improvement in residual volume after free flow, detrusor pressure at maximum flow, minimum voiding detrusor pressure, urethral resistance algorithm and Schafer's class with no significant changes in I-PSS. In the finasteride group no statistically significant differences were noted for I-PSS and bladder outlet obstruction parameters. In the surgery group significant improvement was noted in symptom score and obstruction parameters (table 3).

Changes in detrusor overactivity (see figure). Overall detrusor overactivity was present in 53 patients (52%) at baseline and 41 (40%) at followup, including 28 who had it at baseline. Detrusor overactivity was present in 9 patients (45%) at baseline and 11 (55%) at followup, including 9 who had it at baseline, in the watchful waiting group ($p = 0.17$); in 7 (35%) at baseline and 6 (30%) at followup, including 4 who had it at followup, in the alfuzosin group ($p = 0.37$); in 10 (62.5%) at baseline and 10 at followup, including 7 who had it at baseline, in the finasteride group ($p = 1$); in 6 (46%) at baseline and 4 (30%) at followup, including 1 who had it at baseline, in the transurethral prostate incision group ($p = 0.48$); and in 21 (68%) at baseline and 10 (31%) at followup, including 9 who had it at baseline, in the prostatectomy group ($p = 0.02$).

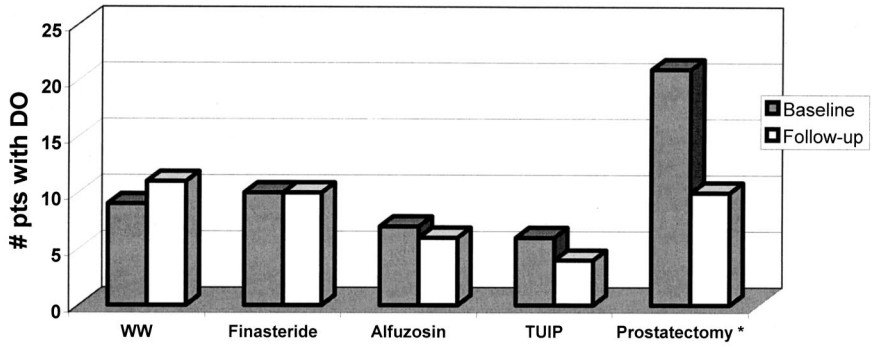
DISCUSSION

Detrusor overactivity is one of the most common causes of lower urinary tract symptoms, and is often associated with bladder outlet obstruction and benign prostatic enlargement. Urodynamic studies of patients with bladder outlet obstruction due to benign prostatic enlargement have revealed detrusor overactivity in more than 50% of men undergoing prostatectomy.[6,7] Following prostatectomy two-thirds of patients with previous detrusor overactivity had normal detrusor function on postoperative cystometrogram.[8,9] However, in a series of 100 patients with bladder outlet obstruction Abrams observed 63% conversion from unstable to stable detrusor after prostatectomy, and the only factor that was predictive of conversion was the severity of the preoperative detrusor instability.[9]

Although these data seem to demonstrate a relationship between detrusor overactivity and bladder outlet obstruction, the pathophysiology of the former remains unclear. Different studies have shown that increased bladder pressure such as that observed with bladder outlet obstruction can lead to partial denervation of the detrusor muscle resulting in detrusor overactivity caused by post-junctional supersensitivity.[10,11] To date followup studies of patients who underwent prostatectomy reveal reinnervation of the bladder and recovery of detrusor stability after relief of obstruction.[9,11,12] Other studies have suggested that abnormal sensory stimuli

TABLE 3. *(Continued)*

Median (mean ± SD) (p value)					
Finasteride		Transurethral Incision of Prostate		Prostatectomy	
Baseline	Followup	Baseline	Followup	Baseline	Followup
16 (17.3 ± 6.7)	15 (15.8 ± 7.3) (0.518)	18 (17.6 ± 5)	12 (10.8 ± 5.5) (0.003)	17 (17 ± 5.5)	6 (8.1 ± 6.2) (0.0000)
4 (3.4 ± 1.2)	3 (3.3 ± 1.4) (0.722)	3 (2.9 ± 1)	2 (1.8 ± 1.4) (0.04)	3 (3.3 ± 1.3)	1 (1.3 ± 1.2) (0.0000)
50 (45 ± 18)	54 (50 ± 20) (0.325)	28 (25 ± 15)	30 (28 ± 19) (0.421)	55 (50 ± 25)	30 (28 ± 10) (0.03)
254 (287 ± 138)	258 (252.2 ± 139.6) (0.224)	200 (225.6 ± 121)	298 (316 ± 178) (0.07)	275 (314.5 ± 155)	288 (360.7 ± 297) (0.74)
35 (65 ± 79.3)	20 (45.1 ± 65) (0.345)	60 (113 ± 146.7)	60 (93.4 ± 111) (0.55)	30 (104 ± 129)	50 (38 ± 71) (0.001)
8 (9.7 ± 3.9)	9 (11.1 ± 5.5) (0.19)	9 (9.3 ± 3.4)	14 (14 ± 7.2) (0.03)	12 (12 ± 4.3)	22 (21.4 ± 10.4) (0.0002)
300 (290 ± 62)	303 (307.8 ± 99.2) (0.5)	350 (361 ± 139.6)	340 (338 ± 124) (0.80)	300 (316 ± 130)	320 (356 ± 120) (0.12)
8 (8.3 ± 3)	9 (9.6 ± 4.3) (0.28)	7 (7.1 ± 3.5)	11 (10.7 ± 4.4) (0.05)	7 (8.8 ± 4.5)	16 (17.8 ± 7.3) (0.0000)
37 (39 ± 43)	0 (28.2 ± 53.8) (0.5)	10 (59.2 ± 97.3)	80 (87.1 ± 75.1) (0.32)	35 (73 ± 94)	0 (19 ± 78) (0.001)
61 (66.2 ± 15.3)	59 (67.2 ± 28.6) (0.44)	63 (60.7 ± 16.5)	50 (52 ± 11.7) (0.22)	65 (72 ± 25)	47 (48.5 ± 12.3) (0.0000)
33 (37.6 ± 14.2)	36 (36.8 ± 17.6) (0.42)	41 (38.9 ± 14.4)	38 (37.9 ± 13.1) (0.81)	50 (44.5 ± 17.5)	31 (29.4 ± 15.2) (0.001)
104 (103 ± 20.7)	112 (101.6 ± 29.7) (0.26)	100 (115 ± 17.4)	102 (103 ± 27) (0.20)	100 (113 ± 36)	113 (114 ± 19) (0.24)
38 (34.7 ± 11)	33 (38.4 ± 20.3) (0.26)	38 (38.4 ± 12.2)	26 (28.1 ± 8.6) (0.03)	38 (38.9 ± 12.5)	16 (18 ± 9) (0.0000)
3 (3.1 ± 0.7)	3 (3.1 ± 1.2) (1)	3 (3 ± 0.9)	2 (1.8 ± 0.8) (0.007)	3 (3 ± 1)	1 (1.5 ± 1) (0.0000)
60 (61.9 ± 12)	58 (58.6 ± 20.6) (0.28)	65 (58.7 ± 16.9)	49 (51.2 ± 11.7) (0.20)	62 (64 ± 16)	40 (42 ± 13) (0.000)



Changes in detrusor overactivity in different treatment groups. *WW*, watchful waiting. *TUIP*, transurethral incision of prostate. *DO*, detrusor overactivity.

from an anatomically altered prostatic urethra, as in patients with benign prostatic enlargement, can induce detrusor overactivity.[6, 9, 13] Permanent surgical ablation of sensory stimuli from the prostatic urethra would be beneficial. The incidence of detrusor overactivity consistently increases with age in more than 50% of men older than 70 years but not suffering from obstruction.[14] Holm et al observed that changes in detrusor nerve density and fibrosis observed in bladder outlet obstruction are not distinguishable from those of aging, suggesting that detrusor overactivity and bladder outlet obstruction are unrelated events occurring in elderly men with an incidence increasing with age.[15] Other mechanisms proposed include altered adrenoceptor function, neurotransmitter imbalance and a myogenic deficit.[16] All of these findings seem to indicate that detrusor overactivity is not just a consequence of bladder outlet obstruction, but might be related to many other factors, and probably what is known only represents the tip of a large iceberg.

Our study confirmed a high prevalence (50%) of detrusor instability in men with bladder outlet obstruction, which was not related to age, symptom severity, detrusor power or obstruction. However, different treatments have shown different impacts on detrusor overactivity during a long time. As far as the "natural evolution" of detrusor overactivity is concerned, the followup of our patients on watchful waiting seems to show long-term persistence with no spontaneous resolution and few new cases. However, it must be noted that bladder outlet obstruction, which some consider a possible cause of detrusor overactivity, also remained unchanged. Also the finasteride group showed no significant change in detrusor overactivity with no change in symptom score or bladder outlet obstruction. Detrusor overactivity seems to remain unchanged for a long time when bladder outlet obstruction and symptoms are stable.

In the alfuzosin group no significant differences were noted in the total number of patients with detrusor overactivity at baseline (7) and at followup (6). However, 5 of 7 patients with detrusor overactivity at baseline presented with a stable detrusor after treatment. This phenomenon might be explained by the direct effect of α-blockers on the detrusor muscle as described by Perlberg and Caine, who proposed an α-adrenergic contractile response in patients with detrusor overactivity compared to a β-adrenergic response in those with a stable detrusor.[17] Moreover Reuther and Aagaard noted that detrusor overactivity disappeared in patients treated with phenoxybenzamine and reappeared when treatment was discontinued.[18] Furthermore, the effect of alfuzosin in relieving bladder outlet obstruction may also have a role in the resolution of detrusor overactivity even without significant change in the total I-PSS.[19] Our finding of a long lasting reduction of bladder outlet obstruction after a median of 15 months of treatment with alfuzosin may be related to a chronic reduction in the dynamic component of bladder outlet obstruction with consequent significant decrease in residual urine. The nonsignificant improvement of symptom scores may also be explained by the persistence of detrusor overactivity with irritative symptoms.

In the surgery group a statistically significant improvement in all obstruction parameters as well as significant relief of detrusor instability was observed. A total of 62% of patients with detrusor overactivity before surgery presented with normal bladder behavior 1 to 5 years (mean 2) after surgery, and a small number of patients with de novo detrusor overactivity were observed during followup. Therefore, surgery seems to have a significant effect on the evolution of this condition. However, in our surgical group it was evident that transurethral incision of the prostate and prostatectomy significantly reduced bladder outlet obstruction but detrusor overactivity was permanently healed with fewer new cases only in the prostatectomy group. This finding might be explained by a major sensory denervation effect, presumably accomplished with prostatectomy compared to transurethral incision of the prostate. This concept is also supported by the CLasP study, which showed that prostatectomy using the laser technique produced less reduction in obstruction but the same reduction in detrusor instability as that observed after transurethral resection of the prostate.[20] However, the CLasP study also provided evidence of detrusor overactivity recurrence after prostatectomy, with a significant increase in detrusor instability several years after surgery despite a sustained long-term reduction in obstruction. This effect appears to be unrelated to patient age.

In our study the rate of de novo detrusor overactivity was only 9% in the prostatectomy and 29% in the other groups. However, we analyzed evolution of bladder outlet obstruction after a medium-term followup, which was probably not enough to allow sensory reinnervation of the urethra and bladder neck, or the natural aging effect on detrusor behavior. Furthermore, persistence of bladder outlet obstruction in other groups of patients may also explain a higher likelihood of de novo detrusor overactivity. It is also likely that in our study detrusor overactivity was relieved by a denervation effect on the bladder neck and trigonal area, and on the prostatic urethra obtained by surgery as first proposed by Abrams et al.[6] These findings were also supported by Chalfin and Bradley, who selectively ablated sensory stimuli from the prostatic urethra with perineal prostatic lidocaine injection in patients with obstructed bladder outlet obstruction and detrusor overactivity.[13] They demonstrated that the sensory block alone without relief of obstruction can eliminate detrusor overactivity.

It is likely that surgery damages the fine system of sensory fibers beneath the mucosa of the prostatic urethra and bladder neck region. Sensory denervation may reduce the abnormal sensory input of the voiding reflex typical of detrusor overactivity. This hypothesis is corroborated by our finding that patients who received alfuzosin or underwent transurethral incision of the prostate experienced significant relief of bladder outlet obstruction without change in detrusor overactivity. Thus, only when complete denervation of the bladder neck and urethra was accomplished, as after prostatectomy, was long lasting relief of detrusor overactivity obtained. Limitations of the current study must be acknowledged. Our study was not prospective and as such data might be subjected to selection bias. Patients who experienced a worsening of symptoms during followup might not be included in the series. However, this selection bias was minimized by comparing the evolution of detrusor overactivity in 5 homogenous groups of patients treated with different options.

CONCLUSIONS

Detrusor overactivity is a common finding in men with bladder outlet obstruction, which seems to persist for a long time when obstruction is left untreated or is treated with medical therapy only. Surgical treatment of bladder outlet obstruction, particularly prostatectomy, significantly reduces the incidence of detrusor overactivity and lessens the chances of new appearance for up to 5 years. The most likely explanation is the denervation effect of prostatectomy on the bladder neck and prostatic urethra.

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