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Original Article: Clinical Investigation

De novo detrusor underactivity after laparoscopic radical prostatectomy

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Objective: The aim of this study was to investigate bladder function following laparoscopic radical prostatectomy, with a focus on de novo detrusor underactivity.

Methods: Records on pre- and postoperative urodynamic studies were retrospectively investigated in 110 patients who underwent laparoscopic radical prostatectomy. Patients exhibiting de novo detrusor underactivity were selected on the basis of an overt strain voiding pattern during the postoperative pressure flow study with detrusor pressure at a maximum flow rate <10 cm H_2O accompanied by an increase in abdominal pressure. In these patients, a follow-up urodynamic study was performed to assess subsequent long-term changes in the bladder function.

Results: Of the 110 patients, 10 (9.1%) were observed to exhibit de novo detrusor underactivity during the postoperative urodynamic study. During the voiding phase of the pre- and postoperative pressure flow study in these 10 patients, the mean detrusor pressure at maximum flow rate showed a significant decrease postoperatively from 57.6 to 3.0 cm H_2O ($P < 0.001$), although the mean abdominal pressure at maximum flow rate significantly increased from 23.1 to 102.5 cm H_2O ($P < 0.001$). The follow-up urodynamic study performed on seven patients at 36 months following surgery revealed no significant change in each urodynamic parameter. De novo detrusor underactivity persisted even over the long term following surgery, and no improvement in bladder function was observed.

Conclusions: Detrusor contractility may be impaired during radical prostatectomy. Postoperative detrusor underactivity following radical prostatectomy seems to be an irreversible phenomenon persisting even over the long term.

Key words: detrusor underactivity, laparoscopy, radical prostatectomy, voiding dysfunction.

Introduction

The widespread use of serum prostate-specific antigen (PSA) in prostate cancer screening has resulted in a rapid increase in the detection rate of early prostate cancer; consequently, the frequency of radical prostatectomy has also increased in recent years. Moreover, since minimally invasive therapies, such as brachytherapy[1,2] and heavy ion therapy,[3] have been widely applied, there has been an increasing interest in improvement in the quality of life due to the prevention of postoperative complications such as urinary incontinence and erectile dysfunction. These factors have been seriously considered in radical prostatectomy; consequently, various operational procedures have been improved.[4–6]

Several studies have assessed storage dysfunction after radical prostatectomy. Changes in the lower urinary tract function, that is, a decline in the urethral sphincter function, a decrease in bladder compliance, and detrusor overactivity are known to develop independently or compositely to cause postoperative incontinence.[7–11] However, there are few reports describing the voiding dysfunction that occurs after radical prostatectomy; furthermore, postoperative detrusor underactivity has not been thoroughly investigated. Moreover, there are almost no reports regarding the evaluation of lower urinary tract function after laparoscopic radical prostatectomy. Therefore, this study investigated the development and clinical significance of de novo detrusor underactivity after laparoscopic radical prostatectomy. Moreover, temporal changes in detrusor underactivity were also investigated.

Methods

In this study, we retrospectively examined 110 patients with localized prostate cancer in clinical stages ranging from T1c to T2b who underwent laparoscopic radical prostatectomy and pre- and postoperative urodynamic studies in our hospital between January 2003 and December 2006. All of the patients underwent laparoscopic radical prostatectomy in an antegrade fashion and via a retroperitoneal approach. Based

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Table 1 Backgrounds of 10 cases with de novo detrusor underactivity

Case	Age	Preoperative PSA (ng/mL)	Resected prostate weight (g)	pT stage	Past illness or complication	Postoperative incontinence
1	65	11.5	21	pT2a	No	Mild
2	69	9.3	38	pT2b	Appendectomy	No
3	71	2.3	37	pT2a	TURP	No
4	60	10	28	pT0	No	No
5	62	11	17	pT3a	Appendectomy	No
6	68	13.7	70	pT2b	No	No
7	76	17.8	38	pT2b	No	No
8	72	10.4	55	pT2b	Hypertension	Mild
9	72	7.4	59	pT2b	Hypertension	No
10	65	15.8	21	pT2b	No	No
Mean	67	10.9	38.4	–	–	–

PSA, prostate-specific antigen; TURP, transurethral resection of the prostate.

on the findings in the pre- and postoperative pressure flow study (PFS), patients exhibiting de novo detrusor underactivity were selected. The bladder and urethral function of these patients were retrospectively investigated in detail based on the medical records of the prostatic cancer stage, clinical course, surgery, and the urodynamic studies. Moreover, the selected patients were observed over a long period of time and the temporal changes in detrusor underactivity were examined.

The lower urinary tract function during storage and voiding phase was investigated with urodynamic studies. The urodynamic studies consisted of PFS and urethral pressure profile (UPP). These were conducted in the patients 2–3 days before and 3–5 months after the surgery. In the PFS, the intravesical pressure, intrarectal pressure (abdominal pressure), detrusor pressure, uroflowmetry (UFM), and residual volume immediately after voiding were measured. Besides the PFS, free UFM in combination with measurement of the residual volume was conducted before and after the operation.

In the PFS, after emptying the bladder, 8-Fr and 6-Fr (single J catheter) catheters were individually inserted through the urethra. The intravesical pressure was measured using the 6-Fr catheter while a physiological saline solution was injected through the 8-Fr catheter at a rate of 50 mL/min into the bladder of the patients, who were in the standing position. Moreover, the intravesical pressure during voiding was measured using the 6-Fr catheter after removing the 8-Fr catheter. The intrarectal pressure was measured using a balloon catheter that was inserted through the anus. The initial intravesical and abdominal pressures were set to zero immediately before bladder filling. In the UPP, the maximum urethral closing pressure (MUCP) was measured using a 6-F microtip transducer catheter when the bladder was empty. The definitions used in the urodynamic study conformed to the standards recommended by the International Continence Society.[12]

In this study, the patients with an apparent pattern of abdominal strain during voiding (an increase in the abdominal pressure) and a PdetQmax of less than 10 cmH_2O were defined as the patients with detrusor underactivity. The patients with de novo detrusor underactivity were selected based on the findings in the pre- and postoperative urodynamic studies.

The PFS were conducted in the patients with de novo detrusor underactivity from 15 to 45 months (average, 36 months) after the operation, and the temporal changes in the detrusor underactivity were investigated. The significant differences were statistically investigated using the Student's *t*-test.

Results

Among the 110 patients who were assessed with pre- and postoperative urodynamic studies, de novo detrusor underactivity was observed in 10 patients (9.1%). The backgrounds of these 10 patients with de novo detrusor underactivity are shown in Table 1. No significant differences were observed between these 10 patients and the other patients with respect to age (average age: 67.0 and 66.4 years, respectively) or preoperative PSA values (average value: 10.9 and 14.2 ng/mL, respectively). In all the patients with detrusor underactivity, the pathological stage of prostate cancer was below pT3a (pT0: 1, pT2a: 2, pT2b: 6, and pT3a: 1) with no infiltration of the cancer observed in the bladder neck. In case of the other patients, 59.6% of the cases had prostate cancer below stage pT2, 30.3% had stage pT3a, and 10.1% had stage pT3b.

Table 2 Changes of parameters on urodynamic studies in a total of 100 patients excluding 10 patients with de novo detrusor underactivity

	Preoperative	Postoperative	*P*
Mean MUCP (cmH_2O)	65.2	42.8	<0.001
(SD)	(20.3)	(15.6)	
Mean FDV (mL)	110.3	115.1	0.65
(SD)	(44.0)	(48.1)	
Mean MCC (mL)	258.0	249.2	0.34
(SD)	(78.4)	(67.7)	
Mean PdetQmax (cmH_2O)	55.6	35.9	<0.001
(SD)	(24.5)	(15.5)	
Mean PabdQmax (cmH_2O)	10.7	8.9	0.24
(SD)	(3.5)	(3.6)	
Mean Qmax (mL/s)	11.6	15.4	0.004
(SD)	(4.0)	(5.1)	

FDV, first desire to void; MCC, maximum cystometric capacity; MUCP, maximum urethral closing pressure; PabdQmax, abdominal pressure at maximum flow rate; PdetQmax, detrusor pressure at maximum flow rate; Qmax, maximum flow rate; SD, standard deviation.

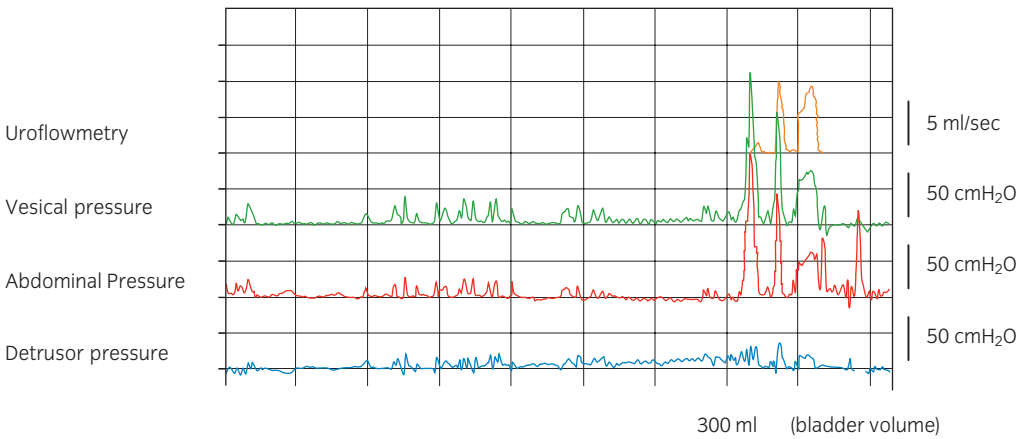

Fig. 1 Postoperative pressure flow study (PFS) in a patient with de novo detrusor underactivity. The figure shows the postoperative PFS of Case 6 in Table 1. A flow curve in uroflowmetry during voiding phase demonstrated an intermittent voiding pattern, suggesting strain voiding. There is a marked increase of abdominal pressure without detrusor contraction. Excretion of urine was accomplished by abdominal straining.

Moreover, complications such as rectal injury were not observed during the operation, and no serious postoperative complications developed in any of the 10 patients with de novo detrusor underactivity. None of the 10 patients with de novo detrusor underactivity had an episode of neurological disorder or diabetes.

Urodynamic studies were conducted between 2 and 5 months (average, 3.8 months) after the operation. Table 2 showed changes of urodynamic parameters in a total of 100 patients except 10 patients with de novo detrusor underactivity. Mean MUCP and PdetQmax were significantly reduced after surgery. On the other hand, there was no significant difference in first desire to void (FDV), maximum cystometric capacity (MCC) or PabdQmax between pre- and post-operation.

Detrusor overactivity was noted preoperatively in 28 patients, and postoperatively in 36 patients. Detrusor overactivity appeared as a de novo dysfunction in 23 (20.9%) of 110 patients. Detrusor overactivity was noted preoperatively in 28 patients and disappeared in 15 patients (53.6%) in the postoperative examination. Detrusor overactivity was not observed in 10 patients with de novo detrusor underactivity.

Detrusor underactivity could be precisely diagnosed based on the marked increase in the abdominal pressure and decrease in the detrusor pressure while voiding as observed during the PFS. As observed in Figure 1, because the UFM

Table 3 Voiding function on urodynamic studies in 10 patients with de novo detrusor underactivity, at preoperative, postoperative and long-term examinations

Case	PdetQmax on PFS (cmH_2O)			PabdQmax on PFS (cmH_2O)		
	Preoperative	Postoperative	Long-term	Preoperative	Postoperative	Long-term
1	48	0	8	5	80	65
2	52	0	6.5	50	120	77.5
3	60	3	8	30	70	85
4	50	5	ND	0	50	ND
5	34	0	5	30	100	85
6	100	0	ND	0	200	ND
7	62	5	11	25	60	55
8	55	10	ND	35	95	ND
9	72	5	4	45.5	150	80
10	43	2	4	10	100	90
Mean	57.6	3.0	6.6	23.1	102.5	76.8
P		<0.001			<0.001	

ND, no data; PabdQmax, abdominal pressure at maximum flow rate; PdetQmax, detrusor pressure at maximum flow rate; PFS, pressure flow study.

Table 4 Storage function on urodynamic studies in 10 patients with de novo detrusor underactivity, at preoperative, postoperative and long-term examinations

Case	MUCP (cmH_2O)			FDV (mL)			MCC (mL)		
	Preoperative	Postoperative	Long-term	Preoperative	Postoperative	Long-term	Preoperative	Postoperative	Long-term
1	47	28	39	85	89	95	309	303	223
2	56	60	56	80	97	102	203	251	205
3	59	56	54	32	95	62	232	300	232
4	53	51		50	77		302	301	
5	65	50	48	83	150	135	198	232	220
6	49	29		110	108		178	172	
7	48	35	38	57	65	66	185	198	115
8	54	79		153	155		203	203	
9	57	51	48	112	133	187	218	252	306
10	55	45	45	105	112	132	230	255	260
Mean	54.3	48.4	46.9	86.7	108.1	111.3	225.8	246.7	223.0
P		0.27			0.16			0.32	

FDV, first desire to void; MCC, maximum cystometric capacity; MUCP, maximum urethral closing pressure.

assessed during the PFS demonstrated an intermittent voiding pattern, strain during voiding could be presumed. Based on the evaluation of PdetQmax, although the preoperative detrusor contraction in all of the patients was adequate (average: 57.6 cmH_2O), the postoperative detrusor contraction was remarkably decreased in all of them (average: 3.0 cmH_2O) ($P < 0.001$) (Table 3). On the contrary, PabdQmax increased from a preoperative value of 23.1 cmH_2O to a postoperative value of 102.5 cmH_2O ($P < 0.001$). Although the mean maximum flow rate in free UFM significantly increased from a preoperative value of 12.2 mL/s to a postoperative value of 24.3 mL/s ($P < 0.001$), the flow curve demonstrated an intermittent pattern in nine of 10 patients with a low mean flow rate of 6.8 mL/s. No residual urine was observed in any of the patients postoperatively.

No significant changes were observed in the storage-function-related urodynamic parameters, such as the bladder capacity at the FDV, the MCC, or the MUCP (Table 4).

No incontinence was observed in eight patients, and slight incontinence, for which patients required a pad per day, was observed in two patients. Only one patient complained of

subjective voiding symptoms such as weak stream and voiding difficulty.

The PFS were repeated in seven out of 10 patients between 15 and 49 months (average, 36 months) after the operation, excluding one patient who developed cerebral infarction after the operation, one patient who underwent radical cystectomy due to bladder cancer, and one patient with insufficient follow up; furthermore, the temporal changes in the bladder and urethral function were investigated. Consequently, the voiding status was excellent, and no incontinence was observed in the seven patients. In the PFS, although the detrusor underactivity was observed to be prolonged (Table 3), no changes were observed in Qmax, FDV, or MCC; moreover, none of the patients complained of an ingravescence of the voiding status (Table 4).

Discussion

The first surgical treatment of prostate cancer was performed by Millin *et al*. in 1947;[13] since then, a variety of improved operative methods have been devised. The surgical management of the dorsal vein complex and the procedure for the preservation of the neurovascular bundle were improved upon by Walsh *et al*.[4] Consequently, the incidence of postoperative complications, such as erectile dysfunction and incontinence, has been decreasing. However, postoperative incontinence is one of the complications that continue to occur at a constant rate and negatively affect the quality of life of the patients.

Many reports regarding incontinence after radical prostatectomy have stated that postoperative incontinence was mainly caused by a perioperative injury to the urethral sphincter and urethral sphincter insufficiency was observed in 8–71% of the patients.[14–18] Bladder dysfunctions, such as a decrease in bladder compliance and detrusor overactivity, have been suggested as the other causes of postoperative incontinence.[10,11,14–18] Urethral sphincter insufficiency is considered to be the direct result of a perioperative injury to the external urethral sphincter at the apex of the prostate and is partially affected by an abscission of the neurovascular bundle. The preservation of the sphincter function has been attempted by performing various surgical procedures for the apex and by preserving the neurovascular bundle.[19,20]

In contrast, there are few reports regarding voiding dysfunction after radical prostatectomy. Some reports have indicated that strain voiding was observed after radical prostatectomy, and the frequency of abdominal strain during voiding after an open radical prostatectomy was observed to be 48%[8] and 29.5%.[10] On the other hand, we identified only three reports[17,18,21] in which the postoperative detrusor underactivity was investigated based on the findings of a urodynamic study. Groutz *et al*. reported that PFS were conducted in 83 patients who had undergone open radical prostatectomy, and that postoperative detrusor underactivity developed in 28.9% of the patients. Furthermore, although the frequency of the postoperative detrusor underactivity development was reported to be 28.6%[17] and 43%,[18] temporal changes in the postoperative detrusor underactivity were not reported. In addition, there has been no report on the vesicourethral function investigated based on urodynamic studies, following laparoscopic radical prostatectomy.

There might be an argument on interpretation of the urodynamic data in 10 patients we selected as detrusor underactivity. It was difficult to diagnose detrusor underactivity only by evaluating the maximum flow rate or the flow curve obtained with UFM. PFS demonstrated that excretion of urine was carried out almost by abdominal straining alone without significant detrusor contraction. After radical prostatectomy, the voiding dynamics are different from those in the preoperative condition, because postoperatively the flow-controlling zone shifts from the proximal urethra to the external sphincter with a decrease of urethral resistance. Therefore, it could be argued that the patients undergoing radical prostatectomy can void without significant detrusor contraction as in women, despite preserved detrusor contractility. However, in the present study, the patients evacuated urine by straining with abnormally high abdominal pressure rise under no or minimal detrusor contraction. In addition, the flow curves were not good with an intermittent pattern despite a high maximum flow rate. Based on these interpretations of the urodynamic data, we considered that detrusor contraction in the present 10 patients was impaired. In these patients, although excellent bladder contraction was observed in the preoperative PFS, apparent detrusor underactivity and high abdominal pressure were observed in the postoperative PFS. Therefore, we consider that the postoperative detrusor underactivity may have developed due to some manipulations during radical prostatectomy. The denervation[21] caused by neural injury to the trigone of the bladder during the dissection of the bladder neck was considered as one of the causes of postoperative detrusor underactivity. However, the 10 patients had a localized prostatic cancer without accompanying infiltration in the peripheral region, and no distinct perioperative complications were observed. Therefore, the cause of the postoperative detrusor underactivity remains unknown. However, as the incidence of detrusor underactivity after laparoscopic radical prostatectomy was only 9.1% in our study and this incidence was lower than that in previous reports[17,18,21] for open radical prostatectomy, the minimal invasiveness of laparoscopic radical prostatectomy may be suggested as the reason for the low incidence.

All of the 10 patients with postoperative detrusor underactivity voided without residual urine and their maximum flow rates were excellent. Moreover, almost no incontinence was observed in the patients, that is, the postoperative detrusor underactivity did not clinically cause any serious

problems in the 10 patients. However, since the postoperative detrusor underactivity is considered to be irreversible, the upper urinary tract function must be carefully monitored over long periods of time.

Although the number of patients included in our study was small, we consider that patients with subjective voiding difficulty, residual urine, and strain voiding must be evaluated using the PFS. In the future, manipulations during radical prostatectomy should be investigated as the probable cause of the development of postoperative detrusor underactivity.

Conclusion

Among the 110 patients who underwent laparoscopic radical prostatectomy, de novo detrusor underactivity was observed in 10 patients (9.1%). Although the lack of detrusor contractions was compensated for by the abdominal pressure and no residual urine was observed, detrusor underactivity persisted for a long time after the operation and was considered to be irreversible. Preoperative detrusor contractions were excellent, so detrusor underactivity was considered to develop as a result of surgical manipulations during laparoscopic radical prostatectomy. However, the exact cause of postoperative detrusor underactivity could not be determined.

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