Chronic Spinal Cord Injury with Intractable Urinary Incontinence

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CASE REPORT

A 58-year-old woman had suffered from urinary frequency and urge incontinence for 5 years. She had had an epidural abscess that resulted in C5 incomplete tetraplegia. Although she can walk with a crutch, her voiding problem causes her much distress. Her voiding volume was around 50 mL and she had to void every half an hour. Residual sensation, urgency and urge incontinence could not be relieved even though she took a lot of medicine to control her overactive bladder. Severe bladder spasm, headache and increased spasticity of her extremities were also noted when she held urine for a short time. Clean intermittent self-cathe-terization (CISC) was used to empty her bladder but the post-void residue (PVR) was around 20 mL. These intractable symptoms prohibited the patient from going outdoors and from working, and she became depressed.

Currently, she has to have an indwelling catheter in place during sleep. She barely drinks water in the daytime. However, she still has to perform CISC more than 20 times during the day. Urodynamic study revealed detrusor overactivity with high voiding pressure and increased urethral sphincter activities during involuntary detrusor contractions. Voiding cystourethrography showed a contracted bladder with grade 1 right vesicoureteral reflux and external sphincter obstruction due to detrusor sphincter dyssynergia (DSD) (Fig. 1). Although no vesicoureteral reflux or hydronephrosis was noted, the diuretic renal scan for effective renal plasma flow (ERPF) showed delayed excretion from both kidneys (Fig. 2).

This patient was referred for further management. A videourodynamic study was performed at her admission. The result showed a contracted bladder with detrusor overactivity and DSD. The voiding pressure was high and she could not empty her bladder completely. Intravesical botulinum toxin A (BTX-A) injections or augmentation enterocystoplasty were recommended and she chose the latter for a permanent correction of her DSD. Augmentation enterocystoplasty was performed with a 40 cm segment of terminal ileum. During the operation, the bladder was found to be relaxed and the capacity during anesthesia was around 250 mL. A Foley cathe-ter was inserted for 2 weeks after surgery. During the first postoperative week, frequent strong detrusor contractions occurred and she was successfully treated with alpha-blocker and baclofen. Two weeks after the operation, the videourodynamic study was repeated immediately after removal of the catheter (Fig. 3). Small bladder capacity and poorly distended augmentation was noted. The patient had a limited increase in bladder capacity (100-120 mL) in the initial 5 days. Detrusor contractions occurred, and caused bladder discomfort and urinary incontinence at small bladder volume. Due to the limited progress in increase of bladder capacity, she was advised to receive intravesical BTX-A 200 U injections for improvement of capacity and also to prevent the contracture effect of the anastomosis.

During surgery, 200 U of BTX-A dissolved in 20 mL normal saline was injected into the bladder wall at 40 sites. By the third postopera-

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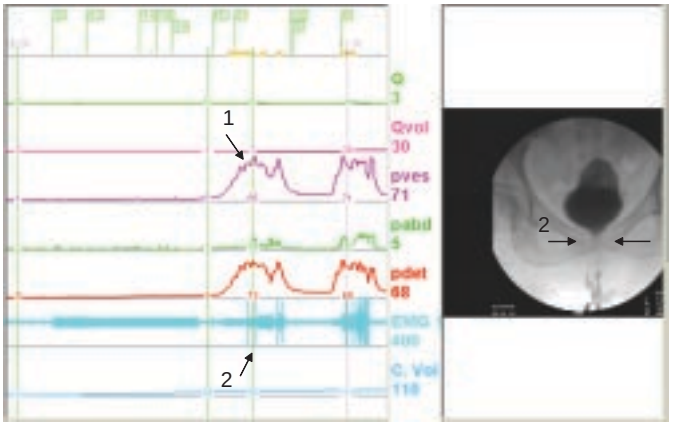

Fig. 1. Preoperative videourodynamic study revealed detrusor overactivity occurred at a volume of 70 mL (1) and urethral sphincter dyssynergia (2).

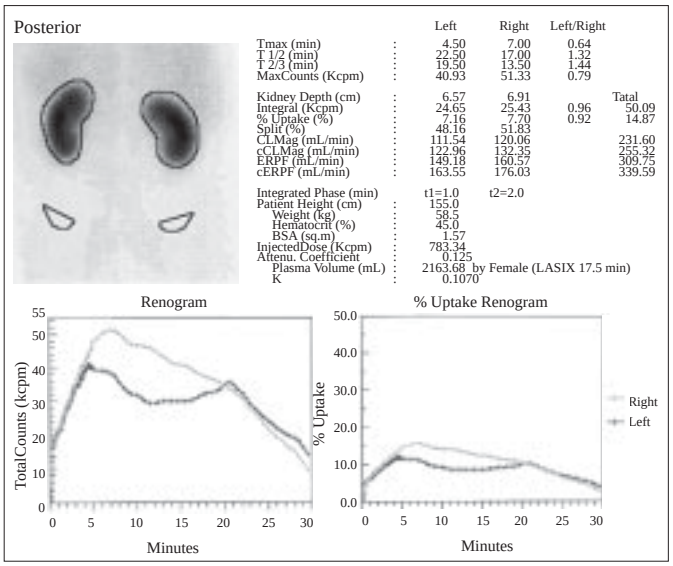

Fig. 2. ERPF study revealed normal renal function and a mildly delayed excretion of both kidneys.

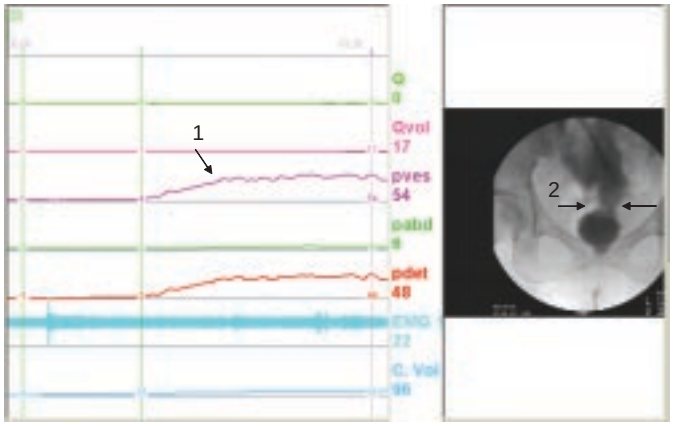

Fig. 3. Postoperative videourodynamic study revealed uninhibited detrusor contraction occurred at a volume of 36 mL; the detrusor contraction pressure was decreased (1) but a narrow anastomosis was noted in the enterocystoplastic bladder (2).

tive day, the bladder capacity had increased to 200 mL, and further increased to 350 mL by the 7th postoperative day. Although the patient could not empty her bladder, less bladder pain and less generalized discomfort were noted after the BTX-A injections. The PVR was around 250 to 300 mL and she had to catheterize herself 5-6 times a day.

COMMENTS

Chronic spinal cord injury at high level results in detrusor overactivity with DSD. DSD will cause functional bladder outlet obstruction and lead to a contracted bladder [1]. When the bladder trabeculation is severe, the vesicoureteral competence mechanism may be damaged and result in vesicoureteral reflux. From the urodynamic tracing and cystography, the bladder compliance was not poor, therefore aggressive medical treatment for detrusor overactivity might be helpful. However, when oral antimuscarinics fail or adverse effects are intolerable, intravesical treatment using resiniferatoxin instillation or BTX-A injection may be necessary to decrease detrusor contractility and increase bladder capacity [2]. Some patients might not benefit from such intravesical management of detrusor overactivity due to a severely contracted bladder or diminished bladder reserve capacity. Under such conditions, bladder augmentation using enterocystoplasty or autoaugmentation may be indicated to effectively increase bladder capacity and improve quality of life [3].

In this case, because the bladder capacity was rather small (less than 50 mL) and there was autonomic dysreflexia associated with a full bladder, intravesical BTX-A injection may have been tried first. Resiniferatoxin might not have been able to increase her bladder capacity to an adequate volume for a better quality of life due to limited improvement [4]. BTX-A, at a dose of 300 units, has been reported to increase bladder capacity to 2 to 3 times that of the baseline volume in neurogenic detrusor overactivity [5]. However, if intravesical BTX-A injection fails to obtain such a good result or repeat intravesical injections become an economic burden, bladder augmentation can be a good treatment choice.

Bladder augmentation using a segment of 40 cm of the terminal ileum can increase the bladder capacity to 600 to 1000 mL and the intravesical pressure can be reduced to less than 20 cm of water at 6 to 12 months after treatment [6]. Patients can regain continence and bladder fullness sensation through reduced intravesical pressure and obtain bladder sensation from the small intestine. Most of the patients can use abdominal pressure to void with adjuvant CISC before sleeping. The upper urinary tract function can be preserved and the quality of life improved. Mucus secretion, bladder stone formation, loose stools and other intra-abdominal symptoms might be possible complications of enterocystoplasty, although most of the complications can be resolved easily [7].

It was surprising that the bladder capacity did not increase rapidly after enterocystoplasty. One possible cause is the persistent contractions of the bladder after surgery. The bladder was activated due to increased sensory activity after cystotomy. Although the bladder was opened into a clam shape and enterocystoplasty was performed with a wide anastomosis, the anastomosis contracted due to persistent bladder overactivity and a small bladder volume remained after the operation. After surgery, as we can see, the enterocystoplasty had an hourglass appearance, which may prohibit pressure transmission from the bladder to the augmented part and cause bladder hyperreflexia and high intravesical pressure. Intravesical BTX-A injections can relax the bladder wall and prevent further contracture of the anastomosis. Once the augmented part has decreased in peristaltic pressure and increased the capacity, the intravesical pressure will be reduced. The result might have been better if we had injected BTX-A before the enterocystoplasty was performed.

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