Int Urogynecol J (1995) 6:31–33

International Urogynecology Journal

Case Report

Bladder Dysfunction After Repeat Laparoscopic Uterine Nerve Ablation (LUNA)

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Abstract: The authors report a case of voiding dysfunction with reduced sensation and areflexia 13 months after a repeat LUNA due to pelvic nerve injury. Anatomic distortion and increased vascularity were likely contributing factors. Repeat procedures may expose patients to a risk of such injury due to anatomic distortion.

Keywords: Laparoscopy; LUNA; Neural injury; Pelvic pain; Voiding dysfunction

Introduction

Laparoscopic uterine nerve ablation (LUNA) has been shown to provide pain relief in 60%–80% of patients with primary dysmenorrhea and endometriosis [1–3]. We report a case of persistent bladder dysfunction 13 months after a repeat procedure.

Case Report

A 29-year-old gravida 2 para 2 woman was referred to the combined gynecology/urology clinic of the University of Michigan Medical Center. She had been unable to void urine since undergoing a LUNA 10 months previously. She had no sensation of bladder filling and was self-catheterizing every 4–6 hours. She first underwent laparoscopy 5 years previously because of chronic dysmenorrhea and non-cyclic pelvic pain. This revealed filmy adhesions between the left lateral rectosigmoid junction and the adjacent pelvic side wall. Hysteroscopy and endometrial biopsy were also performed and were normal. Laparoscopy was repeated 12 months later because of continuing pain unresponsive to medical treatment. On this occasion a LUNA was performed using a potassium–titanylphosphate (KTP) laser. In addition a small stellate peritoneal scar on the left pelvic side wall was vaporized. There were no perioperative complications. The patient experienced symptomatic relief for approximately 3 months after this. Three years later the LUNA was repeated using a KTP laser. Once again the left-sided pelvic adhesions were noted. In addition, the greater omentum was adherent to the left uterosacral ligament. Laser adhesiolysis was performed. The left uterosacral ligament was thickened and the right was hypoplastic. Both uterosacral ligaments were divided at the level of their insertion into the cervix. Division of the left ligament proved difficult due to its thickness, vascularity and the presence of adhesions. It was also noted that the left ureter appeared to be lying lower in the pelvis, within 1 cm of the left uterosacral ligament. The left ligament was divided piecemeal and bleeding vessels were identified and coagulated without undue difficulty and with minimal blood loss. The patient was discharged from hospital later that day. She was readmitted 3 days later with overflow incontinence and painless urinary retention. Catheter drainage revealed a volume of 1700 ml. A Foley catheter was inserted and placed on continuous drainage. An intravenous pyelogram was performed and was normal. Pelvic ultrasonography revealed a 2.2 cm left-sided hematoma-like mass. The patient was comenced on carbachol, terazosin and intermittent self-catheterization. She was discharged

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from hospital 3 days later. Repeat ultrasonography demonstrated resolution of the mass. The patient experienced return of sexual orgasm at 3 months and some pelvic pain at 6 months, without improvement in bladder sensation or function.

Clinical examination 10 months after surgery revealed normal S2–S4 sensation and reflexes. A fluoroscopic-guided urodynamic study was performed. There was no sensation of bladder filling. There was some reduction in terminal compliance, with the bladder pressure rising from 10 to 21 cmH_2O between 400 and 500 ml. The bladder was areflexic. The urethral guarding reflex was normal, with a steady rise in maximal urethral pressure from 80 to 110 cmH_2O. Cystourethroscopy was normal. A concentric electromyography needle electrode was then placed in the right ventral quadrant of the external anal sphincter. Normal insertional activity and relative EMG silence at rest were observed. A normal recruitment pattern was also demonstrated in response to tightening of the anal sphincter. An 18 Fr Foley catheter with a bipolar stimulating electrode mounted at the tip was inserted into the empty bladder. Using a pulse duration of 0.2 msec and a frequency of 1–2 Hz the pelvic nerve was tested. The sensory threshold was elevated (120 V) and no evoked activity was detected on the anal sphincter EMG.

The patient was reviewed 3 months later. She had recently noticed some sensation of bladder fullness but had not voided spontaneously. Urodynamic evaluation revealed the first bladder sensation at 450 ml and mild discomfort at 600 ml. A reduction in terminal compliance and a normal urethral guarding reflex were once again noted. The bladder remained areflexic. Neurourologic testing of the pelvic nerve revealed an improvement in sensory modalities, with a threshold of 50 V and discomfort at 120 V. Once again, however, no evoked activity was detected on the anal sphincter EMG. A bipolar stimulating electrode was also applied to the clitoris. Stimulation parameters were as above. Both pudendal nerve sensory threshold and latency were normal.

Discussion

The autonomic innervation of the pelvic viscera is complex. Parasympathetic fibers arising from the S2 to S4 spinal cord level and sympathetic fibers from T11 to L2 give rise to the efferent or motor component of the pelvic and hypogastric nerves respectively. Afferent fibers also run in each nerve. The role of pelvic afferent fibers in the generation of the micturition reflex and the sensation of bladder filling is well established [4]. However, it is not clear whether sympathetic afferents have any important role in conveying bladder sensation of any type. This is supported by the findings of Learmonth in man, showing that only the pelvic afferents are important, and the observation by Torrens that no bladder sensory abnormalities follow sympathectomy [5,6].

The hypogastric nerves enter the pelvis in the presacral fascia 1–2 cm behind and below the ureter [7]. The parasympathetic pelvic nerves initially lie deep to the fascia that covers the piriformis muscle. At the level of the ischial spine they enter the posterior fascial sheath of the hypogastric visceral vessels, where they are joined by the hypogastric nerves to form the pelvic plexus [7]. In the female the bulk of this plexus lies below the broad ligament [7]. Pain fibers, which are stimulated by bladder distension, spasm or inflammation, are carried predominantly by parasympathetic fibers [8]. Both uterine and bladder afferent pathways pass through the pelvic plexus. Even with gross dissection of the uterosacral ligaments, neural components may be difficult to identity [9]. At their cervical attachments there is a preponderance of sympathetic fibers, in addition to a rich vascular supply [9]. The afferent supply to the feline uterus has been demonstrated to travel in sympathetic fibers, whereas cervical afferents are found in the pelvic and pudendal nerves [10]. Counsellor noted that the sympathetic fibers entered the uterus through the uterosacral ligaments [11]. It is, however, likely that a proportion of uterine afferents in women lie outside the uterosacral ligaments, thus accounting for postoperative failures of both open and laparoscopic uterosacral denervation procedures [2,12].

Although bladder dysfunction has not been described after LUNA, both bladder and bowel dysfunction have been reported in up to 10% of presacral neurectomies [13]. Gynecologic-related bladder dysfunction is, however, more classically associated with radical hysterectomy. Postoperative voiding dysfunction characterized by a poorly contractile or acontractile detrusor commonly occurs. Although traditionally this has been attributed to bladder overdistension injury [14,15], there is no good evidence for this as the mechanism of detrusor dysfunction. In fact, it has been shown experimentally that short- or long-term overdistension produces only transient nerve terminal changes and no changes in visceral smooth muscle [16,17]. Parasympathetic denervation may eventually give rise to a poorly compliant areflexic bladder, which may lead to upper tract deterioration [18]. Simultaneous sympathetic neural injury may lead to reduced urethral pressures [19]. Less extensive removal of the lateral and inferior segments of the cardinal ligament at radical hysterectomy minimizes the risk of bladder neural injury [20]. Neurourologic abnormalities, however, have been reported in over 20% of patients after simple total abdominal hysterectomy [21].

In the case described above, repeating the LUNA procedure apparently resulted in injury to the pelvic nerve afferent and efferent neurons. Anatomic distortion and increased vascularity were the likely contributory factors. The major motor neuron producing intraurethral pressure is the pudendal nerve, which innervates the rhabdosphincter. The pudendal nerve is not implicated in the injury described in this case report.

The other motor nerve involved in the reflex is the efferent sympathetic neurons travelling, not only in the pelvic plexus, but also in the sympathetic chain. The afferent neurons involved in this reflex arc are carried in the pelvic nerve. It is unlikely that this patient's injury was 100% complete. This is compatible with the clinical symptoms and neurourologic findings of diminished bladder sensation, areflexia and a normal guarding reflex. Many afferent nerves do not carry sensory information which can be appreciated at a cortical level: a small number of intact afferent neurons may activate an intact efferent guarding reflex and the micturition reflex can be totally blocked with a selective cryoneurolysis, which preserves many of the afferent and efferent neurons [22,23]. In addition, there is also evidence to suggest that local reflex pathways, which might not be injured in this circumstance, may be responsible for the guarding reflex [24]. Although postoperative bladder overdistension did occur, it was not the primary injury. Painless retention of 1700 ml in this case is virtual proof of a significant prior bladder denervation; if overdistension injury actually occurs, the only pathological evidence of associated neural injury would be a transient afferent neuronal degeneration, which would be expected to recover very quickly.

To date, the results of repeat LUNA procedures have not been widely reported. In the light of this case we feel that there may be a significant risk of neural injury associated with repeat procedures.

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EDITORIAL COMMENT: The authors report detrusor areflexia and reduced sensation, as evidenced by delayed sensation of filling and a high sensory threshold to electrical stimulation, in a patient subsequent to a second uterosacral nerve ablation. As the authors describe, injury to the hypogastric nerve must have resulted from the procedure(s). Since the original description by Ingelman-Sundberg (*Acta Obstet Gynecol Scand* 1959;38:145–153) of transvaginal hypogastric nerve resection for the treatment of urge incontinence (unstable bladder), the effect of hypogastric nerve damage has been well known. It is certainly unusual to find detrusor dysfunction after uterosacral ligament transection, in that this procedure accompanies each hysterectomy and the incidence of this problem nowhere approaches that of hysterectomy. Anatomical distortion from the previous procedure must have played a role in the genesis of this injury. The continued return of function indicates a partial injury with a positive prognosis.