HHS Public Access

Author manuscript

Obstet Gynecol. Author manuscript; available in PMC 2016 May 17.

Published in final edited form as:

Obstet Gynecol. 2012 February ; 119(2 Pt 2): 428–431. doi:10.1097/AOG.0b013e31822c96cb.

Genitofemoral and Perineal Neuralgia After Transobturator Midurethral Sling

Brent A. Parnell, MD, **Elisabeth A. Johnson, NP, PhDc**, and **Denniz A. Zolnoun, MD, MPH**

Department of Obstetrics and Gynecology, Divisions of Female Pelvic Medicine and Reconstructive Surgery and Advanced Laparoscopy and Pelvic Pain, the University of North Carolina at Chapel Hill, Chapel Hill, North Carolina

Abstract

Background—Midurethral slings successfully treat stress urinary incontinence through a minimally invasive vaginal approach. Postoperative pain related to sling placement can occur and poses both diagnostic and treatment dilemmas.

Case—Four years after transobturator midurethral sling placement, the patient presented with complaints of left labial pain and dyspareunia since surgery. Using sensory mapping and a nerve stimulator, the problem was identified in the distribution of the genitofemoral nerve. Conservative therapy with a centrally acting neuromodulatory drug and nerve block relieved the pain.

Conclusion—Postsling neuralgia diagnosis using sensory mapping and a nerve stimulator aids in indentifying the nerve involved and in successful conservative treatment with a nerve block.

Post operative pain, including dyspareunia, is one of the most common complaints associated with pelvic reconstructive procedures.[1] Surprisingly little is known about the prevalence of postoperative neuropathy after gynecologic procedures. In transobturator sling procedures, postoperative peripheral neuropathies are well described, with a rate of 9.4%.[1] To date, the predisposing risk factors, etiology, and mechanism of this postoperative pain remain poorly understood. We describe a case of postoperative neuropathy likely related to trauma to the genitofemoral nerve after a transobturator sling procedure.

Case

A 57-year-old woman presented with a 4-year history of sharp left labial pain. The pain began immediately after placement of an out-to-in transobturator sling. The procedure was performed for treatment of stress urinary incontinence (SUI). She remembers that, when she first tried to stand in the recovery room, she felt as though a razor sliced through her left labia and upper groin and as if a needle remained in the vaginal area.

Over the subsequent 4 years, the patient continued to experience sharp left upper labial pain when moving her left leg. Triggers included sitting, crossing her legs, and changing

Corresponding author: Denniz A. Zolnoun, MD, MPH, Old Clinic Building, Campus Box 7570, Chapel Hill, NC 27599-7570; denniz_zolnoun@med.unc.edu.

Financial Disclosure: The authors did not report any potential conflicts of interest.

positions. The pain radiated down the medial thigh to her foot. Friction on the labia also increased the pain, so she used daily lubrication to prevent rubbing. She also experienced dyspareunia with a dullness and numbness, preventing orgasm. She was started on carbamazepine and noticed a decrease in the intensity of the pain from excruciating to a dull, irritating sensation. Notably, before this surgery, she had no history of pain and maintained a satisfying sex life.

Dorsiflexion of the left ankle replicated the pain, and the patient rated the pain as a 1–2 on a 10-point Likert pain scale. Elevation of the left knee increased the pain to a 2–3, whereas flexion of the thigh increased the pain to an 8–9. On bimanual examination, the shooting pain returned during palpation of the medial ridge of the inferior pubic ramus on the left. Transvaginal palpation 2 cm lateral to the pubic ramus (corresponding with the anatomical location of genitofemoral nerve) produced the same pain and associated radiation to the left medial thigh.

Sensory testing showed hypoalgesia to gentle stroke and mechanical hypoalgesia in the S3 distribution of the perineal region as well as hypoalgesia of the lateral aspect of the foot, inner aspect of the leg, and medial thigh. This also was seen in the genitofemoral nerve distribution on the mons during pinprick assessment, where the patient had areas of decreased sensation interspersed with areas of hypersensitivity to pinprick. She also exhibited focal hyperalgesia in the perirectal region in the S3 distribution. Finally, abnormal sensory perception was noted while moving from the lateral vulvar skin of the labia majora to the more medial aspect of the vulvar mucosa (vestibule) on the left. Normally, the mucosa is less sensitive to pinprick perception compared with hairy skin.

After discussing treatment options, the patient opted for empiric trial of a nerve block performed on the left genitofemoral nerve along with continuing the carbamazepine. In the suprapubic region, two finger breadths above the pubic symphysis and two finger breadths lateral to the midline was infiltrated with 5 cc of 1% lidocaine along the skin and down to Scarpa's fascia. Subsequently, an 80-mm blunt-tip needle was inserted and carried toward the fascia using a nerve stimulator with a current of 2.5 mA, which gradually was increased to 4 mA to reproduce the painful burning sensation. Once the pain was reproduced, the area was injected with 10 cc of a 1:1 dilution of 1% lidocaine and 0.5% ropivacaine. The vaginal hand assisted in directing the needle to ensure full coverage. After the block, repeat assessment confirmed full anesthetic effect, defined as absence of pain with left hip abduction and lateral rotation; before the injection, the patient had been unable to perform these movements because of pain.

Six weeks later, the patient returned for a second block and reported a sustained improvement in her pain of approximately 50%. Pressure applied in the suprapubic region over the genitofemoral nerve reproduced the pain but to a lesser degree than during the previous visit. Sensory mapping revealed continued hypoalgesia along the genitofemoral distribution on the left side in addition to a new finding of numbness in the perineal nerve distribution on the left. A block then was performed in similar fashion to the previous block of the genitofemoral nerve from the suprapubic approach along with a diagnostic block of the perineal branch of the pudendal nerve (in light of the new finding) using the transvulvar

approach. Pressure in the region of the genitofemoral nerve no longer produced pain, confirming a full block. During follow-up of the perineal nerve block, there was no associated improvement, suggesting that the sensory abnormalities in the perineal area were a centrally mediated process (rather than a peripheral process).

Currently, the patient's pain remains well controlled, with a subjectively reported 75% improvement from baseline using carbamazepine alone. To date, she has undergone a total of five blocks: two diagnostic (attempting to identify the offending peripheral nerve[s], genitofemoral compared with perineal) and three therapeutic genitofemoral nerve blocks. Before the patient's most recent block, her sensory mapping was drastically different, revealing that the original region of hyperalgesia as mapped with gentle stroke of a cotton swab was absent. She continued to have an area of hypoalgesia focalized to the ipsilateral mons region. Otherwise, the dermatomal distribution of sensation was normalized. Similarly, examination of the legs revealed more than 80% normalization with remaining patchy hypoalgesia in the inner thigh and the lateral aspect of the big toe.

Comment

Vulvar innervation involves multiple nerves from various levels of the spinal cord. The pudendal nerve carries motor, sensory, and autonomic fibers to the pelvis from the sacral roots S2–S4. The genitofemoral and ilioinguinal nerves originate from L1–L2 and have sensory innervation of the mons pubis, thigh, and labia majora (Fig. 1). Coverage of the vulva also can include the inferior cluneal nerve, which originates from S1–S3.[2] Pain resulting from entrapment or injury of the genitofemoral or ilioinguinal nerves can present with localized vulvar pain or as lower-extremity pain. Sensory innervation of the lower extremities and perineum includes the lumbar nerve roots, so referred pain to various lower extremity structures can aid in identifying the offending nerve.

Injury to nerves can result in a variety of responses, including hyperalgesia (increased sensitivity to pain), hypoalgesia (decreased sensitivity to pain), allodynia (pain response to nonpainful stimuli), and paresthesias (tingling or pins and needles sensation).[2] For various neuropathic sensory disturbances, the response can be any combination of these expressions of pain, which represents an ongoing neuropathic pain response necessitating ongoing therapeutic intervention. Two distinct mechanisms interact to produce the disparate responses within a single patient: central sensitization causing hyperalgesia and peripherally mediated painful hypoalgesia.[3] Identification of those with severe neuropathic-type pain in the acute postoperative period would allow for early intervention, improving the patient's chance for recovery. Carbamazepine successfully treats the centrally mediated portion of acute and chronic pain in many patients,[4] and local anesthetics may improve the peripheral component. If compression is thought to be the origin of the neuropathic pain, early decompression also could preserve nerve function while improving pain.[5]

Training for pelvic surgery typically does not prepare physicians for postoperative diagnosis and management of neuropathic pain. The majority of these patients present with no motor deficit, making them difficult to diagnose because diagnosis requires a detailed neurologic examination. In 2010, it is estimated that 18 million women had urinary incontinence; by

2050, an estimated 28 million women will have this disorder.[6] Slings continue to be the most commonly performed surgery for SUI. If pain affects even a small percentage of women undergoing this procedure, the prevalence of postoperative neuropathic pain will increasingly demand the attention of clinicians. Many gynecologists may be comfortable managing immediate perioperative pain, but, until we understand more about this process, women with chronic pain may benefit from care by a specialist who treats chronic pain.

Midurethral slings continue to be the mainstay for treatment of SUI, with long-term success rates of 90%.[7] The goal of treating SUI is to improve quality of life by decreasing symptoms of incontinence, which can be troublesome. The type of pain experienced in the case described here and in other women with postsling pain can eradicate the improvement in quality of life seen after treatment of SUI symptoms. Clear strategies to treat this type of pain must continue to be developed to help women with postsling neuralgia. Managing postoperative pain after placement of midurethral sling typically involves expectant management with follow-up, physical therapy, anti-inflammatory medications, “trigger point” injections, and surgical excision of the mesh. Neuralgias, such as in the current case, tend to cause chronic, burning pain in the distribution of a specific nerve and may best respond to conservative treatment using neuroleptic medications, physical therapy, and nerve blocks to manage symptoms. This case demonstrates how careful diagnosis of the involved nerve, in conjunction with regional nerve block, may improve chronic pain in a patient with postsling neuralgia.

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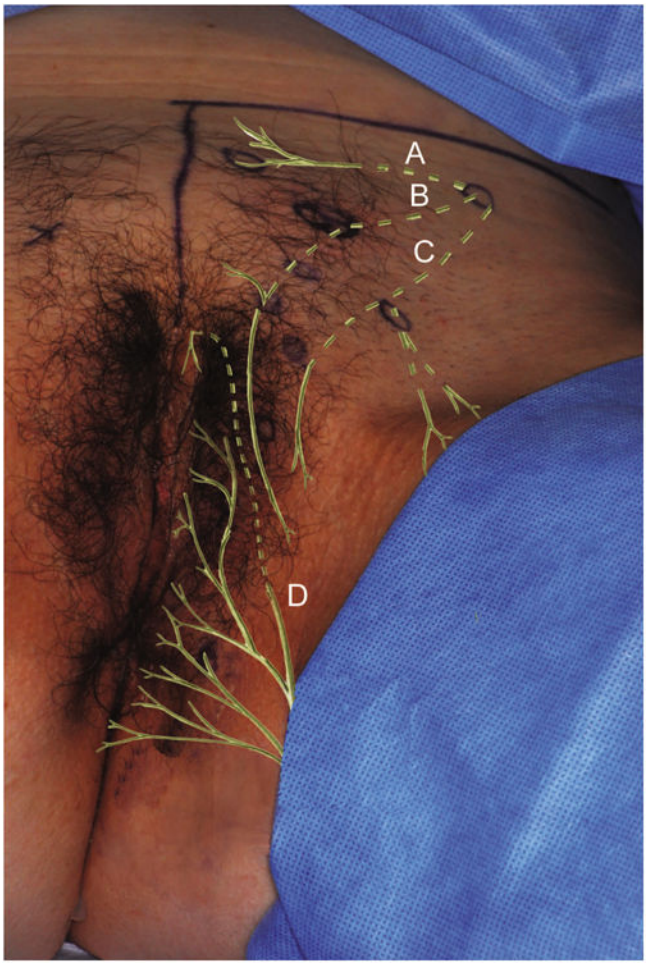

Fig. 1.

A. Anterior cutaneous branches of the iliohypogastric nerve. **B.** Anterior labial branches of the ilioinguinal nerve. **C.** Genitofemoral nerve (both the genital and femoral braches). **D.** Dorsal nerve of the clitoris (continuation of pudendal nerve shown as *dashed lines* deeper in the muscles of the urogenital diaphragm). The course of the specified nerves is delineated based on quantitative sensory testing and selective nerve block in this patient.