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Review Article

Tension-free Vaginal Tapes and Pelvic Nerve Neuropathy

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ABSTRACT Obturator nerve neuropathies after tension-free vaginal tape or transobturator tape are considered to be caused by nerve trauma, although it is unclear whether these are accidents or whether these injuries are inherent to the procedure of tape insertion. Two cases show that obturator nerve neuropathy can occur after tension-free vaginal tape without direct trauma to the obturator nerve possibly as a consequence of excessive fibrotic reaction or persisting low-grade inflammation. PubMed Entrez, Cochrane Library, and up-to-date databases were searched for obturator and pudendal neuropathy and for neuropathies associated with tension-free vaginal tape–transobturator tape and the symptoms, diagnosis, and therapy of the pudendal and obturator nerve neuropathies are reviewed. Based on data, our experience, and data available in literature, we can conclude that, if conservative obturator nerve block confirms the diagnosis of obturator nerve neuropathy and symptoms recur shortly thereafter, a laparoscopic neurolysis can be proposed as therapy. Journal of Minimally Invasive Gynecology (2008) 15, 262–267 © 2008 AAGL. All rights reserved.

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Entrapment neuropathy may occur whenever a nerve passes through a narrow fibrous tunnel such as the obturator canal for the obturator nerve or Alcock canal for the pudendal nerve. Both the obturator nerve entrapment syndrome and the Alcock syndrome can be idiopathic or can be a result of compression.

Nerve injuries are classified into 3 groups [1]. Neuropraxia is a nerve contusion causing a functional injury with recovery within 6 weeks [2]. Axonotmesis occurs when the neural elements distal to the injury sites are exposed to wallerian degeneration, while the supporting structures remain undisturbed with functional recovery within 6 months to 1 year [3]. Neurotmesis is the most severe injury with a complete interruption of the nerve [1].

In gynecology, most nerve injuries are iatrogenic. Accidental lesions of the femoral or obturator nerve are well-known complications of oncologic surgery. Evidence has accumulated during the last decade concerning the importance of the orthosympathetic and parasympathetic nerves, especially during oncologic and severe endometriosis surgery, and nerve-sparing surgery was introduced. Neuropathies caused by endometriosis, especially of the sciaticus with monthly sciatic neuralgia, were recognized for a long time but the direct evidence has remained scarce. Only recently has the dissection of the sacral roots suggested that pain mediated by endometriotic nerve compression or invasion might be more common than anticipated.

Obturator and pudendal nerve neuropathy are considered rare but well-known pathologies in gynecology. Recently, pudendal nerve neuropathy was related to sling procedures for stress urinary incontinence such as tension-free vaginal tape (TVT) and transobturator tape (TOT). Two personal cases and the recent debate in the Listserv of the American Association of Gynecologic Laparoscopists prompted us to review symptoms, diagnosis, and therapy of the pudendal and obturator nerve neuropathies together with the available

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evidence that sling procedures might cause obturator neuropathy.

Review of Obturator and Pudendal Nerve Neuropathies

Obturator Nerve Anatomy

The obturator nerve arises from the second, third, and fourth lumbar nerve roots. These fibers unite in the posterior part of the psoas muscle and descend outward and downward over the sacrum or pelvic brim to the obturator canal, after which the nerve divides into an anterior and posterior division. The anterior division innervates the skin of the medial proximal thigh, the hip joints, and the pectineus, gracilis, adductor longus, and brevis muscles. The posterior division supplies the obturator externus and adductor magnus (occasionally the adductor brevis also), and carries sensory afferents from the knee joint [2]. An accessory obturator nerve, found in 8% to 30% of women [3], supplies the pectineus and the hip joint, and communicates with the anterior division of the obturator nerve [4].

Obturator Nerve Neuropathy

The obturator nerve can be compressed in the pelvis but especially in the obturator tunnel, where idiopathic compressions were reported [5–8]. Other causes of entrapment are pelvic fractures [9–11], compression by malignancies [12], endometriosis [13], obturator hernias [14–16], and rarely by an aneurysm of the hypogastric artery [17] or by a cyst of the hip [18,19]. Obturator nerve injury can occur during delivery, especially forceps delivery [20–22], and is known as obstetric obturator nerve palsy. A single case report related obturator tunnel syndrome to an osteitis of the pubic bone [23]. Most obturator nerve injuries, however, occur during obturator hernia repair [14,24], hip surgery [25–27], or during TVT suburethral sling procedures [28,29], and other pelvic surgery [30–35]. The lithotomy position can strain the obturator nerve [36], although rarely [37]. Obturator injury during laparoscopy surgery is rare, with only 3 reports of nerve fulgurations during pelvic lymphadenectomy [38,39] and 1 nerve injury during tubal ligation, presumably by accidental coagulation [40].

The symptoms of a nerve section are those derived from innervation. Most reports describe obturator neurapraxia and axonotmesis with more vague symptoms. The Howship-Romberg sign refers to pain extending in the inguinal region and anterointernal side of the thigh, going down to the internal side of the knee and, occasionally, into the hip. This pain typically is exacerbated by extension and abduction or inward thigh rotation. Important concomitant signs are increased pain by putting weight on the lower extremity and impeded walking [41]. In addition, sensory disorders can occur such as paresthesia (e.g., tingling sensations, numbness, or pins and needles), electric shocks, burning sensations, allodynia, and intolerance to skin contact (e.g., from wearing clothes) [27,41]. Clinical examination can reveal dysesthesia to contact and hypoesthesia to pinpricks or heat and cold, whereas motor disorders are generally minor or absent, except in rare cases of motor loss in the adductor muscles. Diagnosis is essentially clinical, and other examinations contribute little to the diagnosis. Signs of denervation on electromyography are not very specific whereas standard radiography, magnetic resonance imaging, and computed tomography scans can only diagnose a tumor or obturator or lumbar disk hernia, which compresses the nerve. Important is the local infiltration with an anesthetic, which is diagnostic and can be therapeutic [11,42–47].

The treatment of choice is the obturator nerve block, with similar results whether performed as ultrasound or computed tomography guided [7,43–46]. Although the available data are limited, it is suggested [2] that obturator nerve block is more effective for acute-onset obturator neuropathy [26]. If ineffective or for severe injuries [27], especially when associated with pelvic trauma or when an intraoperative laceration is suggested, surgical exploration is necessary. If necessary, nerve grafting and repair may be attempted [47,48]. Otherwise, total nerve section or destruction by radiofrequencies or cryoanalgesia may be considered [49–51].

Pudendal Nerve Anatomy

The pudendal nerve arises from sacral nerves S2, S3, and S4; traverses the sacral foramen; and divides into autonomic and somatic nerve fibers. The autonomic part forms the pelvic plexus that innervates the bladder and the urethral smooth muscle. The somatic fibers of the S2 to S4 roots form the pudendal nerve. Exceptionally, some somatic fibers from S2 and S3 travel with the pelvic plexus, and innervate the levator ani muscle and the striated external urethral sphincter [52]. The main trunk of the pudendal nerve is formed just proximal to the ischial spine medial and caudal to the sciaticus, curves around the ischial spine, travels between the sacrospinous ligament and the sacrotuberous ligament, to enter the ischiorectal fossa where the pudendal nerve travels in Alcock canal, which is a fascia sheath formed by duplication of the obturator fascia below the falciform process and the insertion of the sacrotuberous ligament [53,54]. In most cases the inferior rectal nerve arises from the pudendal nerve at the beginning of the pudendal canal [55]. In the pudendal canal the pudendal nerve divides into the perineal nerve and the dorsal nerve of the penis or clitoris. The former divides into several motor and sensory branches that innervate the perineum, the perineal muscle, and the external anal sphincter. The latter perforates the transversus perineal muscles to enter the shaft of the penis or the clitoris [56].

Pudendal Nerve Neuropathy

Pudendal nerve neuropathy is generally caused by pudendal nerve entrapment, which can be idiopathic or a

consequence of compression (e.g., biking, sitting for an extended period of time, hematoma, pelvic varices [57], pelvic floor prolapse, endometriosis, neoplastic compression) or stretching (e.g., descending perineum, pelvic and vaginal surgery, delivery) in Alcock canal [58–66]. Contributing factors are a change in the shape or orientation of the ischial spine by some athletic activities during youth [67]. The sacrotuberous ligament can act like a lobster claw when the pudendal nerve traverses the interligamentous space [68] and ligamentous expansions forming a perineural compartment [53] that can be tightly adherent and difficult to detach [56]. Other factors were described such as a dysmetabolic diseases favoring compression inside the canal [64], and postherpetic neuropathy, peripheral polyneuropathy, or postradiotherapy neuropathy [69].

The diagnosis of pudendal nerve neuropathy is essentially clinical, although the diagnosis remains difficult in the absence of pathognomonic clinical symptoms, tests, or imaging [70]. Only the operative finding of nerve entrapment and postoperative pain relief can confirm the diagnosis of Alcock syndrome [71]. In very severe cases diagnosis could be made by pudendal nerve terminal motor latencies [72] or denervation signs on electromyography. Therefore, a multidisciplinary working party has set forward a set of diagnostic criteria (Nantes criteria) [70] emphasizing the difficulty of diagnosis. The 5 diagnostic criteria are: (1) pain in the anatomic territory of the pudendal nerve; (2) that the pain increases by sitting; (3) that the patient is not woken up by the pain; (4) no objective sensory loss on clinical examination; and (5) a positive anesthetic pudendal nerve block.

Exclusion criteria are: purely coccygeal, gluteal, or hypogastric pain; exclusively paroxysmal pain; exclusive pruritus; and presence of imaging abnormalities that justify the symptoms (imaging can reveal an intercurrent disease clearly unrelated to the neuralgia, for which treatment will not modify the course of the neuropathy). Associated symptoms as buttock pain upon sitting, referred sciatic pain, suprapubic pain, urinary frequency and/or pain on a full bladder, and dyspareunia can be present and do not exclude the diagnosis. Some clinical signs can help [73] such as a painful Alcock canal on rectal examination and a painful skin-rolling test (the skin of the perineum is pinched just beneath the level of the anus and then rolled to the front searching for a sharp pain at 1 level).

With a difficult and unclear diagnosis the first approach to therapy is conservative management with medical therapy [72,74] and pudendal nerve block [53,72], which are mainly effective in acute syndromes and often for a period of 1 to 3 months only. For chronic pudendal neuralgia, unresponsive to analgesia and nerve block, pudendal decompression surgery [72,73,75–81] should be considered. If decompression surgery fails, therapies as sacral ligament clamp [82], pudendal neuromodulation [72], or magnetic stimulation [83] have been suggested. When everything has failed, the surgical section of the pudendal nerve can be done by open [84] or laparoscopic [85] surgery.

Case Reports

Case Report 1

A 77-year-old nulliparous woman was seen with pain after a TVT. Her medical history was uneventful with total abdominal hysterectomy and bilateral adnexectomy for chronic pelvic pain 30 years previously. Two years earlier the patient had undergone a TVT for urinary stress incontinence, and some months after this intervention the patient had pain extending in the right inguinal region and anterointernal side of the right thigh, going down to the internal side of the right knee and hypoesthesia in the same region. Moreover, she typically had difficulty sitting because of pain in the lower buttock. The patient was treated with infiltration of local anesthetic around the obturator nerve. This confirmed the obturator nerve involvement but symptom relief was of short resolution only.

Our clinical examination confirmed the symptomatology of an obturator neuropathy, with specific tenderness over the obturator area during vaginal examination and buttock pain on sitting. Because of severe pain, impairing the quality of life, and because infiltrations were ineffective, surgical exploration was undertaken. By laparoscopy, the space of Retzius was opened and the obturator nerves were bilaterally identified. The right TVT mesh was identified and dissected from the pubic bone and Cooper's ligament. During this dissection severe fibrosis was found around the mesh, extending over a large area up to the obturator nerve. At no place did the mesh come in closer proximity than 1.5 cm of the nerve. A real compression or kinking of the right obturator nerve was not found, but after dissection the distance between the tape and the nerve had increased from 1.5 to 2.5 cm. Subsequently, the right part of the mesh was resected from 1.5 cm above the urethra up to the fascia. The remaining mesh was fixed to the Cooper's ligament as done during a Burch procedure to maintain the tension on the urethra. The postoperative period was uneventful except urinary retention that was resolved spontaneously in a few days with bladder training. Within 48 hours, while still in the hospital, the patient declared less pain and that she could sit again. At follow-up after 1 month the symptomatology was greatly improved and after 6 months the neuropathy was completely resolved.

Case Report 2

A 59-year-old woman, with medical history of a vaginal repair for cystocele and rectocele 8 years earlier, underwent a TOT procedure for urinary stress incontinence. During this procedure bleeding occurred from a superficial varicose vein on the left side, which was treated with bipolar coagulation and compression with a tampon. At 6 weeks after surgery the stress urinary incontinence had disappeared

completely, but the patient had dyspareunia, perineal pain on the left side that increased when sitting, and urinary frequency with the sensation of a full bladder. These symptoms persisted after treatment with nonsteroidal anti-inflammatory drugs. An obturator nerve irritation was confirmed 12 weeks after surgery by the injection of a local anesthetic around the nerve, after which symptoms disappeared immediately. Surgical exploration was planned but as a last resort a conservative therapy with local infiltration with corticoids (methylprednisolone acetate) and local anesthetic (lidocaine) was attempted. After a first injection symptoms disappeared for 10 days. Therefore, a second infiltration was performed after which symptoms disappeared for some 20 days. After 2 further infiltrations the symptom of obturator neuralgia had completely disappeared without further recurrence. We, therefore, do not have surgical findings proving a mechanical obturator nerve neuropathy after TOT. This case report strongly suggests, however, that whenever obturator nerve neuropathy occurs after TOT/TVT, the diagnosis should be confirmed by local anesthesia and conservative medical treatment should be attempted. This patient was finally cured by 4 infiltrations with corticoids and a local anesthetic.

Discussion

Minimally invasive suburethral sling procedures such as TOT and TVT are the standard surgical treatment in women with stress urinary incontinence, affecting some 13 million adult women in the United States [86]. The TVT procedure was introduced in 1996 and in 1999 with an initial 2-year cure rate of 84% [87,88]. Transobturator tape procedure "outside-in" was introduced [89] in 2001 with a 1-year cure rate of 84% [90]. In 2003, the transobturator technique "inside-out" (TVT-O) [91] was introduced [92,93] with similar results. Complication rates are low and include bladder perforation [92,94], urge incontinence [95], vascular injuries with an obturator hematoma or a retrosymphysial hematoma [94,95], external iliac vein perforation (1 case reported during TVT-O) [96] and external iliac artery laceration (1 case reported during TVT-O) [97], vaginal and urethral erosion [95], bowel injuries [97], and ilioinguinal nerve entrapment (1 case reported during TVT) [98]. All obturator nerve neuropathies reported were caused by nerve injury during tape insertion (i.e., in 25 cases after TVT-O [99,100], groin and thigh pain were reported; in 4 cases the diagnosis of obturator neuropathy [101] was made, in 2 cases after TOT [102] and in 3 cases after TVT [99,100]). In conclusion, nerve injuries during tape insertion were described although it is unclear whether these were accidents or whether these injuries were inherent to the procedure. Vascular and other lesions, moreover, document that erroneous insertions, much too lateral, occur.

To our knowledge, obturator nerve neuropathies developing progressively after TVT/TOT were not yet reported. We also expected to find in the first case report a tape inserted much too laterally in close proximity of the obturator nerve. This case, therefore, is the first unequivocal laparoscopically proved indication of an obturator neuropathy after a correctly placed TVT with at least 1.5 cm distance between the mesh and the nerve. The causal relationship is, moreover, proved by the disappearance of symptoms after dissection and mesh resection. We can only speculate about pathophysiology. It might be that the fibrotic reaction around a mesh is exaggerated in some women or that a low-grade inflammatory reaction can cause obturator nerve irritation. The fact that we only could retrieve 1 second case, although not documented by laparoscopy, and that during the discussion at Listserv only 5 cases, also poorly documented, were discussed, at least strongly suggests that obturator nerve neuropathy, besides the injuries, after TVT/TOT must be an extremely rare complication.

This case report strongly suggests that obturator nerve neuropathy after TVT/TOT exists. The diagnosis should be suggested by the symptomatology and by the local painful examination of the obturator canal. If conservative obturator block confirms the diagnosis and symptoms recur shortly thereafter, a laparoscopic exploration with neurolysis and eventual partial tape resection should be considered by an experienced endoscopist given the potential difficulty of surgery.

In conclusion, obturator nerve neuropathies after TVT/TOT exist, probably caused by exaggerated fibrotic response, low-grade inflammation, or both. If not resolved by obturator block, a laparoscopic exploration should be considered.

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