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Percutaneous Femoral Nerve Stimulation for Overactive Bladder: A Case Report

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Background: Peripheral nerve stimulation has been used to treat overactive bladder. A possible mechanism of action may be retrograde neuromodulation of the parasympathetic system via the sacral plexus.
If the retrograde neuromodulation theory is correct, then stimulation of any nerve not arising from the sacral plexus should not improve overactive bladder symptoms.

Case Report: We present a case of femoral nerve stimulation for neuropathic pain. While the stimulation did not relieve the neuropathic pain, it did successfully treat our patient's hyperactive bladder.

Conclusions: The femoral nerve arises in part from L2, the lowest level of the sympathetic system. The sympathetic system is involved with relaxation of the bladder. This report shows that nerves not arising from the sacral plexus can relieve overactive bladder symptomology. A refinement of the retrograde neuromodulation theory may be called for, with the inclusion of sympathetic neuromodulation as an alternative means to treat hyperactive bladder.

Key words: Overactive bladder, tibial nerve stimulation, femoral nerve stimulation, peripheral nerve stimulation, mechanism of action, case report

BACKGROUND

Overactive bladder is defined as '"urinary urgency, with or without frequency and nocturia, with or without urgency or urinary incontinence, in the absence of urinary tract infection or other obvious pathology" (1). It is thought to affect approximately 17% of the population with a prevalence that increases with age (2). First-line conservative therapies include bladder and pelvic floor muscle training. Second-line pharmacologic therapies target the autonomic nervous system with the goal of relaxing the detrusor muscles. Examples include antimuscarinics and β3 agonists, such as mirabegron and vibegron. Being systemic agents, their side effect profile may limit compliance. More localized therapies include detrusor injections with botulinum toxins.

Within the last few decades, peripheral nerve stimulation has become accepted as a third-line therapy for overactive bladder. Initially, sacral nerve stimulation was regarded as the gold standard for overactive bladder that was resistant to more conservative therapies. However, since being approved by the US Food and Drug Administration in the late 1990s, advances in sacral nerve stimulation have been minimal (3). In addition, shortcomings exist including cost, device size, and radiation exposure when placing the lead near the sacral nerves. The greatest shortcoming of sacral nerve stimulation, however, is a complication rate estimated to be 30%-40%, leading to device removal and replacement (4,5).

Posterior tibial nerve stimulation has gained favor as

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a therapy which has limited complications and which is cost effective (4). The posterior tibial nerve is a distal branch of the sciatic nerve. The sciatic nerve arises from L4 to S3 and innervates the posterior lower extremity. Posterior tibial nerve stimulation is believed to provide retrograde neuromodulation to the sacral nerve plexus, leading to control of bladder hyperactivity, either by influencing bladder contraction or sphincter control (6).

Posterior tibial nerve stimulation has evolved since its inception in the early 2000s. Several different approaches are now employed to modulate the nerve, each with its own set of advantages and disadvantages. Both transcutaneous and percutaneous technologies have been employed. Transcutaneous surface electrode and percutaneous needle electrode tibial nerve stimulation techniques are minimally invasive. Both approaches rely on patients completing repeated sessions for maximum benefit. For example, a common protocol for transcutaneous tibial nerve stimulation technologies involves having patients complete a series of 30-minute weekly sessions (6).

We provide a case report of a patient who received relief from overactive bladder following femoral nerve (L2-L4) stimulation.

CASE PRESENTATION

A 22-year-old woman was bitten by a dog in the anterior left thigh with the bite puncturing the skin. Later, she noted pain at the site and developed atrophy of the thigh with hypersensitivity. Complex regional pain syndrome was ruled out and she was diagnosed with neuropathy of the left anterior femoral cutaneous nerve. She did not respond to physical therapy, acupuncture, topical lidocaine, or a lumbar sympathetic injection. Flexion of the hip reliably caused fasciculations in the left thigh. Walking, lifting and carrying worsened her pain.

Approximately 6 months after the incident, she noted urinary urgency and frequency. The extent of the urgency and frequency increased and she consulted with an urologist. She was documented to have frequency at one hour with leakage if waiting too long. She underwent a cystometrogram with electromyography and was diagnosed with overactive bladder. Mirabegron was not helpful.

There was not felt to be any causal relationship between the anterior femoral cutaneous nerve neuropathy and the overactive bladder.

Because of the failure of conservative therapies to help with her anterior femoral cutaneous nerve neuropathic pain, she consented to a trial of femoral nerve stimulation. A percutaneous lead (Curonix) was placed over the femoral nerve (Fig. 1). She underwent a 5-day trial of left femoral nerve stimulation. She did not get relief from her neuropathic pain, but the urinary frequency resolved. After completion of the trial, the urinary frequency increased gradually over several days, returning to its previous level of symptomatology.

DISCUSSION

Overactive bladder syndrome affects millions of people in the United States and is equally prevalent in men and women. Urinary urgency is the defining feature in overactive bladder syndrome and includes the desire to urinate, incontinence, and nocturia. However, out of those symptoms, patients find nocturia to be the most annoying (7).

Recent studies have shown that percutaneous sacral nerve and transcutaneous posterior tibial nerve techniques are similarly efficacious in treating overactive bladder (8-11). However, relapse symptoms can occur with these methods and maintenance schedules are necessary for continued effectiveness of the transcutaneous approach. To overcome these issues, novel implantable posterior tibial nerve stimulation technologies, utilizing either inductive or high frequency electromagnetic coupling to power the electrodes, have been developed to provide patients with more options when noninvasive measures fail and as an alternative to sacral stimulation (12).

Wang et al (2) performed a meta-analysis and systematic review to evaluate the safety and efficacy of percutaneous tibial nerve stimulation for overactive bladder (2). They looked at 28 studies enrolling a total of 2,461 patients. Outcomes included daily voiding frequencies, urgency episodes, and nocturia frequency. Percutaneous posterior tibial nerve stimulation for overactive bladder was found to produce statistically significant improvement in overactive bladder symptomatology.

Neural control of the bladder is extremely complex, involving both peripheral and central control (13). The peripheral control involves sacral parasympathetic nerves (S2-S4) dealing with contraction; sympathetic neurons traveling with the iliohypogastric nerve (T10-L2) dealing with relaxation; and sphincter control maintained via the pudendal (S2-S4) and coccygeal (S4-S5, Co1) nerves. Central autonomic control involves multiple areas in the brain. Thus, the concept of retrograde neuromodulation is introductory to a more sophisticated understanding of the mechanism of action of bladder control, yet to be

developed. Variations on the theory include inhibition of the micturition reflex, which would again require sacral stimulation (3,14).

Currently, the best evidence is for tibial nerve stimulation being effective for overactive bladder, although the pudendal nerve has also been studied (15,16). The implication is that feedback into the sacral plexus provides relief of an overactive bladder. If overactive bladder symptoms can be controlled by stimulation of a nerve interacting with the sympathetic, thoracolumbar, system, dealing with bladder relaxation rather than sphincter control or bladder contraction, that finding would provide evidence that the concept of retrograde neuromodulation needs to be refined and that sympathetic neuromodulation might be an option to treat overactive bladder.

Peripheral nerve stimulation, primarily sacral and posterior tibial nerve stimulation, has proven to be effective to treat overactive bladder. Posterior tibial nerve stimulation is believed to work by retrograde neuromodulation of the sacral plexus. We present a case where femoral, L2-L4, stimulation controlled overactive bladder symptoms. Interestingly, the lumbar sympathetic nerve innervation to the bladder originates from the T10-L2 nerve roots. This case suggests that a mechanism other than sacral nerve neuromodulation, likely through modulation of the sympathetic rather than parasympathetic system, is involved in the control of overactive bladder. The nature of this mechanism is not clear but is likely similar to medical treatment of overactive bladder, relaxing the bladder.

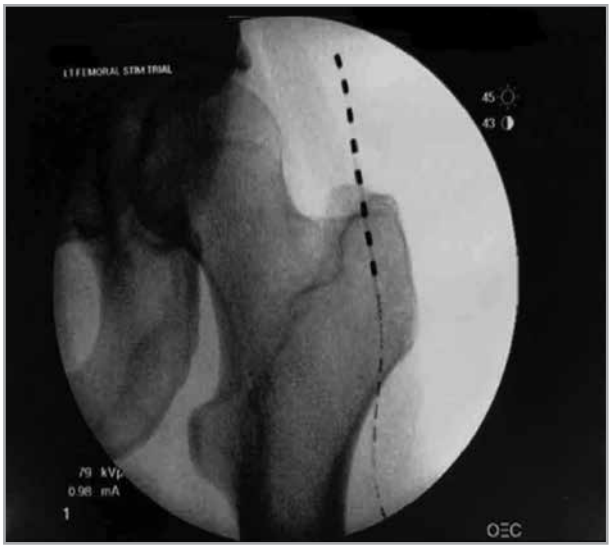

Fig. 1. Trial percutaneous nerve electrode, left femoral nerve.

CONCLUSION

Femoral nerve stimulation was not successful in treating this patient's neuropathy, but it did unexpectedly resolve her overactive bladder symptoms. This report suggests that the stimulation of sympathetic nerves may provide an alternative means of treating overactive bladder and that any theory attempting to explain how peripheral nerve stimulation affects an overactive bladder needs to include consideration of the sympathetic nervous system.

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