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Rare Adverse Effect of Spinal Cord Stimulation: Micturition Inhibition

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Objectives: In current medical literature, most of the reported complications of spinal cord stimulation concern technical problems, such as lead malfunction, migration, breakage, or internal pulse generator dysfunction, whereas reports about the side effects on internal organ function caused by spinal cord stimulation are rare.

Methods: In this clinical report, we describe uncommon side effects owing to spinal cord stimulation in a patient with chronic neuropathic pain. Our patient developed unexpected urinary retention during electrical epidural stimulation.

Results and Discussion: This case report highlights the incomplete knowledge about the mechanism of action of spinal cord stimulation and its influence on the interactions between the autonomic nervous system and voluntary control of urinary function. The complete recovery of bladder function after the interruption of stimulation suggests that electrical stimulation caused the adverse effects in this clinical case.

Key Words: spinal cord stimulation, SCS, side effects, chronic pain, internal organ function

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Spinal cord stimulation (SCS) was first used by Shealey et al[1] in 1967 and it is a key treatment option in chronic, neuropathic pain. It is accomplished by positioning an epidural electrode connected to an internal pulse generator that delivers low intensity electrical impulses to the posterior structures of the spinal cord. The mechanism of action of spinal cord stimulation is still not fully understood, and even if Melzak and Wall's Gate Control Theory remains one of the best explanations of SCS mechanism of action, not all effects of electrical stimulation of dorsal columns have been fully clarified. The placement of a spinal cord stimulator is usually offered to a patient with chronic pain, when other treatment options have proven to be ineffective. In current medical literature, most of the reported complications concern technical problems, such as lead malfunction, migration, breakage or internal pulse generator dysfunction, whereas reports about the side effects on internal organ function caused by SCS are rare.[2–4] In this clinical report, we describe the unexpected development of neurologic bladder and micturition dysfunction simultaneously with the onset of pain relief after the beginning of an electrical stimulation of the caudal segment of the spinal cord.

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MATERIALS AND METHODS

A 68-years-old male patient was subjected to laminectomy of the D12 vertebral body owing to an angioma causing compression of the spinal cord at the D12 level and resulting in cauda equina syndrome. Although neurologic functions were fully recovered within 3 days after surgery, the patient started reporting paroxysmal pain episodes limited to the lateral edge of the left foot and characterized by intermittent periods of sudden spontaneous pain bursts alternating with relief.

Pain episodes happened twice about every 2 hours (mainly during the night) and they were often associated with urinary bladder repletion, whereas pain relief followed micturition. Pain was evaluated as "strong" on the Verbal Rating Scale and as a 10 in intensity on the Numerical Rating Scale.

The patient was initially treated with pharmacologic therapy by combining oral gabapentin (900 mg/die) with amitriptyline (15 mg/die) and tramadol (400 mg/die), but it was not successful as the patient developed consistent side effects. He came to our Pain Unit for clinical evaluation 2 years after the symptoms began appearing. A physical exam at the pain site showed no anatomical abnormalities and no alteration of sensitivity in the lumbosacral dermatomes (no hypoesthesia, hyperesthesia, or mechanical or thermal allodynia). An MRI showed narrowing of the spinal canal (lumbosacral stenosis) with signs of an earlier spine surgery, but there was no evident sign of neural damage. Motor function and reflexes in the lower limbs and perineal muscles resulted not affected, nor was sphincterial function found affected. Neurophysiologic exams, including Somato Sensitive Evoked Potentials and Motor Evoked Potentials did not show any transmission abnormalities along somatosensitive spinal pathways and in motor pyramidal tracts, whereas Electromiography revealed an axonal neuropathy affecting the cauda roots, more on the left side, at the S1 to S2 level. Given the physical and instrumental examinations, the patient was diagnosticated with central neuropathic pain syndrome.

Initially, preexisting therapy was modificated by adding oxcarbazepine (300 mg bid), nortriptyline, and tramadol SR (200 mg/die). As pharmacologic therapy was not very effective and poorly tolerated, the patient,after informed consent,was submitted to a spinal cord stimulation trial.

A multipolar electrode (8 leads) was inserted using a paramedian approach at L3 to L4 interspace in posterior

epidural space up to the D12 level and slightly on the left side with respect to the sagittal plane. Intraoperative stimulating tests were conducted to provoke paraesthesias to assess the correct placement and functioning of the catheter with the subsequent settings: active electrodes 3(+) 4(−) 5(+), amplitude 2.9 V, pulse width 270 μseconds, frequency 60 Hz.

During the postoperative period, stimulation immediately produced complete pain relief (VAS 0) but was associated with the development of urge incontinence episodes about every hour. Three days after the implantation of the neurostimulator, acute urinary retention complicated the patient's clinical course, and the urologist diagnosed neurogenic bladder syndrome.

Since urinary retention was hypothesized to be induced by neurostimulation, a decision was made to stop the stimulation trial and to leave the electrodes in place. In the next 15 days, urinary retention persisted (treated by self-catheterization), the patient did not recover his bladder function and he referred the continuous absence of any stimulus to urinate. Pharmacologic therapy was interrupted too to prevent any other factor from affecting bladder function also because the pain relief was always constant and complete.

Twenty days after the interruption of the stimulation trial, pain appeared again with the same initial characteristics; and in the next 10 days, bladder function was completely recovered. After the complete recovery, the decision was made to start again the stimulation trial, whereas limiting the use of the stimulating catheter only to acute bursts of pain; however, despite this limited use, the neurogenic bladder dysfunction immediately reappeared. Finally, given the impossibility to obtain complete pain relief without preventing urinary retention and owing to the patient's desire, the stimulating catheter was removed.

DISCUSSION

Although it is well known that SCS could have a positive effect on diseases like multiple sclerosis, interstitial cystitis, and dysfunctional bladder disorders,[5–11] only a few reports and experimental studies exist about stimulation-related adverse effects on bladder function.[12,13]

The pelvic plexus with its sympathetic (hypogastric nerves, sacral sympathetic trunk) and parasympathetic (pelvic splanchnic nerves) sources provides and distributes the autonomic nervous supply for the pelvic organs. In the human model, stimulation of the sympathetic hypogastric plexus fibers from T10 to L2 induces urinary retention by causing the relaxation of the bladder detrusor muscle and the contraction of the intrinsic sphincter, whereas stimulation of parasympathetic fibers from S2 to S4 by modulating external sphincter relaxation causes urinary incontinence.[5,6] Moreover, somatic fibers brought by the pudendal nerve and originating in the sacral ventral horns (S2 to S4) from the Onufrowicz nucleus innervate the striated external sphincter that is contracted at rest preventing the urine leakage.[6]

Nashold et al[11] showed that spinal stimulation induces contractions of the detrusor muscle and bladder external sphincter at the same time (resulting in urinary retention), because the nervous structures are too close to each other to be selectively stimulated. Moreover, interactions between nervous structures can be much more complicated[14] at the spinal and supraspinal level, and an imbalance between inhibiting and stimulating signals could cause alterations of urinary function (urinary retention or incontinence).

In this case, physical and instrumental exams suggest damage at the vertebral level between T11 and L1 (corresponding to the lower lumbar sacral spinal segments) with an effective residual bladder function. Epidural stimulation at that level for chronic pain treatment could have highly modified the balance between sympathetic and parasympathetic efferent signals, hence the bladder function. As neurostimulation usually does not cause any adverse effects on the urinary function, when carried out at the T11-L1 level, the preexisting anatomical damage was an essential factor to the development of the complications in this clinical report.

This case report highlights the incomplete knowledge about the mechanism of action of SCS and its influence on the interactions between the autonomic nervous system and voluntary control of urinary function. Furthermore, the complete recovery of bladder function after interruption of stimulation suggests that electrical stimulation is responsible for causing the adverse effects in this clinical case.

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