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*Case Report*

# Spinal cord stimulation-induced gastroparesis: A case report

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## ABSTRACT

**Background:** Spinal cord stimulation (SCS) involves the utilization of an implantable neurostimulation device, stereotypically used in the treatment of patients with chronic neuropathic pain. While these devices have been shown to have significant clinical benefits, there have also been documented potential complications, including the risk of infection, fractured electrodes, electrode migration, and lack of symptom improvement. In addition, there has been minimal documentation on gastrointestinal (GI) side effects after SCS implantation.

**Case Description:** A 42-year-old patient with chronic axial and radicular neuropathic pain in her back and left leg status post multiple lumbar surgeries underwent implantation of an open paddle lead in the T8–T9 region. After the procedure, the patient endorsed a 50% decrease in pain at the 6-week follow-up with no further concerns. However, at the 18 months follow-up, the patient endorsed severe constipation when the SCS was turned on, leading to subsequent evaluation by gastroenterology, motility studies, and a thorough bowel regimen. Symptoms persisted, and the patient ultimately opted for the removal of the SCS implant at 21 months after the initial surgery.

**Conclusion:** While the exact mechanism behind the GI side effects endorsed in this patient is unknown, current literature postulates a variety of theories, including a SCS-induced parasympathetic blockade of the GI tract. Further, investigation is needed to determine the exact effects of SCS on the GI tract.

**Keywords:** Chronic neuropathic pain, Gastroparesis, Spinal cord stimulation

## INTRODUCTION

Spinal cord stimulation (SCS) is an implantable neurostimulation modality used for the management of chronic neuropathic pain.[16] It has been found to be effective at managing ischemic limb pain, chronic axial and radicular spinal pain, complex regional pain syndrome, and refractory anginal pain.[4,7] SCS has been documented in the literature to be particularly efficacious in patients with treatment-resistant neuropathic pain, allowing complex pain medication regimens to be modified or reduced greatly due to newfound pain control provided by SCS.[29] Some researchers have even hypothesized that SCS could potentially play an important role in the multifaceted treatment of spinal cord injuries, perhaps aiding in neuroregeneration and neural repair.[28]

SCS as a therapeutic intervention is not without complications. Current literature details complications of SCS including infection, long-term hardware failure, fractured electrodes, electrode migration, epidural hematomas at implantation sites, urinary complications, and allergic cutaneous reactions to SCS devices.[2,5,16,17,20,22-24] However, little literature exists on gastrointestinal (GI) symptoms related to SCS. In this case report, we describe a patient with an unremarkable prior GI history who underwent SCS implantation and subsequently developed significant GI symptoms.

## CASE DESCRIPTION

A 42-year-old female was seen at our hospital with chronic axial and radicular neuropathic pain in her back and left leg following multiple lumbar surgeries. Her verbal analog pain score averaged 8 on a scale of 0–10. In the past, she had used gabapentin, diazepam, long-acting morphine, hydrocodone/acetaminophen, and meloxicam without sustained relief. Lumbar epidural steroid injections, sacroiliac joint injections, and facet injections did not provide relief. At the time of presentation to our center, the patient had undergone a trial of SCS by an outside physician, which she stated reduced her pain by about 50%. Given this, it was deemed reasonable for the patient to consider a permanent SCS system implant.

The patient underwent an open paddle lead implant in the midline dorsal epidural space covering T8–T9 along with a primary cell generator. There were no complications from surgery. Six weeks out from the SCS system implantation, the patient reported that at rest, the SCS provided about 50% pain reduction. She was alternating between burst and sub-perception tonic programs. The patient did not complain of GI symptoms at the 6-week follow-up after the surgery. However, she presented 18 months after the implant with complaints of severe constipation when the SCS was turned on. She had been evaluated extensively by a gastroenterologist, undergone various motility studies, and reported having very consistent issues with poor bowel movements and abdominal pain only when the stimulator was on. When the stimulator was kept off for several days, her ability to voluntarily empty her bowels did recover. She tried a tonic stimulation program for 2 days but had a recurrence of symptoms. She then turned the stimulator off, reporting that her constipation symptoms improved markedly once the device was off again. When the stimulator was off, pharmacological treatment with laxatives, magnesium (750 mg/day), and cascara facilitated bowel movements. With the stimulator on, she had reasonable control of her baseline pain but required significant therapy to continue having bowel movements. She was told by a gastroenterologist that she would end up needing a colostomy bag if motility did not improve.

After several months of having the stimulator off and seeing continued improvement in her GI symptoms, the patient expressed her desire to have the device explanted since she was unable to use it. The procedure to remove the SCS system was performed about 21 months after the original system implantation. At subsequent visits to our hospital, the patient no longer had gastroparesis issues but recently underwent bowel surgery with diverting ileostomy for chronic rectal prolapse.

## DISCUSSION

A search of the PubMed database revealed only two case reports in the literature as of April 2021 that discuss GI effects resulting from SCS.[13,25] One of these is a series of two patients who both experienced significant GI side effects after implantation of the SCS system. One patient experienced severe nausea and diarrhea whenever the stimulator was turned on, refractory to treatment with antiemetic or antidiarrheal medications.[25] This patient's symptoms resolved within a few hours after discontinuation of the stimulator.[25] The second patient developed progressively worsening gastroesophageal reflux symptoms, flatulence, and diarrhea associated with stimulator use that also resolved after ceasing stimulation.[25] A second case report described a patient who suffered from irritable bowel syndrome. On implantation of the SCS, the patient reported a reduction in diarrheal episodes and associated abdominal pain.[13] Another recent study assessing the effect of SCS in patients with chronic nausea, vomiting, and refractory abdominal pain saw patients reporting reduced abdominal pain scores after SCS,[12] supporting the findings of the aforementioned case report.

It is known that significant GI symptoms can result from spinal cord injury.[10] These may include abdominal bloating and constipation, as well as delayed gastric emptying and reduced motility, related to specific spinal cord levels of injury.[11,19] However, the patient we report had no evidence of spinal cord injury related to the implant procedure or otherwise. The patient's symptoms began after implantation, solely occurring when the stimulator was turned on, without any other associated adverse neurologic symptoms.

A case report by La Grua describes a patient with SCS and concomitant GI symptoms.[14] This patient complained of constipation and abdominal pain during a trial with SCS. The patient's symptoms reduced after decreasing the intensity of the stimulation settings and disappeared following cessation of the stimulation. La Grua hypothesized that these symptoms may be related to a reversible and functional block of parasympathetic outflow to the GI system by the electrical stimulation, causing abdominal distension, abdominal pain, and constipation that are observable only a few hours after the onset of stimulation, and disappear after suspending stimulation. A clinical report by La Grua and Michelagnoli describes a case of micturition inhibition in a patient with

SCS that resolved with the suspension of SCS and reappeared when SCS was started again, further supporting La Grua's hypothesis that parasympathetic blockade may be responsible for the GI and urinary symptoms seen with SCS.[15]

Parasympathetic innervation to the distal colon largely originates from the sacral spinal cord, primarily from the S1 to S4 regions.[1,8] Those axons then either innervate postganglionic neurons directly within the myenteric plexus of the distal colon or within the ganglia of the pelvic plexus, which then innervate neurons within the myenteric plexus or the rectal nerves in the rectum. Stimulation of the pelvic or rectal nerves can induce an increase in motility through the activation of cholinergic pathways.[6] This parasympathetic innervation to the colon plays an important role in regulating colonic motility, particularly before defecation. The effect of SCS on GI function has been evaluated using animal models as well. Tu *et al.* showed that alteration of SCS parameters and lead location can change the rate of gastric emptying in canines.[26]

Damage to these parasympathetic neurons can result in irregular colonic motility as well as constipation.[3] However, over the course of patient care, there was no clear damage to any parasympathetic neurons. Other potential hypotheses based on other studies include overstimulation of sympathetic pathways leading to predominant inhibitory effects and slowing of gastric emptying or perhaps some innate physiological vulnerability to external stimuli of GI muscarinic receptors creating a more functional dysmotility overall.[3,27] In addition of note, there are several reports on using electrical sympathetic blockade as a mechanism to treat disorders such as complex regional pain syndrome and angina.[9,18,21] Accordingly, parasympathetic blockade is a probable explanation for our patient's GI symptoms, but one of a few existent hypotheses.

## CONCLUSION

SCS has been used for many years as a modality for pain management. In the case study presented, the patient's GI symptoms directly correlated with the amplitude of stimulation from the SCS device. Although the exact mechanisms through which the GI symptoms occurred are unknown, a potential mechanism could involve SCS-induced parasympathetic blockade of the GI tract. Future studies are necessary to specifically examine the effects of SCS on the GI system.

### Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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### Conflicts of interest

There are no conflicts of interest.

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