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Uncommon Dermatological Reaction to Spinal Cord Stimulation—Prurigo Nodularis

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Background: Spinal cord stimulation (SCS) is a widely used treatment for chronic neuropathic pain, demonstrating effectiveness in managing resistant symptoms in some cases. However, dermatological complications, though rare, can occur.

Case Report: This case report examines a patient who developed diffuse itching and skin rash with features of prurigo nodularis after undergoing surgical revisions and stimulation adjustments. The rash improved upon deactivating the pulse generator and reappeared upon reactivation, suggesting a direct connection between the neurostimulation therapy and dermatological symptoms.

This raises the possibility that SCS may trigger dermatological reactions rather than allergic ones. The material of the SCS device was not identified as the cause through allergy testing, reinforcing the neurostimulation as a potential trigger.

Conclusions: This case emphasizes the importance of evaluating neurostimulation as a possible cause of itching in patients presenting unexplained rashes after therapy before opting for explantation. Clinicians should consider changing settings or temporarily deactivating the device as part of the diagnostic process when other causes are excluded. Further research is required to understand the underlying mechanisms better.

Key words: Spinal cord stimulation, prurigo nodularis, neuropathic pruritus, chronic neuropathic pain, dermatological reactions

BACKGROUND

Spinal cord stimulation (SCS) is a well-established therapeutic approach for managing refractory chronic neuropathic pain. The therapy involves the implantation of epidural leads that deliver electrical pulses to the spinal cord, modulating pain signals before they reach the brain (1,2). This method has shown significant efficacy in reducing pain and improving the quality of life in many patients, particularly those with neuropathic pain syndromes that are resistant to conventional treatments.

While SCS therapy has become a cornerstone in managing chronic pain, it is not without complications. Dermatological reactions are rare, and when they do occur, they are typically attributed to allergic reactions to the materials used. These allergic responses are generally manageable with medication adjustments or device material explantation (3-5). In contrast to the limited literature focusing on material-induced allergic reactions, this case report presents a novel and unique occurrence of pruritus nodularis potentially linked directly to the SCS itself rather than the device's materials.

Prurigo nodularis (PN) is a chronic skin disorder characterized by intensely itchy, hyperkeratotic nodules, often resulting from repeated scratching. Potential causes include neurological dysfunction, such as abnormal nerve signaling or central sensitization, which may amplify the itch-scratch cycle (6). Additionally, immune

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dysregulation, as seen in atopic conditions or autoimmune disorders, and systemic diseases, like chronic liver or kidney disease, can contribute to its development. Lesions in the central or peripheral somatosensory pathways can lead to neuropathic pruritus, where persistent abnormal signaling results in chronic itch—a key factor in PN pathogenesis (7). Psychological factors, including chronic stress, anxiety, and psychiatric conditions, may further exacerbate the itch-scratch cycle, worsening the condition. Additionally, infections, metabolic disorders, and medication-induced reactions have been implicated in some cases (8).

Itch-specific pathways have complex interactions with pain processing in the spinal cord (7). Tonic activation of proprioceptors in the skin is required to induce scratching behavior, whereas short-lasting stimulation causes withdrawal (9). Recent studies (7,10) suggest that neuropeptides, such as substance P and calcitonin gene-related peptide, can promote inflammatory processes and intensify pruritus and brain-derived neurotrophic factor, as well as changes in sensory nerve fibers, and play significant roles in this process. Sensory nerve fibers transmit sensory information, including pain and itch, from the peripheral nervous system to the central nervous system (7). In PN, these nerve fibers can become hypersensitive, amplifying the response to ordinarily harmless stimuli and intensifying the itch sensation. These neurochemical changes can heighten the perception of itch, contributing to the persistence and exacerbation of pruritus (11).

Furthermore, central sensitization, a phenomenon observed in chronic pain conditions, has been proposed as a mechanism underlying chronic pruritus in PN, leading to amplified itch responses to stimuli within the spinal cord and brain, even without ongoing peripheral stimulation (7). In this context, PN can be understood not merely as a dermatological disorder but as a complex neurocutaneous syndrome driven by altered neural processing of itch stimuli, which, in our case report, could be triggered by SCS.

To our knowledge, no previous documentation in the literature connects skin reactions, such as neuropathic pruritus, leading to PN related to the activation of the neurostimulation mechanism, particularly in the absence of material-related allergies. This case report explores the potential link between SCS and itching, emphasizing the need for further research to clarify the mechanisms behind these responses.

CASE REPORT

A 34-year-old woman presents with an 11-year history of persistent neuropathic pain in the left hand, wrist, and forearm, accompanied by functional impairment. The pain originated from a sports injury in 2013, with an acute fracture of the left scaphoid initially missed on examination, leading to a delay in therapy. Osteosynthesis of the scaphoid was performed in 2014, but a lack of symptom relief resulted in multiple orthopedic interventions, including several revisions, radioulnar fixation, and scaphoid denervation, ultimately exacerbating symptoms.

The patient describes stabbing, pulsatile, and burning sensations, primarily on the dorsal aspect of the wrist, radiating toward the elbow. Examination elicited paresthesia of the dorsal aspect of the second to fifth fingers, along with reduced wrist strength due to protective posturing.

Despite extensive treatments—including anti-inflammatories, opioids, anticonvulsants, localized capsaicin therapy, pulsed radiofrequency therapy, and physical therapy—she experienced minimal pain relief, ongoing functional impairment, and a significant reduction in quality of life.

Neurostimulation Therapy

Given the neuropathic pain characteristics and resistance to conventional therapies, the patient underwent an SCS trial in 2016 with the implantation of a single epidural neurostimulation lead (Octrode, Abbott, Plano, TX, United States) connected to an external pulse generator (Fig. 1). Following a successful trial period, which resulted in more than 50% reduction in pain and improved functionality, a permanent pulse generator was implanted (Prodigy MRI, Abbott, Plano, TX, United States).

The patient benefited from stimulation for > 5 years. Over time, she developed a longstanding history of left-sided omalgia due to recurrent acromioclavicular dislocations, requiring multiple surgical shoulder stabilizations. Subsequently, she developed neuropathic pain in the left shoulder. As a result, in September 2021, the system was expanded with the implantation of a second SCS lead to address the neuropathic shoulder pain, which contributed to improving pain and function (Fig. 2).

Between 2022 and 2023, the patient required surgical revisions due to device-related complications, including the replacement of both leads (Vectris, Medtronic Inc.,

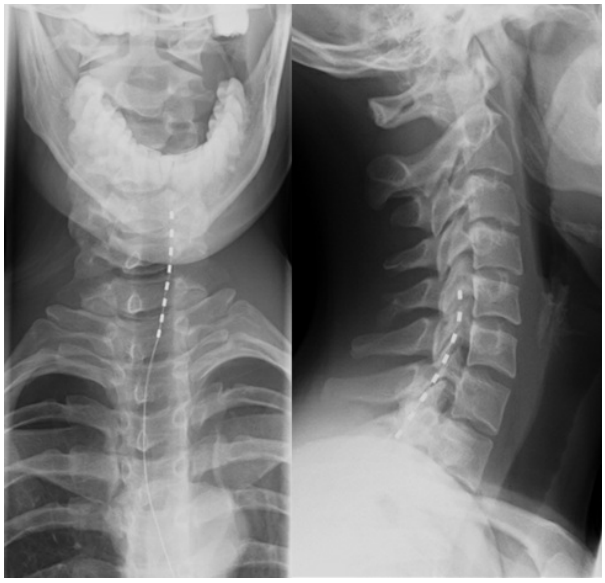

Fig. 1. AP and lateral radiographs of the initial SCS lead placement 2016.
AP, anteroposterior; SCS, spinal cord stimulation.

Minneapolis, MN, United States) and an implantable pulse generator (IPG) exchange (Intellis, Medtronic Inc., Minneapolis, MN, United States) due to battery depletion. In September 2023, the lateral epidural lead was replaced due to lead breakage (Fig. 3a and b). Figure 3c shows the updated position of the lead in the magnetic resonance imaging (MRI) scan conducted in November 2024, illustrating its relation to the spinal cord.

Dermatological Complications

Following the last surgical revision in 2023, the patient developed a diffuse papular rash accompanied by severe itching with features of PN on both legs (Fig. 4a). Initially, the rash was suspected to be a medication side effect, leading to the discontinuation of all medications, including cannabis therapy, and the initiation of antihistamines and oral corticosteroids due to concerns about an allergic reaction. However, the itching worsened, resulting in erythematous, excoriated papules that later spread to the back and torso, ultimately diagnosing PN.

To rule out a psychiatric or psychological cause for the scratching behavior, the patient underwent comprehensive psychiatric and psychological evaluations during an inpatient multimodal pain therapy program, which found no evidence of underlying psychiatric comorbidity or compulsive behavior contributing to the dermatological symptoms.

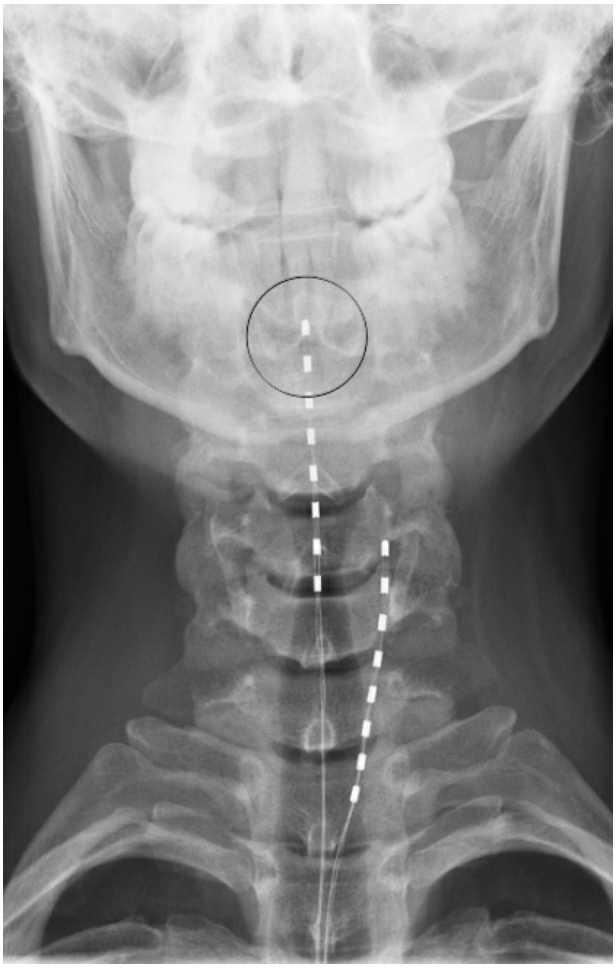

Fig. 2. AP radiograph of the lead position with implantation of the second lead medially up to C2.
AP, anteroposterior.

Despite a comprehensive dermatological evaluation—including extensive macroscopic and histopathological examinations, comprehensive laboratory diagnostics, and prolonged treatment with corticosteroids and antihistamines—the symptoms persisted without an identifiable underlying cause.

A potential link between the SCS revision and the onset of the rash was considered, initially suggesting an allergic reaction to the SCS material. However, this hypothesis was deemed unlikely, as the patient had previously tolerated leads and an IPG of the same material and design without adverse reactions. Furthermore, allergy testing for the materials in direct contact with the patient's tissue yielded negative results, ruling out a material-induced allergic response.

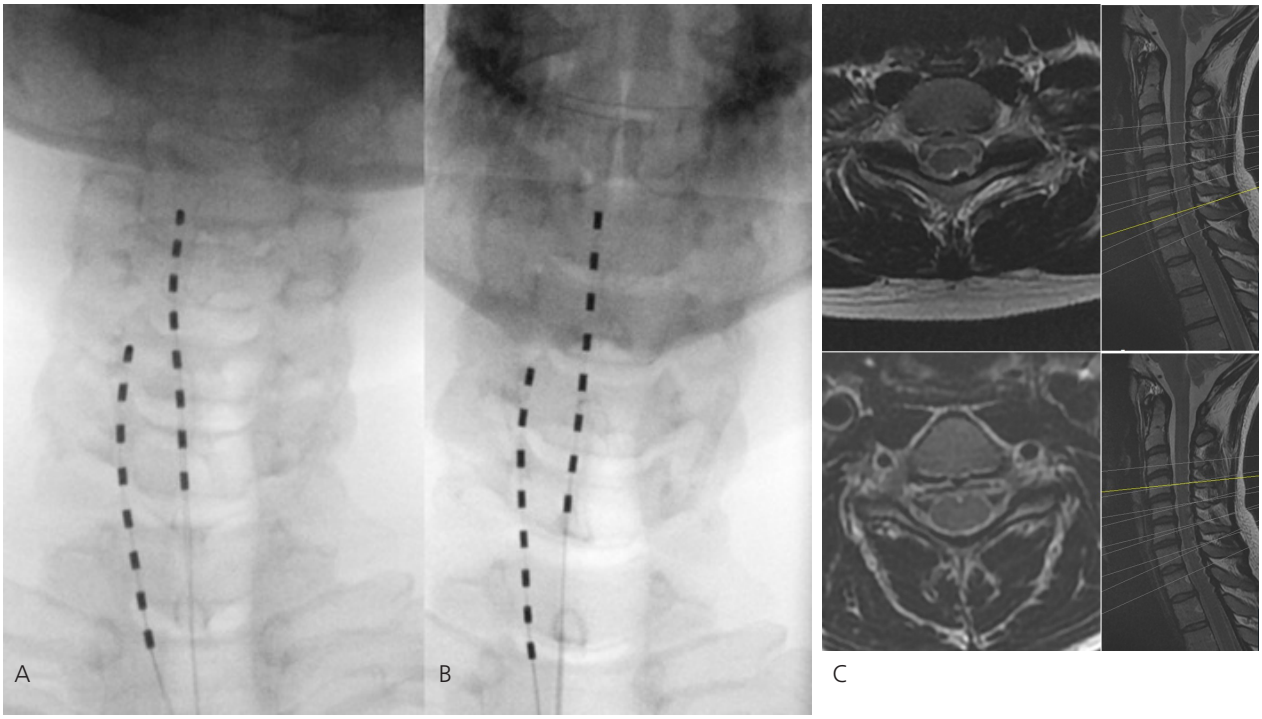

Fig. 3. Intraoperative fluoroscopy documenting the position of the leads before (a) and after (b) the SCS revision in 2023. (c) MRI scan from November 2024 demonstrating the position of the leads in relation to the cervical spinal cord.
SCS, spinal cord stimulation; MRI, magnetic resonance imaging.

Neurostimulation as a Potential Trigger

Given the unclear etiology, it was hypothesized that neurostimulation might be responsible for the dermatological symptoms. To investigate this, the IPG was initially reprogrammed (Fig. 4a-c). Since no improvement was observed with different settings, the IPG was deactivated for 2 weeks, resulting in a rapid and significant improvement in the rash (Fig. 4d). To assess causality further, neurostimulation was reactivated, and a follow-up appointment was scheduled one week later. Upon reactivation, itching and rash reappeared significantly, reinforcing the hypothesis that SCS was the underlying trigger (Fig. 4e).

Further reprogramming attempts were made with adjustments to various parameters, including different frequency ranges (20 Hz-300 Hz), pulse width, amplitudes, and polarities in both leads, even in areas not directly covering the pain region (Fig. 4). However, activation of the medial dorsal column lead consistently triggered itching, followed by skin reactions on the trunk and legs within hours. When stimulation was applied to the laterally placed lead targeting the dorsal horn on the left side, localized skin reactions were confined to the area of paresthesia coverage on the radial side of the left hand and thumb, while an improvement was observed in the legs (Fig. 5).

To rule out other potential causes of neuropathic pruritus, an MRI of the cervical spine was performed, which revealed no relevant pathological structural changes that could explain the symptoms.

Since the causal relationship between the SCS and the itching with skin reactions was established, the medial leads were deactivated, leaving only the lateral lead active to address the original neuropathic pain in the radial hand area. As the skin reaction was localized to a restricted region of the hand, it was tolerated, and the patient requested that the lateral stimulation remain active. She was able to endure the shoulder pain without stimulation and had no alternative therapy options.

Over time, however, the pain in the left shoulder increased significantly, and pain medication provided only limited relief. The shoulder pain became progressively more debilitating, eventually prompting the patient to seek alternative treatment options.

Subsequently, she underwent a 14-day suprascapular peripheral nerve stimulation (PNS) trial. The PNS trial

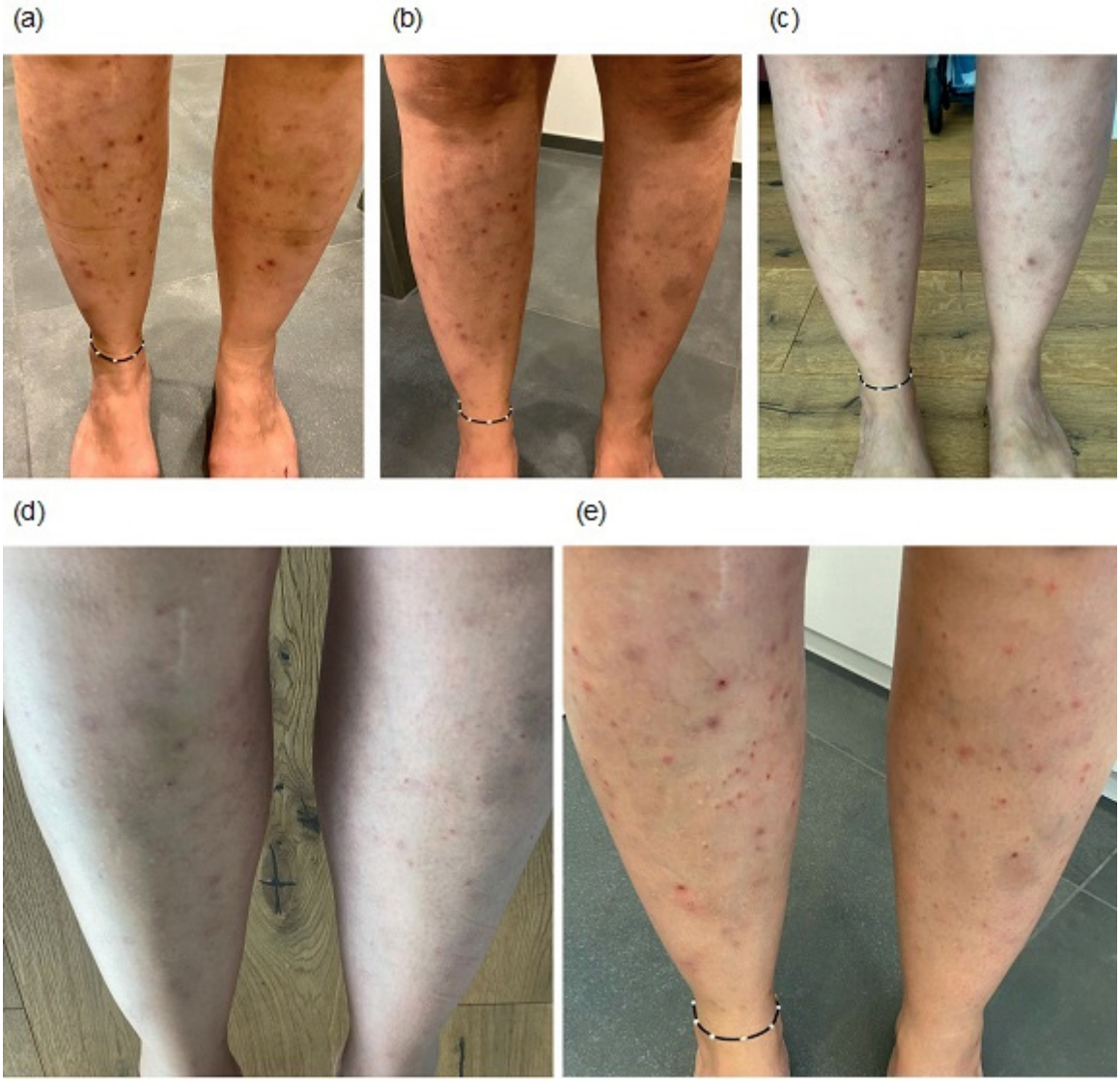

Fig. 4. (a) Onset of itching and skin rash after the SCS revision in 2023. (b) Reprogramming to a high-frequency setting and (c) different polarities used, aside from those covering the pain area. (d) Improvement of dermatological changes one week after deactivation of the IPG and (e) recurrence of itching one week after restarting the system.
SCS, spinal cord stimulation; IPG, implantable pulse generator.

provided effective pain relief for the shoulder without causing itching or rash, leading to the implantation of a permanent lead on the left suprascapular nerve in February 2025 (Vectris, Medtronic Inc., Minneapolis, MN, United States). In combination with the existing epidural lateral SCS lead, both leads were connected to the implanted SCS pulse generator to address both hand and shoulder pain. Tonic stimulation was preferred and well tolerated. The PNS has proven effective in alleviating shoulder pain, and to date, no dermatological side effects like those previously observed have occurred. However, due to residual pain in the hand, the lateral lead of the SCS lead was reactivated. Following this reactivation, a minor rash reappeared on the dorsoradial aspect of the hand, but the patient perceives it as mild and tolerable. Overall, pain relief has been successfully restored, allowing the patient to discontinue opioid medication.

DISCUSSION

SCS has been widely recognized as an effective treatment for chronic neuropathic pain, particularly in patients refractory to traditional pain management therapies. The mechanism of action involves modulating pain signals transmitted through the spinal cord, offering significant relief from pain and improving functionality (1,2). Most reported dermatological side effects of SCS involve allergic reactions to device materials, such as metals in the leads and IPG (3-5). This is the first reported case of dermatological responses directly linked to the stimulation mechanism rather than the device components.

This case also highlights one rare and potentially underreported complication of SCS therapy—the development of neuropathic pruritus leading to PN due to the SCS effect. SCS modulates pain perception by altering neural signaling within the spinal cord's dorsal columns and dorsal horn. Emerging evidence suggests that neuromodulation can influence sensory pathways beyond nociception, potentially affecting pruriceptive processing (1,2).

In dermatological reactions associated with SCS, allergies are often suspected, leading to the explantation of the system (3-5). However, in the current case, the patient benefited from neurostimulation therapy, and given her poor tolerance of pain medications, finding an alternative solution to premature explantation was crucial. Due to the significant improvement in her quality of life with SCS, it was essential to conduct a thorough diagnostic workup to rule out secondary causes and explore ways to manage the dermatological symptoms while preserving the therapeutic benefits of the system.

The hypothesis that neurostimulation could trigger neuropathic pruritus requires further investigation to understand the underlying mechanisms fully. In this case, the rapid and significant improvement of the rash upon deactivation of the IPG, followed by its reappearance upon reactivation, strongly supports the idea that SCS may have been the primary cause. Additionally, the localized skin reactions observed when stimulation was directed at localized areas in the upper limb further implicate the neurostimulation mechanism as the trigger. The development of PN, in this case, may be linked to altered nerve signaling and central mechanisms triggered or modulated by SCS.

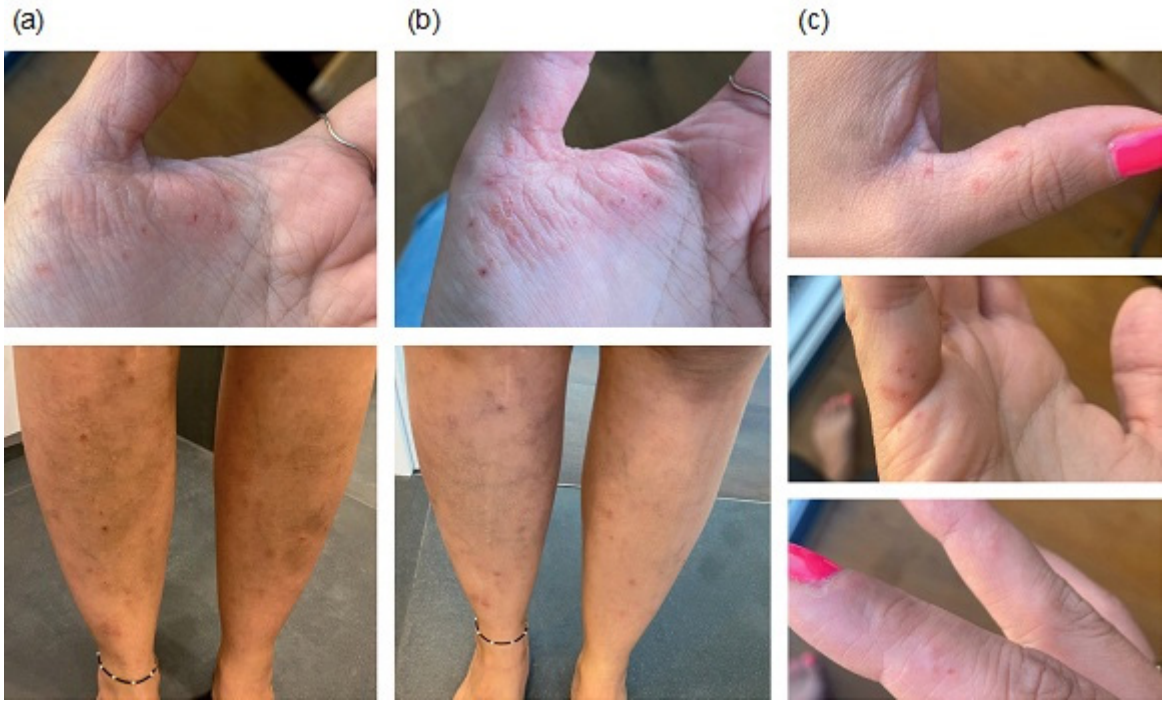

Fig. 5. (a) Skin reaction upon activation of the lateral SCS lead, showing improvement in the legs, trunk, and torso, while persisting in the left hand. (b, c) Further improvement of the skin lesions after 2 weeks, with residual lesions restricted to the radial side of the left hand.
SCS, spinal cord stimulation.

When SCS activates dorsal column axons, action potentials are transmitted orthodromically and antidromically, leading to segmental and supraspinal effects. The electrical stimulation modifies the membrane potential of neurons and other cells within the affected areas, influencing their electrochemical properties and the release of neurotransmitters in the spinal cord (1). SCS-induced alterations in neuropeptide levels could have enhanced pruriceptive signaling, possibly due to maladaptive plasticity leading to disinhibition and disrupting the balance between excitation and inhibition. Given that SCS typically activates inhibitory pathways, it would be expected to counteract neurogenic inflammation and alleviate pruritus by decreasing dorsal horn excitability. SCS also promotes the release of inhibitory neurotransmitters, such as gamma-aminobutyric acid and glycine, suppressing excitatory transmission, while its antidromic activity in sensory fibers may further dampen nociceptive and pruriceptive responses. Therefore, the paradoxical occurrence of pruritus suggests alternative mechanisms, including maladaptive neuroplasticity, aberrant antidromic signaling, or central sensitization.

Low-frequency electrical stimulation in the skin has been linked to pruritus induction, and the severity of pruritus was intensity-dependent, suggesting that specific neuromodulation settings may inadvertently trigger itch-related pathways (11). However, in this case, the high-frequency stimulation and further intensity adjustment in subthreshold settings did not resolve the issue and seemed to promote the same reaction. This indicates that both low and high frequencies, at least at the level of the spinal cord, may trigger similar pruritic responses in this case.

The temporal association between SCS activation and the onset of itching and skin reaction, and the resolution of symptoms upon deactivation, supports the hypothesis that SCS contributed to the pruritic response. Furthermore, the overlap of the skin reaction with the paresthesia coverage—depending on whether the lateral or medial (dorsal column lead) was stimulated—suggests that SCS promotes a unified mechanism of itching and skin reaction at the spinal cord level. The difference does not lie in the type of reaction but in its localization, indicating that the effect remains the same, with only the affected area changing. This supports the notion that SCS directly modulates itching pathways.

The onset of PN after revision surgery in a patient with long-term SCS use remains unexplained. One possible explanation is that the surgical procedure itself, by inducing tissue damage and inflammation, could lead to the release of additional inflammatory mediators, further amplifying both peripheral and central sensitization and/or maladaptive neuroplasticity, enhancing the likelihood of pruritus development in a patient with existing neuroplastic changes from long-term chronic pain.

This case highlights the need for further investigation into the potential unintended sensory effects of SCS, particularly in patients presenting with unexplained pruritic skin conditions. The solution was to deactivate the medial dorsal column lead and implant an additional PNS lead on the suprascapular nerve to cover the shoulder pain in combination with the lateral SCS lead. This approach allowed for the continuation of effective pain management while addressing the dermatological symptoms associated with the neurostimulation therapy. By combining SCS with PNS, the patient could relieve pain without exacerbating diffuse skin reactions; the localized pruritus in the hand was acceptable and provided a tailored solution to her complex clinical situation.

CONCLUSIONS

This case provides valuable insight into a rare and potentially overlooked SCS therapy complication. Further research into the mechanisms of neurostimulation-induced itching and skin reactions is necessary to understand this phenomenon better and to guide management strategies for patients experiencing these complications.

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