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CASE REPORT

Successful spinal cord stimulation for neuropathic below-level spinal cord injury pain following complete paraplegia: a case report

Tim A Reck and Gunther Landmann

INTRODUCTION: Neuropathic pain is common in patients with spinal cord injury (SCI) and often difficult to treat. We report a case where epidural spinal cord stimulation (SCS) below the level of injury has been successfully applied in a patient with a complete spinal cord lesion.

CASE PRESENTATION: A 53-year-old female presented with neuropathic below-level SCI pain of both lower legs and feet due to complete SCI below T5. Time and pain duration since injury was 2 years. Pain intensity was reported on numeric rating scale with an average of 7/10 (0 meaning no pain, 10 meaning the worst imaginable pain), but also with about 8–10 pain attacks during the day with an intensity of 9/10, which lasted between some minutes and half an hour. SCS was applied below the level of injury at-level T11-L1. After a successful 2 weeks testing period the pulse generator has been implanted permanently with a burst-stimulation pattern. The average pain was reduced to a bearable intensity of 4/10, in addition attacks could be reduced both in frequency and in intensity. This effects lasted for at least three months of follow-up.

DISCUSSION: Even in case of complete SCI, SCS might be effective. Mechanisms of pain relief remain unclear. A modulation of suggested residual spinothalamic tract function may play a role. Further investigation has to be carried out to support this theory.

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INTRODUCTION

Pain is a common complication in patients with spinal cord injury (SCI). The prevalence of pain in general is reported in about 74% in SCI.[1] The prevalence of neuropathic pain in a recent review is established at 53%.[2] Neuropathic pain in SCI can be classified as at-level or below-level, according to its localization referring to the level of the lesion.[3] Among individuals with SCI and pain treated in a multidisciplinary pain centre the prevalence of neuropathic at-level and below-level SCI pain was found in 53 and 42% respectively.[4] Neuropathic pain is classified as 'pain arising a as direct consequence of a lesion or disease of the somatosensory system'.[5] Criteria for SCI neuropathic pain have been proposed including history of a spinal disease or lesion confirmed by a diagnostic test, pain location at or below the neurological level of injury, presence of negative or positive sensory signs in the area of pain compatible with the spinal cord or root lesion and exclusion of other pain causes.[6]

Neuropathic pain following SCI is considered as one of the most distressing and disabling complications.[7] Neuropathic pain appears to be persistent despite various treatment and treatment remains difficult and inadequate.[6] Treatment options include pharmacological, interventional and psychological approaches, at the best embedded in an interdisciplinary pain treatment setting.[8] In this context, very little is known about the use spinal cord stimulation (SCS). Some study groups that evaluated the efficacy of SCS therapy included patients with paraplegia, unfortunately no detailed data about the paraplegia (AIS score, level of lesion) is provided.[9–11]

Since its first mention almost 50 years ago,[12] the electrical inhibition of pain using stimulation of the spinal cord has become a well-established method in the treatment of a variety of pain conditions.[13,14] SCS is a synonym for dorsal column stimulation. The main indications for the use of SCS, preferably in a setting of interdisciplinary pain treatment, are neuropathic pain conditions such as complex regional pain syndrome (CRPS) or peripheral nerve injuries as well as mixed neuropathic/nociceptive conditions such as failed back surgery syndrome (FBSS).[15–18]

CASE REPORT

A 53-year-old female presented to our pain clinic with a 2-year history of neuropathic below-level SCI pain following a complete spinal cord lesion. This lesion had set in due to a toxic reaction to local anesthetic which has been administered epidurally as anesthesia for elective knee surgery in 2014. Right after the onset of the sensory and motoric symptoms a burning and stabbing pain developed in both lower legs and feet. The patient was assessed by a neurologist to establish the neurological and pain diagnosis. A complete paraplegia below the level T5, AIS A (according to the International Standards for Neurological Classification of SCI by the American spinal cord injury society[19]) was found. Neurophysiology showed absent cortical somatosensory evoked potentials (SEP) of the tibial nerve while SEP of the median nerve showed normal cortical responses. The MRI scan revealed an extensive diffuse spinal cord lesion with signal hyperintensity along the thoracic spine level. Pain intensity on numeric rating scale was reported between 3 and 7/10 (0 meaning no pain, 10 meaning the worst imaginable pain) with an average of 7/10, but also with about 8 to 10 attacks during the day with an intensity of 9/10, which lasted between some minutes

Centre for Pain Medicine, Swiss Paraplegic Centre, Nottwil, Switzerland.
Correspondence: TA Reck (tim.reck@paraplegie.ch)
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and half an hour. The patient was already on a multimodal treatment approach prior to admission. Medical treatment with antiepileptics (gabapentin, pregabalin, oxcarbazepine), antidepressants (amitriptyline, nortriptyline, duloxetine) or opiods (oxycodone), either did not relief the pain sufficiently or had to be stopped due to side effects. Concomitant psychotherapy helped in terms of a better pain acceptance but could not modify pain intensity. After an intensive discussion within the pain team the decision was made to start trial with SCS applied below the level of spinal cord lesion. There was debate on the fact, whether epidural SCS at that level might be appropriate since a complete spinal cord lesion was established.

In a first intervention two eight-pole Octrode leads (St Jude Medical, Saint Paul, MN, USA) have been placed percutaneously bilateral between T11 and L1. Intraoperative testing did not lead to any paresthesia felt by the patient. A pattern of burst-stimulation had been installed for a two-week testing period. During this testing phase it could be clearly determined, that with a current of more than 0.5 mA the pain increased whereas with a current between 0.25 and 0.5 mA a reproducible pain reduction occurred. The average pain was reduced to a bearable intensity of 4/10, but in addition attacks could be reduced both in frequency and in intensity. The further parameters of the stimulation were a burst-frequency of 40 Hz with an intra-burst-frequency of 500 Hz and a duration of each pulse of 1000 mcs. After the testing period a permanent generator (Proclaim 7, St Jude Medical, Saint Paul, MN, USA) had been implanted. In the follow-up, 3 months later the patient described an unchanged positive effect of the stimulation.

DISCUSSION

We described a successful treatment of neuropathic below-level SCI pain by SCS in a person with a complete spinal cord lesion. From the pathophysiological point a toxic reaction to the local anesthetic has been addressed to be the cause for the paraplegia. Toxic reactions of the spinal cord to local anesthetics leading to permanent neurologic deficits are a rare but potentially severe complication.[20]

Current guidelines suggest SCS as relatively safe, minimally invasive and reversible. Randomized controlled studies support the efficacy of spinal cord stimulation in FBSS and CRPS. Similar studies of neurostimulation for peripheral neuropathic pain, postamputation pain, postherpetic neuralgia, and other causes of nerve injury are needed.[14] Medical treatment of neuropathic SCI pain is remains difficult and inadequate.[6] The evidence of efficacy of SCS treatment in neuropathic SCI pain is limited, therefore current treatment guidelines do not recommend this treatment and suggest that further research is required.[21,22] One publication with regard to SCS application in paraplegic pain found that SCS application might be helpful and found better effects in at-level SCI pain and in incomplete lesions.[9] A wide variety of success rates for SCS in patients with SCI pain was reported.[23]

SCS-electrodes are placed percutaneously or via a small laminotomy in the epidural space to the posterior columns of the spinal cord. The area of the spinal cord to be stimulated for coverage of the legs corresponds to the level T11-L1 of the bony vertebral column, which has shown in many patients with unimpaired sensory properties to be the proper site for stimulation the legs.[24,25] The complete mechanism of action of SCS therapy for pain treatment is yet not fully understood but it seems, among others, to activate the inhibitory neurons in the posterior horns of the spinal cord.[26,27] In general SCS as an minimal invasive treatment method is considered to be relatively safe compared to motor cortex stimulation, which goes along with an operation at the brain with potential risks and complications.[14]

Neuropathic below-level SCI pain is regarded as a central neuropathic pain syndrome (CNP).[3] A wide range of mechanisms has been discussed but have not been completely understood.[6,28,29] This may lead to unsatisfying success in treatment in many cases. Neuropathic pain mechanisms in below-level SCI pain involve a dysfunction of spinothalamic tract (STT) since many authors agreed on the fact that STT-damage is a necessary condition for the development of CNP.[30–33] It has been shown that damage to the STT following SCI is related to enhanced neuronal excitability and reduced descending pain inhibition leading in turn to chronic CNP.[29] The contribution of these mechanisms in complete spinal cord lesion is unclear because measurements in patients with complete lesion were performed at lesion level.[29] One study elucidated the role of residual STT-function in patients with central pain below the level of a clinically complete SCI.[34] In the present case the efficacy of the epidural SCS remains unclear since a complete spinal cord lesion is assumed by clinical, neurophysiological and imaging assessment. While in complete lesions no communication of the affected part of the spinal cord with the brain takes place, the effect of SCS below the level of injury is hard to explain. There is literature that supports SCS therapy in patients with SCI.[9–11] Unfortunately none of these publications provide details concerning the level of the lesion or the AIS score of the patients examined. In our case there is an unusual lesion presentation because of the non-traumatic nature and the finding of extend signal changes on MRI scan with preserved continuity of the spinal cord. Whether this fact may be important for the efficacy of SCS in the current case is unclear. Since a residual STT-function even in patients with complete spinal cord lesions contributes to below-level SCI neuropathic pain[34] it may be assumed that this residual STT function could be a target of SCS-efficacy in our case.

CONCLUSION

We have described a case where SCS has been applied in a patient with CNP due to SCI and could show a promising reducing effect on pain intensity. Despite the mechanism of SCS is not fully understood we postulate that in patients with neuropathic SCI pain with resistance to a multimodal pain therapy approach this method may be applied. In the current case we suppose in addition that a suggested residual STT-function might be responsible for the positive effects of SCS. Further investigation has to be carried out to support this theory.

COMPETING INTERESTS

The authors declare no conflict of interest.

PUBLISHER'S NOTE

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