Cervical cord compression due to delayed scarring around epidural electrodes used in spinal cord stimulation

Report of 2 cases

Phong Dam-Hieu, M.D., Ph.D.,1 Elsa Magro, M.D.,1 Romuald Seizeur, M.D.,1 Alexandre Simon, M.D.,1 and Bertrand Quinio, M.D.2
1Department of Neurosurgery; and 2Pain Clinic, Faculty of Medicine, University of Brest, France

The authors report on 2 cases of delayed compression of the cervical spinal cord by dense scar tissue forming around epidural electrodes implanted for spinal cord stimulation (SCS). This complication has not been previously reported.

Myelopathy developed in 2 patients 5 and 16 years after the surgical placement of a cervical epidural electrode. Prior to myelopathy, both patients experienced a tolerance phenomenon. Magnetic resonance imaging in both cases showed severe compression of the spinal cord by the electrode. At surgery, thick scar tissue surrounding the electrode and compressing the posterior aspect of the cord was discovered. Both patients experienced a full recovery following the removal of both scar tissue and the electrode.

Medical and paramedical staff dealing with SCS should be aware of this severe and delayed complication. In addition, the development of epidural fibrosis can explain the occurrence of tolerance.

(DOI: 10.3171/2009.10.SPINE09193)

KEY WORDS • spinal cord stimulation • complication • myelopathy • fibrosis • tolerance

Spinal cord stimulation is considered an efficient and safe method of treating intractable chronic pain from various origins such as failed back surgery syndrome, CRPS, angina pectoris, and peripheral vascular disease.7–9,12,13,16,17,24,26 Although not rare, complications related to SCS are in most cases benign and can be technical or biological.1,19,20,23,25,27 Technical complications include electrode migration or breakage and pulse generator failures. Biological complications include infection, CSF leakage, and pain located at the incision, electrode, or receiver site. Neurological postoperative complications are exceptional and are related to epidural hematoma or injury to the nerve roots or spinal cord.4,12 We report on 2 cases of progressive cervical cord compression occurring several years after surgical placement of a quadripolar paddle electrode in the cervical epidural space and necessitating electrode removal. At surgery, a thick band of fibrosis embedding the electrode was found in both cases. To our best knowledge, this severe and delayed complication due to the development of dense fibrosis around the implanted epidural stimulating electrode has not been reported in patients treated with SCS.

Aside from documented complications, some patients experience progressive loss of pain control despite the presence of a fully functioning stimulating system, and this phenomenon has been called “tolerance.” The origin of this phenomenon is largely unknown, although the development of fibrosis around the electrode has been advocated. However, this hypothesis has not yet been confirmed through the evaluation of autopsy or surgical exploration findings.10

Before presenting with neurological deficits, both patients experienced a period of tolerance, which could be explained by the development of surrounding fibrosis.

Case Reports

Case 1

History and Examination. This 66-year-old woman presented in 2007 with a 4-month history of progressive gait disorder and walking disability. Neurological examination revealed quadriparenesis associated with sensory impairment and a pyramidal syndrome. Twenty years earlier, she had been in a traffic accident in which she suffered partial avulsion of the right brachial plexus. Given the intractable neuropathic pain in her right super-
rior limb, SCS was initiated in 1991 through the surgical implantation of an epidural quadripolar paddle electrode (Resume, Medtronic) at the C4–5 level. This treatment allowed significant pain reduction (upwards of 80%). Although stimulation continued to produce overlapping paresthesias in the painful area, pain recurred progressively over the 2 years following stimulator implantation. Finally, the patient switched off her stimulator in 1995 and was subsequently lost to follow-up until 2007.

Magnetic resonance imaging revealed severe compression of the posterior aspect of the cervical spinal cord by the epidural electrode.

**Operation.** At surgery, the electrode was embedded in a thick and solid fibrotic mass. The mass was fairly easily detached from the posterior aspect of the dura mater and removed en bloc. Histological examination demonstrated fibrosis with chronic inflammatory reaction and without specificity.

**Postoperative Course.** Following surgery, symptoms completely resolved within 2 months.

**Case 2**

**History and Examination.** Severe Type 1 CRPS of the right forearm and hand developed in this 58-year-old man 1 year following surgical treatment of carpal tunnel syndrome. Treatment by SCS was proposed to the patient in 2001. Stimulation via a quadripolar electrode (Symix, Medtronic) surgically implanted at the C4–5 level allowed a significant reduction in both pain levels (> 75%) and trophic disorders. Because of a tolerance effect, which appeared 6 months later, the intensity of stimulation was progressively increased to provide pain relief.

At the end of 2006, the patient experienced motor deficit in the left inferior limb and dysesthesias and hypesthesia of the left hemibody (sensory level T-4). Examination of the CSF revealed an elevated protein level (1.24 g/L) and normal cytology.

Magnetic resonance imaging demonstrated compression of the cervical spinal cord by the epidural electrode. The degree of actual compression was difficult to assess given the artifact caused by metallic components within the electrode; however, the neurological disorders continued to progress. Consequently, both the cervical electrode and the pulse generator were removed.

**Operation.** During surgery, we found a 9-mm-thick band of scar tissue between the electrode surface and the posterior aspect of the dura mater. The scar tissue was easily peeled off of the dura mater.

**Postoperative Course.** Immediately following surgery, neurological disorders started to regress and completely disappeared within 3 months. Unfortunately, symptoms related to CRPS recurred.

**Discussion**

Spinal cord stimulation has been shown to be an effective treatment option for controlling chronic pain. This therapy is gaining in popularity because of its reversibility, cost-effectiveness, minimal invasiveness, and low complication rate. In a recent review focusing on the safety and efficacy of SCS, Cameron examined the literature available over a 20-year period and found that the most frequently reported complications were generally minor and correctable. Although minimally invasive, the surgical placement of a paddle electrode in the epidural space represents a spinal procedure carrying inherent and potential risks. However, since the introduction of SCS in 1967 and despite the increasing number of procedures performed each year, no case of direct compression of the cord by the electrode has been reported. Neurological complications are exceptionally observed and can result from intraoperative root or spinal cord injury or spinal cord compression by intraspinal clot or epidural hematoma. Two cases of paraplegia following SCS have been reported. In the first case, paralysis was after the occurrence of a bacterial infection located at the lead tip of the electrode. In the second case, a massive epidural hematoma developed following the surgical placement of a thoracic paddle electrode. Temporary paralysis was reported by Law in 1.8% of 625 electrode operations and...
related to myelopathy that may have resulted from cord ischemia, which was caused by vasospasm triggered by pain within or near the spinal canal.

Late spinal cord compression by extensive epidural scar tissue developing around the electrode has not been previously described. In 1983 Reynolds and Shetter reported the case of a 26-year-old man who had presented with a pyramidal syndrome only 3 months after the placement of a Resume electrode at the C3–4 level. At surgery, a dense fibrosis surrounding the wire and the electrode plate was found. Krainick et al. reported on a patient with a cervical electrode in whom spinal cord and radicular compression developed after 2 years; however, no precision as to the origin of compression was specified.

In our cases, neurological impairment was delayed and occurred several years (5 and 16 years) after the surgical placement of an electrode. Thus, spinal cord compression was not related to the surgical procedure but rather to the progressive development of spondylosis and/or scar tissue around the electrode. The latter mechanism probably played the main role because the thickness of scar tissue was measured at 9 and 12 mm, and thus reduced the spinal canal diameter by more than 50%.

Two types of epidural electrodes are currently used for SCS. The percutaneous placement of 4-contact electrodes is a less invasive procedure but is associated with higher migration rates compared with laminectomy electrodes, which can be anchored directly to the dura mater.

Compared with the thoracic spine, the cervical spine is characterized both by greater mobility and a smaller spinal canal diameter. Because a preexisting cervical spinal stenosis could worsen after the placement of an electrode in the epidural space, dimensions of the spinal diameter must be evaluated before electrode implantation. Cervical spinal stenosis can be radiologically assessed by measuring the spinal canal diameter. In addition to MR imaging and/or CT scanning, a simple method of assessment is to determine the spinal canal/vertebral body ratio on plain films (Torg ratio). Significant cervical spinal stenosis has been defined as a ratio of < 0.82 (normal values between 0.973 and 1.102). In our 2 cases, the Torg ratios calculated on plain films obtained after the lead implantation were 0.98 and 1. The mean normal sagittal diameter of the spinal canal is 18.9 mm in men and 17.2 mm in women. Therefore, a 1-mm-thick paddle electrode inserted into the epidural space alone cannot induce mechanical cord compression in patients with no cervical canal stenosis. Magnetic resonance imaging must be performed when myelopathy is suspected and clearly demonstrates spinal cord compression by the electrode. The degree of compression is exaggerated by image distortion caused by the presence of metallic components within the electrode.

The tolerance phenomenon, defined as a progressive loss of pain control despite the presence of a fully functioning stimulating system, is regularly observed in patients with SCS. For Kumar et al., the origin of this phenomenon is still under debate. It is believed that it may be due to the plasticity of pain pathways in the spinal cord, thalamus, or cortex. The development of fibrosis around the electrode was suggested at an early stage by Nashold and Friedman in 1972, followed by several other authors, but has not been proven through the evaluation of autopsy or surgical exploration findings. In the patient in Case 2, tolerance appeared 6 months follow-
ing the beginning of SCS. In Case 1, the patient switched off her generator after 4 years given the lack of adequate pain control. In both cases, the thickness of the scar tissue could easily explain the development of tolerance.

Tolerance in association with the complete absence of responsiveness despite an increase in stimulation to the maximum available intensity should prompt a search for significant epidural fibrosis.

Our cases demonstrated that symptomatic spinal cord compression due to the development of delayed epidural fibrosis can occur several years after an SCS procedure. Medical and paramedical staff dealing with patients treated with SCS using laminectomy electrodes should be aware of the possibility of such a complication. Despite the severity of spinal cord compression, functional prognosis remains good with the possibility of full recovery once the electrode and epidural fibrous tissue are removed, as observed in our cases.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

References


Manuscript submitted February 27, 2009.
Accepted October 22, 2009.

Address correspondence to: P. Dam-Hieu, M.D., Ph.D., Department of Neurosurgery, University Medical Center, Bd Tanguy-Prigent, F29609 Brest, France. email: phongdamhieu@hotmail.com.