Spinal stimulator peri-electrode masses: case report

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The authors describe a case of delayed spastic quadriparesis caused by a peri-electrode mass following the implantation of a minimally invasive percutaneous spinal cord stimulator (SCS). Prior reports with paddle-type electrodes are reviewed, and a detailed histological and pathophysiological comparison with the present case is made.

The patient developed tolerance to a cervical percutaneous SCS 4 months after implantation, followed by the onset of spastic quadriparesis 9 months after implantation. The stimulator was removed, and contrast-enhanced MRI revealed an enhancing epidural mass where the system had been placed, with severe spinal cord compression. Decompression was carried out, and the patient experienced neurological improvement. Pathological examination revealed fibrotic tissue with granulomatous and multinucleated giant cell reactions. No evidence of infection or hemorrhage was found. Professionals treating patients with SCSs or contemplating their insertion should be aware of this delayed complication and associated risk factors.

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The first use of a spinal cord stimulator (SCS) in a human occurred in 1967 to treat a patient with intractable right-sided chest and abdominal pain secondary to bronchiogenic carcinoma.34 Since that time, thousands of devices have been implanted for an expanding list of indications, including failed back surgery syndrome, complex regional pain syndrome, ischemic limb pain, neuropathic pain, chronic low-back pain, refractory angina, and many other less common indications.2,27,28,38,42

Complications associated with the procedure may include electrode migration, infection, hardware malfunction, hematoma, and, rarely, neurological deficit.2,34,35,38

Allergic reactions with cutaneous manifestations to the components of SCS systems have been reported.3,7,26,36

Six cases are reported in the literature of a mass forming around paddle electrodes, causing spinal cord compression and myelopathy between 14 and 22 months after implantation (Table 1).4,6,18,30,41 We review this literature and report a case of cervical cord compression with spastic quadripareisis secondary to a mass around percutaneous electrodes that occurred less than 1 year after implantation.

Case Report

History and Physical Examination

This 41-year-old woman suffered from chronic intratable neck pain. She was managed medically for many years, followed by anterior cervical discectomy and fusion at C5–6 and C6–7. The patient continued to have pain refractory to further medical treatment. Next, the patient had a successful SCS trial and underwent permanent placement of a dual electrode system. Prior to the procedure, MRI of the cervical and thoracic spine without contrast was performed, confirming the absence of canal stenosis or other contraindication.

There were no postoperative complications, and the patient reported full strength and adequate pain control. Slowly, the effectiveness dwindled over the course of 4 months, requiring multiple reprogramming sessions. Nine months after implantation, the patient developed weakness and clumsiness of her upper extremities; she was then referred for neurosurgical evaluation in our clinic.

During the initial consultation, the patient complained of intractable neck and bilateral shoulder pain. This was associated with complaints of coldness, numbness, and clumsiness of her hands, as well as gait instability. Visual inspection revealed marked atrophy of the hand intrinsic muscles. The patient’s gait was spastic. Cranial nerve examination was unremarkable. Motor testing revealed 4/5 strength in the upper-extremity muscles except for 3/5 weakness of hand grip. Strength was diminished (4+/5) throughout the
lower extremities. Reflex testing showed symmetric hyporeflexia in the upper and lower extremities with an absence of Hoffman’s sign or Babinski reflex. The device was evaluated using cervical spine and abdominal radiographs as well as a cervical spine CT scan. These studies did not reveal any migration or malposition of the electrodes or disconnection between electrodes, extensions, and generator. Evaluation of soft-tissue details was severely limited by artifact from the electrode; no mass could be seen within the spinal canal. The electrodes entered the spinal canal at the level of T-2 with stimulation contacts positioned in the posterior cervical canal extending from C-2 to C5–6.

Management

The percutaneous system was removed in the operating room through an incision overlying the generator and a small dorsal skin incision over the anchor point at T-3. The leads were removed with gentle traction, and no resistance was met. MRI of the cervical spine with and without contrast was obtained after removal. There were no postoperative complications, and the clinical examination findings remained stable. The MRI showed a posterior cervical epidural mass extending from the level of C-2 to the superior aspect of T-1 (Fig. 1 upper). The mass was isointense to the cord with areas of central hypointensity and heterogeneous contrast enhancement. It caused severe spinal cord compression, most profound at C3–4 and C4–5, with T2 hyperintensity within the cord at these levels.

The patient was readmitted for cervical decompression. Prior to surgery, she reported a subjective increase in her hand weakness and that she was unable to ambulate, partially relying on a wheelchair for the previous week. On examination, her hand intrinsic strength remained stable but her lower extremities had declined further with 4+/5 strength throughout except for the anterior tibialis muscles at 2/5.

After preparing and draping the patient, we performed a midline incision from C-2 to T-1 with dissection carried down to the spinous processes and laterally to the facet joints. Laminectomy was performed from the caudal portion of C-2 to C-7 and a dense, firm, tan, fibrous mass was then encountered. Sagittal and axial ultrasonography was performed and showed obliteration of the ventral and dorsal subarachnoid space. The mass was densely adherent to the dura, and no dissection plane could be exploited. Ultrasonography was repeated after subtotal resection and indicated ventral and dorsal CSF with a pulsatile spinal cord. The operation was terminated, and the wound was closed in layers in the usual fashion.

Histopathological Findings

Routine cultures were sent at the time of surgery and were negative for bacterial or fungal growth. A specimen was sent for pathological examination. Microscopic examination after H & E staining showed dense fibroconnective tissue with marked chronic inflammation, multinucleated giant cells, and several noncaseating granulomas (Fig. 2). Slides were stained and examined for fungal, yeast, and bacterial organisms. None of these organisms were identified. No malignancy or hemosiderin deposit was identified.
Postoperative Course

Following resection, the patient had improvement in the legs and hand intrinsic muscle weakness over the course of 4 days. She was ambulatory with a walker at the time of discharge to inpatient rehabilitation. At the 2-week postoperative clinic follow-up, the wound was healthy and the patient was ambulating without assistance. The strength in her hands and legs had improved to 4/5. Repeat cervical MRI was performed 6 weeks after surgery and showed adequate decompression (Fig. 1 lower). The patient was receiving no benefit from the stimulator prior to removal and was being managed medically by her pain physician with oral narcotics, benzodiazepines, tizanidine, and pregabalin. The pain continues to be controlled with this regimen, and she declines consideration of further surgical management.

Discussion

Spinal cord compression after SCS implantation is a rare occurrence and may be caused by epidural hematoma, infarction, iatrogenic injury, epidural abscess, and tumor. Common to all these entities is the need for accurate imaging of the area of concern to characterize the cause and formulate a course of treatment.

Imaging modalities usually considered in evaluating SCS include radiography, CT scanning, and postmyelography CT scanning. However, these studies are suboptimal and do not afford the resolution needed to appropriately evaluate soft tissues compared with MRI. Our patient underwent high-resolution cervical CT scanning; the artifact created by the SCS electrodes was such that no useful interpretation could be made. In our experience, the dispersion artifact created by SCS electrodes during CT scanning makes this modality of little clinical utility.

The gold standard for evaluation would be MRI; unfortunately, most SCSs are incompatible with this modality. Uncomplicated MRI has been performed in patients with an SCS in place. However, there is inherent danger that cannot be overlooked. One early report detailed a case of neurological injury to a patient who was believed to have suffered an electrical injury via an SCS after entering the magnetic field of an anti-theft device in a store. Pulsed radiofrequency diathermy was thought to cause brainstem lesions and vegetative state in a patient with bilateral subthalamic nucleus electrodes. Ruggera et al. performed an in vitro experiment using pulsed radiofrequency diathermy and found a 2.8°C temperature increase after 1.49 seconds with an SCS electrode. This heating is seen similarly with MRI where the system acts as an antenna and energy from the radiofrequency magnetic field is absorbed and concentrated. Other possible complications of MRI with incompatible stimulators include device damage, reset, and spontaneous discharge. Our patient was receiving no benefit from the stimulator, which made the choice to remove prior to undergoing MRI simple. One could have advocated for a more invasive procedure that included exploratory laminectomy during the initial surgery. In this case, our patient was not experiencing an acute decline, and a more conservative approach was taken. Had the device been MRI compatible, the mass could have been diagnosed sooner, thus possibly preventing a second operation. It is important to counsel patients on the lack of compatibility prior to SCS implantation.

Six cases of spinal cord compression from a peri-electrode mass have been reported, all believed to have involved a paddle-type electrode. Reports described scar tissue forming around electrodes placed via cervical laminectomy, presenting between 14 months and 16 years after implantation. Our patient presented with neurological complications after 9 months, which is much earlier than prior reports, and is the only case in this review associated with percutaneous electrodes. This device has a smaller surface area and is placed in a significantly less invasive manner, creating minimal disruption to the native anatomy.

Prior histological descriptions include reports of scar tissue, fibrosis, and foreign body giant cell reaction (Table 1). The histology in our case was consistent with that in...
prior reports. In all prior cases except one, a plane could be exploited to completely resect the mass. Wada and Kawai\(^\text{41}\) reported “severe adhesion between the dura and lamina was caused by scar tissue.” Complete resection of the mass was not achieved in the present case because of the dense adhesion to the dura.

Most reports described the development of tolerance preceding symptomatic compression, defined as a progressive decrease in clinical response despite increasing or maximal stimulation in the absence of malposition or malfunction. The average latency to tolerance is 12.8 months (range 4–24 months); in the present case it was 4 months. Proposed mechanisms for tolerance include neural plasticity at various sites of the pain pathway and local fibrosis around the electrodes.\(^{8,15-17,21,24,31}\)

The pathophysiological mechanism of peri-electrode masses is unknown. Dam-Hieu et al. proposed that the deficit in their patients was a result of both the fibrotic mass and spondylosis, although the former was thought to be the primary factor.\(^6\) Cervical laminectomy can be complicated by postlaminectomy kyphotic deformity.\(^{19,40}\) Risk factors include preexisting loss of lordosis, kyphotic deformity, and violation of the facet joints at the time of surgery. Pre-existing instability with the addition of a stimulator could lead to the development of repetitive local trauma and progressive scarring. Spondylosis and loss of cervical lordosis is seen in other reports of cervical peri-electrode masses.\(^{18,41}\) The mass in our patient extended the entire length of the electrodes within the spinal canal from C-2 to T-2 and included the fused segments at C5–6 and C6–7. The involvement of fused segments suggests that spondylosis is not the only factor in the development of peri-electrode masses, although a contributory role cannot be excluded.

Allergic cutaneous reactions to stimulator components have been described, resulting in local dermatitis and, rarely, systemic dermatitis.\(^{7,26,36}\) Manufacturers may provide samples for patch testing to aid in identifying the offending agent. A search of the literature relevant to spinal stimulators, deep brain stimulators, and pacemakers did not reveal any reports of allergic reactions manifesting as a peri-electrode mass. Our patient had no cutaneous manifestations suggesting allergic response. The platinum and iridium alloy composition of the contacts is common for stimulation electrodes because it has a high Warburg capacitance (thus low electrode-tissue capacitance), low allergenicity, and reportedly minor tissue capsule formation compared with many other materials such as copper and steel.\(^{9,13}\) Reactions to platinum and iridium have been reported, largely limited to industrial workers with chronic exposure in recycling centers processing catalytic converters.\(^{1,5,12}\) Testing should include evaluation for immediate and delayed hypersensitivity through skin prick and patch testing, respectively.\(^{39}\) In our patient the reaction involved the entire length of the electrode within the spinal canal and was not isolated to the area around the contacts. A reaction to the insulation is possible, however, one would expect it to extend beyond the spinal canal.

Epidural hematoma can be considered in the differential diagnosis, although it is usually an early complication and is unlikely to develop later. A small subclinical hemorrhage could have occurred during implantation, and the mass formed as a result of continued organization. Histological sections in our patient did not reveal any hematoidin or hemosiderin deposition, arguing against hemorrhage in our patient, although sampling error cannot be excluded.

Contamination during implantation of an SCS system may produce an inflammatory reaction. Some centers implant SCSs in interventional suites outside the rigorous sterile techniques and other procedures mandated in an operating room.\(^{29}\) Additionally, a common technique when accessing the epidural space is to use approximately 5 ml of saline to dissect the plane. This is believed to reduce epidural blood vessel damage or cannulation, but it can also introduce contamination.\(^{31}\) Our patient had no subjective or objective evidence compatible with infection. In our experience, when an SCS system component becomes infected, the infection tracks along the device as is seen with intrathecal drug infusion systems. No reaction or mass was found outside the spinal canal, and cultures of the mass along with specialized staining of the histological sections revealed no evidence of infection. Again, sampling error cannot be excluded.

The development of a peri-electrode mass is a rare complication of SCS therapy. In this review the average latency to tolerance was 12.8 months (range 4–24 months); the average latency to clinical presentation for neurological deficit was 54.4 months (range 9–192 months). The small number of cases precludes a complete understanding of the pathophysiology but appears to be an exaggerated inflammatory reaction. Inciting events of inflammatory reactions could include foreign body reaction, subclinical allergic response, infection, hemorrhage, and dynamic instability with local repetitive trauma. We recommend assessing patients preoperatively for spinal deformity, canal diameter, cord compression, and dynamic instability. It is our belief that the paucity of cases does not support a mandate for long-term follow-up or routine surveillance imaging in all patients. Surgeons and pain physicians should be vigilant for the development of tolerance, especially in the first 2 years after implantation. Neurological deterioration should be thoroughly investigated. These cases further highlight the need for MRI-compatible devices.

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