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Paraplegia in a Patient with an Intrathecal Catheter and a Spinal Cord Stimulator

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This article is accompanied by a Highlight. Please see this issue of ANESTHESIOLOGY, page 27A.

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NEW technical devices are adopted frequently and enthusiastically, while possible serious risks are overlooked. This has happened with intrathecal catheters connected to infusion pumps[1] and spinal cord stimulators.[2]

Herein, we describe a sudden spinal cord derangement that occurred in a patient in whom both an intrathecal catheter infusing morphine by an implanted computerized pump and a spinal cord stimulator had been inserted.

Case Report

A 73-yr-old man described an acute onset of motor and sensory loss in January 1991 but still suffered from thoracic back pain,

depression, and severe headaches. The patient had a 15-yr history of thoracic radicular pain with failed therapeutic response to different treatments including transcutaneous electrical nerve stimulation, oral and intramuscular opioids, and physical therapy. In 1987, he had multiple-level thoracic posterior rhizotomies (D8–D12 on the right and D10–D11 on the left). In July 1990, he underwent implantation of a spinal cord stimulator with the electrode inserted at D7 in the epidural space (RESUME/ITREL Spinal Cord Stimulator, Minneapolis, MN). Neither the radicular distribution nor the spine pain were relieved.

Subsequently, on October 17, 1990, a SynchroMed Infusion System (Medtronic, Minneapolis, MN) was implanted into the subarachnoid space through the L2–L3 interspace, following the manufacturer's protocol, under general anesthesia. This procedure apparently was uneventful. The specific location of the catheter was determined by fluoroscopy during the procedure. The tip of the catheter was localized approximately at D9. The patient was able to obtain partial relief of his pain from the infusion of 11 mg/day of preservative-free morphine, supplemented with oral analgesics.

On January 11, 1991, he felt sudden severe pain on the thoracic spine. He returned to the neurosurgery clinic for an increase in the flow rate of the pump (no documentation is available as to the increased dosage) and a refill with 18 ml of preservative-free morphine (10 mg/ml). Later that night, he obtained partial relief. Three days later, the plantar aspect of his right foot became numb. The next morning, he awakened with paraplegia below D7 and complete sensory loss below D6. He was hospitalized at his local hospital. A lumbar puncture did not reveal abnormal findings, but a myelogram showed "some feature at D9–D10 to the cord lying in the canal creating a void in contrast filling."

Three days after the onset of paraplegia, he was transferred to the center where the stimulator and the pump had been implanted. Physical findings were consistent with paraplegia and a D6 sensory loss level. No other signs or symptoms were observed. Somatosensory evoked potentials showed normal median nerve potential. No cortical response could be obtained from either posterior tibial nerve. No increased uptake was seen on a galliun computed tomography scan. Spinal fluid showed a small increase in the proteins with an increase in α_2 globulins and a sedimentation rate of 37. No bacteriologic or fungal growth was observed from blood or other body fluid samples, including the indwelling morphine solution.

With the operative diagnosis of "transverse myelopathy with acute paraplegia," a bilateral laminectomy from D7 through D11 was performed on on January 18, 1991. At approximately D7, the electrode of the RESUME/ITREL spinal cord stimulator was found and removed. There was no evidence of abscess or granuloma in the area. When the dura was opened, there was a segment of adhesive arachnoiditis. The arachnoid, under magnification, was dissected from the spinal cord, correlating with the findings of the myelogram of a filling defect at this level. One segment of the spinal cord measuring approximately 1–1.5 cm was soft. After dissection of the almost gel-like arachnoid from the dorsum of the spinal cord, there was a small disruption of the pia that permitted what appeared to be necrotic spinal cord to exude. The catheter of the Medtronic morphine pump also was removed. Specimens of the arachnoid and spinal cord were submitted for culture and histopathologic study. The reports showed arachnoidal fibrosis and variable meningothelial hyperplasia. The few vessels observed showed no vasculitis. After surgery, he was transferred to a rehabilitation center for 7 weeks without change of the level of paraplegia. Morphine was administered through an intravenous patient-controlled pump for analgesia and to avoid a withdrawal syndrome.

Magnetic Resonance Imaging, which was obtained in December 1993, showed marked atrophy of the thoracic spinal cord at D6 extending caudally to the D10 level (fig. 1) with evidence of syrinx formation (figs. 1 and 2). The thoracic cord from D10 through the conus was noted to be slightly smaller than normal. Severe degenerative disc changes were seen at L2–L3, L3–L4, and L4–L5. No significant areas of spinal canal stenosis were identified. There was a cyst in the filum terminale.

Discussion

In 1976, Yaksh and Rudy reported on the safety of long-term catheterization of the intrathecal space in animals.[3] Bromage[4] and DuPen[5] have warned of the risks of neurotoxicity associated with intraspinal opioid administration but focused their concern on the neural injury that resulted from inclusion of preservatives in spinal drug preparations.

In humans, administration of spinal opioids using an implanted pump was introduced first for the management of intractable cancer pain.[6] Epidural and intrathecal routes have been used, each with advantages and disadvantages.[7] An important number of "predictable complications" were reported from both procedures.[8,9] Nitescu *et al.* described some complications following the placement of 157 catheters into the intrathecal space of 142 patients: In most instances,

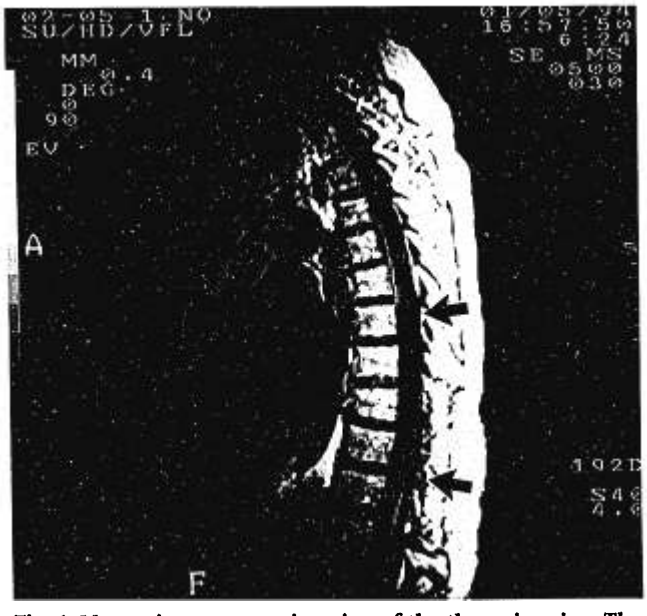

Fig. 1. Magnetic resonance imaging of the thoracic spine. The spinal cord ends proximally at D6 (upper arrow). The distal end is shown at D11 (lower arrow). From D7 to D10, a distorted, undefined structure links both points.

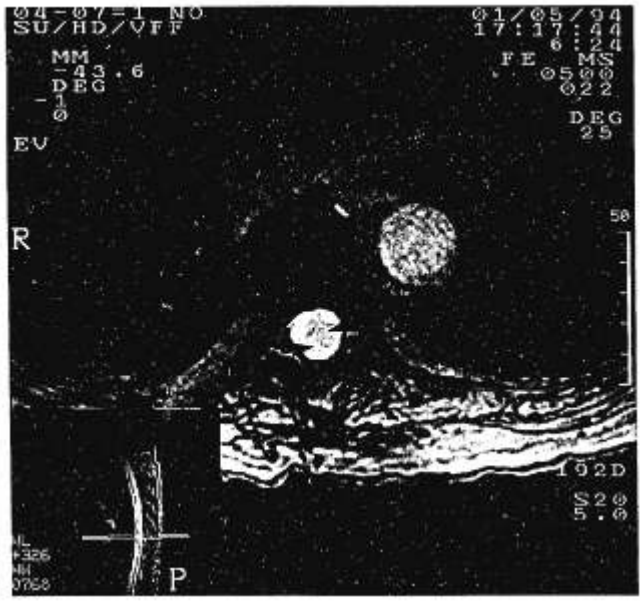

Fig. 2. Axial view of the D8 level showing a syrinxlike cavity formation (arrows). The typically well delineated and centrally located image of the spinal cord is absent.

problems were related to the placement procedure, but no neurologic sequelae could be ascribed to these intraoperative complications.[10] Clinically, unsuspected degeneration of the posterior column, perhaps related to intraspinal infusion of morphine or, more probably, a paraneoplastic phenomenon has been observed *postmortem* in two patients with implanted pumps.[11,12] Coombs *et al.*[11] found dural thickening with focal necrosis and a few chronic inflammatory cells at *postmortem* examinations of cancer patients who had long-term epidural catheters. However, there was no reported spinal cord compression in any of these patients.

In a laboratory model using ewes, a focal chronic pericatheter granulomatous reaction was demonstrated, and in several of these cases it progressed to spinal cord compression.‖ Similar findings were reported in a canine model of the subarachnoidal infusion of diazocine, with spinal cord compression even in control dogs receiving saline, and more severe abnormalities (fibrotic syrinx formation and spinal cord infarcts) limited to the animals receiving an active drug solution.[13] The chronic intrathecal infusion of a morphine solution or saline in monkeys has been noted to produce mononuclear cell infiltration in the leptomeninges and cauda equina within 6 weeks. The effects of the infusion and the trauma of the catheter insertion have been difficult to separate.[14]

North *et al.*[15] reported a case of a 42-yr-old woman with a history of previous lumbosacral spine operations. Spinal cord stimulation had been attempted twice, at midthoracic and upper thoracic levels without a lasting benefit. A programmable infusion pump (SynchroMed, Medtronic) and an intrathecal catheter were implanted at D11 to infuse morphine because of persistent low back and lower limb pain. Two months after pump implantation, the patient developed progressive paraparesis culminating in paraplegia and incontinence 1 month later. There was no sensation to pinprick below the D11 level on the left and the D10 level on the right, the lower limbs were flaccid, areflexic with bilateral Babinsky. A myelogram showed nearly a complete block to the flow of contrast at D10–D11. With computed tomography, extensive arachnoid fibrosis without evidence of extradural compression was noted. A laminectomy was performed identifying a darkly pigmented and partially liquefied mass measuring 6 × 8 × 40 mm communicating with an intramedullary cavity containing creamy liquid material. These findings were similar to the case described here, in which a sequence of surgical spine procedures, the placement of a neurostimulator, and finally, the insertion of a permanent catheter in the subarachnoid space were attempted to treat severe thoracic radicular pain.

Also, in the current case, the appearance of the clinical symptoms was sudden, and the first symptom was severe pain that could be partially relieved through an increase in the flow rate of the pump. Later, the patient had numbness in the plantar aspect of his right foot, and the next day he awakened with motor and sensory loss below T6. In both cases, the findings at surgery and the histologic reports represented arachnoidal fibrosis near the area of the tip of the catheter and below the electrode of the neurostimulator. A remarkable coincidence is that, a syrinx-like formation in the magnetic resonance imaging could be correlated with the pathology described in a canine model receiving morphine infusions that developed spinal cord infarct and syrinx-like formation.[13]

To perhaps recognize the development of this complication in its early stages, we recommend that a brief neurologic examination be performed and recorded, to be compared with previous findings, every time the

‖ Coombs DW, Colburn RW, DeLeo JA, Hoopes PJ: Observations on spinal morphine infusion in the ewe. Presented at the Eighth Annual Scientific Meeting of the American Pain Society, Phoenix, Arizona, October 26–29, 1989.

pump is refilled. If an alteration in the examination is revealed, imaging studies could be performed to establish a proper diagnosis.

The clinical and pathologic findings in this patient are similar to a case reported by North *et al.*[15] Both of these patients had neurostimulators and a computerized pump functioning simultaneously. The manufacturer recommends in the equipment brochure that no other programmable medical device be used if a pump is to be inserted.#

As far as we know, in both cases, the spinal cord stimulator was present, attached to the dural sac, but was not being used. A case similar to the current one occurred in another institution 3 yr before but was reported in the medical literature.

The paucity of reported complications, however, does not necessarily imply their absence. It may be necessary to establish a National Registry of Spinal Cord Stimulators and of Implantable Spinal Catheter-Pump units to accurately determine their effectiveness as well as their complications.

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