Anesthesiology
1998; 88:1672-5

Paraspinal Fluid Extravasation from Long-term Epidural Catheter Delivery System

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LONG-TERM use of epidural and intrathecal catheters in cancer and nonmalignant pain has increased substantially over the past 20 yr. Although they provide effective analgesia for many patients, these catheter delivery systems also have produced many complications. Infections and fibrosis have been reported most commonly and with great variation, dependent to a great degree on the catheter system chosen.[1,2]

We present a case involving the long-term use of an epidural catheter for a patient with non-Hodgkin's lymphoma. Infection, fibrosis, and an unusual paraspinal fluid extravasation from the epidural space are described and discussed.

Case Report

A 68-yr-old woman was admitted to the hospital in September 1994 for increasing abdominal distension, increasing diffuse abdominal pain, nausea, and decreased oral intake during the previous weeks.

Her primary diagnosis, established in 1991, was stage 4 non- Hodgkin's lymphoma. Subsequently she received chemotherapy, consisting of six cycles of cyclophosphamide, doxorubicin, vincristine, and prednisone and intermittent oral cyclophosphamide therapy with prednisone. Control of pain consisted of 4 mg oral hydromorphone every 3 h and fentanyl patches given at a dosage of 150 μg/h.

In April 1994 the Pain Control Center was consulted for management of increased low back pain. Computed tomographic (CT) scan results were normal. On discharge the fentanyl patches had been changed to slow-release morphine sulfate, 30 mg by mouth every morning, 45 mg by mouth in the evening, and 60 mg by mouth at bedtime in combination with hydromorphone, 2 mg by mouth every 4 h for breakthrough pain.

Three months later she was admitted with uncontrolled abdominal, pelvic, low back, and leg pain. Systemic opioids did not control her pain adequately, and side effects were noted. A permanent epidural catheter and port (polyurethane; Deltec®, Minneapolis, MN) implanted by a lumbar approach (L3-L4) after a 2-day trial significantly decreased her pain. The tip of the epidural catheter was noted at T9. The patient was discharged and given epidural morphine sulfate at 10 mg/h.

It is noteworthy that CT scan of the spine at this time revealed a compression of the L1 vertebral body >50% of the original height and collapse of L3.

In October 1995 the patient fell and required hospitalization. A local anesthetic agent was added to the epidural infusion, and she was discharged receiving 0.1% morphine sulfate and 0.075% bupivacaine at 10 ml/h with 5-ml bolus doses every 15 min if needed.

She was admitted in June 1996 for abdominal distention and back pain. A CT scan of the chest and abdomen revealed a fluid density mass within the right paraspinous location posterior to the azygoesophageal recess (fig. 1). This measured 2 × 4 cm and ≈6 cm in superior/inferior extent and communicated with the epidural space. There were associated destructive changes involving T8, T9, and T10 vertebral bodies (fig. 2). An abnormal fluid density that compressed the thecal sac posteriorly and laterally was noted (fig. 3), and areas of sclerosis were seen within the vertebral body, suggesting a degree of chronicity to the epidural process. On subsequent CT, contrast was injected *via* the epidural catheter, and a tract was visualized leading to the paravertebral fluid collection. The contrast remained localized to the paraspinal space. During this hospitalization gram-positive cocci 2+ and leukocytes 2+ were obtained from the epidural catheter. Antibiotic treatment was provided, and the catheter and infusion maintained. Pain control was adequate in the abdomen, and abdominal distention resolved. She was discharged to home.

Twelve days later she was admitted with purulent discharge from the epidural port-a-cath site. A staphylococcal species, coagulase negative and β-lactamase negative, was cultured. The patient was started on vancomycin and rifampin. The epidural infusion was stopped, and pain control was accomplished *via* an intravenous patient-controlled

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Received from the Department of Anesthesiology, Pain Control Center, and the Department of Internal Medicine, Section of Hematology/Oncology, Wake Forest University School of Medicine, Winston-Salem, North Carolina. Submitted for publication November 10, 1997. Accepted for publication January 30, 1998.

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Key words: Cancer pain; complications; epidural; fluid extravasation; infection.

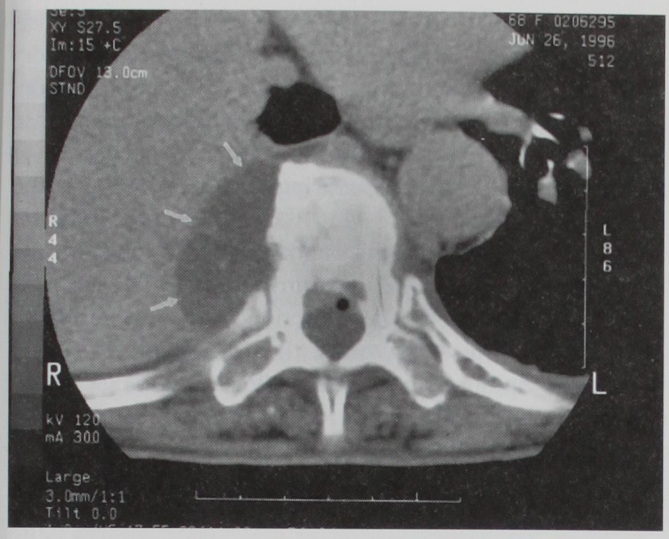

Fig. 1. Paraspinal fluid collection is noted at T7 (*arrows*). This represents fluid from the epidural infusion. The dark circle within the spinal canal represents the magnetic resonance imaging shadow adjacent to the tip of the epidural catheter.

analgesia of morphine sulfate at 1.5 ml/h with 1-ml bolus doses every 15 min.

She remained stable and was discharged in 7 days. She completed 14 days of intravenous antibiotic agents at home. Pain control continues until now (21 months after discharge) *via* intravenous patient-controlled analgesia of morphine sulfate, basal rate 90 mg/h and bolus doses of 150 mg, with satisfactory results. It is noteworthy that the epidural catheter has not been extracted. The patient is predominantly bedridden but has intact motor, sensory, bladder, and bowel function.

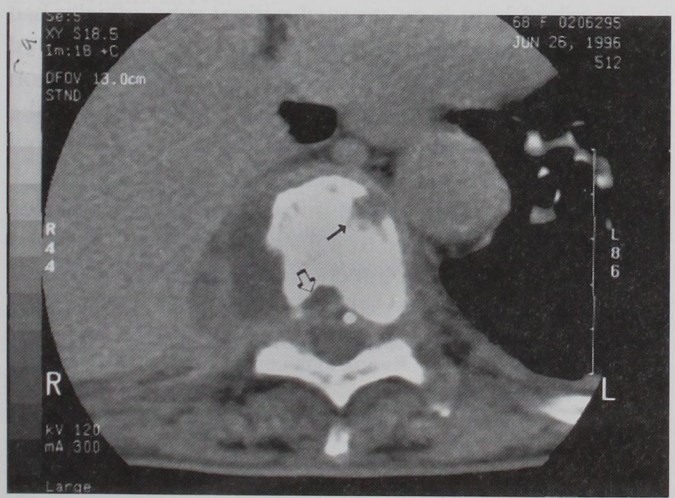

Fig. 2. Destructive changes in the vertebral body of T8 are noted. The paraspinous fluid mass is also present. The catheter is noted in the epidural space.

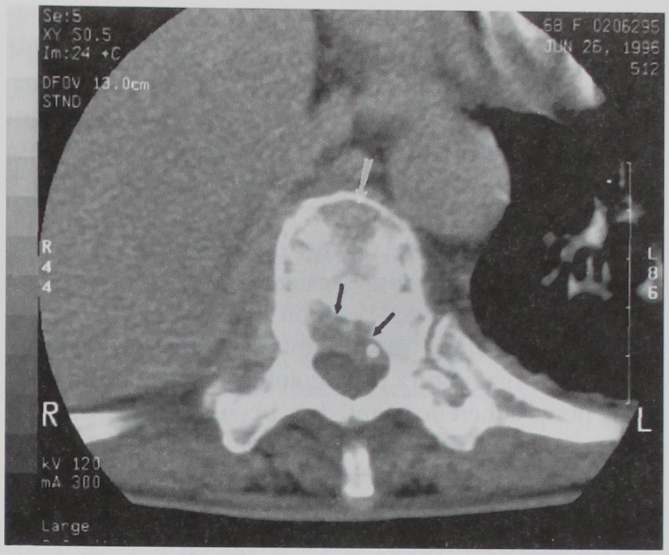

Fig. 3. Abnormal fluid density and fibrosis are noted within the epidural space, compressing the thecal sac posteriorly and laterally. Sclerosis within the vertebral body is also apparent.

Discussion

This case illustrates several management issues that commonly are present during long-term use of spinal, epidural, or intrathecal catheters, including fibrosis, infection, and extravasation of an epidural infusion into paraspinal tissue. This patient's catheter has been in place much longer than the median duration of catheters reported in other series (47–96 days) and in our experience (93 days).[3–5] Many of the complications with spinal catheter delivery systems do not manifest during the initial 3-month use; however, with longer use, many problems and management issues can develop.

Signs of epidural or intrathecal infection can be subtle and nonspecific.[1–2,6] This patient never demonstrated an elevation of her leukocyte count nor did she develop temperature elevations. Nonetheless her clinical condition continued to deteriorate despite increasing analgesic medications. Detection of an occult infection can be difficult in these patients and often requires multiple attempts at discovery.

The presence of gram-positive cocci from an aspirate of the epidural catheter was suspicious but not confirmatory of a clinical infection. Seeding of catheters without clinical correlation can occur; however, our practice treats these situations to avoid or delay future possible sequelae. The presence of a staphylococcal species from the portal site further confirmed an infectious process.

Standard management for infection of any implanted

hardware requires removal of the equipment.[1,7] Regarding intraspinal catheter delivery systems, this decision must be weighed against the analgesic advantages that the patient may be experiencing. Some patients with life-shortening disease processes do not want their pain-relieving systems removed, if possible, despite an increased risk of infectious complications.

This patient requested that her catheter not be removed. Our policy demands close consultation with the infectious disease department, and decisions depend on the individual clinical situation. This patient was treated with vancomycin and rifampin (a drug combination with good foreign body adherence) and has been able to keep her catheter free of any further infection for 16 months.

Other case reports have stated similar success with antibiotic treatment of spinal catheter systems.[2,8–10] If a potentially infected catheter system is not going to be removed, close monitoring of the patient's clinical situation and close consultation with infectious disease specialists is required. In this particular case we were able to comply with the patient's request that the catheter not be removed because she did not at any time appear clinically ill or septic from the infectious process.

The unique characteristic of this case demonstrates the distribution of epidural, infused solutions into the paraspinous space in the presence of vertebral metastatic disease. It is important to note that this patient had no evidence of any spinal disease in the thoracic spine at the time of epidural implantation. The subsequent development of vertebral metastatic disease with ultimate destruction of the canal occurs in a significant minority of cancer patients. The actual likelihood of developing this complication varies greatly with the type of tumor, and ultimate destruction depends on the aggressiveness of the tumor. When spinal catheters are in place, the clinician must remain vigilant and realize the potential for vertebral metastasis.

Unexplained back pain, an unusual increase in pain, or new onset of radiculopathy are all common symptoms of vertebral metastasis and often require specific treatment *versus* an increase in analgesic medications. This patient complained of new, severe, unremitting back pain that did not resolve with increasing doses of her epidural infusion. The lack of a response to a change in the infusion demanded the subsequent work up, including the CT scan.

This patient was found on CT scan to have significant epidural fibrosis and destruction of bony vertebral elements resulting in loss of normal spinal anatomy. Although epidural catheters can certainly produce significant epidural scarring,[11,12] it is more probable that the fibrosis seen in this case resulted from the tumor's ongoing destruction of the vertebrae. Vertebral metastasis and destruction can produce a significant inflammatory response with extensive fibrosis within the spinal canal.

The fluid collection, measuring $2 \times 4 \times 6$ cm in the paraspinous space of T8–T10, communicated with the epidural space. The leaking of analgesic solution from the epidural space undoubtedly contributed to the poor pain relief that the patient was experiencing at the time she sought care. It is noteworthy that a collection of fluid within the epidural space was believed radiologically to compress the intrathecal space. This was unlikely to be of clinical significance because the fluid space communicated with the paraspinous fluid collection. In other cases, however, in which no extravasation occurs, intraspinal pressure can increase greatly during continuous infusion, particularly in the presence of extensive fibrosis. This increased intraspinal pressure can produce a significant hyperpathia.[13,14]

The care of patients with cancer similar to this one continues to evolve at our medical center. We currently prefer the use of intrathecal catheters to preclude the necessity of large epidural volumes during continuous infusion. Intrathecal location maintains the dura exterior to the catheter and provides an added membrane to prevent extravasation of fluid. The intrathecal space also provides a significant dose-sparing effect compared with the epidural space for hydrophilic opiates and local anesthetic agents. Finally, the practitioner using intrathecal catheters does not have to worry about migration of the catheter from the epidural space.

Described here is an unusual patient who initially had no tumor involvement of the spinal column and achieved excellent long-term analgesia with an epidural infusion of opiates and local anesthetic agents. Subsequently she developed vertebral body metastases, bony destruction of the vertebral column, and infection at the site of her epidural catheter. The epidural infusion clearly extravasated from the epidural space to paraspinal tissue and resulted in poor analgesia. The infection was successfully treated without removal of the catheter, and the patient has done well for >16 months after this occurrence.

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Anesthesiology
1998; 88:1675–7

Intraoperative Bronchospasm Induced by Stimulation of the Vagus Nerve

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INCREASED peak inspiratory pressure and bronchospasm during general anesthesia can have many etiologies, including the patient's intrinsic disease and mechanical, chemical, or neurogenic causes. We present a case of bronchospasm induced by direct stimulation of the vagus nerve.

Case Report

A 56-yr-old man underwent a left glomus vagal tumor resection during general anesthesia. Preoperative medical history was significant for restrictive lung disease resulting from asbestosis. In addition there was a long smoking history, obesity (5′6″; 260 lbs), and significant cervical stenosis at C5–C6. Previous pulmonary evaluation showed mild asbestosis, requiring no treatment. No pulmonary function tests were obtained preoperatively. The patient denied any history of wheezing or use of any medications.

Because of the severe cervical stenosis, the patient was orally intubated awake with a fiberoptic scope. Placement of a 7.0-mm ID endotracheal tube (ETT) was attempted initially, although the ETT could not be passed through the cords. A 6.0-mm ID ETT was subsequently placed without difficulty. The patient was induced using thiopental and fentanyl, and the lungs were mechanically ventilated. Wheezing was noted immediately after intubation and induction and was treated by deepening the anesthetic depth. Anesthesia was maintained with 70% N_2O/30% O_2 and isoflurane (end tidal concentration stable at 0.9%). After intubation, hookwire electrodes were placed in the vocal cords during direct laryngoscopy. Despite pretreatment with 0.2 mg of glycopyrrolate, the patient continued to produce copious amounts of secretions during and after intubation. No muscle relaxant was used so that a nerve stimulation monitor could be used to identify nerves during the dissection and tumor removal. The patient was positioned with the head 180° away from the anesthesia machine with the head turned to the right. The patient was ventilated with 600 cc tidal volume with peak inspiratory pressures (PIPs) ranging from 55 to 60 mmHg.

The case progressed uneventfully with periodic stimulation of the hypoglossal, vagus, and spinal accessory nerves during dissection until approximately 10 h after incision when the patient developed

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Received from the Department of Anesthesia, University of Pennsylvania, Philadelphia, Pennsylvania. Submitted for publication November 11, 1997. Accepted for publication February 9, 1998.

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Key words: Complications; evoked potentials; neck dissection.