EPIDURAL abscess is a rare occurrence; the diagnosis is made in fewer than two cases per 10,000 admissions at major hospitals. Even more rare is the occurrence of epidural abscess after obstetric epidural anesthesia. We present what we believe to be the fourth reported case of epidural abscess after obstetric epidural anesthesia.

Case Report

A 34-yr-old, 109-kg, gravida 2 para 1 patient presented at term with spontaneous onset of labor. She received epidural anesthesia for labor and delivery. She was placed in the lateral position, the skin was prepared with betadine solution three times using sponge pads for application, and a sterile fenestrated drape was placed. The betadine and sponge pads were contained in the disposable epidural tray. Lidocaine, 1%, was used for local anesthesia, and the tip of an 18-G thin-wall Tuohy needle was placed on the first attempt into the epidural space via the L3-L4 interspace using loss of resistance to air. The epidural catheter was advanced 4 cm cephalad without difficulty, and the Tuohy needle was removed. A sterile foam pad was placed over the insertion site, and plastic tape was used to cover the area. After a negative test dose of lidocaine with epinephrine, an infusion of 0.0625% bupivacaine and 2.1 mcg/ml fentanyl was begun at 12 ml/h. A bacterial filter was used for all infusions and injections through the epidural catheter. One hour, 25 min later an infant with an Apgar score of 9/10 was delivered spontaneously. The epidural catheter was left in place to be used the next day to provide anesthesia for tubal ligation.

Twenty-six hours after delivery, the epidural catheter was used for uneventful epidural anesthesia (2% lidocaine with epinephrine 1:200,000) for tubal ligation, and the catheter was removed intact after the procedure. Two hours, 10 min after the injection of the lidocaine, the patient was observed to have full motor and sensory recovery from the epidural anesthetic. The patient had no complaints when discharged from the hospital the following day; she had been afibrile throughout her hospital stay.

On the 4th postpartum day, the patient began to experience low back pain and pain with ringing sensations in both lower extremities. Over the next 2 days, she developed subjective weakness of both lower extremities; she had difficulty standing because of the pain. Neurosurgical evaluation revealed severe back pain with dysesthesia in both legs. Her temperature was 98.8°F, and she denied having chills or fever for the previous 6 days. The site of the epidural catheter insertion was erythematous and indurated, and it exuded purulent material found to be filled with gram-positive cocci. She had normal sphincter tone and was essentially intact neurologically. Motor function of the lower extremities was difficult to evaluate because of her extreme pain but was believed to be slightly reduced. Magnetic resonance imaging (MRI) revealed an epidural mass effect at the L1-L2 interspace level consistent with epidural abscess (fig. 1). The mass enhanced peripherally with gadolinium DTPA, as would be expected with an abscess (fig. 2).

Surgical exploration was performed on the 6th postpartum day. A cutaneous dermal sinus tract at L1-L2 was traced from the skin through the subcutaneous fatty tissues and paraspinal muscles through the ligamentum flavum, into a bilateral posterior extradural abscess at L1-L2. The dura mater was intact. The abscess was drained and the cultures grew Staphylococcus aureus. Pathologic examination of the surgical specimen revealed subacute and chronic inflammation and granulation tissue consistent with epidural abscess, no evidence of osteomyelitis was seen. The organism was sensitive to cefazolin.

Except for a mild fever that lasted 2 days and then subsided, the patient did well postoperatively. She was discharged on the 8th postoperative day (14th postpartum day). By the time of discharge, her back pain and lower extremity pain had resolved, and she was walking without difficulty. There were no neurologic sequelae found at her 6-week follow-up visit.

Discussion

A review by Eisen et al. of 9,532 obstetric epidural anesthetics revealed no epidural infections. A retro-
dural catheter had been placed for labor and delivery in this patient, it would have been implicated as the cause of the abscess.9

Another type of infection after obstetric epidural anesthesia includes bacterial meningitis, two cases of which have been reported by Ready and Helfer.9 Both patients were treated with antibiotics without surgical intervention and recovered completely.

Unlike previous obstetric cases, our case may represent an epidural abscess caused by the introduction of skin contaminants (S. aureus) via the epidural catheter. The catheter had been inserted at L3-L4 and advanced 4 cm; the abscess was found at L1-L2, the approximate level of the catheter tip. No other area of infection was discovered that might have served as a source for bacteremia and hematogenous spread of the infecting agent. Also, the epidural catheter remained in place for 26 h postpartum. Because there was no infusion of anesthetic agent during this period of time, the potential antimicrobial effect of the local anesthetic10 was not present. However, local anesthetics also can interfere with normal lymphocytic function fundamental in defense against infection11; therefore, it would be difficult to conclude that the lack of local anesthetic infusion contributed to the formation of the epidural abscess.

Of the three previously reported cases of epidural abscess related to epidural anesthesia, the case reported by Ngan Kee et al.5 most closely resembles our report.

Fig. 1. Sagittal view of the lumbar magnetic resonance image (T1-weighted) showing an epidural mass effect at L1-L2 consistent with epidural abscess (arrow).

Fig. 2. Axial view of the gadolinium-enhanced magnetic resonance image at L1-L2 showing a posterior epidural mass with peripheral enhancement (arrow) and anterior displacement of the thecal sac.
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Their patient was asymptomatic until the 5th postpartum day after cesarean section, when she experienced high fever and bacteremia with blood cultures positive for *S. aureus*. She had difficulty urinating accompanied by severe back and leg pain and experienced paresthesia and weakness of both legs. Laminectomy revealed an epidural abscess extending to the left paravertebral muscle. Postoperatively, she had persistent radicular pain, from which she eventually recovered.

The case reported by Crawford, on the other hand, probably involved hematogenous spread from a postpartum endometritis. A high vaginal swab was taken on the 2nd postpartum day because of fever; cultures grew β-hemolytic streptococci (Lancefield Group B). Sixteen days postpartum, she was admitted to the hospital with severe back pain and stiffness with signs of possible meningeal irritation. She was febrile, and blood cultures grew Group B β-hemolytic streptococci of the same phage type as the culture from the high vaginal swab taken on the 2nd postpartum day. At laminectomy, superficial infection was not evident, and the extradural catheter tract was well healed. Laminectomy revealed three small fluid collections under pressure, cultures of which showed no growth. Histologic examination of the tissues removed during surgery showed no evidence of infection or abscess formation. The patient experienced complete recovery. We agree with Crawford's conclusion that this was a case of extradural infection. Ngan Kee et al., although mentioning Crawford's case, did not consider it an example of epidural abscess.

Although surgery was the approach used to drain the abscess in our case, Tabo et al. recently reported a technique involving percutaneous drainage of epidural abscess in two patients. The abscesses were identified using MRI; epidural needles and catheters were placed using fluoroscopic control. Drainage and irrigation followed by antibiotic administration achieved a successful result in both cases. The authors acknowledge that the usual method of treatment is immediate surgical laminectomy once the diagnosis of epidural abscess is made but chose their method because of the posterior-lateral location of the infection and because they were clearly identified by MRI. The percutaneous drainage of the abscesses was attempted as a preliminary noninvasive measure, and in both cases, surgery was not needed. They further acknowledge the danger of spreading the infection by irrigation and by perforation of the dura mater.

We believe the promptness with which surgical intervention was accomplished in our patient was a key factor in the prevention of permanent neurologic sequelae. Previous reports emphasize the fact that, if surgical exploration is delayed until a neurologic deficit is present, the prognosis for full recovery is poor. A 23% mortality has been reported in one series of epidural abscess.

Irreversible paraplegia, loss of sphincter function, and anesthesia may occur suddenly once the early signs of fever and severe back pain occur. The mechanism for this precipitous loss of neurologic function in many patients has been debated. The suggestion has been made that cord compression works in synergy with venous thrombosis and thrombophlebitis in the spinal cord to produce the sudden, catastrophic neurologic deterioration. Feldenzer et al., however, noted that of several hundred cases of spinal epidural abscess reviewed, only three showed histologic evidence of vascular thrombosis. They further stated that the dura appears to provide an excellent barrier against the inflammatory thrombotic effect of the epidural abscess on spinal vessels. Feldenzer et al. developed an experimental model by inducing *S. aureus* spinal epidural abscess in rabbits. Both histopathologic and microangiographic analysis suggested that arterial or venous occlusion did not accompany the initial neurologic deficit. They concluded that cord compression and ischemia, rather than infection-induced thrombosis, is responsible for the rapid neurologic deterioration. This ultimately leads to the severe, permanent neurologic sequelae. The clinical implication is that, in the early stages of neurologic deficit and spinal cord compression, prompt operative decompression may provide the best opportunity for functional recovery, thus explaining our choice of surgical intervention in this case rather than percutaneous epidural irrigation, as proposed by Tabo et al.

The diagnosis in our case was confirmed using MRI. The original MRI revealed an epidural mass effect that could have been interpreted as either hematoma or abscess (fig. 1). Gadolinium enhancement showed increased signal intensity, confirming the presence of an epidural abscess (fig. 2). MRI is as sensitive (91%) as myelography with computed tomography (92%). Furthermore, MRI eliminates the need for myelography in the diagnosis of epidural abscess. Lumbar puncture carries the risk of neurologic deterioration if performed below the level of block caused by an epidural abscess; there also is a possibility of inducing meningitis if the needle traverses the epidural abscess.
Unintended Transthoracic Pulmonary Artery Cannulation: A Complication of Central Line Insertion

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Central venous cannulation is associated with a number of complications, including unintended arterial puncture. The likelihood of such a complication is increased in the presence of abnormal or distorted anatomy, and it is important that the anesthesiologist be able to recognize and safely manage these complications. To our knowledge, there have been no reports of unintended transarterial pulmonary artery cannulation during central venous catheter insertion. We report such a case occurring in a patient presenting for major spinal instrumentation.

Case Report

The patient, a 14-yr-old, 32-kg girl with a history of a thoracic meningomyelocele (closed in the neonatal period) presented for Luque rod insertion for correction of an extreme thoracolumbar kyphoscoliosis. She was otherwise in good health, was taking no medications, and was known to have a latex allergy. A right-sided ven-

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