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Two cases of spinal epidural abscess with granulation tissue associated with epidural catheterization

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Abstract
Two cases of spinal epidural abscess are reported whose abscesses became granulated after epidural catheterization. Although emergency surgical intervention was performed almost within 24 h after the diagnosis of epidural abscess in case 1, the patient revealed a poor outcome. After laminoplasty, case 2 received lumbar epidural catheterization, and he had a complete recovery. The abscesses were recognized to spread around the catheter insertion site of the operative procedure in both cases, and MRI in case 2 showed the connection between the epidural abscess and the interspinous space where the catheter had been inserted. Methicillin-sensitive *Staphylococcus aureus* (MSSA) was identified at the operative field in both cases. Also, MSSA was identified at the subcutaneous abscess around the catheter in case 1 and at the catheter tip in case 2. Those findings suggest the midpoint of the abscess is the puncture site and that MSSA is found in or around the catheter. Infection at epidural catheterization seems to be caused by catheter insertion or skin contamination after catheterization. As those catheterizations were completed in the outpatient theater, we conclude that epidural catheterization should be performed in the operating room or with a restricted aseptic technique.

Key words Epidural catheter · Skin contamination · Granuloma

Introduction

Reports of epidural abscess have been increasing for the past decade, and the prognosis is sometimes poor [1–4]. We report two cases that needed surgical intervention because the abscesses were granulation tissue.

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Case 1

A 55-year-old man complained of herpetic neuralgia that was identified on his right scapular region (T3). After sterilization of the skin by 0.5% chlorhexidine gluconate with 80% ethanol three times, an epidural catheter was inserted through the interspinous space between T5 and T6, and a patient-controlled analgesia apparatus (Bard Ambulatory PCA, C; R Bard Insurance, North Reading, MA, USA) was connected to the catheter after admission on the 8th day after onset. On the 4th day of catheter insertion, the patient had a mild fever (37.4°C), and the skin around the site where the epidural catheter had been inserted revealed flaring and swelling; therefore, the catheter was removed and a culture was prepared from its subcutaneous small abscess. Culture of the abscess around the catheter showed methicillin-sensitive *Staphylococcus aureus*. On the same afternoon, he complained of a tingling pain on the abdominal wall and high back skin with movement, but no neurological disorder was detected. Cefotiam hydrochloride 2 g/day was administered intravenously, but his body temperature reached 38.0°C, and leukocytosis (WBC 18 800 mm^{3-1}) was recognized the next morning. On the night of the 6th day after catheter insertion, he complained of numbness of bilateral thighs with mild neck rigidity. As hypesthesia beneath the L1 dermatome without motor weakness was recognized, a computed tomogram of the thoracic vertebrae was obtained; however, it revealed no apparent disorder. Because the spinal segment level of hypesthesia reached T4 and magnetic resonance imaging (MRI) was not available, a myelogram was taken on the evening of the 7th day.

After recognition of the interruption beneath the T7 level of the contrast media on the myelogram, emergency laminectomy (T3–T7) was performed on the same night. A granulation abscess was seen in the spinal epidural region compressing the spinal cord anteriorly

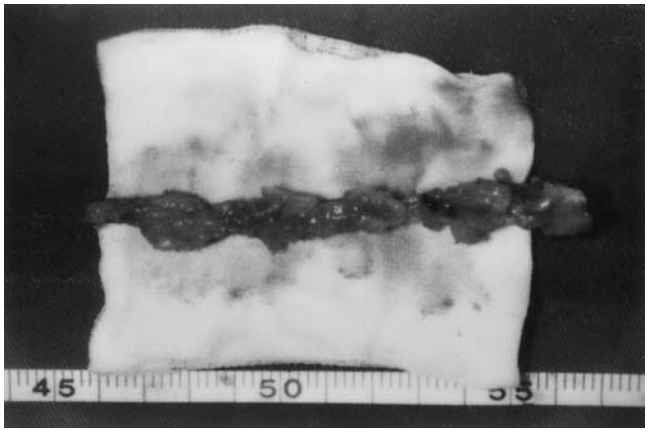

Fig. 1. Extracted thick and broad granulation tissue

between T3 and T7. As much of the granulation abscess with hypertrophied ligamentum flavum as possible was removed. Figure 1 shows the hypertrophied ligamentum flavum, which has a thick, long configuration. Bacterial culture of the abscess isolated methicillin-sensitive *Staphylococcus aureus*. The abscess and inflammatory granuloma were diagnosed pathologically as an inflammatory granulocyte infiltration. Two doses of cefotiam hydrochloride 2 g and gentamicin sulfate 40 mg per day were administered for a week postoperatively.

Although the decompression surgery was performed almost within 24 h of the onset of the epidural abscess when the neurological signs and symptoms were detected, his paraplegia was not definitely improved. One month after the laminectomy between T3 and T7, motor weakness of both legs and the sensory deficit beneath T8 were improved, manual muscle test (MMT) of lower extremities became 1-2/5, and analgesia was lowered to T12. Three months after the operation, MMT of the ankle joints was 3 of 5. He became able to walk on crutches, but genitourinary impairment remained.

Case 2

A 29-year-old man admitted to the orthopedic ward was diagnosed as having lumbar disk herniation. He had complained of left sciatica for 5 months. MRI showed L3–L4 and L4–L5 disk herniation and left L4 root deficit, and the computed tomographic myelogram also indicated compressed spinal cord at the L4–L5 disk ventrally and a blurred root sleeve.

After sterilization with 10% povidone-iodine twice for 60 s followed by sodium thiosulfate with 40% ethanol, an epidural catheter was inserted through the L2–L3 interspinous space medially and passed 5 cm cephalad. Continuous infusion was started with a Two Day Infusor (Baxter Healthcare, Deerfield, IL, USA) at 2 ml·h^{-1} on the 10th day of the insertion. Although sciatica improved at rest, the patient complained of lumbago and pain around the catheter insertion on the 12th day. As body temperature increased to 37.6°C and the lumbago and the pain around the site of catheter insertion became worse, the catheter was removed and its tip used to prepare a culture on the 13th day. Culture of the tip revealed methicillin-sensitive *Staphylococcus aureus*. Leukocytosis was indicated as WBC was 15 800·mm$^{3-1}$ and C-reactive protein (CRP) was 19.8 mg·dl^{-1}. Besides lumbago, the patient complained of throbbing headache, nausea, and rigor, and Kernig sign was recognized on the 15th day; therefore, cefotiam hydrochloride 2 g per day was administered. On the 18th day after catheter insertion, MRI suggested that the lumbar epidural abscess at L1–L3 had compressed the spinal cord anteriorly (Fig. 2A) and contained gas (Fig. 2B) in T_1-weighted MR. It was not monotonous and was uniformly enhanced by gadolinium-diethylenetriaminopentoacetic acid (DTPA). Figure 2C shows the L2–L3 interspinous high-density legion that connects the epidural mass in the T_2-weighted MR image. The neurological deficit was not obvious, but the compression was so severe that decompressive surgery was performed. After laminoplasty from L1 to L4, the outer dura mater covered by degenerated granulation tissue was removed as much as possible. Culture of the granulation identified methicillin-sensitive *Staphylococcus aureus*. Panipenem-betamipron 1 g per day for 3 days followed by cefotiam hydrochloride 4 g/day for 11 days was administered after the operation, simultaneously with amikacin sulfate for 14 days. Three days after the operation, he became afebrile, and WBC revealed 10 600·mm$^{3-1}$, with CRP reduced to 2.4 mg·dl^{-1}. He was able to walk without any assistance at the 16th postoperative day and was discharged 1 month after the operation.

Discussion

Although epidural analgesia is commonly employed in many circumstances, the epidural analgesia technique is normally performed aseptically. However, subcutaneous abscess and epidural abscess are sometimes reported. The instance of epidural abscess has increased statistically in recent decades [1]. The most frequent causes are intravenous drug abuse, diabetes mellitus, multiple medical illnesses, trauma, prior spinal surgery, morbid obesity, and spinal nerve block, as reported by Rigamonti et al. [1]. They also described the outcomes of 75 patients: 50 patients (66%) had a good outcome, 11 had a fair outcome, 6 patients had a poor outcome, and 8 patients died.

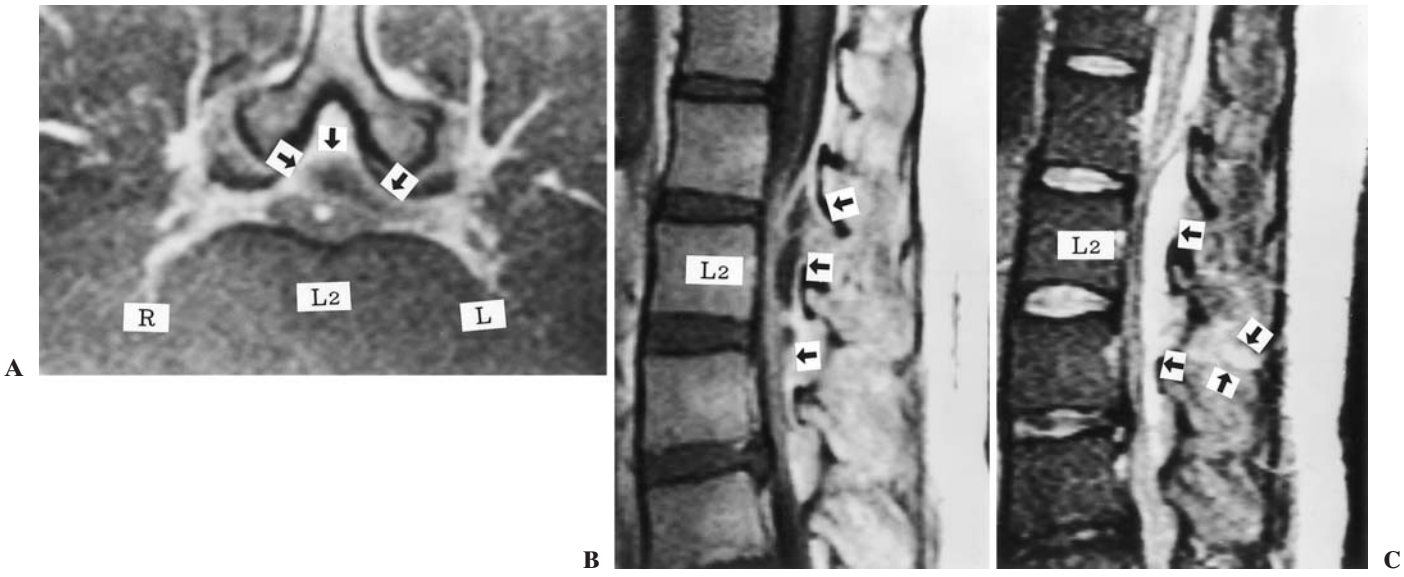

Fig. 2. **A** The isointense magnetic resonance (MR) abnormal tissue (*arrows*) to the spinal cord and cerebrospinal fluid (CSF) (T_1 weighted) compressed the cauda equina anteriorly from L1 to L3 (uniformly enhanced with gadolinium-DTPA) (axial view). **B** The thecal sac is compressed anteriorly by the iso-MR intensity abnormal tissue (*arrows*) (T_1 weighted; sagittal view). **C** Hyperintensity abnormal tissue in the epidural space and the L2–L3 interspinous space (*arrows*) are seen in the T_2-weighted MRI (sagittal view)

Tung et al. [2] reported that an epidural mass was designated as a "phlegmon" if the abnormal tissue enhanced uniformly or if enhancement was only slightly heterogeneous, and as an "abscess" if there was any area of peripherally or nonenhancing tissue. Several investigations of a radiologic–pathological correlation [3] support the designation of epidural inflammatory masses as either "phlegmon" or "abscess," based on the pattern of contrast enhancement. Predominantly homogeneous contrast enhancement correlates with phlegmonous tissue, which consists of vascularized granulation tissue with microabscesses, but little or no drainable pus. In contrast, a primarily peripheral pattern of contrast enhancement (or rare nonenhancing epidural inflammatory mass) is consistent with a frank abscess containing a liquefied, purulent core.

During the past several years, many reports of spinal epidural abscess (SEA) following epidural analgesia have been published. In a review of SEA, Reihsaus et al. [3] report that the prognosis of SEA following epidural analgesia was poorer than that of SEA without epidural intervention. Complete recovery was experienced by 43% of patients with spontaneous SEA and 38% of patients with SEA following anesthesiological interventions in the epidural space. Paresis or paralysis developed in 27% of SEA patients following anesthesiologic interventions, which was significantly higher than in patients with spontaneous SEA (15%). All in all, there has been no improvement in outcome for patients with SEA following anesthetic or analgesic procedures in the epidural space. A possible explanation for this observation could be that signs of the infection in the epidural space are confused with consequences of the operation or the disorder for which the operation was performed, which could in turn lead to delays in the diagnosis of SEA. Wang et al. [4] reported that overall recovery rate for patients with paresis/plegia after epidural abscess was 20%. No patients with paresis/plegia following a thoracic abscess recovered in contrast to a 50% recovery rate for patients with lumbar epidural abscess.

As seen in our case 1, there was a broad phlegmonous tissue in the thoracic epidural space, which consists of vascularized granulation tissue with microabscess, and there was no improvement in outcome.

The pathogenesis of SEA following epidural catheterization has been speculated by Wang et al. [5] to be that patients with previous epidural catheterization should not develop a spontaneous abscess unrelated to the epidural catheter, because the abscesses in all patients were found at the same level as the previous catheter, and they occurred within a relatively short time interval after epidural catheter insertion.

Tay and Lee [6] suggest that catheter-related SEA is (1) introduced directly during needle or catheter insertion, and that (2) spread of infection may occur directly from an adjacent site of infection, (3) indirectly via hematogenous spread from a distant focus, or (4) after injection of contaminated fluid; particularly with the use

of multidose vials, the degree of catheter manipulation and colonization and migration of microorganisms along percutaneous catheters are major factors.

Phillips et al. [7] also speculated that infection of the epidural space may occur at the time of insertion of the epidural catheter or by subsequent contamination by one of three different ways: through contamination of the skin site and subsequent spread along the catheter track, by hematological spread, or by intraluminar contamination via a contaminated syringe or contaminated local anesthetic solution.

These reports suggest that the abscesses in all patients were found at the same level as the previous catheter in the MRI examination. In case 1, the abscess was spread between T3 and T7, the center of which (T5) was the catheter insertion interspace site (T5–T6). Our case 2 showed a simultaneous low-density legion between the posterior lumbar epidural space and the L2–L3 interspinous space. This result suggests that hematological spread or intraluminar contamination is unlikely to be the genesis of the abscess, that the abscess was formed by direct bacterial contamination at catheterization or through contamination of the skin site and subsequent spread along the catheter track as reported by Phillips et al. Because our two cases both had subcutaneous abscesses around the catheters, inflammatory changes were mainly seen around the catheters.

In case 1, as within 5 days the epidural infection had occurred and the subdural abscess was recognized, the most possible explanation for abscess formation, that the subcutaneal infection progressed into the epidural space, is more acceptable than the contamination of the perfusate through the interspinous region along the catheter.

As seen in the sagittal MRI (Fig. 2C) in case 2, it is possible that bacterial contamination at the needle insertion site or bacterial invasion from the skin is more appropriate than perfusate infection through the catheter.

After the experience of case 1, we changed skin sterilization from three times with 0.5% chlorhexidine gluconate with 80% ethanol to twice with 10% povidone-iodine sterilization for 60 s followed by sodium thiosulfate containing 40% ethanol. However, evidence that povidone-iodine has a greater sterilization effect than chlorhexidine gluconate with 80% ethanol has not been found.

After case 2, dressing change was altered from once per 3 days to every other day, and a bacterial filter (Flatfilter 0.2 μm; Pall, East Hills, NY, USA) was connected to the catheter. To the skin around the catheter, a patch containing chlorhexidine gluconate (Biopatch; Johnson-Johnson, Somerville, NJ, USA) was attached, and it was changed on alternate days. Phillips et al. did not state that daily sterilization and dressing change are needed; they only recommended daily inspection and a closed apparatus connection under sterile conditions, and that the insertion of the catheter be performed in the operating room (OR). Catheter insertion for our two cases was performed in the outpatient clinic, not in the OR, although there has been no epidural abscess among patients in many daily epidural anesthesia procedures in the OR. As Phillips et al. recommended, catheter insertion should be completed in the OR.

Prophylaxis of the spinal epidural abscess during epidural catheterization, sterile technique in the OR, and daily observation of the site of the catheter insertion are adequate. Laboratory data such as white blood cells, C-reactive protein, and erythrocyte sedimentation rate must be checked, and we must be aware of signs and symptoms such as fever, back pain, radicular pain, and dysesthesia, with immediate MRI examination needed during epidural catheter insertion.

References

1. Rigamonti D, Liem L, Sampath P, Knoller N, Numaguchi Y, Schreibman DL, Sloan MA, Wolf A, Zeidman S (1999) Spinal epidural abscess: contemporary trends in etiology, evaluation, and management. Surg Neurol 52:189–197
2. Tung GA, Yim JWK, Mermel LA, Philip L, Rogg JM (1999) Spinal epidural abscess correlation between MRI findings and outcome. Neuroradiology 41:904–909
3. Reihsaus E, Waldbaur H, Seeling W (2000) Spinal epidural abscess: a meta-analysis of 915 patients. Neurosurg Rev 232:175–204
4. Wang LP, Hauerberg J, Schmidt JF (2001) Long-term outcome after neurosurgically treated spinal epidural abscess following epidural analgesia. Acta Anaesthesiol Scand 45:233–239
5. Wang LP, Hauerberg J, Schmidt JF (1999) Incidence of spinal epidural abscess after epidural analgesia: a national 1-year survey. Anesthesiology 91:1928–1936
6. Tay SM, Lee R (2001) Case report: catheter-related epidural abscess. Ann Acad Med Singap 30:62–65
7. Phillips JMG, Stedeford JC, Hartsilver E, Roberts C (2002) Epidural abscess complicating insertion of epidural catheters. Br J Anaesth 89:778–782