A Case of Extensive Sacral Decubitus Ulcer Complicated by an Epidural Abscess

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CASE REPORT

A 62-year-old man developed aspiration pneumonia. He had suffered from an acute subdural hematoma and brain contusion caused by a traffic injury 6 years previously. He had been bedridden for rigid paralysis and could not communicate at all. During the treatment of pneumonia, his decubitus ulcer got worse and became infected. The patient was subsequently transferred to our hospital.

On this admission, the sacral decubitus ulcer measured 10 × 8 cm in size and had formed a huge pocket (Fig. 1), accompanied by pus. He had a high C-reactive protein (CRP) level, anemia, and prominent malnutrition.

A pocket incision and debridement of the wound were performed on the second day after his admission. After debridement, the size of the ulcer was 30 × 20 cm and the pocket, which measured 15 cm in depth, still remained in the cranial side of the ulcer (Fig. 1). Exposure of the spinal bone was not observed during operation, and no deep tissue infection was observed on plain computed tomography (CT). After surgery, we continued to administer intravenous antibiotics and irrigate the wound with physiological saline.

The amount of wound exudate gradually decreased, and granulation tissue formed on the surface of the ulcer. The CRP levels also decreased, and hemodynamic stability was achieved. We switched the intravenous administration of antibiotics to oral administration on hospital day 20. However, we resumed intravenous antibiotic therapy on hospital day 23 because the CRP levels increased again. Contrast-enhanced CT and gadolinium-enhanced magnetic resonance imaging (MRI) revealed a lesion with a ring-formed contrasting effect in the L5/S1 level vertebral canal (Figs. 2 and 3), which indicated an epidural abscess. As the abscess did not decrease in size, despite intravenous antibiotic therapy for 3 weeks, surgical treatment was performed for the epidural abscess. Laminectomy was performed to open the spinal canal, and fat-like granulation tissue and pus were observed in the vertebral canal. The defect was then covered with a gluteal perforator-based flap. There was no recurrence of the decubitus ulcer at 5 months postoperatively (Fig. 4) or any recurrence of the epidural abscess on contrast-enhanced CT.

DISCUSSION

An epidural abscess is a rare disease with a frequency of 2 to 25 cases per 100,000 hospital admissions, and its mortality rate is 5%.1 The compression of the spinal cord between the abscess and the bones of the spinal column can cause severe symptoms and permanent complications and may lead to death.1–3 Few cases of epidural abscess complicated by a decubitus ulcer have been previously reported.1–3

The classic triad of a spinal epidural abscess is a fever, spinal pain, and neurologic deficit. However, only a minority of patients with epidural abscesses complete this triad.

Once a spinal epidural abscess is suspected, then imaging of the spinal column is imperative. MRI with gadolinium enhancement is highly sensitive (more than 90%) and is the most useful imaging modality.4 In the present case, the lesion was detected by both contrast CT and contrast MRI.

Disclosure: The authors have no financial interest to declare in relation to the content of this article. The Article Processing Charge was paid for by the authors.
Conservative treatment with antibiotic therapy alone without surgery may be selected in patients with no symptoms of a serious loss of the spinal cord or cauda equina function. In those cases, frequent neurological examinations, blood testing, and MRI examinations are required. In the present case, communication was impossible and the patient was in a state of rigid paralysis because of the sequelae of the cerebral contusion and acute subdural hematoma, and the staging of the neurological symptoms was unclear. We initially administered antibiotic therapy; however, the abscess did not decrease in size on imaging, and thus, debridement, laminectomy and flap surgery were performed.

Because many of the decubitus patients are bedridden or paraplegic, there is no way to know the neurological symptoms of the lower limbs. An epidural abscess by itself...
is rare; however, in the case of a decubitus ulcer complicated by an epidural abscess, the diagnosis may be delayed because the symptoms, except for a fever, are difficult to notice. In the present case, no back pain or neurological symptoms in the lower extremities were detected. If we had not been suspicious of the repeatedly elevated CRP levels and imaging results and wound closure had been performed unaware of the presence of an epidural abscess, then the symptoms of the infection would have worsened.

In cases of a decubitus ulcer without symptoms of infection or elevated CRP levels, debridement and wound closure with a flap can be performed simultaneously. However, wound closure of a decubitus ulcer with severe infection and sepsis should only be performed after a complete examination, including imaging and control of the infections.

In the present case, it remained unclear whether the abscess was the cause of the infection/ulcer or a consequence. During the treatment of pressure ulcers, osteomyelitis as a deep infection is sometimes encountered. However, an epidural abscess is rare. Therefore, an epidural abscess should be kept in mind in the differential diagnosis of deep tissue infection of a severe decubitus ulcer of the back and sacral region.

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