Spinal subdural abscesses are very rare. As detailed in a review by Bartels, et al., the condition was first described in 1927, and since that time only slightly more than 50 cases have been reported. We present the first case, to our knowledge, in which an unintended durotomy (or dural tear) that occurred during a routine laminectomy may have contributed to the spread of systemic infection into the subdural space.

CASE REPORT

History. A previously healthy 70-year-old woman presented with a 2-month history of progressive weakness and numbness in the lower extremities. Admission MR images demonstrated a severe degenerative central canal stenosis at the T10–11 spinal level. A laminectomy was performed, and the patient experienced initial improvement of her symptoms. Nevertheless, by 2 years postoperatively recurrent leg weakness and paresthesias had developed, and she had progressive gait difficulties. She did not report bladder, bowel, or sexual dysfunction at presentation.

Examination. The patient had bilateral leg weakness, which was more marked in the proximal muscle groups (4+/5). There was decreased sensation to pinprick below the T-10 dermatome. Reflexes were hyperactive in the lower extremities, although the Babinski sign was negative. An MR imaging study revealed recurrent/residual spinal cord compression at the T-11 level that was caused by degenerative canal stenosis (Fig. 1).

Revision Operation. A revision laminectomy and epidural decompression (T10–11) were performed. At surgery, the stenosis was found to be caused by a combination of calcified hypertrophic ligamentum flavum and epidural scar tissue that was tightly adherent to the dura mater. During dissection of this scar tissue, a small dural tear was created in the thecal sac at the T-11 level. No cerebrospinal fluid leak was apparent; the arachnoid layer appeared to be intact. The defect was repaired with Gelfoam (Pharmacia, Kalamazoo, MI) and Tisseel (Baxter Corp., Mississauga, Ontario, Canada).

Postoperative Course. The patient reported a significant resolution of her sensory symptoms and a slight improvement in her motor strength. On the 1st postoperative day she experienced a high fever (39°C), right upper-quadrant abdominal pain, shortness of breath, hypoxia, and hypotension. Her white blood cell count was normal. A chest x-ray film revealed bilateral lower lobar collapse consistent with pneumonia. Blood cultures were positive for Staphylococcus aureus. Medical and infectious disease consultants diagnosed nosocomial pneumonia and sepsis, and the patient was treated with broad-spectrum, intravenously administered antibiotic drugs (2 g cefotaxime and 600 mg clindamycin every 8 hours). Once antimicro-
bial sensitivities were determined, the antibiotic regimen was changed to 400 mg moxifloxacin taken orally twice a day. The patient’s vital signs stabilized and her symptoms improved.

Ten days later, the patient began to experience low-back pain with the sensation radiating into her buttocks and thighs. A fluctuant superficial mass was palpable at the wound. Neurological findings were unchanged. An MR image (Fig. 2) revealed an extensive subdural collection extending from the surgical site at T-11 to the sacral region. Although the white blood cell count remained normal, the erythrocyte sedimentation rate and C-reactive protein level were elevated (84 mm/hour and 39.2 mg/L, respectively). The superficial fluid collection was aspirated, and culture results were consistent with *S. aureus*.

At about this time, the patient experienced acute respiratory insufficiency. Doppler ultrasonography studies of the legs revealed deep venous thrombosis, and pulmonary embolism was confirmed on computerized tomography scans of the chest. Intravenous heparin therapy was administered. Because of the medical fragility of the patient and the extensive nature of the spinal subdural collection, we believed that she would not tolerate an extensive laminectomy and drainage procedure at that time. The moxifloxacin was discontinued and the patient was placed on a regimen of intravenously administered meropenem (1 g every 8 hours). After initiation of this broad-spectrum antibiotic therapy, the patient’s back and leg pain improved. She never showed symptoms or signs of meningitis.

A follow-up MR image was obtained to assess the status of the subdural collection 1 week later. This study demonstrated a slight increase in the size of the empyema (Fig. 3). By this time, the patient’s neurological condition had begun to deteriorate. She reported the recurrence of back pain with radiation down both legs. This progressed over 24 hours to include bilateral leg paresthesias and intermittent urinary incontinence. The intravenous heparin was discontinued and a superior vena cava filter was inserted in preparation for surgery.

**Second Operation.** We performed a lumbar laminectomy (L2–4). The absence of abnormal material in the epidural space was confirmed, and the dura was found to be bulging and nonpulsatile. A midline durotomy was performed, and copious amounts of thick pus were aspirated from the subdural space. No cerebrospinal fluid was encountered, and the arachnoid membrane beneath the empyema appeared markedly thickened and sclerotic. The dura was sutured closed in a watertight fashion.

**Second Postoperative Course.** There was marked postoperative improvement in the patient’s neurological status, with almost complete resolution of her lower-extremity symptoms. She was able to ambulate initially with a walker, and then unassisted, by 1 week postsurgery. A follow-up MR image obtained 26 days postoperatively demonstrated almost complete resolution of the empyema despite the subtotal lumbar exposure (Fig. 4). After the antimicrobial sensitivities were known, the antibiotic regimen was changed to 2 g cloxacillin administered intra-
Spinal subdural empyema

includes fever with or without back pain; the second stage
symptoms appear in stages of progression analogous to
simultaneously. We believe that the anatomical barrier of the
epidural abscess and subdural empyema may occur simul-
migration of the organism into the subdural space. Spinal
dural space was seeded hematogenously, with subsequent
fections, we postulate that it is most likely that the epi-
dural abscesses are much more common than subdural in-
secondary to nosocomial pneumonia. Because spinal epi-
duration was disrupted during the initial surgery by a dural
ear, facilitating subdural extension of the infection. An-
other possibility, although less likely, is that the surgi-
cal wound was contaminated at the time of surgery. In ei-
other case, however, the spread of infection to the subdural
space would have been aided by the dural tear. To our
knowledge, there are no other reported cases of this asso-
ciation.

The symptoms of spinal subdural empyema include fe-
ver, back pain, radiculopathy, and neurological deficits
that vary according to the spinal level of the lesion. These
symptoms appear in stages of progression analogous to
those seen in epidural spinal abscesses. The first stage
includes fever with or without back pain; the second stage
consists of neurological symptoms such as motor defi-
cit, sensory loss, and sphincter dysfunction; and the third
manifests as paralysis and complete sensory loss below
the level of the lesion. The rate of progression through
these stages is unpredictable. Interestingly, the duration of
symptoms before treatment does not appear to affect the
outcome.3 Our patient presented in the aforementioned
manner, although treatment was successfully implement-
ed before progression to the third stage.

The diagnostic modality of choice for spinal subdu-
ral empyema is Gd-enhanced MR imaging. The effec-
tiveness of contrast-enhanced computerized tomography
scanning is not widely reported in the literature, but
is considered to be less sensitive and specific than MR
imaging.

It is generally recommended that spinal subdural empy-
ema be treated with immediate, complete surgical drain-
age followed by parenteral antibiotic therapy, given the
unpredictable course of progression of the condition and
the potential for a poor outcome. In the five reported cases
of nonsurgically treated patients prior to 1992 (to our
knowledge there have been no nonsurgically treated cases
reported since then), four died and the one survivor had
only a partial neurological recovery.3 In our case, immedi-
ate surgery was not advisable because of the patient’s
medical condition. Despite a 2-week delay before surgery
and a limited surgical drainage, the patient made a nearly
complete neurological recovery. Sathi, et al.,5 also report-
ed favorable results after performing a limited surgical
drainage of a spinal subdural abscess. When surgical treat-
ment is implemented in a timely fashion the results are
generally good, and nearly all patients who survive will
have significant or complete neurological recovery.3

DISCUSSION

Spinal subdural empyema is a rare and serious condi-
tion. Bartels, et al.,1 reported a mortality rate of 25%, with
some neurological improvement seen in 47.7% of cases,
and complete recovery in only 27.3%. Hematogenous
spread from a distant focus is the most common route of
infection; direct extension of local infection is less
common. The most frequent causative agent is S. aureus,
which is involved in more than 80% of known cases.3,6

In our case, the patient suffered an S. aureus septicemia
secondary to nosocomial pneumonia. Because spinal epi-
dural abscesses are much more common than subdural in-
fections, we postulate that it is most likely that the epi-
dural space was seeded hematogenously, with subsequent
migration of the organism into the subdural space. Spinal
epidural abscess and subdural empyema may occur simul-
taneously.3 We believe that the anatomical barrier of the
dura was disrupted during the initial surgery by a dural
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Venous every 8 hours and 300 mg oral rifampin. This
therapy was administered for a total of 10 weeks after the
drainage procedure. There has been no recurrence of in-
fection.

CONCLUSIONS

Spinal subdural empyema is a rare disease with an un-
predictable progression and potentially devastating conse-
quences. When such an infection is suspected, urgent in-
vestigation with neuroimaging is highly recommended.
Immediate and complete surgical decompression, irriga-
tion, and drainage is the treatment of choice, and should
be followed with appropriate antibiotic therapy.

In our case, a dural tear is postulated to have facilitated
the subdural spread of infection in the presence of system-
ic sepsis. This case also demonstrates that in instances in
which the patient is medically fragile, delaying surgery to
allow for medical optimization of the patient’s condition
may be feasible. Ultimately, surgical decompression is ne-
necessary.

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