Spinal subdural abscess

Case report

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Only 44 cases of spinal subdural abscess have been reported to date. The authors present another case and review the relevant literature. The findings of intraspinal gassification or computerized tomography scans and Escherichia coli as the causative organism have not previously been described in relation to spinal subdural abscess. Most frequently, Staphylococcus aureus is the responsible organism. Hematogenous spread of infection from a distant source often takes place. In a surprising number of incidences, iatrogenic causes are the primary foci of spinal subdural abscess.

Spinal subdural abscess is an unpredictable disease, with an unfavorable outcome if left untreated. If there is suspicion of a spinal subdural abscess, urgent radiological examination followed by immediate surgical drainage and appropriate antibiotic therapy is warranted.

Key Words • abscess • Escherichia coli • intraspinal gassification • spinal lesion

Spinal subdural abscesses are rare. Most neurosurgical textbooks omit the subject or mention it only briefly. Recent experience with a patient exhibiting this abscess stimulated us to conduct a search of the literature. Since the first description by Sittig in 1927, only 44 such cases have been described. These cases are reviewed and our case is presented. The need for proper diagnosis and prompt surgical intervention is stressed since treatment of spinal subdural abscess has a high probability of success, whereas the outcome of nonsurgically treated patients is bleak.

Case Report

This 55-year-old man with insulin-independent diabetes mellitus and moderate radicular pains in the left arm due to cervical spondylarthrosis was referred to our neurosurgical unit. The patient presented with progressive quadriplegia that was more pronounced on the left side, urinary incontinence, and sensory loss, particularly on the right side. These symptoms had developed within a 24-hour period. During the 2-week period before admission, he had suffered high fevers with chills and lancinating pains in all limbs. He had no history of instrumentation, such as catheterization or cystoscopy.

Examination. On admission, the patient's general physical examination showed no abnormalities except for a fever of 38.4°C. There were no signs of peripheral infection. Neurological examination revealed spastic paresis below the C-6 level, particularly pronounced on the left side. The triceps reflex was absent on both sides, while the tendon jerks of the legs were hyperreactive with bilateral Babinski's sign. Sensation was diminished below the T-1 level. A stiff neck was also noted.

The patient's erythrocyte sedimentation rate was elevated (80 mm/hr; normal: < 5 mm in 1st hour), as were the number of leukocytes (13.2; normal < 10.0 × 10⁹) and the blood glucose level (18.6 mmol/liter; normal 3.5 to 6.5 mmol/liter). All other specimen values fell within the normal ranges. Cultures of blood, urine, and sputum showed no growth.

A computerized tomography scan disclosed air in the spinal canal from C-5 to C-7 on the left side (Fig. 1). Discrimination between epi- and subdural localization was not possible. Because a spinal abscess was suspected and therefore surgical intervention was warranted, no further radiological examination, such as myelography, was carried out.

Operation. An emergency C5–7 laminectomy was performed. No epidural abscess was found. The strongly
tensed but nonpulsating dural sac was opened at the C6-7 level. The spinal cord was found to be enlarged, with an altered appearance. On the left side, a subdural empyema around the C6-7 roots was excised. To exclude intramedullary abscess formation, the spinal cord was punctured at the midline, where the swelling was most prominent; however, no pus could be aspirated. After closure of the dura, the wound was closed in layers.

**Postoperative Course.** Cultures of the empyema were positive for *Escherichia coli*. Intravenous administration of co-trimoxazole (960 mg twice daily), metronidazole (500 mg three times a day), and cefuroxime (2.5 gm four times a day) was started. The patient's neurological condition gradually improved. Two weeks after surgery, the administration of metronidazole and co-trimoxazole was stopped but the cefuroxime was continued for 6 weeks. Four weeks after surgery, the patient was referred to a rehabilitation center. Five months later, although his gait was spastic, he could walk without assistance. The intrinsic musculature of the left hand was still severely paretic (1/5), while the strength of the musculature of the right hand was nearly normal. He still complained of root pains in the left arm. Magnetic resonance imaging disclosed cervical spondylarthrosis with instability, a pathologically broadened myelin sheath from C-4 to T-1, and an abnormal signal intensity, interpreted as the result of an infectious process (Fig. 2).

In cooperation with an orthopedic surgeon, an intercorporal spondylodesis was performed from C-4 to C-7. The cervical spine was immobilized by a Minerva brace for a period of 3 months. Postoperatively, the patient's brachialgia disappeared. He has regained the ability to perform normal daily activities and to work full time at his former job.

**Discussion**

**Incidence of Spinal Subdural Abscess**

The exact incidence of spinal subdural abscess is unknown. Including our patient, only 45 cases have been reported. The relative scarcity of reported cases of spinal in comparison with cranial subdural abscesses has been ascribed to the absence of sinuses in the spine, the width of the epidural spinal space acting

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Onset of Symptoms</th>
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<tbody>
<tr>
<td></td>
<td>Initial</td>
</tr>
<tr>
<td>fever</td>
<td>25 (55.6%)</td>
</tr>
<tr>
<td>spinal/limb pain</td>
<td>38 (84.4%)</td>
</tr>
<tr>
<td>motor deficit</td>
<td>4 (8.9%)</td>
</tr>
<tr>
<td>sensory loss</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>sphincter dysfunction</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

* Late symptoms are defined as those presenting just prior to death or therapy.
Spinal subdural abscess

FIG. 3. Graph showing age distribution of 44 patients with spinal subdural abscess. The age and sex of one patient are unknown.

as a filter, and the centripetal direction of the spinal blood flow in contrast with the predominantly centrifugally oriented bloodstream in the head.\textsuperscript{11,26}

The male:female ratio of the cases reported was 1.1:1. The age of the patients at diagnosis ranged from 9 months to 77 years. In nearly 50\% of the reported cases, the patients were between 49 and 70 years of age. Spinal subdural abscess occurred more frequently in men in the fifth and sixth decade of life, while women were more affected in the seventh decade (Fig. 3).

Clinical Presentation

The symptoms of spinal subdural abscess include fever, spinal or root pain, and neurological deficit, depending on the location (Table 1). In 55.6\% of the cases, the initial symptom was fever, while 84.4\% presented with spinal and/or radiating pain. A combination of fever and spinal and/or root pain was present in 37.8\% of the cases. During the course of the disease, 82.2\% of the patients developed motor deficit, 57.8\% sensory loss, and 53.3\% sphincter disturbances.

Analogous to the stages of progression of symptoms proposed by Heusner\textsuperscript{17} for spinal epidural abscess, symptoms of spinal subdural abscess appear to occur in a certain sequence; Stage 1: fever, either in combination with or without spinal and/or root pain; Stage 2: motor deficit, sensory loss, and/or sphincter disturbances; and Stage 3: paralysis and complete sensory loss below the level of the lesion. In our case, symptoms developed in the order outlined above. The rate of progression from one stage to another is not predictable.

The duration of symptoms from their onset until patient death or therapeutic intervention ranged from 1 day to approximately 1 year. A division between acute (duration ≤ 1 week), subacute (2 to 8 weeks), and chronic (> 8 weeks) spinal subdural abscess can be made. Most spinal subdural abscesses were subacute (28 cases), six were chronic, and five acute; in six cases, the duration was not known. The location of the spinal subdural abscesses, upon which the extent of symptoms is dependent, is illustrated in Fig. 4.

Differential diagnosis includes acute transverse myelitis, spinal epidural abscess, epidural hematoma, vertebral osteomyelitis, and intraspinal tumor.\textsuperscript{2,7,9,24,26}

Neuroradiographic Examination

Myelographic studies may confirm the presence of an intraspinal space-occupying process. In 28 of the 30 cases in which myelography was performed, a blockage with or without intradural irregularities was found. The myelographic features are not as characteristic as some have described.\textsuperscript{24,34} In the remaining two cases, only intradural irregularities were found. Computerized tomography was performed in only seven cases; in five, the scans revealed an intraspinal space-occupying lesion. Intraspinal gasification, as demonstrated in our case, has not been described before. Spinal subdural abscess was diagnosed using magnetic resonance imaging in only one case.\textsuperscript{20}

Treatment

In conformity with the old adage ubi pus, ibi evacua, surgical drainage followed by appropriate antibiotic therapy is the optimum treatment (Table 2). In the surgically treated group, 32 (82.1\%) of the 39 patients

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Surgical</th>
<th>Conservative</th>
<th>Total</th>
</tr>
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<tbody>
<tr>
<td>Complete recovery</td>
<td>12 (27.3%)</td>
<td>0 (0%)</td>
<td>12 (27.3%)</td>
</tr>
<tr>
<td>Improved</td>
<td>20 (45.4%)</td>
<td>1 (2.3%)</td>
<td>21 (47.7%)</td>
</tr>
<tr>
<td>Death</td>
<td>7 (15.9%)</td>
<td>4 (9.1%)</td>
<td>11 (25%)</td>
</tr>
<tr>
<td>Total</td>
<td>39 (88.6%)</td>
<td>5 (11.4%)</td>
<td>44 (100%)</td>
</tr>
</tbody>
</table>

* The treatment in one patient was unknown.

J. Neurosurg. / Volume 76 / February, 1992
survived; in contrast, one (20%) of five patients in the conservatively managed group survived. All surviving patients improved or completely recovered. In no patient did the symptoms worsen, and in none was the outcome affected by the duration of symptoms (Table 3). Therefore, we recommend immediate surgical decompression and drainage if a spinal abscess is suspected after clinical and neuroradiographic examination. Appropriate antibiotic therapy using susceptibility testing of the cultured organism should follow surgery.

The issue of coexistent epi- and subdural abscesses should be addressed. If the dura is found to be tense but not pulsating after evacuation of an epidural abscess, intradural inspection is warranted. The consequences of ignoring a nonpulsating dura, including clinical deterioration and subsequent surgery, are well demonstrated by the case described by Sittig.

Frank pus, whether encapsulated or not, was most often encountered at surgery or autopsy (Fig. 5). The presence of frank pus in 62.5% of the chronic spinal subdural abscesses is remarkable. As expected, granulation tissue has not been found in patients with acute spinal subdural abscess.

Pathogenesis

The causative agent was identified in all but 12 cases (Table 4). Staphylococcus aureus was the responsible organism in 25 cases; Escherichia coli had not been encountered as the causative organism before our case. Organisms producing spinal subdural abscess reach the subdural space via several routes. Most frequently, hematogenous spread from a distant focus takes place with peripheral infections such as furunculosis being the primary source (Fig. 6). Direct extension of a contiguous infection is a different but infrequent route. The number of patients with spinal subdural abscess related to iatrogenic causes, such as lumbar puncture, injection of a local anesthetic agent, and discography, is striking. In 10 cases, accompanying factors or diseases that diminished the patient's resistance to infections were present. These are summarized in Table 5.

![Graph showing the relationship between the duration of symptoms and the finding at surgery or autopsy in 41 patients.](image)

**Fig. 5.** Graph showing the relationship between the duration of symptoms and the finding at surgery or autopsy in 41 patients. Acute = duration ≤1 week; subacute = duration 2 to 8 weeks; chronic = duration ≥8 weeks.

![Graph showing the foci in 45 patients with spinal subdural abscess.](image)

**Fig. 6.** Graph showing the foci in 45 patients with spinal subdural abscess.


**Spinal subdural abscess**

<table>
<thead>
<tr>
<th>Disease or Factor</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>lues</td>
<td>1</td>
</tr>
<tr>
<td>diabetes mellitus</td>
<td>6</td>
</tr>
<tr>
<td>Crohn's disease</td>
<td>1</td>
</tr>
<tr>
<td>rheumatoid arthritis</td>
<td>1</td>
</tr>
<tr>
<td>intravenous drug abuse</td>
<td>1</td>
</tr>
<tr>
<td>unknown</td>
<td>34</td>
</tr>
<tr>
<td>total</td>
<td>45</td>
</tr>
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</table>

**Conclusions**

Spinal subdural abscess is a rare but treacherous disease. The rate of progression of symptoms is unpredictable and outcome may be unfavorable. Therefore, if a spinal subdural abscess is suspected (for example, as in the case of the febrile patient with spinal pain after a lumbar puncture), urgent radiological examination is warranted, followed by immediate surgical drainage and appropriate antibiotic therapy. The coexistence of epi- and subdural abscesses should be noted.

**References**


Manuscript received March 28, 1991. Accepted in final form July 16, 1991. Address reprint requests to: Ronald H. Bartels, M.D., Department of Neurosurgery, University Hospital Nijmegen, Reinier Postlaan 4, 6500 HB Nijmegen, The Netherlands.