Rare Concurrent Retroclival and Pan-Spinal Subdural Empyema: Review of Literature with an Uncommon Illustrative Case

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Key words
- Cranial subdural empyema
- Retroclival empyema
- Spinal subdural empyema
- Streptococcus mitis
- Subdural empyema

Abbreviations and Acronyms
CSE: Cranial subdural empyema
CSF: Cerebrospinal fluid
CT: Computed tomography
ED: Emergency department
FLAIR: Fluid attenuation inversion recovery
IV: Intravenous
MRI: Magnetic resonance imaging
SSE: Spinal subdural empyema

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INTRODUCTION

Lowenburg first classified spinal subdural empyema (SSE) as a distinct clinical entity in 1918. Because of the rarity of this condition, the amount of literature concerning it is limited. Subdural empyema is a loculated collection of purulent material between the dura and arachnoid mater. It can present as a cranial subdural empyema (CSE) or very rarely as SSE.1 The lack of barriers in the spinal subdural space increases the risk for infection spread, so an SSE can spread to multiple levels within the spine and produce extensive spinal cord injury, including infarction, necrosis, and profound neurologic deficit within 48–72 hours. Delays in diagnosis and treatment are directly related to morbidity and mortality in patients with intracranial and SSE. CSE and SSE are neurosurgical emergencies, often requiring prompt surgical intervention. Subdural empyemas mostly occur secondary to other head and neck infections or to surgical procedures, with Staphylococcus aureus being the most common etiological agent. Although the epidemiology, etiology, pathophysiology, and symptomatology of CSE and SSE differ somewhat and occur separately, they rarely manifest together. To the best of our knowledge, only 3 reported cases of concurrent cranial and spinal empyemas have been caused by bacteria other than Streptococcus mitis.4,5

S. mitis, a member of the viridans type of streptococci and part of the normal bacterial flora of the human oropharynx, gastrointestinal system, skin, and female genital tract, is known to cause meningitis, subacute bacterial endocarditis, sepsis, and septic shock.6-10 S. mitis has never been recognized in the literature as a causative organism for CSE or SSE. The aim of this article is to review the literature concerning the clinical presentation, clinical course, and availability of various treatments for managing concurrently occurring SSE and CSE. On the basis of the literature, we propose a treatment plan for this very rare but potentially fatal condition. We also present an illustrative rare case of S. mitis causing copresentation of retroclival and pan-spinal subdural empyema.

METHODS

The literature in the Medline database was reviewed. Terms included, but were not
limited to, subdural empyema, cranial subdural empyema, spinal subdural empyema, viridans streptococci, Streptococcus mitis, skull base empyema, and retroclival empyema. A detailed search was undertaken to highlight the epidemiology, pathophysiology, risk factors, preceding associations, clinical presentation, diagnostic modalities, and treatment strategies for patients with CSE, SSE, or both. Only relevant journal articles written in English and related to our search key words were chosen for inclusion in our literature review, which produced a total of 72 articles including 5 book chapters, 13 case reports, 4 case series, 31 original articles, 17 review articles, and 2 meta-analyses.

RESULTS
Copresentation of CSE and SSE is extremely rare, and only 3 cases have so far been reported in the literature, caused by various etiologic agents as summarized in Table 1.4,5,20 Two of the patients recovered with no neurologic deficit, whereas 1 fatality was reported. All 69 articles were included in our review and the 3 case reports with copresentation of SSE and CSE were studied in detail. We also illustrate a case from our institution, which depicts the causality of copresentation by a rare etiologic bacterial agent.

Baker et al.4 reported a case of a young woman with multiple sclerosis who presented with generalized headache, neck pain, and third-nerve palsy and was diagnosed with CSE and SSE involving the entire spine.4 The patient did not survive, and the causative organism was identified as a beta-hemolytic streptococcus. The infection was believed to have originated from decubitus ulcers in the sacral area that disseminated into osteomyelitis.

Pompucci et al.5 reported on a 65-year-old woman with history of subacute lumber pain and sciatric neuritis who presented with acute altered mental status and meningismus. The subsequent imaging workup demonstrated multiple subdural abscesses along the entire spine with right frontal lobe and bilateral cerebellar involvement. After Streptococcus intermedius was identified as the causative agent, ampicillin was administered. Her condition deteriorated after remaining stable for a few days, and then a suboccipital craniectomy was performed to drain the CSE in the posterior fossa. The patient’s condition resolved with complete resolution of the SSE after 8 weeks of antibiotic therapy in the hospital.

Kamat et al.20 presented the case of a 2-year-old girl with an infected congenital dermal sinus and a history of recurrent meningitis. On presentation, the child was clinically stable with no neurologic signs or symptoms. Initial magnetic resonance imaging (MRI) of the spine revealed only a dorsal intradural sinus tract connected with an intraspinal mass. A few hours after the initial MRI, the child developed meningitis, flaccid quadriaparesis, and septic shock and went into cardiac arrest requiring resuscitation. She was given meropenem. Subsequent computed tomography (CT) and MRI of the brain demonstrated hydrocephalus requiring an external ventricular drain, and an intramedullary lesion involving the entire neural axis. The L5, S1, T11, and T12 vertebrae were subjected to emergency laminectomies and the infected dermoid cyst was removed. Culture revealed Proteus mirabilis infection, which was sensitive to meropenem. On follow-up serial MRIs there was no recurrence, and this child was discharged on day 21 with no residual neurologic deficits.

Illustrative Case
A 55-year-old, right-handed, white woman had a 2-week history of back pain. She had a history of “dental-cleaning” shortly before she sought care to her primary care physician for back pain and was sent home with analgesics (ibuprofen and hydrocodone) and muscle relaxants (Flexeril [cyclobenzaprine]). A few days later, she came to the hospital emergency department (ED) with increasing back pain. MRI of the spine was performed and initially was read as normal. Three days later, a code-sepsis was called, the patient was placed on empiric intravenous (IV) antibiotic therapy, and an infectious disease specialist was consulted. Lumbar puncture was performed but no cerebrospinal fluid (CSF) could be withdrawn. An infectious disease specialist diagnosed her with bacterial meningitis and changed her antibiotic to IV ceftriaxone. A day later, 4 days after the initial radiology reading, the MRI reading was changed to show pathologic changes suspicious for neoplastic or inflammatory disease. The neurologist at this point consulted the neurosurgeon, who transferred patient to intensive care unit and ordered an MRI of the entire spine to exactly outline the infection. The MRI reconfirmed thoracolumbar abscess, as well as a significant left iliopsoas and a smaller right iliopsoas abscess.

The patient was taken to the operating room by neurosurgeon for emergent evacuation of her thoracolumbar spinal abscess. During the operation, the epidural space was normal, so patient underwent T2–L5 laminectomies and durotomy. Extensive subdural empyema was encountered and was evacuated. Less-extensive surgical procedures such as limited/intermittent laminectomy or laminotomy are typically used in patients with epidural abscesses, but not in subdural abscesses/empyema, that requires durotomy to access the subdural space. As the spinal cord was edematous, and closure of the dura would compress the edematous spinal cord, the dura was expanded with a dural patch. After surgery and along with IV broad-spectrum antibiotics, dexamethasone was added to decrease the severe spinal cord edema (Figure 1). Cultures grew S. mitis, which was sensitive for ceftriaxone, and the IV antibiotic regimen was tailored based on the culture results, managed by the infectious disease specialist. As S. mitis is part of the oral flora, dental workup was performed and confirmed dental abscess, which was evacuated by a maxillofacial surgeon.

The patient’s condition improved significantly, and she remained neurointact postoperatively. Considering the extent of infection involving both soft tissue and bone, neurosurgery recommended 6–8 weeks of IV antibiotic therapy. Neurosurgery also recommended drainage of the significant iliopsoas abscess to decrease the burden of infection and for better response to antibiotic therapy. Patient was evaluated by orthopedic surgeon for her iliopsoas abscess, who deferred drainage of the iliopsoas abscess, due to declining levels of inflammatory markers. Postoperative surveillance MRI of the entire spine only showed enhancement around the operative field and meningeal enhancement in the cervicocranial meninges, and fluid attenuation inversion...
recovery (FLAIR) series did not show any signs of edema or inflammation in the cervical spinal cord or brainstem, findings that, given clinical and laboratory improvement and lack of neurodeficits, did not require surgical intervention. Surveillance MRI of pelvis with contrast showed slightly smaller but still significant left iliopsoas abscess, but the orthopedic surgeon wished for conservative treatment (Figure 2). The patient stayed neurointact clinically, and her laboratory inflammatory parameters continued to improve. She was recommended rehabilitation but wanted to be discharged home. Four weeks after surgery, she returned to the hospital’s ED with severe neck stiffness, fatigue in all extremities, and a severe radicular left arm pain. She stated that she had received 4 weeks of IV antibiotic therapy that was switched to oral antibiotic therapy under the direction of her infectious disease specialist on discharge. She appeared in a very septic, toxic, and meningitic state. MRI of brain and entire spine showed extensive subdural abscess extending throughout the cervical cord and superiorly, to a large retroclival empyema with severe edema of the brainstem and cervical spinal cord. The FLAIR MRI series showed very significant edema in the brainstem (Figure 3). Her infectious disease specialist saw the patient in the ED and immediately switched her oral antibiotic regimen to IV antibiotic (ceftriaxone). In view of the previous subdural empyema, the location of this new infection was suspected to be subdural.

One week after discharge and 5 weeks after surgery, she returned to the hospital’s ED with severe neck stiffness, fatigue in all extremities, and a severe radicular left arm pain. She stated that she had received 4 weeks of IV antibiotic therapy that was switched to oral antibiotic therapy under the direction of her infectious disease specialist on discharge. She appeared in a very septic, toxic, and meningitic state. MRI of brain and entire spine showed extensive subdural abscess extending throughout the cervical cord and superiorly, to a large retroclival empyema with severe edema of the brainstem and cervical spinal cord. The FLAIR MRI series showed very significant edema in the brainstem (Figure 3). Her infectious disease specialist saw the patient in the ED and immediately switched her oral antibiotic regimen to IV antibiotic (ceftriaxone). In view of the

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**Table 1. Demographics, Symptoms, Treatments, and Follow-Ups Reported in the Literature for Patients with Copresentation of CSE and SSE**

<table>
<thead>
<tr>
<th>Authors</th>
<th>Age, years</th>
<th>Sex</th>
<th>Presenting Symptoms</th>
<th>Causative Organism</th>
<th>Origin of Disseminating Infection</th>
<th>Treatment</th>
<th>Outcome</th>
<th>Follow-Up</th>
</tr>
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</table>
| Baker et al., 2003⁴      | 33         | Female | • Long-standing multiple sclerosis  
• Deteriorating level of consciousness  
• Focal neurologic symptoms  
• Generalized malaise and anorexia  
• Generalized headache and neck pain | Group C beta-hemolytic Streptococcus and anaerobes | Sacral and ischial decubitus ulcers | • External ventricular drain was inserted  
• No further intervention as the result of poor prognosis | Died on second day of admission | Not applicable                   |
| Pompucci et al., 2007⁷   | 65         | Female | • History of 3-week lumbar pain and sciatica  
• Rapid onset of lethargy and confusion  
• Neck stiffness and meningismus  
• Focal neurologic deficits | Streptococcus intermedius | Unknown                       | • Vancomycin and rifampin as initial antibiotics  
• Antibiotic regimen changed to ampicillin based on culture results  
• Suboccipital craniectomy | Complete disappearance of spinal and cranial empyema | Well and without neurologic deficit at 1-year follow-up |                          |
| Kamat et al.²⁰          | 2          | Female | • No presenting symptoms  
• Hours later developed meningism, fluctuating quadriparesis, septic shock, and cardiac arrest requiring resuscitation | Proteus mirabilis | Infected dermal sinus | • Started on meropenam  
• Emergent L5–S1 and T11–T12 laminotomies along with removal of dermoid | Discharged on day 21 with no residual neurologic deficit | Not available                   |

CSE, cranial subdural empyema; SSE, spinal subdural empyema.

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empyema with thick wall was found anterior to the brainstem and consistent with the preoperative MRI. The central soft part of the abscess was evacuated and the abscess walls were meticulously dissected off of the cranial nerves VII-XI, brainstem, and clivus (Video 1). Cervical cord showed severe adherences and abscess. Here the spinal cord was significantly edematous and indurated. The abscess was evacuated and the dura was expanded with a DuraGen patch (Integra Lifesciences, Plainsboro, New Jersey, USA) (Video 1).

Postoperatively, the patient survived, and the infection was controlled with aggressive and timely surgical debulking, decompression of the brain, and spinal cord and reinstitution of IV antibiotic therapy. The patient developed quadriparesis, a right third-nerve palsy, left facial palsy, and hoarseness as a result of the natural history of her severe brainstem and spinal cord infection. After a few weeks of improvement in the hospital she was transferred to a rehabilitation facility. Since then, she has been in a dependent situation.

**DISCUSSION**

**Epidemiology and Etiologies of Subdural Empyema**

SSE and CSEs are rare neurosurgical emergencies and often require prompt surgical removal. A range of 15%–20% of intracranial infections present as CSE.21-23 CSE mostly occurs in children and young adults. There is a 62% male predominance, which is not yet fully understood.24 CSE often occurs as a complication of other head and neck infections such as meningitis, otitis, and sinusitis, and of neurosurgical procedures.25-27 Meningitis is the most common cause of CSE in infants.20,27 In older children and adults, the infections most commonly leading to CSE are sinusitis and otitis, followed by osteomyelitis, which directly spreads through the Haversian channels.24,26,29 SSE is very much rarer than CSE, with fewer than 100 cases reported in the literature.4-5,20-23 It commonly presents in older persons between 60 and 70 years with a male-to-female ratio of 0.7.3 In our illustrated case, the patient had a history of dental abscesses, which could have been the source of infection and dissemination.

In a nationwide, prospective, observational cohort study of 1034 patients in the Netherlands from 2006 to 2011, Jim et al.25 found subdural empyema in 28 (2.7%) patients with bacterial meningitis. *Streptococcus pneumoniae* was identified as the causative agent in 93% (26) of these cases and *Streptococcus pyogenes* in 3% (1), whereas the one remaining patient had negative CSF cultures. In a similar observational cohort study including 1322 patients with community-acquired meningitis in the Netherlands, Lucas et al.24 found 26 (2%) of the cases to be caused by *S. pyogenes*, and among these 26 cases, subdural empyema occurred in 8 (26%).

The most common causative organisms for subdural empyema originating from paranasal sinuses are anaerobic and microaerophilic streptococci; in particular, *S. milleri* was identified as the causative agent in 17% of these cases.4 Other causative bacteria in this study included *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Hemophilus influenzae*, *Escherichia coli*, *Pseudomonas aeruginosa*, and *Klebsiella*...
A total of 17.6% of CSE cases had sterile cultures.24 CSE, occurring as a complication of head trauma or surgery, is mostly caused by S. aureus, anaerobes, coagulase-negative staphylococci, or Gram-negative organisms.3,4 However, SSE is mostly caused by S. aureus, particularly in iatrogenic cases.3 SSE can develop via spread from other parts of the body hematogenously or from contiguous infections or via iatrogenic causes such as lumbar puncture or injection of anesthetics or steroids into the spine. Comorbid conditions (such as diabetes mellitus, Crohn’s disease, rheumatoid arthritis, AIDS, or tumors) can compromise the immune system and predispose to abscess formation.3,4,11,49 SSE secondary to decubitus ulcers and halo orthosis placement have also been reported in the literature.4,47,50

In this illustrative case, S. mitis was identified as the causative organism. S. mitis, previously known as Streptococcus mitior, is a mesophilic, gamma-hemolytic species of Streptococcus viridans, commonly found in the oropharynx. Viridans streptococci are the most common cause of subacute bacterial endocarditis.11 S. mitis and other viridans streptococci are primarily known to cause subacute bacterial endocarditis or upper respiratory tract infections in persons with poor oral hygiene or patients undergoing dental procedures.8-11 It is also known to cause meningitis in patients with sinusitis.8,13,55 The underlying factors in patients acquiring S. mitis pneumonia are old age, diabetes, alcoholism, lung cancer, and hypothyroidism, whereas the same factors have been associated in patients with meningitis.17,56 To the best of our knowledge, this is the first reported case of S. mitis being the causative organism of subdural empyema presenting with retroclival and pan-spinal empyema together.

Clinical Presentation of Subdural Empyema
In most cases, CSE is located over the cerebral convexity. Infratentorial empyema is rare, constituting only 0.6% of
intracranial suppurations. In our review of the literature, we found no reports of intracranial empyemas located in the skull base or at the retroclival space. In our illustrative case, the location of the subdural empyema extended from the presemesencephalic area, along the prepontine and premedullary regions and through the foramen magnum into the anterior cervical epidural space. The presemesencephalic, prepontine, and premedullary areas are extremely rare locations for the development of an abscess.

Clinically, CSE often presents with rapid onset of meningeal irritation, altered mental status, focal neurologic signs, increased intracranial pressure, and seizures. It can also present with aural or nasal discharge, Pott’s puffy tumor, and subfalcine herniation syndrome. In contrast, SSE can present with fever, back or neck pain, bladder dysfunctions, motor deficits, and para- or tetraparesis. It can be differentiated from CSE if back or neck pain develops after an insult to the spine as a result of trauma, surgery, or infection. Although very rare, CSE and SSE are to be suspected if there are signs and symptoms of both brain and spinal injury.

Diagnosis of Subdural Empyema

Laboratory diagnosis of both CSE and SSE includes the nonspecific parameters white blood cell count, C-reactive protein level, and erythrocyte sedimentation rate, all of which are usually elevated. Because of the difficulties associated with accessing the location, the low diagnostic value of changes in CSF cell count, glucose levels, and protein levels, and the risk of herniation in CSE cases, lumbar puncture is not considered a standard diagnostic modality. CSE can be missed by a CT scan but is detectable by MRI, especially in cases of posterior fossa involvement. Therefore, because of its high sensitivity for detecting lesions in the disc space, subdural space, epidural space, and adjacent soft tissue, MRI with gadolinium contrast is considered the gold standard for diagnosis. However, CT is more sensitive for conditions involving the bone. We recommend performing MRI of the whole spine and brain in all patients with subdural empyema to rule out the involvement of other parts of the central nervous system.

Treatment Modalities for Subdural Empyema

Treatment of CSE and SSE includes surgical drainage followed by an appropriate antibiotic regimen. Cases characterized by nonfocal neurologic deficits, small empyema at locations other than the infratentorial region, or rapid responses to prompt IV antibiotic therapy can be managed conservatively with serial surveillance CT/MRI scans with contrast.

For SSE, laminectomy including one or more levels might be necessary for drainage, diagnosis, and quicker response to antibiotics. Dexamethasone is recommended as prophylaxis against an occlusive thrombophlebitis of the spinal draining veins and can only be started after an IV antibiotic regimen has been commenced. Residual pus collection should be monitored for antibiotic sensitivity and any residual pus resistant to treatment should be re-explored.

Concurrent SSE and SSE can be in continuity or in separate locations. For better treatment outcomes, both need to be drained and the patient needs to be treated with broad-spectrum IV antibiotics until an antibiotic therapy specific to the causative agent can be formulated. We propose a treatment plan for craniospinal empyema that is summarized in Figure 4.

For concurrent CSE and SSE, conservative treatment with antibiotics alone is not recommended. In such cases, a combined aggressive surgical and medicinal approach is recommended instead. Pompecci et al. reported on the use of initial antibiotic treatment for a few days with the placement of an external ventricular drain after cranial and pan-spinal involvement had been identified on MRI. The patient’s condition deteriorated after a few days, and an emergent suboccipital craniectomy was required.

In our illustrative case, emergency T2–L5 laminectomies were performed to release the pressure from the spinal cord and cauda equine. Besides the compressive nature of the empyema, it is well known that infection risks causing spinal venous thrombophlebitis resulting in a secondary infarct if left untreated. On preoperative MRI, the appearance suggested either epidural or subdural location. This can only be confirmed during surgery. If an epidural location is suspected, for long segment sections, small laminotomies can be performed for evacuation because the consistency of the epidural abscess is light. If no epidual infection is encountered and subdural infection is suspected, laminectomies and durotomies are needed as in this case. The thoracic spinal cord was extremely edematous secondary to infection, so the dural space was expanded with a dural patch.

Although patient did very well after this surgery, she could have developed neurologic deficit secondary to the edematous spinal cord or a spinal cord infarct secondary to an occlusive spinal venous thrombophlebitis that the infection could have caused. With the infection extending through the thoracolumbar spine, thoracic cord, thecal sac, and also a spinal osteomyelitis, long IV antibiotic treatment was required. Her IV antibiotic treatment was focused to cover the causative bacteria, after the cultures grew S. mitis, and she was managed by an infectious disease specialist for her antibiotic regimen. She underwent surveillance MRI with contrast during her hospital stay that showed good operative results and response to IV antibiotic therapy. She showed meningeval enhancement on her craniovertebral meninges, but given that the FLAIR was normal, there was no significant mass effect on the spinal cord or brainstem, she was clinically neuro intact, and her inflammatory parameters were normalizing on IV antibiotics, a new surgical intervention was not indicated. Her large iliopsoas abscess was not treated surgically. Surveillance MRI showed still a significant iliopsoas abscess that had slightly decreased in size. The orthopedic surgeon did not see any surgical indication, given her clinical and laboratory improvement. Her dental abscess was, however, drained.

Five weeks later, she returned with a recurring infection superior to the previous spinal location. Her cervical cord and brainstem were severely affected, with significant edema as seen on the FLAIR-MRI in Figure 3. This time she was in a toxic state. She was meningeal and appeared septic. Without surgical intervention, she was at high risk of mortality. Surprisingly, she was found to be on oral antibiotics, and she stated that her IV antibiotic regimen had been changed to oral 4 weeks after the previous surgery on discharge. The infectious disease specialist saw her in
the ED and immediately switched her oral antibiotic to IV antibiotic. On vital indication, emergent right retrosigmoid craniectomy and C1–C2 laminectomies were performed to evacuate the subdural empyema. Duroplasty also was performed to expand the cervical dura as the dura could not be closed on the severely edematous cervical cord.

The use of allogenic material is not preferred but in unique cases in which there is no option, the spinal cord needs to have dural coverage. Otherwise, the muscular layer risks to grow into the spinal cord pia with severe adherences, in access of potential risk of pseudomeningocele. Follow-up of this case illustrates that with correct antibiotic therapy, there was no recurrent infection at the locations allogenic material was used. Viridans streptococci have recently shown increased resistance to antibiotics such as penicillin, cephalosporin, erythromycin, and tetracycline, and S. mitis is comparatively more resistant to antibiotics than other viridans species.6,8,12

Because of the life-threatening nature of these infections and the resistant nature of S. mitis, we recommend using a minimum of an 8-week IV antibiotic course to preclude recurrence. Pompucci et al.5 reported that an 8-week IV antibiotic treatment course until discharge ensured full recovery and no recurrence on 1-year follow-up.

The patient initially had received 4 weeks of IV antibiotic treatment and was converted to oral antibiotics on the recommendation of the infectious disease specialist without notification to neurosurgery. She also was discharged without drainage of the iliopsoas abscess per orthopedic surgeon’s recommendation, despite neurosurgery recommendations of longer term IV antibiotic treatment and evacuation of the large iliopsoas abscess. One week after conversion to oral antibiotics, she returned with recurrence of infection and concurrent lesions involving the retroclival space and spinal cord. She was placed on the IV antibiotic regime again by infectious disease. It is reasonable to suggest that only 3 weeks of initial IV antibiotic therapy combined with the resistant nature of S. mitis could have contributed to the recurrence of the infection in our patient.

The duration of antibiotic treatment of osteomyelitis, or infected bone, varies with age: the younger the patient, the shorter the treatment needed.61,62 Typically, the recommended duration of antibiotic treatment for osteomyelitis is 2 weeks for children and 4–6 for adults.61–64 Earlier studies defining the durations of antibiotic therapy for most forms of osteomyelitis were performed during the 1970s and 1980s.65–67 A meta-analysis by Stengel et al.66 and a review by Lazzarini et al.67 evaluating effectively-documented trials of 6 or more patients published from 1968 through 2000, including 93 studies and nearly 2500 patients, demonstrated that most studies used 6 weeks of therapy. They also found that the few studies with treatment prolonged up to 6 months showed no clear improvement in outcomes. Presently, the recommendations for duration of curative antimicrobial therapy for most patients

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**Figure 4.** Treatment algorithm for cranial and spinal subdural empyemas. SE, subdural empyema; MRI, magnetic resonance imaging; IV, intravenous.
with osteomyelitis who have received “stage-appropriate” surgical interventions remain a minimum of 4–6 weeks, whereas patients with more extensive infections and limited surgery can require longer treatment.24,65 In clinical practice, a “goal-directed” approach is often adopted, with the use of clinical assessment and normalization of inflammatory markers (C-reactive protein and/or erythrocyte sedimentation rates) to define the duration of therapy. There is no literature specifically addressing cranial osteomyelitis. Hence, on the basis of our own experience, we recommend a minimum of 8 weeks of antibiotic treatment until the infection is completely resolved as seen on MRI and with normal inflammatory parameters. It is important to emphasize that some meningeal enhancement continues to be visible on contrasted MRI long after treated infection and is not to be mistaken with active infection.

Role of Corticosteroid Therapy

The use of corticosteroid therapy in central nervous system lesions in particular infectious lesions is highly debated, and there are various studies supporting pros and cons of adjunctive corticosteroid treatment. In addition, there is lack of strong evidence supporting corticosteroid use for cranial and spinal abscesses with brain/spinal cord edema, without any controlled, randomized clinical trials reported in the literature. In an overview of brain abscesses, Muzumdar et al.68 recommended to avoid corticosteroid therapy unless the patient demonstrates signs of signs of meningitis or disproportionate cytotoxic edema posing a life-threatening situation. A recent Cochrane review on the effect of corticosteroids on mortality with bacterial meningitis concluded that dexamethasone did not significantly reduce mortality, although steroids significantly reduced mortality in cases of meningitis caused by S. pneumoniae as compared with cases of meningitis caused by other bacteria.69 Obviously due to the rarity of spinal and especially cranial empyema, it is difficult to perform prospective randomized trials with high power. Given the severity of the reported case and the serious medical history, it is reasonable to use steroids concurrently with IV antibiotic therapy.

Recommendations Based on Our Experience and Literature Review

CSE and SSE or concurrent CSE and SSE is a neurosurgical emergency that requires immediate and timely intervention. Delay in diagnosis can pose significant morbidity as it did in our case and high risk of mortality. This illustrated case had several delays, including the delay in diagnosis by her primary care physician, and the delay in correct MRI reading. To avoid neurologic damage, the decompression of empyema should be considered as soon as the diagnostic workup is done. The initial antibiotic regimen needs to include broad-spectrum covering gram-positive and gram-negative bacteria. In cases of concurrent cranial and spinal empyema, we recommend simultaneous aggressive surgical and medical management to reduce the infectious burden. Steroid therapy can be used concurrent with IV antibiotic therapy. Indications for surgery are for diagnosis, debulking of the infective burden to enhance antibiotic therapy and to decrease local mass effect. Hence, indications for surgery vary from case to case. Moreover, when dealing with such cases, it is important to consider that without surgery, the risk of mortality and morbidity is very high.

These empyemas are adjacent to bony structures such as the vertebrae and cranial bone and hence are osteomyelitis and not only soft-tissue infections. Therefore, we highly recommend long-term IV antibiotic therapy of 6–8 weeks to prevent its recurrence after surgery. To monitor the effectiveness of therapy, the surveillance should be multifactorial, including patient’s clinical status, serial biochemical markers (C-reactive protein, procalcitonin, white blood cell count with differential) and follow-up MRI for radiologic status. Adjunctive corticosteroid therapy in cases of cranial or spinal empyema lacks evidence. It may benefit in reducing cerebral edema surrounding the abscess. A word of caution is that steroid therapy can decrease the enhancement of the abscess wall on CT. Therefore, because of MRI’s superiority in the diagnosis and surveillance of infections, MRI with contrast is preferred to CT with contrast for surveillance if steroid have been used. Important factors to be considered in this case are patient’s preoperative dental health, the delay of the primary care physician in diagnosis, the radiologist’s delay in correctly interpreting the initial MRI results before neurosurgery was contacted, the decision not to drain the iliospsoas abscess and the shortening of IV antibiotic treatment after the first surgery.

Conclusions

Concurrent CSE and SSEs are rare, and only 3 cases of concurrent SSE and CSE have previously been reported. CSE in the retroclival region has not been reported until now. S. mitis has never been reported as causative agent. Treatment consists of emergent decompression of the brain and spinal cord, and IV antibiotics. Steroids along with IV antibiotics can be considered. To preclude recurrence, at least 8 weeks of IV antibiotics treatment is recommended, with close surveillance until the infection is completely resolved.

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