Subdural empyema and other suppurative complications of paranasal sinusitis

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Suppurative intracranial infection, including meningitis, intracranial abscess, subdural empyema, epidural abscess, cavernous sinus thrombosis, and thrombosis of other dural sinuses, are uncommon sequelae of paranasal sinusitis. A high index of suspicion is necessary to identify these serious complications. We present a patient with subdural empyema in whom the diagnosis was delayed, followed by a discussion of suppurative complications of sinusitis. The case shows the rapid progression of subdural empyema, which represents a true neurosurgical emergency requiring prompt diagnosis and management.

Case presentation

A 63-year-old white man presented to the emergency department because of lethargy. The patient was well until 4 days before admission when he complained of cough, fever, and feeling poorly. Over the next few days, he became progressively lethargic. In the emergency department, the patient complained of neck pain but was unable to relate other symptoms.

The patient's past medical history was notable for diabetes mellitus, hypertension, hyperlipidaemia, and daily alcohol use. He had a remote history of post-traumatic splenectomy. 8 years ago, he had recurrent episodes of pancreatitis eventually requiring drainage of a pseudocyst. 5 years before admission, he was treated for a group F streptococcal liver abscess. Medications at the time of admission included quinapril hydrochloride, hydrochlorothiazide, and gemfibrozil.

On examination, the patient was lethargic, but could be aroused with verbal stimulation. His temperature was 37·2°C, heart rate 66 beats per min, blood pressure 180/64 mm Hg, and respiratory rate 18 breaths per min. His pupils were equal, round, and reactive. Over the next few days, he became progressively lethargic. In the emergency department, the patient complained of neck pain but was unable to relate other symptoms.

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On examination, the patient was lethargic, but could be aroused with verbal stimulation. His temperature was 37·2°C, heart rate 66 beats per min, blood pressure 180/64 mm Hg, and respiratory rate 18 breaths per min. His pupils were equal, round, and reactive, and his neck was supple. The patient was oriented only to person and place; cranial nerves were intact. His left arm was flaccid, but the right arm and both legs showed normal strength. The remainder of his physical examination was normal.

The white blood cell count was 22 200 per μL with 26% band forms, 48% segmented neutrophils, and 10% lymphocytes; haemoglobin was 14·8 g/dL, and platelets 453 000 per μL. Chemistries were remarkable for a sodium level of 129 MEq/L. Pre and post-contrast cranial computed tomography (CT) (figure, A–C) showed extensive pansinusitis with a 1 cm right-sided subdural fluid collection, associated mass effect, and overlying soft-

Figure: Serial computed tomography scans showing progressive subdural collection

(A, B, C) Initial contrast CT scan showing subdural fluid collection and near complete opacification of bilateral ethmoid and maxillary sinuses (indicated by *). There is a heterogeneous pattern of mixed hyperdense and hypodense signal overlying the right cerebral convexity. (D, E, F) Non-contrast cranial CT 6 hours later: subdural empyema now easily visible, with midline shift and obliteration of the contralateral basilar cisterns (D), consistent with subuncal herniation.
meningitis. Because of the CT findings, a lumbar puncture was not done. 6 h after admission, the patient deteriorated acutely, developed a dilated right pupil, and required endotracheal intubation. Repeat cranial CT without contrast showed progression of the right subdural fluid collection with increased right-to-left midline shift of 1.8 cm and subuncal herniation (figure, D–F). At emergent craniotomy, a large subdural empyema was evacuated. The patient also underwent bilateral maxillary antrostomy, bilateral anterior ethmoidectomy, and right sphenoidectomy. Gram stain of the empyema showed many polymorphonuclear white blood cells, many Gram-positive cocci, and rare Gram-negative rods. Culture of the empyema grew alpha-haemolytic Streptococcus spp. Sinus cultures from operative specimens also grew alpha-haemolytic Streptococcus spp and Fusobacterium spp. The antibiotic regimen was changed to meropenem.

By hospital day 2, the patient’s sodium level had returned to normal. He was febrile for several days post evacuation of the empyema then became afebrile. Following surgical evacuation, his mental status waned and waned, but slowly improved, and he was extubated on hospital day 10. The patient received 3 weeks of parenteral antimicrobials and required a percutaneous gastrostomy tube for feeding because of impaired swallowing. At hospital discharge he could answer simple questions and sit up with assistance but was unable to walk. 1 year later, he was much improved and had only mild residual left arm weakness. He developed a seizure disorder and remained on anti-epileptic medications.

**Review and discussion**

The case shows the rapid progression of subdural empyema and the need for prompt diagnosis and aggressive management of this disease. Although the presence of sinusitis, fever, altered level of consciousness, focal neurological findings, and compatible intracranial imaging suggested this diagnosis, recognition was delayed. This delay was possibly caused by several factors including a low index of suspicion, occurrence in an older man (which is somewhat atypical), and the clinician’s reliance on the interpretation of CT imaging done by a radiologist who was unaware of the clinical context. The rapid progression to herniation emphasises the need for emergent neurosurgical intervention.

**Intracranial complications of sinusitis**

Bacterial sinusitis is common and resolves without sequelae in the majority of cases. Suppurative complications can occur, although they are much less common since the advent of antibacterial agents. Additionally, the use of CT and other cranial imaging has allowed for earlier diagnosis and treatment of complicated sinus disease. Because the intracranial suppurative sequelae of sinusitis can be neurologically devastating, clinicians should retain a high index of suspicion for this uncommon but serious disease.

The suppurative complications of sinusitis can be divided into orbital and intracranial infections. Orbital complications include orbital cellulitis, subperiosteal abscess (Pott’s puffy tumour), orbital abscess, and cavernous sinus thrombosis. Sinusitis, particularly of the ethmoid air cells, is the most common cause of these severe orbital infections. Orbital infections present with periorbital oedema, chemosis, visual loss, restricted eye movement, and proptosis. The latter three findings help to differentiate orbital infections from preseptal cellulitis, which is confined to structures anterior to the orbit. Diplopia results from involvement of the cranial nerves, most often the sixth cranial nerve. Orbital complications of sinusitis and intracranial infection coexist in up to 45% of cases. Therefore, patients exhibiting orbital signs should undergo cranial imaging.

The intracranial complications of paranasal sinusitis include subdural empyema, epidural abscess, intracerebral (intraparenchymal) abscess, meningitis, cavernous sinus thrombosis, and thrombosis of other dural sinuses. It is not uncommon for a patient to exhibit more than one focus of infection—intracranial or orbital, or both. Most cases of sinusitis are uncomplicated and occur in the outpatient setting. In view of the common occurrence of sinusitis and the rarity of complications, the incidence is presumed to be low, although precise estimates are unavailable since many cases of sinusitis do not present for medical treatment. The literature on intracranial complications of sinusitis consists mainly of case reports and single-institution case series, with the exception of one large series involving two institutions. The small number of cases acquired at one institution over many years is a testament to their rarity. In patients hospitalised with sinusitis, the reported rate of intracranial complications varies from 3.7% to 47.6%.

By selecting for severe sinus disease, these series clearly overestimate the incidence of intracranial complications. Overall, sinus disease is the presumed underlying cause of about 10% of intracranial suppuration. Subdural empyema, however, is much more strongly associated with underlying sinusitis and is also the most common sinusitis-associated intracranial infection. Most case series of complicated sinusitis are in the surgical literature. Since meningitis from sinusitis rarely requires surgery, this complication might be under-represented in these case series.

The pathogenesis of intracranial complications of sinusitis includes two major mechanisms: direct extension and, more commonly, retrograde thrombophlebitis via the valveless diploe veins. The close anatomical proximity of the sinuses to the intracranial
cavity allows for direct extension of infection to bone (ie, osteomyelitis) and subsequent erosion into the epidural space. On occasion, there is further penetration of purulence through the dura to the subdural space. Retrograde thrombophlebitis is facilitated by the shared venous drainage of the sinuses and the intracranial structures. Because these veins are valveless, thrombophlebitis that begins in the veins draining the sinuses can pass retrograde into the cavernous sinus and other dural venous sinuses. If infection reaches the subdural space, it spreads easily over the convexities of the brain owing to a lack of septations. The implantation of the arachnoid granules represents the only barrier in this space. Contralateral spread under the falx cerebri can occur. Thrombophlebitis associated with subdural empyema can lead to venous stasis with thrombosis, infarction, and subsequent cerebral inflammation. The resultant oedema is a major contributor to the neurological deterioration seen with undrained subdural pus.12 The pathogenesis of subdural empyema in infants is different, with most cases resulting from infection of subdural effusions that develop from an initial meningitis.13

The clinical presentation of complicated sinusitis depends on the site of bacterial seeding (table). The presentation can be acute and fulminant with bacterial meningitis, cavernous sinus thrombosis, and subdural empyema. Brain abscesses and epidural abscesses typically have a more indolent presentation, although rupture of a brain abscess into the ventricular system can precipitate an acute neurological deterioration. The anatomical considerations mentioned above are important determinants of the clinical course. For example, purulence in the epidural space is constrained by the dura, which is adherent to the calvarium. Therefore, clinical presentation of epidural abscess is insidious with fever and headache evolving over weeks. By contrast, seeding of the subdural space leads to rapid spread of purulence because of lack of anatomical constraints.

Most complicated sinusitis occurs in young men in the second or third decade of life, often with no underlying medical problems.5,9–11 This predisposition for young men has been explained by the vascularity of the diploic system in this age group, which is at its maximum,10 as well as the continued development of the frontal sinus that occurs during that time. The male to female ratio is reported to be from 1:3–1 to 4:5:1. The most common symptoms in patients presenting with intracranial complications are fever and headache. Symptoms or history of sinusitis may or may not be present.2,9,10,14 Altered mental status and focal neurological deficits are frequent. Seizures occur in 8–20% of cases.3,10,15 Other symptoms include meningismus, decreased visual acuity or other ocular complaints including photophobia. “Silent” (asymptomatic) complications have also been described,7 in which intracranial purulence is detected incidentally when imaging is done to evaluate the sinuses. Most intracranial complications result from frontal, ethmoid, or sphenoid sinusitis; sinusitis is often bilateral.10,17 Sphenoid sinusitis is strongly associated with cavernous sinus thrombosis,18 which manifests as proptosis, periorbital oedema, and chemosis caused by obstruction of the superior ophthalmic vein. Meningitis arises most often from ethmoid or sphenoid sinusitis.19 Mortality from intracranial sinusitis-associated infection in the pre-CT era was as high as 66%,13 but has decreased to 2–7%.2,10,15 Many patients are left with residual neurological deficits, including seizure disorders, particularly if diagnosis or intervention is delayed.10 Therefore, early diagnosis and treatment remain a priority.

Subdural empyema

Much of the clinical presentation and epidemiology of subdural empyema parallels that of intracranial complications in general. Most cases occur in the second decade of life5,7,22,23 in patients who are otherwise healthy. As with other intracranial complications, males have a marked predisposition, with a male-to-female ratio of 3:1,20–22 and as high as 8:1 for sinus-associated cases.1,24

### Table: Intracranial complications of bacterial sinusitis

<table>
<thead>
<tr>
<th>Site of complication</th>
<th>Usual involved sinuses</th>
<th>Clinical presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meningitis</td>
<td>Sphenoid, ethmoid</td>
<td>Acute and rapidly progressive, fever, meningismus, headache with or without signs of cerebral dysfunction</td>
</tr>
<tr>
<td>Subdural empyema</td>
<td>Frontal</td>
<td>Rapidly progressive; meningeal signs plus increased intracranial pressure; can lead to focal neurological defects and coma in 24 to 48 h if untreated; seizures</td>
</tr>
<tr>
<td>Brain abscess</td>
<td>Frontal, ethmoid</td>
<td>Indolent with symptoms caused by increased intracranial pressure; classic triad of fever, headache, focal neurological findings present in only a minority; seizures</td>
</tr>
<tr>
<td>Cavernous sinus thrombosis</td>
<td>Sphenoid, ethmoid, frontal</td>
<td>Subacute when sinusitis is the underlying cause; periorbital swelling, fever, headache, diplopia, proptosis; involvement of contralateral eye is a late finding</td>
</tr>
<tr>
<td>Epidural abscess</td>
<td>Frontal</td>
<td>Slowly growing, indolent onset; may be asymptomatic except for headache; signs of increased intracranial pressure eventually develop</td>
</tr>
</tbody>
</table>

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mood without focal neurological symptoms.2,9,24 The most
presentation could be subtle changes in personality or
empyema is located in the frontal lobe, the clinical
white-matter interface is displaced inwardly.25 Mass eff ect
is generally caused by oedema and ischaemia rather than
stiff ness. More ominous fi ndings can evolve rapidly and
common symptoms are headache, fever, and neck
progression is a hallmark of subdural empyema and
neurological defi cits or cranial nerve palsies, hemiparesis,
include depressed level of consciousness, focal
diagnosis. It is also the test of choice for visualisation of
of more widespread availability and the need for a rapid
several patterns, most often low DWI signal, or mixed signals.
Benign subdural eff usions have a low signal on DWI,
similar to cerebrospinal fi uid (CSF).29,30 Most empyemas
are located within the frontal lobe24,25 and can be
Although rare, infratentorial empyemas have also been described as complications of parasanal
sinusitis.12,33

Complete blood count almost universally reveals
leucocytosis, with increased polymorphonuclear cells and
band forms.5,7,13,17,21 Erythrocyte sedimentation rate is often
less than 100 mm/h.7,21,22 Lumbar puncture in patients
with subdural empyema is contraindicated, particularly if
mass eff ect is present on CT or if the patient has
papilloedema.3 Neurological deterioration and trans-
tentorial herniation after lumbar puncture are well
described, and have led to death.5,7,13,21 However, in patients
who have a lumbar puncture done, the CSF shows a
parameningeal formula, with elevated protein, normal
glucose, and pleocytosis with polymorphonuclear
predominance.5,7,13,21–23 The CSF Gram stain usually does
not show organisms and CSF cultures are negative more
than 85% of the time.5,7,13,17,21,22 The CSF may be normal.20
Infections are often polymicrobial.3,21 Anaerobic Gram-
positive cocci, Streptococcus spp, Staphylococcus spp, and
anaerobic Gram-negative bacilli are the most commonly
isolated organisms from empyema material5,21,22,24,25,26
(panel 2). No single organism predominates, although members of the Streptococcus milleri group (including

mass eff ect from the abscess.25 The oedema can cause
effacement of the basilar cisterns and flattening of the
cortical sulci.25 The sinuses might appear opacifi ed, with
air-fiuid levels and bony erosion evident in some cases.2
MRI appearance is similar; T1-weighted images show
mass eff ect and hypointense areas of purulence, which
are hyperintense on T2-weighted images.19 The abscess
usually has a hyperintense rim on non-contrast
T1-weighted images. Diffusion-weighted images (DWI)
on MRI may be helpful in differentiating subdural and
epidural empyemas.29,30 Subdural empyemas have high
signal on DWI, whereas epidural empyemas show several
patterns, most often low DWI signal, or mixed signals.

Radiographic imaging should be done in all patients in
whom subdural empyema is suspected. Although
magnetic resonance imaging (MRI) is more sensitive in
showing parenchymal abnormalities such as abscess,19
cranial CT is often the fi rst neuroimaging done because of
more widespread availability and the need for a rapid
diagnosis. It is also the test of choice for visualisation of the
parameningeal sinuses and associated bony abnormalities.
In early cases of subdural empyema, CT might not show a
fiuid collection,5,7,13,17,21,22 so consideration should be given
to repeated CT imaging or MRI as the clinical scenario
dictates. When visualised on CT, the empyema appears
as a thin, hypodense subdural lesion, with linear
enhancement of the medial surface.5,26 The grey-matter/
white-matter interface is displaced inwardly.25 Mass eff ect
is generally caused by oedema and ischaemia rather than

Panel 1: Common signs and symptoms in patients with subdural empyema

- Fever
- Headache
- Altered mental status
- Hemiparesis
- Nausea or vomiting
- Seizures
- Meningismus
- Periorbital oedema
- Papilloedema
- Pott’s fippy tumour
- Other: dilated pupil, ocular palsies, cranial nerve palsies, dysphasia or aphasia, eyelid abscess, homonymous
hemianopsia, cerebellar signs, hydrocephalus

occur when a subdural eff usion related to meningitis
becomes infected. When sinusitis is the cause, the frontal
sinus is most often implicated,6,21 though pansinusitis and
involvement of the posterior ethmoid cells are also
common. Concomitant intracerebral abscess occurs in 6% to
22% of cases6,20,22 and epidural abscess in 9% to 17%.22

The presenting symptoms of subdural empyema are
reflective of increased intracranial pressure, meningeal
irritation, and cerebritis (panel 1).5–7,13,17,20–22,24,26,28 If the
empyema is located in the frontal lobe, the clinical
presentation could be subtle changes in personality or
without focal neurological symptoms.2,9,24 The most
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Panel 2: Associated pathogens in subdural empyema

**More common**
- Streptococcus milleri
- Other streptococci and enterococci
- Aerobic Gram-negative bacilli*
- No growth

**Less common**
- Streptococcus pneumoniae
- Staphylococcus aureus, coagulase-negative staphylococci
- Anaerobic Gram-positive cocci†
- Anaerobic Gram-negative bacilli‡

Streptococcus anginosus, Streptococcus intermedius, and Streptococcus constellatus) are over-represented.2,9,22,24,34 Anaerobes are more likely to be present if the empyema is associated with sinusitis compared with other causes of subdural empyema. The presence of anaerobes and a conspicuous absence of pathogens associated with acute sinusitis (Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis) suggest that most subdural empyemas arise from chronic sinusitis. Operative cultures do not yield growth in 7–53% of cases, 4,20–22 presumably because of previous administration of antibiotics or failure to use proper anaerobic culture technique.

Antimicrobial therapy should be directed against the aforementioned organisms and include broad-spectrum activity against aerobic and anaerobic cocci and bacilli. Recommended empiric therapy is a third-generation cephalosporin plus metronidazole, which offers broad coverage and good CSF and abscess penetration. Once antimicrobial identification and susceptibility results are available, antimicrobial therapy can be tailored to the pathogens recovered from cultures. Antimicrobial therapy in the case discussed may have been unnecessarily broad. It should be recognised, however, that cultures might be negative and continuation of empiric antimicrobial therapy, including anaerobic coverage, is reasonable. The appropriate duration of therapy has not been studied in randomised, controlled trials, but available series suggest at least 2 weeks of intravenous therapy should be given; parenteral or oral therapy is frequently continued for up to a total of 6 weeks of antimicrobials.6,20–22 If adjacent osteomyelitis is present, prolonged parenteral therapy should be considered (eg, minimum of 6–8 weeks). Adjunctive care includes prophylactic anticonvulsants,3,15 and management of oedema and intracranial hypertension with measures such as corticosteroids,1,15 ventriculostomy, and intravenous mannitol.29

Surgical management of subdural empyema is an integral part of therapy and should be done without delay. Isolated medical management has been successful in only a few case reports.6,32 The goals of surgical intervention are decompression of the brain and complete evacuation of purulence. There is controversy in the neurosurgical literature regarding the preferred surgical intervention—burr hole versus craniotomy.4,20,33 Some patients treated with a burr hole require additional surgery or conversion to craniotomy.12,13,20,22 Currently, both procedures are acceptable, and the determination of which procedure to do should be made in consultation with the neurosurgeon. Multiple burr holes with surgical irrigation and decompressive craniectomy are other surgical options.22 In addition to drainage of intracranial purulence, definitive management of the infected sinuses should be done, preferably at the same time as empyema drainage. The choice of surgical approach depends on the involved sinus and can include maxillary irrigation, external frontoethmoidectomy, sphenoid sinusotomy, antral washout, and frontal trephine. The development of endoscopic sinus surgery had made this the most popular otolaryngologic intervention in subdural empyema in recent years.35

Before the availability of antibiotics, subdural empyemas were almost always fatal, even with surgical drainage. Antimicrobials decreased the mortality rate to 15–6–41%.2,7,11,13,21 Although the impact of CT diagnosis on mortality has not been studied directly, mortality rates are lower (6–15%) since the advent of CT imaging.6,7,19,22 Survivors may exhibit substantial morbidity, including seizures in 12–37.5% of cases,17,20,21 and residual neurologic deficits in nearly half of cases.6,47

Conclusions
Although often considered a benign disease, paranasal sinusitis can on rare occasions lead to serious, potentially life-threatening complications. The astute clinician should consider such complications in any patient presenting with fever, headache, and neurological deficits, regardless of a history of sinusitis. Early diagnosis with aggressive medical and surgical management can lead to improved outcome, most notably for subdural empyemas that evolve rapidly and are true neurosurgical emergencies.

Conflicts of interest
We declare that we have no conflicts of interest.

References
5 Kaufman DM, Miller MH, Steigbigel NH. Subdural empyema. A Medline search of the literature from 1966 to the present was done to identify relevant English language articles. Several search strategies were used. The search term “sinusitis” was combined with (1) “subdural empyema”, (2) “brain abscess or epidural or intracranial or intracerebral”, (3) “cavernous sinus thrombosis”, (4) “meningitis”, and (5) “intracranial complications”. Retrieved references were manually reviewed for relevance, favouring case series and larger studies over case reports. Studies of exclusively pediatric cases were not included. The references of selected papers were also reviewed.

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