Article published online: 2022-09-14

Case Report

# Rapidly Developing Subdural Empyema in an Adult with Sinusitis: A Neurosurgical Threat Alert

**Abstract**

Subdural empyema (SDE) is collection of pus in the potential space between the dura and arachnoid layers of the meninges. Leading causes of SDE are sinonasal and otomastoid infections. Commonly affecting patients in the second and third decades, SDE could have a fulminant course with immediate complications and delayed morbidities including hydrocephalus, focal deficits, and epilepsy.

**Keywords:** *Multidetector computed tomography, sinusitis, subdural empyema*

**Kabilan Chokkappan, Rahul Lohan**

*Department of Diagnostic Radiology, Khoo Teck Puat Hospital, Singapore*

## Introduction

Sinonasal inflammatory disorders usually have an innocuous clinical course; however, rarely they can affect the orbits, underlying bones, adjacent veins, and the intracranial structures. Intracranial extension is the most dangerous complication of these disorders. The most common form of intracranial involvement is subdural empyema (SDE) with bacterial sinusitis being its leading cause. SDE makes for 15%–22% of intracranial infections overall. Males are affected more frequently than females in most of the reported series. Unlike in adults, it occurs commonly secondary to bacterial meningitis in young children. Although SDE is universally fatal if untreated,[1] with current improvements in the management, the mortality is reported to be between 6% and 35%. More than 50% are inflicted with long-term morbidities. Morbidity rate was close to 70% for those who were operated after 3 days, compared to 10% in those who got operated before 72 h, emphasizing the need for early recognition and prompt surgical management.

We present a case of rhinosinusitis that within a few hours progressed into large SDE with severe mass effect and midline shift. The case highlights the importance of having a high index of clinical suspicion and low threshold for imaging in cases of sinusitis that present with subtle neurological signs and symptoms. We emphasize on the role of imaging in optimal management of these cases. The imaging helps in identification of catastrophic neurological complications early in the course of disease and thus guides the treatment approach. Although SDE *per se* is not very uncommon, the presented case is unique as there was sudden development of the SDE with corresponding rapid neurological deterioration.

## Case Report

A 45-year-old man presented to Accident and Emergency (A and E) Department with 2-day history of fever, chills, productive cough, runny nose, and gradual onset headache. He had a temperature of 39.8°C, heart rate of 115 beats/min (bpm), blood pressure of 130/70 mmHg, and respiratory rate of 18/min. The throat was injected. Examination of his ears and oral cavity was unremarkable. His total white blood cell (WBC) count was slightly raised (15.45 × $10^9$/L). The chest radiograph was normal. After receiving symptomatic treatment for presumed upper respiratory tract infection, he was discharged from the A and E. After 6 h, the patient was brought to the A and E following eight episodes of witnessed seizures. On examination, he was febrile and in status epilepticus with heart rate of 173 bpm and blood pressure measuring 94/57 mmHg. His cardiac auscultation was normal. On neurological examination, the patient was confused, his consciousness was impaired, reflexes were preserved, pupils were equal and reactive, and no papilledema was present. His WBC count increased to 31 × $10^9$/L with predominant neutrophils (84%). C-reactive protein (CRP) levels were

**How to cite this article:** Chokkappan K, Lohan R. Rapidly developing subdural empyema in an adult with sinusitis: A neurosurgical threat alert. Asian J Neurosurg 2018;13:458-61.



***Address for correspondence:***
*Dr. Kabilan Chokkappan, Department of Diagnostic Radiology, Khoo Teck Puat Hospital, Tower B, Level 2, 90 Yishun Central, Singapore 768828, Singapore.*
*E-mail: dr.kabilan@yahoo.com*

**Access this article online**

**Website:** www.asianjns.org

**DOI:** 10.4103/ajns.AJNS_97_16

**Quick Response Code:**

grossly elevated (245.6 ml/L). He was intubated. An urgent noncontrast computed tomography (CT) of the brain [Figure 1] revealed subtle fullness in the right frontal region with mild sulcal effacement. The paranasal sinuses showed a diffuse mucosal thickening.

In view of inconclusive CT findings and sudden clinical deterioration, a lumbar puncture was performed to look for signs of intracranial infection. The cerebrospinal fluid (CSF) analysis revealed an infective picture (leukocytosis and increased protein levels). Meanwhile, his condition further deteriorated and Glasgow Coma Scale (GCS) dropped from 10 to 7. In the view of sudden neurological worsening, a contrast-enhanced magnetic resonance imaging (MRI) of the brain [Figure 2] was performed which showed a large peripherally enhancing right frontotemporoparietal subdural collection with smaller collections in the left frontal region. There was severe mass effect in the right cerebral hemisphere with midline shift. Fluid-filled paranasal sinuses showed mucosal hyperenhancement. A small bony defect in the posterior wall of the left frontal sinus was seen with adjacent meningeal enhancement and right frontal parenchymal edema. A diagnosis of sinogenic meningoencephalitis with multiple SDEs was inferred from MRI. Retrospective inspection of the initial CT images of the brain confirmed the bone defect in the corresponding location of the left frontal sinus [Figure 3]. Antibiotic treatment was started with intravenous administration of vancomycin and ceftriaxone.

He underwent right decompressive craniectomy and evacuation of SDE. Frank pus was drained which contained *Streptococcus intermedius* sensitive to multiple antibiotics. In the same sitting, he underwent functional endoscopic sinus surgery (FESS) to address the sinusitis. The pus from the infected sinus had the same organism and antibiotic sensitivity as the empyema. Postoperatively, he was treated with intravenous antibiotics (intravenous meropenem and vancomycin for 2 weeks) and stayed in the intensive care unit for 5 days. His condition improved gradually based on the neurological examination (GCS of 10) with vital signs becoming normal, and he could be extubated on his fourth postoperative day. The follow-up CT [Figure 4] showed improvement of mass effect and cerebral edema. After 20 days of hospital stay, he was discharged with good neurological status. The inflammatory markers demonstrated a gradual decline and the CRP was

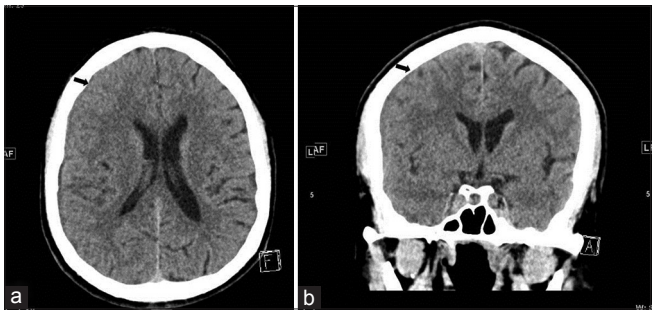


**Figure 1: Axial (a) and coronal (b) sections of noncontrast computed tomography brain show mild effacement of right frontal sulci (black arrows), with no discernible mass effect or midline shift**

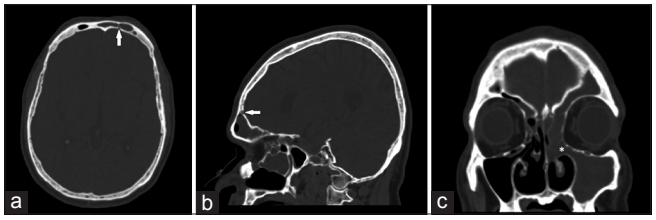


**Figure 3: (a and b) Bone window computed tomography images of the head showing the focal bone defect (arrow in image A and B) in the posterior wall of the left frontal sinus. (c) Coronal image showing the extent of sinusitis. Left osteomeatal unit was completely obstructed (arrow) by the mucosal thickening**

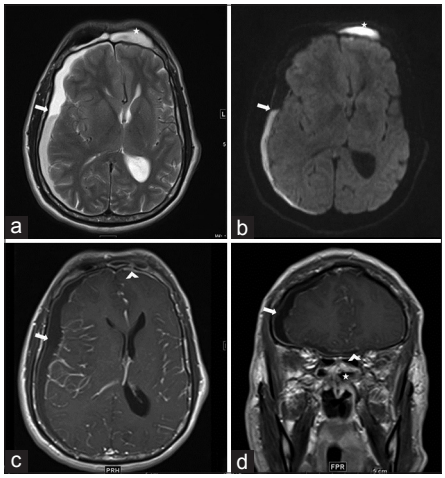


**Figure 2: Contrast-enhanced magnetic resonance imaging taken 8 h after the initial computed tomography. (a) Axial T2-weighted image shows the right convexity subdural collection with layering of fluid (white arrow). Marked mass effect with midline shift to the left is seen. The left frontal sinus (white asterisk) is fluid filled. (b) Diffusion-weighted imaging shows restricted diffusion of the subdural collection (white arrow) and the sinusitis (white asterisk). (c) Contrast-enhanced axial T1-weighted image shows thin peripheral enhancement of the subdural collection. There is increased enhancement of the pachymeninges in the left frontal region that continues with enhancing mucosal lining of the left frontal sinus through a bony defect (arrowhead) in the posterior wall of the sinus. (d) Coronal contrast-enhanced T1-weighted image shows a large right convexity and a small left basal subdural empyemas connected to each other at the basal subfalcine region**

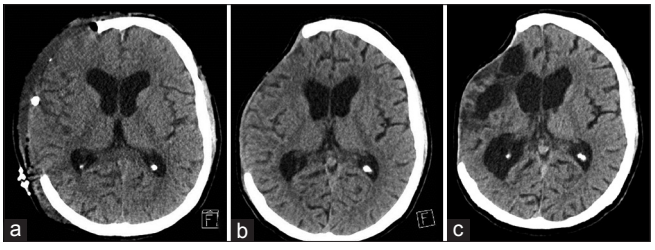


**Figure 4: (a) Immediate postcraniectomy computed tomography scan axial section showing resolution of mass effect and the midline shift as the subdural empyema has been evacuated. (b) Follow-up computed tomography after 9 months showing stable craniectomy changes and complete resolution of mass effect. (c) Two-year follow-up study showing more pronounced encephalomalacic changes and parenchymal loss of the right cerebral hemisphere**

51.4 mg/L at the time of discharge. There was no change in his personality or cognitive functions. No motor or sensory deficits were observed upon discharge.

Unfortunately, he developed scar epilepsy and his follow-up CTs demonstrated a significant parenchymal loss and gliosis [Figure 4]. This could be secondary to ischemia and cerebral infarctions that may develop secondary to increased intracranial pressure and thrombosis of cortical veins as in many of the intracranial infections. The patient is currently under neurosurgical follow-up with his epilepsy being managed by oral sodium valproate and levetiracetam.

## Discussion

The rarely encountered complications of bacterial sinusitis are subdivided into local manifestations (such as mucocele, pyocele, and recurrence) and extension of sepsis to the adjacent orbital or intracranial structures. Orbital complications include orbital cellulitis, subperiosteal abscess, intraorbital abscess, and osteomyelitis. The intracranial complications comprise meningitis, encephalitis, epidural or SDE, cerebral abscess, and cavernous or other dural venous sinus thrombosis.[2] The true incidence of intracranial sepsis in sinusitis is unknown as majority of the uncomplicated sinusitis does not get medical attention.[3] By virtue of their anatomical relationship, different forms of paranasal sinusitis are associated with specific intracranial complications. Osteitis and subperiosteal abscess are generally the complications of frontal sinusitis; meningitis and cavernous sinus thrombosis are associated with sphenoethmoid sinusitis and orbital inflammation with ethmoid sinusitis. Acute progressive headache, orbital manifestations, and failure to respond to usual medical treatment of sinusitis are indicative of possible intracranial spread of infection and demand prompt radiological assessment.[2,4]

SDE represents collection of pus between the dura and arachnoid layers of the meninges.[4] The peak incidence of SDE is second and third decades with a male predilection. Sinonasal and otomastoid infections are the leading causes, followed by meningitis, previous head injury, neurosurgeries, and infection of subdural hematoma.[3] From the sinonasal region, the infection can spread through direct or indirect routes. The direct spread occurs following the erosion of sinus wall or through preformed pathways such as congenital or acquired skull defects and the natural skull foramina.[5] The more commonly implicated mechanism, however, is the indirect spread through retrograde septic thrombophlebitis of valveless emissary veins. The oral commensals, *Streptococcus milleri* group, are the most commonly encountered causative organisms.

The clinical manifestations of the SDE reflect the underlying pathophysiological processes such as meningeal irritation, cerebral edema, increased intracranial pressure, and mass effect. The usual symptoms are headache, fever, vomiting, lethargy, neck stiffness, seizures, and focal neurology. Unlike other sinogenic intracranial complications such as epidural and intracranial abscesses, the SDE has fulminant clinical course due to rapid spread of purulence in a space that lacks anatomical constraints.[3] Long-term sequelae of SDE include hydrocephalus, residual hemiparesis, and epilepsy. CT is the imaging modality of choice when immediate surgical management is contemplated.[5] CT not only demonstrates the size and mass effect of the empyema but also evaluates the status of the paranasal sinuses. SDE appears as a hypodense subdural collection with peripheral rim enhancement on CT. Initial CT may be negative or nonspecific as in our case. A repeat contrast CT or MRI is recommended in cases of persisting clinical suspicion. MRI is the gold standard in evaluating intracranial infections. The SDE is often hyperintense on diffusion-weighted imaging (DWI). This feature differentiates it forms epidural empyema and subdural effusion which follow CSF signal on DWI.[6] MRI is superior to CT in evaluating the orbital inflammation and status of the dural venous sinuses.

Medical management of SDE includes early initiation of antibiotic therapy, antiedema measures, and treatment of associated seizures. In most cases, the medical management alone is insufficient and associated with high mortality.[3] Early surgical intervention by burr hole or craniotomy evacuation is the key to timely recovery and salvage of maximal neurological function. Simultaneous treatment of the sinusitis by FESS is preferred.[3] Brief note

**Table 1: Complications of subdural empyema**

| Complications | Management |
|---|---|
| Recurrent SDE | Second surgery may be necessary.[7] Open craniotomy if the first surgery is burr hole evacuation |
| Subdural effusions | Depending on the symptoms, size, and mass effect surgical drainage may be necessary with or without placement of shunt tube |
| Late seizures | Maintenance anticonvulsant therapy |
| Cavernous sinus thrombosis from septic thrombosis of adjacent cerebral veins | Aggressive intravenous antibiotic therapy with broad-spectrum coverage. Use of anticoagulants may be beneficial[8] |
| Increased intracranial pressure | Conservative or neurosurgical antiedema measures |
| Hydrocephalus | Majority needs placement of CSF shunt |
| Cerebral infarction | Comprehensive management of the intracranial infection source and mass effect |
| Cranial osteomyelitis | Surgical debridement, sequestrectomy, and reconstruction with muscle flaps or vascularized bone grafts[9] |
| Residual neurological deficits (like hemiparesis, aphasia) | Rehabilitation therapy |

SDE – Subdural empyema; CSF – Cerebrospinal fluid

on the list of complications of SDE and their management is provided in Table 1.

SDE is a rare albeit life-threatening complication of sinusitis. Nonspecific initial presentation, vague neurological signs, and unremarkable initial imaging examinations can potentially delay the diagnosis of this rapidly progressing entity. High index of clinical suspicion and prudent use of imaging are essential for appropriate management of this dreaded complication.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

## References

1. Renaudin JW, Frazee J. Subdural empyema – Importance of early diagnosis. Neurosurgery 1980;7:477-9.
2. Bruner DI, Littlejohn L, Pritchard A. Subdural empyema presenting with seizure, confusion, and focal weakness. West J Emerg Med 2012;13:509-11.
3. Osborn MK, Steinberg JP. Subdural empyema and other suppurative complications of paranasal sinusitis. Lancet Infect Dis 2007;7:62-7.
4. Waseem M, Khan S, Bomann S. Subdural empyema complicating sinusitis. J Emerg Med 2008;35:277-81.
5. Kombogiorgas D, Seth R, Athwal R, Modha J, Singh J. Suppurative intracranial complications of sinusitis in adolescence. Single institute experience and review of literature. Br J Neurosurg 2007;21:603-9.
6. Tsuchiya K, Osawa A, Katase S, Fujikawa A, Hachiya J, Aoki S. Diffusion-weighted MRI of subdural and epidural empyemas. Neuroradiology 2003;45:220-3.
7. Niklewski F, Petridis AK, Al Hourani J, Blaeser K, Ntoulias G, Bitter A, *et al.* Pediatric parafalcine empyemas. J Surg Case Rep 2013;2013. pii: Rjt067.
8. Levine SR, Twyman RE, Gilman S. The role of anticoagulation in cavernous sinus thrombosis. Neurology 1988;38:517-22.
9. Pincus DJ, Armstrong MB, Thaller SR. Osteomyelitis of the craniofacial skeleton. Semin Plast Surg 2009;23:73-9.