

Rapidly Developing Subdural Empyema in an Adult with Sinusitis- A Neurosurgical Threat Alert!

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Abstract

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 commonest form of intracranial involvement is subdural empyema with bacterial sinusitis being its leading cause.

We present a rare case of rhinosinusitis that, within a few hours progressed into a large subdural empyema 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.

Keywords: Subdural empyema; Sinusitis; Multidetector computed tomography

Case Report

A 45 year old man presented to accident and emergency (A&E) department with two days history of fever, chills, productive cough, runny nose and gradual onset headache. He had temperature of 39.8°C, heart rate of 115 beats per minute (BPM), blood pressure of 130/70 mmhg and respiratory rate of 18 per minute. 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 x10⁹/L). The chest radiograph was normal. After receiving symptomatic treatment for presumed upper respiratory tract infection, he was discharged from the A & E. After six hours, the patient was brought to the A&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. His WBC count increased to 31 x 10⁹/L with predominant neutrophils (84%). CRP (C-reactive protein) levels were grossly elevated (245.6 ml/L). He was intubated. An urgent non-contrast computed tomography (CT) of the brain (Figure 1) revealed subtle fullness in the right frontal region with mild sulcal effacement. The paranasal sinuses showed 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 (leucocytosis and increased protein levels). Meanwhile, his condition further deteriorated and GCS dropped to 7.

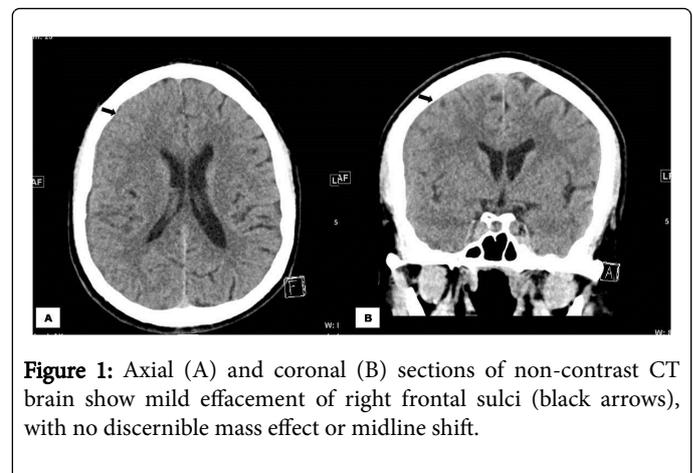


Figure 1: Axial (A) and coronal (B) sections of non-contrast CT brain show mild effacement of right frontal sulci (black arrows), with no discernible mass effect or midline shift.

In the view of sudden neurological worsening, a contrast enhanced magnetic resonance imaging (MRI) of brain (Figure 2) was performed which showed a large peripherally enhancing right fronto-temporo-parietal 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 oedema. A diagnosis of sinogenic meningo-encephalitis with multiple subdural empyemas (SDE) was inferred from MRI. Retrospective inspection of the initial CT images of the brain confirmed the bone defect in the corresponding location of left frontal sinus (Figure 3).

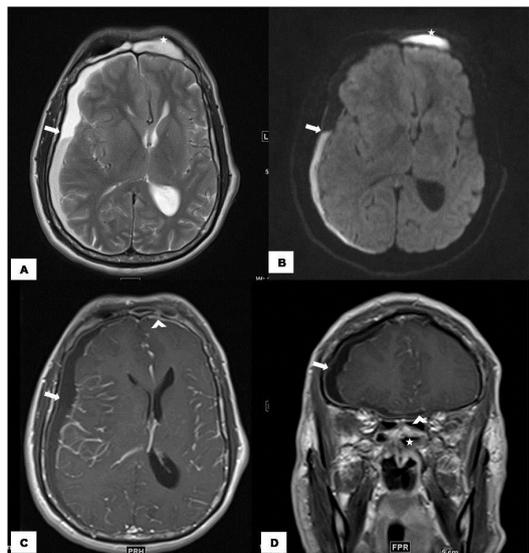


Figure 2: Contrast enhanced MRI taken 8 hours after the initial CT. A) Axial T2 weighted image shows the right convexity subdural collection with layering of fluid (white arrow). Marked mass effect with midline shift to the left is seen. The left frontal sinus (white asterisk) is fluid filled. B) Diffusion weighted image 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 (arrow head) 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 subfalci region.



Figure 3: A,B) Bone window CT 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.

He underwent right decompressive craniectomy and evacuation of subdural empyema. 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. Post-operatively, he was treated with intravenous antibiotics (intravenous Meropenem and Vancomycin for 2 weeks) and stayed in intensive care unit for five days. His condition improved gradually and the follow up CT (Figure 4) showed improvement of mass effect and cerebral oedema. After 20 days of hospital stay, he was discharged with good neurological status. The inflammatory markers demonstrated a gradual decline and the CRP was 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.



Figure 4: A) Immediate post-craniectomy CT scan axial section showing resolution of mass effect and the midline shift as the subdural empyema has been evacuated. B) Follow-up CT after nine months showing stable craniectomy changes and complete resolution of mass effect. C) 2 year follow-up study showing more pronounced encephalomalacic changes and parenchymal loss of the right cerebral hemisphere.

Unfortunately he developed scar epilepsy and his follow-up CTs demonstrated significant parenchymal loss and gliosis (Figure 4). 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 sub divided into local manifestations (like mucocoele, pyocoele and recurrence) and extension of sepsis to the adjacent orbital or intracranial structures. Orbital complications include orbital cellulitis, subperiosteal abscess, intra-orbital abscess and osteomyelitis. The intracranial complications comprise of meningitis, encephalitis, epidural or subdural empyema, cerebral abscess and cavernous or other dural venous sinus thrombosis [1]. The true incidence of intracranial sepsis in sinusitis is unknown, as majority of the uncomplicated sinusitis does not get medical attention [2]. 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 sphenothmoid 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 [1,3].

SDE represents collection of pus between the dura and pia layers of the meninges [3]. 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 [2]. 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 like congenital or acquired skull defects and the natural skull foramina [4]. The more commonly implicated mechanism however is the indirect spread via 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 oedema, 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 subdural

empyema has fulminant clinical course due to rapid spread of purulence in a space that lacks anatomical constraints [2]. Long term sequelae of SDE include hydrocephalus, residual hemiparesis and epilepsy. CT is the imaging modality of choice when immediate surgical management is contemplated [4]. 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 from epidural empyema and subdural effusion which follow CSF signal on DWI [5]. MRI is superior to CT in evaluating the orbital inflammation and status of the dural venous sinuses.

Medical management of subdural empyema includes early initiation of antibiotic therapy, anti-oedema measures and treatment of associated seizures. In most cases the medical management alone is insufficient and associated with high mortality [2]. 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 [2].

SDE is a rare albeit life threatening complication of sinusitis. Non-specific 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.

References

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