# Pitfalls in the determination of intracranial spread of complicated suppurative sinusitis

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## Abstract

We present two cases of suppurative sinusitis that presented to our casualty department over a one-week period. Both patients suffered complications of the disease secondary to extension of the inflammatory process beyond the bony confines of the sinus. Neither of the patients had a previous history of sinus disease. The first patient deteriorated suddenly 24 hours after admission. The initial computed tomography (CT) scan failed to demonstrate a developing subdural empyema. This complication was confirmed following repeat scanning 24 hours later and the patient required urgent neurosurgical intervention and drainage. The second patient presented with periorbital cellulitis secondary to sinusitis and suffered a grand mal seziure on admission. Once again initial CT scan changes were subtle and significant intracranial extension was not noted until the subsequent magnetic resonance imaging (MRI) scan was performed.

The purpose of this paper is to highlight the potential dangers over reliance on CT scanning in diagnosing early intracranial spread of sinus disease and we emphasise that the clinician must interpret any radiological investigations in light of the associated clinical findings.

Key words: Sinusitis, complications; Subdural empyema; Tomography, X-ray computed

## Introduction

Intracranial suppuration is a well recognised, albeit rare, complication of paranasal sinusitis and is still seen despite the widespread use of antiboitics. Due to the intimate relationship of the paranasal sinuses to the orbit and brain, rapid spread of infection to these organs still poses a threat to sight and life.

Clearly, early diagnosis and rapid intervention is the mainstay of treatment and accounts for the improvements in morbidity and mortality seen in the management of this disease over the past 30 years. Plain radiographs may be helpful in demonstrating sinus opacification, subtle changes in the calvarium or lateral shift of the pineal gland but they are largely non-diagnostic and have been supplanted by the use of CT and MRI scanning in the determination of suppurative extension (Gardiner, 1986). Difficulties arise however in the management of those patients in whom one clinically suspects intracranial suppurative disease, yet have normal or only subtle changes on CT scanning.

In *Case 1* sinogenic infection spread only to the cranial cavity whereas in *Case 2* inflammation involved both the orbital and intracranial contents.

# **Case reports**

#### Case 1

A 16-year-old male presented with a four-week history of left frontal headache which had worsened over the preceding 48 hours and was associated with nausea and vomiting. On admission his temperature was 38.5°C, pus was present in the left middle meatus and he had a tender left frontal sinus. He was fully orientated in time, place and person and had no focal neurological signs. A CT scan demonstrated left-sided pansinusitis with enhancement of the anterior falx (Figure 1). There was no evidence of intracranial collection. The patient was commenced on high dose broad spectrum antibiotic cover and remained stable for the first 24 hours following admission. Over the next 12 hours he started to deteriorate. The patient became disorientated in place and time, developed generalised increased muscle tone and an upgoing right plantar response. Under general anaesthesia, emergency bilateral antral lavage and a frontal trephine was performed through an external Howarth approach. Frank pus was present in both the left maxillary antrum and the

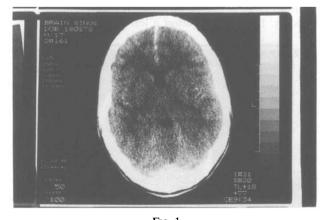
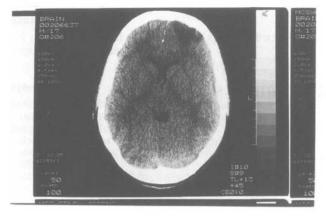


FIG. 1 Initial CT scan for *Case 1* fails to demonstrate any intracranial abnormality.

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#### FIG. 2

CT scan of Case 1 undertaken 28 hours later demonstrates expanding subdural empyema.

frontal sinus. A portex drain was placed in the frontal sinus and a post-operative cranial CT scan was performed. This repeat CT scan confirmed the clinical suspicion of intracranial extension of disease and demonstrated a wedge-shaped area of hypoattenuation in the left frontal region consistent with a venous infarct (Figure 2). The patient was transferred the National Neurosurgical Centre where he underwent a craniotomy and evacuation of a subdural empyema. He made a full recovery with no residual neurological impairment.

#### Case 2

A 16-year-old female presented to the A and E department with a five-day history of frontal headache, left periorbital swelling and photophobia. Apart from a recent upper respiratory tract infection she had no previous history of sinus disease. She had been treated by her local general practitioner with amoxycillin/clavulanic acid. While in the A and E department she developed tonic clonic seizures associated with incontinence. A presumptive diagnosis of intracranial extension of suppuration was made and the patient was intubated and ventilated. Metronidazole and cefotaxime were commenced intravenously. An emergency cranial CT scan demonstrated pansinusitis with narrowing of the cortical sulci and possible reduction in size of the lateral ventricles (Figure 3). No intracranial collection was noted. Following consultation with the neurosurgical department the patient was transferred to the intensive care unit and ventilated for 24 hours. During this period a left external ethmoidect-

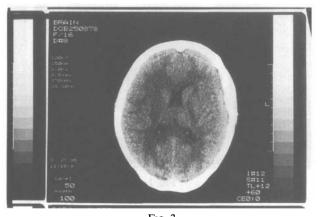
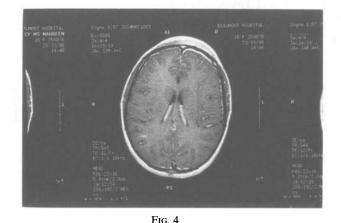


FIG. 3 CT scan of *Case 2* fails to demonstrate intracranial extension of disease process.



MRI scan of *Case 2* demonstrates left frontal dural enhancement.

omy, frontal sinus trephine and bilateral antral lavage was performed in theatre. Polypoidal inflamed mucosa were present in the left ethmoid and frontal sinus. Frank pus was aspirated from the right maxillary sinus and  $\beta$  haemolytic *Streptococcus* group A were cultured later. Prior to extubation an MRI scan was preformed 24 hours after the initial CT scan. This scan revealed significant enhancement in the left frontal dural region suggestive of cerebritis (Figure 4). There was no intracranial collection of pus. The patient was subsequently extubated and made a complete recovery. The periorbital swelling settled over one week with full return of normal vision and eye movement.

# Discussion

The ability of sinogenic infection to invade the cranial cavity has long been recognised and the first successful treatment was recorded by De la Peyronie in 1699 (Pattisapu and Parent, 1987). The infectious process usually spreads from the frontal sinus to the cranial contents by extension along communicating veins. Deposition and proliferation of organisms in the subdural space can result in the rapid accumulation of a subdural empyema. This is the most frequent intracranial complication of frontal sinusitis. Further retrograde extension of septic emboli from the sagittal sinus to the cortical veins can lead to a brain abscess.

Although there has been no reduction in the incidence of frontal sinusitis since the introduction of antibiotics, the morbidity and mortality resulting from suppurative complications of sinogenic infection has improved significantly since the development of antibiotics. Prior to the introduction of penicillin subdural empyema was almost universally fatal (Schiller et al., 1948). Mortality figures subsequently remained relatively static until the newer developments in diagnostic radiology became widespread over the past 20 years. Before the advent of CT scanning mortality was frequently reported in the region of 40 per cent. Mortality is currently reported in various series as ranging from seven per ecnt to 25 per cent (Zimmerman et al., 1984; Wackym et al., 1990; Bok and Peter, 1993). Clearly although this represents a great improvement there are still a significant number of patients who succumb or suffer long term morbidity as a result of delay in early diagnosis and rapid implementation of early treatment.

In *Case 1* the initial subtle change on the CT scan was not interpreted as representing an expanding subdural empyema and on this supposition conservative treatment was instituted. With the subsequent development of focal neurological signs it was apparent that intracranial extension had occurred and that neurosurgical intervention would be required. This decision was made in light of the evolving neurological findings and the initial CT scan was unhelpful in making this decision. In retrospect, if the initial investigation had been an MRI scan, the developing subdural empyema may have been noted prior to the onset of clinical signs, which would have facilitated earlier neurosurgical intervention.

Skelton *et al.* (1992) retrospectively reviewed 10 cases of children who suffered sinogenic subdural/extradural empyema and noted that in 50 per cent of cases the initial CT scan was reported as normal. Wetterling *et al.* (1987) also reported a case in which initial CT scanning failed to demonstrate significant subdural empyema in a patient, resulting in delayed intervention and possibly contributing to the fatal outcome.

Komori *et al.* (1992) reported on a similar case in a 14year-old boy in which CT scanning failed to detect a subdural empyema that was clearly seen on MRI. They also concluded that contrast-enhanced MRI was a more powerful and better diagnostic tool than contrast-enhanced CT scan.

We feel that the clinician should be aware that initial CT changes may be very subtle and thus easily overlooked when attempting to determine early intracranial extension of suppurative sinus disease. In cases where such complications are suspected one should be aware that a negative CT scan does not outrule intracranial pus and MR imaging should be sought or CT scan repeated in 24 hours if MRI is not available. The radiological findings must be interpreted in the light of clinical findings and if a clinical suspicion of intracranial extension exists, even with only subtle radiological changes, immediate neurosurgical consultation should be sought.

Only if the clinician carries a high index of suspicion and persists in accurate imaging can we hope to see a continuing decline in the mortality associated with this disease process.

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