

From Sinus Infection to Subdural Empyema: A Diagnostic Pitfall with Atypical Non-Contrast CT Findings in a Pediatric Epileptic Patient

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Background: Subdural empyema (SDE) is a rare pyogenic infection between the dura mater and arachnoid mater, accounting for 5–25% of intracranial infections in children, with mortality up to 9%. Early diagnosis, antimicrobial therapy, and surgical drainage are essential.

Case Presentation: A 14-year-old male with epilepsy and prior head trauma presented with recurrent tonic-clonic seizures, headache, and seven-day fever (39.5°C). Labs showed leukocytosis and elevated CRP. Non-contrast CT initially suggested cerebral infarction; subsequent CT revealed right-sided pansinusitis. Intraoperative purulent material prompted conversion from burr hole to craniotomy, confirming SDE. The patient received six weeks of intravenous penicillin G and metronidazole, with continued valproic acid. He was discharged on day 14 with marked neurological improvement, and uneventful cranioplasty was performed three months later.

Conclusion: SDE must be considered in pediatric patients with sinusitis and head trauma history. Early recognition and prompt intervention are critical to prevent severe complications.

Keywords: Subdural Empyema, Epilepsy, Pansinusitis, Traumatic Brain Injury, Case Report

INTRODUCTION

Subdural empyemas (SDEs) are time-sensitive neurosurgical emergencies associated with significant morbidity and mortality, often resulting from delayed diagnosis and treatment [1]. These lesions may arise from a variety of causes, including complications of otitis media and sinusitis, distant bacterial infections, head trauma, and neurosurgical

procedures [2]. The cornerstone of treatment is prompt surgical drainage [3]. Source control and targeted intravenous antibiotic therapy are also essential components of management [1,4]. Owing to advances in neuroimaging, antimicrobial therapy, and sepsis management, the mortality rate of SDE has decreased to 6%–15%. Nevertheless, complications such as seizures and various forms of paresis continue to affect a substantial proportion of patients [1,4–6].

On imaging, SDE typically appears as a hypo- or isodense subdural collection with peripheral rim enhancement [7–9]. This report describes a child with sinusitis who presented with an atypical hypodense subdural collection on non-contrast CT that mimicked cerebral infarction. The purpose of this report is to emphasize that, under certain circumstances, SDE may present as a hypodense lesion on non-contrast CT, resembling a subdural collection or effusion. This case highlights the importance of considering the clinical context when interpreting atypical imaging findings, as SDE is a neurosurgical emergency that requires prompt diagnosis and treatment.

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CASE REPORT

A 14-year-old male presented to our emergency department with recurrent generalized tonic-clonic seizures. Following the seizures, he was unresponsive, with a Glasgow Coma Scale (GCS) score of 9 (E2V2M5), accompanied by significant aphasia but no motor weakness in either extremity. Seven days before admission, he developed a persistent throbbing headache accompanied by fever (39.5 °C). Three months before admission, he had sustained head trauma but did not seek medical attention. The patient had been diagnosed with epilepsy at the age of five years and had been taking valproic acid 500 mg twice daily (1,000 mg/day) regularly since then. Written informed consent was obtained from the patient's legal guardian.

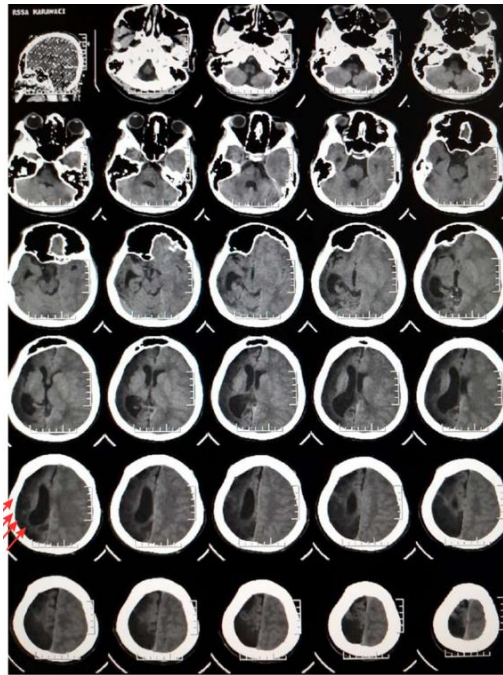


Figure 1. Non-contrast CT scan revealed large hypodense lesion expanded from the frontotemporoparietal of the right brain, the left lateral ventricle had disappeared, the midline was shifted to the right.

Laboratory investigations revealed leukocytosis, an elevated neutrophil-to-lymphocyte ratio, and increased C-reactive protein levels. A lumbar puncture was initially recommended; however, the patient's family declined the procedure. A non-contrast CT scan revealed a large crescentic hypodense lesion in the right frontotemporoparietal region, suggestive of cerebral infarction (Figure 1). Further evaluation demonstrated right-sided pansinusitis.

Due to the patient's worsening clinical condition and the high index of clinical suspicion, emergency surgery was performed. The procedure was initially started with a burr hole to drain the presumed subdural collection. Upon encountering purulent fluid, the procedure was converted to a craniotomy, confirming the diagnosis of SDE. The bone flap was left off because of persistent cerebral oedema observed intraoperatively and concern for possible cerebral venous thrombosis.

Postoperatively, the patient received intravenous penicillin G at a dose of 3 million units every 4 hours (approximately 18 million units/day) and intravenous metronidazole at 7.5 mg/kg every 6 hours (375 mg per dose based on a body weight of 50 kg) for a planned duration of six weeks. Valproic acid therapy was continued to prevent recurrent seizures. The patient remained hospitalized for 14 days and was discharged on postoperative day 14 with marked neurological improvement and a substantially reduced seizure frequency.

At the 3-month follow-up, the patient had completely recovered from aphasia, with no persistent speech impairment, motor weakness, or cognitive dysfunction. Cranioplasty was performed successfully without any complications. Valproic acid therapy was continued for the management of the underlying

From Sinus Infection to Subdural Empyema: A Diagnostic Pitfall with Atypical Non-Contrast CT Findings in a Pediatric Epileptic Patient

epilepsy. At the final follow-up, 7 months after the initial hospital admission, the patient remained seizure-free.

DISCUSSION

SDE is a recognized complication of bacterial meningitis, occurring in approximately 2.7% of cases and often associated with poor neurological outcomes [1,9]. SDE is characterized by the accumulation of purulent fluid between the dura mater and the arachnoid mater and may occur in infants with meningitis. In older children, conditions such as sinusitis, mastoiditis, and otitis media are also common causes of SDE [2,10]. Both subdural effusions and SDE may develop following meningitis. Subdural effusions are more common, particularly in children, are typically asymptomatic, and often resolve spontaneously [11]. In contrast, SDE is less common, tends to progress rapidly, and usually requires urgent surgical drainage [11]. In the present case, the collection appeared as a crescentic hypodense lesion on non-contrast CT, mimicking cerebral infarction. Without contrast-enhanced imaging, the characteristic rim-enhancing appearance of SDE could not be evaluated. Given the atypical hypodense appearance on non-contrast CT, the lesion may have represented an early stage of infection before the development of a well-formed inflammatory capsule [12,13].

From a pathophysiological perspective, the body attempts to localize infection to limit its spread. In empyema, an inflammatory membrane forms around the purulent collection [12]. This feature also helps distinguish SDE from subdural hematoma (SDH) on imaging. SDH typically appears as a hypodense crescent-shaped collection that is not limited by the cranial sutures. Although a membrane may also develop in SDH, it occurs less frequently [12,13]. In contrast, SDE usually appears as a low-density crescent-shaped collection over the cerebral convexity with a membrane that demonstrates contrast enhancement. This prominent membrane enhancement is a distinguishing imaging feature of SDE compared with subacute or chronic SDH [12,13]. Yuan et al. demonstrated that gadolinium-enhanced magnetic resonance imaging (MRI) can accurately and sensitively delineate both the empyema collection and its inflammatory capsule [6]. Another useful modality for differentiating SDE from SDH is diffusion-weighted imaging (DWI). Both hematomas and empyemas may appear as hyperintense fluid collections on DWI; however, restricted diffusion strongly favors the diagnosis of empyema [14,15]. This restricted diffusion is attributed to the highly viscous, purulent contents of the empyema [14,15]. Once the diagnosis of empyema is established, immediate neurosurgical intervention is required.

Similarly, brain abscess formation progresses through four sequential stages: early cerebritis, late cerebritis, early capsule formation, and late capsule formation [16]. The earliest stage, typically occurring within the first 1–3 days after the primary infection, is characterized by absent or minimal contrast enhancement on neuroimaging. However, patients presenting during this stage are uncommon, and when they do present, they usually exhibit substantial perilesional edema, a finding that was absent in our patient (Figure 1) [17,18]. Therefore, the non-enhancing appearance of the SDE in this case may represent an early stage of infection, possibly during the cerebritis phase before complete inflammatory capsule formation [1,13].

Laboratory findings such as leukocytosis may support the diagnosis of SDE, and bacterial infections are often associated with a left shift in the differential white blood cell count [19]. SDE may also be associated with elevated inflammatory markers, including erythrocyte sedimentation rate, C-reactive protein, and procalcitonin. However, blood tests are indirect diagnostic measures compared with cerebrospinal fluid (CSF) analysis [19]. Microbiological evaluation of both blood and empyema fluid is essential, as identification of the causative pathogen allows antibiotic therapy to be tailored appropriately, thereby improving patient outcomes [2].

The management of subdural empyema requires a multidisciplinary approach involving specialists in neurosurgery, infectious diseases, neurology, rehabilitation, and psychiatry [10]. Monotherapy is generally insufficient for the treatment of SDE. Instead, combination antibiotic therapy is recommended and should be adjusted according to microbiological culture results and antimicrobial susceptibility testing. Consideration should also be given to bacterial resistance, treatment duration, and

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potential adverse effects, with antibiotic therapy typically continued for 3–6 weeks [19]. Surgical management involves burr holes or craniotomy to achieve adequate drainage and debridement. Although craniotomy is associated with better outcomes and lower recurrence rates, it requires informed consent from the patient or the patient's family [9]. Important prognostic factors include the patient's preoperative neurological status, the timeliness of surgical intervention, and the aggressiveness of treatment. Patients who are awake and alert generally have more favorable outcomes, whereas those presenting in a coma have a higher risk of mortality [20]. In our case, the patient received an appropriate course of antibiotic therapy; however, his condition had already deteriorated because of SDE. Following timely craniotomy and drainage, together with continued antibiotic therapy, the patient's clinical condition improved markedly.

This report has several limitations. Contrast-enhanced CT and MRI were not performed, precluding detailed radiological characterization of the subdural collection and assessment of meningeal enhancement. MRI was unavailable because of limited resources at our institution, which we acknowledge as a major limitation of this case. In addition, no microbiological specimens were obtained because cultures were not collected during surgery.

CONCLUSION

To reduce morbidity and mortality, subdural empyema (SDE) must be recognized and treated promptly, as it is a treatable neurosurgical emergency. In most cases, SDE appears on imaging as a peripherally enhancing subdural collection. However, this report describes a case in which SDE presented as an atypical hypodense collection on non-contrast CT, mimicking cerebral infarction. Distinguishing SDE from subdural hematoma can be challenging, particularly when imaging findings are atypical. Therefore, a high index of clinical suspicion is essential. When the diagnosis remains uncertain on non-contrast CT, SDE should be considered, and contrast-enhanced CT or gadolinium-enhanced magnetic resonance imaging (MRI) should be performed. Patients presenting with severe headache, fever, and deteriorating mental status, particularly those with a history of sinusitis, warrant careful evaluation. Early diagnosis of SDE is critical for improving treatment outcomes. This case underscores the importance of maintaining a high index of clinical suspicion and proceeding with timely surgical intervention when the clinical presentation strongly suggests SDE, even when the initial imaging findings are atypical.

DISCLOSURES

Ethical approval

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the local Ethics Committee of RSUP Dr. Kariadi, number: 16724/EC/KEPK-RSDK/2026.

Consent to participate

The patients gave consent to use their information and images for research purposes. *Consent for publication*

Conflict of interest

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper

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Artificial intelligence

The authors affirm that no artificial intelligence tools were used in the writing, editing, or content generation of this manuscript.

CONTRIBUTIONS

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From Sinus Infection to Subdural Empyema: A Diagnostic Pitfall with Atypical Non-Contrast CT Findings in a Pediatric Epileptic Patient

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