



# Treatment-refractory *Escherichia coli* subdural empyema caused by infection of a chronic subdural hematoma in an infant

Jason A. Chen<sup>1</sup> · Dimitrios Mathios<sup>2</sup> · Joaquin Hidalgo<sup>2</sup> · Alan R. Cohen<sup>2</sup>

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## Abstract

**Introduction** Subdural empyema (SDE) is a neurosurgical emergency that is typically treated with surgical drainage, either by burr hole or by craniotomy. *Escherichia coli* is an uncommon cause of SDE and is associated with infection of a pre-existing subdural hematoma.

**Case report** We report the case of an otherwise healthy, immunocompetent 4-month-old infant girl with an *E. coli*-infected subdural hematoma. The infection persisted despite aggressive neurosurgical treatment that included drainage of the subdural space through burr holes and, subsequently, a wide craniotomy was performed. Ultimately, after 30 days, the SDE resolved with good neurological outcome. A review of prior literature indicates that infected subdural hematomas (including those caused by *E. coli*) are typically less aggressive and respond to burr hole drainage.

**Conclusion** We illustrate the fulminant progression of the SDE in the face of neurosurgical treatment. Our experience suggests lowering the threshold for wide craniotomy in these incompletely understood cases.

**Keywords** Subdural empyema · Subdural hematoma · Intracranial abscess · *Escherichia coli*

## Introduction

Subdural empyema (SDE) typically occurs following otorhinogenic infections (sinusitis, otitis media, mastoiditis), penetrating head injuries, or neurosurgical procedures. In infants, the frontal sinus is undeveloped and meningitis is the most common cause [1, 2]. Occasionally, infection of an existing chronic subdural hematoma causes SDE in children and adults. *Escherichia coli* and *Salmonella* species appear to be the most common organisms involved in this specific etiology, whereas Gram-positive organisms typically predominate [3, 4]. The optimal method of surgical drainage, whether by burr hole or craniotomy, has not been firmly established [5].

Here, we present a case of an isolated, bilateral SDE caused by *E. coli* in an infant, refractory to multiple neurosurgical

treatments. This case extends the spectrum of presentation of this rare disease entity and provides insight into its natural history and management.

## Report of a case

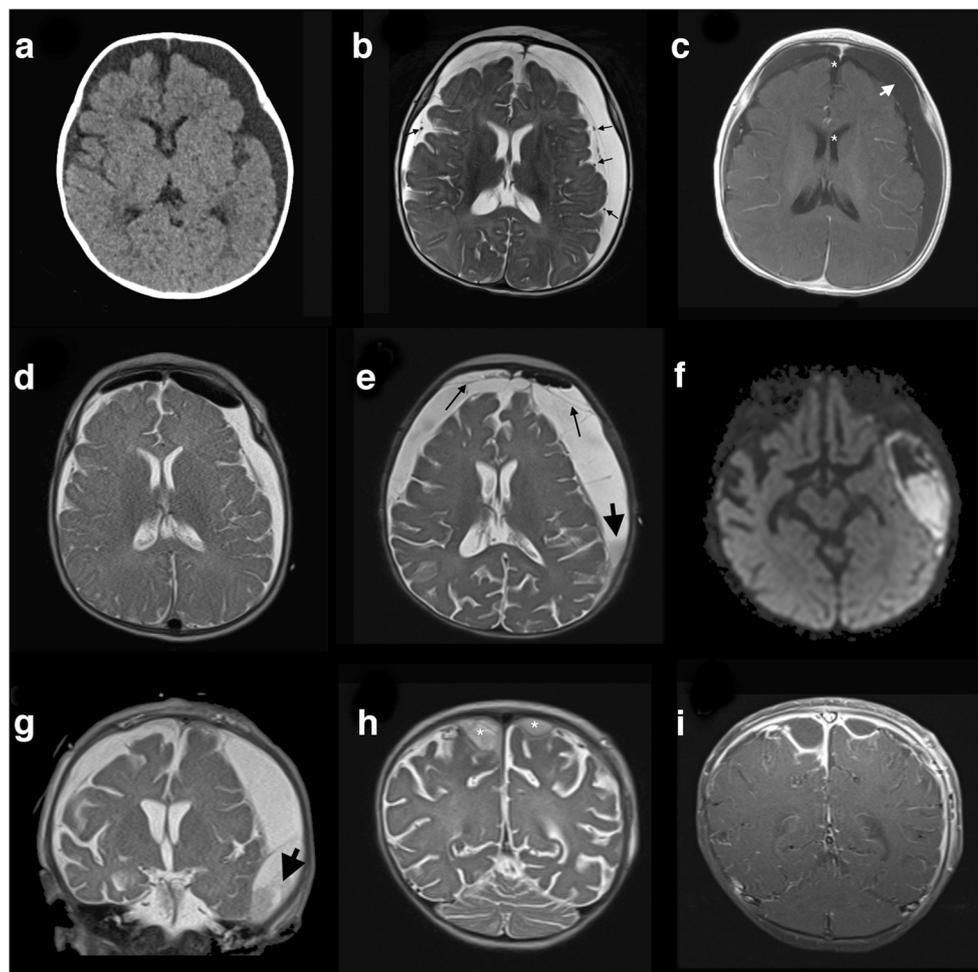
A 4-month-old girl presented with 1 day of emesis, lethargy, and fever. Birth history was unremarkable. Four weeks prior to admission, the patient presented to an outside hospital with emesis; head ultrasound revealed a small frontal intracranial fluid collection, and laboratory workup (including a complete blood count and urinalysis) was normal. She was discharged and the emesis resolved. Ten days prior to admission, the patient was seen for a well-child check and her head circumference had increased from 41 cm (65th percentile) to 43.5 cm (98th percentile) over 1 month.

The patient was evaluated at an outside hospital. Based on clinical suspicion for meningitis, the patient was started on high-dose vancomycin and ceftriaxone. Chest radiography, urinalysis, and lumbar puncture were normal. A non-contrast CT of the head revealed bilateral, prominent extra-axial spaces in the frontotemporal convexities and rightward midline shift (Fig. 1a), and the patient was transferred. The parents denied

✉ Alan R. Cohen  
alan.cohen@jhmi.edu

<sup>1</sup> Department of Neurosurgery, David Geffen School of Medicine, UCLA, Los Angeles, CA, USA

<sup>2</sup> Division of Pediatric Neurosurgery, Department of Neurosurgery, The Johns Hopkins University School of Medicine, Baltimore, MD, USA



**Fig. 1** Neuroimaging throughout hospital course of subdural empyema. **a** At presentation, non-contrast CT in the axial plane shows bilaterally enlarged frontotemporal extra-axial spaces, left greater than right, and mild left-to-right midline shift. **b** T2-weighted MRI in the coronal plane demonstrates enlargement of the subdural space; cortical veins within the subarachnoid space can be seen (thin arrows). **c** Post-contrast T1-weighted MRI in the coronal plane again demonstrates enlargement of the subdural and subarachnoid spaces; the subdural fluid (arrow) is hyperintense relative to the subarachnoid and intraventricular CSF (asterisk). **d** Status post left burr hole evacuation and subdural drain placement (post-admission day 2): T2-weighted MRI in the coronal plane demonstrates decreased size of the subdural space and post-operative pneumocephalus. **e** Eight days after initial burr hole evacuation (post-admission day 9): interval increase in

size and development of loculations (thin arrows) within the subdural space. Subdural debris is noted in the dependent area of the subdural collection (thick arrow). **f** Three days after bilateral subdural drain placement (post-admission day 12): trace diffusion-weighted imaging (DWI) in the axial plane demonstrates diffusion restriction within a left temporal lobe abscess. **g** T2-weighted MRI in the coronal plane demonstrates the temporal region abscess with mass effect on the adjacent left temporal lobe (thick arrow). **h** Seven days after left temporal craniotomy (post-admission day 19): T2-weighted MRI in the coronal plane demonstrates resolution of temporal region abscess but interval development of parasagittal subdural abscesses (asterisks). **i** Post-contrast T1-weighted MRI shows contrast enhancement of the abscess rim

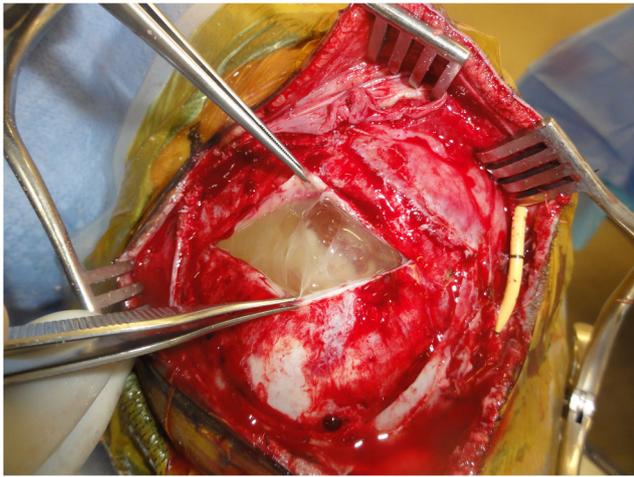
any history of head trauma, urinary tract symptoms, or gastroenteritis.

On admission, the patient was somnolent but spontaneously moved all extremities. The anterior fontanelle was full. A complete blood count was normal. CSF obtained by lumbar puncture showed no white blood cells or red blood cells, glucose of 83 mg/dl (58% of serum level), and protein of 36.1 mg/dl; the opening pressure was 15 cm H<sub>2</sub>O. There were no retinal hemorrhages or bone fractures. Workup for immunodeficiency, platelet function, and coagulation abnormalities was unremarkable. CSF, peripheral blood, and urine cultures (from the outside hospital and repeated on admission) were

negative. An MRI more clearly visualized the bilateral, slightly T1 hyperintense, subdural fluid collections (Fig. 1b, c), which did not appear loculated. The head circumference was measured at 45.5 cm (>97th percentile) and remained stable for the remainder of the admission.

### Surgical treatment

Initial surgical treatment comprised urgent unilateral subdural drain placement, as the fluid appeared communicating throughout. A burr hole was placed at the left posterior aspect



**Fig. 2** Intraoperative photograph of left temporal craniotomy (post-admission day 12): cruciate opening of the dura following temporoparietal craniotomy demonstrating copious subdural pus

of the anterior fontanelle. The dura was opened, revealing yellow fluid under pressure. The fluid was drained and an antibiotic-impregnated silicone catheter was placed in the subdural space. Laboratory studies and cultures of the purulent fluid revealed heavy *E. coli*, with 12,639 WBC/ $\mu$ l (neutrophil predominant), 3000 RBC/ $\mu$ l, <2 mg/dl glucose, and 727.8 mg/dl protein. The patient was continued on vancomycin and ceftriaxone until a polymicrobial infection was ruled out, and subsequently narrowed to ceftriaxone.

Post-operatively, yellow fluid continued to drain with decreasing subdural fluid (Fig. 1d). Daily fluid cultures revealed persistent *E. coli*. On the 9th post-admission day, the drain output decreased and the subdural fluid collections increased, with loculation (Fig. 1e). Therefore, we revised the drain and placed a second contralateral drain. Frank subcutaneous pus oozed from the previous drain site. The wound and subdural space were irrigated with gentamicin-infused saline and closed.

Subsequent fluid cultures were negative for *E. coli*, but the patient's fevers and lethargy persisted. On the 12th post-admission day, an MRI revealed increasing subdural fluid with apparent loculations, despite broadening antibiotic

coverage to include ciprofloxacin (Fig. 1f, g). Therefore, we performed a wide left temporoparietal craniotomy for washout of the purulent collection. The dura was tense with thick yellow pus and a surrounding rind (Fig. 2). Following washout, a drain was placed in the subdural space prior to closing the wound.

On the 19th post-admission day, an MRI showed bilateral ring-enhancing parasagittal subdural abscesses (Fig. 1h, i). We created two parietal burr holes 2 cm lateral to midline and evacuated thick yellow-green pus. Cultures of the pus grew light *E. coli*. The patient's fevers subsided and she improved clinically. The infection resolved by the 29th post-admission day (Fig. 3) and the patient was discharged to inpatient rehabilitation with IV ciprofloxacin and ceftriaxone, without any deficits at 1-month follow-up. By 7 months of age, the patient was developmentally normal and her head circumference had decreased to 43.5 cm (66th percentile).

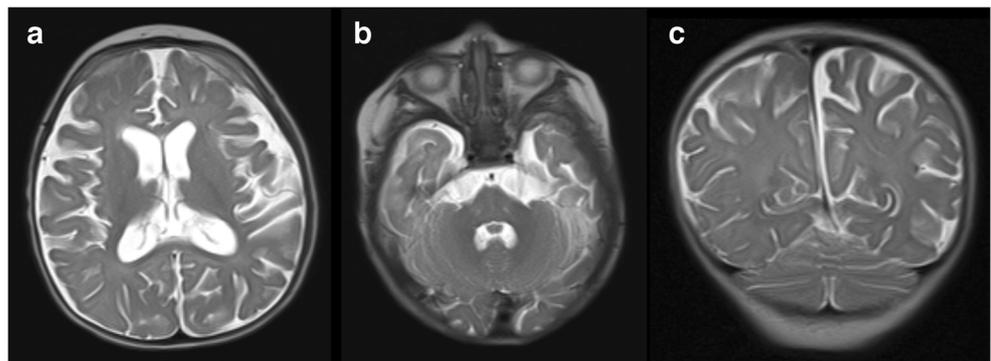
## Discussion

We present a case of SDE caused by *E. coli* that required aggressive surgical management. The etiology of SDE was presumed to be infection of a pre-existing chronic subdural hematoma, as evidenced by symptoms and ultrasound findings prior to admission and aspiration of yellow-tinged fluid during initial drainage.

Literature review identified nine cases of *E. coli*-related SDE in infants (Table 1). The main etiologies were meningitis [1, 6] and infected chronic subdural hematoma [7–12]. Each of the cases of infected chronic subdural hematomas (as well as our case) lacked a history of preceding trauma. In most cases, no primary infection was identified; in one case, a urinary tract infection was the presumed source. *E. coli* bacteremia can be caused by urinary tract infection or gut translocation [13] and we speculate that this could be the case for our patient.

The treatment of SDE consists of identification of the primary infection and organism, antibiotic treatment, and drainage of purulent material and normalization of ICP [14]. In

**Fig. 3** T2-weighted MRI demonstrating resolution of subdural empyema, post-admission day 29, in the **a** axial plane at the level of the third ventricle, **b** axial plane at the level of the prior temporal lobe abscess, and **c** coronal plane at the prior parietal abscesses. The subdural and subarachnoid spaces have markedly reduced in size



**Table 1** Case reports of *Escherichia coli*-related subdural empyema in infants (0–2 years of age)

Ref.	Age	Sex	Presentation	Primary source	Risk factors	SDH etiology	Treatment	Outcome
This case	4 months	F	Fever, emesis, lethargy	Unknown	Chronic subdural hematoma	Unknown	Burr hole drainage and subdural drain placement; craniotomy and subdural drain placement	Resolution of subdural empyema
Hsu et al. 2015	5 months	M	Fever, seizure, irritability	Urinary tract	Chronic subdural hematoma	Unknown	Burr hole drainage and subdural drain placement	Resolved without neurological deficits
Hendaus and Khalifa 2013	1 month	M	Intermittent fever, generalized tonic-clonic seizures, irritability, lethargy	Meningitis	Meningitis	N/A	Antibiotics alone	Resolved without neurological deficits
Imura et al. 2010	6 months	M	Fever, focal seizure	Unknown	Chronic subdural hematoma	Unknown	Burr hole drainage and subdural drain placement	Resolved without neurological deficits
Fender et al. 2000	5 months	M	Fever, increased HC, irritability, poor feeding	Not reported	Not reported	N/A	Endoscopic evacuation	Subsequent development of cavernomas
Chang et al. 1997	3 months	M	Fever, seizures	Meningitis	Meningitis	N/A	Not reported	Resolved without neurological deficits
Chang et al. 1997	4 months	M	Fever, seizures	Meningitis	Meningitis	N/A	Not reported	Resolved without neurological deficits
Miedema et al. 1996	7 months	M	Not reported	Pneumonia/bacteremia/meningitis	Chronic subdural hematoma, prior <i>Haemophilus influenzae</i> meningitis	Unknown	Not reported	Resolved without neurological deficits
Hamada et al. 1993	11 months	M	Fever, headache	Unknown	Chronic subdural hematoma	Unknown	Burr hole drainage and subdural drain placement	Resolved without neurological deficits, subsequent contralateral subdural hematoma
Smith and Hendrick 1983	15 months	F	Not reported	Surgical wound	Prior neurosurgery (craniotomy for tumor resection, VP shunt placement)	N/A	Not reported	Death from septic shock

infants with SDE without loculations or significant brain edema, burr hole drainage is often satisfactory, particularly in SDE related to meningitis or chronic subdural hematoma; in these cases, infection of pre-existing intracranial fluid collections is restricted to the subdural space [1]. Previous patients with *E. coli*-infected subdural hematomas were treated by subdural drainage via burr hole; typically, patients recovered without neurological deficits. Others attributed this to a protective rind encapsulating the SDE [9], which we also observed.

This unusual case illustrates an aggressive *E. coli* SDE in an infant without obvious risk factors. We described the progression from a subdural fluid collection to eventual infection, loculation, and abscess formation despite escalating treatment. This case emphasizes the need for appropriate surgical management—including a low threshold for wide craniotomy—and close post-operative observation.

### Compliance with ethical standards

**Conflict of interest** On behalf of all authors, the corresponding author states that there is no conflict of interest.

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