Bacterial meningitis in children: Neurologic complications

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INTRODUCTION

Bacterial meningitis continues to result in substantial morbidity and mortality despite the availability of effective antimicrobial therapy. The risk of dying or of developing complications is related to the age and underlying condition of the patient, the causative pathogen, the severity and duration of illness at the time of presentation, and, occasionally, delays in the initiation of antibiotic therapy. (See "Bacterial meningitis in children older than one month: Treatment and prognosis".)

The neurologic complications of bacterial meningitis in children will be discussed here. The neurologic complications of bacterial meningitis in neonates and adults, and the prevention of neurologic complications in children, are discussed separately. (See "Bacterial meningitis in the neonate: Neurologic complications" and "Neurologic complications of bacterial meningitis in adults" and "Bacterial meningitis in children: Role of dexamethasone".)

OVERVIEW

Complications of bacterial meningitis can be divided into systemic and neurologic. Systemic complications, such as septic shock, disseminated intravascular coagulation, acute respiratory distress syndrome, and septic or reactive arthritis, are usually the consequence of the bacteremia that frequently accompanies meningitis. (See "Bacterial meningitis in children older than one month: Clinical features and diagnosis", section on 'Clinical findings'.)

The neurologic complications of meningitis may be sudden or gradual in onset and can appear at any time after the onset of symptoms, including after the completion of therapy. Impaired mental status is seen in most patients at presentation, and seizures are more often seen during the acute episode. (See "Bacterial meningitis in children older than one month: Clinical features and diagnosis".)

Although many neurologic complications are severe and readily apparent, others may be subtle or inapparent during the early phases of infection. Sensorineural hearing loss, for example, is typically not appreciated until the patient has recovered from the acute illness or, in young children, when a hearing assessment is performed. (See "Bacterial meningitis in children older than one month: Treatment and prognosis", section on 'Hearing evaluation'.)

EPIDEMIOLOGY

Incidence — In the available studies, the incidences of short- and long-term neurologic and developmental complications have varied widely depending upon the time of the study and the duration of follow-up [1-3].

Early complications — Reported incidence rates of early complications (ie, occurring during the hospitalization or within the first month after diagnosis) are as follows [4-7]:

- Seizures – Approximately 30 percent (see 'Seizures' below)
- Subdural effusion and empyema – Approximately 8 to 15 percent (see 'Subdural effusion and empyema' below)
- Arterial stroke – Approximately 10 to 15 percent (see 'Cerebrovascular complications' below)
- Cerebral edema – 9 percent (see 'Cerebral edema' below)
- Ventricular enlargement or hydrocephalus – Approximately 5 to 15 percent (see "Hydrocephalus in children: Physiology, pathogenesis, and etiology", section on 'CNS infections')
- Venous sinus thrombosis – 5 percent (see 'Cerebrovascular complications' below)
- Brain abscess – 3 percent (see "Pathogenesis, clinical manifestations, and diagnosis of brain abscess")

Long-term disabilities — In a study of 3623 infants and children diagnosed with bacterial meningitis from 1987 to 2021 who were followed for a median of 24 years, rates of post-meningitis neurodevelopmental disabilities were as follows [3]:

- Behavioral and emotional disorders – 10.2 percent (incidence rate of 4.9 per 1000 person-years)
- Hearing loss – 8.1 percent (incidence rate of 4 per 1000 person-years)
- Vision impairment – 8.0 percent (incidence rate of 3.9 per 1000 person-years)
- Seizures – 6.9 percent (incidence rate of 3.3 per 1000 person-years)
- Cognitive impairment – 6.4 percent (incidence rate of 3.1 per 1000 person-years)
- Motor disability (including cerebral palsy) – 5.9 percent (incidence rate of 2.8 per 1000 person-years)

In a literature review of published reports (1970 to 2010), approximately one-half of 1433 survivors of pediatric meningitis beyond the neonatal period had at least one sequela at ≥5 years of follow-up [2]. Intellectual disability and behavioral disorders (eg, attention deficit hyperactivity disorder [ADHD]) accounted for 78 percent of the reported sequelae, neurologic disorders (seizures, motor disabilities, cerebral palsy) for 14 percent, hearing loss for 7 percent, and vision impairment for 3 percent.

Microbiology — In the study described above, the distribution of bacterial etiologies (when specified) among children with long-term disabilities was as follows [3]:

- *Haemophilus influenzae* (25 percent)
- *Streptococcus pneumoniae* (16 percent)
- *Neisseria meningitidis* (14 percent)
- Group A or group B *Streptococcus* (9 percent)
- Staphylococcal spp (3 percent)

Pneumococcal meningitis appears to be associated with an increased incidence of certain complications (particularly empyema and hemiparesis) in the post-13-valent pneumococcal conjugate vaccine (PCV13) era [5,8]. In one study, empyema occurred in 1 percent (1 of 67) of children with pneumococcal meningitis in the three years before introduction of PCV13 compared with 16 percent (9 of 58) of children during the three years after PCV13 introduction [8]. Similarly, hemiparesis was noted in 1 percent (1 of 76) of children in the pre-PCV13 period compared with 12 percent (8 of 69) of children the post-PCV13 period. The reasons for the increased rates of empyema and hemiparesis after PCV13 was introduced are not understood. It is unclear whether the shift to expanded PCV vaccines (PCV15 and PCV20) will impact these risks. (See "Pneumococcal vaccination in children", section on 'Invasive disease'.)

Risk factors for adverse outcomes — A major risk factor for adverse outcomes is the duration and/or progression of illness before effective antibiotic therapy, emphasizing the importance of early diagnosis and treatment [9-11]. Other factors related to outcome include the patient's age;

children less than 12 months of age are more likely to have neurologic sequelae than older children [9,12,13]. *S. pneumoniae* is associated with worse outcome compared with other pathogens [1,3,14-17]. This may result in part from the persistence of potent biologic activity in the debris of killed bacteria. The debris may stimulate the host inflammatory response, which in turn contributes to tissue damage. (See "Pathogenesis and pathophysiology of bacterial meningitis".)

A number of studies have attempted to identify predictors of adverse outcome in children with bacterial meningitis. Clinical features that have been associated with increased risk of neurologic complications or sequelae include:

- Low cerebrospinal fluid (CSF) glucose concentration [9,14]
- Thrombocytopenia [10]
- ≥2 days of symptoms before admission [14,18]
- $\geq 10^7$ colony forming units (CFU)/mL of CSF [19-21]
- Mechanical ventilation and/or a Pediatric Risk of Mortality score ≥20 within the first 24 hours [10,22]

The presence of neurologic complications in the acute phase also predicts long-term neurologic sequelae. These include:

- Seizures that are prolonged, are complicated, or begin >72 hours after admission [6,23,24]
- Focal neurologic deficits in patients who are not postictal [6]
- Ataxia [14,25-27]
- Hydrocephalus on computed tomography (CT) scan [10]

MAJOR NEUROLOGIC COMPLICATIONS

Cerebral edema — Cerebral edema can be caused by vasogenic, cytotoxic, or interstitial mechanisms [28]. The net effect of these processes is an increase in the total fluid volume of the brain, leading to a rise in intracranial pressure (ICP).

Cerebral perfusion pressure, normally maintained by an autoregulatory mechanism, becomes dependent upon peripheral blood pressure during meningitis because autoregulation is impaired [29,30]. Cerebral edema itself can increase the ICP and secondarily reduce cerebral blood flow.

- **Clinical features** – Cerebral edema and elevated ICP initially manifest with headache, confusion, irritability, nausea, and vomiting. Papilledema may be appreciated on physical

examination. More severe intracranial hypertension is characterized by severely depressed mental status (coma); cranial nerve palsy, particularly involve the abducens (VI) nerve; and bradycardia with hypertension (the Cushing reflex). In the most severe cases, this can progress to herniation of the cerebellar tonsils, leading to death.

- **Evaluation** – The possibility of cerebral edema should be considered in all critically ill patients with meningitis, particularly those with severely depressed mental status, asymmetric pupillary examination, or other cranial nerve palsy. The diagnosis is confirmed with neuroimaging, though urgent empiric treatment may be warranted if there are highly concerning clinical findings.

- **Management** – Management of elevated ICP is discussed in greater detail separately. (See "Elevated intracranial pressure (ICP) in children: Management".)

  Careful attention to fluid management is an important aspect of care in children with bacterial meningitis, and avoiding excessive hypotonic fluid administration may reduce the risk of cerebral edema. Fluid management in children with bacterial meningitis is discussed separately. (See "Bacterial meningitis in children older than one month: Treatment and prognosis", section on 'Fluid management'.)

Subdural effusion and empyema — Subdural effusions occur in 10 to 45 percent of children with acute bacterial meningitis [8,24,31-33]. Empyema is a less common but more serious complication [34].

Clinical manifestations of subdural effusions are often subtle or absent. In very young children, bulging fontanelles may be a sign of this complication, while in older children, subdural effusions can rarely produce increased ICP and a shift of intracranial structures when the fluid collection is large. By contrast, subdural empyema is generally associated with altered mental status as well as signs of increased ICP [34].

Both of these entities can be visualized on imaging, which should be performed at presentation in patients with altered consciousness or other risk factors. Imaging should also be performed in patients who deteriorate neurologically (see "Bacterial meningitis in children older than one month: Clinical features and diagnosis", section on 'Neuroimaging' and "Bacterial meningitis in children older than one month: Treatment and prognosis", section on 'Neuroimaging'). Ultrasound, CT, and magnetic resonance imaging (MRI) can identify these collections; MRI is the most sensitive and is sometimes required to distinguish them (image 1) [34].

In most children, subdural effusions produce few symptoms and require no treatment [33]. However, development of subdural empyema requires drainage (picture 1).

Seizures — Seizures occur in 20 to 30 percent of children with acute bacterial meningitis [16,35]. The pathogenesis of seizures in meningitis is not well understood. Although fever may be a cofactor in very young children, cerebrovascular inflammation or secondary neurochemical changes are presumably the cause of most seizures. In one study, as an example, the occurrence of seizures correlated with bacterial counts of greater than 10^7 colony forming units (CFU)/mL in the cerebrospinal fluid (CSF) sample prior to treatment [21].

Seizures in children with bacterial meningitis are focal, often with secondary generalization [6].

Seizures should be managed with antiseizure medication. (See "Seizures and epilepsy in children: Initial treatment and monitoring", section on 'Acute symptomatic seizure'.)

- Seizures that occur early in the course of bacterial meningitis and that are easily controlled are rarely associated with permanent neurologic sequelae. Such patients generally do not require long-term antiseizure medication.

- By contrast, seizures that are prolonged, are difficult to control, or begin more than 72 hours after hospitalization are more likely to be associated with epilepsy and other neurologic sequelae, suggesting that a cerebrovascular complication may have occurred [6,16,36]. Such patients should be evaluated with neuroimaging and may be candidates for more prolonged treatment.

Persistent neurologic deficits and other major neurologic sequelae such as stroke and empyema are predictive of late afebrile seizures (epilepsy) in children [6,37]. (See 'Cerebrovascular complications' below.)

Hearing loss — Hearing loss after bacterial meningitis may be transient or permanent. Transient hearing loss may be secondary to a conductive disturbance in many affected patients [38]. However, sensorineural hearing loss (transient or permanent) can result from damage to the eighth cranial nerve, cochlea, or labyrinth, induced by direct bacterial invasion and/or the inflammatory response elicited by the infection [38-42]. Many, but not all, patients with acute hearing loss have associated MRI abnormalities (enhancement and/or fluid-attenuated inversion recovery [FLAIR] signal) in the affected labyrinth [43,44].

Permanent sensorineural hearing loss occurs in 5 to 10 percent of children with bacterial meningitis overall and up to 30 percent of those with pneumococcal meningitis [1-3,8]. In one large cohort, hearing loss developed in 7 percent of cases, one-quarter of which were detected after the routine follow-up period had ended [14]. All of the children with hearing loss had one or more of the following risk factors at presentation:

- Symptoms for ≥2 days before admission
- Absence of petechiae
- CSF glucose concentration ≤10.8 mg/dL (0.6 mmol/L)
- *S. pneumoniae* infection
- Ataxia

Hearing loss is two to three times more common in children with pneumococcal meningitis than in those with other forms of bacterial meningitis [14,39]. In a multicenter study of 161 children with pneumococcal meningitis treated in the United States from 2007 through 2013 (three years before and after the introduction of the 13-valent pneumococcal conjugate vaccine [PCV13]), 31 percent developed hearing loss [8]. Ataxia is commonly associated with hearing loss in children, since both are related to bacterial labyrinthitis [14,25-27].

Cranial nerve palsy — Cranial nerve palsies can result from compression due to brain swelling or perineuritis due to the adjacent meningeal inflammatory reaction [45]. The VI nerve is the cranial nerve most commonly affected in meningitis, probably because its long intracranial segment adjacent to the brainstem is highly vulnerable to elevated ICP and the inflammatory reaction that can occur with meningitis. Cranial nerves III, IV, and VII may also be affected. Cranial nerve deficits related to meningitis are usually transient. (See "Third cranial nerve (oculomotor nerve) palsy in children" and "Fourth cranial nerve (trochlear nerve) palsy" and "Facial nerve palsy in children" and "Sixth cranial nerve (abducens nerve) palsy", section on 'Clinical manifestations'.)

Bacterial meningitis can induce arachnoiditis around the optic nerve, which can lead to transient or permanent visual loss. Optic atrophy that results in irreversible total blindness is a rare complication of severe meningitis [46].

Motor deficits — Hemiparesis, quadriparesis, and other motor deficits can complicate bacterial meningitis. Most motor deficits improve or resolve with successful treatment of the meningitis, but long-term disability can occur [1,2,6,35].

Paresis typically results from an intracranial abnormality (eg, cortical vein or sagittal vein thrombosis, cerebral artery spasm, subdural effusion or empyema, hydrocephalus, cerebral infarct or abscess, cerebral edema).

Paresis resulting from meningitis generally improves with time. In a review of 235 children with bacterial meningitis, hemiparesis or quadriparesis was noted in 30 patients (12 percent) shortly after discharge but persisted in only 5 (2 percent) one year after discharge [35].

Cerebrovascular complications — Thrombosis, vasculitis, acute cerebral hemorrhage or infarction, and aneurysm formation of cerebral vessels are potential complications of bacterial meningitis [23,47-50]. These diverse processes can manifest similarly as a focal abnormality, such as hemiparesis or focal seizures [50]. (See "Ischemic stroke in children and young adults: Epidemiology, etiology, and risk factors", section on 'Infection' and "Cerebral venous thrombosis: Etiology, clinical features, and diagnosis", section on 'Acquired risk factors'.)

Developmental delay and intellectual disability — Survivors of childhood meningitis are at increased risk for developmental delay, learning difficulties, and intellectual disability [2,3]. This is true even for those who do not have acute neurologic complications during the acute illness. Parents/caregivers and schoolteachers should be aware of possible speech and language delays and problems understanding language-based material [35,51]. Early identification and intervention may help to minimize the long-term impact of these problems [52]. (See "Developmental-behavioral surveillance and screening in primary care", section on 'Early intervention or special education services'.)

Intellectual disability is a well-recognized complication of bacterial meningitis in children and can range from mild to severe [13,53-59]. However, few studies have utilized appropriate controls and sufficient follow-up to assess the risk. (See "Intellectual disability (ID) in children: Clinical features, evaluation, and diagnosis".)

Several studies have evaluated the intelligence quotient (IQ) of survivors of bacterial meningitis compared with their siblings or other control children [35,51,60,61]. Although not all of the studies found a difference in the mean IQ compared with controls (usually siblings), a greater proportion of children who had meningitis had IQ <70 [35,53,61].

In a report of a cohort of 130 children who were evaluated 7 and 12 years after meningitis, after adjustment for sociodemographic variables, scores on measures of intelligence, learning, and neuropsychologic skills were consistently below those of age- and grade-matched controls, even though meningitis survivors achieved scores within the normal range [13,53]. Onset of meningitis before 12 months of age was associated with decreased performance on tests requiring language and executive skills [13]. The occurrence of acute neurologic complications and duration of symptoms before diagnosis were not predictors of outcome at 12 years.

In a case-control study of a British cohort of 461 teenagers who had bacterial meningitis in infancy and 289 matched controls (recruited when the cases were five years old), 8 percent of cases attended special schools (compared with none of the controls); this rate is more than four times the national average [54,58]. One-quarter of cases did not pass any General Certificate of Secondary Education examination, compared with 7 percent of controls.

In a population-based Danish cohort, by age 35 years, survivors of childhood meningitis were less likely to have completed high school, attain higher education, or achieve economic self-sufficiency than the comparison cohort [62].

Behavioral and emotional problems — Survivors of childhood bacterial meningitis may have increased risk of behavior and emotional problems over time (eg, attention deficit hyperactivity disorder [ADHD], mood disorders, and social difficulties) [2,3,55,56,63,64]. Whether the frequency of behavior problems varies according to pathogen is not clear; one retrospective review found the prevalence of behavior problems among survivors of non-*H. influenzae* type b meningitis to be similar to that in the general population [65].

UNUSUAL COMPLICATIONS

A variety of unusual neurologic complications can occur in children with meningitis. These unusual complications are rare and include:

- Spinal cord complications, such as transverse myelitis or infarction, presumably as a direct result of local vascular changes with secondary cord ischemia [66-68] (see "Transverse myelitis: Etiology, clinical features, and diagnosis")
- Brain abscesses, which are rare in patients with meningitis due to *S. pneumoniae*, *H. influenzae*, and *N. meningitidis* but occur with higher frequency with certain rare pathogens, such as *Enterobacter sakazakii* (also known as *Cronobacter* [69]) or *Citrobacter* spp [39] (see "Pathogenesis, clinical manifestations, and diagnosis of brain abscess")
- Severe permanent hydrocephalus (see "Hydrocephalus in children: Physiology, pathogenesis, and etiology", section on 'CNS infections')
- Aneurysm formation of focal intracranial vessels, presumably secondary to inflammatory changes in the blood vessel wall [70]
- Cortical visual impairment [48,71-73]

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Bacterial meningitis in infants and children".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or email these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient education" and the keyword[s] of interest.)

- Basics topic (see "Patient education: Bacterial meningitis (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Incidence and risk factors** – Permanent neurologic sequelae occur in up to one-half of children with bacterial meningitis.

  A major risk factor for adverse outcomes is the duration and/or progression of illness before effective antibiotic therapy. Other risk factors include age less than 12 months and pneumococcal infection. (See 'Risk factors for adverse outcomes' above.)

- **Acute seizures and epilepsy** – Seizures occur in 20 to 30 percent of children with acute bacterial meningitis. Seizures that are prolonged, are difficult to control, or begin more than 72 hours after hospitalization are more likely to occur with other major neurologic sequelae; such patients are also more likely to develop epilepsy. (See 'Seizures' above.)

  Seizures should be managed with antiseizure medication. (See "Seizures and epilepsy in children: Initial treatment and monitoring", section on 'Acute symptomatic seizure'.)

- **Hearing loss** – Permanent sensorineural hearing loss occurs in 5 to 10 percent of children with bacterial meningitis overall and up to 30 percent of those with pneumococcal meningitis. (See 'Hearing loss' above.)

- **Other long-term sequelae** – Survivors of childhood meningitis are at increased risk for developmental delay, learning difficulties, and behavioral problems, even if they did not have acute neurologic complications or physical sequelae. (See 'Developmental delay and intellectual disability' above.)

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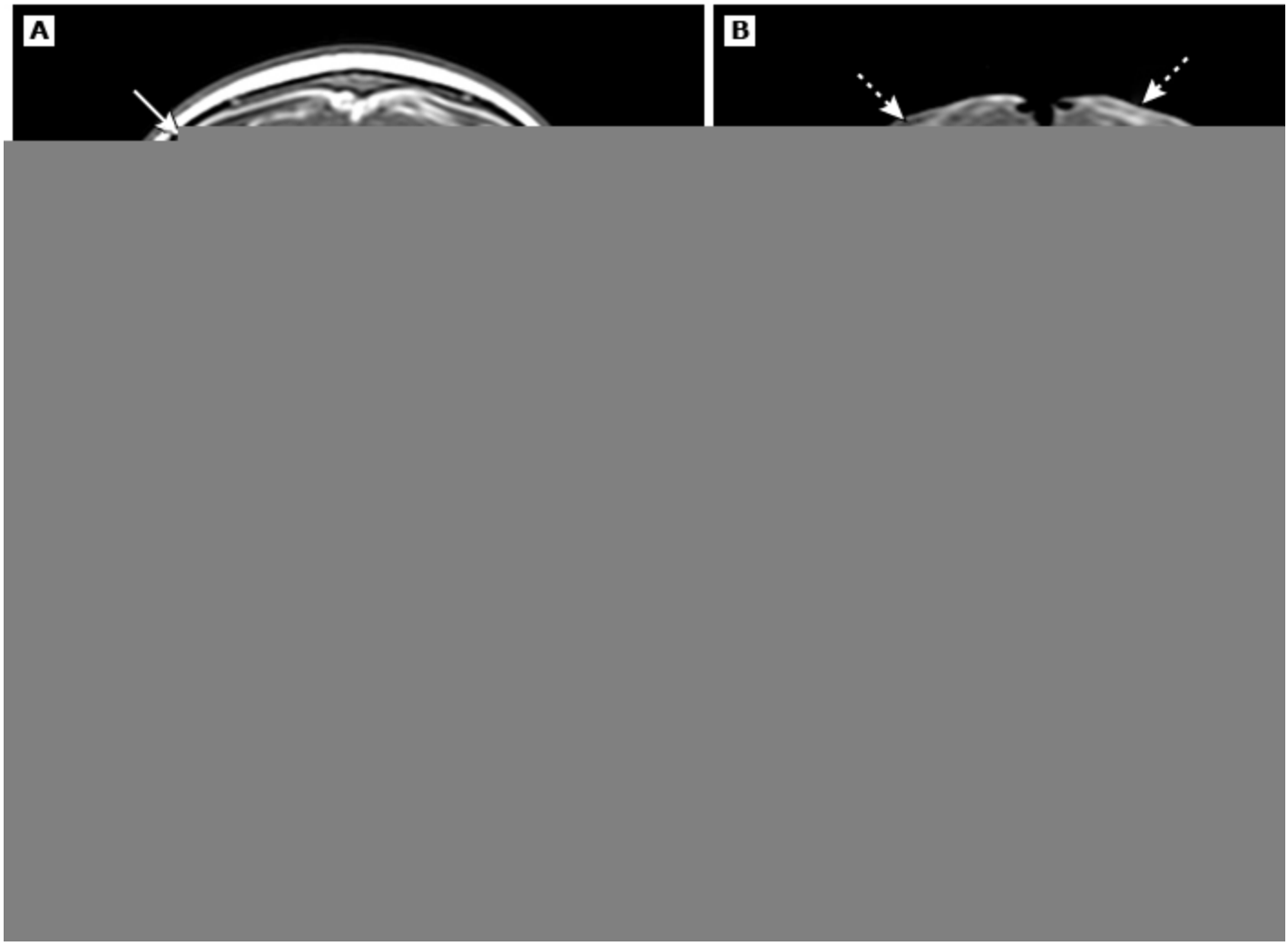
Topic 6000 Version 21.0

GRAPHICS

Magnetic resonance images of bilateral subdural empyema in a child with *Haemophilus influenzae* meningitis



(Panel A) Post-contrast T1 weighted axial image demonstrating leptomeningeal and dural enhancement (arrows) with hypointense subdural collection.

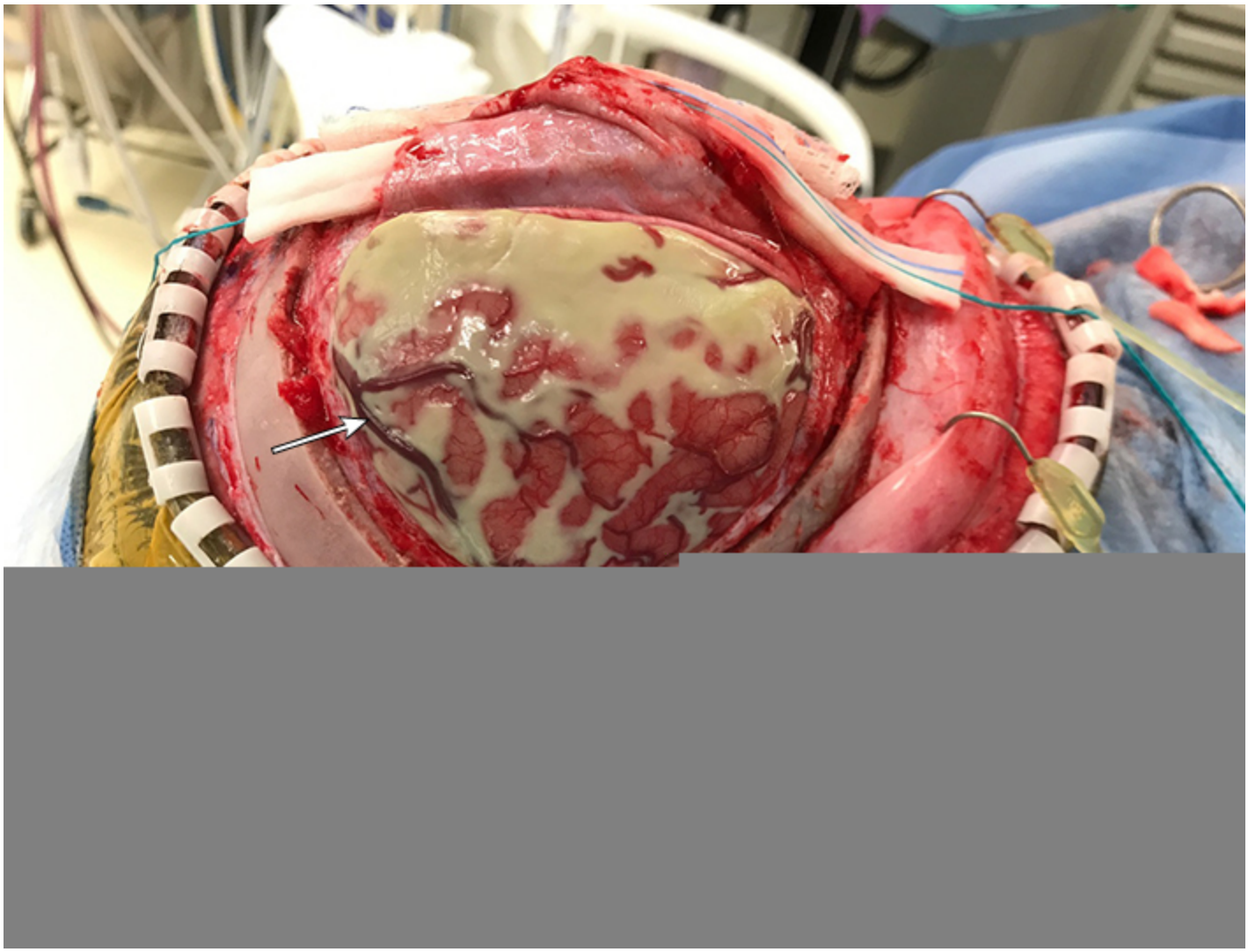
(Panel B) FLAIR axial image demonstrating hyperintense proteinaceous subdural collections (dashed arrows) overlying bifrontal lobe surfaces.

FLAIR: fluid-attenuated inversion recovery.

Courtesy of Sherri B Birchansky, MD, Texas Children's Hospital and Baylor College of Medicine.

Graphic 117506 Version 2.0

Bifrontal craniotomy for drainage of bilateral subdural empyema



Intraoperative photograph of a child undergoing bifrontal craniotomy for drainage of bilateral subdural empyema. Note the extensive purulent material over the surface of the exposed brain. Thrombosed superficial veins are also visible (arrow). The child developed subdural empyema as a complication of *Haemophilus influenzae* meningitis.

Courtesy of Sandi K Lam, MD, MBA, Texas Children's Hospital and Baylor College of Medicine.

Graphic 117505 Version 1.0

Contributor Disclosures

Sheldon L Kaplan, MD Grant/Research/Clinical Trial Support: Pfizer [Streptococcus pneumoniae]. Other Financial Interest: Elsevier [Textbook honoraria – Pediatric infectious diseases]. All of the relevant financial relationships listed have been mitigated. **Morven S Edwards, MD** Other Financial Interest: Texas State University personal services agreement [Chagas disease]. All of the relevant financial relationships listed have been mitigated. **Douglas R Nordli, Jr, MD** No relevant financial relationship(s) with ineligible companies to disclose. **Alejandro A Rabinstein, MD** Grant/Research/Clinical Trial Support: Chiesi [Clevidipine infusion for ICH and hypertension]. Consultant/Advisory Boards: AstraZeneca [Secondary stroke prevention; anticoagulation reversal]; Brainomix [AI for stroke diagnostics]; Novo Nordisk [Stroke risk]; Shionogi [Stroke neuroprotection]. Other Financial Interest: Boston Scientific [Adverse event adjudication committee member for stroke risk reduction device in patients with atrial fibrillation]. All of the relevant financial relationships listed have been mitigated. **Janet L Wilterdink, MD** No relevant financial relationship(s) with ineligible companies to disclose. **Carrie Armsby, MD, MPH** No relevant financial relationship(s) with ineligible companies to disclose.

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Conflict of interest policy