Intracranial Infection Mimics Acute Stroke in an Adolescent Male

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CE Earn Up to 7.5 Hours. See page 468.

An 18-year-old male adolescent was brought to the emergency department by ambulance from a residential substance use disorder treatment program. His chief complaint was weakness. Medics reported that approximately 2 hours prior to arrival at the emergency department, the patient told program staff that he had weakness in his right hand and a headache. The patient maintained normal vital signs during transport and demonstrated no objective neurologic deficits. The patient's medical history included polysubstance use disorder (alcohol, marijuana, and benzodiazepines), migraine headaches, and a recent sinus infection. The patient reported that he recently completed treatment for the sinus infection with an unknown antibiotic. The medics reported that the patient had no known use of tobacco products, inhalants, cocaine, or methamphetamines but that previously he may have used IV drugs. The patient had no known history of immunosuppression, hospitalizations, surgeries, or trauma. Current medications included acetaminophen, ibuprofen, and gabapentin for chronic headache pain.

In triage, the patient's vital signs were as follows: blood pressure, 120/72 mm Hg; heart rate, 95; respirations, 20 breaths per minute; oxygen saturation via pulse oximetry, 100% on room air; and oral temperature, 36.8°C. The triage nurse noted that the patient was stable, cooperative, and tearful, with a Glasgow Coma Scale score of 15.

The primary emergency nurse assumed care 10 minutes after the triage assessment. The primary nurse observed the patient lying awake on a stretcher with an anxious affect. The patient made intense eye contact and followed the nurse with his gaze but was unable to state his name or provide any historical information. The patient only responded with a limited verbal response of "yes" to several questions during this initial assessment. The patient's behavior represented a significant mental status change from the triage assessment. The patient's Glasgow Coma Scale score was now 13, compared with 15 during triage.

The primary nurse was aware that the differential diagnosis for patients presenting with altered mental status in the emergency department is broad and includes a wide range of pathologic, toxicologic, traumatic, and psychogenic conditions.[1] This patient's acutely altered mental status strongly suggested an underlying organic pathology, substance intoxication, or psychogenic condition. Substance intoxication, acute infection, and neurologic illnesses are frequent etiologic factors for altered mental status in children presenting to the emergency department.[1] Substance intoxication is the most frequent primary cause of altered mental status in adolescents, but not in children younger than 12 years.[1]

Further inspection of the patient revealed no obvious signs of trauma or incontinence to suggest injury or seizure. Pupils were equal and reactive bilaterally. The cardiopulmonary physical examination was normal without evidence of heart murmur or respiratory impairment. He had no neck rigidity, petechial rash, or skin lesions to indicate an underlying infectious pathology. A random blood glucose level was in the normal range.

Stroke Screening

The primary nurse performed emergent stroke screenings, including the BE-FAST (Balance, Eyes, Face, Arms, Speech, and Time) mnemonic and the vision, aphasia, neglect (VAN)

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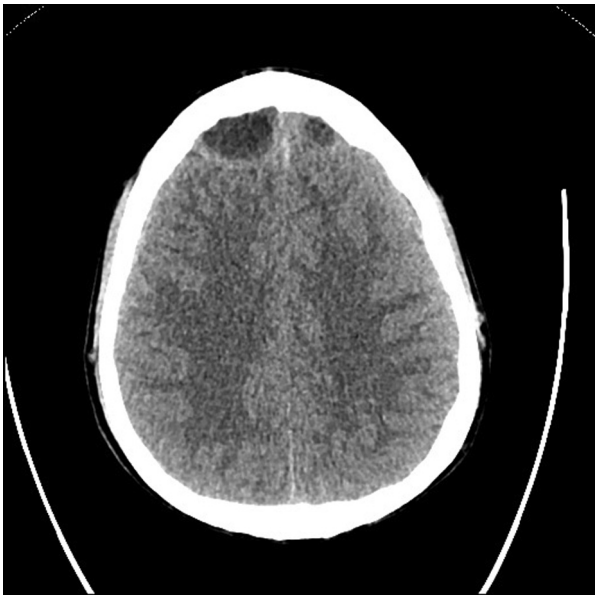

FIGURE 1

Computed tomography6 image showing a 10.6-cm bifrontal lentiform collection suspicious for epidural abscess with mass effect.

assessment. The BE-FAST mnemonic is a quick stroke screening tool that is useful in identifying focal motor weakness indicative of cerebral stroke and also is inclusive of balance and vision symptoms suggestive of potential stroke of the vertebrobasilar circulation of the posterior fossa.[2] VAN (https://www.strokevan.com/van-test) is a novel stroke screening tool.[3] VAN is an experimental tool used to reduce door-to-computed tomography (CT) angiography times and to increase the identification of patients eligible for endovascular therapy who present to the emergency department with emergent proximal large vessel occlusion.[3] In this case, the patient's neurologic examination revealed profound expressive aphasia, severe right-sided weakness, and poor right hand grasp. Left arm and leg extremity strength was fully intact. Given the patient's rapid neurologic deterioration and positive VAN screening, the nurse immediately activated the stroke team per the hospital protocol.

The neurologist's initial examination revealed a National Institutes of Health Stroke Scale score of 8.[4] Results of the National Institutes of Health Stroke Scale can range from 0 to 42, with a score of 5 to 15 indicating moderate stroke symptoms and scores greater than 20 indicating severe stroke symptoms.[4] Urgent head and neck CT, performed within 15 minutes from stroke activation, revealed a 10.6-cm frontal fluid collection of concern for intracranial epidural abscess with dural sinus thrombosis and significant underlying mass effect on brain parenchyma without intracranial hemorrhage (Figure 1). Additionally, the patient had extensive paranasal sinus disease, with erosion of the posterior wall of the right frontal sinus creating communication with the epidural space. Perfusion CT showed decreased arterial perfusion in the left frontal lobe without a clear occlusion. As an infectious etiology became evident, the patient's afebrile finding in triage needed to be interpreted along with the patient's report to the medics of over-the-counter acetaminophen and ibuprofen use for headache, with unknown timing of the last dose.

The patient's laboratory results were significant for a mildly elevated white blood cell count and significantly elevated erythrocyte sedimentation rate and C-reactive protein. The stroke team discussed the administration of IV alteplase (r-tPA). Ultimately, the r-tPA was not administered because the proximate cause of the patient's neurologic deficits was an intracranial infection. Further, because of the elevated risk of bleeding, administration of r-tPA would preclude urgent neurosurgical intervention.

Intracranial Infection

After CT imaging, the patient's temperature was 38.2°C rectally. Broad-spectrum IV antibiotics were indicated for both the patient's intracranial infection and presumed frontal sinus osteomyelitis. Ceftriaxone, 2 g, vancomycin, 1 g, and metronidazole, 500 mg, were prescribed and expeditiously initiated. Magnetic resonance imaging (MRI) and magnetic resonance angiography confirmed extension of the epidural infection into the subdural space.

Intracranial infection is an uncommon yet life-threatening sequelae of acute bacterial sinusitis.[5,6] Acute viral or bacterial sinusitis occur as a complication of upper respiratory infection in about 5% to 10% of pediatric patients.[5] Intracranial infections occur in an estimated 10% of children hospitalized with acute bacterial sinusitis. The frontal sinuses are most frequently involved, with the development of sinogenic intracranial infection.[5] Epidural abscesses are pus accumulations in the potential space between the skull and fibrous dura mater.[5,6] Predisposing factors to the development of intracranial infections include systemic infection (eg, endocarditis and bacteremia); mechanical disruption of the cranial and facial bones due to surgery, trauma, or localized infection; and immunosuppression.[7] The clinical presentation of sinus-related intracranial infection may be insidious and often is characterized by the persistence of nonspecific symptoms including headaches, fever, nausea, and

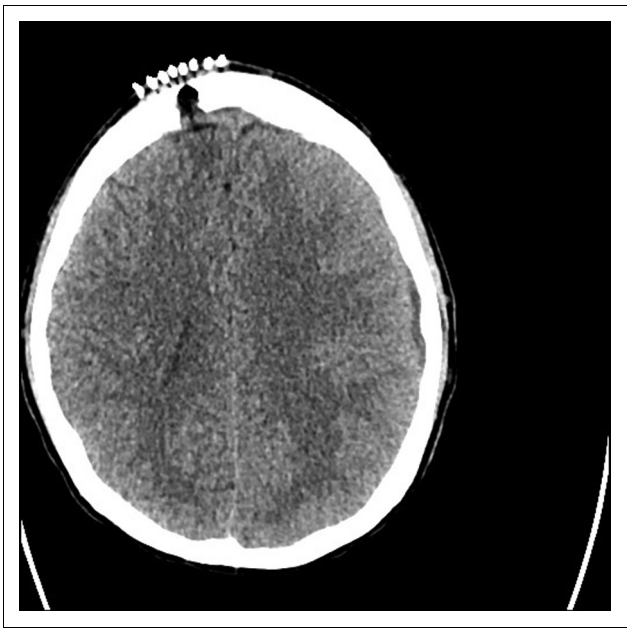

FIGURE 2

Computed tomography image following frontal epidural abscess evacuation without residual abscess.

vomiting.[5] Headache is the cardinal presenting symptom of brain abscess, whereas fever and altered mental status often are absent.[7] Children with sinus-related intracranial infection may not exhibit purulent rhinorrhea or other nasal symptoms that frequently are seen with sinusitis.[5] Nonfocal symptoms increase diagnostic challenges for health care practitioners in recognizing intracranial infection, especially in young children who are unable to verbalize symptoms or in patients who are poor historians.[7] Focal neurologic deficits, altered mental status, and seizures occur less frequently with sinogenic intracranial infection than with other forms of intracranial infection.[5] The presence of fever with symptoms of increased intracranial pressure (such as headache, nausea, vomiting, visual changes, lethargy, and irritability in children) should raise suspicion in the ED clinician for possible intracranial infection.[5-7] Common abnormal laboratory findings with intracranial infections include nonspecific elevations in inflammatory markers (erythrocyte sedimentation rate and C-reactive protein) and an elevated white blood cell count with a predominant neutrophilia.[5]

Sinogenic intracranial infection can develop via bloodborne spread of pathogens or by direct communication between the infected sinus cavity and the intracranial space and tissues. Contiguous spread of infection from the sinuses is frequently caused by *Streptococcus* species, often with polymicrobial involvement. Common causative pathogens include *Streptococcus sanguis, Streptococcus mutans, Streptococcus pneumoniae*, *Enterobacteriaceae*, *Staphylococcus aureus*, and anaerobes (*Prevotella*, *Bacteroides*).[7,8] Empiric antibiotic therapy for intracranial infection includes a third- or fourth-generation cephalosporin combined with metronidazole.[7,8] Vancomycin is added for clinical suspicion of staphylococcal co-infection.[7,8] Glucocorticoid therapy may reduce edema, but data showing benefit are lacking.[8] Although CT imaging with contrast offers a quick and readily available mode of detection of brain abscess, MRI should be performed to further assess the extent of intracranial infection and the presence of any associated complications.[7] Prompt initiation of antibiotic therapy and neurosurgical intervention are crucial interventions to prevent rapid increase in brain abscess volume with possible brain herniation, sepsis, and death.[5,7,9] Initiation of antibiotic therapy is warranted as soon as there is clinical suspicion of brain abscess, because treatment delay can worsen patient outcomes.[7] Acute management of patients with sinogenic intracranial infection requires skillful coordination of care by emergency nurses, emergency prescribing providers, neurosurgeons, infectious disease specialists, and otolaryngology providers.[5]

Intracranial complications of pediatric sinusitis include subdural empyema, epidural abscess, cerebral abscess, and meningitis.[5] Less frequently observed complications are cavernous sinus thrombosis, frontal bone osteomyelitis (Pott's puffy tumor), encephalitis, and cerebral infarct.[5] Younger children are more likely to experience orbital infections related to sinusitis, whereas adolescents experience more intracranial complications, possibly because of rapid frontal sinus growth in adolescence and increased frequency of frontal and anterior ethmoid infections.[5,9] The prevalence of intracranial complications of pediatric sinusitis is highest in adolescent boys, although exact causation is unknown.[5]

The mortality rate of intracranial infections in youth (<18 years) is 3% to 6%, whereas the rate of long-term sequelae is notably higher, ranging from 7% up to 27% to 35%.[5,8] Morbidity increases with delayed recognition and treatment, co-occurring orbital involvement, dural sinus thrombosis, sepsis, meningitis, and other secondary infections.[5,6,9] Potential long-term neurologic sequelae of intracranial infection in children include epilepsy, hearing loss, visual deficits, hemiparesis, aphasia, cranial nerve palsies, deficits of learning and cognition, and developmental delay.[9]

The management of orbital and intracranial complications of acute sinusitis in pediatric patients are associated with significant health care costs.[6] In one hospital review, the mean cost of inpatient care related to pediatric sinusitis complications was more than $20,000 (United States

dollars) per patient because of surgical interventions, hospital pediatric intensive care admissions, and pharmacy costs.[6] Early recognition of complications remains paramount to improve long-term pediatric health outcomes.[6,9] Globally, pediatric complications of acute bacterial sinusitis occur at higher rates in developing countries and among medically compromised and socioeconomically disadvantaged groups; further research is needed to quantify impact and incidence in vulnerable populations.[5,9]

Patient Case Disposition

Approximately 5 hours after the initial ED presentation, following urgent MRI/magnetic resonance angiography and resource mobilization, the patient was taken to the operating suite where neurosurgeons performed an urgent burr hole craniotomy to evacuate the epidural abscess and subdural empyema. Sinus surgery was subsequently performed to wash out and debride the affected sinuses. Antibiotic therapy was continued in the ICU while awaiting abscess and blood culture findings. Anticoagulation therapy was initiated, once doing so was safe from a surgical perspective, with administration of IV heparin to treat sagittal sinus thrombosis. Repeat head imaging demonstrated resolution of the abscess with no recurrence of the fluid collection (Figure 2). Epidural abscess cultures collected during surgery isolated *Streptococcus intermedius*. Blood cultures had one bottle detect positive findings for *S intermedius*. Transthoracic echocardiography ruled out structural heart defects, endocarditis, or cardiac valve vegetations.

The patient's neurologic deficits fully resolved during acute hospitalization. A peripherally inserted central catheter was placed prior to discharge, and the patient was transferred to a rehabilitation facility to complete 6 weeks of antibiotic therapy with IV ceftriaxone and oral metronidazole, along with anticoagulation with apixaban. Because the patient was at high risk for secondary meningitis as a result of dehiscence of the posterior wall of the right frontal sinus, he was vaccinated to prevent against invasive *Streptococcus pneumoniae, Haemophilus influenzae* type B, and *Neisseria meningitidis* infections.

Emergency Nursing Implications

Clinical recognition of intracranial complications of sinusitis may be delayed, leading to increased patient morbidity and potential catastrophic sequelae. Children and adults who are unable to adequately communicate symptoms or have poor access to health care are at increased risk for serious complications. When persons treated for acute bacterial sinusitis experience unresolved headaches, fevers, or constitutional symptoms, clinical concern should be raised for sinogenic intracranial infection, including brain abscess; these patients warrant timely re-evaluation.[5,7,9] Intracranial infections also may clinically mimic acute stroke. Other conditions that often mimic stroke include hypoglycemia, Todd's paralysis, seizure, cerebral neoplasm, complex migraine, substance intoxication, peripheral vestibular disorder, and conversion disorder.[10]

Emergency nurses should become familiar with the use of emergent stroke screening tools and consider stroke in patients of any age who present with sudden onset of altered mental status or new focal neurologic signs and symptoms. Serial neurologic assessments by emergency nurses are essential to identify early clinical deterioration, guide appropriate interventions, and improve patient outcomes. The emergency advanced practice registered nurse should maintain current knowledge of historical and clinical risk factors for bacterial sinusitis, intracranial infection, and stroke in children and adults; conduct a thorough patient history, review of systems, and physical examination; formulate relevant differential diagnoses; and follow institutional and evidence based diagnostics and clinical management guidelines in collaboration with the supervising emergency physician.

Emergency nurses who discharge patients with acute bacterial sinusitis should stress the importance of completing all antibiotic doses and emphasize that the patient should immediately return to the emergency department if symptoms worsen or do not fully resolve. Emergency nurses also should teach parents to eliminate child exposure to secondhand smoke and advise them to fully vaccinate their children. Free, evidence-based, and easy to read teaching literature for patients and families regarding appropriate antibiotic use and prevention, recognition, and management of sinus infections is available at https://www.cdc.gov/antibiotic-use/community/for-patients/common-illnesses/sinus-infection.html.[11]

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