Objective: Closed head injury rarely predisposes patients, particularly children, to the development of dural sinus thrombosis. In addition, most cases of sinus thrombosis are subacute in nature. The following is a case report of a precipitous course of dural sinus thrombosis after closed head injury in a pediatric trauma patient.

Description: A 14-year-old African American girl presented to the emergency department as a trauma activation. She was an unrestrained rear-seat passenger involved in a motor vehicle collision during which she was ejected. She experienced loss of consciousness and was found unresponsive at the scene. She was intubated and then transported via helicopter to the level I trauma center. Initial head computed tomography (CT) revealed no acute intracranial abnormality. The head CT was repeated 15 hours later when nursing noted the patient to be acutely hypertensive and tachycardic with an 8-mm nonreactive right pupil. The repeated CT showed interval development of extensive dural sinus thrombosis, cerebellar edema and infarct, as well as mild downward transtentorial herniation. Because of multiple coexistent injuries and complications, treatment of the dural sinus thrombosis was difficult. The hospital course was complicated and there was very little improvement in the patient's condition.

Conclusions: The multisystem injured trauma patient poses many clinical challenges. Treatment of dural sinus thrombosis is difficult and controversial and requires an investigation into possible risk factors for a hypercoagulable state. Clinical outcomes vary from excellent to dismal.

Key Words: dural sinus thrombosis, pediatric head injury, pediatric trauma

CASE

A morbidly obese 14-year-old African American girl presented to the emergency department as a trauma activation. She was an unrestrained rear-seat passenger involved in a motor vehicle collision during which she was ejected and severely injured. At the scene, the patient was unconscious and responsive only to painful stimuli. Cervical spine stabilization and immobilization were instituted, intravenous access was obtained, and she was intubated in accordance with the rapid sequence intubation protocol of the emergency medical services. She was then transported via helicopter to the level I trauma center. According to emergency medical services providers, the patient had no significant medical/surgical history, she was not taking any medications, and she had no known allergies. Social history and family history were unknown. The patient was unable to provide any history.

On arrival to the emergency department, the patient was assessed in accordance with the standard advanced trauma life support protocol. The patient’s initial Glasgow Coma Scale was 3TP (eye, 1; verbal, 1; motor 1; intubated; and paralyzed). Her pupils were noted to be equal and round as well as reactive to light. The remainder of the physical examination was notable for a 4-cm laceration on the right frontal scalp, a 1.5-cm laceration on the left eyelid, a large abrasion on the left side of the face, abrasions on all extremities, a deep wound on the right upper extremity, an absent left radial pulse, and an obvious deformity with an open fracture of the left radius and ulna. Her initial vital signs were rectal temperature of 37.5°C, blood pressure of 193/113 mm Hg, heart rate of 116 beats per minute, respiratory rate of 20 breaths per minute, and an oxygen saturation of 100%. She was given intravenous fluids, pain medication, and sedation medication, with return of vital signs to acceptable limits.

After the initial assessment and stabilization, the patient underwent multiple radiographic studies. The initial head computed tomography (CT) revealed no acute intracranial abnormality, bilateral frontal scalp lacerations, bilateral parietal scalp soft tissue thickening, mild asymmetric prominence of the right lateral ventricle without hydrocephalus, and no calvarial fracture. Cervical spine CT was negative for any fracture. Computed tomography of the abdomen and pelvis demonstrated a grade 3 to 4 spleen laceration, free fluid in the pelvis, a questionable left kidney laceration versus infarction, and bilateral parenchymal lung densities. Chest radiograph demonstrated right upper lobe collapse versus opacification. Multiple films of all extremities revealed a fracture of the middle one third of the left ulna and radius as well as a Salter II fracture of the left radius.

The patient was admitted to the pediatric intensive care unit (ICU) where she remained intubated and received supportive therapy, monitoring, and interventions as appropriate. Suddenly, after 15 hours, she developed hypertensive emergency with a blood pressure of 240/120 mm Hg, tachycardia to 216 beats per minute, and a dilated right nonreactive pupil. She emergently underwent repeated CT imaging. Head CT revealed an interval development of a hyperdensity consistent with thrombus throughout the superior sagittal sinus, straight sinus, left transverse sinus to the junction of the left transverse and left sigmoid sinus, as well as right transverse and sigmoid sinuses to the junction of the right sigmoid sinus and the right internal jugular vein. In addition to the dural sinus thrombosis, there was an interval development of edema at the cerebellar hemispheres, the right temporal region, and the right caudate head/internal capsule region. This was consistent with venous infarct/ischemia and was without parenchymal hemorrhage. Mild hydrocephalus and mild downward transtentorial herniation were also noted. Head/neck CT angiography was suboptimal but without evidence for vascular injury. Intracranial pressure monitoring was initiated, and she was treated aggressively with mannitol, 3% saline, and antihypertensive medications. Given her condition and other injuries, she was not a candidate for anticoagulation therapy and interventional radiology thrombolysis was deemed too risky.

The patient experienced extensive dural sinus thrombosis and subsequently had a complicated and protracted ICU stay. Secondary to the combined efforts of multiple specialists, the patient...
left the ICU after 2 months and was eventually transferred to a rehabilitation facility. A repeated head CT scan at that time revealed a progressive patchy encephalomalacia of the cerebral and cerebellar hemispheres as well as the basal ganglia consistent with venous infarction after dural sinus thrombosis. Persistent thrombus was noted within the superior sagittal sinus and the left transverse sinus. She had regained the ability to open her eyes and blink in response to commands but was unable to perform any other functions. Every attempt was made to determine any underlying cause for the thrombosis, but no known disorder of coagulation was discovered in the patient or in her immediate family. Unfortunately, there was very little improvement in her condition and, overall, her prognosis is poor.

**DISCUSSION**

Dural sinus thrombosis, also known as cerebral venous thrombosis (CVT), is a thrombosis or clot within the brain's venous system. This system begins with venules and veins that then drain into the great dural sinuses. Although the first description of CVT occurred in the French literature in 1825, the true incidence is still unknown. The first report of posttraumatic thrombosis occurred in 1915. Lacking good epidemiologic data, it is difficult to accurately determine the true incidence. However, the incidence is likely higher than what was originally thought. Bousser and Ferro stated that it is a “rare type of cerebrovascular disease affecting about 5 people per million and can occur at any age” and that it is responsible for 0.5% of all strokes. Cerebral venous thrombosis seems to be slightly more common in women than in men and there is no apparent predilection for race. Thrombosis is much more common in adults than in children.

Thrombus formation is often multifactorial. The 3 main factors that predispose to clot formation are known as Virchow triad. This triad consists of endothelial injury, stasis or turbulence of blood flow, and blood hypercoagulability. Hypercoagulable states are classified as either genetic or acquired. Known genetic causes include mutations in factor V, antithrombin III deficiency, protein C or S deficiency, fibrinolysis defects, homocysteinemia, and allelic variations in prothrombin levels. Acquired causes include prolonged immobilization, myocardial infarction, tissue damage, cancer, disseminated intravascular coagulation, heparin-induced thrombocytopenia, antiphospholipid antibody syndrome, atrial fibrillation, nephrotic syndrome, hypercoagrogenic states, oral contraceptive use, sickle cell anemia, smoking, advancing age, and obesity. After formation, a clot will propagate, embolize, dissolve via fibrinolytic activity, or recanalize. The significance of a venous thrombus is dependent on its location, and detrimental effects are the result of congestion and edema in the vascular beds distal to the obstruction.

Dural sinus thrombosis is often difficult to diagnose. Signs and symptoms are often vague and nonspecific and may include headache, papilledema, focal neurologic deficit, cranial nerve palsies, seizure, altered mental status, amnesia, or coma. Symptomatic in 35% of patients, subacute (<1 month) onset in 40% of patients, and progressive/chronic onset in 25% of patients. Neuroimaging is required to definitively diagnose CVT. Head CT may be normal in 25% of patients, and magnetic resonance imaging is now considered the criterion standard for diagnosis. Treatment occurs on a case-to-case basis with identification and management of any underlying cause, anticoagulation and antithrombotic therapy as deemed appropriate, as well as symptomatic treatment of elevated intracranial pressure, seizures, etc. Mortality has been reported to be from 5% to 30% with a generally good outcome overall. It has been reported that 15% to 25% of patients will have some lasting deficit. Poor outcomes are more likely to be associated with intracranial hemorrhage, a decreased mental status defined as a Glasgow Coma Score of lower than 9, focal neurologic deficits, seizures, and infarct. It has been reported that up to 80% of patients are functionally independent in the long-term. Dural sinus thrombosis is less common in children than in adults but has been reported in the literature to be secondary to dehydration, metabolic acidosis, sepsis, localized central nervous system infection, and cyanotic heart disease as well as in various hypercoagulable states. Children often have worse functional outcomes.

This case is reported because of the paucity of literature on dural sinus thrombosis in pediatric trauma patients and the complexity of treatment. The etiology of posttraumatic dural sinus thrombosis is unclear. Meena et al and D’Alise et al reported 2 separate cases of young adults with dural sinus thrombosis after head trauma from motor vehicle collisions. Both cases describe a subacute course, with signs, symptoms, and diagnosis occurring approximately 10 days after the initial injury. Rich et al reported in 1993 of a 4-year-old boy who developed thrombosis of the straight, superior sagittal, and transverse sinuses 2 weeks after minor head trauma without loss of consciousness. He was subsequently diagnosed with inherited protein S deficiency. This literature postulates that endothelial damage and intramural hemorrhage secondary to rupture of the sinusoids at the site of entry of the draining veins may initiate the thrombotic process after a closed head injury. It is also postulated that an underlying inherited pathology will potentiate the formation of a thrombus precipitated by trauma. The case presented here is unique for 2 reasons. First, the patient experienced a massive thrombosis of her dural sinus system acutely after an initial negative head CT. The second reason is that a genetic or an acquired predisposition to hypercoagulability was not identified. This patient also reinforces the premise of a case-to-case management. This multisystem trauma patient developed an acute dural sinus thrombosis. Anticoagulation was contraindicated secondary to her other potentially life-threatening injuries, and symptomatic treatment was simultaneously managed by the trauma surgeons, neurosurgeons, neurologists, and pediatric intensivists. Unfortunately, this patient experienced a poor outcome without any clear etiology.

**CONCLUSIONS**

Closed head injury rarely predisposes patients, particularly pediatric patients, to the development of dural sinus thrombosis. In addition, most cases of dural sinus thrombosis are subacute in nature. However, thrombosis formation is unpredictable and the diagnosis should be entertained in any symptomatic patient or in any patient who deteriorates clinically, regardless of the time frame. Treatment of dural sinus thrombosis is difficult and somewhat controversial, and it requires an investigation into possible risk factors for hypercoagulability. The multisystem injured trauma patient poses additional clinical challenges, and each case must be managed individually. Clinical outcomes are not easily predicted and vary from excellent to dismal.

**REFERENCES**


