

Cerebral Venous Sinus Thrombosis

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Abstract: Cerebral venous sinus thrombosis (CVST) is a rare and potentially deadly condition. Common etiologies include hypercoagulable diseases, low flow states, dehydration, adjacent infectious processes, oral contraceptives, hormonal replacement therapy, pregnancy, and puerperium. Symptoms include nausea, seizures, severe focal neurological deficits, coma, and headache (the most common presenting symptom). Anticoagulation is the mainstay of treatment for CVST. Transvenous clot lysis can be performed using injected thrombolytic agents and specialized catheters for clot retrieval.

Cerebral venous sinus thrombosis (CVST) is a rare and potentially deadly condition. Known conditions that increase the risk of CVST include hypercoagulable states, dehydration, adjacent infectious processes, low cerebral blood flow, oral contraceptives, hormone replacement therapy, pregnancy, and puerperium (Benamer & Bone, 2000; Buccino et al., 2001; Chaloupka, Mangla, & Huddlem, 2000; deBruijn, Budde, Teunisse, de Haan, & Stam, 2000; Ekseth, Bostrum, & Vegfors, 1998; Frey, Muro, McDougall, Dean, & Jahnke, 1999; Patel et al., 2003; Soleau, Schmidt, Stevens, Osborn, & MacDonald, 2003). Each of these conditions is associated with a higher risk of venous thrombus formation, but exactly why the cerebral venous sinus system is involved over other veins is unclear (Chaloupka et al.). A specific age group has not been identified as at risk in the literature, and it is not uncommon that a precipitating condition will not be identified (Baker, Opatowsky, Wilson, Glazier, & Morris, 2001; Chaloupka et al.).

As the thrombus enlarges, it causes venous congestion and leads to cerebral edema with mass effect and a resultant increase of intracranial pressure. If the thrombus is untreated, the intracranial pressure continues to rise and the vascular supply is compromised, leading to ischemia. This contributes to worsening of neurological status, frequently at an unpredictable and accelerated rate. Complete obstruction of the venous system can occur with exacerbation of cerebral edema, vascular compression, and brain herniation, leading to death. Monitoring in an

intensive care unit (ICU) may be indicated depending on the person's neurological status and diagnostic results (Benamer & Bone, 2000; Chaloupka et al. 1999; Frey et al., 1999). Medical management begins with systemic anticoagulation therapy with heparin and intravenous (IV) hydration (Baker et al. 2001; Chaloupka et al.; Chow et al., 2000). Direct clot lysis or clot retrieval for rapid recanalization of the affected sinus can now be considered, as a result of recent advancements in interventional neuroradiology (Baker et al.; Benamer & Bone, 2000; Buccino et al., 2001; Buccino, Scoditti, Patteri, Bertolino, & Mancina, 2003; Chaloupka et al.; Chow et al.; Ekseth et al., 1998; Frey et al.).

Intensive care techniques, such as intracranial pressure monitoring, drainage of cerebrospinal fluid, osmotic medications, and barbiturate coma, may be instituted in patients with severe cerebral edema, cerebral infarction, intracranial hypertension, unstable or deteriorating neurological status (Benamer & Bone, 2000; Ekseth et al., 1998). Supportive care is essential for these individuals, including optimizing cerebral oxygenation, promoting venous drainage, providing nutritional support, providing hemodynamic support, and preventing complications. Case studies are presented to demonstrate the range of presenting symptoms, diagnostic evaluation, medical treatment, and nursing care issues.

Pathophysiology

The venous sinus is a cavity formed between the endosteal and meningeal layers of the dura. The sinuses are connected by channels to large cortical veins and together make up the venous system that allows blood to drain from the cranium. Thrombus formation within a venous sinus can create a partial or complete blockage and localized congestion within the venous system and the brain; both are secondary to decreased venous outflow (Chow et al., 2000; Ekseth et al., 1998). Exacerbation of venous congestion causes increased intracranial pressure, massive ischemia, and infarction of cerebral tissue (Ekseth et al.). Hemorrhagic conversion can occur in larger infarctions (Baker et al., 2001).

Symptoms can be vague and nondescript, with either gradual changes or rapid neurological deterioration (Baker et al., 2001; Benamer & Bone, 2000; Buccino et al., 2001; Buccino et al., 2003; Chaloupka et al., 1999; Chow et al., 2000; deBruijn et al., 2000; deBruijn, deHaan, & Stam, 2001; Ekseth et al., 1998; Frey et al., 1999; Patel et al., 2003; Soleau et al., 2003). Because there is limited room for expansion of intracranial components, neurological compromise occurs rapidly. Any increase in intracranial volume will be at the expense of the brain. Complete blockage of the venous

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system leads to compression of cerebral arteries, which is secondary to massive cerebral edema and eventual brain death (Baker et al., 2001).

Clinical Presentation

Presenting symptoms may include nausea, seizures, severe focal neurological deficits, coma, and headache, which is the most common (Baker et al., 2001; Benamer & Bone, 2000; Buccino et al., 2001; Buccino et al., 2003; Chaloupka et al., 1999; Chow et al., 2000; DeBrujin et al., 2000; Ekseth et al., 1998; Frey et al., 1999; Patel et al., 2003; Soleau et al., 2003). Less common symptoms include nystagmus, dysphagia, hearing loss, and cerebellar incoordination (Baker et al., 2001). Symptoms can be very vague and may be overlooked by the individual, resulting in failure to seek medical treatment and the delay of appropriate treatment (Baker et al.; Soleau et al., 2003). The severity of symptoms is directly related to the extent of cerebral venous thrombosis, but the condition of an individual with headache and nausea may rapidly deteriorate as the brain's threshold for compensation is surpassed (Benamer & Bone, 2000; Chaloupka et al.; Ekseth et al.; Frey et al.; Patel et al.).

Papilledema, proptosis, ocular chemosis, cranial nerve III, IV & VI involvement, and focal neurological deficits may be present as symptoms. It is not uncommon for the physical exam to be unremarkable (Buccino et al., 2001; Ekseth et al., 1998; Patel et al., 2003).

Diagnostic Imaging

Computed tomography (CT) of the head is generally the first diagnostic tool to evaluate patients with suspected neurological pathology, even though it lacks the sensitivity and specificity needed to evaluate changes associated with venous sinus thrombosis (Buccino et al., 2003; Chow et al., 2000; Ekseth et al., 1998; Frey et al., 1999; Patel et al., 2004; Soleau et al.). CT venogram (CTV) is useful at identifying changes within the venous system, if available. Magnetic resonance imaging (MRI) is more sensitive and specific to the changes seen with venous sinus thrombosis. The magnetic resonance venogram (MRV) is the diagnostic tool of choice (Buccino et al., 2001; Buccino et al., 2003; Ekseth et al.; Frey et al.; Patel et al.). Cerebral angiography is generally not performed unless MRI and MRV are unavailable, or the patient is unable to have a magnetic resonance scan. Cerebral angiography is the only diagnostic tool that can evaluate the patency of cortical veins (Buccino et al., 2001; Ekseth et al., 1998; Patel et al.).

Treatment

Anticoagulation with heparin is the first line of treatment in CVST (Baker et al.; Buccino et al., 2001; Buccino et al., 2003; Chow et al.; deBrujin et al. 2000; Ekseth et al.). This treatment is aimed at preventing thrombus extension to maintain venous pathways and has been shown to be the most effective treatment modality, though its risks and ben-

efits need to be evaluated on an individual basis (Buccino et al., 2001; Buccino et al., 2003; Ekseth et al.; Soleau et al., 2003). The use of anticoagulation in intracranial ischemia or hemorrhage is controversial, though the extensive use of systemic heparin in the ischemic stroke population has not demonstrated an increased risk of hemorrhagic conversion (Baker et al., 2001; Buccino et al., 2001; Buccino et al., 2003; Chow et al.; deBrujin et al.; Ekseth et al.). Soleau et al. and Buccino et al. (2003), after retrospective review, concurred that in the presence of intracranial hemorrhage, anticoagulation is safe for the treatment of CVST. Treatment not directed at prevention of thrombus extension, clot formation, or clot lysis is ineffective and carries higher risk.

In conjunction with anticoagulation, IV hydration is started to treat dehydration and reduce blood viscosity. Other treatment modalities include corticosteroids or osmotic agents to decrease cerebral edema, sedatives to reduce metabolic needs, and cerebrospinal fluid drainage to decrease intracranial pressure. None of these treatments prevents thrombus propagation or stroke extension. Without anticoagulation, limited improvement in morbidity and mortality is attained (Chaloupka et al., 1999; Chow et al., 2000; Ekseth et al., 1998; Soleau et al., 2003).

Seizure prophylaxis depends on the treating physician. Variables that influence the use of antiepileptic drugs (AEDs) include the presence of intracerebral hemorrhage, location of hemorrhage, and seizure as a presenting symptom (Buccino et al., 2003; Soleau et al., 2003). Buccino et al. (2003) suggested that only recurrent seizures be treated during the acute phase with continuation of AEDs for 1–2 years posttreatment.

Anticoagulation does not treat the acute thrombus; therefore, if there is a severe compromise of venous outflow or occlusion, thrombolytic agents may be used for clot lysis (Chow et al., 2000; Ekseth et al., 1998; Frey et al., 1999; Soleau et al., 2003). Thrombolytic agents, such as recombinant tissue plasminogen activator (tPA), streptokinase, and urokinase, may be used intravenously, though the precise dose that is delivered to the venous sinus is unknown and probably low (Baker et al., 2001; Chow et al.; Ekseth et al.; Frey et al.).

Developments in catheters and interventional neuro-radiology techniques now offer the possibility of venous thrombolysis with either transvenous injection of a thrombolytic agent (tPA or urokinase) into the sinus or using catheters that allow for clot retrieval (Baker et al., 2001; Buccino et al., 2001; Chow et al., 2000; Dowd, Malek, Phatourous, & Hemphill, 1999; Ekseth et al., 1998). The catheter tip is positioned within the thrombosed sinus, and the thrombolytic agent is given in pulsing injections. The starting thrombolytic dose is based on the current dosage being used in acute intraarterial stroke management; if tPA is used, the starting dose is 0.3 mg/kg. The total dose of tPA or other thrombolytics will vary related to the volume of the thrombus and extent of occlusion. Frequently the total

dose is dependent on the comfort of the treating physician, because there is limited research and clinical data on precise dosing.

Transvascular mechanical thrombectomy can be performed using a specialized catheter that provides a vacuum to pull or extract the clot or a micro-guidewire that can assist in clot destruction. Both systems may be used in combination with thrombolytic agents (Baker et al., 2001; Benamer & Bone, 2000; Chaloupka et al.; Chow et al., 2000; Dowd et al., 1999; Frey et al., 1999). Micro-balloons have also been used in venous sinus stenosis and may provide an additional tool in the treatment of CVST by assisting in sinus recanalization (Baker et al.; Chaloupka et al.).

It is not known whether there is a critical window for the timing of thrombolysis or the extent clot lysis needed to improve clinical outcome (Buccino et al., 2001; Chaloupka et al., 1999; Chow et al., 2000). Chaloupka et al. noted clinical improvement in cases where partial venous sinus recanalization was attained. Such improvement suggests that partial recanalization may be adequately beneficial, to the extent that the procedure duration and the total thrombolytic dose administered may both be reduced, thereby decreasing hemorrhage risk.

Identification of venous sinus thrombosis etiology is essential in defining additional treatment and identifying risk factors.

Interventional therapies are not without risks and include vessel wall injury or rupture with subsequent hemorrhage, vessel dissection, femoral artery pseudoaneurysm, hematoma, thrombus advancement, or ischemic stroke extension (Baker et al., 2001; Chaloupka et al., 1999). The overall risk of intracerebral hemorrhage with transvenous thrombolysis is relatively low, though it is not absent (Chaloupka et al.).

Surgical thrombectomy with local use of thrombolytic agents can be performed, but it is considered high risk (Ekseth et al., 1998). Craniotomy is generally performed around the vertex, allowing for access to all portions of the superior sagittal sinus, though careful intraprocedural positioning is required to adequately position the head above the heart to promote venous drainage while minimizing the risk for air emboli formation (Ekseth et al.). Small catheters can be introduced into the thrombus for suction and injection of thrombolytic agents; mechanical removal of the thrombus may be done using small forceps (Ekseth et al.). Both techniques require special care to avoid dislodging the thrombus into the venous system (Ekseth et al.).

Timely diagnosis is essential for providing appropriate treatment and limiting morbidity. Determining the most appropriate treatment modality is difficult and no specific criterion exists to indicate which invasive intervention should be initiated first or to determine which individu-

als are at risk for thrombus extension (Baker et al., 2001; Buccino et al., 2001; Chaloupka et al., 1999; Chow et al., 2000). In general, medical management is instituted promptly and includes anticoagulation, hydration, and careful neurological monitoring. The timing of thrombolysis or surgical treatment depends on clinical status and the degree of venous thrombosis (Buccino et al.; Chaloupka et al.; Chow et al.; Ekseth et al., 1999). Abrupt neurological deterioration or advancing thrombosis on either an MRI or MRV may be critical indicators that invasive intervention is warranted (Chaloupka et al.).

Identification of venous sinus thrombosis etiology is essential in defining additional treatment and identifying risk factors. Common etiologies include hypercoagulable states, dehydration, adjacent infectious processes, oral contraceptives, hormonal replacement therapy, pregnancy, and puerperium. Workup involves obtaining a coagulation panel to rule out hypercoagulation states; reviewing CT or MRI to rule out mastoiditis and intracranial abscess; obtaining sodium, potassium, creatinine, blood urea nitrogen, hematocrit, and hemoglobin levels to rule out dehydration; and reviewing current medications and recent medical conditions to rule out etiology related to hormone use, pregnancy, or puerperium. Additional treatment should be aimed at treating primary etiology.

Prognosis

Morbidity is an estimated 15%–25% with mortality at 10%–50% (Baker et al., 2001; Benamer & Bone, 2000; Buccino et al., 2001; Chow et al., 2000; Ekseth et al., 1998; Frey et al., 1999). Individuals who demonstrate neurological compromise at the time of diagnosis have a significant risk for long-term deficits, though those with venous strokes have demonstrated a greater potential for recovery than arterial strokes (Benamer & Bone; Buccino et al., 2003; Chow et al.; deBruijn, deHaan, & Stam, 2001; deBruijn et al., 2000; Ekseth et al.; Patel et al., 2003; Soleau et al., 2003).

DeBruijn et al. (2000) evaluated 57 individuals 1 year after CVST was treated with systemic anticoagulation and found that 44% had persistent cognitive deficits, 6% were dependent for all care, 40% were unable to return to their previous occupations and mortality overall was 13%. Buccino et al. (2003) evaluated individuals 1–3 years after CVST. In their series, all but two individuals were treated with anticoagulation ($N = 34$). They found that 12% had mild residual hemiparesis or hyperreflexia, 9% demonstrated mild nonfluent aphasia, 18% demonstrated persistent memory problems, 18% showed depression by Beck Depression Inventory, 9% had persistent seizures, and 29% complained of persistent headaches. Both studies lack information correlating outcomes to presenting neurological exam and extent of venous involvement.

Soleau et al. (2003), in a retrospective review, found that overall clinical improvement was noted in 60% of individuals undergoing thrombolytic therapy compared

to 88% with mechanical thrombectomy. Thrombolytic therapy was the most effective for the immediate restoration of venous sinus patency, though 30% had hemorrhagic complications (Soleau et al.).

Indicators of poor prognosis include deep sinus involvement, extension of thrombus into cortical veins, coma as the presenting symptom, rapid rate of symptom progression, more than 10-day delay in diagnosis, hemorrhage on initial CT scan, and presence of papilledema (Buccino et al., 2003; Chaloupka et al., 1999; Chow et al., 2000; deBruijn et al., 2000; deBruijn et al., 2001; Ekseth et al., 1998). Those not treated with anticoagulation also fared worse. Better prognoses were demonstrated in individuals who presented with headache and papilledema alone and who had rapid venous sinus recanalization or development of collaterals (Benamer & Bone, 2000).

Nursing Care

Whether the individual requires admission to the ICU or an acute neurosurgical ward, ongoing astute neurological assessment by the nurse is essential. Subtle and abrupt neurological changes can indicate further compromise to the venous system and the brain, signaling the nurse to alert the physician to coordinate prompt diagnostic workup or adjust treatment.

Ongoing anticoagulation monitoring is required with timely rate adjustments to maintain partial thromboplastin time (PTT) within prescribed goal rate. Adjustments should be made per physician order or hospital policy. Again, use of anticoagulation and target PTT values vary. Variables that influence the use of anticoagulation include physician level of comfort, extent of thrombus, neurological status, and presence of intracerebral hemorrhage. If anticoagulation is used and an acute neurological decline is noted, the anticoagulation should be stopped immediately and not restarted until intracranial hemorrhage is ruled out.

Education is ongoing as it relates to treatment, etiology, and long-term preventative management. If a hypercoagulable state is identified the patient may require long-term anticoagulation and education should focus on the specific disorder and risks and benefits of treatment or lack of treatment. If warfarin is initiated the patient needs to be educated on bleeding precautions, potential food interactions and importance of careful monitoring of international normalized ratio (INR) while on the warfarin. If

hormonal replacement therapy or contraceptives are identified as the only risk factors the female patient should be counseled on the risks of continued hormonal therapy and offered alternative treatment for hormonal replacement or birth control.

Supportive equipment ranges from endotracheal tube placement for airway management, ventilators, nasogastric tubes, nutritional support, sequential stockings, and skin-care to integration of therapy at the bedside. Thrombosis of the venous sinus can cause headaches that can be intense and may require use of analgesics or narcotics. Medications used and dosage is physician dependent. From a global perspective, the nurse coordinates all aspects of supportive care for the individual, aiming at the prevention of complications related to immobility, malnutrition, neurological deficits, and pulmonary compromise.

Case Study 1

Robert is a 39-year-old male who presented with a 5-day history of severe headaches treated initially with antibiotics for suspected sinusitis by his primary-care physician. Robert's headaches did not improve, and he was seen at a local hospital emergency department (ED) for further evaluation. A CT scan of his head was unremarkable for acute blood, mastoiditis, or sinus infection, but the lumbar puncture was suspicious for subarachnoid hemorrhage. An MRI revealed left transverse sinus, sigmoid sinus, and jugular bulb thrombosis with a left temporal lobe venous infarct (Fig 1). The following morning he became acutely confused with receptive aphasia, and he was inconsistently following commands and moving all extremities (Glasgow Coma Scale [GCS] score 14). Robert was then transferred to a different facility and placed in the neuroscience ICU. He was loaded with IV phenytoin, followed by scheduled phenytoin for prophylactic seizure management. Dexamethasone was started at 6 mg every 6

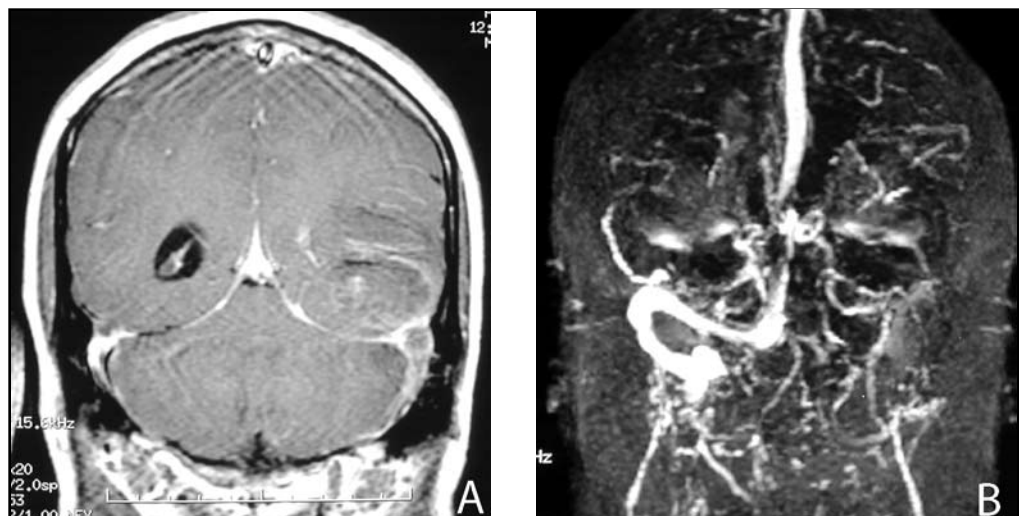


Fig 1. (A) Coronal T1 MRI with contrast showing thrombosis in left sigmoid sinus. (B) MRV showing no flow in left sigmoid sinus.



Fig 2. Follow-up CT demonstrating left temporal hemorrhage

hours, and IV fluids were increased to 150 cc/hr. Robert's past medical history was significant for previous history of deep vein thrombosis that was treated with warfarin for 6 months after right knee surgery. No hypercoagulation workup was obtained at that time.

On the evening of admission, Robert's aphasia worsened with paraphrastic errors, limited comprehension, perseveration, inability to name objects or read, and right

visual field neglect. His motor exam remained stable. A follow-up CT scan of the head demonstrated hemorrhagic conversion of the infarct (Fig 2).

Interventional neuroradiology was consulted on the fourth hospital day to perform a cerebral venography for evaluation of the extent of venous thrombosis. Venography revealed a thrombosed straight sinus, left transverse sinus, and sigmoid sinus (Fig 3). Anticoagulation with intravenous heparin was started immediately with target PTT of 50–65 seconds.

On the evening of the next day, Robert became acutely unresponsive with no speech, eye opening, or motor response; the left pupil was dilated and nonreactive. Anticoagulation was immediately stopped and a mannitol bolus (50 grams) was given, with recovery of the left pupil. Robert was then intubated and placed on mechanical ventilatory support. An emergency CT scan demonstrated no increase in left temporal hemorrhage, though extension of left hemispheric edema with questionable density changes in the upper brainstem suggestive of ischemic changes was noted. MRI was then completed and was negative for acute infarction. Anticoagulation was restarted.

Robert was then taken to the neuroradiology suite for emergency transvenous thrombolytic therapy. Moderate sinus recanalization was noted with 10 mg of tPA and 2000 units of Heparin. Mechanical clot aspiration and disruption were then performed in the left sigmoid sinus or transverse sinus (Fig 4). After the procedure his neurological exam improved. Robert remained intubated, opened his eyes to his name, followed commands bilaterally, and demonstrated a slight, right upper extremity weakness with drift. His pupils were equal and reactive to light.

Robert was extubated on day 11, 6 days after thrombolytic therapy. Robert opened his eyes spontaneously, had a right homonymous hemianopsia and bilateral

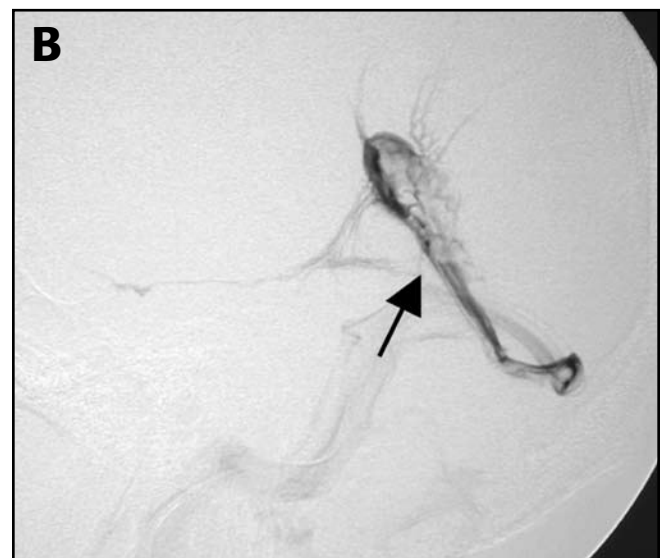
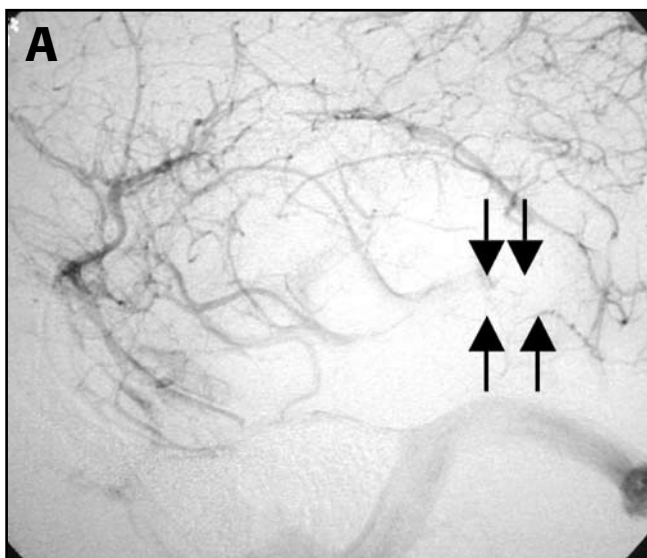


Fig 3. Angiography: (A) Left carotid angiogram, venous phase; shows obliteration of the straight sinus. (B) Superselective angiography of straight sinus following tPA infusion

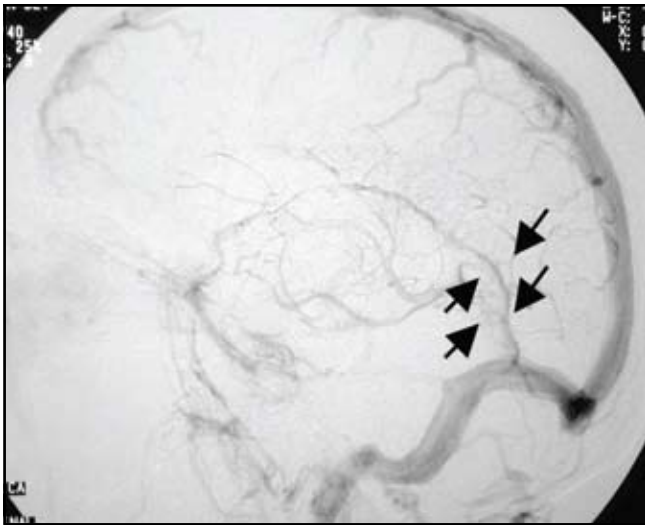


Fig 4. Left carotid angiogram, venous phase, posttreatment shows partially recanalized straight sinus. facial weakness. He was disoriented with fluent inappropriate speech, partial agraphia, dysnomic aphasia, and he followed commands bilaterally with slight weakness of the right upper extremity.

A hematology-oncology consult was obtained for further evaluation. A hypercoagulation panel had been performed on admission and was repeated with no abnormalities noted. The sinus thrombosis was thought to be idiopathic. Because of a second episode of thrombosis, hematology-oncology recommended a minimum of 12 months oral anticoagulation to maintain the INR level at 2%–3%. He was started on warfarin and transferred to the acute neurosurgical ward on day 12. A rehabilitation consult was obtained and Robert was cleared for discharge home with 24-hour supervision, and outpatient speech and occupational therapy on day 22.

At Robert's 3-month follow-up he was alert, oriented to person, place and time, his speech was fluent with anomia with low-frequency words, intact repetition and comprehension, impaired reading with visual alexia, and intact writing skills; he was moving all four extremities equally to command and ambulating independently, and had a right superior quadrant defect in the right temporal field. Phenytoin was weaned at this time. Robert also had returned to work part-time as a sheet metal worker. A follow-up MRI at 8 months demonstrated left temporal encephalomalacia, partial recanalization of the left transverse sinus, sigmoid sinus, and left internal jugular vein with no new focal lesions.

Case Study 2

Mary is a 30-year-old female who presented with a 1-week history of left-side neck pain and headaches that acutely increased in severity on the day of admission with associated nausea and vomiting (GCS = 15). Mary was seen in the ED where a CT scan of the brain demonstrated asymmetry of the posterior fossa within the left transverse

sinus suggestive of a venous thrombosis. An MRI was consistent with left transverse sinus, sigmoid sinus, and left jugular bulb thrombosis (Figs 5 and 6). Mary was admitted to the neurology service and placed in the acute care ward where IV hydration was initiated at 150 cc/hr and anticoagulation was begun with enoxaparin 60 mg subcutaneously twice daily and warfarin 3 mg daily.

Mary's past medical history was unremarkable with the current use of oral contraceptives as her only risk factor. Her status remained stable with improvement of her headaches with hydration. Mary was discharged home 36 hours postadmission. A hypercoagulation panel was unremarkable, though the hematology-oncology service recommended oral anticoagulation for a total of 12 months with a target INR level of 2%–3%. Enoxaparin was discontinued once the INR was within prescribed range on day 5.

One month later, a follow-up MRI showed interval improvement of flow within the left transverse sinus, sigmoid sinus, and left internal jugular vein. Mary returned to work 1 week after discharge.

Discussion

The two cases illustrate the variety of presenting signs and symptoms associated with CVST and the unpredictable nature of symptoms. The second case demonstrates how the appropriate use of imaging establishes early diagnosis prior to severe clinical deterioration and leads to early treatment. Good outcomes were noted in both



Fig 5. Hyperdense left sigmoid sinus due to presence of thrombus

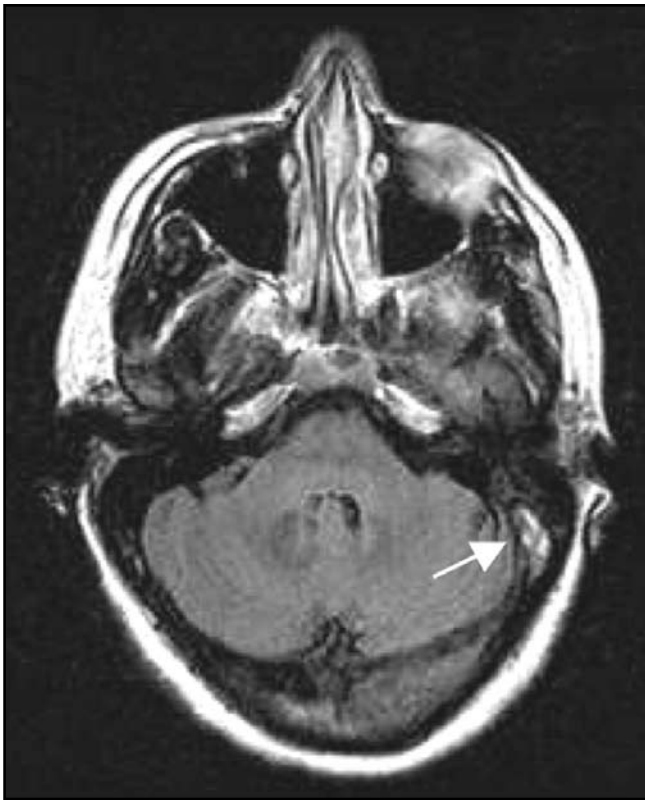


Fig 6. MRI: Thrombus in the left sigmoid sinus

cases, as defined by return to previous occupation, but one would not argue that there was concern with Robert and his potential for long-term disability.

Summary

The first-line treatment in acute CVST remains anticoagulation and hydration. Coordination of treatment modalities should be aimed at defining the best treatment with the lowest risk. Transvascular approaches by interventional neuroradiology offer a means for rapid recanalization of a thrombosed sinus by direct injection of thrombolytic agents and with the availability for clot retrieval with special catheters aimed at improving outcome. Timing continues to be an issue and for now the timing of interventional procedures with CVST is patient defined.

Astute physical assessment by the nurse is essential in identifying changes in this patient population, and if severe neurological compromise is present, critical care is essential. Critical care allows ongoing monitoring, use of supportive equipment, and coordination of treatment modalities. Nursing is not only key in the ongoing assessment but essential in providing coordination of supportive care and education of the patient and family.

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