Acute Treatment of Cerebral Venous Thrombosis

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Opinion statement

• Correct and timely diagnosis of cerebral venous thrombosis (CVT) is imperative in determining the appropriate treatment plan. Clinical suspicion is crucial, followed by radiographic confirmation using CT, MRI, or conventional angiography.
• Emergency and critical care of the patient is important, including control of airway and circulation.
• Initially, the treatment of choice is anticoagulation with heparin.
• Consideration should be given to intrathrombus delivery of thrombolytic agents.
• Eventually, newer techniques such as laser, ultrasound, and suction may be considered.

Introduction

Cerebral venous thrombosis (CVT) is an extremely interesting condition with an unknown occurrence rate and a highly variable presentation and outcome. It is generally thought to be a rare disease, but published numbers vary widely from 16 cases in 12,500 autopsies up to 9% in another patient series. These numbers may increase with technologically improved diagnostic techniques, however, the clinical presentation remains highly variable and consequently these tests may be underused. This clinical heterogeneity is partly the result of the different veins involved, the extent of thrombosis, normal variance of the venous structures, and specific patient characteristics such as age and other systemic factors [1,2].

The range of clinical symptoms includes headache, papilledema, focal deficits, seizures, and altered consciousness. Depending on the site and extent of thrombosis, increased intracranial pressure and infarction can develop. In severe cases, there can be coma and death; mortality is estimated to be from 5% to 30% [3–5].

Diagnostic tests that can be used are CT, MRI, and conventional angiography. Both CT and MRI are non-invasive and are routine tests in brain imaging, which can help diagnose CVT and the brain structures affected. Radiographic signs such as location and characteristics of infarction or cerebral edema, and the empty delta or dense triangle sign help diagnose CVT. With the addition of contrast dye, both can also be used to give venogram studies to help with diagnosis. However, the gold standard remains conventional angiography, because of its better resolution and the ability to diagnose CVT and evaluate the venous structures affected and the clot burden.

Treatment

General medical treatment

• The usual emergency and intensive care principles apply, including the following:
  - 1) Good airway control—intubation and mechanical ventilation should be considered when the patient is unable to control his airway from lack of brainstem reflexes or coma, poor respiratory effort, or poor oxygen delivery or exchange.
- 2) Hemodynamic stability—a balance must be reached between maintaining cerebral perfusion pressure, by keeping the mean arterial pressure elevated to counter increased intracranial pressure, and not risking intracerebral hemorrhage, because elevated blood pressure, especially in the setting of anticoagulation or thrombolytics (potential treatments of CVT), is particularly dangerous.
- 3) Fluid management—because of the potential concerns of increased intracranial pressure, fluids given should be isotonic, such as normal saline (0.9%, which has an osmolality of 308 mOsm/kg), rather than hypotonic fluids such as lactated Ringer’s solution (which has an osmolality of 274 mOsm/kg). Euvolemia should be the goal, because this will best maintain cerebral perfusion pressure as well as treat dehydration, which is one of the potential causes of CVT. This is maintained by reaching a goal central venous pressure (CVP) of 0 to 5 mmHg and maintaining good urine output of 1000 to 1500 mL per day. Generally, this means administering 2 to 3 liters of maintenance fluid per 24 hours.
- 4) Intracranial pressure management—the definitive treatment of the increased intracranial pressure from CVT is reperfusion of the venous structures, which will be discussed later. Otherwise, the typical measures are to improve venous drainage as best as possible by elevating the head of the bed by 30 degrees. Steroids are not indicated, though good pain control and thermal regulation is crucial. Use isotonic fluid to minimize extravasation of fluid across the blood-brain barrier and prevent worsening of cerebral edema. In severe cases consider lasix and mannitol to promote diuresis and decrease intravascular volume and promote osmotic gradients away from the brain. Also, intubation and hyperventilation may be warranted.
- 5) Seizure prophylaxis—the use of seizure prophylaxis is unclear. If seizure occurs or is a presenting symptom, then there is little doubt that an anticonvulsant should be used.

Pharmacologic treatment

Anticoagulation

- The primary goal of anticoagulation is to prevent progression of thrombosis and worsening neurologic function. The downside to anticoagulation, of course, is hemorrhage, particularly if there is an existing hemorrhagic infarction [6, Class II]. However, in clinical series and in a randomized controlled trial with unfractionated heparin, hemorrhage was not a significant problem. Furthermore, particularly in the latter trial, there was such a significantly improved outcome in patients who were treated compared with the placebo patients that the study had to be stopped early [7, Class I]. In the patients that had hemorrhagic infarction, there was also improved outcome in those treated with heparin [8••].
- In another randomized, placebo-controlled trial [9, Class I] with a low-molecular-weight heparin, there was a trend toward improved outcome in the treated patients, though not statistically significant.

Heparin

**Standard dosage** No bolus, but begin continuous intravenous infusion usually at 1000 U/hour (approximately 15 U/kg per hour) and adjust according to partial thromboplastin time (PTT) 1.5 to 2 times control.

**Contraindications** Persons with hypersensitivity or active bleeding.

**Main drug interactions** Digitalis, antihistamines, and tetracyclines may partially counteract anticoagulant effect.