Cerebral Venous Thrombosis Associated with Intracranial Hemorrhage and Timing of Anticoagulation after Hemicraniectomy

Michael A. Pizzi, DO, PhD,*† David A. Alejos, MD,† Jason L. Siegel, MD,*
Betty Y. S. Kim, MD, PhD,‡ David A. Miller, MD,‡ and William D. Freeman, MD*†‡

Background: Cerebral venous thrombosis (CVT) is a rare cerebrovascular event that can present with headache, seizure, and focal neurological deficits. Approximately 30%-40% of patients with CVT also present with intracranial hemorrhage. Current guidelines recommend anticoagulation after CVT even in the setting of intracranial hemorrhage, but the timing of initiation is unclear. We present a case of CVT where timing of anticoagulation was unclear by current guidelines. *Methods:* We conducted a literature search with search terms of "cerebral venous thrombosis," "intracranial hemorrhage," and "anticoagulation." Abstracted information included anticoagulation status and time of initiation of anticoagulation. We present a 30-year-old woman with sudden onset of right hemiplegia, global aphasia, and new-onset seizures diagnosed with left transverse and sigmoid sinus thrombosis with intraparenchymal hemorrhage. The patient was treated with endovascular thrombectomy and decompressive hemicraniectomy due to hemorrhage expansion, and anticoagulation was restarted 8 days after hemicraniectomy. *Results:* The literature review demonstrated a wide variation of timing for anticoagulation initiation in patients with CVT and intracranial hemorrhage. Most started anticoagulation within 24 hours of admission with similar functional neurological recovery. Current guidelines on the treatment of CVT, even with intracranial hemorrhage, recommend anticoagulation. Most reports in the literature state initiation of anticoagulation within 24 hours. However, the literature does not definitively state when to initiate anticoagulation in a patient with CVT, intracranial hemorrhage, thrombectomy, and decompressive hemicraniectomy. *Conclusion:* This case illustrates the challenge of determining when to resume anticoagulation for CVT. **Key Words:** Cerebral venous thrombosis—intracranial hemorrhage—anticoagulation—craniectomy.

Introduction

Cerebral venous thrombosis (CVT) is a rare form of stroke, constituting .5%-1.0% of all cerebral strokes. The symptoms and clinical outcome are highly variable.[1] The incidence is approximately 3 patients out of 1 million, and approximately 76% of the patients are female. Several potential risk factors have been identified including surgery, trauma, pregnancy, puerperium, antiphospholid syndrome, cancer, exogenous hormones, and thrombophilia.[2]

The mortality of CVT has decreased over the last few decades and is currently 5%-10%,[3] and this is because of increased awareness of the diagnosis, improved neuroimaging techniques, and more effective treatment.[1]

From the *Departments of Neurology, Mayo Clinic, Jacksonville, Florida; †Department of Critical Care, Mayo Clinic, Jacksonville, Florida; and ‡Department of Neurosurgery, Mayo Clinic, Jacksonville, Florida.
Received March 16, 2016; revision received May 12, 2016; accepted May 17, 2016.
Address correspondence to William D. Freeman, MD, Mayo Clinic, 4500 San Pablo Road, Jacksonville, FL 32224. E-mail: Freeman.william1@mayo.edu.
1052-3057/$ - see front matter

http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2016.05.025

Mortality is usually attributed to transtentorial cerebral herniation due to mass occupying lesions or generalized cerebral edema.[4] Complications such as subarachnoid hemorrhage (SAH)/intracerebral hemorrhage (ICH) are rare but can further complicate treatment and prognosis.[2,5]

The most frequent, but not specific, symptom of CVT is severe headache, which occurs in more than 90% of adult patients.[1] It usually progresses over days but can sometimes present as a thunderclap headache.[6] Most patients present with signs and symptoms of intracranial hypertension (headache, visual disturbances, papilledema, focal neurological deficits, and/or seizures).[7] Thrombosis of the deep venous system can cause bilateral thalamic lesions causing delirium, amnesia, mutism, or even coma.[8] Given the variety of possible presenting symptoms, the diagnosis of CVT can easily be delayed. According to the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT), there are an average of 4 days between the initial presentation of symptoms and admission to a hospital and 7 days between the initial presentation of symptoms and a diagnosis of CVT.[9]

Computed tomography (CT) or magnetic resonance imaging can be used as the initial test. Magnetic resonance venography (MRV) is the most sensitive imaging modality for CVT. Thrombosed sinuses are best demonstrated as hyperintensities on T1 and T2 sequences.[1] When computed tomography venography (CTV) and MRV are equivocal, cerebral angiography may be indicated if clinical suspicion for CVT is high. A repeat CTV or MRV at 3-6 months is recommended to assess the recanalization of the occluded cortical vein or sinuses in stable patients.[2]

The most recent guidelines state that once CVT has been confirmed, treatment with anticoagulation should be initiated even in the setting of ICH.[2] According to the algorithm detailed in the American Heart Association (AHA)/American Stroke Association (ASA) guidelines, if neurological deterioration occurs despite anticoagulation for CVT, decompressive hemicraniectomy may be considered. However, there are no recommendations for when to resume anticoagulation in this specific patient population.[2] The lack of recommendations for when to resume anticoagulation after decompressive hemicraniectomy after CVT in current guidelines and the paucity of published cases of CVT patients with ICH subsequently undergoing decompressive hemicraniectomy prompted us to present this unique case and engage in a discussion on when to resume anticoagulation in such patients.

Case Presentation

A 30-year-old woman with a past medical history of oral contraceptive use for dysmenorrhea developed severe left-sided headache 4 days before admission. The patient had been taking ibuprofen and naproxen for her headache without relief. On the day of admission, the patient was witnessed by her spouse as having a vacant, unresponsive stare. The patient was noted to have an ataxic gait followed by a witnessed generalized tonic–clonic seizure. The patient had a second witnessed seizure en route to the hospital, where she was intubated for airway protection.

At an outside facility, noncontrast CT of the head demonstrated a 3.7×2.3 mm ICH in the lateral left partial lobe, small adjacent SAH, and a left transverse and left sigmoid sinus thrombus (Fig 1). The patient was transferred to our institution for a possible interventional neurovascular procedure. Cerebral angiogram was performed, revealing a venous thrombosis in the left vein of Labbé and occlusion of the transverse and sigmoid sinuses (Fig 2). Significant thrombus was removed, and sections of the occluded sinuses were recanalized. No thrombolytics were given due to recanalization after mechanical thrombectomy. Anticoagulation with intravenous (IV) heparin was started. Within 12 hours of admission, the patient's condition deteriorated, as noted by asymmetric pupils. Repeat CT of the head was obtained 4.5 hours after endovascular intervention, demonstrating expansion of the ICH (Fig 3). Treatment with heparin was stopped, and the patient received emergent decompressive hemicraniectomy and removal of a left temporal hematoma (Fig 4).

The patient was transferred to the ICU where she improved neurologically, reaching a Glasgow Coma Scale score of 15. A noncontrast CT scan of the head was repeated 8 days after decompressive hemicraniectomy and showed no increase in the size of the ICH. Based on our patient's consistent and stable neurological status and radiographic imaging, we decided to resume anticoagulation. Treatment with IV heparin was then restarted 8 days after decompressive hemicraniectomy. The patient continued to show improvement, with expressive aphasia and dysarthria as her only residual symptoms. Anticoagulation

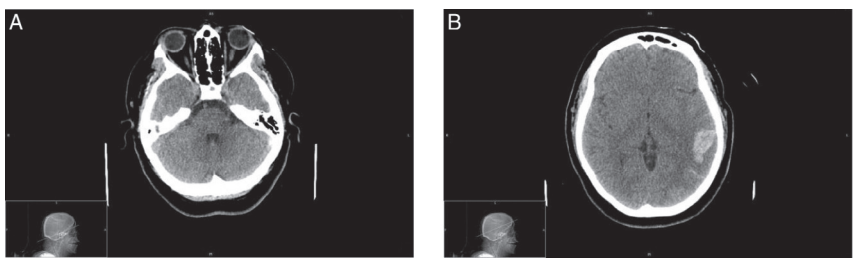

Figure 1. *Initial computed tomography scan of the head without contrast. (A) Hyperdensity in the region of the left transverse sinus. (B) Intraparenchymal hemorrhage of the left temporal lobe.*