DURAL VENOUS SINUS THROMBOSIS (DVST): A RARE CAUSE OF INTRACEREBRAL HEMORRHAGE (ICH)

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LEARNING OBJECTIVES

1. Highlight a case of dural venous sinus thrombosis with full resolution of clinical symptoms after prompt recognition and appropriate clinical management.

2. Review typical clinical presentations and characteristic radiologic findings of dural venous sinus thrombosis.

DISCLOSURE OF COMMERCIAL INTEREST

- Neither I nor my immediate family members have a financial relationship with a commercial organization that may have a direct or indirect interest in the content.

CLINICAL VIGNETTE

A 16-year-old male presented to the emergency room two weeks after a combat sports competition where he sustained traumatic injuries to the head. He reported a painful headache, blurry vision, and nonbloody nonbilious emesis.

- He reported several recent trips to the ER treated with migraine cocktails. The patient and his mother had previously decided to wait for an outpatient MRI due to concerns regarding the risk of radiation exposure. Notably, he was a nonsmoker, nondrinker, and had no family history of malignancy.

- ROS was (+) for generalized weakness, dizziness, & decreased appetite. He also displayed unsteady ambulation during his physical examination.

CLINICAL VIGNETTE IMAGING RESULTS

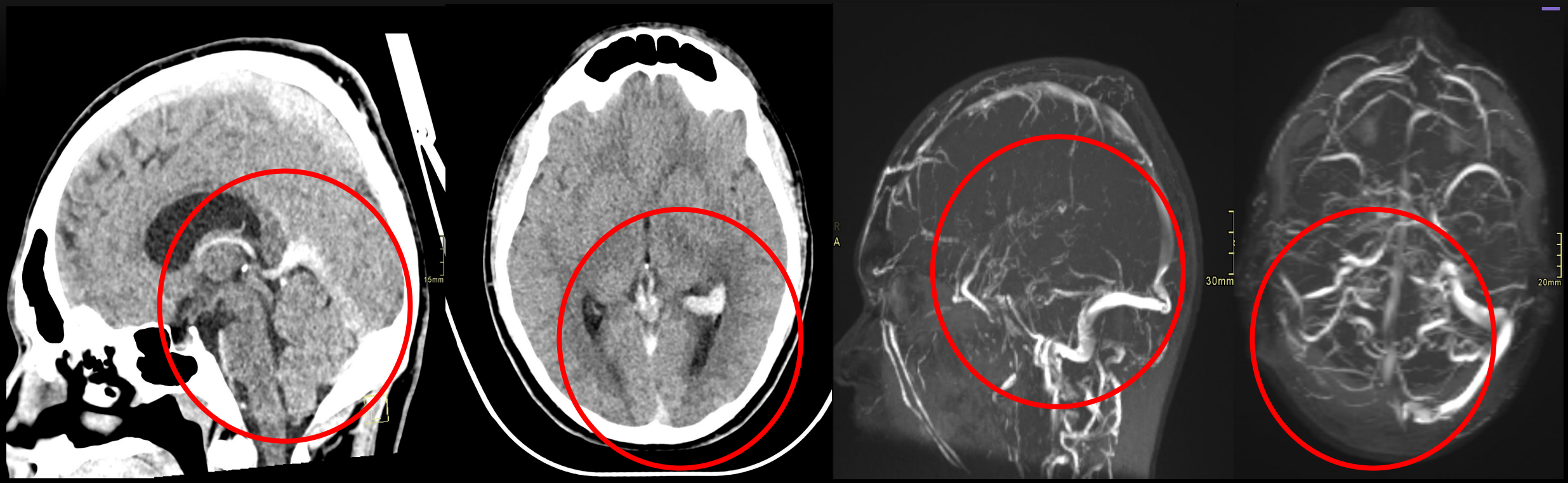
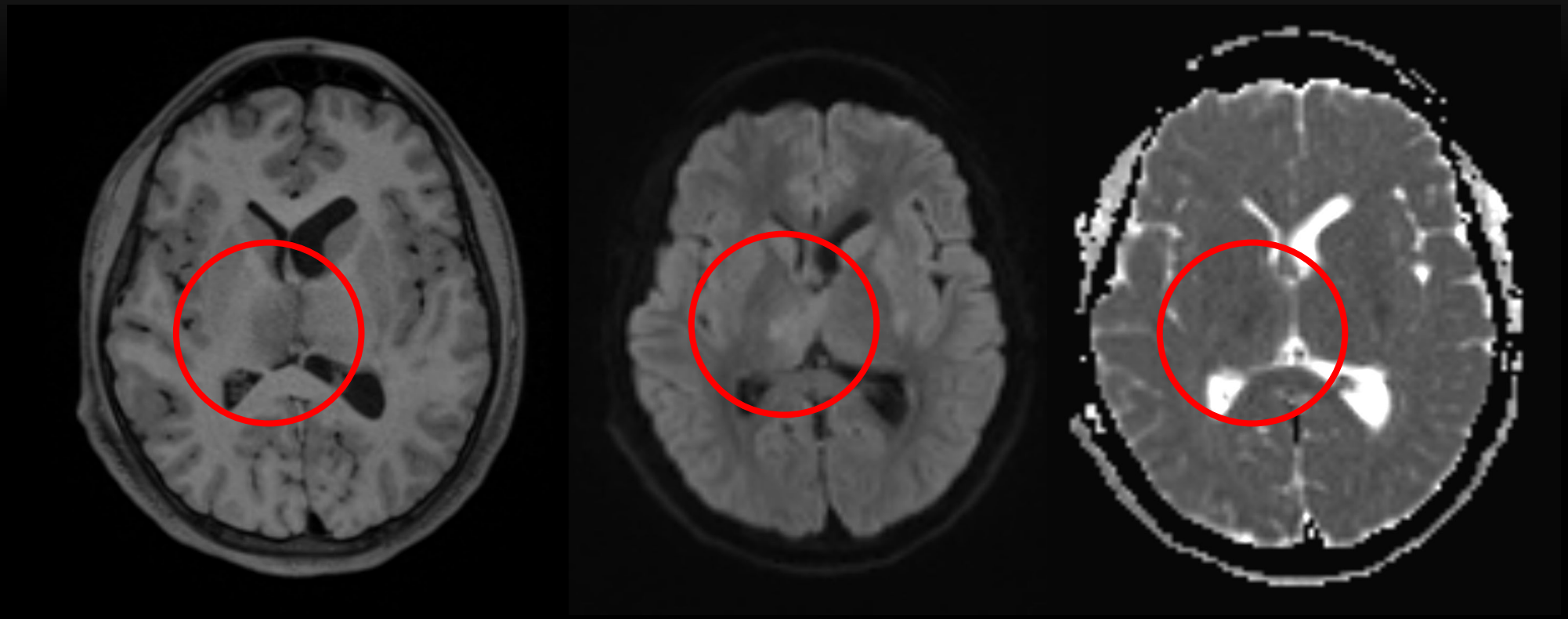


Figure 1 & 2: Sagittal view of the non-contrast head CT displays multifocal hyperdense venous contents, compatible with DVST. An axial view of the same CT illustrates the "cord" sign. Blood products within the left lateral ventricle are also partially visualized.

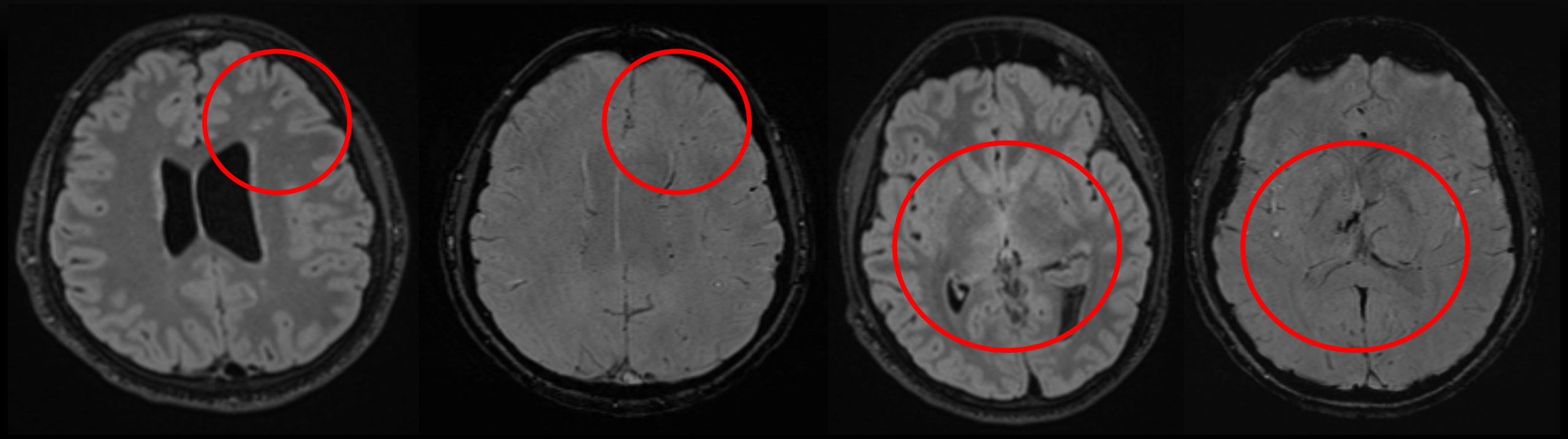
Figure 3 & 4: Volume-rendered axial & sagittal views of the contrast-enhanced brain MRV which displays the corresponding filling defects involving the internal cerebral veins, inferior sagittal sinus, vein of Galen, straight sinus, and torcula.

CLINICAL VIGNETTE IMAGING RESULTS



Figures 5 – 7: Axial views of T1W, DWI, and ADC sequences of the contrast-enhanced brain MRI display a T1 hypointense right thalamus with corresponding true restricted diffusion. This is compatible with right thalamic infarction secondary to deep cerebral venous thrombosis.

CLINICAL VIGNETTE IMAGING RESULTS



Figures 8 – 11: Axial views of T2W and SWI sequences of the contrast-enhanced brain MRI displays right thalamic and left frontal lobe T2W hyperintensity with corresponding SWI susceptibility, consistent with subacute hemorrhage/microhemorrhage given the clinical history.

The left intraventricular hemorrhage is also partially visualized within figures 10 & 11.

CLINICAL VIGNETTE OUTCOME

The patient was treated with anticoagulation therapy during and after hospital discharge.

At his follow up evaluation 1 month after discharge, he had no laboratory studies to indicate a predisposition to thrombophilia and no longer exhibited any neurologic or intracranial bleeding symptoms.

CLINICAL VIGNETTE FOLLOW-UP IMAGING RESULTS

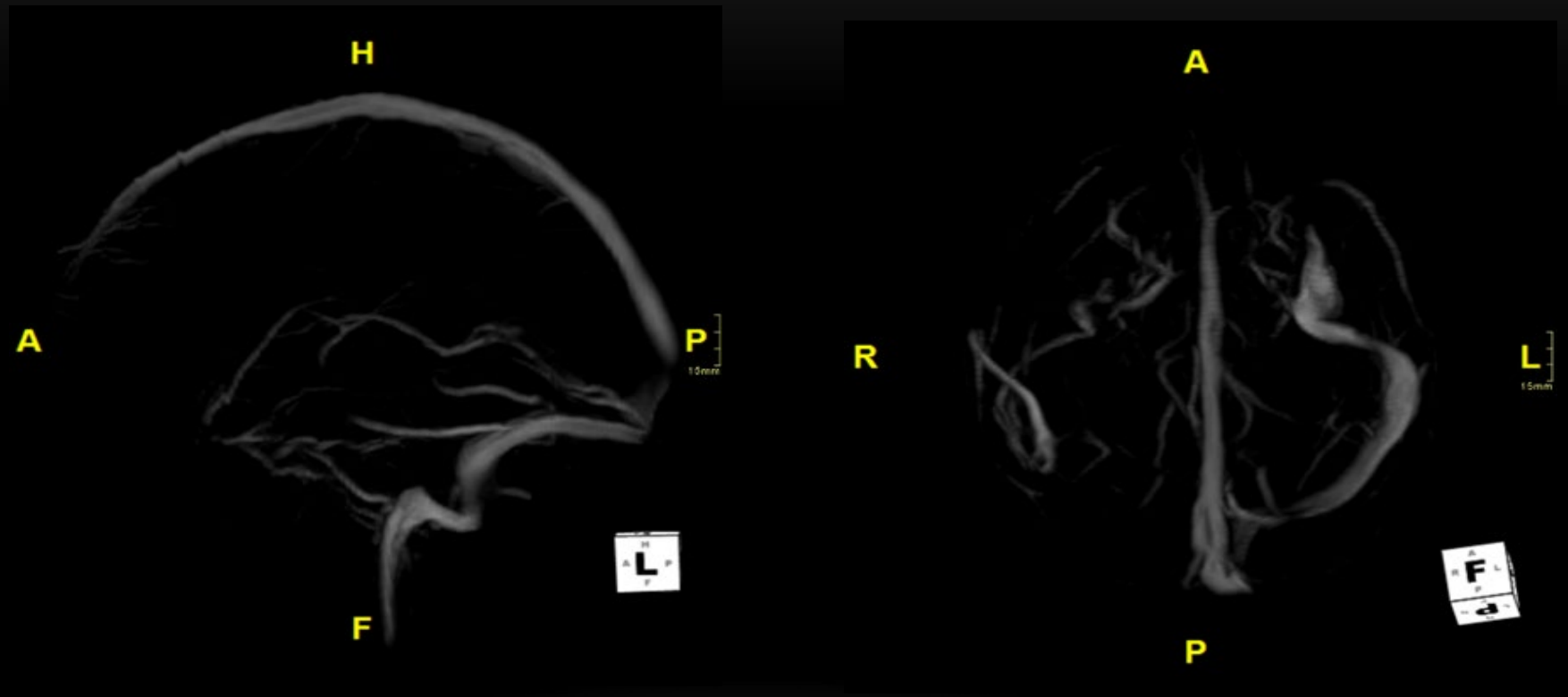


Figure 12 & 13: Volume-rendered axial & sagittal views of the follow up contrast-enhanced brain MRV which displays interval recanalization of the internal cerebral veins and vein of Galen, as well as partial recanalization of the straight sinus and torcula.

TAKE HOME POINTS – DVST RISK FACTORS & SYMPTOMS

Follow the principles of Virchow's triad: Hypercoagulability, stasis, and endothelial damage.

- These include a **hypercoagulable state** (Commonly due to mutations involving factor 5 Leiden, factor C and S, antithrombin, &/or prothrombin, as well as other causes such as pregnancy, contraceptive use, or use of hormonal replacement therapy), **trauma, infections, and malignancies.** ~25-30% of all DVST's have no clear etiology.

- The most common symptom reported is **headache,** which occur in 80% of patients. About 25% of the time, patients with DVST have headaches with no other symptoms. **Seizures** occur in 40% of patients, followed by **papilledema**, which occurs about 28% of the time.

TAKE HOME POINTS – DVST PROTOCOL & RADIOLOGIC FINDINGS

- **Non-contrast CT** can be used acutely to exclude hemorrhage and/or infarction, while concurrently diagnosing and/or ruling out other etiologies. **CT venography (CTV)** can localize the lesion more accurately and serves as a more sensitive alternative if necessary. **Magnetic resonance imaging (MRI)** is also helpful in diagnosis and like CT, will appear differently depending on the acuity.

- A common clue to the diagnosis on non-contrast CT is an increased density within the thrombosed venous sinus or cortical vein if acute, commonly referred to as the "**cord**" **sign**. Meanwhile, on contrast-enhanced CT, a filling defect will be visualized and is referred to as the "**empty delta**" **sign** when present within the superior sagittal sinus.

- If the deep cerebral veins are involved, there may be associated **thalamic infarcts**, although other considerations such as an artery of Percheron occlusion must be excluded in cases with infarcts bilaterally.

TAKE HOME POINTS – ETIOLOGIES OF ICH

- The most common causes of intracerebral hemorrhage (ICH) include **trauma, small vessel disease, hemorrhagic transformation, tumor, and DVST.** Thus, it is vital to remain vigilant of blood products in these clinical scenarios.

- **Localization** of the hemorrhage is key, as this will assist in the narrowing of your differential diagnosis. Other vital components of information include the **size** of the hemorrhage, **timing** of presentation, and the **modality of imaging**. These can impact your recommendations and guidance of therapy.
    - *For example, intraventricular hemorrhage is of the utmost importance as well due to its high risk of obstructive hydrocephalus. This is often secondary an intracerebral hemorrhage or subarachnoid hemorrhage with ventricular reflux. The recommendations given for our patient differed from the recommendation that would be given for an acute cerebellar hematoma secondary to a hypertensive CVA.*

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