24F with Down Syndrome, presents with confusion & left sided weakness.

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Deep Cerebral Venous Thrombosis with Venous Infarct
• Hyperattenuation of straight dural sinus (red arrow)
• Hypoattenuation of right thalamus and surrounding basal ganglia (blue arrow)
Increased density of straight sinus, extending into the inferior sagittal sinus, vein of Galen, & internal cerebral veins
Magnified view shows hypodense edema of splenium (yellow arrow) contrasted by relatively normal genus of corpus callosum (white arrow).
Axial DWI demonstrates hyperintensity involving the right thalamus (blue arrow)
Postcontrast subtraction MRV demonstrates near complete occlusion of straight dural sinus (red arrow).

The superior sagittal sinus is patent (blue arrow).
Cerebral Venous Thrombosis

Dural Sinus Thrombosis

Deep (Subependymal) Cerebral Vein Thrombosis

Cortical Vein Thrombosis
CT Findings

Non-contrast CT

- Early imaging findings often subtle
- **Hyperdense sinus** (compare to carotid arteries)
  - Usually $> 65$ HU
  - Distinguish thrombus vs. hyperdense sinus from high hematocrit (HCT)
    - HU:HCT ratio in thrombus $1.9 \pm 0.32$ vs. $1.33 \pm 0.12$ in nonthrombus
- $\pm$ hyperdense cortical veins ("cord" sign)
- $\pm$ venous infarct (50%)
  - White matter edema
  - Cortical/subcortical hemorrhages common
  - Thalamus/basal ganglia edema (if straight sinus, vein of Galen, and/or internal cerebral vein occlusion)
CT Findings

- Contrast Enhanced CT
  - "Empty delta" sign (25-30%)
    - Enhancing dura surrounds nonenhancing thrombus
    - "Shaggy," enlarged/irregular veins (collateral channels)
    - gyral enhancement
    - prominent intramedullary veins
- CTA/CTV
  - Filling defect (thrombus) in dural sinus
    - Caution: Acute clot can be hyperdense, obscured on CECT/CTV
      - Always include NECT for comparison
Differential Diagnosis

- Asymmetric anatomy: hypoplasia or atresia of the transverse sinus. The right transverse sinus is larger in 75-80% of cases.
  - If the sinus is small or absent, then the ipsilateral sigmoid sinus groove and jugular fossa should also be small.
- Arachnoid granulations: usually characterized as well-defined focal filling defects within the dural venous sinuses (measuring <10 mm in diameter)
  - more commonly in the lateral aspects of the transverse sinuses
  - Should be isointense to CSF (or nearly so) on all MRI sequences.
Dural Sinus Thrombosis

• Etiology:
  – Trauma (especially skull fractures extending to dural venous sinus)
  – Infection (especially mastoid sinus-dural sinus occlusive disease)
  – Inflammation
  – Hormonal: Peripartum/pregnancy, OCP, steroids
  – Metabolic (dehydration, thyrotoxicosis, cirrhosis)
  – Prothrombotic hematologic conditions: factor 5 Leiden mutation, Protein S deficiency, Prothrombin (factor II) gene mutation, polycythemia
  – Collagen-vascular disorders (APLA syndrome)
  – Vasculitis (Behçet)
  – Malignancy
Dural Sinus Thrombosis

- Pathophysiology
  1. Thrombus forms in dural sinus
  2. Clot propagates into cortical veins
  3. Venous drainage obstructed, venous pressure elevated
  4. Blood-brain barrier breakdown with vasogenic edema, hemorrhage
  5. Venous infarct with cytotoxic edema (DWI: mixed restriction)

- Locations:
  - Transverse Sinus
  - Superior Sagittal Sinus
  - Straight Sinus
  - Sigmoid Sinus
  - Cavernous Sinus

- Associated abnormalities
  - Dural AV fistula; venous occlusive disease may be underlying etiologic factor
Dural Sinus Thrombosis

- Presentation: HA*, n/v, +/- neuro deficit
- F > M, any age
- Up to 50% progress to venous infarction
- Rx: Heparin/Warfarin (even in the setting of venous hemorrhage); mechanical thrombectomy +/- local heparin infusion in more severe cases.
References

1. Statdx