

## Imaging of Cerebral Venous and Sinus Thrombosis

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### Abstract

Early and correct diagnosis of cerebral venous and sinus thrombosis (CVST) is necessary for prompt management of this disorder. Neuro-imaging is a crucial part of diagnosis of CVST. I reviewed the findings of non-enhanced computerized tomography (CT), Contrast enhanced CT, CT venography (CTV), different techniques of magnetic resonance imaging (MRI), MR venography, and digital subtraction angiography. I also reviewed the potential pitfalls in image interpretation. [GMJ.2016;5(Supp.1):43-47]

**Keywords:** Cerebral venous; Sinus thrombosis; Computerized tomography (CT); Magnetic resonance imaging (MRI).

### Introductions

Cerebral venous and sinus thrombosis (CVST) is often underdiagnosed because, it is associated with a wide spectrum of etiologic factors and clinical presentation. Early diagnosis of CVST is currently a major clinical challenge and the correct diagnosis of CVST depends on neurologic imaging. Neuroradiologists play an essential role in patient care by providing early diagnosis through selecting and interpretation of imaging studies. On the other hand, early diagnosis leads to rapid treatment that can be effective in prognosis [1, 2].

The purpose of this article is to review the approach for radiologic investigation and pitfalls associated with the radiologic evaluation of this diagnosis.

### Cerebral sinus thrombosis

CT findings:

Hyperdense dural sinus or cortical vein (“cord

sign”), cortical/subcortical petechial hemorrhages or edema and venous infarct in 50% can be seen [3-6].

#### 1. Contrast enhanced CT

“Empty delta” sign in 25-30% of cases, enhancing dura surrounds non enhancing thrombus and “Shaggy,” irregular veins (collateral channels) can be seen [3-6].

#### 2. CT venography

CT venogram (CTV) shows thrombus as filling defect in dural sinus [3-6].

MR Findings:

#### 1. T1WI

Acute thrombus usually appears in T1 iso-intense or hypointense however subacute thrombus becomes hyperintense and chronic thrombose usually hypointense [6-11] (Figure-1).

#### 2. T2WI

Acute thrombus usually appears in T2 hypointense however subacute thrombus be-

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comes hyperintense and chronic thrombosis usually hypointense [6-9] (Figure 1).

It should be noted in acute phase if thrombus is hypointense, can mimic normal sinus "flow void" on T2WI. On the other hand, chronically occluded, fibrotic sinus eventually appears isointense [6-11].

In proton density (PD) imaging is more sensitive than T2W but less sensitive than FLAIR. In FLAIR thrombus appears hyperintense in subacute and chronic phase [6-11].

### 3. T2\* or GRE

Thrombus in GRE appears hypointense, like blooming (blooming effect). Petechial and/or parenchymal hemorrhages appears Hypointense [6-11].

### 4. DWI

In DWI, clot in 40% appears hyperintense in occluded vessel however DWI/ ADC findings in parenchyma is variable and heterogeneous because in CVST we have mixture of vasogenic and cytotoxic edema; cytotoxic edema may precede vasogenic. Parenchymal abnormalities appears more frequently reversible than in arterial occlusions [12-14].

### 5. Contrast-enhanced T1

Peripheral enhancement around acute clot is prominent, it should be noted chronic sinus thrombosis can enhance due to organizing fibrous tissue, therefore in chronic phase of CVST T1 C+ imaging can be confusing and in spite of clot, filling defect due to fibrotic tissue enhancement, is not prominent [7-11].

### 6. MRV

Absence of flow in occluded sinus on 2D TOF MRV is the main feature. Frayed or shaggy appearance of venous sinus is prominent.

Abnormal collateral channels (e.g., enlarged medullary veins) become prominent. It is better to evaluate standard sequences and source images to exclude artifacts in MRV. Contrast-enhanced MRV (CE-MRV) better demonstrates thrombus, small vein detail, and collaterals, much faster than 2D TOF [6-11].

### Venous infarct

If there is venous infarct, mass effect with mixed hypo-/hyperintense signal in adjacent parenchyma is common. However, venous infarct has four characteristic feature: usually bilateral, parasagittal, in 60% with hemorrhage and is in venous territories not arterial territories [6-14].

Staging, Grading or Classification Criteria for venous ischemia:

Type 1: No abnormality

Type 2: High signal on T2WI/FLAIR; no enhancement

Type 3: High signal on T2WI/FLAIR; enhancement present

Type 4: Hemorrhage or venous infarction [15].

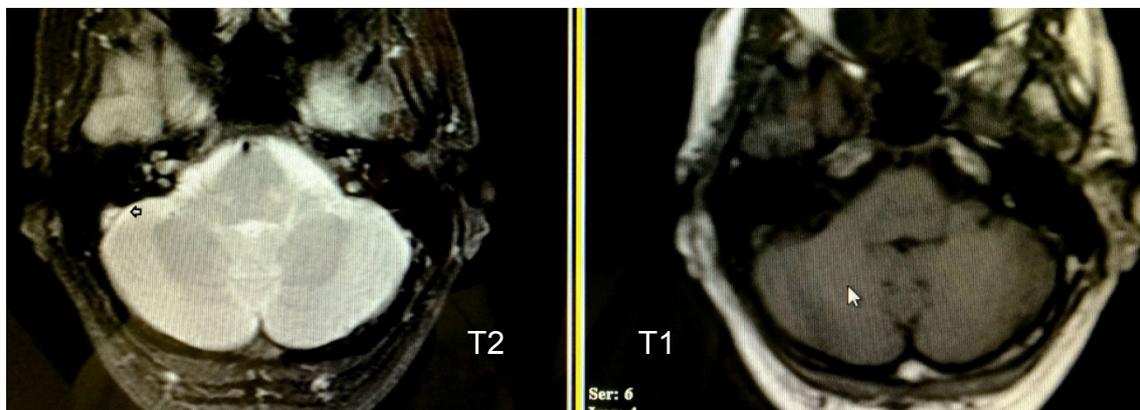
Angiographic Findings:

Occlusion of involved sinus is the main findings. However, slow flow in adjacent patent cortical veins and collateral venous drainage are prominent [3].

### Imaging Recommendations for CVST Best imaging tool:

1- NECT, CECT scans ± CTV as initial screening

2- MRI, MRV (include T2\*, DWI, T1 C+)[5].



**Figure 1.** T1 and T2 MRI image of a 34 y old female with diagnosis of cerebral sinus thrombosis with subacute thrombosis (T1, T2 hyperintense) in right lateral sinus.

Protocol advice: If CT scan is negative, MRI (include T2\*, DWI, contrast-enhanced T1) with MRV should be done.

If MRV equivocal and diagnosis is not definite, DSA should be done.

Potential Pitfalls in Image Interpretation

#### Normal:

Blood in vessels normally slightly hyperdense on NECT scans. Normally veins are slightly denser than brain tissue and in some cases it is difficult to say whether it is normal or too dense. In these cases a contrast enhanced scan is necessary to solve this problem. In infants the brain is usually less dense than in older children and adults. Consequently, relative high density of the blood in the sagittal sinus compared to the brain, which simulates a dense clot sign [5, 7, 10, 16].

#### Anatomic variant [10] may mimic CVT:

Congenital hypoplastic/absent transverse sinus (transverse sinus flow gaps 31%, usually non dominant sinus)

- Right transverse sinus dominant 59%, left dominant in 25%, codominant in 16%
- “High- split” tentorium
- Fat in sinus

#### “Giant” arachnoid granulation [16]:

- Round/ovoid filling defect (clot typically long, linear)
- CSF density/signal intensity
- Arachnoid granulations normal in 24% of CECT, 13% of MR
  - o Transverse sinus most common location by imaging, L>R
  - o SSS most common location for arachnoid granulations on histopath (in lateral lacunae, not well seen by imaging)
- False “empty delta” sign [5, 7:]
- SDH, subdural empyema

#### Neoplasm [5, 7, 16]:

- Venous infarct can enhance, mimic neoplasm
  - Intravascular lymphomatosis (rare)
- Cerebral hemorrhage [5, 7, 16]:
- Mimics venous infarct
  - Amyloid, contusion, hypertensive

#### Thrombus Signal Shine-Through at TOF

MR Venography [8]:

T1 hyperintense thrombus falsely appears as patent flow

Flow Gaps at TOF MR Venography [8]:

Wrong bolus timing [5, 7, 16]

#### Cortical venous thrombosis Definitions

- Superficial cerebral vein thrombotic occlusion with/without associated dural sinus thrombosis (DST)

General Features:

- Best diagnostic clue: “Cord sign” on NECT, T2\* GRE
- Location: Cortical veins (supra- > infratentorial)
- Size: Varies from small to extensive clot
- Morphology: Linear, cigar-shaped thrombus

CT Findings:

CT findings are as sinus thrombosis however:

- If internal cerebral veins (ICV) occlude, thalami and/or basal ganglia become hypodense
  - o Abnormal collateral channels (e.g., enlarged medullary veins)
  - o CECT has Limited value in chronic CVT (organizing thrombosis also enhances)

MR Findings:

MR findings are as sinus thrombosis however in:

- DWI/ ADC imaging findings heterogeneous dependent on presence of ischemia, type of edema, hemorrhage and can distinguish cytotoxic from vasogenic edema. Restriction can be seen in clot occluded veins at time of diagnosis and might be predictive of low rate of vessel recanalization 2 or 3 months later
- T1 C+ in acute/early subacute clot peripheral enhancement outlines clot can be seen and in late clot, thrombus, fibrous tissue often enhances. However in venous infarct patchy enhancement is prominent.
- MRV, may see abnormal collateral channels (e.g., enlarged medullary veins) but in contrast-enhanced MRV (CE-MRV) faster and better depicts non enhancing thrombus & small veins than TOF was seen.

- MR Perfusion, T2\* Gadolinium perfusion may show extensive venous congestion, but without perfusion deficits may play a role in detecting venous congestion vs venous infarction in CVT [6-12, 17]

#### Angiographic Findings:

- Conventional: More accurate than MRI, particularly for isolated cortical vein thrombosis
- DSA when imaging findings inconclusive, if clinical suspicion is high, or if intervention is planned

#### Deep cerebral vein thrombosis (DCVT)

##### Definitions:

- Thrombotic occlusion of deep cerebral veins (Figure-2).
- DCVT usually affects both ICVs +/- vein of Galen (V of G), straight sinus (SS)
- More widespread dural sinus thrombosis (DST) and cortical vein occlusion may occur

##### Best diagnostic clue:

Hyperdense ICV on NECT +/- bithalamic hypodense edema, variable DST

- Location: Clot in ICV +/- V of G, SS, basal veins of Rosenthal
  - o Bilateral ICV thrombosis »» unilateral
  - o Deep gray nuclei, internal capsule, medullary WM typically affected
  - o Variable involvement of midbrain, upper cerebellum (V of G, SS territory)



**Figure 2.** MRI T2 image of a 42 y female with confusional state and diagnosis of deep cerebral vein thrombosis with bilateral thalamus infarct.

- Morphology: Cigar shaped, “cord-like”

- T1WI

- o Clot: Early T1 isointense, later hyperintense

- o Most conspicuous sequence if clot subacute
- o Venous hypertension: Hypointense swelling of thalami, basal ganglia

- o Venous infarct: Hypointense edema, may be hemorrhagic

- T2WI

- o Clot: Often T2 hypointense mimicking flow void (“pseudo flow void”), much later hyperintense [6-12 ,18].

#### Angiographic Findings:

- o Unlike quite variable superficial veins, deep cerebral veins are always present on angiography

- In DCVT, occluded ICVs don’t opacify

- Collateral venous channels (e.g., pterygoid veins) enlarge

Magnetic resonance direct thrombus imaging (MRDTI) is a new modality that may improve the diagnosis of CST or CVT through its ability to visualize thrombus directly by exploiting the short T1 relaxation time of methemoglobin within the thrombus. Utilizing MRBTI in CVST was found to approach the high diagnostic accuracy of conventional imaging. The utility of MRBTI may also extend to monitoring for thrombus progression/resolution and to calculate thrombus volume as well [19].

#### Conclusion

In conclusion, best diagnostic tool for CST is NECT, CECT scans ± CTV as initial screening and MRI, MRV (include T2\*, DWI, T1 C+) and if MRV is equivocal, DSA. For cortical vein thrombosis T2\* is the best modality and for deep vein thrombosis hyperdense ICV on NECT and hyperintense T1WI (subacute) +/- bithalamic hypodense edema is the best clue for diagnosis. Venous infarct has four characteristic features: usually bilateral, parasagittal, in 60% with hemorrhage and is in venous territories (not arterial territories). New modality such as MRDTI can lead to high diagnostic accuracy for CVST in future.

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