Key points in the CT and MRI evaluation and interpretation of cerebral venous thrombosis

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Learning objectives

- To review the CT and MRI techniques used in cerebral venous system thrombosis;
- To present the most common and particular signs seen in cerebral venous thrombosis (CVT).
- To discuss and illustrate the common pitfalls, tips and tricks in diagnosis of CVT.

Background

Definition

Cerebral venous thrombosis represents the thrombotic occlusion of major dural sinuses or cerebral veins.

Epidemiology

Cerebral venous sinus thrombosis is a disorder whose epidemiology has changed over the past few decades. It is no longer regarded as a rare condition.

CVT is responsible for 1% of strokes.
The population subgroup with the highest risk appears to be women of childbearing age, particularly in the postpartum period. Another high-risk group appears to be neonates or young infants.

There is no reliable data on racial or geographical distribution but researches conducted in India on CVST claim that this disease is more common in underdeveloped countries of Asia than the western world [1].

Etiology

Many causative conditions have been described in cerebral venous thrombosis. These may be seen alone or in combination. At least 1 risk factor can be identified in >85% of patients with cerebral venous thrombosis [1].

- **Infectious causes:**
  - local: abces, meningitis, empyema, otitis, mastoiditis, sinusitis;
  - general: sepsis;
- **Non-infectious causes:**
Pathophysiology

The three main components of Virchow's Triad (damage to vessel wall, disorders of coagulation and stagnant flow) have different weights than in case of an arterial stroke [2]- Fig. 1 on page 6.

Two major pathophysiological mechanisms contribute to the clinical presentation of cerebral venous thrombosis [3]- Fig. 2 on page 6. First, thrombosis of cerebral veins or sinuses can result in increased venular and capillary pressure. As local venous pressure continues to rise, decreased cerebral perfusion results in ischemic injury and cytotoxic edema, disruption of the blood-brain barrier, leads to vasogenic edema, and venous and capillary rupture then culminates in parenchymal hemorrhage.

Obstruction of cerebral sinuses may also result in decreased cerebrospinal fluid absorption (which is normally absorbed through arachnoid granulations into the superior sagittal sinus) leading to increased intracranial pressure. Consequently, increased intracranial pressure worsens venular and capillary hypertension and contributes to parenchymal hemorrhage and vasogenic and cytotoxic edema.

Anatomy

Intracerebral venous system is composed of (Fig. 3 on page 6):

Superficial cerebral veins:

- Superior - drain frontal and parietal lobes into superior sagittal sinus;
  - Troland vein is the largest;
- inferior - drain the temporal lobe (vein of Labe) and part of the occipital lobe into transvers sinus, inferior petrosal sinus and cavernous sinus;

Deep venous system:

Internal cerebral veins drain septum pellucidum, thalamo-striate veins and superior choroidal vein. They unite with the basal veins (of Rosenthal) to form the vein of Galen. Dural venous sinuses are found in between layers of dura mater and drain finally into internal jugular veins.

- Superior group: Superior sagittal sinus, inferior sagittal sinus, transverse sinuses, straight sinus, sigmoid sinuses.
- Inferior group: cavernous sinuses, petrosal sinuses, intercavernous sinuses, pterygoid plexus, clivus plexus, sphenoparietal sinuses [4].

Location:
• The main cerebral sinuses involved are: Superior sagittal sinus >> transverse and sigmoid sinuses >> straight sinus;
• deep venous system: 8%;
• isolated cortical vein: exceptional.

Clinical features

The clinical onset is polymorph: headache, focal neurological deficits, seizures, papilledema and consciousness impairment.
The symptoms and signs depend to some extent on which sinus is affected and to an important extent on whether the thrombotic process is limited to the dural sinus or extends to the cortical veins (Fig. 4 on page 7).
Onset of symptoms and signs may be acute, subacute, or chronic.
Four major syndromes have been described: isolated intracranial hypertension, focal neurological abnormalities, seizures, and may be present in combination or isolated[5].

Diagnosis

The diagnosis of CVT is typically based on clinical suspicion and imaging confirmation.
Measurement of D-dimer has little diagnostic role in exclusion of DVT or pulmonary embolus when used with pretest probability assessment. If there is a strong clinical suspicion of CVT, a normal D-dimer level should not preclude further evaluation [5].
Examination of the cerebrospinal fluid (CSF) is typically not helpful in cases with suspected CVT. An elevated opening pressure may be a clue for diagnosing CVT in patients who present at the emergency department with headaches.

Imaging methods

• **CT.** Routine head CT requires additional spiral acquisitions at 40-45 seconds after injection of intravenous contrast, with maximum 1 mm slice thickness. CT venography is comparable to MR venography in the diagnosis of CVT.
• **MRI.** The routine MRI protocol for the brain includes: axial T2 SE, axial/coronal Flair, sagittal, axial T1SE, axial T2*, DWI sequences. The most commonly used MRV techniques are time-of-flight (TOF) MRV and contrast-enhanced magnetic resonance- 3DT1+Gd with multiple reformatations-MPR and MIP reconstructions [6].

Imaging findings

The radiological findings are divided in:

• **Direct signs:** direct visualization of the thrombus: cord sign, hyperdense sinus sign, loss of flow void in SE or FSE sequences of a dural sinus, empty delta sign, lack of enhancement of a dural sinus or a cortical vein;
• **Indirect signs**: infarction, hemorrhage, oedema [9].

**Prognostic**

Death rates in different studies range between 5% and 30% and probably depend more on case mix than on treatment. Causes of death can be the underlying condition, the brain lesion, secondary complications, or a combination of these [2]. Poor prognosis factors are: coma, cerebral hemorrhage, malignancy, male sex, age >37 years, mental status disorder, thrombosis of the deep cerebral venous system, papilledema and diagnostic delay >10. Good outcome was associated with an isolated intracranial hypertension presentation and a delta sign on CT [2]. Recurrence of cerebral venous thrombosis is rare (2.8%) [7].

**Complications** [5]

*Hydrocephalus* usually caused by failure of CSF absorption; is rarely obstructive resulting from hemorrhage into the ventricular system.

*Intracranial Hypertension* primarily caused by venous outflow obstruction and tissue congestion compounded by CSF malabsorption.

*Dural Arteriovenous Fistula*. The relationship between the 2 entities is rather complex, because (1) dural fistulas can be a late complication of persistent dural sinus occlusion with increased venous pressure, (2) the fistula can close and cure if the sinus recanalizes, and (3) a preexisting fistula can be the underlying cause of CVT.

*Seizures*: Focal or generalized post-CVT seizures can be divided into early or remote (occurring >2 weeks after diagnosis) seizures. Can require continuous antiepileptic treatment.

*Visual Loss*: Severe visual loss due to CVT rarely occurs (2% to 4%).

**Treatment**

Acute phase therapy for cerebral venous thrombosis focuses on anticoagulation, management of sequelae such as seizures, increased intracranial pressure, and venous infarction, and prevention of cerebral herniation. Organized care is one of the most effective interventions to reduce mortality and morbidity after acute stroke (Fig. 5 on page 7).

In general, thrombolytic therapy is used if clinical deterioration continues despite anticoagulation or if a patient has elevated intracranial pressure that evolves despite other management approaches. Many invasive therapeutic procedures have been reported to treat CVT. These include direct catheter chemical thrombolysis and direct mechanical thrombectomy with or without thrombolysis. There are no randomized controlled trials to support these interventions compared with anticoagulation or with each other [5].
**Fig. 1:** Figure 1: Virchow Triad

![Virchow Triad Diagram]

**Fig. 2:** Figure 2: Pathophysiology of cerebral venous thrombosis. References: modified after Piazza G, Cerebral venous thrombosis, Circulation. 2012;125:1704-1709

![Pathophysiology Diagram]
**Fig. 3:** Figure 3: Anatomy of intracerebral venous system. References: http://www.radiologyassistant.nl/en/p4befacb3e4691/cerebral-venous-thrombosis.html

<table>
<thead>
<tr>
<th>Location</th>
<th>Symptoms/Signs</th>
</tr>
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<tbody>
<tr>
<td>Cavernous sinus</td>
<td>Orbital pain, Chemosis, Proptosis, Cranial nerve palsies (III-IV)</td>
</tr>
<tr>
<td>Superior sagittal sinus</td>
<td>Motor deficits, Seizures</td>
</tr>
<tr>
<td>Inferior sagittal sinus</td>
<td>Motor deficits, Seizures</td>
</tr>
<tr>
<td>Straight sinus</td>
<td>Motor deficits, Mental status changes</td>
</tr>
<tr>
<td>Transverse sinus</td>
<td>Intracranial hypertension (headache), Tinnitus, Cranial nerve palsies, Aphasia (if left-sided)</td>
</tr>
<tr>
<td>Internal jugular vein</td>
<td>Neck pain, Tinnitus, Cranial nerve palsies</td>
</tr>
</tbody>
</table>

**Fig. 4:** Figure 4: Major clinical syndromes according to location of cerebral venous thrombosis.
Fig. 5: Figure 5: Proposed algorithm for the management of CVT. References: Modified after Saposnik G et al., Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American, Stroke. 2011;42:1158-1192
Findings and procedure details

Imaging methods

CT evaluation. For the CT examination we used a 16-slice MDCT system. CT-venography was preceded by a non-enhanced CT acquisition (sequential, caudo-cranial direction, orbito-meatal plane, with 5 mm slice thickness and slice reconstructions of 1,5 mm thickness). 40 seconds after the start of 80-100 ml of iodinated nonionic contrast medium, iv. injection at a rate of 2.5-3 ml/sec begins the postcontrast acquisition (spiral, axial plane, 0,625-1 mm slice thickness). MPR, MIP and VRT reconstructions were used to aid the diagnosis.

MRI evaluation was performed using a 1,5 T magnet. Besides the cerebral routine exam which consists of:

- axial T1 SE;
- axial T2 FSE;
- axial/coronal FLAIR;
- T2*;
- DWI;
we used:
- 2DTOF sequence and
- 3DT1 FSPGR postcontrast (0,1 mmol/kg Gadolinium-based contrast media). Recon: MPR and MIP

Imaging findings

- CT findings

Unenhanced CT

Direct signs:

There are a couple of signs described in the literature: cord sign (given by dense cortical veins)- Fig. 6 on page 13, dense triangle sign (given by hyperdensity of the superior sagittal sinus)- Fig. 7 on page 13.

- Fresh thrombus - hyperdense - see Fig. 8 on page 14, Fig. 9 on page 14, Fig. 10 on page 15.
- Old thrombus ( > 14 days) - isodense
- Rarely - hypodense
Indirect signs:

Venous infarction: - Fig. 11 on page 15, Fig. 12 on page 16, Fig. 13 on page 17

- The most common indirect sign in CVT;
- Hypodense areas which do not respect an arterial territory;
- Cortico-subcortical or profound location;
- Can be multiple;
- Can turn hemorrhagic --> multifocal hyperdensities (Fig. 14 on page 17).

Cerebral oedema (Fig. 15 on page 18)

- Can be diffuse or localised;
- Can be both cytotoxic and vasogenic in aetiology;
- Hypodense areas.

Contrast enhanced CT

"Empty delta sign"- Fig. 16 on page 18

- in 25-30% of cases;
- Enhancing dura surrounds non enhancing thrombus in the SSS.

Thrombus = filling defect inside the enhanced venous vessel (Fig. 17 on page 19, Fig. 18 on page 19).

- MRI findings

Unenhanced MRI

Normal appearance of cerebral sinuses (Fig. 19 on page 20).

Direct signs:

Thrombus appearance (Table nr.1)- Fig. 20 on page 20, Fig. 21 on page 20.

Table no.1

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Acute stage &lt;5 days</th>
<th>Subacute stage 5-15 days</th>
<th>Chronic stage &gt;2-3 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb</td>
<td>DeoxyHb</td>
<td>MetHb IC then EC</td>
<td>Organnized thrombus</td>
<td></td>
</tr>
<tr>
<td>CT</td>
<td>#</td>
<td>#</td>
<td># then#</td>
<td>#</td>
</tr>
</tbody>
</table>
T1       #       #       #       #
T2/Flair #       #       # then#  #
T2*      #       #       # then#  #
DWI      #       #/#      #/#      #/#

• Hypointensity in T2* sequences (Fig. 23 on page 21)
• Loss of flow-void in T2 FSE sequences (Fig. 22 on page 21)
• Absence of flow in occluded sinus on 2D TOF MRV (Fig. 24 on page 22)
• 40% of clots have restriction of water diffusion (Fig. 25 on page 22)
• Chronic thrombus can enhance; hiperintensity on DWI may predict lack of recanalization.

Indirect signs:

Venous infarction:

• subcortical/ cortico-subcortical edematous lesions;
• topography of venous infarction: SLS (parasagittal, fronto-parietal distribution; rather bilateral and asymmetrical), transverse sinuses (isolated temporal distribution), deep cerebral system (often bilateral and symmetrical lesions of the thalamus and basal nuclei), isolated cortical vein (in the drained territory);
• non-arterial distribution;
• hyperintense area in T2/Flair, hypointense in T1, with restriction of water diffusion;
• may turn to hemorrhagic (petechial --> hematoma) infarct, in 60-80% of cases --> present hyperintensities in T1 or subarachnoid hemorrhage (Fig. 27 on page 24)
• can associate subdural or subarachnoid hemorrhage- (Fig. 26 on page 23)

Cerebral oedema (Fig. 28 on page 24)

• effacement of cerebral sulci and basal cisterns, ventricular compression;
• the vasogenic oedema has no restriction of water diffusion;
• the cytotoxic oedema has restriction of water diffusion;

2D T1 and 3D T1 postgadolinium sequences (Fig. 30 on page 25)

• reveal the lack of enhancement of the thrombosed sinus;
• organizing fibrous tissue from chronic thrombosis can enhance;
• adjacent leptomeningeal enhancement ;
• abnormal collateral channels.

Comparison CT venography/ MRI (Table nr.2):
• Direct signs: CTV>>>MRI;
  - better spatial resolution (source images and 3D);
  - cortical veins;
  - no flow artifacts;
  - VRT reconstructions: parenchyma + veins.
• Indirect signs: MRI>>>CTV.

Table no.2

<table>
<thead>
<tr>
<th>CTV</th>
<th>MRI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Advantages</strong></td>
<td><strong>Disadvantages</strong></td>
</tr>
<tr>
<td>- without irradiation</td>
<td>- flow/irradiation movement</td>
</tr>
<tr>
<td>accessible</td>
<td>- iodine contraste artifacts</td>
</tr>
<tr>
<td>easy</td>
<td>- time reduced</td>
</tr>
<tr>
<td>fast</td>
<td>- 3D not operator-dependent</td>
</tr>
<tr>
<td>agitated patients</td>
<td>consuming accessibility</td>
</tr>
<tr>
<td>- quality of MPR and 3D</td>
<td></td>
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</tbody>
</table>

**Differential diagnosis:**

• **Hyperdense sinus in children and young adults:** In infants the brain is usually less dense than in older children and adults. This results in a relative high density of the blood in the sagittal sinus compared to the brain, which simulates a dense clot sign (Fig. 31 on page 26)

• **Arachnoid granulations:** focal rounded filling defects with a characteristic anatomic distribution: usually identified in the lateral part of the transverse sinus, near the entrance sites of the Labbé vein (Fig. 32 on page 26)

• **Hypoplasia** and **atresia** of the lateral sinuses: asymmetry right-left of the lateral sinuses on the MRA (Fig. 33 on page 27)

• **False "empty delta sign"** : subdural haematoma, great bifurcation of the Torcular Herophili (the transverse sinuses take a more vertical direction which can produce a false image of delta in axial sequences);

• **Arterial ischemic stroke** - ischemic area that respects arterial territory (Fig. 34 on page 27)
• **Tumoral invasion** (especially meningiomas, sinonasal tumors, neuroblastoma, clivus chordoma) /compression (brain/bone metastases) - Fig. 35 on page 28.

The **IMAGING REPORT** must answer at the following questions:

- Are there any density/signal abnormalities involving the intracranial venous system?
- Are there any filling defects involving the venous sinuses or cortical/deep veins?
- Are there any parenchymal injuries?
- Do we detect signs of severity: intracranial hypertension, other associated injuries?

**Images for this section:**

![Image](https://example.com/image.png)

**Fig. 6:** Cord sign: NECT-hyperdensity of thrombosed cortical veins. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 7:** Dense triangle sign. NECT - hyperdensity of the thrombosed superior sagittal sinus. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

**Fig. 8:** NECT- Hyperdensity of the right sigmoid and transverse sinuses. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 9:** NECT - Hyperdensity of the superior sagittal sinus and cortical veins. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

**Fig. 10:** NECT- hiperdensity of right transverse, sigmoid sinuses and right internal jugular vein. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 11:** Venous infarction. NECT (1) and MRI (2,3,4) - right parietal venous infarction with thrombosed cortical vein nearby References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania

**Fig. 12:** Venous infarction. NECT- small right parietal venous infarction with thrombosed cortical vein nearby; hyperdense superior sagittal sinus References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 13:** NECT- Left thalamic venous infarction with hyperdense left internal cerebral vein. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

**Fig. 14:** NECT-Right parietal hemorrhagic infarction, hyperdense cortical vein nearby. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 15: NECT: Multiple bilateral supratentorial venous infarcts with important edematous component, mass effect and hemorrhagic areas on the left fronto-parietal zone, complicated with subarachnoid hemorrhage (1 a,b); bilateral frontal venous infarcts (2) in a context of superior longitudinal sinus (SLS) thrombosis. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 16:** Empty delta sign- CECT - enhancing dura surrounds non enhancing thrombus in the SLS. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

![Image of CT scans showing empty delta sign and enhancing dura surrounding thrombus in the SLS.](image)

**Fig. 17:** CECT: Filling defects of the superior sagittal sinus and transverse sinuses. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

![Image of CT scans showing filling defects in the superior sagittal sinus and transverse sinuses.](image)
**Fig. 18:** CECT: Small filling defect of the Torcus Herophili extended into the superior sagittal sinus. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

**Fig. 19:** MRI: Normal appearance of cerebral sinuses

**Fig. 20:** MRI - subacute thrombosis of the right transverse sinus. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 21:** MRI - acute thrombosis of the right transverse and sigmoid sinuses. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania

**Fig. 22:** MRI - Loss of normal flow-void in T2 FSE sequences. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania
Fig. 23: MRI - Hypointensity of the right sigmoid sinus in T2* sequences. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

Fig. 24: 2D TOF MRV, MIP reconstructions and T1WI+contrast: Lack of flow of the right lateral sinuses and internal jugular vein. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 25: Thrombosed cortical veins- utility of MR-DWI and ADC maps: restricted diffusion and corresponding low signal on the ADC map of the thrombosed cortical veins. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

Fig. 26: Venous infarction: Right frontal cortico-subcortical area hyperintense on T2-WI/Flair images, hypointense on T1WI with focal subarachnoid hemorrhage; hypersignal of
the SLS in T2 FSE sequence. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

**Fig. 27:** Right operculo-parietal hemorrhagic infarct with mass effect over the right lateral ventricle. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

F, 22 years old, postpartum, headache, Intracranial hypertension
**Fig. 28:** MRI - Diffuse cerebral oedema. Hypersignal of the SLS, straight sinus and left transverse sinus. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

**Fig. 29:** MRI - Thrombosis of the superior sagittal sinus, left lateral sinuses and torcular. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
**Fig. 30:** Cerebral venous thrombosis. MRI - T1WI + contrast: Lack of enhancement of the right transverse sinus (1), superior sagittal sinus (2), right transverse and sigmoid sinus (3). References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

**Fig. 31:** Hyperdense SLS in children. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 32: Arachnoid granulations. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

Fig. 33: Venous sinus hypoplasia: 2D TOF MRV-Right lateral sinuses and right internal jugular vein hypoplasia. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Fig. 34: NECT: First CT- acute ischemic stroke: hipodense cortico-subcortical area in the left frontal lobe; 5 days later: hemorrhagic transformation. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.

Fig. 35: MIP sagittal reconstruction: Meningioma invading the superior sagittal sinus (1); Mip sagittal reconstruction and T1 FSWI sagittal: bone metastasis compressing the superior sagittal sinus. References: Radiology and Imaging Department Fundeni Clinical Institute, Bucharest, Romania.
Conclusion

- MRI is the method of choice for the diagnosis and follow-up of CVT, especially in children and young patients.

- In cases with limited access or contraindications of MRI evaluation, CT is the most common imaging study used, fast and reliable.

- It is essential to know the anatomy of the intracranial venous system and the radiological signs of the CVT in order to establish a prompt and correct diagnosis.

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