Imaging of cerebral venous thrombosis, pearls and pitfalls

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Learning objectives

To show imaging findings of cerebral venous thrombosis and to highlight possible pitfalls in this sometimes difficult diagnosis.
Background

Cerebral venous thrombosis (CVT) involves 0.5/100,000 people/year and usually affects young individuals (78% < 50 years). CVT may involve the superior sagittal sinus (62%), right and left transverse sinuses (44.7% and 41.2%, respectively), straight sinus (18%), cortical veins (17%), deep venous system (11%), cavernous sinuses (1.8%) and cerebellar veins (0.3%).

Risk factors for CVT include: thrombophilia, obesity, oral contraceptives, estrogenic replacement therapy, pregnancy and puerperium, cancer, medicaments (e.g. Tamoxifen and chemotherapeutics) and oto-mastoiditis/sinusitis (especially in newborns and children).

Clinical presentation is variable. In the majority of the cases symptoms are due to an increase in intracranial pressure as a consequence of impaired venous outflow that causes intense headache (present in about 90% of the patients), nausea and vomiting. Focal neurologic symptoms may appear in a progressive way as a consequence of ischemic and/or haemorrhagic complications and vary according to the involved cerebral area; CVT is responsible for about 1% of all strokes. Seizures occur in about 40% of the cases. Given the progressivity of symptoms onset patients typically refer to the hospital some days after the event.

Normal D-dimer levels have high negative predictive values for excluding CVT; anyway, two studies have reported that D-dimer levels may be normal in up to 25% of positive cases, in particular when thrombi are very small (e.g. in isolated cortical veins thrombosis). The diagnosis of CVT is radiological and is based on computed tomography (CT) and magnetic resonance imaging (MRI). Imaging studies may be performed also for excluding late complications and in particular dural arteriovenous fistulae that may develop in up to 3% of the cases.

Treatment is based on anticoagulant drugs (i.e. intravenous heparin followed by oral anticoagulants). Endovascular therapies (mechanical thrombectomy and local thrombolysis) may be considered only in life-threatening cases with clinical deterioration despite pharmacological treatment.
Findings and procedure details

COMPUTED TOMOGRAPHY

Unenhanced CT represents the first line imaging modality for the evaluation of patients with new neurological symptoms. Anyway, unenhanced CT is normal in about 70% of the patients affected by CVT and, therefore, it cannot be considered reliable for this diagnosis.

Intravenous contrast material administration is mandatory whenever CVT is suspected; indeed, CT venography with subsequent multiplanar reconstructions is extremely accurate in the identification of CVT.

Imaging findings

On unenhanced CT, hyperdensity within a dural sinus or a cortical vein is the only manifestation of CVT and is better appreciable on reconstructions perpendicular to the involved segment, but it is present only in 25-65% of the cases; unenhanced CT sensitivity is slightly higher for deep cerebral venous thrombosis.

Unenhanced CT is able to accurately recognize haemorrhagic complications that may follow CVT. On the other hand, unenhanced CT accuracy in the detection of ischemic complications varies according to lesion's age. Ischemic lesion that doesn't respect a vascular territory or is located in the strict proximity of a dural sinus must raise the suspicion of CVT and mandates further examinations.

The key finding for the diagnosis of CVT is the presence of a filling defect within the involved vessel, usually associated with a certain degree of dural enhancement, on contrast-enhanced CT; this association may generate to the so-called "empty delta sign", i.e. a triangular filling defect surrounded by contrast material within the superior involved sinus, which, anyway, is present in only 35% of the cases.

Pitfalls

On unenhanced CT, false positive cases are prevalently due to haemoconcentration, which determines homogeneous hyperdensity of the whole cerebral venous system. Partial volume artefacts from adjacent bony structures may also be responsible for false positive cases, but multiplanar reconstructions reduce their significance.
On unenhanced CT, false negative cases may have various explanations. First of all, thrombus hyperdensity may be observed in the acute stage only; indeed, thrombi become progressively hypodense during the subacute and chronic stages.

On contrast-enhanced CT, arachnoid granulations may protrude within a dural sinus, but their round shape enables to distinguish them from thrombi. Some degree of sinus hypoplasia/aplasia is very common (20-40% of the population) and should not be confused with CVT; indeed, in these cases the vessel is uniformly thinned and do not present filling defects.

Markedly hyperdense thrombi may be difficultly recognizable within the contrast-filled vessel if a proper visualization window isn't used; moreover, post-contrast images must always be compared with pre-contrast ones. Partial volume artefacts may hide thrombi in small veins, in particular on maximum intensity projections. Moreover, chronic thrombi may show a certain degree of enhancement.

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<thead>
<tr>
<th>Cause</th>
<th>False positives</th>
<th>False negatives</th>
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<td>Hyperdense acute clot</td>
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<td>Haemoconcentration</td>
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<td>Subacute/chronic thrombi</td>
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<td>Filling defect</td>
<td>Clot surrounded by contrast enhancement</td>
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<td>contrast artefacts</td>
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MAGNETIC RESONANCE IMAGING

Unenhanced MRI is increasingly performed in patients with neurological symptoms that remain unexplained after CT and is extremely accurate in CVT diagnosis. Anyway, MRI findings are frequently overlooked if the examination is performed without precise clinical indications and there are a lot of possible pitfalls a radiologist must be aware of.

Contrast-enhanced MRI represents the gold standard for diagnosing CVT.
**Imaging findings**

Sinus hyperintensity on T1-weighted, T2-weighted and PD-weighted images with the subsequent absence of flow-void artefact is highly suspicious for CVT, but is not present in the acute phase (first 5-7 days) after thrombus formation. Also hyperintensity on FLAIR images has high sensitivity in detecting subacute CVT, but its specificity is low.

A prominent markedly hypointense tubular structure with blooming artefacts on T2* images is quite constantly present in patients with CVT, also in the acute stage; therefore, T2*-weighted images are the most accurate for diagnosing CVT on unenhanced MRI. Moreover, T2* weighted images might be the only ones able to identify and isolated cortical vein thrombosis. Anyway, these sequences are not routinely included in a "standard" brain MRI protocol.

The clot may show diffusion restriction on high b-value diffusion-weighted images, but the sensitivity of this finding is only 4-40%. Moreover, diffusion restriction has been observed only in T1- and FLAIR-hyperintense clots.

**Time of flight (TOF)/phase contrast MR venography** has high sensitivity in diagnosing CVT; indeed, flow absence/reduction is always present in CVT. Anyway false positive results are extremely frequent. 2D TOF MR venography is more accurate than 3D TOF MR venography and phase contrast MR venography in diagnosing CVT.

Unenhanced MRI depicts CVT both ischemic and haemorrhagic complications with higher accuracy than CT. About 25% of the patients with CVT show oedema of adjacent parenchyma, whereas up to 40% of them show oedema associated with petechial or confluent haemorrhage. Diffusion coefficient restriction is usually minor in venous infarctions in comparison with arterial ones.

On contrast-enhanced T1-weighted images CVT manifests as a filling defect within a cortical vein/sinus.

**Pitfalls**

During the first 5-7 days after its formation, the thrombus appears substantially isointense on T1-weighted images and hypointense on T2-weighted ones, possibly leading to false negative findings. Moreover, a marked T2-hypointensity, simulating flow void, may be appreciable in about 13% of the cases. On the other hand, T1- and T2-hyperintensity may be observed in case of slow flow within a sinus. T1-weighted images performed without and adequate flow-compensation often show hyperintensity within dural sinuses.

Hyperintensity on FLAIR images may be observed within hypoplastic sinuses and in case of slow flow. Chronic thrombi may appear hypointense on FLAIR images.
Some authors reported absence of blooming artefacts on T2*-weighted images in chronic thrombi.

Sinus hypoplasia results in a marked lumen reduction on TOF MR venography, but the vessel appears uniformly tapered and doesn't show abrupt changes. Moreover, flow speed alterations and in-plane flow may cause false positive findings. Given the wide anatomical variability, cortical veins thrombosis is virtually undetectable on TOF MR venography.

Thrombus hyperintensity on T1-weighted images may be responsible for false negative cases on contrast-enhanced MRI. Anyway, the performance of pre- and post-contrast 3D gradient-echo T1-weighted images with subsequent subtracted reconstructions enables to avoid this possible pitfall.
**DIGITAL SUBTRACTION ANGIOGRAPHY**

Digital subtraction angiography (DSA) has progressively lost its role in the diagnosis of CVT parallel to the technical improvements on CT and MRI.

DSA findings associated with CVT include lack of sinus visualization/filling defect, delayed visualization of venous structures, upstream venous congestion and reversed venous flow.
Fig. 8: Superior sagittal sinus thrombosis: typical MRI findings in the acute (yellow arrows) and subacute (blue arrows) phases.

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**Fig. 9:** Chronic left sigmoid sinus thrombosis (yellow arrows) with dural fistula (blue arrows): MRI findings.

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**Fig. 10:** Acute cortical vein thrombosis (yellow arrows): MRI findings.

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Fig. 11: Subacute cortical vein thrombosis (yellow arrows): MRI findings.

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**Fig. 12:** Subacute superior sagittal sinus thrombosis: empty delta sign (yellow arrow) on contrast-enhanced T1-weighted images.

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**Fig. 13:** Subacute superior sagittal sinus and cortical veins thrombosis (yellow arrows) with intraparenchymal haemorrhage (blue arrows).

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**Fig. 14:** Acute straight sinus thrombosis (yellow arrow) with bilateral thalamic ischemic stroke (blue arrows).

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**Fig. 15:** Subacute superior sagittal sinus and cortical veins thrombosis (yellow arrows) with left frontal ischemic stroke (blue arrows).

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Fig. 16: False positive case: lack of flow in the right transverse and sigmoid sinuses on phase-contrast images (yellow arrow) that, however, show physiological enhancement after contrast material administration. A small arachnoid granulation (blue arrow) is also appreciable on contrast-enhanced images.

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Fig. 17: False positive case: hyperintensity of a cortical vein suspicious for thrombosis on FLAIR images (yellow arrow) in a patient with left temporal headache. The finding is not confirmed on the other imaging sequences (blue arrows).

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**Fig. 18:** Pitfall on contrast-enhanced images: a subacute thrombus (yellow arrows) in the right transverse sinus appears markedly hyperintense on T1-weighted images and might be misinterpreted as contrast enhancement after gadolinium injection. Subtraction reconstructions clearly highlight the filling defect.

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**Fig. 1:** Subacute superior sagittal sinus thrombosis (yellow arrows): unenhanced CT findings.

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Fig. 2: Acute internal cerebral vein (blue arrow) and straight sinus (yellow arrow) thrombosis: unenhanced CT findings.

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**Fig. 3:** Subacute isolated cortical vein thrombosis (yellow arrow): unenhanced CT findings.

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Fig. 4: Intraparenchymal haemorrhage (blue arrow) in subacute superior sagittal sinus thrombosis: unenhanced CT findings.

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Fig. 5: Empty delta sign (yellow arrow) on contrast-enhanced CT in subacute superior sagittal sinus thrombosis.

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**Fig. 6:** Chronic left sigmoid sinus thrombosis (yellow arrows): unenhanced CT (a) and contrast-enhanced CT (b) findings.

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**Fig. 7:** Subacute right transverse sinus thrombosis (yellow arrows): typical MRI findings.

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Conclusion

Both contrast-enhanced CT and MRI show extremely high accuracy in the detection of CVT and must be considered the gold standard for this diagnosis. Unenhanced MRI is relatively accurate in CVT diagnosis but is limited by a lot of pitfalls; T2*-weighted images are the most accurate for this diagnosis and are particularly useful in the detection of cortical veins thrombosis.
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References