Spectrum of CT and MR Findings for Cerebral Venous Sinus Thrombosis

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Learning objectives

1. Review aetiology, clinical presentation and basic pathophysiology of cerebral venous sinus thrombosis.

2. Consolidate cerebral venous anatomy and venous drainage of the cerebral parenchyma.

3. Identify the spectrum of imaging findings on CT and MR imaging including distinguishing common pitfalls from true pathology.
Background

Cerebral venous sinus thrombosis (CVST) is a rare but potentially life-threatening condition, which predominantly affects the young, primarily women of childbearing age and children [9]. With a prevalence of 3-4 cases per million and up to three times more common in women, early detection and rapid administration of treatment are essential to reduce morbidity and mortality [7]. Many predisposing factors have been implicated in the development of CVST including oral contraceptives, coagulopathies, pregnancy and puerperium, systemic diseases, cancer, ear-nose-throat infections, vasculitis, CNS infections and other CNS disorders [4]. In up to 35% of cases of CVST no predisposing factor is identified [10]. Four major presenting syndromes have been identified including raised intracranial pressure, focal neurological deficits, seizure and encephalopathy with 95% of patients presenting with headache [3].

In CVST thrombus formed from hypercoagulable state, stasis or vein wall damage causes occlusion of the cerebral sinus or cortical vein with subsequent congestion of the upstream cortical veins and sinuses [13]. This congestion compromises blood flow to the area of parenchyma drained by the congested veins causing neuronal ischaemia [7]. In turn, cytotoxic oedema results from the ischaemic changes [7]. Concurrently the venous congestion causes petechial haemorrhages at the grey white interface and thalami, which induce vasogenic oedema with mass effect [7]. Furthermore due to the venous sinus occlusion there is reduced resorption of CSF, further increasing intracranial pressure [11]. An understanding of this cascade as well as the normal venous drainage of the cerebral parenchyma is key in detecting and interpreting computed tomography (CT) and magnetic resonance imaging (MRI) findings.

The venous drainage of the brain is via superficial and deep venous drainage systems [5]. The superficial cortical veins drain the cerebral cortex to the superior sagittal sinus (Fig 1), which then drains to the torcula, the confluence of the sinuses. Similarly, deep veins from the basal ganglia and thalami drain via the inferior sagittal sinus and straight sinus to the torcula. From this point, venous blood flows through the paired transverse sinuses to the sigmoid sinuses and into the internal jugular veins. The temporal lobe is drained by the vein of Labbe to the ipsilateral transverse sinus [5] (Fig 2). CVST most commonly affects the superior sagittal sinus closely followed by the transverse sinuses with conjoined sinuses often affected by a single thrombus resulting in loss of enhancement or flow void on CT or MRI [12]. Thrombosis of the deep venous system is rare, 11%, but also associated with a poorer prognosis [1].

CT is the most commonly utilised imaging modality for first line assessment of intracranial pathology [4]. Although CT has inherent ionising radiation and iodinated contrast medium may cause adverse effects, the combination of speed of acquisition, accessibility and
sensitivity of CT is to date unmatched by any other imaging modality [5]. MRI has been shown to be the most sensitive at detecting both CVST and related complications but is often limited by longer acquisition times, accessibility and motion and flow artefacts [2]. Angiography remains the gold standard for diagnosis of CVST and provides the added benefit of concurrent intervention however it is an invasive procedure, with added risks of iodinated contrast medium and ionising radiation [10]. With improving imaging techniques, the rate and accuracy of detection of CVST is increasing and thus it is essential that all radiologists be aware of the spectrum of findings associated with CVST and its complications.

PROCEDURE DETAILS:

A 10-year (June 2003 to June 2013) retrospective review of consecutive positive CVST studies was undertaken at an Australian Quaternary Institution. A systematic automated search of the RIS report database was undertaken using key terms and restricted to MR studies. A total of 722 reports were returned by searching the terms "+venous +thrombosis", "+venous +thrombus" and "+venous + thrombosed" with 31 positive cases of cerebral venous sinus thrombosis identified. No further positive studies were identified with a search of all cerebral catheter angiogram studies.

All MR studies were performed on a 1.5 T magnet. T1 and T2 weighted spin echo images were acquired on all patients with 15 patients given gadolinium and 18 of the patients having either 2D or 3D time of flight (TOF) MR venography (MRV). Of the 31 patients, 19 underwent non-contrast enhanced CT (NECT) with 12 contrast enhanced CTs (CECT) of which 6 were dedicated CT venograms. The CT venogram (CTV) protocol at the study institution includes administration of 100 mL of iodinated contrast scanned at a 100 second delay.

The age, presenting complaint and duration of symptoms were recorded from the information provided on the request form (Table 1). Three independent reviewers scrutinised the MR studies each classifying the signal intensity of the thrombus on T1 and T2. Further MR findings included extent of thrombosis and the thrombus and parenchymal signal changes including diffusion restriction, FLAIR intensity and blooming on susceptibility weighted imaging. Where available CT findings were documented and compared to the MR findings to assess clot extent.
Fig. 1: Normal venous anatomy. Superficial cortical veins (blue arrow) drain to the superior sagittal sinus (red) and from there to the Torcula (purple). The deep venous system (yellow) includes the inferior sagittal sinus, Vein of Galen and the straight sinus, which also drain to the Torcula. The paired transverse sinuses (green) drain laterally from the Torcula into the ipsilateral sigmoid sinus (orange) and then into each internal jugular vein. Drainage of the temporal lobes is via the Vein of Labbe (white) directly to the transverse sinus.

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Fig. 2: The superficial cortical veins and superior sagittal sinus drain the convexities of the cerebral hemispheres (green) with the temporal lobes drained via the Vein of Labbe (blue). The anterior portions of the temporal lobes and posterior frontal lobes (yellow) are drained via the Sylvian vein to the basal venous sinuses.

Findings and procedure details

From the collected data the mean patient age was 38 years with an age range of 16 to 67 years. The majority of patients were female (20:11) less than the expected incidence of female predominance documented in the literature, 3:1, which is somewhat estimation as our Institution does not provide an obstetric service [7]. The mean duration of symptoms was 10 days, with a range of 1 to 60 days. Presenting symptoms varied with headache the most common seen in 61% of cases with an identified risk factor documented on the request form in 42% of the cases. The most commonly thrombosed sinus in our patient subset was the transverse sinus, 70%, with multiple contiguous sinus involvement seen in 60% of the patients (Table 1). Of the patients with a NECT, 79% demonstrated a hyperdense sinus, more than the expected average of one third of cases described in the literature [7]. The extent of the thrombus demonstrated with CECT was equal to that shown on MRI in patients in whom both studies were performed in keeping with previous findings demonstrating CTV to be as accurate as MRI [7].

On CT and MRI there are direct and indirect signs of CVST, which are utilised to diagnose sinus thrombosis [10]. These are described below.

**DIRECT SIGNS:**

**Hyperdense sinus:** On NECT early thrombus is hyperdense, expanding the sinus in approximately 30% of cases [10] (Fig 3). This should be asymmetrical and homogenous [16]. With time the thrombus will become less dense becoming isodense to brain in 14-21 days (Fig 4). Asymmetry of the transverse sinuses can be a normal anatomical variant and often a hypoplastic sinus can be a pitfall in diagnosis [10]. On NECT the larger sinus may be relatively hyperdense however with contrast administration the larger sinus will also show dense contrast enhancement and thus patency. Further to this the contrast will highlight the hypoplastic transverse sinus and small draining internal jugular vein [12] (Fig 5). On angiography, a hypoplastic sinus will fill at the same rate as the normal sinus with a thrombosed sinus demonstrating delayed filling [8].

**Cord sign:** A thrombosed cortical vein seen as a cord of hyperdensity over the convexity of the cerebrum (Fig 3). In isolation a cortical vein thrombosis can be difficult to detect and susceptibility weighted imaging is useful to increase the conspicuity of the thrombosed vein [10]. Angiography provides more accurate assessment of the cortical veins and should be performed if there is a high clinical suspicion of venous thrombosis [15].

**Empty delta:** Superior sagittal sinus thrombosis distends the superior sagittal sinus and precludes enhancement of the sinus on post contrast imaging. As a result there is venous congestion within the veins of the dural leaves, which enhance surrounding the unenhanced triangular shaped sinus forming an empty delta appearance [6] (Fig 3).
Filling defect or loss of a flow void within a sinus or cortical vein: Absence of flow voids on FLAIR and T2-weighted spin echo MR images orthogonal to the sinus is suggestive of thrombosis [10]. On CECT and MR there should be a filling defect in the expanded sinus or vein to indicate thrombus (Fig 3). As thrombus ages some enhancement can be seen within the thrombus but to a lesser degree than that of the adjacent sinus [15]. Arachnoid granulations can cause filling defects in the cerebral venous sinuses however these can be distinguished from thrombus as they have lobulated margins and inhomogeneous central enhancement [10] (Fig 6).

INDIRECT SIGNS:

Cerebral haemorrhage occurs at the grey white interface as a result of venous congestion and increased venous pressure (Fig 7) [7]. Subarachnoid haemorrhage can also result.

Oedema can cause reduction of the ventricle size due to mass effect which can be difficult to distinguish from normal in young patients with normally small ventricles [10]. On NECT the cerebral parenchyma is hypodense with FLAIR and T2 hyperintensity on MRI (Fig 7). The oedema can be both cytotoxic and vasogenic in aetiology [7].

Infarction in a non arterial distribution involving the subcortical region but sparing the cortex, often multifocal with or without haemorrhage is suggestive of venous obstruction and congestion [10]. The area affected should be in keeping with the area drained by the thrombosed veins and sinuses (Fig 7).

With MRI of CVST the thrombus signal intensity follows that of aging blood product evolving through the stages of oxyhaemoglobin, deoxyhaemoglobin, methaemoglobin and hemosiderin [9] (Table 2). Thus in the hyperacute phase venous thrombus will be isointense on T1 and hyperintense on T2 weighted imaging (Fig 9). As the thrombus evolves the T1 appearance remains isointense with T2 becoming hypointense to cerebral parenchyma in the acute phase (Fig 10). In the early (Fig 11) and late (Fig 12) subacute phases methaemoglobin causes the T1 signal to be hyperintense with early T2 hypointensity and late T2 hyperintensity. Chronic thrombi demonstrate T1 and T2 isointensity (Fig 13). Within our patient subset thrombus signal intensity correlated with the reported duration of patients' symptoms as documented on the request forms (Fig 14).
**Table 1:** Distribution of sites of venous thrombosis

<table>
<thead>
<tr>
<th>Location of Thrombus</th>
<th>No. of Patients n=31 (%)</th>
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<tbody>
<tr>
<td>Superior Sagittal Sinus</td>
<td>16 (51%)</td>
</tr>
<tr>
<td>Transverse Sinus</td>
<td>22 (71%)</td>
</tr>
<tr>
<td>Sigmoid Sinus</td>
<td>14 (45%)</td>
</tr>
<tr>
<td>Deep Venous System</td>
<td>5 (16%)</td>
</tr>
<tr>
<td>Cortical Vein</td>
<td>2 (1%)</td>
</tr>
<tr>
<td>Contiguous Sinus Involvement</td>
<td>19 (61%)</td>
</tr>
</tbody>
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**Fig. 3:** Direct signs of cerebral venous sinus thrombosis. Hyperdensity in the unenhanced sinus and cortical vein is suggestive of thrombosis (A and B). After contrast administration the thrombus forms a filling defect in the sinus with the empty delta sign showing the central thrombus and enhancing dural leaves (C). The contrast thrombus interface is seen in partially thrombosed sinuses (D).

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Fig. 4: Hyperdense right transverse sinuses (red arrows) on unenhanced CT in a 44 yr female patient with 1 day of visual changes and papilloedema (A), a 19 yr female with a 4 day history of headache and new seizure (B) and a 21 yr female with progressively worsening migraines over a month. As the thrombus ages it becomes less dense in keeping with aging blood product.

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Fig. 5: This patient has a hypoplastic left transverse sinus giving the dominant right transverse sinus, an expanded hyperdense appearance on unenhanced CT (A). Post contrast the right transverse sinus enhances normally (B) and the left transverse, sigmoid (C) and internal jugular vein are all small but homogeneously enhancing.

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Fig. 6: Arachnoid granulations also known as Pacchionion granulations (A), have lobulated margins with inhomogeneous central enhancement, which distinguishes them from cerebral venous sinus thrombosis (B).

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Fig. 7: Cerebral venous infarction is an indirect sign of cerebral venous sinus thrombosis represented by cerebral haemorrhage (A and C), oedema (B and D) and infarction (E and F) in a venous distribution.

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**Fig. 8:** CT venogram showed similar results to MR imaging in demonstrating cerebral venous sinus thrombosis. Complete thrombosis of the left transverse sinus, sigmoid sinus and internal jugular vein seen on CT venogram (A,B and C) is depicted equally effectively on MRI, coronal T2 (D), axial 2D TOF MR (E) and 3D TOF MR reconstruction (F) in the same patient.

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Fig. 9: This 44 yr male patient presented with a 1 day history of reduced visual acuity, papilloedema and diplopia. On MR imaging hyperacute thrombus is isointense on T1 (A) and hyperintense on T2 (E). The filling defect is demonstrated on 2D TOF MR (D) and 3D TOF reconstruction (F). Signs of raised intracranial pressure were identified in this patient, namely the empty sella sign (B) and bilateral increased CSF around the optic nerves with flattening of the optic discs (C).

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**Fig. 10:** 42 yr female presents with a 2 day history of occipital headache and photophobia. Unenhanced CT showed hyperdensity of the deep venous system (A) which is hypointense on T2W MR (B). 3D TOF MRV shows loss of the inferior sagittal sinus and straight sinus (C). Oedema is seen in the left thalamus on FLAIR (D) which has a small area of restriction on DWI (E) and ADC (F) in keeping with a venous infarction secondary to an acute thrombus.

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**Fig. 11:** 45 yr female presents with a 2 day history of facial paraesthesia, headache and vomiting. The subacute thrombus remains hyperdense on unenhanced CT (A) and hypointense on T2 (D). ON FLAIR there is increased signal in the left sigmoid sinus (B) with the superior sagittal sinus, left transverse sinus, left sigmoid sinus and left internal jugular vein missing on 3D TOF MRV (C and F). Surpigenous blooming on susceptibility weighted imaging over the cerebral convexity is in keeping with cortical vein thrombosis (E).

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Fig. 12: 21 yo female presents with 3 day history papilloedema and headache. There is increased signal intensity on FLAIR (A), T1 (B) and T2 (C) imaging in the right transverse and sigmoid sinuses with blooming on susceptibility weighted imaging (D), filling defect on 2D TOF MRV (E) and attenuation of the sinuses on 3D TOF MRV (F). This is in keeping with a late subacute thrombus.

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**Fig. 13:** 62 yo male known to be a factor V leiden heterozygote presents with progressing headache over the preceding 17 days. This chronic thrombus is isointense on T1 (E) and T2 (B) with FLAIR hyperintensity (A and C). Again the filling defect and absent sinus are evident on 2D TOF (D) and 3D TOF MRV (F).

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Table 2: MR signal intensity of aging blood product.

<table>
<thead>
<tr>
<th></th>
<th>Duration</th>
<th>T1</th>
<th>T2</th>
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<tbody>
<tr>
<td>Hyperacute</td>
<td>&lt; 24 hrs</td>
<td>Iso</td>
<td>Hyper</td>
</tr>
<tr>
<td>Acute</td>
<td>1-3 days</td>
<td>Iso</td>
<td>Hypo</td>
</tr>
<tr>
<td>Early subacute</td>
<td>&gt; 3 days</td>
<td>Hyper</td>
<td>Hypo</td>
</tr>
<tr>
<td>Late subacute</td>
<td>&gt; 7 days</td>
<td>Hyper</td>
<td>Hyper</td>
</tr>
<tr>
<td>Chronic</td>
<td>&gt; 14 days</td>
<td>Iso</td>
<td>Hyper/Iso</td>
</tr>
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Fig. 14: Correlation of duration of presenting symptoms with expected thrombus age. Patients were stratified into hyperacute, acute, late subacute, chronic thrombus age groups based on thrombus signal intensity which correlated with duration of presenting symptoms.

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Conclusion

Cerebral venous sinus thrombosis is a rare and life threatening condition, which requires rapid detection and treatment to reduce morbidity and mortality. CECT and MRI have provided accurate diagnosis and detection of subsequent secondary changes in the cerebral parenchyma in all cases indentified in our patient group. The spectrum of imaging findings have been demonstrated in this group of 31 cases with thrombus MR signal intensity correlating thrombus age with duration of patient symptoms. With improving imaging techniques the role of angiography may be limited to therapeutics as opposed to its historical diagnostic role.
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References


