

MRI and MRV in Cerebral Venous Thrombosis

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Abstract

Although Cerebral venous thrombosis is an uncommon cause of stroke among the young, it is being increasingly recognised. A prothrombotic risk factor is identified in the majority of the patients. In most instances it is seen in women in the post-partum period and those on oral contraceptives. Magnetic Resonance Imaging (MRI) has improved our ability to diagnose this condition however the variability of radiological and clinical presentation remains a challenge. MR in combination with MR venography (MRV) is the single most sensitive diagnostic technique. The MR appearance of the thrombus within the dual sinus or cortical vein is variable and is largely dependent on its age. The loss of the normal flow void on spin echo T2 images is a sensitive parameter. Thrombus on MRV is seen as loss of high flow signal from the sinus. Focal parenchymal changes occur in approximately 50% of cases and are due to oedema and infarction, with or without haemorrhage. Appearances of these lesions too are dependent on their age. Diffuse changes of raised intracranial pressure with gyral effacement may also be present.

MR in Cerebral Venous Thrombosis

Cerebral venous thrombosis (CVT) is an uncommon disorder. The improvements in imaging techniques and increased awareness among the clinicians and radiologists have however led to the diagnosis being considered more often. As the clinical course and the radiological findings are both highly variable, diagnosis remains difficult. Magnetic Resonance (MR) imaging has assumed the central role in the diagnosis and follow-up of these patients.¹

Venous strokes as opposed to arterial strokes are primarily a disease of the young, with young adults and children being most often affected. Prothrombotic risk factors can be identified in approximately 85% of patients. Infections, dehydration, pregnancy and oral contraceptive use are among the more common acquired risk factors. Three quarters of the adult patients are women. This increased risk seems to be entirely associated to oral contraceptive use and peri/post partum period.¹

The Normal Venous Anatomy and MR Venography

The cerebral venous system is composed of superficial and deep channels. The superficial system consists of superior and inferior saggital sinuses along with the superficial cortical veins. The deep system consists of the deep cerebral veins, the vein of Galen, the straight sinus, the transverse and sigmoid sinuses.

The arrangement of the cortical veins is highly variable. They can be divided into three groups, the anterior group draining into the cavernous sinus, the lateroventral group draining into the lateral sinus, and the mediadorsal group draining into the superior saggital sinus. There are multiple anastomotic channels which are equally variable. The more constant parts of this anastomotic network are the veins of Labbe, which connect the middle cerebral veins to the lateral sinus and the great vein of Trolard, connecting the middle cerebral veins to the superior saggital sinus.

The superior saggital sinus (SSS) commences at the foramen cecum anteriorly and runs along the inner surface of the calvarium in the mid saggital plane to the internal occipital protuberance. The anterior part is narrow and may be completely absent. The SSS drain the superficial surface of the cerebral hemispheres via the cortical veins.

At the internal occipital protuberance, the SSS joins the straight sinus and divides into the two transverse sinuses running laterally. The confluence of sinuses at the internal occipital protuberance is known as the trochlea of Herophili. The sizes of the lateral sinuses are often asymmetric with the left transverse sinus being smaller (or even absent) than the right one. The transverse sinuses run forward to become the sigmoid sinuses which in turn drain into the internal jugular veins.

The straight sinus is formed by the inferior saggital sinus and the great vein of Galen. The vein of Galen in turn is formed by the confluence of the deep cerebral veins and the basal veins (of Rosenthal).

The veins of the posterior fossa also show considerable variation in their arrangement. They may be divided into three groups. Anterior group; draining to the petrosal sinuses, posterior group; draining to the straight

sinus and the trocula and the superior group; draining to the vein of Galen.

In addition to the arrangement of the venous channels, understanding their structure is also important in understanding the patho-physiology of CVT. The cerebral venous channels lack valves. This allows bidirectional flow of blood and lets potentially infected blood from the scalp and mastoids to enter the intracranial circulation. Absence of tunica muscularis allows the veins to distend and remain dilated in response to even extended occlusion. Arachnoid granulations project into the dural sinuses and are the site of absorption of the cerebro-spinal fluid.²

MR venography is a non invasive technique which

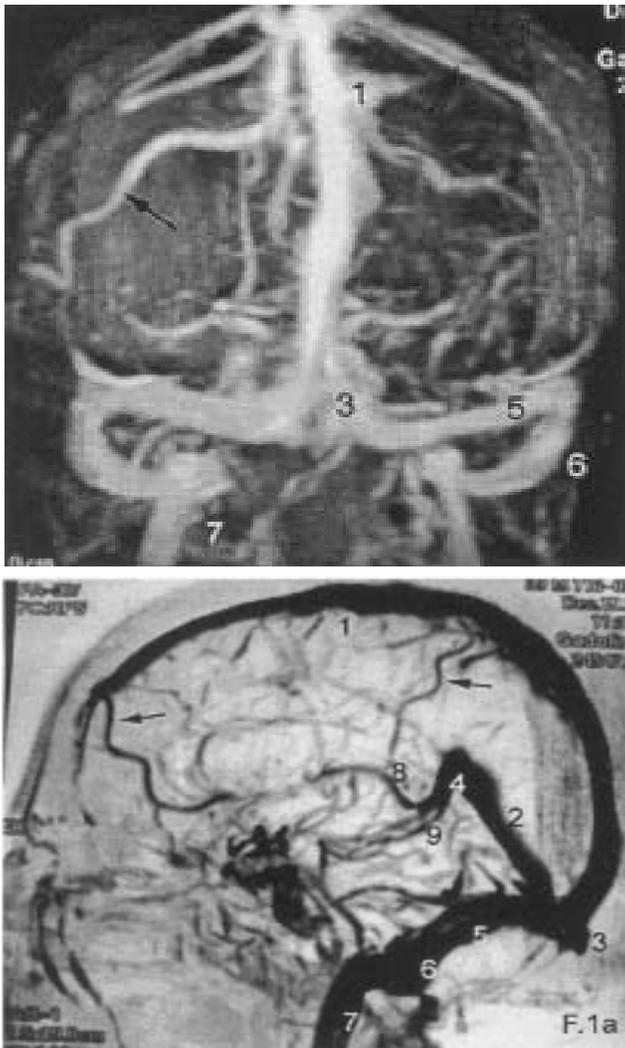


Figure 1. Normal cerebral magnetic resonance venogram. Three-dimensional time of flight images obtained after intravenous injection of gadolinium in the sagittal (A) and coronal (B) planes. There is visualization of dural venous sinuses and superficial and deep cerebral veins. 1 = superior sagittal sinus, 2 = straight sinus, 3 = torcular herophili, 4 = vein of Galen, 5 = lateral sinus, 6 = sigmoid sinus, 7 = internal jugular vein, 8 = internal cerebral vein, 9 = basal vein of Rosenthal. The arrows point to superficial cerebral veins. (From: Wasay M, Azeemuddin M. Neuroimaging of cerebral venous thrombosis. *J Neuroimaging*, 2005;15:118-128.)

allows visualisation of the intracranial venous channels. Although this may be done without the use of intravenous contrast, contrast based techniques improve resolution.³ Studies have shown contrast based techniques to be better than non contrast techniques and equivalent to conventional digital subtraction angiographic (DSA) images in the visualisation of cerebral venous anatomy. (Figure 1).⁴

The great variations in the venous anatomy however make interpretation of images especially in the context of CVT difficult. Non visualisation or asymmetry of cortical veins and transverse sinuses especially need to be critically analysed.

MR Appearances in Cerebral Venous Thrombosis (CVT)

MR has been used in the diagnosis of CVT for approximately two decades⁵ and is the modality of choice when the diagnosis of CVT is considered clinically.^{1,4,6,7} The most common sites of involvement are the transverse sinuses and the SSS, but any venous structure may be involved either in isolation or in combination with another structure.⁸

The MR features are varied. This variability reflects the spectrum of patho-physiological changes seen after venous occlusion. There are two primary processes. The first process is the development of localised oedema and venous infarction. This is usually seen after the occlusion of cortical veins. The second process is the development of intra-cranial hypertension. This usually occurs due to the occlusion of large dural venous sinuses. These two processes are not exclusive and often occur together.^{1,4,9}

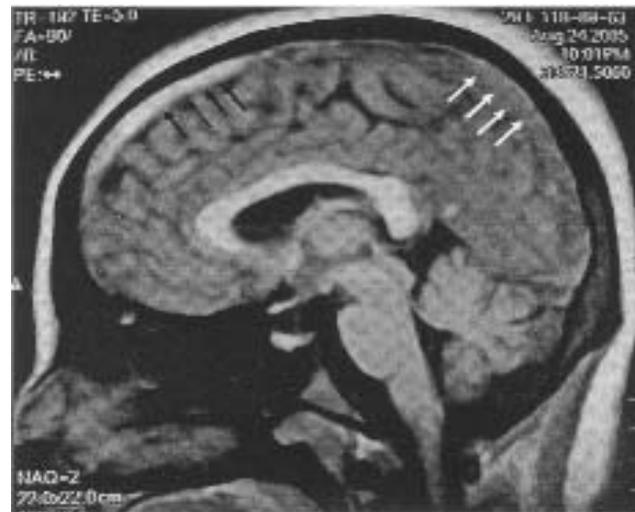


Figure 2. Non Enhanced T1 weighted sagittal image. There is extensive thrombus in the superior sagittal sinus. The thrombus is giving variable signal with the older thrombus in the anterior part giving a hypertintense signal (Black arrows, and fresh thrombus in the posterior part giving isointense T1 signal (Black arrows).

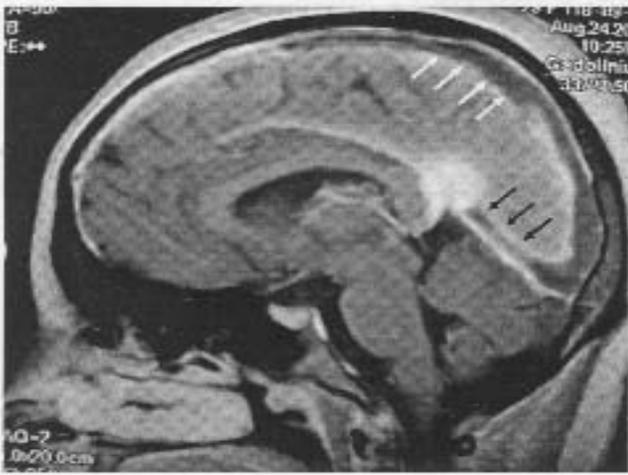


Figure 3. T1 post contrast sagittal image (Same patient as Fig 2). Extensive filling defect due to thrombus in the superior sagittal sinus (White arrows) and the straight sinus (Black arrows).

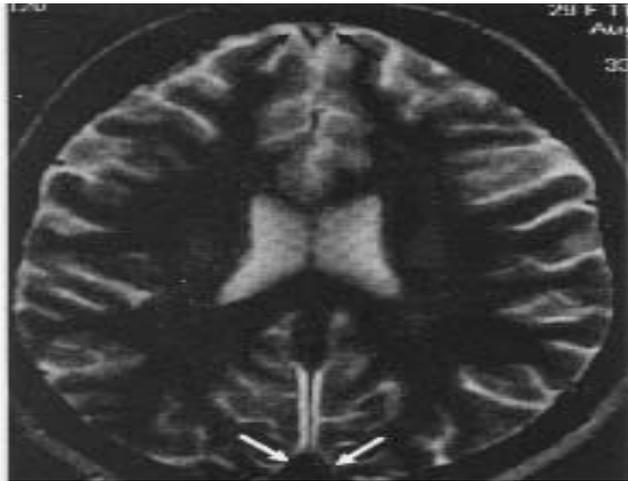


Figure 4. T2 weighted axial image. There absent signal void in the anterior superior sagittal sinus (Black arrows) representing thrombus, with normal flow void (White arrows) in the posterior part of the superior sagittal sinus.

Changes in the Venous Structures and MR Venography

MR in combination with MR venography is the single most sensitive diagnostic technique.^{1,3,4,6,7,9} Demonstration of the thrombus in the dural sinus or vein is pathognomonic of CVT. The signal from the thrombus within the dural sinus or cortical vein is variable (Table 1).⁴ However by the time most patients are scanned the thrombus shows hyper-intense signal both on T1 as well as T2 weighted images (Figure 2 and 3). The T2 signal

Table 1. Signal from intra-luminal thrombus depending on age of thrombus.

Age of Thrombus	T1	T2	Reason
Less than 5 days	Iso	Hypo	Deoxyhaemoglobin
5-15 Days	Hyper	Hyper	Methaemoglobin
Longer than 15 days	Low	Low	Re-Cannalisation



Figure 5. Contrast enhanced MRV (Same patient as Fig 2) demonstrating patchy, turbulent flow in the superior sagittal sinus (White arrow heads) and absent flow in the straight sinus (White arrows).

abnormality which replaces the normal signal void from the sinus is a more reliable sign of thrombosis (Figure 4) as very slow flowing blood may occasionally give rise to give high signal on spin echo T1 weighted images even in the absence of any thrombus. Additionally the thrombus may be iso-intense on T1 weighted images in the first 5 days.^{1,4,10} Therefore the loss of the normal flow void on spin echo T2 images is a much more sensitive parameter. Care should however be taken to ensure that the image being evaluated is a spin echo T2 (TR>1500ms, TE>75ms) sequence as gradient echo images which use short TR are prone to the same pitfall.¹⁰

Thrombus on MRV is seen either as loss of high flow signal from the sinus, in cases of complete occlusion of the sinus or frayed and patchy flow signal in the presence of non occlusive thrombus (Figure 5). The demonstration of thrombus in cortical veins is very difficult due to the high degree of anatomic variations.

Parenchymal Changes

The parenchymal changes are of two types. Changes secondary to intracranial hypertension and focal changes of oedema and infarction.

Changes secondary to intracranial hypertension are non-specific, with generalised cerebral oedema with effacement of basal cisterns and sulci. The size of ventricles is variable but is usually enlarged.

Focal changes occur in approximately 50% of cases. The location of the parenchymal changes may not correspond to the site of the sinus involved.¹¹ However the lesions do correspond to cortical or deep venous involvement¹² (Figure 6). Focal gyral oedema and effacement are the earliest parenchymal changes seen in cases of CVT. These appear as hyper-intense areas on T2

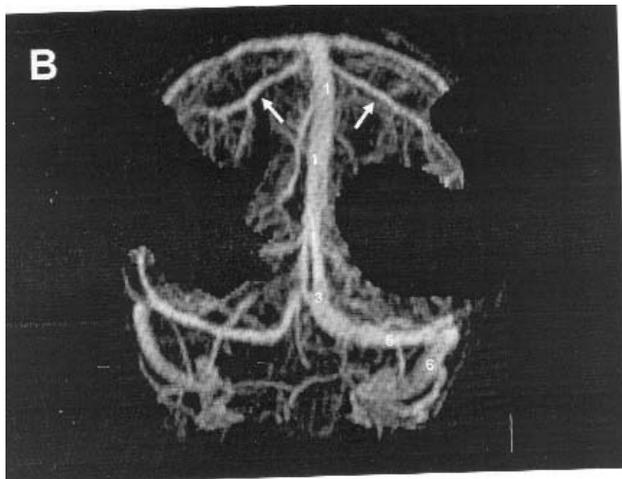
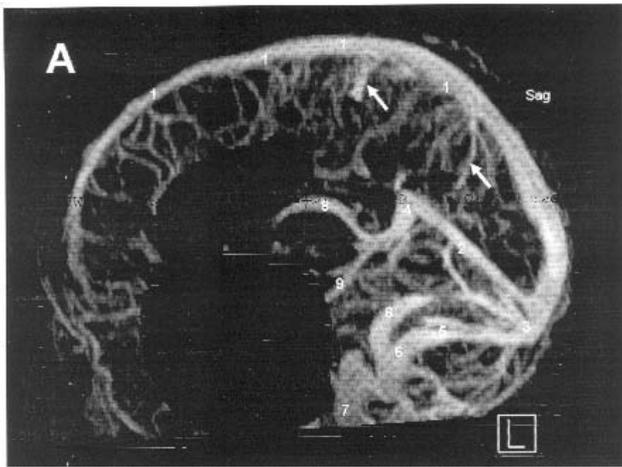


Figure 6. 6A: T2 weighted axial image. Bilateral hyperintense signal in the thalami representing infarctions (Black arrows). No evidence of haemorrhage or cortical abnormalities. 6B: DSA venography demonstrating normal dural venous sinuses but complete absence of flow in the deep venous system (Black arrows) representing thrombosis.

Table 2. MR signal from parenchymal haematomas depending on the age of the haematoma.

Age of Haematoma	T1	T2	Reason
Less than 24 hours	Iso	Hyper	Oxyhaemoglobin
1-3 days	Hypo	Hypo	Deoxyhaemoglobin
3-7 days	Hyper	Hypo	Intra-cellular methaemoglobin
8-14 days	Hyper	Hyper	Extra-cellular methaemoglobin
Longer than 14 days	Hypo	Hypo	Haemosidrin

and FLAIR sequences. These changes may persist for up to six months after the resolution of clinical symptoms.¹² They do not necessarily proceed to venous infarction. Infarction may occur with or without haemorrhage. The haemorrhages are usually seen in the para-sagittal location and may be considerably distal to the visualised thrombus. The infarcts are typically multiple and do not correspond to arterial territories. Thrombosis of the Galenic system and the deep cerebral veins leads to thalamic infarction which is usually patchy and bilateral.

The appearances of these lesions are dependent on their age and the presence of haemorrhagic component. Non haemorrhagic infarcts are progressively hyper-intense on T2 and hypo-intense on T1 weighted images. Haemorrhagic infarcts follow the pattern of other cerebral haematomas (Table 2). Recent studies have shown T2* images to be superior to spin echo T2 images in the diagnosis of CVT.¹³

Conclusion

CVT is a potentially fatal condition which if recognised and treated appropriately has excellent prognosis.⁸ MRI with MRV is the best diagnostic modality for CVT. The combination of hyper intense T1 and T2 signals from the sinus, associated with a defect seen on the MR venogram, in the appropriate clinical setting are diagnostic of CVT. These changes may be seen with or without parenchymal changes.

Caution should be exercised in the interpretation of the MR images to avoid potential pitfalls.

References

1. Stam J. Thrombosis of cerebral veins and sinuses. *N Engl J Med* 2005;352:1791-8.
2. Williams PL, Warwick R, eds. *Veins of the Brain in Gray's Anatomy*, 35th ed. Longman Group, 691.
3. Liang L, Korogi Y, Sukahara T, Onomichi M, Shigematsu Y, Yang D, et al. Evaluation of the intracranial dural sinuses with a 3D contrast enhanced MP-RAGE sequence: Prospective comparison with 2D-TOF MR venography and digital subtraction angiography. *Am J Neuroradiol*, 2001;22:481-92.
4. Wasay M, Azeemuddin M. Neuroimaging of cerebral venous thrombosis. *J Neuroimaging* 2005;15:118-28.
5. Macchi PJ, Grossman RI, Gomori JM, Goldberg HI, Zimmerman RA, Bilaniuk LT, et al. High field MRI of cerebral venous thrombosis. *JCAT* 1986;10:10-15.
6. Lafitte F, Boukobza M, Guichard JP, Hoefel C, Reizine D, Illet O, et al. MRI and MRA or the diagnosis and follow up of cerebral venous thrombosis. *Clin Radiol* 1997;52:672-79.
7. Dormont D, Anxionnat R, Evrard S, Louaille C, Chiras J, Marsault C. MRI in cerebral venous thrombosis. *J Neuroradiol* 1994;21:81-99.
8. Ferro JM, Canhao P, Stam J, Bousser MG, Barinagarrementeria F; for the ISCVT investigators. Prognosis of cerebral vein and dural sinus thrombosis. Results of the international study on cerebral vein and dural sinus thrombosis (ISCVT) *Stroke*. 2004;35:664-70.
9. Connor SEJ, Jarosz JM. Magnetic resonance imaging of cerebral venous thrombosis. *Clin Radiol* 2002;57:449-61.
10. Ayanzen RH, Bird CR, Keller PJ, McCully FJ, Theobald MR, Heiserman JE. Cerebral MR venography, normal anatomy and potential diagnostic pitfalls. *Am J Neuroradiol* 2000;21:74-8.
11. Bergui M, Bradac GB, Daniele D. Brain lesions due to cerebral venous thrombosis do not correlate with sinus involvement. *Neuroradiology* 1999;41:419-24.
12. Rottger C, Trittmacher S, Gerriets T, Bales F, Kaps M, Stotz F, et al. Reversible MR imaging abnormalities following cerebral venous thrombosis. *Am J Neuroradiol*, 2005;26:607-13.
13. Selim M, Fink J, Linfante I, Kumar S, Schlaug G, Caplan LR. Diagnosis of cerebral venous thrombosis with echo-planar T2*-weighted magnetic resonance imaging. *Arch Neurol* 2002;59:1021-26.
14. Isensee C, Reul J, Thorn A. Magnetic resonance imaging of thrombosed dural sinuses. *Stroke* 1994;25:29-34.