

Cerebral venous thrombosis: making the most of imaging

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Radiological diagnosis of cerebral venous thrombosis is aimed at confirming the presence of thrombus within the venous system and identifying any secondary effects of the thrombosis upon the brain parenchyma or cerebrospinal fluid pathways. The techniques include standard arterial catheter angiography, magnetic resonance imaging (MRI), MR venography (MRV) and computed tomography (CT). Because of the varied and confusing ways in which cerebral venous disease presents, clinicians and radiologists need a simple, accurate test that can either confirm or reliably exclude it. For many years, thrombosis within the venous system was diagnosed from the venous phase of a cerebral angiogram. Although new non-invasive methods were assessed against this 'gold standard' they are probably superior.

MRI IMAGING

MRI is widely used in the diagnosis of cerebral venous thrombosis but has important limitations. MR venography, the first non-invasive technique to simulate cerebral angiography, is performed either by time-of-flight or by phase-contrast techniques, each of which has limitations.

With the T1 weighted time-of-flight MRV technique, blood which is flowing at the preset rate expected for venous blood is bright. However, if the thrombus is already bright on T1 weighting (subacute) then a clot will simulate flow in the venographic sequence^{1,2}. If a vein is acutely thrombosed the MRV shows no signal or flow at the expected anatomical site of that vein. Lack of flow, however, may be due to anatomical absence of the vein, so one must then perform axial and/or sagittal T1 and T2 weighted MR imaging to confirm that the vein in question is present. In some cases a small transverse sinus can be identified only by sagittal imaging³. T1 and T2 imaging is necessary to show clot within the vein, since absence of flow in a vein on MRV does not necessarily equate with thrombosis, nor does the apparent presence of flow signify normality or absence of thrombosis.

Signal characteristics on T1 and T2 weighted imaging vary with the age of the clot. The signals from thrombus of 1–7 days' maturity are very similar to those seen in normal veins, in which the signal varies with the degree of

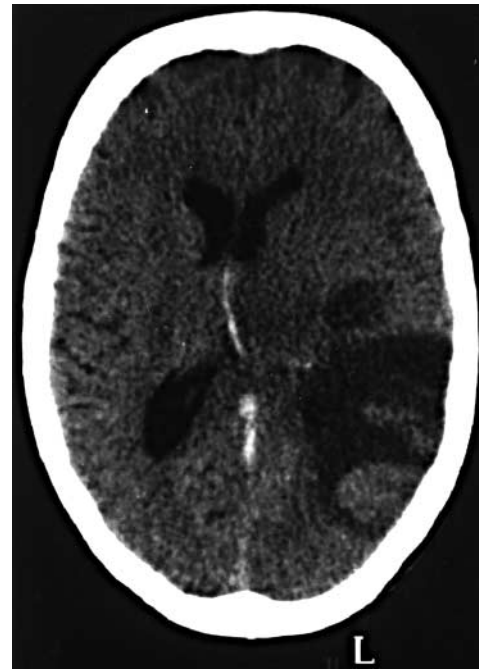


Figure 1 Cord sign in internal cerebral veins, vein of Galen and straight sinus. The sagittal sinus is not hyperdense because that clot is older and has been present for several days. Note the extensive low-attenuation venous infarction in the left temporoparietal area and the more subtle more recent ischaemia in the thalamus secondary to acute propagated clot in the deep venous system

turbulence or sluggishness of the flow and with inflow/outflow effects as the blood moves into and out of the magnetic field. By the second week and during the subsequent subacute phase, the thrombus usually produces a bright signal on both T1 and T2 imaging in the plane of the thrombosed vein or sinus⁴.

Demonstration of subacute thrombosis is straightforward with MR but in the acute phase MRV will often be needed to prove or exclude the presence of thrombosis. The subtleties of the signal changes due to the age of the clot and the difficulties of interpreting the venogram demand a carefully selected multisequence protocol in all cases; moreover, the observer has to be very expert in interpreting signals obtained with the particular scanner with which he or she is working⁴.

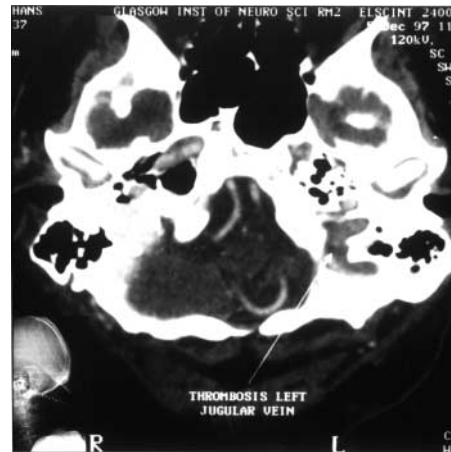
Difficulty can also arise from the demands of the techniques on the patient. The imaging, which requires

complete stillness, takes in excess of 30 minutes even with modern scanners, and many patients with acute venous thrombosis are unable to cooperate. MR techniques are better suited to the examination of relatively well patients in the chronic stage of the disease.

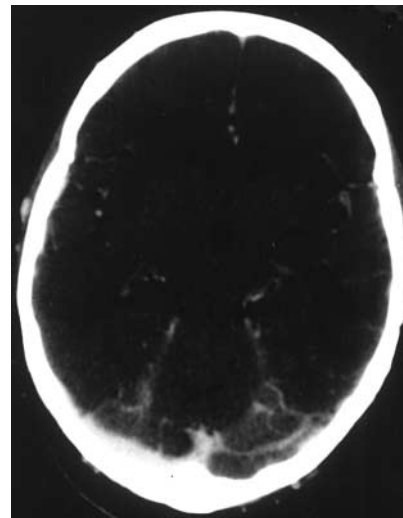
MR imaging is more sensitive than CT in the detection of the associated parenchymal changes, particularly in identifying microhaemorrhage, which is one of the hallmarks of venous infarction, much more frequent in venous disease than arterial disease^{5,6}. Nevertheless, CT displays the great majority of parenchymal abnormalities, and detection of microhaemorrhage on its own will seldom suffice to confirm the venous origin.

RESURGENCE OF CT

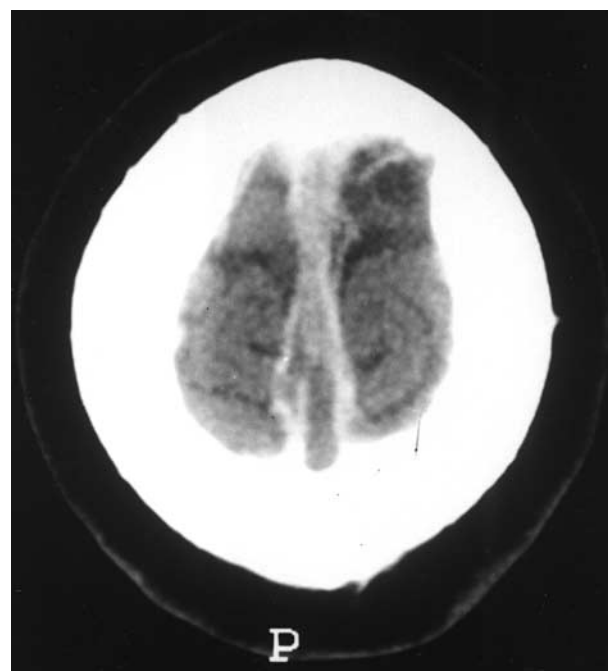
During the 1980s CT scanning was routinely used in the diagnosis of cerebral venous disease. Two signs reported at that time remain accepted as pathognomonic of thrombosis. Because they were originally found only in a minority of patients with cerebral venous thrombosis⁷ there were doubts about their value in routine diagnosis; however, it became clear that with thinner CT slices and higher doses of intravenous contrast these signs could be detected much more frequently⁸. The first CT sign is the *cord* or *dense triangle* sign^{9,10}, which refers to the presence of hyperintense acute thrombus within a vein or sinus (Figure 1). Since the cord sign represents newly formed thrombus it will seldom be seen in patients with subacute or chronic disease. It was present in 55% of patients in a Glasgow series who had unenhanced CT. The second sign is the *empty delta* sign which indicates mature thrombus in the dura-covered sinuses; the name refers to the triangle of enhancing dura surrounding non-enhancing thrombus¹⁰ (Figure 2). The exact cause of the enhancement is not established; it may be due to contrast enhancement of the dural covering or the presence of small venous collaterals within the walls of the sinuses, or it may represent partial recanalization of the sinus. With helical scanning, use of very thin sections, and a high dose of intravenous contrast, the venous sinuses and cerebral veins are demonstrated clearly¹¹⁻¹³. The veins can be examined on the axial acquired, or source, images and these images can be post-processed by a maximum intensity projection (MIP) technique. This produces a pseudo-three-dimensional projection of the veins which superficially resembles the flow venograms provided by MRV and cerebral angiography¹¹. The venous images are however not



(a)



(b)



(c)

Figure 2 (a) Non-enhancement in a thrombosed non-dominant left jugular vein; (b) non-enhancing thrombus in a dominant left transverse sinus (delta sign); (c) filling defect of established non-enhancing thrombus in the sagittal sinus (note that cortical veins still enhance normally)

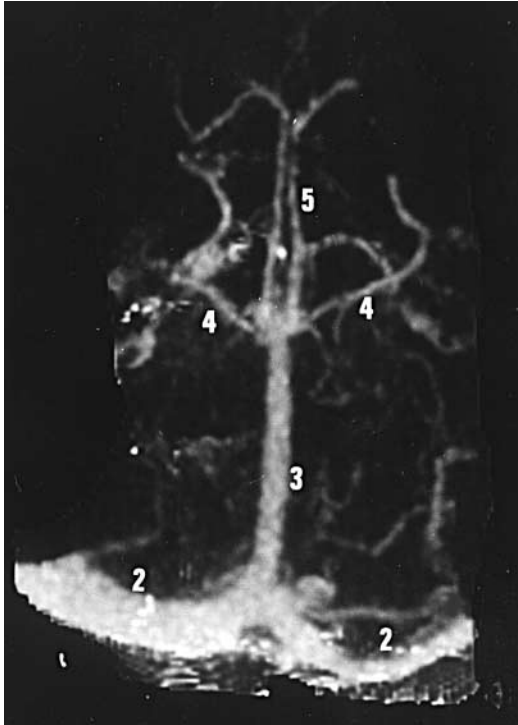


Figure 3 Computerized tomography venogram, axial projection, showing the appearance of normal transverse sinuses (2), straight sinus (3), basal veins of Rosenthal (4) and the paired internal cerebral veins (5)

produced by flow but by the enhanced wall and contents of the sinuses (Figure 3).

Since 1997 a CT technique similar to that described by Casey¹¹ has been used routinely in the Institute of Neurological Sciences, Glasgow, and the number of patients diagnosed as having venous thrombosis has increased from 2–3 to 10 per year—seemingly a higher rate than is obtained by researchers who use MR and angiography^{14–16}. Our studies show that normal veins and sinuses can be detected just as well by non-specialist radiologists as by consultant neuroradiologists. The speed and simplicity of the CT technique means that it can be performed even on acutely ill restless patients and it can be read by non-specialist radiologists. These features increase the likelihood of diagnosing venous thrombosis in the acute stage, at a time when treatment is most likely to be effective.

DISTRIBUTION OF THROMBOSIS

Cerebral venous thrombosis is often referred to as sagittal sinus thrombosis because the sagittal sinus is presumed to be most frequently involved. In fact, of 40 consecutive patients with venous thrombosis seen at the Institute of Neurological Sciences, Glasgow, 39 had thrombosis of the transverse sinus. Of these, about half also had thrombosis of the sagittal sinus, and just under half of those with sagittal sinus thrombosis had involvement of the deep venous

system. These observations indicate that thrombosis progresses from proximal to distal veins. In the Glasgow series there were no patients who had isolated thrombosis involving only the deep venous system. In each of the 12 patients with deep venous thrombosis there was thrombosis of the vein into which the straight sinus drained. In 11 patients this was the sagittal sinus at the torcula, and in one patient it was the thrombosed non-dominant transverse sinus into which the straight sinus drained exclusively. This venous arrangement is a common anatomical variant, present in about one-third of people¹⁷. Careful scrutiny of the illustrations and the text of previous reports of isolated deep vein thrombosis shows that many patients had thrombosis proximal to the deep veins and the word 'isolated' may refer to the lack of sagittal sinus thrombosis^{18–20}. Isolated deep venous thrombosis, occurring without thrombosis of the sinus draining the straight sinus, seems to be extremely rare. A systematic review of the appearances of all veins on the source images is necessary to determine the presence and degree of patency of veins. This is not possible with cerebral angiography, MRV or MR because none can exclude the presence of thrombus in a partially recanalized sinus or vein.

Analysis of the distribution of the delta and the cord signs on CT discloses additional patterns. The delta sign was present in all 30 patients who had CT with contrast enhancement; it was present in the transverse sinus in 26 patients, the sagittal sinus in 15 and the straight sinus in only 3. The cord sign, signifying acute thrombosis, was present in 18 of the 32 patients who underwent CT scan without contrast enhancement. The cord sign was present in the sagittal sinus in 14, the straight sinus in 9, the vein of Galen in 8 and the internal cerebral veins in 6 but it was seen in only 10 of the transverse sinuses and 6 of the sigmoid sinuses. In other words, the distribution of the cord sign is the converse of that of the delta sign. This observation supports the concept of propagation of thrombosis from mature clot in the proximal sinuses to acute thrombus in the distal sinuses and veins. It also indicates that proximal clot may be tolerated by the patient, with secondary or propagated acute thrombosis in the distal veins precipitating admission to hospital.

The propagation of thrombus and hence the clinical presentation was also influenced by anatomical factors. If there was thrombosis of the non-dominant transverse sinus, usually the left, the patient most often presented with symptoms of ischaemia involving the temporoparietal region and with imaging evidence of posterior temporal ischaemia. This is presumed to be due to extension of the venous thrombosis into the vein of Labbe draining into the non-dominant sinus. In no patient with thrombosis of the non-dominant transverse sinus was there sagittal sinus thrombosis. Conversely, when there was thrombosis of the

dominant or equally dominant transverse sinus there was almost always thrombosis of the sagittal sinus. This is the common pattern found in patients who present subacutely or chronically with idiopathic intracranial hypertension.

Caution is needed in interpretation of CT images post-processed with the MIP projection. Because the projection uses, as its name suggests, the brightest pixel to project the image of the veins, the enhancing wall of a thrombosed sinus can be displayed as an apparently patent sinus. Cerebral angiography, too, can be misleading: when there is partial recanalization of a sinus, it can show apparently normal flow within the sinus when cross-sectional CT images have been shown to contain clot within a partly recanalized sinus. The accolade of 'gold standard' should probably go to the source images of high-resolution dynamic CT scanning, since these more definitively and directly confirm the presence of clot.

CONCLUSIONS

CT conducted with a detailed technique that includes plain and dynamic sequences and careful analysis of the source images is a simple and effective method to diagnose acute, subacute and chronic venous thrombosis. The new data from CT indicate that the disease usually starts in the proximal sigmoid and transverse sinuses and that deep vein thrombosis does not occur without thrombosis of the vein into which the straight sinus drains. Thrombosis of the non-dominant lateral sinus presents most frequently with focal signs of cerebral ischaemia, whereas thrombosis of a dominant lateral sinus progresses to involve the sagittal sinus. CT is a much less complex technique than MR and is practicable in many acute situations to confirm or exclude thrombosis. The best application of MRI may be in the follow-up of patients already shown by CT to have disease of the cerebral veins.

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