CLINICAL APPLICATIONS OF VASCULAR IMAGING

Imaging of Cerebral Venous Thrombosis: Current Techniques, Spectrum of Findings, and Diagnostic Pitfalls

James L. Leach, MD • Robert B. Fortuna, MD • Blaise V. Jones, MD
Mary F. Gaskill-Shipley, MD

Cerebral venous thrombosis is a relatively uncommon but serious neurologic disorder that is potentially reversible with prompt diagnosis and appropriate medical care. Because the possible causal factors and clinical manifestations of this disorder are many and varied, imaging plays a primary role in the diagnosis. Magnetic resonance (MR) imaging, unenhanced computed tomography (CT), unenhanced time-of-flight MR venography, and contrast material–enhanced MR venography and CT venography are particularly useful techniques for detecting cerebral venous and brain parenchymal changes that may be related to thrombosis. To achieve an accurate diagnosis, it is important to have a detailed knowledge of the normal venous anatomy and variants, the spectrum of findings (venous sinus thrombi and recanalization, parenchymal diffusion or perfusion changes or hemorrhage), other potentially relevant conditions (deep venous occlusion, isolated cortical venous thrombosis, idiopathic intracranial hypertension), and potential pitfalls in image interpretation.

© RSNA, 2006

Abbreviations: ADC = apparent diffusion coefficient, GRE = gradient-recalled echo, MIP = maximum intensity projection, TOF = time of flight

RadioGraphics 2006; 26:S19–S43 • Published online 10.1148/rg.26si055174 • Content Codes: CT MR NR

1From the Department of Radiology, University of Cincinnati College of Medicine, 234 Goodman St, Cincinnati, OH 45246 (J.L.L., R.B.F., M.F.G.S.); the Neuroscience Institute, Cincinnati, Ohio (J.L.L., M.F.G.S.); and Cincinnati Children’s Hospital Medical Center, Cincinnati, Ohio (B.V.J.). Recipient of a Cum Laude award for an education exhibit at the 2004 RSNA Annual Meeting. Received September 19, 2005; revision requested January 13, 2006 and received February 17; accepted April 6. All authors have no financial relationships to disclose. Address correspondence to J.L.L. (e-mail: james.leach@uc.edu).

See also the article by Rodallec et al (pp S5–S18) and the commentary by Phillips (pp S42–S43) in this issue.

© RSNA, 2006
Introduction
Cerebral venous thrombosis is a relatively uncommon disorder, with an estimated annual incidence of between two and seven cases per million in the general population (1). The incidence likely was underestimated before the advent of accurate noninvasive imaging methods. It is estimated that five to eight cases may be seen per year at a typical tertiary-care referral center (2). Accurate and prompt diagnosis of cerebral venous thrombosis is crucial, because timely and appropriate therapy can reverse the disease process and significantly reduce the risk of acute complications and long-term sequelae. Since the possible causal factors and clinical manifestations of thrombosis are many and varied, imaging plays a primary role in the diagnosis. A wide range of cross-sectional imaging methods and venographic techniques may be used to detect abnormalities in the brain parenchyma as well as the cerebral veins and venous sinuses. This article provides a survey of common findings in cerebral venous thrombosis and in several other disorders that may include a venous thrombotic process as a component. To provide a context for the discussion of abnormal findings, the normal venous anatomy and variants also are reviewed, and potential pitfalls related to image interpretation are described.

Causal Factors
More than 100 causes of venous thrombosis have been described in the literature (1). Causal factors may be classified as local (related to intrinsic or mechanical conditions of the cerebral veins and dural sinuses) or systemic (related to clinical conditions that promote thrombosis). Local processes that alter the venous flow (e.g., sinus trauma, regional infection such as that in mastoiditis, and neoplastic invasion or compression) may potentiate the development of thrombosis. Systemic causes include protein S and protein C deficiencies, a peripartum state, oral contraceptive use, and hypercoagulable states secondary to malignancy. In as many as 25% of cases, no cause is identified (1).

Clinical Manifestations
The clinical manifestations of cerebral venous thrombosis vary, depending on the extent, location, and acuity of the venous thrombotic process as well as the adequacy of venous collateral circulation (2). Generalized neurologic symptoms (e.g., headache, experienced by 75%–95% of patients) and focal neurologic deficits, including seizure, may result. Focal neurologic symptoms are more often seen in patients with parenchymal changes observed at imaging than in those without such changes. Because thrombosis and endogenous thrombolysis and recanalization may occur concurrently, the clinical manifestations may fluctuate in as many as 70% of patients, adding to clinical uncertainty (2). Intracranial hypertension occurs in 20%–40% of patients with cerebral venous thrombosis and should be excluded in patients with the specific complex of symptoms (2).

Anatomic Distribution
A review of the magnetic resonance (MR) imaging literature published in 1995–2004 (3–14) enables a general description of the anatomic distribution of thrombosed cerebral venous structures identified primarily at MR imaging (Table 1). Multiple locations of thrombosis, particularly in the contiguous transverse and sigmoid sinuses, are found in as many as 90% of patients (11). Cortical venous involvement is seen in 6% of patients but is likely to be underreported when the dominant imaging finding is dural sinus involvement.

Pathophysiology
The pathophysiology of brain parenchymal involvement in venous occlusion differs from that in arterial occlusion. Parenchymal changes may be
secondary to cytotoxic edema, vasogenic edema, or intracranial hemorrhage. The primary underlying mechanism is likely to be increased venous pressure. If collateral pathways of venous drainage are insufficient, especially in the presence of cortical venous involvement, subsequent parenchymal changes may occur. If venous pressure continues to increase, with a consequent diminishment in arterial perfusion pressure, cell death may ensue. If adequate collateral pathways develop or recanalization occurs before cell death or intracranial hemorrhage, the parenchymal changes may resolve partly or completely. Vasogenic and cytotoxic edema patterns may coexist (15–17).

Imaging Techniques and Findings

Normal Venous Anatomy

The intracranial venous system may exhibit a wide range of normal variations. The use of high-spatial-resolution noninvasive venographic methods at contrast material–enhanced MR imaging and computed tomography (CT) allows the visualization of a greater number of venous structures than is possible with cross-sectional (parenchymal) imaging. Greater familiarity with the details of venous anatomy and broader knowledge of anatomic variations are important for accurate interpretation of venographic images (18). The images in this section of the article were obtained with a previously described contrast-enhanced MR venographic technique (19).

According to traditional descriptions, the cerebral venous system consists of the deep venous system, superficial venous system, and dural venous sinuses (with their superior and inferior components) (18). The dural venous sinuses are enclosed in the leaves of the dura and serve as the major drainage pathway of the cerebral veins (Fig 1). The superficial veins of the cerebrum empty into the dural sinuses and are variable in morphologic structure and location. Superiorly draining (ascending) superficial veins are named for the area of cortex that they drain (20). Inferiorly draining (descending) superficial veins include the Labbé vein and the sylvian (superficial middle cerebral) veins (Fig 2). Although the venous
drainage territories of the superficial cerebral
veins are variable, general drainage areas can be
identified (21) (Fig 3).

The deep system includes the vein of Galen,
the internal cerebral veins, and their tributaries;
the Rosenthal vein (basal vein) and its tributaries;
and the medullary and subependymal veins,
which drain the hemispheric white matter (Fig 4).
The deep system drains the inferior frontal lobe;
most of the deep white matter of the frontal, tem-
poral, and parietal lobes; the corpus callosum; the
upper brainstem; the basal ganglia; and the thala-
mus (21). The parenchymal alterations that occur
with deep venous occlusion typically involve the
thalami, probably because the primary venous
pathways that drain the thalami extend directly
into the internal cerebral veins (Fig 5). The basal
dural sinuses are complex and are interconnected
with the cavernous sinus complex. Multiple emis-
sary channels in the skull base connect with the
sigmoid sinus and jugular bulb. These struc-
tures are much more commonly seen at contrast-
enhanced MR venography than at time-of-flight
(TOF) MR venography (Fig 6).

MR and CT Venography
Venous thrombi can be detected with direct visu-
alization on MR and CT parenchymal images or
with various venographic techniques. The most
commonly used venographic techniques currently
include unenhanced TOF MR venography, con-
trast-enhanced MR venography, and CT venog-
raphy. Phase-contrast MR venography is less of-
ten used, because of its dependence on operator-
deﬁned velocity encoding parameters.

TOF MR venography is the method most
commonly used for the diagnosis of cerebral ve-
nous thrombosis. Two-dimensional TOF tech-
niques are used to evaluate the intracranial ve-
nous system because of their excellent sensitivity
to slow flow and their diminished sensitivity to
signal loss from saturation effects compared with
the sensitivities of three-dimensional TOF tech-
niques (22). Because two-dimensional techniques
are most sensitive to flow that is perpendicular to
the plane of acquisition, the coronal plane, axial
Figure 3. Axial MR image series with a color
overlay represents the major superficial cortical ve-
nous drainage territories according to Meder et al
(21). Most of the superior cerebrum (green) is
drained primarily into the superior sagittal sinus,
which also receives drainage from the parasagittal
cortical regions at lower levels. The sylvian veins
drain blood from the peri-insular region (yellow)
into the basal dural sinuses. The transverse sinuses
receive blood from the temporal, parietal, and oc-
cipital lobes (blue). The Labbé vein, if dominant,
may drain much of this territory. Parenchymal ab-
normalities such as hemorrhage or edema in this
territory may be indicative of thrombosis of the
transverse sinus or Labbé vein.
and coronal planes, or an oblique plane are often used for image acquisition. Venous flow in the plane of image acquisition may produce saturation and resultant nulling of the venous signal at TOF MR venography, a potential pitfall for image interpretation and diagnosis. A close assessment of the source images is mandatory to

Figure 4. Lateral MIP image from contrast-enhanced MR venography shows the major components of the deep venous system: the thalamostriate vein (1), septal vein (2), internal cerebral vein (3), basal vein (Rosenthal vein) (4), and vein of Galen (5).

Figures 5, 6. (5) Axial MR image with color overlay shows the drainage territory of the deep cerebral veins (internal cerebral vein, vein of Galen) (pink), in which parenchymal abnormalities due to deep venous occlusion typically are found. The deep white matter (medullary) venous drainage territory (blue) also is shown. (6) Basal dural sinuses. Anteroposterior MIP image from contrast-enhanced MR venography, with the superficial and deep veins removed for better visualization, shows the cavernous sinus complex (1) and, connecting the lateral dural sinuses with the cavernous sinus, the superior petrosal sinuses (2), which arise from the junction of the transverse and sigmoid sinuses and extend along the petrous ridge, and the inferior petrosal sinuses (3), which arise from the distal portion of the sigmoid sinus or jugular bulb and extend along the clivus. Also visible are the superficial middle cerebral vein (sylvian vein) (4), which in this case extends into the cavernous sinus, and the emissary veins and occipital venous plexus complex (5).
accurately evaluate venous morphologic features and reduce the potential for diagnostic error (Fig 7).

Contrast-enhanced MR venography with elliptic centric ordering is a more recently developed venographic method in which the paramagnetic effect of intravenous gadolinium is used to shorten T1 and provide positive intravascular contrast enhancement (19). Small-vessel visualization is improved at contrast-enhanced MR venography, compared with that at TOF MR venography (19). Depiction of the dural sinuses is also superior with contrast-enhanced MR venography because of a decrease in the effects of turbulent flow on vessel contrast (Fig 7) (19).

CT venography is a rapid, readily available, and accurate technique for detecting cerebral venous thrombosis (Fig 8) (23,24). CT venography provides a highly detailed depiction of the cerebral venous system, superior to that available with conventional TOF MR venography, and has at least equivalent accuracy for the detection of cerebral venous thrombosis (23). Drawbacks of CT venography include the difficulty of reconstructing maximum intensity projection (MIP) images.
from the source image data sets, a process that requires the subtraction of bone adjacent to the venous sinus; it is very difficult to subtract all of the adjacent bone without also subtracting part of the sinus (25). However, source images and multiplanar reformatted images can be displayed quickly for evaluation. Investigators in a recent study advocated the use of a matched mask bone elimination technique to improve the quality of MIP images reconstructed from CT venographic data (26).

With the continuing development of multidetector CT, the use of CT venography probably will become more widespread. However, because the technique involves the use of ionizing radiation, CT venography may have limited use in pregnant patients and children, in whom the radiation dose may be a cause for particular concern, as well as in patients with renal failure, contrast material allergy, or another contraindication to the use of iodinated contrast material. To our knowledge, the comparative accuracies of CT venography, MR imaging, and contrast-enhanced MR venography have not yet been assessed.

Venous Sinus Abnormalities

**Thrombosis.**—The classic finding of sinus thrombosis on unenhanced CT images is a hyperattenuating thrombus in the occluded sinus (Fig 9). However, variability in the degree of thrombus attenuation makes this sign insensitive; hyperattenuation is present in only 25% of sinus thrombosis cases (27). Increased attenuation in the venous sinuses also may be seen in patients with dehydration, an elevated hematocrit level, or a subjacent subarachnoid or subdural hemorrhage. In most cases, a close comparison of sinus attenuation with arterial attenuation can help differentiate between a physiologic increase in sinus attenuation and increased attenuation due to thrombosis. Increased attenuation in the sinus may be the only finding suggestive of sinus thrombosis on unenhanced CT images, and patients with this sign should be further evaluated with contrast-enhanced CT, MR imaging, or both, in the proper clinical scenario.

A well-described finding of sinus thrombosis at contrast-enhanced imaging is the empty delta sign, a central intraluminal filling defect that
represents a thrombus surrounded by contrast-enhanced dural collateral venous channels and cavernous spaces within the dural envelope (Fig 10) (27,28). The filling defect is typically seen on multiple sections in contrast-enhanced CT and MR imaging studies. Initial reports described the appearance of the empty delta sign on CT images in 29% of patients with sinus thrombosis detected at contrast-enhanced CT (27). On thin-section helical CT images and volumetric MR images, the empty delta sign could be expected to appear in an even higher percentage of cases.

Unenhanced MR imaging is more sensitive for the detection of venous thrombi than is unenhanced CT. The absence of a flow void and the presence of altered signal intensity in the sinus is a primary finding of sinus thrombosis on MR images. Slow or turbulent flow also may cause a signal intensity alteration in the sinus. A close assessment with multiple pulse sequences should be

Table 2

<table>
<thead>
<tr>
<th>Imaging Technique and Signal Characteristic</th>
<th>0–5 Days</th>
<th>6–15 Days</th>
<th>&gt;15 Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1-weighted sequence</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperintensity</td>
<td>30 (17/57)</td>
<td>71 (57/80)</td>
<td>39 (23/59)</td>
</tr>
<tr>
<td>Isointensity</td>
<td>68 (39/57)</td>
<td>29 (23/80)</td>
<td>54 (32/59)</td>
</tr>
<tr>
<td>Hypointensity</td>
<td>2 (1/57)</td>
<td>0 (0/80)</td>
<td>7 (4/59)</td>
</tr>
<tr>
<td>T2-weighted sequence</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperintensity</td>
<td>25 (14/57)</td>
<td>52 (40/77)</td>
<td>43 (33/76)</td>
</tr>
<tr>
<td>Isointensity</td>
<td>11 (6/57)</td>
<td>32 (25/77)</td>
<td>45 (34/76)</td>
</tr>
<tr>
<td>Hypointensity</td>
<td>65 (37/57)</td>
<td>16 (12/77)</td>
<td>12 (9/76)</td>
</tr>
</tbody>
</table>

Sources.—References 3, 9, 10, 29, 30.
Note.—Data are the percentage of thrombi with the specified signal characteristic at the given time after symptom onset. The numbers in parentheses are the number of segments with the specified signal intensity among the number of segments reported within the given time interval after symptom onset. A total of 80 thrombosed venous segments were reported. Not all studies included cases for each time interval (thrombus stage) and each pulse sequence.

*The signal characteristic is the signal intensity of thrombi in relation to that of gray matter.

Figure 10. (a) Contrast-enhanced CT image in a patient with superior sagittal sinus thrombosis shows a central filling defect in the superior sagittal sinus (arrow), surrounded by intensely enhanced dura mater. (b) Coronal reformatted image from contrast-enhanced MR venography in another patient shows a nonenhanced thrombus (arrows) surrounded by enhanced sinus walls and dural cavernous spaces. The thrombus extends from the superior sagittal sinus through the sinus confluence and into the right transverse sinus.
made. In addition, the administration of contrast material and the application of venographic techniques often are necessary for confident diagnosis.

The signal intensity of venous thrombi on T1- and T2-weighted MR images varies according to the interval between the onset of thrombus formation and the time of imaging (Table 2) (3,9,10,29–32). The change in signal intensity is thought to be related to the paramagnetic effects of the products of hemoglobin breakdown in the thrombus (31,32).

In the acute stage of thrombus formation (0–5 days), the signal is predominantly isointense on T1-weighted images and hypointense on T2-weighted images because of deoxyhemoglobin in red blood cells trapped in the thrombus (Fig 11). A venous thrombus in the acute stage may have a signal intensity that mimics a normal flow state, and such a finding may lead to diagnostic error (33). The signal may be very hypointense on T2-weighted images and may be mistakenly thought to indicate a flow void. According to some estimates, in 10%–30% of cases of sinus thrombosis, the thrombus at initial presentation or imaging examination is in the acute stage of formation (3,33,34). Contrast-enhanced MR venography or CT venography is usually necessary to achieve a definitive diagnosis at this stage.

In the subacute stage of thrombus development (6–15 days), the signal is predominantly hyperintense on both T1-weighted images and T2-weighted images because of methemoglobin
in the thrombus (Fig 12) (30,32). Subacute-stage thrombus has been found in 55% of patients at clinical presentation with cerebral venous thrombosis (3,34). This stage of formation is the easiest stage at which to detect a thrombus on MR images, as the signal intensity of the sinus is most different from that in normal flow states. The finding of increased signal intensity on both T1-weighted images and T2-weighted images is almost always abnormal.

Chronic thrombosis with incomplete recanalization of the sinus may present a diagnostic challenge at MR imaging (Fig 13). As many as 15% of patients in whom sinus thrombosis is diagnosed at MR imaging may have a chronic (>15-day-old) thrombus (3,34). Compared with the MR signal in normal brain parenchyma, the signal in a chronic thrombus is typically isointense or hyperintense on T2-weighted images and isointense on T1-weighted images; however, significant variability in thrombus signal intensity exists (3,9,10,29,30). The signal intensity may be similar to that of very slowly moving oxygenated blood.

On images acquired after gadolinium administration, marked contrast enhancement may be observed that resembles the enhancement typically seen in a normal sinus (35). Sinus enhancement is presumably secondary to an organized thrombus with intrinsic vascularization (35) as well as to slow flow in dural and intrathrombus collateral channels. Contrast enhancement of the sinus on MR images does not definitively indicate patency, and venography usually is necessary for a definitive diagnosis. Although the findings on images obtained with contrast-enhanced venographic techniques reportedly have been misleading in cases of chronic sinus thrombosis (35), in our experience chronic thrombosis has been well depicted and correctly diagnosed with contrast-enhanced MR venography with elliptic centric ordering. Recanalized vessels and filling defects

Figure 12. Subacute thrombus of the superior sagittal sinus. (a, b) Axial T1-weighted (a) and axial T2-weighted (b) MR images show an area of abnormal increased signal intensity in the superior sagittal sinus (arrow). (c) Sagittal source image from contrast-enhanced MR venography shows filling defects (arrows) due to a thrombus.
that represent a nonrecanalized and nonvascularized thrombus are particularly well depicted on source images (Fig 13d). However, if image acquisition is delayed at contrast-enhanced MR venography, thrombus enhancement may increase to a level that simulates patency of the sinus (19).

As gradient-recalled echo (GRE) sequences are increasingly used in MR imaging protocols to detect the presence of blood breakdown products, their usefulness in depicting intraluminal thrombi in cerebral venous thrombosis has been appreciated (29). In stages of thrombus evolution in which paramagnetic products such as deoxyhemoglobin and methemoglobin are present, the sensitivity of GRE sequences to magnetic susceptibility produces blooming artifacts in the thrombosed venous segments on GRE images.
GRE imaging sequences may be an important diagnostic aid in acute-stage thrombosis, when the signal intensities on T1- and T2-weighted images may be more subtle (24). There has been recent interest in evaluating the appearance of intraluminal venous thrombi on diffusion-weighted images (3). Signal hyperintensity in thrombosed sinuses on diffusion-weighted images, with corresponding diminishment in the mean apparent diffusion coefficient (ADC) values, has been described in 41% of patients with sinus thrombosis (3). The duration of clinical symptoms was longer and complete recanalization was less frequent in patients with restricted diffusion in the thrombus (3).

**Figure 14.** Magnetic susceptibility effects on MR images in a 27-year-old postpartum woman with a severe headache for 6 days. (a) T1-weighted spin-echo image shows an area of isointense signal (arrowheads) indicative of a thrombus in the right transverse sinus. (b) GRE image shows significantly diminished signal intensity and apparent enlargement of the sinus in the same area (arrows). No flow was demonstrated at TOF MR venography.

**Figure 15.** Oblique MIP image from TOF MR venography in a patient with a history of extensive thrombosis of the superior sagittal sinus and left transverse sinus 2 years before. Note the irregular appearance of the superior sagittal sinus (arrows), which corresponds to incomplete recanalization of the sinus. The source images showed residual intrasinus defects, intrasinus channels, and dural collateral channels.

**Figure 16.** Frontal MIP image from axial TOF MR venography in a patient with prior extensive thrombosis and variable recanalization of the superior sagittal sinus, straight sinus, and both transverse sinuses. Veins of the face and scalp were subtracted for better visualization of the irregular cortical venous collateral vessels (arrows), which drain primarily to the superficial middle cerebral veins and to the sphenoparietal, cavernous, and inferior petrosal sinuses. Tentorial collateral vessels also are visible (arrowhead).
Recanalization.—An irregular appearance of the sinus, with multiple intrasinus channels and dural collateral vessels, may be seen on MR venographic images and is characteristic of incomplete recanalization (Fig 15) (28). Complete recanalization may occur more often in thrombosis of the superior sagittal sinus and straight sinus than in thrombosis of the transverse and sigmoid sinuses after anticoagulation therapy (4). In patients who undergo anticoagulation therapy for sinus thrombosis, progression of recanalization is not typical after 4 months of therapy (4). Complete recanalization is not necessary for clinical recovery, and the extent of recanalization may not correlate closely with the clinical outcome (5).

Numerous venous collateral pathways may form after sinus thrombosis (Fig 16). Collateral vessels may form in the dura that surrounds the occluded sinus, arising from cortical veins that drain to nonthrombosed sinuses or from intermediate veins (ie, diploic, emissary, meningeal). The intermediate veins may form a dominant collateral system in the presence of extensive thrombosis (27).

Table 3
Focal Parenchymal Abnormalities in Patients with Cerebral Venous Thrombosis

<table>
<thead>
<tr>
<th>Parenchymal Abnormality</th>
<th>No. of Patients*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edema</td>
<td>148 (25)</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>190 (32)</td>
</tr>
<tr>
<td>Total</td>
<td>338 (57)</td>
</tr>
</tbody>
</table>

Sources.—References 6–9, 13, 14, 17, 32, 34, 36.
*Numbers are based on 587 cases reported in the literature from 1999 to 2004, primarily cases diagnosed at MR imaging. Numbers in parentheses are the percentage of patients.

Parenchymal Abnormalities
Focal brain abnormalities have been identified in as many as 57% of patients with cerebral venous thrombosis (Table 3). Parenchymal lesions are better depicted and more commonly identified at MR imaging than at CT (17,30). Focal edema (without visible hemorrhage) is visible on CT images in approximately 8% of cases and on MR images in 25% of cases (6–9,13,14,17,27,32,34,36) (Fig 17). Diffusion-weighted MR imaging
techniques allow subclassification of parenchymal abnormalities as either primarily vasogenic edema (with increased ADC values presumably related to venous congestion) or primarily cytotoxic edema (with decreased ADC values related to cellular energy disruption). Hemorrhage may occur with both types of edema, and various patterns may coexist in the same region (7,8). In view of the variable nature of the parenchymal abnormalities that may occur in cerebral venous thrombosis, the use of the term venous infarct in reference to these lesions should be discouraged because that term implies irreversibility. In contrast with arterial ischemic states, many parenchymal abnormalities secondary to venous occlusion are reversible. Although parenchymal changes may occur in areas of the brain that are directly drained by the occluded venous sinus, in some patients the parenchymal changes may not closely correlate with the location of venous occlusion (6,9).

Parenchymal swelling without abnormalities in attenuation or signal intensity on images may occur in as many as 42% of patients with cerebral venous thrombosis (17). Sulcal effacement, diminished cistern visibility, and a reduction in ventricular size may occur. Patients with brain swelling and without parenchymal signal intensity changes tend to have intrasinus pressures in the intermediate range (20–25 mm Hg); however, intrasinus pressures also may be markedly elevated (6). Such patients typically have more prominent clinical symptoms than would be expected on the basis of imaging findings (6).

Diffusion Change.—Although diffusion-weighted imaging may provide important information in the evaluation of patients with cerebral venous thrombosis, research involving the use of diffusion-weighted imaging to assess cerebral venous thrombosis has been limited. In approximately half of all patients who have lesions with increased T2 signal intensity associated with cerebral venous thrombosis (excluding those with hemorrhage), diffusion-weighted images show regions with diminished ADC values (Fig 18).
Information from diffusion-weighted imaging in these patients is consistent with edema related to combined vasogenic and cytotoxic processes. Patients with diminished ADC values more often have parenchymal sequelae, while those with normal or increased ADC values usually do not (7,37,38). Areas without diminishment in ADC may primarily represent vasogenic edema from venous hypertension. Complete or nearly complete resolution of edema in patients with cerebral venous thrombosis and diminished ADC values also has been reported (38).

Contrast Enhancement.—Parenchymal enhancement in 1%–29% of cases of cerebral venous thrombosis has been reported (17,27,39). The enhancement is typically gyral in location and may extend into the white matter (Fig 19). Parenchymal enhancement, which indicates disruption of the blood-brain barrier, may be seen in areas of cytotoxic or vasogenic edema and in the presence of either irreversible or reversible brain abnormalities. Increased tentorial enhancement (likely related to dural venous collaterals), adjacent leptomeningeal enhancement, and prominent cortical venous enhancement (secondary to venous congestion) also may be visible after the administration of contrast material.

Perfusion Changes.—Little has been published about MR perfusion imaging in patients with cerebral venous thrombosis. In a study performed in patients with normal ADC values, the most common finding was prolongation of the mean transit time with normal relative cerebral blood volume in areas of the brain that were drained by the thrombosed sinus (40). Alterations in mean transit time were seen in areas of the brain with and without parenchymal signal intensity alterations or hemorrhage. In patients who underwent follow-up MR perfusion imaging after treatment and sinus recanalization, the perfusion abnormalities had resolved by the time of follow-up. In an additional case of deep venous occlusion, Keller et al found an increase in relative cerebral blood volume with a prolonged time to peak enhancement in the thalami; areas involved with extensive vasogenic edema; and elevated ADC measurements (41).

Perfusion changes in patients with cerebral venous thrombosis may reflect both venous congestion and diminishment of cerebral blood flow, which may be reversible before permanent brain parenchymal damage occurs (40).

MR Spectroscopic Findings.—Little information exists regarding MR spectroscopic findings in cerebral venous thrombosis. In one patient with deep venous occlusion and extensive thalamic edema, proton MR spectroscopic findings included a very mild lactate peak with preserved N-acetylaspartate levels (42). Arterial infarction generally produces a more pronounced diminishment in neuronal metabolites and a pronounced elevation of lactate levels. Very early arterial infarction may have a similar appearance, with an elevated lactate level and variably preserved N-acetylaspartate levels (43). To our knowledge, the use of MR spectroscopy to differentiate between venous infarction and arterial infarction has not been assessed.
Hemorrhage.

—Parenchymal hemorrhage can be seen in one-third of cases of cerebral venous thrombosis (6–9,13,14,32,34,36) (Fig 20). Flame-shaped irregular zones of lobar hemorrhage in the parasagittal frontal and parietal lobes are typical findings in patients with superior sagittal sinus thrombosis and should prompt additional imaging evaluations (eg, with MR venography or CT venography). Hemorrhage in the temporal or occipital lobes is more typical of transverse sinus occlusion. Hemorrhage in cerebral venous thrombosis is typically cortical with subcortical extension. Smaller zones of isolated subcortical hemorrhage also may be seen (44) and may be accompanied by minimal edema. MR imaging with GRE sequences is sensitive in the depiction of these zones of parenchymal hemorrhage.

The mechanism of hemorrhage in cerebral venous thrombosis is multifactorial. Hemorrhage may be precipitated by continued arterial perfusion in areas of cell death, as can be seen at reperfusion in arterial ischemia. Elevation of venous pressure beyond the limit of the venous wall also is likely operative. In one study, hemorrhage was noted in patients with intrasinus pressures higher than 42 mm Hg but not in those with lower pressures (6).

Deep Venous Occlusion

Thrombosis of the internal cerebral veins, vein of Galen, or straight sinus has been observed in approximately 16% of patients with cerebral venous thrombosis (3–14). Most such patients present with symptoms of elevated intracranial pressure that may rapidly escalate to a coma (45,46). The manifestations may mimic those of encephalitis. Prompt and accurate diagnosis and treatment are critical.

Thalamic edema is the imaging hallmark of this condition, and it may extend into the caudate regions and deep white matter (Fig 21). Thalamic edema may be seen in as many as 76% of CT and 86% of MR studies (45). At MR imaging, a thrombus usually is visible in the straight sinus, vein of Galen, or internal cerebral veins (45).

**Figure 20.** Axial CT images from three patients with sinus thrombosis. (a) Typical irregularly shaped right frontal lobe hemorrhage (arrows) in a patient with superior sagittal sinus thrombosis. (b) Right temporal lobe hemorrhage with extensive edema (arrow) in a patient with right transverse sinus thrombosis. (c) Small subcortical hemorrhages (arrows) in the left frontal and parietal lobes in a patient with superior sagittal sinus thrombosis.
Hemorrhage is noted in 19% of patients and typically is located in the thalami (45). Unilateral thalamic edema may occur but is rare (47). Despite its extensive nature, the edema may be primarily vasogenic (with elevated ADC values) and may resolve without sequelae (41). Although historical mortality rates among patients with this condition are estimated to be between 22% and 37%, as many as 54% of patients may have no neurologic sequelae (45).

Isolated Cortical Venous Thrombosis

Isolated cortical venous thrombosis is a relatively rare entity; fewer than 20 cases have been reported in the imaging literature (48). Many patients with isolated cortical venous thrombosis have underlying coagulation abnormalities or chronic inflammatory conditions such as inflammatory bowel disease (49).

Typical parenchymal findings are areas of focal cortical edema or hemorrhage, which may be nonspecific. The finding of an adjacent thrombosed venous structure, which may be the most specific sign of this disorder, has been mentioned in most recent descriptions of this entity (Fig 22) (48,50). On CT images, this finding has been referred to as the “cord sign”; on MR images, it has been called the “hyperintense vein sign” (48). Blooming artifacts within the thrombosed veins can be a very useful adjunct finding on GRE images. It is important to assess the signal intensity or attenuation of the adjacent cortical veins when evaluating a focal cortical area of edema or hemorrhage, particularly if these findings are in an atypical arterial vascular distribution. The
hyperintense vein sign may be more difficult to
detect in acute-stage venous thrombosis (48).
MR venograms may show an asymmetric absence
of flow signal or diminished enhancement in the
thrombosed cortical veins. Evaluation of the MR
venographic source images is typically necessary
to make this determination (48).

Idiopathic Intracranial Hypertension
Idiopathic intracranial hypertension (also known
as pseudotumor cerebri) is a syndrome of in-
creased intracranial pressure without an obvious
explanation such as a mass lesion. Symptoms and
signs include headache, nausea, vomiting, papill-
edema, cranial nerve palsy, and visual changes.
Sinus thrombosis may occur as part of the intra-
cranial hypertension syndrome and should be ex-
cluded with detailed imaging (MR imaging and
MR venography or CT venography) in all pa-
tients with such manifestations.

Bilateral stenoses of the transverse sinuses,
without definitive evidence of current or prior
thrombosis, have been described in 93% of pa-
tients with idiopathic intracranial hypertension
identified at contrast-enhanced MR venography
(51). The stenosis typically is found in the lateral
segment of the transverse sinus (Fig 23) (51). Bi-
lateral flow restrictions in the transverse sinuses
rarely occur in patients with normal cerebrospinal
fluid pressure; only 1.8% of such patients are af-
fected by bilateral stenoses (52). Results of an-
other investigation showed bilateral transverse
sinus flow gaps in 65% of patients with idiopathic
intracranial hypertension but in none of the con-
trol patients (53). An improvement of symptoms
occurred after placement of intrasinus stents in
patients with idiopathic intracranial hypertension
and sinus stenosis (54). Transverse sinus stenoses

Figure 23. Pseudotumor cerebri in a 47-year-old woman with elevated cerebrospinal fluid pressure
documented at lumbar puncture. Oblique volume-rendered images from contrast-enhanced MR venog-
raphy show a hypoplastic right transverse sinus (arrowhead in a) and prominent smooth-bordered ste-
noses in distal portions of both the right (arrows in a) and the left (arrow in b) transverse sinuses. The
morphologic structure of the stenoses is suggestive of extraluminal compression. Parenchymal images
showed no alteration in signal intensity that might be indicative of acute or subacute thrombosis.

Figure 24. Transverse sinus atresia. Oblique
MIP image from coronal TOF MR venography
shows the complete absence of the medial part of
the left transverse sinus (arrows), a finding con-
firmed at contrast-enhanced MR venography. No
abnormal signal intensity was noted on images
obtained with other MR sequences.
also may resolve spontaneously after cerebrospinal fluid diversion in patients with idiopathic intracranial hypertension (55,56). Abnormally enlarged dural sinuses in patients with intracranial hypotension may return to normal size after blood-patch treatment and return of cerebrospinal fluid pressure to normal levels (56). Whether sinus stenoses are a cause or a result of increased intracranial pressure is a topic of ongoing study. There appears to be a dynamic relationship between cerebrospinal fluid pressure and venous sinus caliber (56).

**Potential Pitfalls in Image Interpretation**

Variants of normal venous anatomy that may mimic sinus thrombosis have been well described (57,58). These can be subdivided into venous anatomic variants that mimic occlusion (sinus atresia or hypoplasia), asymmetric or variant sinus drainage (occipital sinuses, sinus duplication), and normal sinus filling defects (arachnoid granulations, intrasinus septa).

**Sinus Hypoplasia and Atresia**

Hypoplasia and atresia of the transverse sinuses occur frequently. In one anatomic study performed with conventional angiography, asymmetric transverse sinuses were seen in 49% of cases, with a partial or complete absence of one transverse sinus in 20% of cases (59). In most cases, the right transverse sinus was larger than the left (58,59). In a study performed with TOF MR venography, the left transverse sinus was atretic in 20% of cases and relatively hypoplastic in 39%. The right transverse sinus was atretic in 4% of cases and relatively hypoplastic in 6% (60). The medial part of the transverse sinus is more commonly atretic or hypoplastic (Fig 24).

**Flow Gaps at TOF MR Venography**

Flow gaps commonly appear on TOF MR venographic images and may cause confusion in diagnosis; their appearance on as many as 31% of coronally acquired TOF MR venographic images has been reported (61). Flow gaps most commonly appear in the nondominant transverse sinus and are correlated with a normal but small sinus as depicted at conventional angiography (61). The combination of a small sinus size, a slow or complex flow pattern, and an image acquisition plane that is not perpendicular to the sinus likely results in this finding (61). A close assessment of the source images is mandatory to accurately evaluate venous structures and reduce the potential for diagnostic error. The lack of a thrombus signal within the sinus on MR images is a helpful clue for avoiding this pitfall. Flow gaps are a much less common problem with the use of contrast-enhanced MR venographic or CT venographic techniques (Fig 25) (19).
Variant Anatomy of the Sinus Confluence

Variant anatomy of the torcular herophili is common and may lead to diagnostic error, particularly in the interpretation of CT images (59,62). A high or asymmetric bifurcation may resemble an intrasinus thrombus (Fig 26). In one study, a high bifurcation or asymmetric deviation of the sinuses at the confluence produced a “pseudo empty delta sign” in 18% of patients examined with contrast-enhanced CT (62). The careful assessment of sequential images and the documentation of venous continuity are necessary to avoid this pitfall.

Arachnoid Granulations

Arachnoid granulations are normal structures that protrude into the dural sinus lumen or lateral lacunae. When they are prominent, they may simulate sinus thrombosis. Their occurrence throughout the dural sinuses has been described, but they are most commonly seen in the transverse and superior sagittal sinuses on anatomic images (63,64). With typically used clinical imaging protocols, arachnoid granulations are usually identified in the transverse sinus, specifically in the lateral part of the transverse sinus, near the entrance sites of the Labbé vein and the lateral tentorial sinus (Fig 27). As the contrast resolution of cross-sectional imaging techniques has improved, filling defects consistent with arachnoid granulations have been seen with increasing frequency. In a study of three-dimensional contrast-enhanced GRE MR imaging with a 1.25-mm section thickness, filling defects consistent with arachnoid granulations were depicted in 90% of patients in the superior sagittal sinus, transverse sinus, and straight sinus (65).

It is important to differentiate between arachnoid granulations and filling defects secondary to sinus thrombosis. Arachnoid granulations typically have signal intensity and attenuation similar to those of cerebrospinal fluid and appear as focal rounded filling defects with a characteristic anatomic distribution. Although arachnoid granulations are a normal anatomic structure, if they are large and appear in the dominant sinus or only in the transverse sinus, they could cause venous obstruction and lead to symptoms of venous hypertension (66).

Thrombus Signal Shine-Through at TOF MR Venography

An intrasinus thrombus in the subacute stage may have markedly increased signal intensity on MR images that may be misinterpreted as evidence of flow on TOF MR venograms (Fig 28) (30,67). A close evaluation of MR venographic source images usually allows differentiation, as the thrombus signal is typically not as intense as the flow-related signal. T1-weighted MR images in such cases depict an abnormal increase in signal intensity within the sinus.

Sinus Signal Intensity Variations

In comparison with the homogeneous flow void that is usually seen in arterial structures on standard MR images, the signal intensities of venous structures may range widely. Slow flow states, complex flow patterns, normal anatomic varia-
tions, and normal physiologic variations in dural sinus flow can create confusing imaging appearances (10,67,68).

Flow-related enhancement secondary to unsaturated protons entering the imaging section may produce an increased relative signal intensity on MR images, particularly on the first image in a multisection sequence (67,68). The lack of persistence of this artifactual finding in sections with other orientations helps differentiate it from thrombosis.

Slowly flowing or stagnant blood may appear with increased signal intensity on MR images (67,69,70). This may occur even in normal patients (69). The signal intensity of stagnant blood is typically isointense to that of brain parenchyma on T1-weighted images and hyperintense on T2-weighted images. Asymmetric signal intensity and enhancement normally can be seen in the transverse sinus, sigmoid sinus, and jugular bulb. This asymmetric signal intensity is more common on the left and is likely secondary to physiologic effects of left brachiocephalic vein compression during the respiratory cycle (71).

**Summary**

Cerebral venous thrombosis is a relatively uncommon but serious neurologic disorder. Imaging plays a primary role in diagnosis. Prompt and appropriate medical therapy is important because brain parenchymal alterations and venous thrombus formation are potentially reversible. MR imaging, TOF MR venography, contrast-enhanced MR venography, and CT venography are the most useful techniques for diagnosis of this condition. Knowledge of normal venous variations and potential pitfalls related to image interpretation are important for achieving an accurate diagnosis.

**References**


Intracranial hypertension occurs in 20%–40% of patients with cerebral venous thrombosis and should be excluded in patients with the specific complex of symptoms.

On images acquired after gadolinium administration, marked contrast enhancement may be observed that resembles the enhancement typically seen in a normal sinus.

In contrast with arterial ischemic states, many parenchymal abnormalities secondary to venous occlusion are reversible.

Thalamic edema is the imaging hallmark of this condition [deep venous occlusion], and it may extend into the caudate regions and deep white matter.

Arachnoid granulations are normal structures that protrude into the dural sinus lumen or lateral lacunae. When they are prominent, they may simulate sinus thrombosis.