MR of Deep Cerebral Venous Thrombosis

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ABSTRACT: Two cases of deep cerebral venous thrombosis are presented with specific reference to the CT and MR findings. The MR findings are discussed, with comparison to the findings of superficial cerebral sino-venous occlusion.

RÉSUMÉ: Résonance magnétique nucléaire dans la trombose veineuse cérébrale profonde Nous présentons les observations faites par CT scan et NMR chez deux cas de trombose veineuse cérébrale profonde. Nous discutons des observations faites par NMR et nous comparons ces observations à celles faites dans les cas d'occlusion de sinus veineux cérébraux superficiels.

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Cerebral venous thrombosis is an uncommon disorder, most frequently reported to involve the superficial sino-venous system of the brain. The radiographic features of deep cerebral venous thrombosis are different from those of superficial sino-venous thrombosis, and have been previously described. Although MR has recently been reported to be a useful tool in evaluating cerebral sino-venous thrombosis, the MR findings in deep cerebral venous thrombosis have not been as well documented. Two patients with this condition underwent CT, MR and angiography. The radiographic findings are described and are compared with the findings reported in the literature.

Materials and Methods

Axial CT scans were obtained with and without contrast enhancement on either a GE-9800 Quick CT Scanner or a Picker 1200SX CT Scanner. Digital intra-arterial angiography was performed in both cases on a Siemens Angioscope Digitron 3VA unit. MR studies were obtained on a 1.5 T Philips Gyroscan MR unit. In both cases, coronal and parasagittal T1-weighted (TR =550ms, TE = 30ms) as well as axial T2 (TR = 2100ms, TE = 60ms) and proton-density (TR = 2100ms, TE = 30ms), spin-echo images were obtained.

Case Reports

Patient 1

A 19-year-old previously well woman on the birth control pill presented with a 2 to 3 day history of severe headaches, nausea and vomiting, having been found staring and uncommunicative. Examination demonstrated right-sided hemiparesis and mild right-sided hyperreflexia. Lumbar puncture revealed elevated intracranial pressure and slightly increased CSF protein content.

CT on admission revealed high-density thrombus in the deep cerebral veins and marked hypodensity of deep cerebral structures (Figure 1). Angiography performed within two hours of the CT confirmed occlusion of deep cerebral veins, left transverse sinus and superior sagittal sinus (not shown).

MR was obtained three days following admission, and demonstrated changes of thrombosis within the deep cerebral veins and sinuses (Figure 2A and B). The deep venous changes were somewhat less extensive than would have been expected on the preceding CT study, a finding which may be due to either rapid recanalization of veins in the case of vessels showing flow-void, or to isointense thrombus in the vessel lumen. The findings of sinus thrombosis were better appreciated with MR than with CT. Associated changes of brain injury including hemorrhage were well assessed on long TR images (Figure 3).

The patient was treated with steroids and improved dramatically, and was discharged from hospital on the fifth day. Laboratory studies revealed a relative protein-C deficiency. Follow-up MR study one month later showed hemosiderin deposition from previous hemorrhage and resumption of flow-void throughout the deep veins and sinuses (not shown).

Patient 2

A 21-year-old woman on the birth control pill presented three weeks prior to admission with headaches, nausea and vomiting, attributed to a viral illness. Contrast enhanced CT at this time was normal. Two days prior to admission, the patient developed increasing headaches, nausea and vomiting and decreasing level of consciousness, and was mute with no evidence of paresis or reflex changes on admission. Lumbar puncture revealed mildly elevated CSF pressure and increased protein.

CT on admission demonstrated high-density thrombus in the deep cerebral veins and straight sinus with hypodensity of deep cerebral structures (Figure 4A). CT performed four days after, following treatment with steroids and Mannitol, demonstrated hemorrhagic areas in the deep cerebral structures with increased surrounding hypodensity and moderate enlargement of the ventricles (Figure 4B). Angiography the next day confirmed occlusion of the deep cerebral veins (not shown).

MR ten days after admission demonstrated thrombosis of the deep cerebral veins and straight sinus (Figure 5A), subacute hemorrhage in

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the thalami and basal ganglia and associated surrounding edema (Figure 5B and Figure 6). Ventricular dilatation with possible transependymal CSF resorption was also noted (Figure 6).

The patient eventually improved clinically and was discharged approximately one month following admission with slight memory difficulty, remaining somewhat slow to respond but otherwise normal.

Discussion

The CT findings of cerebral sino-venous thrombosis can be divided into direct and indirect signs. The direct signs relate to visualization of hyperdense thrombus within the lumina of involved vessels, and include the "cord sign" or "dense vein sign", seen in both cortical veins and venous sinuses.[2-5] The "empty delta" or "empty triangle" signs are seen following contrast enhancement in sinus thrombosis.[2-4] Indirect signs are due to either distended cortical or dural venous collaterals, or to associated brain injury. Venous collaterals may be manifested by tentorial enhancement[2-5] and dilated intramedullary veins.[2] Brain injury is most commonly manifested as diffuse cerebral edema with small lateral ventricles and pronounced gray/white matter differentiation.[3,5,7] Non-hemorrhagic venous infarct (hypodensity, not in an arterial or watershed distribution) and hemorrhagic venous infarcts (subcortical, irregular and often multifocal hematomas) also occur, as does gyral enhancement.[2-6]

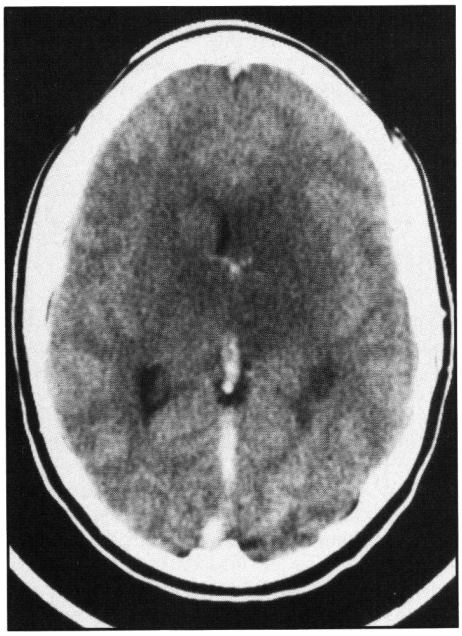
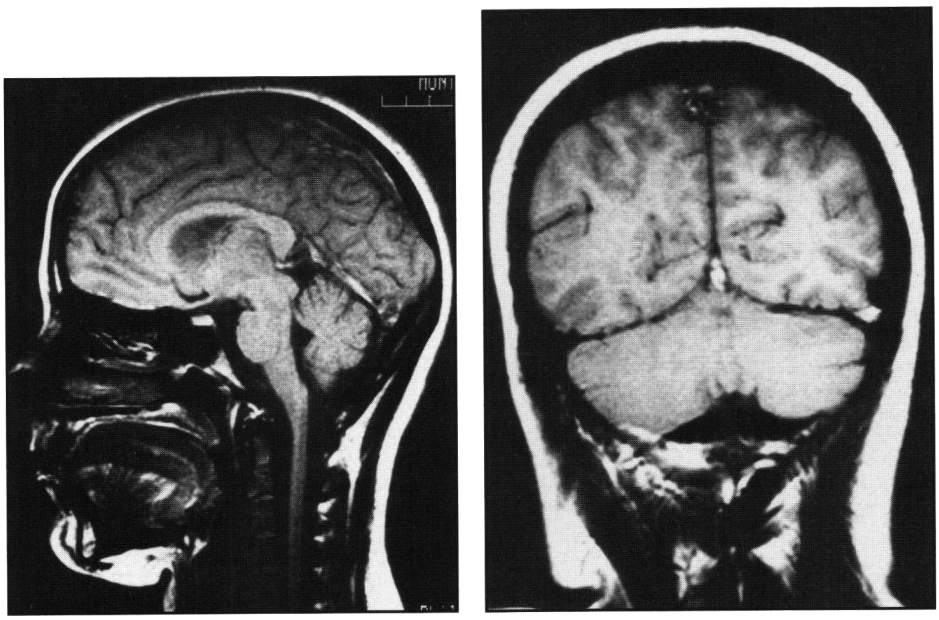


Figure 1 — Patient 1 — Non-enhanced CT on admission. Hypodensity in the basal ganglia, thalami, left lentiform and caudate nuclei and left internal capsule with slight compression of left lateral ventricle. Hyperdensity in the thalamostriate and internal cerebral veins and straight sinus.

In deep cerebral venous thrombosis, CT findings can again be divided into direct and indirect signs. Hyperdense thrombus visualized in the vein of Galen, straight sinus, internal cerebral veins or thalamostriate veins are direct signs.[8,9] Indirect signs include uni- or bilateral low-density venous infarction in the thalamic or basal ganglia region; ring or homogeneous contrast enhancement in the venous infarction; hemorrhagic infarction in basal ganglia; ipsi- or bilateral ventricular dilatation; and hemorrhage in the cistern of the velum interpositum.[8-10]

MR findings in sino-venous thrombosis include the direct findings of intraluminal venous thrombus. This is most often reported as increased signal intensity in superior sagittal sinus, transverse and sigmoid sinuses on T1-weighted spin-echo images, representing subacute thrombosis.[11-12] Macchi et al divide venous thrombosis into initial, intermediate and late stages based on signal intensity on T1 and T2-weighted spin-echo images.[13] Initial venous thrombosis is manifested as absence of flow-void, with vessels appearing isointense on T1 and hypointense on T2. Intermediate stage thrombosis, several days following ictus, demonstrates absence of flow, with the vessels appearing hyperintense initially on T1 then on T2-weighted images, with hyperintensity progressing to fill the vessel lumen. This hyperintensity persists in multiple imaging planes in both T1 and T2-weighted images. In late stages,



A B

Figure 2 (A and B) — Patient 1 — T1-weighted SE MR (TR = 550ms, TE = 30ms) three days after admission. Hyperintensity in straight sinus, posterior sagittal sinus, left transverse and sigmoid sinuses and posterior left internal cerebral vein. Normal flow-void seen in right internal cerebral vein and vein of Galen.

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greater than two weeks duration, vascular recanalization occurs with resumption of flow-void in previously thrombosed vessels.

Indirect MR findings include changes of hemorrhagic infarction,[11,13] as well as dilated venous collaterals, including transcortical medullary veins.[13,14]

Our two cases of deep cerebral venous thrombosis demonstrate many of the typical CT findings of this entity. The MR studies in our patients demonstrate direct findings of venous thrombosis, with the most striking finding being hyperintensity involving internal cerebral veins, vein of Galen, straight sinus and other venous sinuses on both T1 and T2-weighted spin-echo images. In one patient, however, while hyperintensity was present in the superficial venous sinuses and posterior left internal cerebral vein and absence of flow-void was noted in the anterior left internal cerebral vein, flow-void persisted in the right internal cerebral vein and vein of Galen. This may represent either absence of thrombosis in these veins initially, perhaps unlikely in view of the hyperdensity on the initial CT study, or rapid recanalization of thrombosed vessel with resumption of flow-void. Variability of vein recanalization raises a possible source of false negative results for detection of intraluminal thrombus when imaging several days following the acute ictus. This may be difficult to avoid, as these patients are frequently ill and restless on presentation and so unable to undergo MR immediately. A second potential source of false negative results lies in the marked hypointensity seen in acute thrombus on T2-weighted images due to the presence of deoxyhemoglobin, which can mimic flow-void. These areas should appear isointense on T1-weighted images. False positive diagnoses of venous thrombosis can be caused by paradoxical enhancement of slow flowing venous blood in otherwise normal patent veins. This enhancement is predominantly an entry-section phenomenon which is most pronounced on T1-weighted images. It may be minimized by long TR imaging sequences and scans in different planes.

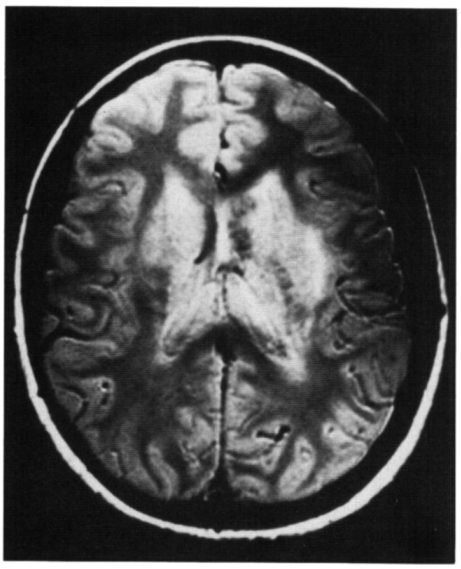
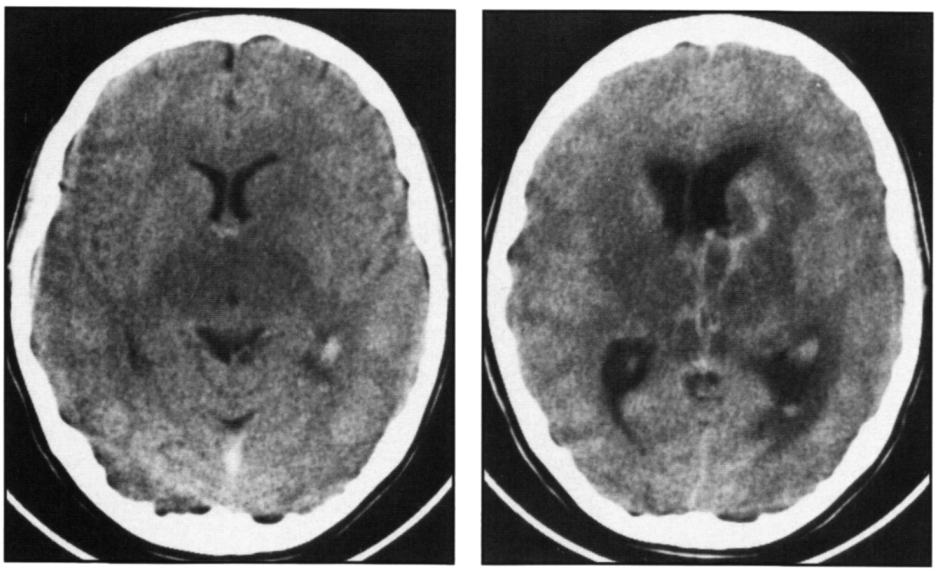


Figure 3 — Patient 1 — T2-weighted SE MR (TR = 2100ms, TE = 60ms) three days after admission. Hypointensity in head of left caudate and genu of left internal capsule indicative of acute hemorrhage.[1] Hyperintensity in basal ganglia and thalami, internal and external capsules.

Other MR imaging techniques may be of value in assessing cerebral vasculature. Gradient-echo imaging sequences demonstrate flowing blood as a high-signal intensity and may be used to demonstrate vascular patency, but intraluminal thrombus in clotted vessels may also appear as a high-signal intensity, mimicking normal blood flow.[15] Careful correlation of spin-echo and gradient-echo images is needed.

Various techniques of Magnetic Resonance angiography have the capability to demonstrate blood flow in patent vessels.[16,17] The signal contributed by stationary tissues, including that of thrombosed vessels, would be removed from the images such that clotted veins would not be visualized. The use of these techniques may obviate the need for conventional angiography.

In both our cases, the indirect findings of venous thrombosis were very striking. A marked amount of basal ganglionic, thalamic, internal and external capsular edema was demonstrated, as evidenced by hyperintensity on T2 and less striking hypointensi-



A B

Figure 4 — Patient 2 — (A) Non-enhanced CT on admission and (B) four days after admission. Hypodensity in both thalami, internal capsules and head of left caudate nucleus with hyperdensity in thalamostriate and internal cerebral veins, vein of Galen and straight sinus. Later CT demonstrates hemorrhage in the head of left caudate, internal capsule and both thalami. Increased surrounding hypodensity, now with moderate lateral ventricular dilatation.

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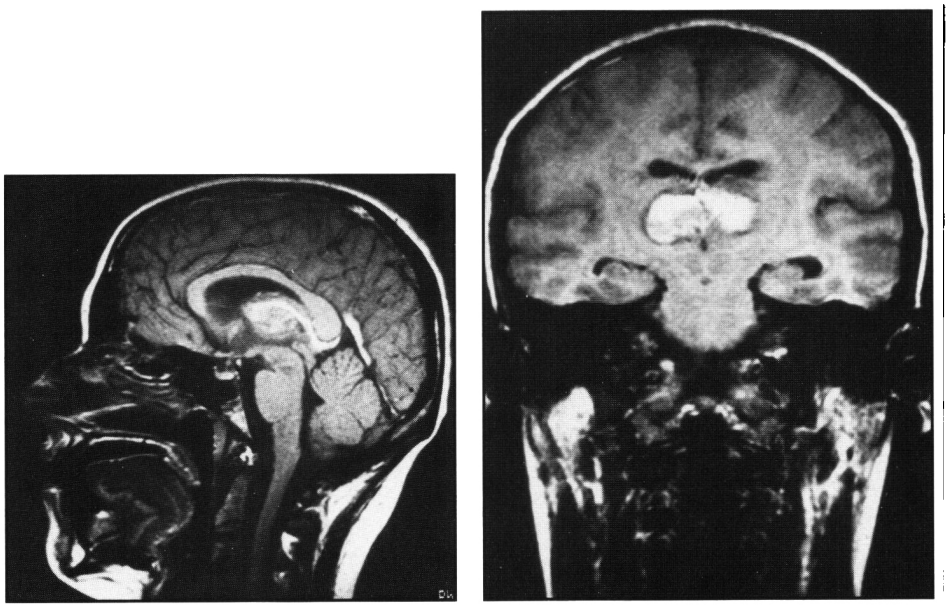

A B

Figure 5 (A and B) — Patient 2 — T1-weighted SE MR (TR = 550ms, TE = 30ms) ten days after admission. Hyperintensity in the internal cerebral veins, vein of Galen, straight sinus and mid-posterior sagittal sinus. Globular hyperintensity with rim hypointensity, characteristic of subacute hemorrhage[1] in medial thalami, as well as in left caudate nucleus, internal capsule and globus pallidus (not shown).

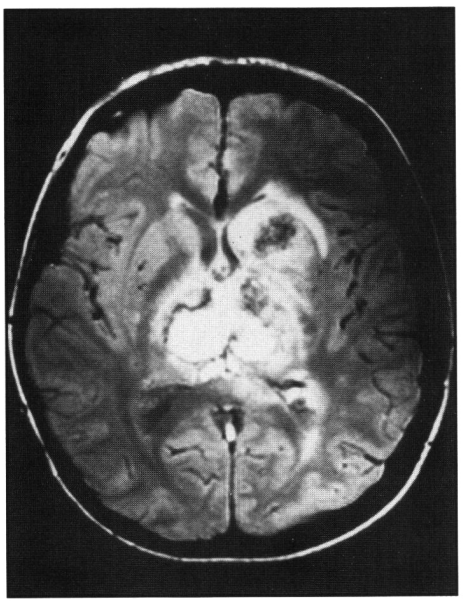

Figure 6 — Patient 2 — Proton-density SE MR (TR = 2100ms, TE = 30ms) ten days after admission. Hyperintensity with rim hypointensity in the medial thalami and internal capsules indicating subacute hemorrhage. Hypointensity of left putamen, anterolateral left thalamus, left internal capsule and body of left caudate nucleus, which appeared hypo- to isointense on T1-weighted images (not shown), indicating more acute phase hemorrhage. Hyperintensity throughout remainder of deep cerebral structures. Mild bilateral lateral ventricular dilatation with hyperintensity adjacent to the anterior horns which, on the right, is not contiguous with white matter edema elsewhere, a pattern suggesting transependymal CSF resorption.

ty on T1-weighted images. In both cases, frank basal ganglionic, internal capsular and thalamic hemorrhage was demonstrated. The hemorrhage was subacute in one patient and more acute in the other. Unfortunately, CT was not obtained on the same day as the MR studies and direct comparison could not be made, but the sensitivity of MR to subacute hemorrhage may make detection of hemorrhage a more frequent occurrence in venous thrombosis than was previously suspected from CT. This may have some impact on the treatment of these patients, in whom anticoagulation is sometimes considered.

Changes in the configuration in the ventricular system were seen in both of our patients, with slight compression of the left lateral ventricle in one of our patients, likely due to the surrounding edema. Bilateral lateral ventricular dilatation was seen in the second case, with hyperintensity adjacent to anterior horns of the lateral ventricles. Although this appearance is nonspecific and may be due to edema from venous congestion, the non-contiguity with basal ganglia and white matter edema elsewhere suggests interstitial edema from transependymal resorption of CSF, frequently seen in obstructive hydrocephalus. The changes suggest outlet obstruction to the lateral ventricles at the foramina of Monro or third ventricle, due to compression from basal ganglionic edema and/or hemorrhage.

In summary, these cases demonstrate MR to be exquisitely sensitive to the brain edema and/or hemorrhage frequently found in venous infarction associated with cerebral venous thrombosis. Ventricular changes from either mass effect or compression of the foramina of Monro are also well evaluated. The direct finding of intraluminal thrombus is corroborated, but must be evaluated carefully in patients suspected of cerebral venous thrombosis in view of the potential pitfalls discussed. Gradient-echo imaging sequences and Magnetic Resonance Angiography should enhance the specificity of MR imaging in this condition.

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