MR Staging of Acute Dural Sinus Thrombosis: Correlation with Venous Pressure Measurements and Implications for Treatment and Prognosis

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PURPOSE: To correlate parenchymal brain changes, venous sinus pressure measurements, and outcome in 29 patients with acute dural sinus thrombosis. **METHODS:** A retrospective review of 29 patients with angiographically proved acute dural sinus thrombosis was made from January 1989 to December 1993. MR examinations were performed on either a 0.5- or 1.5-T superconductive scanner in multiple planes. Direct dural sinus venography, cerebral angiography, and MR venography were performed. Venous sinus pressure measurements were obtained in 11 of 29 patients. **RESULTS:** We identified five distinct stages of brain parenchymal changes; each stage correlated with increasing intradural sinus pressure. The pressures measured in this study ranged from 20 to 50 mm Hg. Brain parenchymal changes were reversible up to stage III if thrombolytic treatment was performed. Beyond stage III, there were some residual changes, even after thrombolysis. All stage V patients died. **CONCLUSION:** Acute dural sinus thrombosis leads to distinct stages of parenchymal changes, the severity of which depends on the degree of venous congestion, which, in turn, is closely related to intradural sinus pressure. As intradural sinus pressure increases, progression from mild parenchymal change to severe cerebral edema and/or hematoma may occur if thrombolysis is delayed.

Index Terms: Thrombosis, dural sinus; Brain, magnetic resonance; Brain, pressure

The clinical diagnosis of acute dural sinus thromboocclusion can be difficult and is frequently delayed because the clinical manifestations and radiologic findings may not be specific. Acute dural sinus occlusion may lead to severe venous congestion and massive venous infarction, with potentially fatal consequences if the occlusion is not correctly diagnosed and promptly treated.

The purpose of this paper is to report our experience with 29 patients who have had various magnetic resonance (MR) and computed tomographic (CT) patterns of parenchymal changes, as a result of acute dural sinus and/or cerebral venous thrombosis, and to correlate the clinical and radiologic patterns with the intradural sinus pressure.

Materials and Methods

A retrospective review of 29 patients with angiographically proved acute dural sinus and cerebral venous thrombosis from January 1989 to December 1993 was made. Sequential CT and/or MR examinations of the brain were correlated with clinical symptoms and venous sinus pressure.

MR examinations were performed on either a middle-field 0.5-T or high-field 1.5-T superconductive scanner. All patients had a noncontrast MR examination including relatively T1- and T2-weighted spin-echo images. Thirteen patients also underwent contrast-enhanced studies with intravenous injection of gadopentetate dimeglumine, and 9 patients had follow-up examinations. Seven patients had two-dimensional time-of-flight MR venography.

TABLE 1: Stages of severity of dural sinus thrombosis

Stage	Parenchymal Changes	Symptoms	Pressure Range
I	No parenchymal change	Severe headache, papilledema, weakness, changed mentation, drowsiness, right hemiparesis (one patient only)	One patient placed in this category had pressure measurements taken: left transverse sinus = 14 mm Hg; superior sagittal sinus = 17 mm Hg
II	Brain swelling, sulcal effacement and mass effect, no signal change	Increased headache, double vision, seizure, decreased mentation, extreme drowsiness, difficulty rousing, right lower extremity weakness (one patient)	Four patients had measurements taken: 20–25 mm Hg
III	Increased intensity of signal change as mild to moderate edema	Inability to rouse, obtundation, hemiparesis, seizure	Three patients had measurements taken: 32–38 mm Hg
IV	Severe edema, with or without hemorrhage	Hemiparesis, seizure, loss of consciousness, coma	Three patients had measurements taken: 42–51 mm Hg
V	Massive edema and/or hemorrhage	Coma, response to deep pain only	No measurements were taken

On noncontrast CT or T1-weighted MR, special attention was given to the presence or absence of mass effect (cortical sulci effacement and/or ventricular compression), hemorrhage, and signs of dural sinus thrombosis, including the absence of flow void phenomena. T2-weighted images were evaluated for parenchymal signal changes and contrast-enhanced studies for any abnormal vascular and/or parenchymal enhancement. The follow-up studies in 19 patients were evaluated for evidence of residual changes, including brain swelling, atrophy, and hemosiderin.

All patients had cerebral angiography and/or dural sinus venography. Dural sinus venography was performed via the percutaneous transfemoral route using a 5F angiographic catheter as a guide, advanced through the inferior vena cava, right atrium, and superior vena cava to the jugular bulb. A multiside hole Tracker 18 catheter (Target Therapeutics, Fremont, Calif) was advanced into the dural sinus coaxially through the 5F guiding catheter. Cases of unilateral transverse sinus thrombosis were approached from the contralateral transverse sinus through the torcular herophili. This approach was technically easier than retrograde through the ipsilateral sinus and allowed measurement of the intrasinus pressure proximal to the thrombosis. Bilateral transverse sinus thrombosis was approached either from the right side or the side of incomplete occlusion. Superior sagittal sinus thrombosis was approached via the transverse sinus and the torcular then superior sagittal sinus.

Eleven of 29 patients had dural sinus pressures measured directly using a Tracker 18 end-hole catheter proximal to the thrombus and connected to a pressure transducer at ear level with the waveform displayed on either an Alpha 9 pressure monitor (Spacelabs, Chatsworth, Calif) or a Hewlett Packard M1092A monitor (Hewlett Packard, Waltham, Mass).

Thrombolysis was performed with urokinase, 80 000 IU in 5 mL of sterile water, after an initial bolus of 250 000 IU. Intradural sinus pressure measurements were taken before, during, and after completion of thrombolysis (1).

One of five stages of severity was assigned to each patient by one of three neuroradiologists (F.Y.T., A.M.W., and W.T.C.Y.) after a retrospective evaluation of the patient's imaging studies, dural sinus pressures, and clinical symptoms (Table 1). The imaging criteria were as follows: in stage I there are no parenchymal changes; in stage II there is brain swelling, sulcal effacement, and mass effect without signal changes to indicate edema; in stage III, attenuation and signal intensity changes compatible with mild to moderate edema; in stage IV, severe edema with or without small hemorrhage; and in stage V, massive edema or hemorrhage.

Results

Among our 29 patients, 17 were female and 12 male with ages ranging from 6 days to 77 years (average, 33.3 years) (Table 2). Predisposing factors for acute dural sinus thrombosis were as follows: 2 patients had hypercoagulopathy; 4 were postpartum; 3 were taking oral contraceptives; 4 had histories of intravenous drug use; 2 were dehydrated infants; 1 had posterior fossa surgery 3 days earlier; and the remaining 13 patients had no identifiable cause.

Eleven of the 29 patients received only anticoagulation and/or other supportive treatment. In this group, 8 patients were female and 3 male with ages ranging from 9 months to 77 years (average, 31 years). Three of the 11, classified as stage I, had good recoveries with anticoagulation only. Follow-up MR showed incomplete resolution of thrombosis in 1, but the other 2 had insignificant thromboses after 2 weeks. Six of these 11 patients died from either massive hemorrhage or edema and were classified as

TABLE 2: Twenty-nine cases of acute dural sinus thrombosis

Patient	Age/Sex	Cause	Signs and Symptoms	Duration	Confirmation Method	Parenchymal Stage	Venous Pressure	Site of Thrombosis	Treatment	Outcome
1	37 y/F	Oral contraceptives	Headache and papilledema	8 d	MR venography	I	...	L transverse sinus	Anticoagulant	Neurologically normal
2	77 y/F	Undetermined	Lethargy, R hemiparesis	1 wk	MR venography	I	...	Superior sagittal sinus	Anticoagulant	Neurologically normal
3	42 y/M	Undetermined	Increasingly severe headache	4 d	MR venography	I	...	Superior sagittal sinus, R transverse sinus	Anticoagulant	Neurologically normal
4	48 y/F	Undetermined	Weakness, decreasing mentation	1 d	Venography	I	...	L transverse sinus	Urokinase	Neurologically normal
5	35 y/F	Oral contraceptives	Increasingly severe headache, papilledema, decreased mentation	3 d	MR and direct venography	I	17 mm Hg / 14 mm Hg	Superior sagittal sinus, L transverse sinus	Urokinase	Neurologically normal
6	31 y/F	Postpartum VonWillebrand disease	Decreased mentation, severe headache	2 d	Venography	II	20 mm Hg	R transverse and sigmoid sinus	Urokinase	Neurologically normal
7	23 y/F	Drug use	Headache and neck pain, drowsiness	1 wk	Venography	II	23 mm Hg	Superior sagittal sinus	Urokinase	Neurologically normal
8	54 y/F	Undetermined	Increasing headaches, increasing frequency of seizures, drowsiness	1 wk	Angiography and venography	II	24 mm Hg	L transverse sinus	Urokinase	Neurologically normal
9	38 y/M	Drug use	Papilledema, R lower extremety weakness, seizure, drowsiness	4 d	Venography	II	25 mm Hg	Superior sagittal sinus, R transverse sinus	Urokinase	Neurologically normal
10	46 y/M	Drug use	Double vision, headache, decreased mentation	3 d	Angiography and venography	II	...	Superior sagittal sinus, R transverse sinus	Urokinase	Neurologically normal
11	52 y/M	Alcoholism	R hemiparesis, semi-coma after head trauma	2 d	Venography	II	...	Superior sagittal sinus	Urokinase	Neurologically normal
12	10 mo/M	Dehydration	Decreasing mentation and sucking power	6 d	Venography	II	...	Superior sagittal sinus	Urokinase	Neurologically normal
13	23 y/F	Postpartum	Inability to rouse	5 d	Venography	III	32 mm Hg	Superior sagittal sinus	Urokinase	Neurologically normal
14	58 y/M	Undetermined	Increasing L side weakness and obtundation	7 d	Venography	III	38 mm Hg	L transverse sinus	Urokinase	Neurologically normal
15	46 y/M	Postoperative R cerebellopontine angle tumor	Lethargy and obtundation	3 d	MR and direct venography	III	35 mm Hg	R transverse sinus	Urokinase	Neurologically normal

Table continues

TABLE 2: Continued

| Patient	Age/Sex	Cause	Signs and Symptoms	Duration	Confirmation Method	Parenchymal Stage	Venous Pressure	Site of Thrombosis	Treatment	Outcome
16	37 y/F	Postpartum state	Photophobia, headache, decreased mentation, obtundation	3 d	MR and direct venography	III	…	Superior sagittal sinus and straight sinus	Urokinase	Neurologically normal
17	6 d/M	Dehydration	Semicoma	5 d	Venography	III	…	L transverse sinus	Urokinase	Neurologically normal
18	43 y/F	Hypercoagulability	Lethargy and obtundation	4 d	Venography	III	…	L transverse sinus	Urokinase	Neurologically normal
19	25 y/F	Oral contraceptives	Coma	3 d	Angiography and venography	IV	42 mm Hg	Superior sagittal sinus and R transverse sinus	Urokinase	Neurologically normal
20	32 y/M	Alcoholism	Speech deficit, ataxia, diplegia, then coma for 5 d	1 wk	Venography	IV	48 mm Hg	L transverse sinus	Urokinase	Neurologically normal
21	27 y/M	Hypercoagulability	Seizure, loss of consciousness, R hemiplegia, sudden coma	2 d	Venography	IV	51 mm Hg	Superior sagittal sinus	Urokinase	Neurologically normal
22	22 y/F	Postpartum state	Coma	4 d	MR venography	IV	…	Superior sagittal and straight sinus	R frontal surgery for removal of large hematoma	Neurologically normal
23	40 y/F	Undetermined	Dysarthria, then coma	10 d	Angiography, MR, and direct venography	IV	…	Superior sagittal sinus (misdiagnosed as basilar artery thrombosis with normal arteriogram, but venous phase showed thrombosis of the superior sagittal sinus)	Anticoagulant only	Bilaterally blind
24	52 y/M	Undetermined	Blindness, progressive decreasing mentation, sudden coma	5 d	Angiography	V	…	Superior sagittal sinus	None	Died 3.5 wk later
25	60 y/F	Undetermined	L hemiplegia, decreasing mentation, sudden coma	2 d	Angiography	V	…	Superior sagittal sinus and L transverse sinus	None	Died 1.5 mo later
26	29 y/M	Undetermined	Severe headache, decreasing mentation, sudden coma	3 d	MR and angiography	V	…	Superior sagittal sinus	None	Died within 3 wk
27	21 y/F	Undetermined	Coma, response to deep pain only	2 d	Angiography	V	…	Straight sinus and deep vein	None	Died 2 wk later
28	18 y/M	Trauma	Coma	1 d	Angiography and MR	V	…	Superior sagittal sinus	None	Persistently vegetative, died 1 mo later
29	9 mo/F	Trauma	Lethargy, coma	2 d	MR and MR venography	V	…	Superior sagittal sinus	None	Died 3 d into coma
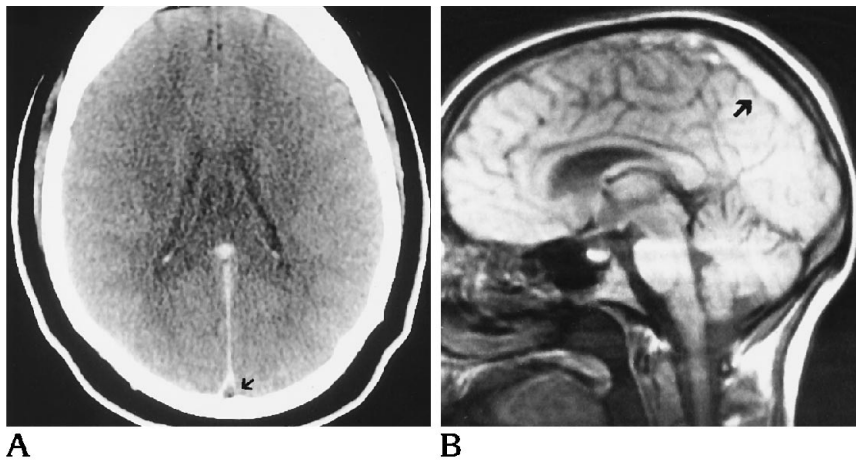
Fig 1. Patient 1, stage I.

A, Contrast-enhanced CT shows a filling defect in the superior sagittal sinus (*arrow*). The brain parenchyma is normal.

B, Spin-echo T1-weighted MR shows increased signal intensity in the superior sagittal sinus (*arrow*).

stage V at the time each diagnosis was made. Two of the patients classified as stage IV were included in the 11 patients. One received anticoagulation therapy only, and another had a right frontal hematoma removed surgically without anticoagulants. The diagnosis of superior sagittal sinus thrombosis was not made until late in each of these last 2 patients.

Eighteen of the 29 patients were treated with urokinase. This group included 9 female and 9 male patients with ages ranging from 6 days to 58 years (average, 32 years). Two patients were stage I; 7 were stage II; 6 were stage III; and 3 were stage IV. These 18 patients (except for 3 stage IV patients) had complete resolution of dural sinus occlusion and had excellent recoveries. The 3 stage IV patients had mild residual deficits with memory problems or mild ataxia.

The approximate duration of urokinase infusion varied between 2 to 4 hours. The urokinase dose varied from a minimum of 500 000 IU to a maximum of 2 million IU.

Imaging Findings

Three of the 29 patients had normal brain parenchyma on imaging studies (Fig 1). Twenty-six patients had mass effect and cortical sulcal effacement in addition to other findings. Nine of these 26 patients had only mass effect with sulcal effacement and/or ventricular compression (Fig 2). Fourteen patients had hyperintense parenchymal signal changes on T2-weighted MR images (Fig 3). Nine of these 14 patients had parenchymal hemorrhage with a
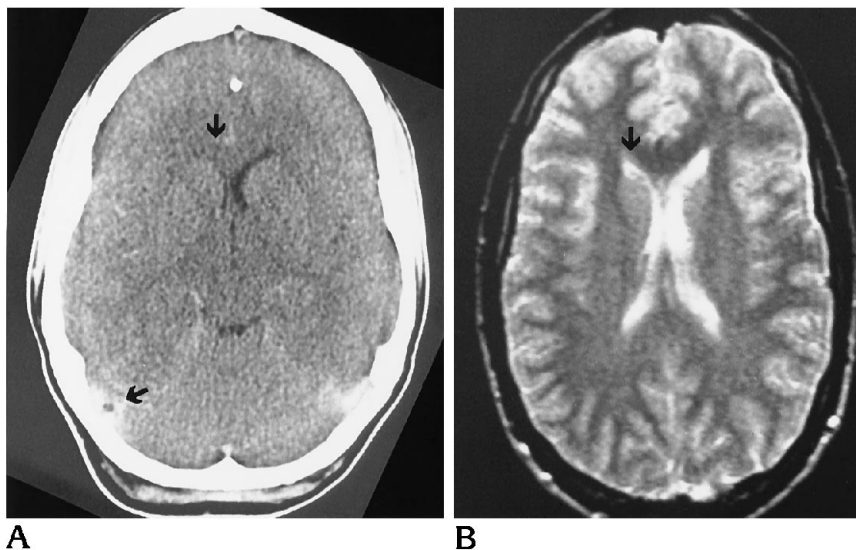

Fig 2. Patient 6, stage II.

A, Contrast-enhanced CT shows a filling defect in the right transverse sinus (*arrow*). The right lateral ventricle is smaller than the left.

B, Spin-echo T2-weighted MR shows asymmetry of the right lateral ventricle with no abnormal parenchymal signal intensity.
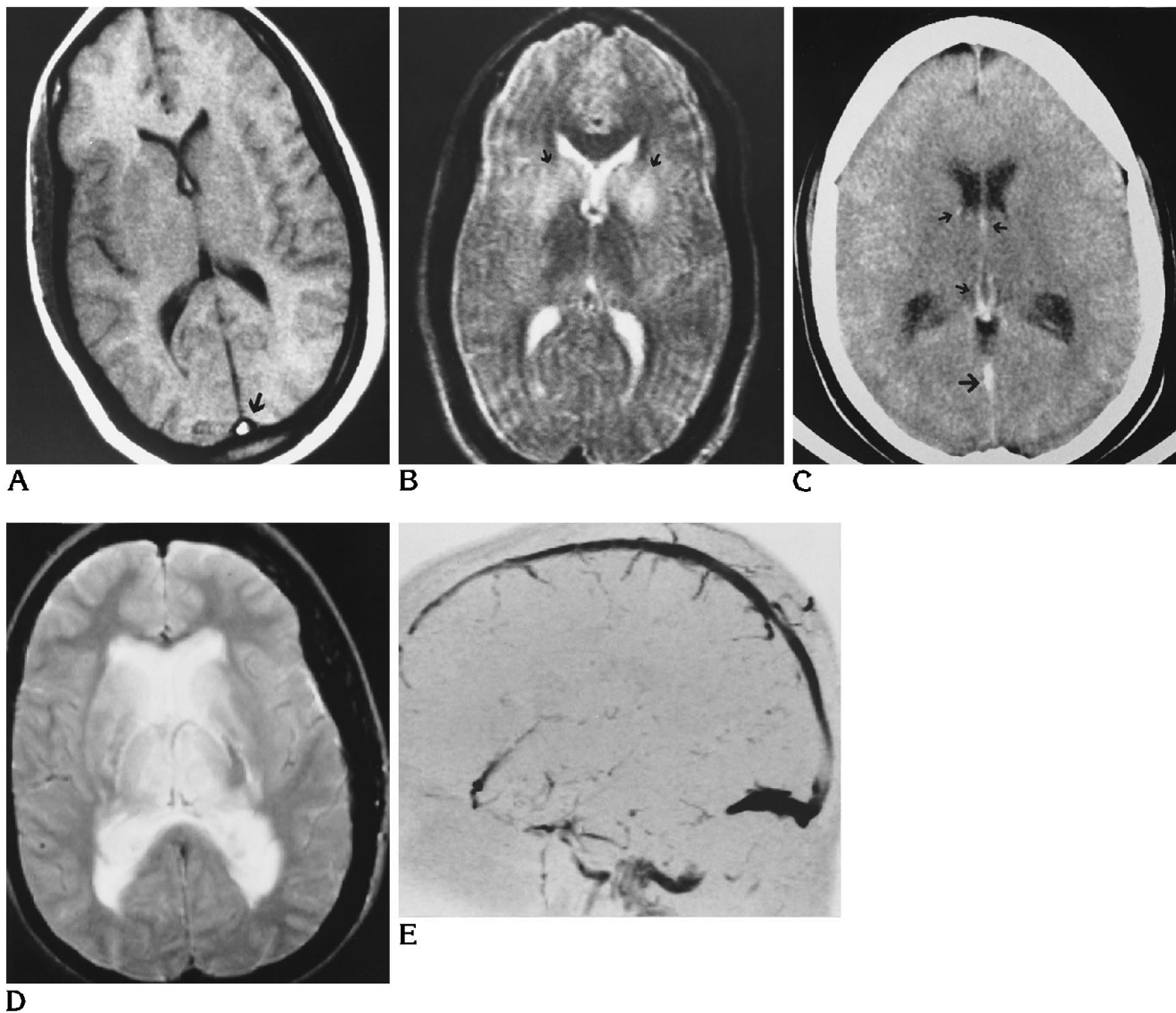
Fig 3. *A* and *B*, Patient 13, stage III (early).

A, Spin-echo T1-weighted MR shows increased signal in the superior sagittal sinus (*arrow*) and small frontal horns.

B, Spin-echo T2-weighted MR shows increased signal in both basal ganglia (*arrows*).

C–E, Patient 16, stage III (late).

C, Noncontrast CT shows increased attenuation in the straight sinus, vein of Galen, and both internal cerebral veins (*arrow*). Decreased attenuation is seen in both thalamic areas (*arrows*).

D, Spin-echo proton-density MR image shows extensive edema in both ganglia and thalamic areas with enlargement of both lateral ventricles. The cerebrospinal fluid signal in the lateral ventricles is more intense than normal because of the obstruction.

E, On 2-D time-of-flight MR venography the vein of Galen, straight sinus, and torcular areas are not seen because of thrombosis.

peripheral rim of low signal intensity. Among these 9 patients, one, patient 15, classified as stage III, had had recent posterior fossa surgery. The small hemorrhage was the result of surgery, not a sequela of dural sinus occlusion. Five patients were classified as stage IV (Fig 4), and 3 were stage V (Fig 5). Three stage V patients had CT without MR. These 3 patients, who did not receive thrombolysis, presented in stage IV

and deteriorated into stage V. Two of these patients had massive hematomas; the third had massive cerebral edema. Eleven patients had abnormal cortical venous enhancement.

Among the 19 patients with MR follow-up, ranging from 3 months to 2 years of age, 5 had persistent mass effect with cortical sulcal effacement. These 5 patients were treated with anticoagulants only. In patients who received
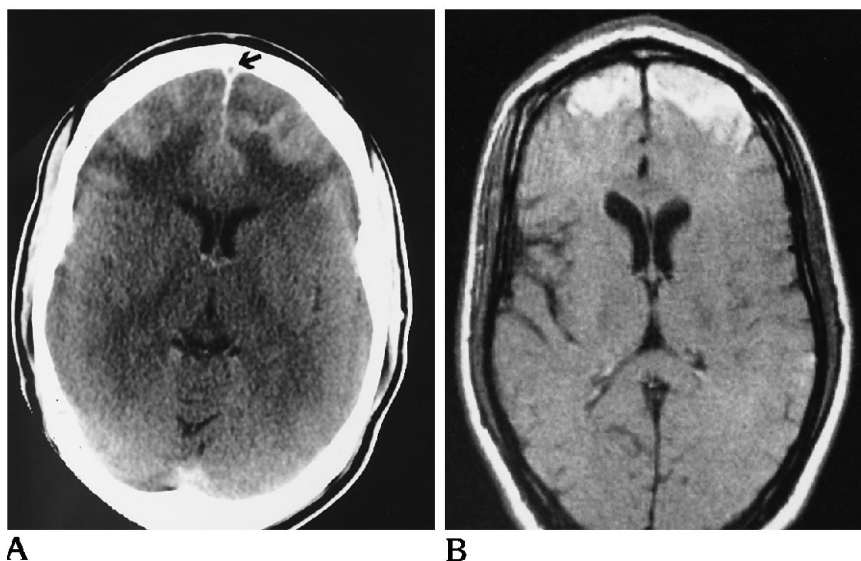
Fig 4. Patient 21, stage IV.

A, Contrast-enhanced CT shows a filling defect in the superior sagittal sinus anteriorly(*arrow*). The patient also had a filling defect posteriorly (not shown). Mixed attenuation is seen in both frontal lobes, indicating hemorrhage and edema.

B, Spin-echo T1-weighted MR image shows high signal intensity in both frontal lobes because of hemorrhage. The left frontal horn is slightly compressed (*arrow*). There is no definite signal abnormality arising from the superior sagittal sinus, unlike in the accompanying CT.

thrombolysis, the mass effect and parenchymal signal changes resolved completely within a few days to 1 week, except in four patients who had signal changes related to residual hemosiderin.

Discussion

Previous reports of CT and MR findings in cerebral venous and dural sinus thrombosis have focused on subacute and chronic cases (2–10). The recent reports of acute dural sinus thrombosis have not adequately described the clinical manifestation or its CT and MR appearance (1, 8–16).

From the analysis of our 29 patients, we have identified five stages of brain parenchymal changes, more than previously described (1, 11, 16). Each stage relates to the dural sinus pressure. The clinical and brain parenchymal changes may be related to the presence of collaterals through deep and cortical veins (1). As collateral flow decreases, more brain changes will be seen, and symptoms will worsen.

Categorizing our patients by stages correlates with clinical symptoms, imaging findings, and outcome. Stage I patients had mild clinical signs and symptoms, including headache, papilledema, and, rarely, seizures. All had normal mentation, and all had good outcomes. Imaging findings included the delta sign of superior sagittal sinus thrombosis on contrast-enhanced CT
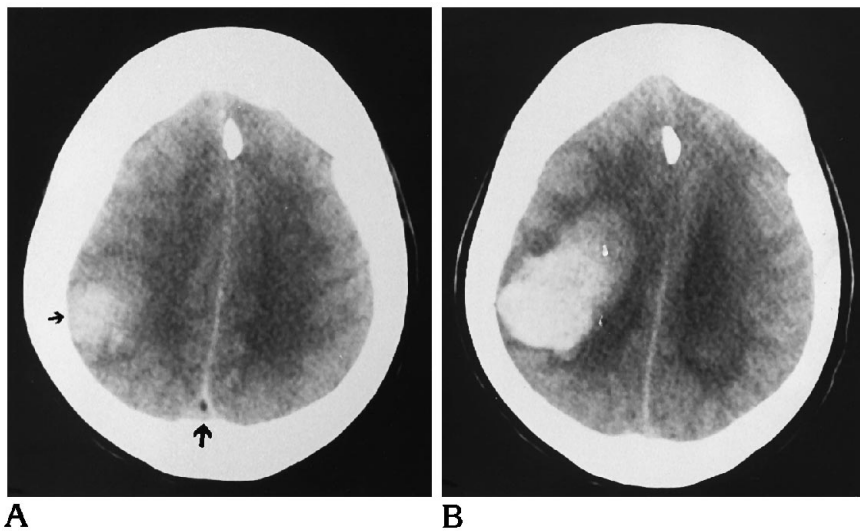

Fig 5. Patient 25, stage V.

A, Contrast-enhanced CT shows a filling defect in the superior sagittal sinus (*arrow*) and a small hematoma in the right posterior parietal lobe.

B, Follow-up noncontrast CT performed 3 days later shows a large hematoma in the right parietal lobe. The previously noted filling defect in the superior sagittal sinus is no longer seen.

or MR. Some stage I cases showed only mild brain swelling with cortical sulcal effacement.

Stage II patients were very drowsy with poor mentation. These patients may have brain swelling, but may not show imaging evidence of cerebral edema, hemorrhage, or abnormal enhancement, in which case their clinical presentation may be more severe than imaging would suggest. The four stage II patients had dural sinus pressures of 20, 23, 24, and 25 mm Hg. Imaging may show obliteration of sulci and sylvian fissures with small lateral ventricles, a picture that may be confused with acute middle cerebral artery occlusion. Additional evidence for dural sinus thrombosis includes a dural sinus filling defect on contrast-enhanced CT, increased attenuation in the venous system or dural sinus on noncontrast CT, or abnormal signal on MR imaging or MR venography. These patients recovered completely after thrombolysis.

Our stage III patients were obtunded or semicomatose. Brain parenchymal changes on imaging seem to correlate with previous reports suggesting that a high venous pressure alters the fluid exchange between the brain's intravascular and extravascular compartments, causing edema (12, 16, 17). Dural sinus pressures were 32, 35, 38, and 42 mm Hg. These patients recovered completely after thrombolysis.

Stage IV patients were comatose. Imaging showed brain edema and hemorrhage presumably caused by the rupture of small cortical veins as a result of venous drainage obstruction. Measured dural sinus pressures were as high as 48 and 51 mm Hg. Although thrombolysis is contraindicated for arterial occlusion in patients with cerebral edema on CT or MR, it is not contraindicated for acute dural sinus thrombosis. Patients receiving direct dural sinus venography and thrombolysis survived because thrombolysis stopped the clinical and radiologic deterioration. In our experience, thrombolysis can reverse early brain edema and swelling if used before hemorrhage occurs. In two stage IV patients, the proper diagnosis was overlooked. One was treated with anticoagulants with resulting massive infarcts in both occipital lobes, and the other had a hematoma removed surgically, resulting in permanent frontal lobe atrophy. These two patients had more parenchymal damage and greater deficits than the other stage IV patients.

All six stage V patients deteriorated from stage IV and demonstrated increasing hemorrhage and edema. In our series, this stage is inevitably fatal. Although dural sinus pressures were not measured, we postulate that they should exceed 50 mm Hg.

Dural sinus pressure seems to increase in 10- to 15-mm Hg increments per stage and correlates with brain parenchymal changes on imaging studies. The higher the pressure, the greater the parenchymal changes, which probably reflect interstitial edema rather than the cytotoxic edema of arterial infarction (12, 16, 17). The severity of injury associated with increasing dural sinus pressure is different in acute versus chronic pressure elevation. In chronic venous obstruction, the sinus pressures may be very high relative to our patients with acute thrombosis (12, 18).

The sites of parenchymal changes usually reflect thrombosis in the adjacent dural sinus. For example, bilateral frontal, parietal, and occipital lobe edema or hemorrhage usually corresponds to superior sagittal sinus thrombosis. However, the site of parenchymal change does not always reflect the site of thrombosis. We had two patients with bilateral basal ganglia edema. One patient had superior sagittal sinus thrombosis; the other had thrombosis of the deep cortical veins. Also, posterior fossa dural sinus thrombosis may cause supratentorial changes as well as edema or hemorrhage of the cerebellum and brain stem.

Hydrocephalus caused by elevated dural sinus and superior vena cava pressures has been well documented (12, 16, 19). The postulated mechanism is increased production and decreased absorption of cerebrospinal fluid. Our series includes cases of hydrocephalus caused by the obstruction of cerebrospinal fluid flow at the foramen of Monro attributable to edema of the basal ganglia and thalamus and obstruction of the aqueduct by brain stem edema. Once brain edema or swelling is diffuse, it may compress and decrease ventricular size rather than cause ventricular enlargement.

Although thrombolysis has been used extensively to treat acute arterial occlusion, acute dural sinus thrombolysis is just beginning. Thrombolysis is not appropriate for patients with edema or hemorrhage associated with acute arterial occlusion, but it is appropriate in cases of venous thrombosis, in which it should be started as soon as possible to reduce ele-

vated dural sinus pressure and relieve venous congestion. The described stages of dural sinus thrombosis occur because of varying degrees of venous congestion and elevated dural sinus pressure. Thrombolysis should not be withheld even if there is cerebral edema or hemorrhage. Even with massive venous infarction, no patient died after thrombolysis, unlike most of the untreated patients. Two patients survived after only anticoagulant therapy, but their neurologic deficits were much worse than in patients receiving even delayed thrombolysis. The other seven patients not treated with thrombolysis died. Stage I patients may be treated solely with anticoagulants; however, if their clinical condition deteriorates, we recommend prompt thrombolysis.

Summary

Acute dural sinus and cerebral venous thrombosis may lead to various stages of parenchymal changes of venous infarction, with the degree of severity depending on the degree of venous congestion and elevated dural sinus pressure. The prognosis of venous thrombosis depends to a significant extent on the use of thrombolytics. Severe neurologic symptoms, including coma, may be reversible if treatment with thrombolytics is started before massive cerebral edema or hemorrhage has developed. Stage I may be treated with anticoagulants alone; however, if the patient deteriorates clinically, we recommend prompt thrombolysis. All other stages should be treated with thrombolysis. A progression from mild brain swelling to severe cerebral edema and/or hemorrhage from increasing dural sinus pressure may occur if treatment is delayed.

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