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

Fong Y. Tsai, Ay-Ming Wang, Violet B. Matovich, Mark Lavin, Bruce Berberian, Tereasa M. Simonson, and William T. C. Yuh

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 mmHg. 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.
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>
<thead>
<tr>
<th>Stage</th>
<th>Parenchymal Changes</th>
<th>Symptoms</th>
<th>Pressure Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No parenchymal change</td>
<td>Severe headache, papilledema, weakness, changed mentation, drowsiness, right hemiparesis (one patient only)</td>
<td>One patient placed in this category had pressure measurements taken: left transverse sinus = 14 mm Hg; superior sagittal sinus = 17 mm Hg</td>
</tr>
<tr>
<td>II</td>
<td>Brain swelling, sulcal effacement and mass effect, no signal change</td>
<td>Increased headache, double vision, seizure, decreased mentation, extreme drowsiness, difficulty rousing, right lower extremity weakness (one patient)</td>
<td>Four patients had measurements taken: 20–25 mm Hg</td>
</tr>
<tr>
<td>III</td>
<td>Increased intensity of signal change as mild to moderate edema</td>
<td>Inability to rouse, obtundation, hemiparesis, seizure</td>
<td>Three patients had measurements taken: 32–38 mm Hg</td>
</tr>
<tr>
<td>IV</td>
<td>Severe edema, with or without hemorrhage</td>
<td>Hemiparesis, seizure, loss of consciousness, coma</td>
<td>Three patients had measurements taken: 42–51 mm Hg</td>
</tr>
<tr>
<td>V</td>
<td>Massive edema and/or hemorrhage</td>
<td>Coma, response to deep pain only</td>
<td>No measurements were taken</td>
</tr>
<tr>
<td>Patient</td>
<td>Age/Sex</td>
<td>Cause</td>
<td>Signs and Symptoms</td>
</tr>
<tr>
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</tr>
<tr>
<td>1</td>
<td>37 y/F</td>
<td>Oral contraceptives</td>
<td>Headache and papilledema</td>
</tr>
<tr>
<td>2</td>
<td>77 y/F</td>
<td>Undetermined</td>
<td>Lethargy, R hemiparesis</td>
</tr>
<tr>
<td>3</td>
<td>42 y/M</td>
<td>Undetermined</td>
<td>Increasingly severe headache</td>
</tr>
<tr>
<td>4</td>
<td>48 y/F</td>
<td>Undetermined</td>
<td>Weakness, decreasing mentation</td>
</tr>
<tr>
<td>5</td>
<td>35 y/F</td>
<td>Oral contraceptives</td>
<td>Increasingly severe headache, papilledema, decreased mentation</td>
</tr>
<tr>
<td>6</td>
<td>31 y/F</td>
<td>Postpartum VonWillebrand disease</td>
<td>Decreased mentation, severe headache</td>
</tr>
<tr>
<td>7</td>
<td>23 y/F</td>
<td>Drug use</td>
<td>Headache and neck pain, drowsiness</td>
</tr>
<tr>
<td>8</td>
<td>54 y/F</td>
<td>Undetermined</td>
<td>Increasing headaches, increasing frequency of seizures, drowsiness</td>
</tr>
<tr>
<td>9</td>
<td>38 y/M</td>
<td>Drug use</td>
<td>Papilledema, R lower extremity weakness, seizure, drowsiness</td>
</tr>
<tr>
<td>10</td>
<td>46 y/M</td>
<td>Drug use</td>
<td>Double vision, headache, decreased mentation</td>
</tr>
<tr>
<td>11</td>
<td>52 y/M</td>
<td>Alcoholism</td>
<td>R hemiparesis, semi-coma after head trauma</td>
</tr>
<tr>
<td>12</td>
<td>10 mo/M</td>
<td>Dehydration</td>
<td>Decreasing mentation and sucking power</td>
</tr>
<tr>
<td>13</td>
<td>23 y/F</td>
<td>Postpartum</td>
<td>Inability to rouse</td>
</tr>
<tr>
<td>14</td>
<td>58 y/M</td>
<td>Undetermined</td>
<td>Increasing L side weakness and obtundation</td>
</tr>
<tr>
<td>15</td>
<td>46 y/M</td>
<td>Postoperative R cerebellopontine angle tumor</td>
<td>Lethargy and obtundation</td>
</tr>
<tr>
<td>Patient</td>
<td>Age/Sex</td>
<td>Cause</td>
<td>Signs and Symptoms</td>
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<tr>
<td>16</td>
<td>37 y/F</td>
<td>Postpartum state</td>
<td>Photophobia, headache, decreased mentation, obtundation</td>
</tr>
<tr>
<td>17</td>
<td>6 d/M</td>
<td>Dehydration</td>
<td>Semicoma</td>
</tr>
<tr>
<td>18</td>
<td>43 y/F</td>
<td>Hypercoagulability</td>
<td>Lethargy and obtundation</td>
</tr>
<tr>
<td>19</td>
<td>25 y/F</td>
<td>Oral contraceptives</td>
<td>Coma</td>
</tr>
<tr>
<td>20</td>
<td>32 y/M</td>
<td>Alcoholism</td>
<td>Speech deficit, ataxia, diplegia, then coma for 5 d</td>
</tr>
<tr>
<td>21</td>
<td>27 y/M</td>
<td>Hypercoagulability</td>
<td>Seizure, loss of consciousness, R hemiplegia, sudden coma</td>
</tr>
<tr>
<td>22</td>
<td>22 y/F</td>
<td>Postpartum state</td>
<td>Coma</td>
</tr>
<tr>
<td>23</td>
<td>40 y/F</td>
<td>Undetermined</td>
<td>Dysarthria, then coma</td>
</tr>
<tr>
<td>24</td>
<td>52 y/M</td>
<td>Undetermined</td>
<td>Blindness, progressive decreasing mentation, sudden coma</td>
</tr>
<tr>
<td>25</td>
<td>60 y/F</td>
<td>Undetermined</td>
<td>L hemiplegia, decreasing mentation, sudden coma</td>
</tr>
<tr>
<td>26</td>
<td>29 y/M</td>
<td>Undetermined</td>
<td>Severe headache, decreasing mentation, sudden coma</td>
</tr>
<tr>
<td>27</td>
<td>21 y/F</td>
<td>Undetermined</td>
<td>Coma, response to deep pain only</td>
</tr>
<tr>
<td>28</td>
<td>18 y/M</td>
<td>Trauma</td>
<td>Coma</td>
</tr>
<tr>
<td>29</td>
<td>9 mo/F</td>
<td>Trauma</td>
<td>Lethargy, coma</td>
</tr>
</tbody>
</table>
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
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...

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**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.
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 appear-
ance (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 col-
laterals 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, pap-
illedema, and, rarely, seizures. All had normal
mentation, and all had good outcomes. Imaging
findings included the delta sign of superior sag-
ittal sinus thrombosis on contrast-enhanced CT.

Fig 4. Patient 21, stage IV.
A, Contrast-enhanced CT shows a fill-
ing defect in the superior sagittal sinus an-
teriorly (arrow). The patient also had a fill-
ing 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 (ar-
row). There is no definite signal abnor-
mality arising from the superior sagittal sinus,
unlike in the accompanying CT.

Fig 5. Patient 25, stage V.
A, Contrast-enhanced CT shows a fill-
ing defect in the superior sagittal sinus (ar-
row) and a small hematoma in the right
posterior parietal lobe.
B, Follow-up noncontrast CT per-
formed 3 days later shows a large hema-
toma in the right parietal lobe. The previ-
ously 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.

References