Effects of balloon angioplasty on perfusion- and diffusion-weighted magnetic resonance imaging results and outcome in patients with cerebral vasospasm

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Object. The aim of this study was to analyze the effects and outcome of transluminal balloon angioplasty (TBA) on brain tissue perfusion by using combined perfusion- and diffusion-weighted (PW/DW) magnetic resonance (MR) imaging in patients with cerebral vasospasm after subarachnoid hemorrhage.

Methods. Ten consecutive patients with cerebral vasospasm treated using TBA were included in this prospective study. Hemodynamically relevant vasospasm was diagnosed using a standardized PW/DW MR imaging protocol. Digital subtraction angiography was used to confirm vasospasm, and TBA was performed to dilate vasospastic arteries. The PW/DW imaging protocol was repeated after TBA. The evaluation of the passage of contrast medium after standardized application using the bolus tracking method allowed for the calculation of the time to peak (TTP) before and after TBA.

Tissue at risk was defined based on perfusion delays in individual vessel territories compared with those in reference territories. In cases with proximal focal vasospasm, TBA could dilate spastic arteries. Follow-up PW/DW MR imaging showed the disappearance of, or a decrease in, the mismatch. A TBA-induced reduction in the perfusion delay of 6.2 ± 1 seconds (mean ± standard error of the mean) to 1.5 ± 0.45 seconds resulted in the complete prevention of infarction; a reduction in the delay of 6.2 ± 2.7 to 4.1 ± 1.9 seconds resulted in the preservation of those brain tissue parts having only small infarcts in the vessel territories. Without TBA, however, the perfusion delay remained or even increased (11.1 ± 3.7 seconds), and the complete infarction of a territory occurred.

Conclusions. Angioplasty of vasospastic arteries leads to hemodynamic effects that can be quantified using PW/DW MR imaging. In cases of a severe PW/DW imaging mismatch successful TBA improved tissue perfusion and prevented cerebral infarction. The clinical significance of PW/DW MR imaging and the concept of tissue at risk is shown by cerebral infarction in vessels not accessible by TBA.

Key Words • subarachnoid hemorrhage • cerebral vasospasm • diffusion-weighted imaging • perfusion-weighted imaging • balloon angioplasty

Cerebral vasospasm and the side effects of aggressive HHH therapy are still major causes of death and disability in patients surviving SAH. One promising treatment for vasospasm and the prevention of tissue infarction is the dilation of spastic arteries by TBA. In a recent retrospective analysis, however, this procedure had no positive effect on outcome. Few other studies have been focused on the hemodynamic effects of TBA to further clarify its role in the treatment of vasospasm.

Methodologically, several questions must be answered when investigating the role of TBA in treating patients with cerebral vasospasm. 1) Is there a reduction in CBF that is caused by the angiographically visible vasospasm? 2) Can this reduction in CBF be reversed by TBA? 3) Is the reversal of a CBF reduction associated with fewer cerebral infarctions attributable to vasospasm?

In this context advances in MR imaging, including the development of PW and DW MR imaging, are of particular value.
Effects of angioplasty on perfusion-weighted imaging in vasospasm

allows one to diagnose tissue at risk for infarction, that is, tissue not yet infarcted but with misery perfusion to such an extent that infarction will ensue if ischemia is not rapidly reversed. At-risk tissue is still salvageable as shown by data from stroke studies of clot lysis. Moreover, perfusion changes and the efficacy of angioplasty can be monitored using PW and DW imaging.

The aim of this prospective study was to analyze the effects of TBA in a consecutive series of patients by using a PW and DW MR imaging-based protocol. We sought to clarify whether TBA, which was used selectively in patients with DS angiography–confirmed cerebral vasospasm and a perfusion deficit on PW/DW imaging, is effective in reversing that deficit and in preventing the infarction of tissue at risk.

Clinical Material and Methods

Patient Selection

Between October 2001 and November 2003, 204 patients with SAH of all clinical grades were admitted to our Department of Neurosurgery. A ventricular drain was placed in patients with a poor clinical grade (Hunt and Hess Scale) and in those with hydrocephalus. Whenever possible, surgery or coil embolization was performed within 72 hours after hemorrhage. Patients received nimodipine (Nimotop; Bayer, Leverkusen, Germany) either orally (60 mg/day × 6) or intravenously (2 mg/hour). Great care was taken to compensate for increased diuresis and natriuresis and to maintain a normovolemic and normotensive state. In selected cases, that is, in poor-grade patients, brain tissue PO₂ (Licox; Integra Neurosciences, Plainsboro, New Jersey) was invasively monitored. Computed tomography was indicated in cases of clinical deterioration. In the absence of structural causes, impending cerebral ischemia attributable to vasospasm was defined as a decrease to less than 15 mm Hg in tissue oxygenation, a 50% decrease in somatosensory evoked potential amplitude, an increase in somatosensory evoked potential latency, or an increase of greater than 150 cm/second in transcranial Doppler flow velocity. Additional signs of clinical vasospasm included a newly developed neurological deficit or a loss of at least two points on the Glasgow Coma Scale in the absence of other identifiable causes. A stepwise HHH protocol—hypervolemia, moderate hypertension, or aggressive hypertension—was followed. The absence of improvement prompted MR imaging studies using combined PW and DW MR imaging, while maintaining the HHH protocol during transportation as well as the imaging period. The results of detailed neurological examinations and any complications were fully documented. Outcome was assessed according to the mRS 6 months after SAH.

Patient Treatment

Between October 2001 and November 2003, 204 patients with SAH of all clinical grades were admitted to our Department of Neurosurgery. A ventricular drain was placed in patients with a poor clinical grade (Hunt and Hess Scale) and in those with hydrocephalus. Whenever possible, surgery or coil embolization was performed within 72 hours after hemorrhage. Patients received nimodipine (Nimotop; Bayer, Leverkusen, Germany) either orally (60 mg/day × 6) or intravenously (2 mg/hour). Great care was taken to compensate for increased diuresis and natriuresis and to maintain a normovolemic and normotensive state. In selected cases, that is, in poor-grade patients, brain tissue PO₂ (Licox; Integra Neurosciences, Plainsboro, New Jersey) was invasively monitored. Computed tomography was indicated in cases of clinical deterioration. In the absence of structural causes, impending cerebral ischemia attributable to vasospasm was defined as a decrease to less than 15 mm Hg in tissue oxygenation, a 50% decrease in somatosensory evoked potential amplitude, an increase in somatosensory evoked potential latency, or an increase of greater than 150 cm/second in transcranial Doppler flow velocity. Additional signs of clinical vasospasm included a newly developed neurological deficit or a loss of at least two points on the Glasgow Coma Scale in the absence of other identifiable causes. A stepwise HHH protocol—hypervolemia, moderate hypertension, or aggressive hypertension—was followed. The absence of improvement prompted MR imaging studies using combined PW and DW MR imaging, while maintaining the HHH protocol during transportation as well as the imaging period. The results of detailed neurological examinations and any complications were fully documented. Outcome was assessed according to the mRS 6 months after SAH.

Patient Selection

If signs and symptoms of vasospasm persisted, patients underwent MR imaging. Once a mismatch of the PW and DW imaging results was identified on TTP mapping, HHH therapy was continued and DS angiography was performed. Ten consecutive patients, eight women and two men, with a mean age of 51.5 ± 3.2 years (range 40–69 years) were enrolled for TBA to treat severe vasospasm refractory to medical treatment. Hunt and Hess grades in patients ranged from III to V, including three Grades IV and two Grades V. Nine aneurysms were located in the anterior circulation or at the PCoA, and one was situated at the basilar tip. Six aneurysms were clipped, and four were occluded with coils (Table 1).

Magnetic Resonance Imaging Protocol

Before and after TBA, MR imaging was performed at 1.5 tesla (Magneton Vision; Siemens, Munich, Germany). The standardized imaging protocol included axial native T₁-weighted imaging, axial T₂-weighted imaging, and axial fluid-attenuated inversion-recovery sequences. In addition, all patients underwent DW and PW imaging. The former was performed using a single-shot spin echo–echo planar imaging sequence (b value 1000 seconds/mm²). Bollus-tracking PW images were acquired using a gradient echo–echo planar imaging sequence. After a standardized intravenous contrast agent injection (0.1 mmol/kg Gd–diethylene triamine pentaacetic acid) at a flow rate of 5 ml/second, 40 T₁-weighted images for each of the 12 slices were obtained at 2-second intervals. In all major vessel territories and adjacent areas of abnormality indicated on DW images, regions of interest were measured for TTP and mean transit time. Results were compared with corresponding values for the contralateral vascular territories. The TTP measurement protocols were assessed immediately after scanning. The mean transit time did not differ from the TTP values, and therefore only the TTP results are provided for further analysis. We considered tissue to be at risk when there was no sign of infarction or only small infarcts on DW images of a major vessel territory together with a perfusion deficit indicating a TTP delay of at least 2 seconds in the same territory compared with the contralateral or ipsilateral major vessel territory—that is, in cases of a PW/DW imaging mismatch.

Digital Subtraction Angiography and TBA

The presence of vasospasm was first verified on DS angiography and then compared with the initial angiogram obtained after SAH. The degree of vasospasm was classified, according to the scheme by Kassell and colleagues, into absent, mild, moderate, or severe, and focal (< 2 cm) or diffuse (> 2 cm). The angiography procedure and TBA are described in detail elsewhere.

Study End Points

The primary end point of the present study was to compare the PW/DW imaging results after TBA with those before TBA. As a secondary end point, we noted the occurrence of new infarctions in vessel territories demonstrating a critical delay on PW imaging and successful TBA, compared with vessel territories revealing a critical delay on PW imaging that were not treatable using TBA. An additional end point included the 6-month posttreatment clinical outcome.

Results

The results are summarized in Table 2. No patient had in-
A fraction of a major vessel territory on baseline MR images before TBA. Only very small, punctate lesions were visible on DW imaging in eight patients. Two patients (Cases 1 and 8) showed acute, newly developed hemiparesis and lethargy, but no lesions were visible on DW images. On the PW images there were large perfusion deficits greater than 2 seconds in eight patients (mean deficit 6.5 ± 0.88 seconds, range 2.1–16.4 seconds). The areas of perfusion deficits were much larger than the small lesions on the DW images. We considered these cases with no or only small lesions on DW imaging to be consistent with TBA. Cases 9 and 10 had no TBA-related deficits, and mRS scores were 0.

### TABLE 1

Summary of characteristics and outcome in 10 patients with cerebral vasospasm

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Hunt &amp; Hess Grade</th>
<th>Fisher Grade</th>
<th>Aneurysm Site</th>
<th>Hrs to Surgery After SAH</th>
<th>TBA Treatment</th>
<th>No. of Days From SAH to TBA</th>
<th>Admin of Nimodipine</th>
<th>mRS Score at 6 Mos Postictus</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>45, F</td>
<td>III</td>
<td>4</td>
<td>MCA</td>
<td>26</td>
<td>clip</td>
<td>3</td>
<td>iv</td>
<td>2 (not returned to work)</td>
</tr>
<tr>
<td>2</td>
<td>43, F</td>
<td>IV</td>
<td>3</td>
<td>ACoA</td>
<td>68</td>
<td>clip</td>
<td>7</td>
<td>no</td>
<td>5 (dependent)</td>
</tr>
<tr>
<td>3</td>
<td>62, M</td>
<td>III</td>
<td>3</td>
<td>ACoA</td>
<td>24</td>
<td>coil</td>
<td>5</td>
<td>iv &amp; oral</td>
<td>1 (retired)</td>
</tr>
<tr>
<td>4</td>
<td>46, F</td>
<td>IV</td>
<td>3</td>
<td>MCA</td>
<td>24</td>
<td>clip</td>
<td>5</td>
<td>oral</td>
<td>1 (back to work)</td>
</tr>
<tr>
<td>5</td>
<td>48, F</td>
<td>V</td>
<td>3</td>
<td>ACoA</td>
<td>15</td>
<td>coil</td>
<td>5</td>
<td>no</td>
<td>2 (back to work)</td>
</tr>
<tr>
<td>6</td>
<td>40, F</td>
<td>III</td>
<td>3</td>
<td>PCoA</td>
<td>29</td>
<td>clip</td>
<td>7</td>
<td>oral</td>
<td>0 (back to work)</td>
</tr>
<tr>
<td>7</td>
<td>69, F</td>
<td>III</td>
<td>3</td>
<td>ACoA &amp; MCA (unruptured)</td>
<td>24</td>
<td>coil &amp; coil</td>
<td>6</td>
<td>iv</td>
<td>5 (dependent)</td>
</tr>
<tr>
<td>8</td>
<td>62, F</td>
<td>V</td>
<td>3</td>
<td>tip of BA</td>
<td>32</td>
<td>coil</td>
<td>11</td>
<td>iv</td>
<td>4 (dependent)</td>
</tr>
<tr>
<td>9</td>
<td>43, M</td>
<td>III</td>
<td>3</td>
<td>ACoA</td>
<td>115</td>
<td>clip</td>
<td>12</td>
<td>oral</td>
<td>0 (back to work)</td>
</tr>
<tr>
<td>10</td>
<td>57, F</td>
<td>IV</td>
<td>3</td>
<td>PCoA</td>
<td>48</td>
<td>coil</td>
<td>6</td>
<td>oral</td>
<td>6 (dead)</td>
</tr>
</tbody>
</table>

* ACoA = anterior communicating artery; admin = administration; BA = basilar artery; iv = intravenous.

### TABLE 2

Results of PW and DW MR imaging before and after angioplasty

<table>
<thead>
<tr>
<th>Case No.</th>
<th>DW Lesion Before TBA</th>
<th>Affected Vessel</th>
<th>Pre-TBA</th>
<th>MRT After TBA</th>
<th>TBA</th>
<th>DW Lesions After TBA</th>
<th>TTP After TBA</th>
<th>ΔTTP</th>
<th>Presence of Infarct/ Extent of Growth</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>no</td>
<td>ant MCA-lt</td>
<td>6.7</td>
<td>2.5</td>
<td>C1-lt</td>
<td>70</td>
<td>no</td>
<td>1.2</td>
<td>-5.5</td>
</tr>
<tr>
<td>2</td>
<td>no</td>
<td>mid MCA-lt</td>
<td>9.4</td>
<td></td>
<td>M1-lt</td>
<td>no</td>
<td>no</td>
<td>1.8</td>
<td>-7.6</td>
</tr>
<tr>
<td>3</td>
<td>no</td>
<td>pst MCA-lt</td>
<td>16.4</td>
<td></td>
<td>no</td>
<td>11.4</td>
<td>-5.0</td>
<td>yes/small</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>white matter-bilat</td>
<td>CC-rt</td>
<td>NA</td>
<td>NA</td>
<td>C1-lt</td>
<td>24</td>
<td>A1-lt</td>
<td>2100</td>
<td>no change</td>
</tr>
<tr>
<td>5</td>
<td>no</td>
<td>MCA</td>
<td>4.2</td>
<td>28</td>
<td>M1-lt</td>
<td>46</td>
<td>no</td>
<td>1.4</td>
<td>-2.8</td>
</tr>
<tr>
<td>6</td>
<td>no</td>
<td>ACA</td>
<td>5.1</td>
<td>4.5</td>
<td>M2-lt</td>
<td>46</td>
<td>A1-lt</td>
<td>0.9</td>
<td>yes/small</td>
</tr>
<tr>
<td>7</td>
<td>border zone of ACA</td>
<td>MCA/ACA-bilat</td>
<td>6.1</td>
<td></td>
<td>ACS-lt</td>
<td>6</td>
<td>ACA-bilat</td>
<td>6.7</td>
<td>+0.6</td>
</tr>
<tr>
<td>8</td>
<td>insula-lt</td>
<td>ant MCA-lt</td>
<td>2.1</td>
<td>6</td>
<td>M1-lt</td>
<td>21</td>
<td>decrease in signal intensity</td>
<td>-0.1</td>
<td>-2.2</td>
</tr>
<tr>
<td>9</td>
<td>no</td>
<td>ACA-lt</td>
<td>5.4</td>
<td>5</td>
<td>not done</td>
<td>384</td>
<td>ACA-bilat</td>
<td>25.8</td>
<td>+20</td>
</tr>
<tr>
<td>10</td>
<td>ACA-bilat (small)</td>
<td>ACA</td>
<td>5.8</td>
<td>5</td>
<td>A1-lt</td>
<td>1.3</td>
<td>1.5</td>
<td>0.8</td>
<td>no/normal</td>
</tr>
</tbody>
</table>

* ant = anterior; CC = corpus callosum; DW lesion = lesion demonstrated on DW imaging; mid = middle; MRT = timing of MR imaging studies; NA = not applicable; pst = posterior.

† Delay demonstrated on PW images of affected vessel territories compared with that for the contra- or ipsilateral territory.

‡ In Case 7, pre-TBA refers to MR images obtained before angiography, and MRT after TBA refers to MR images obtained after angiography.
DW images together with large perfusion deficits as a PW/DW imaging mismatch, signifying tissue at risk for infarction (Figs. 1 and 2).

In one patient (Case 2) PW imaging was not possible because of motion artifacts, and we proceeded with angiography given our high suspicion of a vasospasm. In another patient (Case 3) there was no perfusion deficit greater than 2 seconds, and DS angiography was not done. When he suffered an acute decrease in his level of consciousness 91 hours later, however, angiography and TBA were performed.

In all cases with a PW/DW imaging mismatch, DS angiography data confirmed severe narrowing (> 66%) of the arteries supplying the territories with misery perfusion. We performed angioplasty of spastic segments in nine of 10 patients (Table 2). Control after TBA appeared on angiograms as normal or near-normal diameters (Figs. 1 and 2) of the previously dilated arteries. Although at-risk tissue was revealed on imaging, we did not perform angioplasty in the patient in Case 7 because of diffuse, distal vasospasm.

Follow-up DW images revealed no lesion (Cases 6 and 9) or only very small lesions in all patients who underwent TBA. There was no DW imaging–demonstrated lesioning of a complete vessel territory when the supplying artery was successfully treated with angioplasty (Cases 1–6 and 8–10), although there were large lesions (territory infarcts) on DW images when the supplying arteries had not been dilated (Cases 2, 7, and 8). Transluminal balloon angioplasty effected an improvement in perfusion in the respective territory, as shown on PW images. The perfusion delays (TTP) were shortened to the range of −0.3 to −8 seconds, with a mean improvement of −3.5 ± 0.57 seconds via TBA (Fig. 3). The extent of the tissue perfusion improvements varied and did not include the vessel territories homogeneously so that small areas with a PW/DW imaging mismatch remained after TBA (Table 2 and Fig. 3). The TBA-induced reduction of a large (> 2 seconds) perfusion delay from 4.2, 6.7, 9.4, 2.1, 10.8, 3.1, 5, 8.8, and 5.4 seconds (mean 6.2 ± 1 seconds) to 1.4, 1.2, 1.8, −0.1, 2.8, 0, 0, 3.3, and 3.1 seconds (mean 1.5 ± 0.45 seconds), respectively, resulted in the preservation of tissue at risk (Cases 1, 4, 6, and 8–10). The reduction of a large (> 2 seconds) perfusion delay from 5.4, 16.4, 5.1, 6.1, and 2.2 seconds (mean 6.2 ± 2.7 seconds) to 4.5, 11.4, 2.5, 6.7, and 1.1 seconds (mean 4.1 ± 1.9 seconds) resulted in the preservation of parts of tissue with small infarcts in these vessel territories (Cases 1, 4, 5, and 10). When TBA was not done at all (Case 7) or was not performed in a specific vessel (right ACA and left MCA in Case 8), territory infarcts and two small infarcts (left ACA in Cases 5 and 10) developed. Similarly, in the patient in

Fig. 1. Case 8. Initial T2-weighted (a) and DW (b) MR images obtained before TBA, showing no sign of infarction. A PW MR image (c) obtained before TBA, demonstrating a large perfusion deficit (TTP delay > 10 seconds) in the right posterior MCA territory, which indicates tissue at risk. A DS angiogram (d) obtained 1 hour later, revealing focal vasospasm (arrow) in the right M1 segment. The A2 segment was vasospastic but not suitable for TBA (arrowhead). Follow-up T2-weighted (e) and DW MR images (f) obtained 225 hours after TBA, exhibiting no infarction in the right MCA territory where the angioplasty had reduced the perfusion delay by more than 8 seconds. A PW MR image (g) displaying a perfusion deficit (> 6.2 seconds) and infarction in the left posterior MCA. Note the infarction in the ACA territory (e) with a perfusion deficit of 9.1 seconds. A DS angiogram (h) showing successful dilation of the right M1 segment.
Case 2, perfusion deficits of 8.2 and 8 seconds on the follow-up MR image were related to territory infarcts; however, a deficit of 3.1 seconds was associated with only a small infarct in parts of the MCA territory. In all instances of an increased TTP delay on the follow-up MR imaging study, this increase (that is, the worsening of perfusion) occurred in territories of undilated vessels.

The effect of TBA in resolving the PW/DW imaging mismatch and salvaging at-risk tissue is demonstrated in the patient in Case 8 (Fig. 1); consider the patient in Case 7 with tissue at risk that eventually became infarcted because angioplasty was not performed (Fig. 2). Data in Case 1 of this consecutive series has been already reported in more detail.1

### Treatment Outcome

In three of the five patients who were awake before TBA, the deficit improved immediately after the procedure. In a fourth patient there was temporary worsening of the hemiparesis (Case 4), and a fifth remained temporarily dependent on a ventilator (Case 8). All these patients showed neurological improvement at 6 months (Table 1). Six of nine patients who had undergone TBA scored 2 or better on the mRS 6 months thereafter and were independent. One patient (Case 2) with a large PW/DW imaging mismatch after TBA and one (Case 7) who had not been treated with TBA were severely disabled and dependent 6 months thereafter. The patient in Case 8 remains dependent, but lives at home (Table 2). The patient in Case 10 died during the acute phase due to the infarction of large territories because of distal vessel narrowing that was not amenable to TBA.

### Discussion

This study is the first in which the hemodynamic changes of TBA by using PW and DW imaging as well as outcome has been prospectively examined in a consecutive series of patients. Our findings indicate that angioplasty substantially improves brain tissue perfusion leading to the preservation of tissue that would have otherwise had a high likelihood of infarcting. Obtaining serial MR images, before and after TBA, made it possible to quantify the extent that angioplasty indeed reduced a perfusion deficit caused by arterial narrowing. Successfully reducing by 0.9 to 8 seconds (mean 4.7 ± 0.73 seconds) a large perfusion delay (>2 seconds) caused by vasospasm prevented tissue infarction. Without a preexisting perfusion deficit, as in the MCA territory in the patient in Case 5 (<0.5 seconds), there was only marginal further (0.3 seconds) reduction, underscoring the need for perfusion tests before angioplasty. In four of 16 angioplasties, follow-up DW images revealed new, small lesions in the border zones between treated and untreated territories. Note, however, that the location and shape of these infarcts indicated particularly hemodynamic reasons rather than embolic events due to endovascular treatment.

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Fig. 2. Case 7. Initial T2-weighted (a) and DW (b) MR images showing signs of infarction in parts of the left ACA territory. A PW MR image (c) revealing perfusion deficits of 5.8 and 9.7 seconds (TTP delay) in both ACAs. A DS angiogram (d) featuring a diffuse, distal spasm that precluded a TBA. Follow-up T2-weighted (e) and DW (f) MR images obtained 16 days later, showing complete infarction of the ACA on both sides. A PW MR image (g) exhibiting a perfusion deficit of 7.3 and 25.8 seconds.
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Tissue Perfusion: Symptomatic Vasospasm

We know from PW/DW imaging studies of ischemic stroke that perfusion deficits can cause infarction. These ischemic stroke figures may not be applicable to ischemia caused by vasospasm, because a less severe perfusion deficit may be sufficient to cause infarction given the distinct pathophysiology of vasospasm. In the present study MR imaging results showed that at-risk tissue becomes infarcted when angioplasty is not performed. Combined PW/DW imaging as a tool that identifies symptomatic vasospasm—that is, vasospasm that eventually leads to tissue infarction—is highly valuable. With this method we were able to prove the efficacy of TBA for the improvement of tissue perfusion and the prevention of infarct.

Analyzing the TTP before and after TBA revealed the quantitative relationship between tissue perfusion and tissue preservation. All tissue with a perfusion deficit greater than 3.3 seconds on the MR imaging study obtained post-TBA eventually became infarcted. Infarction also ensued in the patient in Case 7, in whom TBA was not possible as well as in cases of single vessels not treated with TBA (Case 5, left ACA; Case 8, right ACA; and Case 10, left ACA) and when TBA could not sufficiently reduce the perfusion deficit (Case 1, posterior MCA; Case 2, left MCA; Case 4, posterior MCA; and Case 10, right posterior MCA). These findings support the hypothesis of a cause and effect relationship between successful TBA and tissue salvage and implies that angioplasty completely preserved brain tissue in at least six patients (Cases 1, 4, 6, and 8–10) and prevented additional growth of the infarct in other cases (Cases 1, 4, 5, and 10). Despite these remarkable findings on the effect of TBA, more data are needed to calculate the specificity and sensitivity for combined PW/DW imaging.

Angiographically confirmed vasospasm was present in all vessel territories with MR imaging–demonstrated tissue at risk. It is essential to detect at-risk tissue in vasospasm because many cases of arterial narrowing do not lead to ischemia. If no misery perfusion is present, it is unlikely that TBA can enhance perfusion, and thus TBA would create only risks and no benefit. With PW/DW imaging one can open and widen the window of opportunity for TBA, reserving the procedure for patients with still viable tissue at risk.

Alternative Methods

There is a need for diagnostic studies that reliably identify hemodynamic compromise and select patients who will benefit from TBA. Transcranial Doppler flow velocity studies, most commonly used as a daily routine, often proved unreliable and not sensitive or specific enough for the diagnosis of symptomatic vasospasm. Clinical judgment is the mainstay in identifying delayed neurological deterioration, but many patients are comatose or sedated. Microdialysis, thermal–diffusion flowmetry, or measuring brain tissue PO2 are promising invasive techniques. Cerebral blood flow measurements using Xe–computed tomography, single-photon emission computed tomography, positron-emission tomography, or computed tomography perfusion studies have also been used.

Study Limitations

The perfusion measurements in this study are only semi-quantitative in nature, and absolute values of tissue perfusion cannot be calculated at present. It is only possible to compare the regions of interest with their contralateral counterparts or other regions; therefore, global disturbances of perfusion can be missed using the PW MR imaging method. We must make an effort to perform imaging studies in patients, especially those with poor clinical grades, who are intubated and receiving ventilation. Combined PW/DW imaging is a costly, elaborate, and time-consuming procedure. There may be hazards for patients because of the restricted accessibility and monitoring possibilities in the imaging unit. Because of motion artifacts, image quality may not always be sufficient to quantify perfusion indices. This factor limits the clinical value, but with increasing practice the failure rate can be reduced. Incorporating high-quality MR angiography to reveal vasospasm may complete the MR imaging protocol and help to select those patients at-risk tissue and focal proximal arterial narrowing who will most likely benefit from TBA. Although our hypothesis has been tested in a limited number of patients thus far, we believe that the PW/DW imaging protocol enables the diagnosis of symptomatic vasospasm (that is, tissue ischemia caused by arterial narrowing that, if left untreated, leads to infarction) and that the therapeutic implication of proceeding with angioplasty only when there is misery perfusion and still salvageable tissue warrants further study.

Conclusions

Angioplasty has tremendous and lasting effects on brain tissue perfusion and can prevent infarction, thus improving outcome in patients with cerebral vasospasm. The results of this study favor TBA as a treatment option for severe prox-
imal vasospasm and reduced tissue perfusion. Based on these first results, analysis of combined PW and DW imaging studies can help diagnose tissue at risk for cerebral vasospasm and thus may be suitable for selecting patients for TBA. One should consider applying TBA in conjunction with PW/DW imaging more often and not merely as a last-resort measure.

Acknowledgment

The excellent assistance of Marina Eberhardt in preparing this manuscript is thankfully acknowledged. Some details of Case 1 were already published as a short report to propose the concept of tissue at risk in cerebral vasospasm after SAH.1

References

Effects of angioplasty on perfusion-weighted imaging in vasospasm


Manuscript received May 19, 2005. Accepted in final form May 17, 2006.
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