Diffusion- and Perfusion-Weighted Brain Magnetic Resonance Imaging in Patients With Neurologic Complications After Cardiac Surgery

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Background: Neurologic complications after cardiac surgery include stroke, encephalopathy, and persistent cognitive impairments. More precise neuroimaging of patients with these complications may lead to a better understanding of the etiology and treatment of these disorders.

Objective: To study the pattern of ischemic changes on diffusion- and perfusion-weighted magnetic resonance imaging (DWI, and MRPI, respectively) in patients with neurologic complications after cardiac surgery.

Methods: All records were reviewed of our patients undergoing cardiac surgery in the previous year who also underwent postoperative DWI or MRPI. Neurologic symptoms, vascular studies, and the pattern of ischemic changes were recorded. Acute ischemic lesions were classified as having a territorial, watershed, or lacunar pattern of infarction. Patients with multiple territorial infarcts in differing vascular distributions that were not explained by occlusive vascular lesions were classified as having multiple emboli.

Results: Fourteen patients underwent DWI and 4 underwent MRPI. Acute infarcts were found in 10 of 14 patients by DWI as compared with 5 of 12 patients by computed tomography. Eight patients presented with encephalopathy (associated with focal neurologic deficits in 4), 4 with focal deficits alone, and 2 with either fluctuating symptoms or transient ischemic attacks. Among patients with encephalopathy, 7 of 8 had patterns of infarction suggestive of multiple emboli, including 3 of 4 patients with no focal neurologic deficits. Several patients had combined watershed and multiple embolic patterns of ischemia. Findings of MRPI studies were abnormal in 2 of 4 patients, showing diffusion-perfusion mismatch; both patients had either fluctuating deficits or transient ischemic attacks, and their conditions improved with blood pressure manipulation.

Conclusions: In patients with neurologic symptoms after cardiac surgery, DWI is more sensitive to ischemic change than computed tomographic scanning and can demonstrate patterns of infarction that may help us understand etiology. The most common pattern was multiple embolic infarcts. Preliminary experience with MRPI suggests that some patients have persistent diffusion-perfusion mismatch after surgery and may benefit from therapeutic intervention.

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Prevention and treatment of neurologic complications remain a challenge in the management of patients undergoing cardiac surgery. Stroke occurs in approximately 2% to 3% of patients after coronary artery bypass grafting, with higher rates after valve replacement or other cardiac surgical procedures.5,6 Patients with stroke have poorer functional outcomes and incur greater medical costs with longer hospital stays.1,3 Many potential mechanisms have been proposed for stroke after cardiac surgery, including perioperative embolism from the heart or aortic arch, systemic hypoperfusion, ischemia from large-vessel occlusive disease, or a combination of these factors.6,7 Previous studies have identified risk factors for stroke after cardiac surgery,8,9 but few studies have used advanced imaging techniques to investigate patients with stroke in detail.10,11

For editorial comment see page 549

In addition to stroke, other neurologic complications in the postoperative period include delirium, seizures, and persistent cognitive impairment. Approximately 10% of patients experience postoperative encephalopathy, and up to 37% of patients display persistent cognitive decline on neuropsychological testing re-
PATIENTS AND METHODS

We reviewed the medical records and imaging studies of all patients who had DWI performed after cardiac surgery between March 1999 and January 2000. Patients were identified by review of a prospectively maintained cardiac surgery database and a registry of MRI studies. More than 1000 cardiac surgery procedures were performed at our institution a year, and during the study period, 30 patients underwent brain imaging studies (either computed tomography [CT] or MRI), although not all patients received a final diagnosis of stroke. At our institution, DWI has been available since 1997 for selected patients with stroke but became part of the routine sequence for brain MRI in mid 1999. Additionally, MRPI has been available for clinical use since 1999. Medical records were reviewed with attention to stroke risk factors, intraoperative parameters, neurologic deficits, and the clinical impression by the consulting neurologist prior to imaging studies. (All patients in this series were seen in consultation by a neurologist.)

Computed tomographic studies were reviewed by a neuroradiologist who was given the clinical history but not the MRI findings. Only CT scans performed prior to MRI studies were analyzed. The MRI studies were then reviewed with the prior CT scan available. The pattern of stroke was categorized as follows: (1) territorial infarction in the distribution of a cerebral artery; (2) watershed infarction in the border zone between vascular territories (either superficial or deep territories)26,27; and (3) lacunar infarction, defined as a small (<1-cm) infarction in the distribution of a penetrating artery. Embolic infarcts were suspected in patients with territorial infarcts in the absence of large-vessel occlusive disease, particularly if multiple or bilateral territorial infarcts were found.

RESULTS

We identified 14 patients who underwent DWI studies after cardiac surgery between March 1999 and January 2000. There were 9 men and 5 women with an average age of 69 years. Twelve patients underwent coronary artery bypass grafting (1 with simultaneous carotid endarterectomy), 1 had a mitral valve replacement, and 1 patient with Marfan disease underwent repair of a dissecting aneurysm of the ascending aorta. During the study period, there were approximately 1300 cardiac surgical procedures of various types performed, of which 30 were associated with neurologic complications. Of the 14 patients who underwent DWI studies, 13 (93%) had coronary artery disease; 9 (64%), hypertension; 6 (43%), hyperlipidemia; 5 (36%), diabetes; and areas of relatively decreased cerebral perfusion that may be at risk for infarction.26,27 We report our preliminary experience with diffusion and perfusion MRI in patients with neurologic complications after cardiac surgery. In our ser-
4 (29%) were current smokers. The most common neurologic symptom after surgery that prompted neurologic consultation was encephalopathy in 8 patients, 4 of whom also had local neurologic findings on examination. Two patients presented with visual field deficits, 3 with hemiparesis, and 1 with a transient ischemic attack (TIA) of aphasia. At the time of hospital discharge, the final neurologic diagnosis was stroke in 11 patients and seizure, unexplained encephalopathy, and TIA in 3 others, respectively.

Twelve patients underwent CT scans. The first scan was taken on postoperative day 3 on average, with a range from day 0 to 6. Acute infarcts were seen in 5 of 12 scans and were classified as single territorial infarcts in 2 scans and multiple territory infarcts in 3. In contrast, the first DWI study was performed on postoperative day 6, on average (range, day 3-15), and revealed acute infarcts in 10 patients (10 of 11 patients with final clinical diagnosis of stroke). In 4 of 5 patients with acute infarcts demonstrated by CT, DWI revealed additional infarcts in other vascular territories. For example, patient 7 had a single territorial infarct on CT, but MRI revealed several other lesions in both hemispheres (Figure 1).

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>Cardiac Procedure</th>
<th>Neurologic Symptoms</th>
<th>Acute CT Lesion</th>
<th>Acute DWI Lesion</th>
<th>MRPI</th>
<th>Vascular Lesions</th>
<th>Stroke Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/48</td>
<td>Proximal aortic aneurysm repair</td>
<td>Encephalopathy</td>
<td>POD 2: none</td>
<td>POD 3: bilateral watershed, multiple bilateral infarcts in the anterior and posterior circulation</td>
<td>Not done</td>
<td>MRA: dissection extending into both CCAs and L ICA</td>
<td>Bilateral watershed + multiple emboli</td>
</tr>
<tr>
<td>2/M/76</td>
<td>CABG</td>
<td>Encephalopathy</td>
<td>POD 0: R&gt;L occipital infarcts</td>
<td>POD 2: bilateral watershed, multiple bilateral infarcts in the anterior and posterior circulation</td>
<td>Not done</td>
<td>Not done</td>
<td>Bilateral watershed + multiple emboli</td>
</tr>
<tr>
<td>3/M/67</td>
<td>CABG</td>
<td>Encephalopathy</td>
<td>Not done</td>
<td>POD 6: multiple small bilateral infarcts involving anterior and posterior circulations</td>
<td>Not done</td>
<td>Duplex: none</td>
<td>Multiple emboli</td>
</tr>
<tr>
<td>4/M/58</td>
<td>CABG</td>
<td>Encephalopathy</td>
<td>POD 2: none</td>
<td>POD 7: none</td>
<td>Normal</td>
<td>Duplex: none; MRA: intracranial ICA stenosis</td>
<td>No infarct</td>
</tr>
<tr>
<td>5/M/61</td>
<td>CABG (with postoperative hypotension)</td>
<td>Encephalopathy, R hemiparesis</td>
<td>POD 7: none</td>
<td>POD 8: multiple bilateral infarcts, most in deep watershed territory</td>
<td>Normal</td>
<td>Duplex: none; MRA: none</td>
<td>Bilateral watershed + multiple emboli</td>
</tr>
<tr>
<td>6/F/56</td>
<td>CABG</td>
<td>Encephalopathy, L hemiparesis</td>
<td>POD 2: R MCA and R PCA territory infarcts</td>
<td>POD 15: subacute R MCA and R PCA infarcts, acute deep R MCA infarct</td>
<td>Not done</td>
<td>Duplex: none</td>
<td>Multiple emboli</td>
</tr>
<tr>
<td>7/M/66</td>
<td>CABG</td>
<td>Encephalopathy, aphasia</td>
<td>POD 3: L parietal infarct</td>
<td>POD 4: L temporal-parietal infarct, multiple small bilateral infarcts in the anterior and posterior circulation</td>
<td>Not done</td>
<td>Duplex: none; MRA: none</td>
<td>Multiple emboli</td>
</tr>
<tr>
<td>8/F/77</td>
<td>Mitral valve replacement</td>
<td>Encephalopathy, bilateral visual field loss</td>
<td>POD 5: R&gt;L subacute occipital infarcts</td>
<td>POD 5: R&gt;L occipital infarcts, tiny bilateral MCA territory infarcts</td>
<td>Not done</td>
<td>Duplex: R ICA 60%-80% stenosis</td>
<td>Multiple emboli</td>
</tr>
<tr>
<td>9/M/76</td>
<td>CABG</td>
<td>R visual field loss</td>
<td>POD 3: none</td>
<td>POD 3: L&gt;R posterior watershed infarcts</td>
<td>Not done</td>
<td>Duplex: none; MRA: none</td>
<td>Bilateral watershed</td>
</tr>
<tr>
<td>10/F/88</td>
<td>CABG + R CEA</td>
<td>R visual field loss</td>
<td>POD 4: L occipital infarct</td>
<td>POD 6: L occipital infarct, tiny PICA infarct</td>
<td>Not done</td>
<td>Duplex: R ICA stenosis; MRA: L MCA stenosis</td>
<td>Single territorial infarct</td>
</tr>
<tr>
<td>11/M/81</td>
<td>CABG</td>
<td>R hemiparesis</td>
<td>POD 6: none</td>
<td>POD 12: none</td>
<td>Not done</td>
<td>Not done</td>
<td>No infarct (clinically suspected lacunar stroke) No infarct</td>
</tr>
<tr>
<td>12/F/68</td>
<td>CABG</td>
<td>L hemiparesis, later seizures</td>
<td>POD 1: none</td>
<td>POD 4: none</td>
<td>Not done</td>
<td>MRA: L PCA and R intracranial ICA stenosis</td>
<td>Unilateral watershed and contralateral lacuna</td>
</tr>
<tr>
<td>13/F/29</td>
<td>CABG</td>
<td>Fluctuating L hemiparesis</td>
<td>POD 3: none</td>
<td>POD 4: deep R watershed infarct, small L thalamic infarct</td>
<td>Not done</td>
<td>ACA/MCA territory hypoperfusion Extensive L hemispheric hypoperfusion</td>
<td>Duplex: none</td>
</tr>
<tr>
<td>14/M/61</td>
<td>CABG</td>
<td>TIA of aphasia and confusion</td>
<td>Not done</td>
<td>POD 2: none</td>
<td>Not done</td>
<td>Extensive L hemispheric hypoperfusion</td>
<td>TIA with L hemispheric hypoperfusion</td>
</tr>
</tbody>
</table>

* DWI indicates diffusion-weighted magnetic resonance imaging; CT, computed tomography; MRPI, magnetic resonance perfusion imaging; POD, postoperative day; MRA, magnetic resonance angiography; CCA, common carotid artery; L, left; R, right; ICA, internal carotid artery; duplex, carotid duplex ultrasound; CABG, coronary artery bypass grafting; CEA, carotid endarterectomy; PICA, posteroinferior cerebellar artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; ACA, anterior cerebral artery; and TIA, transient ischemic attack.
The pattern of stroke by DWI suggested multiple emboli in 7 patients. Many of the infarcts identified by DWI were small and typically located in the cortex of the hemispheres or in the cerebellum (Figure 2). Infarcts generally seemed equally distributed between right and left hemispheres and involved both anterior and posterior circulations. A few acute lesions were in the territories of penetrating arteries and were small enough to be considered lacunar infarcts. Five patients had a watershed pattern of infarction; 3 of these patients had coexistent multiple territorial infarcts, while 2 had only watershed infarcts. Only 1 patient (No. 5) in our series had significant postoperative hypotension requiring vasopressor agents; DWI showed bilateral deep watershed infarcts as well as a number of small, scattered lesions also suggestive of emboli in other territories. In several of these patients with combined watershed and embolic patterns, the small cortical infarcts seemed to cluster and coalesce in the watershed territory (Figure 2).

Among the 8 patients presenting with postoperative encephalopathy, 7 had multiple infarcts apparent on DWI consistent with emboli, including 3 of 4 patients who had no focal neurologic deficits on examination. One patient had normal findings on both DWI and MRPI studies performed on postoperative day 7. The following day, he dramatically improved, and his delirium was thought to have been metabolic in origin. Three other patients had normal DWI findings: 1 had clinical findings consistent with a lacunar stroke (pure motor hemiparesis); 1 later developed seizures; and 1 had TIAs without a persistent neurologic deficit.

Four patients underwent MRPI studies; the findings in 2 of these were instructive. One patient (patient 14) had several spells of “confusion” and nonsensical speech several days after coronary artery bypass grafting. An MRPI revealed extensive hypoperfusion of the left hemisphere that corresponded with his known chronic left internal carotid artery occlusion. Both the number and doses of antihypertensive medications were reduced. After the patient’s blood pressure rose from 110/60 mm Hg to an average of 140/80 mm Hg, his TIAs ceased. Another patient (patient 13) had a deep watershed infarct apparent on DWI,
but had a larger perfusion abnormality in the middle and anterior cerebral artery territories (Figure 3). This patient had hemiparesis with marked fluctuations in the degree of weakness over 24 hours. Average blood pressure was 130/60 mm Hg at that time. Antihypertensive medications were reduced, resulting in an elevation of average blood pressure to 160/70 mm Hg and an improvement in arm strength that remained through discharge. The 2 remaining patients had normal results on MRPI studies.

COMMENT

To our knowledge, ours is the only series in the literature to report the results of diffusion- and perfusion-weighted MRIs in patients after cardiac surgery. Several pertinent findings emerged: (1) DWI was clearly more sensitive than CT in detecting ischemic lesions; and (2) DWI also made small cortical or periventricular infarcts more conspicuous than did conventional MRI sequences.30 In addition, however, DWI improved the specificity of detecting acute ischemic lesions. Many patients with coronary artery disease have coexistent cerebrovascular disease, with either old infarcts or chronic periventricular ischemic matter changes evident on baseline imaging studies.31 These abnormalities make it difficult to distinguish small, acute infarcts in postoperative patients from chronic lesions. Deep watershed infarcts, for example, are often located in the white matter of the centrum semiovale,39 a region in which many patients have chronic ischemic changes. On the other hand, DWI allows one to distinguish an acute lesion from chronic ischemia (Figure 3), with confirmation by measurement of the apparent diffusion coefficient. In humans, the apparent diffusion coefficient typically remains depressed for about 10 to 14 days, and thereafter normalizes, allowing one to identify lesions that have occurred within the previous 2 weeks.26

The most common finding on DWI in our series was the presence of multiple small infarcts, typically spread throughout the cortical regions of the anterior and posterior circulations and suggestive of a shower of emboli of varying sizes. Most of our patients with a single infarct evident on CT or conventional MRI were found on DWI to have additional lesions. Several patients had a pattern of multiple embolic infarcts that clustered and merged with watershed infarcts. However, an association of watershed infarction with multiple emboli is not surprising because small embolic particles are likely to migrate into distal vascular territories.31 As postulated by Caplan and Hennerici,32 the combination of small embolic particles and hypotension (eg, during cardiopulmonary bypass) can lead to delayed washout of emboli and produce watershed territory infarction. Most of the patients with a pattern of multiple embolic infarcts presented with encephalopathy, either alone or associated with focal neurologic findings. Further study of patients with this pattern of infarction should include assessment of long-term recovery and cognitive function. It would also be interesting to determine
if these patterns correlate with embolic counts from transcranial Doppler studies or the presence of extensive aortic arch atheroma. Because of the retrospective nature of our study, we cannot assess the frequency of unexpected small infarcts in patients with encephalopathy after cardiac surgery; only selected patients underwent DWI studies during the study period.

Finally, we used MRPI in 4 of our patients and found significant perfusion abnormalities with diffusion-perfusion mismatch in 2. In patients presenting with acute ischemic stroke, diffusion-perfusion mismatch is thought to identify a region of brain potentially at risk for infarction, but potentially salvageable by reperfusion.33-35 Reperfusion may be accomplished by thrombolysis, mechanical opening of a stenotic vessel (eg, carotid endarterectomy or angioplasty), or increasing cerebral perfusion by blood pressure elevation. The protocol for blood pressure management after cardiac surgery at our institution has been to maintain relatively low blood pressures to prevent bleeding complications in the mediastinum and at suture lines. Reduced blood pressure, however, may be detrimental to cerebral perfusion in the setting of acute stroke owing to loss of cerebral autoregulation or the presence of high cerebral resistance in patients with chronic hypertension.30

Two of our patients had fluctuating symptoms (recurrent TIAs in one, fluctuating weakness in the other), which corresponded to a region of relative hypoperfusion on MRPI. In both cases, increasing blood pressure by careful reduction of antihypertensive medications was associated with amelioration of symptoms. In studies of patients with acute ischemic stroke, diffusion-perfusion mismatch tends to diminish over time as infarction progresses. An MRPI may therefore be of greater importance if performed much earlier after cardiac surgery than in our series.

In conclusion, our preliminary findings show the value of DWI and MRPI in patients with neurologic complications after cardiac surgery. These new imaging techniques better define the degree and distribution of ischemic injury and may reveal regions of persistent hypoperfusion. This information is important in the study of mechanisms of neurologic injury in patients undergoing cardiac surgery, and is also relevant for the clinician searching for an explanation of neurologic dysfunction in the postoperative patient.

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REFERENCES