Brain Damage After Open Heart Surgery in Patients With Acute Cardioembolic Stroke

Michiyuki Maruyama, MD, Yoshihiro Kuriyama, MD, Tohru Sawada, MD, Takenori Yamaguchi, MD, Tsuyoshi Fujita, MD, and Teruo Omae, MD

We evaluated 14 patients with acute cardiogenic embolism who underwent open heart surgery soon after the onset to determine the cerebral and cardiac factors that influence neurologic outcome. The mean interval from onset of cerebral embolism to surgery was 5.3 (range 1–16) days. Five of the 14 patients had vegetations from infective endocarditis (including prosthetic valve endocarditis) as embolic sources, eight had intracardiac thrombi, and one had atrial myxoma. The diagnosed site of infarction before surgery was based on computed tomographic and/or angiographic findings. Of the 14 patients, four had infarcts due to major artery occlusion, seven due to cortical branch occlusion, and two due to perforating artery occlusion; one patient presented with a transient ischemic attack without computed tomographic abnormalities. Ten patients (71%) showed no clinical aggravation after open heart surgery; however, two patients died of massive cerebral hemorrhage, one died of deterioration of brain edema, and another became comatose from midbrain hemorrhage immediately after surgery. The four patients with clinical aggravation comprised three with septic embolism and one with aseptic occlusion of a major artery. From these results, infective endocarditis and a large infarct appear to be possible aggravating factors when patients with recent cerebral embolism undergo open heart surgery. (Stroke 1989;20:1305–1310)
TABLE 1. Summary of Clinical Data for 14 Patients With Open Heart Surgery After Acute Cerebral Embolism

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Cardiac source</th>
<th>Site</th>
<th>Interval (day)</th>
<th>ECC (hr)</th>
<th>Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>68</td>
<td>F</td>
<td>Thrombi</td>
<td>Perforating branch of basilar artery</td>
<td>10</td>
<td>1.75</td>
<td>Good</td>
</tr>
<tr>
<td>2</td>
<td>58</td>
<td>F</td>
<td>Thrombi</td>
<td>Perforating branch of R MCA</td>
<td>5</td>
<td>2.66</td>
<td>Good</td>
</tr>
<tr>
<td>4</td>
<td>44</td>
<td>M</td>
<td>Myxoma</td>
<td>Cortical branch of R MCA</td>
<td>4</td>
<td>1.66</td>
<td>Good</td>
</tr>
<tr>
<td>6</td>
<td>48</td>
<td>F</td>
<td>Thrombi</td>
<td>Cortical branch of L MCA</td>
<td>16</td>
<td>1.50</td>
<td>Good</td>
</tr>
<tr>
<td>7</td>
<td>58</td>
<td>M</td>
<td>Infective endocarditis</td>
<td>TIA without abnormality on CT</td>
<td>11</td>
<td>1.90</td>
<td>Good</td>
</tr>
<tr>
<td>8</td>
<td>50</td>
<td>M</td>
<td>Thrombi</td>
<td>Cortical branch of R MCA</td>
<td>16</td>
<td>1.75</td>
<td>Good</td>
</tr>
<tr>
<td>9</td>
<td>44</td>
<td>F</td>
<td>Thrombi</td>
<td>Cortical branch of L MCA</td>
<td>1</td>
<td>2.33</td>
<td>Good</td>
</tr>
<tr>
<td>11</td>
<td>36</td>
<td>M</td>
<td>Infective endocarditis</td>
<td>Cortical branch of L MCA</td>
<td>1</td>
<td>3.70</td>
<td>Good*</td>
</tr>
<tr>
<td>13</td>
<td>42</td>
<td>F</td>
<td>Thrombi</td>
<td>L MCA trunk</td>
<td>2</td>
<td>2.30</td>
<td>Good</td>
</tr>
<tr>
<td>14</td>
<td>43</td>
<td>F</td>
<td>Thrombi</td>
<td>Cortical branch of R MCA</td>
<td>1</td>
<td>1.66</td>
<td>Good</td>
</tr>
</tbody>
</table>

Mean±SD 6.7±6.1 2.12±0.17

Aggravation (N=4, coma in 7% and brain death in 21%)

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Cardiac source</th>
<th>Site</th>
<th>Interval (day)</th>
<th>ECC (hr)</th>
<th>Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>32</td>
<td>F</td>
<td>Infective endocarditis</td>
<td>Cortical branch of L MCA</td>
<td>4</td>
<td>2.90</td>
<td>Death from hemorrhage</td>
</tr>
<tr>
<td>5</td>
<td>57</td>
<td>F</td>
<td>Infective endocarditis</td>
<td>L ICA trunk</td>
<td>1</td>
<td>2.20</td>
<td>Coma from hemorrhage</td>
</tr>
<tr>
<td>10</td>
<td>29</td>
<td>M</td>
<td>Thrombi</td>
<td>R MCA trunk</td>
<td>1</td>
<td>2.50</td>
<td>Death from hemorrhage</td>
</tr>
<tr>
<td>12</td>
<td>39</td>
<td>M</td>
<td>Infective endocarditis</td>
<td>R MCA trunk</td>
<td>1</td>
<td>5.60</td>
<td>Death from brain edema</td>
</tr>
</tbody>
</table>

Mean±SD 1.8±1.5 3.30±1.56

Interval, time from cerebral embolism to surgery; ECC, extracorporeal circulation time; F, female; M, male; R, right; L, left; MCA, middle cerebral artery; TIA, transient ischemic attack; CT, computed tomogram; ICA, internal carotid artery.

*Hemorrhagic change on CT after surgery.

sion, seven had infarcts due to occlusion of the cortical branches of the middle cerebral artery, and two had infarcts due to occlusion of perforating arteries. No CT abnormalities were found in the remaining patient with transient ischemic attacks (Table 1). No hemorrhagic lesions were apparent on preoperative CT scans.

Results

Among the 10 patients with postoperative CT scans, five showed no change from the preoperative CT scan, four showed hemorrhagic changes, and one exhibited marked swelling of the brain. After surgery, the clinical condition in 10 patients (71%) was unchanged, two patients died of massive cerebral hemorrhage, one patient died of herniation due to deterioration of brain edema, and one patient fell into a coma due to midbrain hemorrhage (Table 1). These aggravations of the four patients' clinical conditions occurred ≤24 hours after open heart surgery. The cardiac disorders in two patients with hemorrhage and in the patient with brain edema consisted of infective endocarditis, and the other patient with hemorrhage had intracardiac thrombi from a prosthetic valve (Table 1). Two patients with hemorrhage and the patient with brain edema had infarcts due to occlusion of major arteries, and the other patient with hemorrhage had infarction due to septic occlusion of a cortical branch (Table 1). That is, infective endocarditis or a large infarct appeared to be possible factors aggravating clinical condition after surgery. The interval from embolism to surgery in the patients with aggravated conditions was ≤1 day in three and 4 days in one, giving a mean shorter than that of the 10 patients without aggravation, although the difference was not significant (Table 1). The mean duration of extracorporeal circulation of the four patients with aggravated conditions was longer than that of the 10 patients without aggravation, although the difference was not significant (Table 1). Illustrative cases will now be described.

Case 3. A 32-year-old woman was admitted to another hospital after suffering from high fever for a month, and she abruptly developed right hemiparesis. Although the infecting organisms could not be identified in several blood cultures, she was diagnosed as having infective endocarditis from an
FIGURE 1. Computed tomograms (CT scans) of Case 3 on admission (top) showed medium-sized infarct of left temporoparietal lobe (arrows). CT scans immediately after open heart surgery (bottom) revealed hemorrhage at site of former infarct, with bleeding into lateral ventricle.

ultrasonic cardiogram and was administered antibiotics. She was transferred to our hospital for surgical treatment of acute heart failure due to severe aortic valve regurgitation. A CT scan revealed an infarct presumably due to septic occlusion of a cortical branch of the left middle cerebral artery. Angiography was not performed because of heart failure. Due to cardiac emergency, open heart surgery was performed 4 days after her stroke. There were no problems during surgery, and aortic valve vegetations were confirmed. However, 12 hours after surgery the patient abruptly fell into a deep coma with left mydriasis. An immediate CT scan demonstrated massive hemorrhage at the site of the former infarct (Figure 1). She died of brain herniation the next day.

Case 5. A 57-year-old woman with right hemiparesis was diagnosed as having infective endocarditis and septic cerebral embolism due to occlusion of a cortical branch of the left middle cerebral artery by CT and angiography, but the infecting organisms could not be identified by blood culture. In spite of the administration of antibiotics, the vegetation on the aortic valve demonstrated by ultrasonic cardiography grew larger. On Day 10 after admission, she displayed an aggravation of her level of consciousness. On that day, she underwent aortic valve replacement due to medically uncontrollable infection, and the vegetations consisting of cocci were confirmed pathologically. During surgery, she developed right mydriasis and fell into a coma. Angiography (Figure 2) and CT (Figure 3) after surgery revealed that the left internal carotid artery had been occluded just before surgery and that multiple hemorrhages in the right midbrain and elsewhere developed during surgery. The patient has been in a vegetative state since.

Case 14. A 43-year-old woman with mitral stenosis abruptly developed left hemiparesis and speech disturbance. She was admitted to our hospital and diagnosed as having a nonseptic cardiogenic embolism due to occlusion of a cortical branch of the right middle cerebral artery by CT angiography. Intracardiac thrombi were detected by ultrasonic cardiography. To prevent recurrent embolism, open mitral commissurotomy and thrombectomy were performed the next day. She did not have any neurologic aggravation after surgery, and a postoperative CT scan did not show hemorrhagic transformation or deterioration of brain edema (Figure 4).

Discussion

Analysis of neurologic complications encountered after open heart surgery has been reported by many authors.2-5 It is well known that the use of heparin and the hypotension brought about by extracorporeal circulation during such operations can lead to brain damage, such as intracranial hemorrhage or cerebral ischemia. Moreover, in the clinical course of cardiogenic cerebral embolism,6-8 there is a potentially high incidence of hemorrhagic transformation of the infarct and/or deterioration of edema during the acute phase. For these reasons, open heart surgery in patients with recent infarction has been considered to give rise to a high incidence of serious neurologic complications and is even considered to be contraindicated. At present, we do not have adequate information concerning the outcome of patients with recent cerebral infarction who undergo open heart surgery and the clinical factors that determine the prognosis of these patients. Zisbrod et al1 reported the clinical results of open heart surgery in patients with recent cerebral embolism. All their patients except one with atrial myxoma suffered from infective endocarditis; these authors included no patients with aseptic thromboembolism, and details of the cerebral embolism were not fully described. To our knowledge, ours is the first report to include patients with aseptic embolism evaluated closely by neurologists with neuroradiologic examinations.

As a reference concerning the potential for hemorrhagic complications that may be brought about by the use of anticoagulants during surgery, papers describing preventive therapy by immediate anticoagulation following embolic stroke would be instructive. Generally speaking, patients with aseptic cerebral embolism have a better overall prognosis if
FIGURE 2. Left carotid angiogram of Case 5 on admission (left) showed occlusion of left middle cerebral artery branch (arrows). Angiogram after open heart surgery (right) revealed occlusion of left internal carotid artery, which was considered to have become complete just before surgery.

FIGURE 3. Computed tomograms (CT scans) of Case 5 immediately after open heart surgery revealed infarct in left deep terminal zone due to occlusion of left internal carotid artery (arrowheads) and multiple small hemorrhages in right midbrain and elsewhere (arrows) unrelated to site of infarct.
FIGURE 4. Computed tomograms (CT scans) of Case 14 before (left) and after (right) open heart surgery showed infarct due to nonseptic embolic occlusion of cortical branch of right middle cerebral artery. There was no deterioration of infarct on CT scan after surgery.

Anticoagulants are used immediately after the onset. However, the relation between hemorrhage and infarct size remains controversial. Some studies have suggested that the occurrence of hemorrhagic infarction may be related to infarct size. Our results for patients with aseptic embolism were similar to those in the above reports on immediate anticoagulation in aseptic cardioembolic stroke. The outcome after surgery in patients with aseptic embolism was relatively fair. All but one, who died of hemorrhage, did not deteriorate clinically, and this excepted patient had a large infarct before surgery.

On the other hand, the risk of hemorrhage in patients with infective endocarditis should be regarded as different from that of patients with aseptic embolism. Spontaneous intracranial hemorrhage occurs in approximately 5% of patients with infective endocarditis. Hart et al. stated that hemorrhage complicating infective endocarditis is a result of arterial injuries. On this basis, because of the existence of arterial injury unlike that in patients with aseptic embolism, infective endocarditis as a cardiac disorder is considered to be a determinate factor of hemorrhagic complications even with the use of anticoagulants during open heart surgery. Actually, among our patients, three of five with infective endocarditis showed hemorrhagic changes on postoperative CT scans, and two such changes were accompanied by clinical deterioration. Case 3 had a moderate-sized infarct limited to one cortical branch territory, but she died of expansion of secondary hemorrhage, which was diagnosed on postmortem examination as bleeding from a ruptured artery with septic occlusion. Case 5 developed multiple hemorrhages at other sites of infarction during surgery; this bleeding might have originated from an injured arterial wall that was not identified before surgery.

Brain edema accompanying cerebral infarction also contributes to the clinical course. Ischemic brain edema is considered to be most severe approximately 7 days after onset. Therefore, it is considered that hypotension and/or nonpulsatile blood flow brought about by the use of extracorporeal circulation during open heart surgery during the acute phase must promote the development of ischemic edema. Among our patients, only one (with a large infarct, who underwent cardiac surgery ≤ 1 day after onset) deteriorated clinically due to expansion of edema, and no other patient had clinical aggravation resulting from increased edema. Open heart surgery during the acute phase of infarction may promote the development of brain edema to a greater or lesser extent, but it seems that deterioration of edema after surgery is important only in patients with massive infarcts.

Although it is difficult to draw any definite conclusions from our few patients, we suggest that 1) clinical outcome after cardiac surgery in patients…
with recent aseptic cerebral embolism is relatively fair; but 2) a large infarct (irrespective of the etiology of embolism) is accompanied by a high risk of both hemorrhagic transformation of the infarct and expansion of edema, with clinical deterioration after surgery; 3) the possibility of arterial injury in patients with infective endocarditis (especially during intractable infection), even if the infarct is small, increases the incidence of hemorrhagic complications after surgery; and 4) patients with clinical aggravation tend to have a shorter interval from the onset of embolic stroke to surgery and/or a longer duration of extracorporeal circulation during open heart surgery than patients without clinical aggravation. To confirm these impressions, further study of larger groups of patients are needed.

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References


Key Words • cardiovascular diseases • cerebral infarction • embolism • heart surgery
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