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)

Subjects and Methods

Of 2,900 adults with valvular heart disease undergoing open heart surgery at our hospital from December 1980 through November 1988, we evaluated 14 consecutive patients with a history of recent (≤3 weeks) cardiogenic cerebral embolism. These 14 patients underwent open heart surgery for various reasons such as medically uncontrollable infective endocarditis, acute cardiac dysfunction, and frequent recurrence of embolism. The intervals from the onset of cerebral embolism to surgery ranged from 1 to 16 (average 5.3) days. Cardiac sources for the cerebral emboli were diagnosed preoperatively by cardiologists and neurologists based on the clinical symptoms and detailed examinations; computed tomograms (CT scans) were performed in all 14 patients, and cerebral angiography was carried out in 10. During open heart surgery, nonpulsatile extracorporeal circulation under heparin administration was used in each patient. The clinical symptoms and results of radiologic examinations after surgery were recorded; 10 patients had postoperative CT scans.

There were six men and eight women aged 29–68 (average 46.3) years (Table 1). Five patients had vegetations from infective endocarditis (three had prosthetic valve endocarditis), eight had intracardiac thrombi from rheumatic valvular disease or a prosthetic heart valve, and one had atrial myxoma (Table 1). Four patients had infarcts due to internal carotid artery or middle cerebral artery trunk occlusion.
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>Emboli</th>
<th>Interval (day)</th>
<th>ECC (hr)</th>
<th>Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>No aggravation (N=10, 71%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>68</td>
<td>F</td>
<td>Thrombi</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>5</td>
<td>2.66</td>
<td>Good</td>
</tr>
<tr>
<td>4</td>
<td>44</td>
<td>M</td>
<td>Myxoma</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>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>11</td>
<td>1.90</td>
<td>Good</td>
</tr>
<tr>
<td>8</td>
<td>50</td>
<td>M</td>
<td>Thrombi</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>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>1</td>
<td>3.70</td>
<td>Good*</td>
</tr>
<tr>
<td>13</td>
<td>42</td>
<td>F</td>
<td>Thrombi</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>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>Emboli</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>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>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>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>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.

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 cardiology 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 noneptic cardiogenic embolism due to occlusion of a cortical branch of the right middle cerebral artery by CT and angiography. Intracardiac thrombi were detected by ultrasonic cardiology. 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 embolism, 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.
抗凝药物会很流的停止使用。然而，血栓和梗死部位的关系仍然存在争议。一些报告指出，血栓的发生与梗死面积有关。9-11 我们的报告显示，在大多数患者中，梗死面积相对较小。发生了凝固的患者没有表现出恶化，并且除了特殊情况之外，这个患者的梗死面积相对较大。

另一方面，对于伴有感染性心内膜炎的患者而言，出血的风险应被视为与无菌性心内膜炎不同。自发性颅内出血发生率为 5%左右。14-15. Hart et al.14 认为，由于动脉损伤导致出血。在这基础上，因为存在动脉损伤不同于无菌性心内膜炎，感染性心内膜炎是一种导致出血的决定性因素，即使在使用抗凝药物的情况下也是如此。实际上，我们患者的五分之三在手术后出现了出血变化，其中两例伴随临床恶化。Case 3 的中度梗死仅限于一个皮质区域，但由于手术后出血的扩展而死亡，该出血在尸检中被诊断为动脉破裂导致的。Case 5 发展了多个出血区域，这些出血可能来自手术前未诊断的动脉壁。

脑水肿伴随脑梗死也会影响临床过程。缺血性脑水肿在症状后 7 天左右最严重。16-17 因此，出血在手术期间会促进脑水肿的发展。在我们的患者中，只有一个人（在梗死面积较大，手术后 1 天内）由于脑水肿的扩展而恶化，其他患者没有临床恶化。手术期间脑水肿发展可能在较轻或较重程度，但术后脑水肿的恢复仅在大梗死患者中重要。

尽管难以从我们的少数患者中得出任何明确的结论，但我们提出以下假设：1) 手术后的临床结局在无菌性心内膜炎患者中至关重要。
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

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