Aggressive Decompressive Surgery in Patients with Massive Hemispheric Embolic Cerebral Infarction Associated with Severe Brain Swelling

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Summary

Massive hemispheric cerebral infarction, also known as malignant infarction, is characterized by rapid clinical deterioration due to brain swelling and downward transtentorial herniation, and is associated with a mortality of 80%. Early patient selection and establishment of the optimum therapeutic modality are important to improve the outcome. Early clinical, computed tomography (CT), and angiographic characteristics were analysed to identify patients with malignant infarction and external and internal decompression was performed, with unco-para-hippocampectomy if needed, and the outcomes were compared with those of conservative treatment.

Thirty-four of 55 patients admitted with large cerebral infarctions due to embolism showed rapid clinical deterioration due to brain swelling and herniation. These 34 patients were treated under a diagnosis of malignant infarction by decompressive surgical treatment (19 cases) or conservative treatment (15 cases).

CT showed significantly higher infarction volume in patients with malignant infarction (288 ± 62 cm³) compared to patients with non-malignant infarction (200 ± 57 cm³, P < 0.001) and angiography showed a higher incidence of recanalization of the occluded vessels in patients with malignant infarction (58%) compared to patients with non-malignant infarction (15%, P < 0.05). Discriminant analysis revealed that an infarction volume of more than 240 cm³ was predictive of malignant infarction with 76.4% accuracy. Basic clinical characteristics on admission and deterioration were not statistically different between the surgically treated and conservatively treated groups of patients with malignant infarction. The shift of midline structures was significantly improved (14 ± 3.5 to 10 ± 4.7 mm) after surgical treatment (P < 0.05), compared to deterioration (12 ± 5.8 to 15 ± 4.3 mm) after conservative treatment. The mortality was 67% in the conservative group and 16% in the surgical group. Surgical treatment significantly improved the mortality and Glasgow Outcome Scale score (P < 0.01). However, the mean Barthel Index scores of the survivors were not significantly different.

An infarct volume of more than 240 cm³ on CT and angiographic recanalization of the occluded artery are predictors of fatal brain swelling after massive cerebral infarction. Decompressive surgical treatment dramatically improves the mortality of massive hemispheric infarction.

Keywords: Cerebral infarction; brain herniation; decompressive surgery.

Introduction

Acute hemispheric cerebral infarction in the territory of the middle cerebral artery (MCA), and sometimes including the territories of the anterior cerebral artery and/or the posterior cerebral artery (PCA), may cause massive brain swelling associated with raised intracranial pressure (ICP) and brain herniation known as malignant MCA infarction [1, 8, 13, 16, 26]. Patients with malignant MCA infarction suffer progressive clinical deterioration and death, and treatment by only conservative methods results in a mortality of 80% [8, 25, 29]. Administration of brain protective agents such as calcium antagonist, high dose steroid, barbiturate, and hyperosmotic agent cannot prevent the increase in ICP and brain herniation caused by malignant MCA infarction [14, 21, 28]. Surgical intervention has been advocated to improve the mortality and morbidity of malignant MCA infarction [3, 4, 7, 13, 15, 24, 25, 28]. Both external decompression (hemicraniectomy) and internal decompression have been recommended, but the optimum surgical procedure remains unclear.

The present study investigated the early computed tomography (CT) and angiographic factors in patients with severe brain swelling due to embolic cerebral infarction in order to identify the indicators for malignant MCA infarction. This study also assessed the use of aggressive surgical intervention, based on external decompression with duroplasty, but if necessary including internal decompression with unco-para-hippocampectomy to treat downward transtentorial herniation.
Patients and Methods

Fifty-five patients with large supratentorial cerebral infarctions caused by cerebral embolism were admitted to the Department of Neurosurgery, Juntendo University Izu-Izunagaoka Hospital between 1993 and 1998. The diagnosis of cerebral embolism was established in patients with abrupt onset of focal neurological signs and symptoms with a probable source of emboli confirmed by echocardiography and/or electrocardiography. Patients with arrhythmia (atrial fibrillation) or valvar heart disease in whom cerebral angiography or magnetic resonance angiography showed either occlusive changes suggestive of embolus or patency of the vessels supplying the area of infarction without localized arteriosclerotic narrowing were considered to have suffered embolic infarction. All patients were admitted within six hours of the onset of symptoms. Patients aged more than ninety years, patients with any previous disabling neurological disease, and patients in extremely poor general condition such as those with severe cardiac failure were excluded from this study. All patients received adequate oxygen and hydration with osmotic therapy after admission. Steroids were not administered.

The 55 patients with embolic cerebral infarction were divided into two groups according to the clinical course and CT findings. Twenty-one patients who did not show any clinical deterioration, with or without slight brain swelling on CT, were regarded as having non-malignant MCA infarction. The other 34 patients were considered to have malignant MCA infarction, that is massive hemispheric infarction with severe brain swelling, based on the progressive worsening of their neurological condition and pupil asymmetry. CT was performed on admission and repeated when clinical deterioration occurred. CT at clinical deterioration demonstrated brain swelling associated with shift of the midline structures and compression of the perimesencephalic cisterns, suggesting the impending signs of downward transentorial herniation. Follow-up CT studies were also performed three to five times during the first two weeks to evaluate and detect the infarct volume, non-symptomatic haemorrhagic infarct, and non-symptomatic brain shift. CT was repeated in the non-malignant MCA infarction group to detect the maximum midline shift at the point that corresponded to the clinical deterioration in the malignant infarction group, namely at 2–5 days after the ictus. Cerebral angiography was performed in 13 patients with non-malignant MCA infarction and 19 patients with malignant MCA infarction on average 2.8 ± 3.3 days (1–13 days) after the ictus. These CT and angiographical findings were analysed to evaluate the radiological indicators predictive of massive cerebral swelling (malignant MCA infarct).

The 34 patients with malignant MCA infarction were divided into two subgroups. Nineteen patients were treated by external and/or internal decompression immediately after clinical deterioration associated with CT indications of massive cerebral swelling and brain herniation (surgically treated group). Fifteen patients with the same clinical course and CT indications of massive hemispheric infarction continued to be treated by conservative methods (consservatively treated group), because informed consent was not obtained from the patient’s family. Patients were intubated and mechanically ventilated to maintain the appropriate arterial oxygen and carbon dioxide tension if needed and conservative medical treatment was continued.

The principal surgical technique was external decompression (hemicraniectomy) with duroplasty. In brief, a large semicircular skin incision with an additional incision extending posteriorly was made. A large frontotemporoparietal craniectomy was performed and the temporal squama was rongeured out until the floor of the middle cranial fossa was exposed. The dura was opened in a cruciate fashion to allow the brain to expand outward. Duroplasty was performed by lyophilized cadaver dura or artificial dura substitute (Gore-tex®; W. L. Gore & Associates, Inc., Arizona, U.S.A.) If the temporal lobe swelling was extreme, the non-viable temporal lobe was removed. After temporal lobectomy, the operating microscope was introduced to observe the medial temporal structures. Unco- parhippocampectomy was also performed in four patients, in whom these structures showed compression necrosis and were packed into the tentorial incisura, to expose the compressed oculo-motor nerve, PCA, and midbrain [19]. The patients were mechanically ventilated to maintain mild hyperventilation for a few days after the operation.

The infarction volume and the midline shift (distance from the midline to the septum pelliculum) were measured by an investigator unaware of the treatment group using a computerized planimetric technique on the day of deterioration and the day after surgery or the next day for conservative therapy.

Patients were neurologically evaluated using the Glasgow Coma Scale (GCS) [11] on the day of admission, day of clinical deterioration, and one month after the ictus. Functional recovery was evaluated by the Glasgow Outcome Scale (GOS) [10] and the Barthel Index (BI) [18] score at three months. A BI score of 60–95 indicates a patient requiring minimal assistance with daily activities who is almost independent at home; a score of less than 60 implies that the patient is functionally dependent. Patients who died before the end of the three months outcome evaluation period were excluded from BI scoring because inclusion would have required a score for death. Since the GOS includes a rating for death, such patients could be included in the GOS analysis.

All data are expressed as mean ± standard deviation. Results were analysed for statistical significance using the chi-square ($\chi^2$) test, the Mann-Whitney rank sum (U) test, and discriminate analysis, with $P < 0.05$ considered significant. Statistical software, SPSS Release 7.5 (SPSS Inc., Illinois, U.S.A.) was used for these statistical analyses.

Results

Clinical and Radiological Differences Between Non-Malignant and Malignant Cerebral Infarction

Table 1 summarizes the clinical and neuroradiological characteristics of the 21 patients with non-malignant infarction and the 34 patients with malignant infarction. Figure 1 shows representative cases of non-malignant infarction (Fig. 1A) and malignant infarction (Fig. 1B). Age, male/female ratio, and dominant/nondominant affected hemisphere ratio were not significantly different between the two groups. Anti-coagulant (antiplatelet) drugs were administered before the ictus in 3 patients with malignant infarction and in 1 patient with non-malignant infarction. Thrombolytic therapy using intra-arterial injection of tissue plasminogen activator within three hours of the ictus was given to 9 patients with malignant infarction and 2 patients with non-malignant infarction. The incidences of anticoagulant therapy and thrombolytic therapy were not statistically different between the two groups. Seven of these 11 patients treated by thrombolytic therapy showed recanalization (64%) and 8