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# Is decompressive craniectomy for malignant middle cerebral artery infarction of any worth?*

YANG Xiao-feng (杨小锋), YAO Yu (姚 瑜)†‡, HU Wei-wei (胡未伟), LI Gu (李 谷), XU Jin-fang (徐锦芳), ZHAO Xue-qun (赵学群), LIU Wei-guo (刘伟国)

(*Second Affiliated Hospital, School of Medicine, Zhejiang University, Hangzhou 310009, China*)

†E-mail: yu_yao03@126.com

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**Abstract:** Objective: Malignant middle cerebral artery (MCA) infarction is characterized by mortality rate of up to 80%. The aim of this study was to determine the value of decompressive craniectomy in patients presenting malignant MCA infarction compared with those receiving medical treatment alone. Methods: Patients with malignant MCA infarction treated in our hospital between January 1996 and March 2004 were included in this retrospective analysis. The National Institute of Health Stroke Scale (NIHSS) was used to assess neurological status on admission and at one week after surgery. All patients were followed up for assessment of functional outcome by the Barthel index (BI) and modified Rankin Scale (RS) at 3 months after infarction. Results: Ten out of 24 patients underwent decompressive craniectomy. The mean interval between stroke onset and surgery was 62.10 h. The mortality was 10.0% compared with 64.2% in patients who received medical treatment alone ($P$<0.001). The mean NIHSS score before surgery was 26.0 and 15.4 after surgery ($P$<0.001). At follow up, patients who underwent surgery had significantly better outcome with mean BI of 53.3, RS of 3.3 as compared to only 16.0 and 4.60 in medically treated patients. Speech function also improved in patients with dominant hemispherical infarction. Conclusion: Decompressive craniectomy in patients with malignant MCA infarction improves both survival rates and functional outcomes compared with medical treatment alone. A randomized controlled trial is required to substantiate those findings.

**Key words:** Decompressive craniectomy, Cerebral infarction, Middle cerebral artery (MCA)
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## INTRODUCTION

The management of ischemic stroke has improved greatly over the past two decades in terms of stroke treatment. Large multicentre trials and studies provide many evidences of the value of intravenous thrombolysis with rtPA, anticoagulants, antiplatelet agents, and so on. There is strong evidence that outcomes after stroke can be improved and that death or disability from stroke can be reduced with appropriate treatment (Adams *et al*., 2003; Hack *et al*., 2003).

Despite these advances, there is still a subset of patients who deteriorate rapidly after hospital admission for cerebral infarction, with a mortality approaching 80% when treated conservatively (Berrouschot *et al*., 1998; Heinsius *et al*., 1998; Wijdicks and Diringer, 1998). This occurs in 10% to 15% of supratentorial infarction cases (Hacke *et al*., 1996) and is involved in the entire middle cerebral artery (MCA) territory. This "malignant" MCA infarction suffers from progressive clinical deterioration because of increasing brain swelling, raised intracranial pressure (ICP), and brain herniation. This patient subpopulation constitutes a particularly difficult challenge for those charged with their cases. Considering the high mortality yielded by conservative treatment, therapy of malignant MCA infarction should be more aggressive.

‡Corresponding author
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Decompressive craniectomy was first described by Kocher for the treatment of post traumatic brain edema (Bayir *et al*., 2003; Mussack *et al*., 2003; Ziai *et al*., 2003). The rationale for decompressive surgery is to reduce ICP and the vicious circle of extensive edema and further infarction. But the role of surgical decompression in malignant MCA infarction is still controversial and needs to be clearly defined further (Adams *et al*., 2003; Hack *et al*., 2003). We report a series of patients with malignant MCA infarction who underwent decompressive craniectomy because they showed signs of transtentorial herniation despite appropriate medical therapy.

## PATIENTS AND METHODS

### Patient selection

From January 1996 to March 2004, 24 patients with malignant MCA infarction were treated in our hospital. The criteria of malignant MCA infarction was as following: (1) infarction of more than 50% of the MCA territory as defined by computed tomography (CT) and/or magnetic resonance imaging (MRI) with an acute onset of corresponding clinical signs and symptoms; (2) neuroradiological evidence of local brain swelling such as midline shift of 5 mm or more indicating space occupying edema. We excluded patients with small size infarctions, as they usually were treated successfully by conservative methods. All these 24 patients were first treated using standardized conservative management included controlling good blood pressure, hyperosmolar treatment, hypertonic saline solution, hyperventilation and so on (Steiner *et al*., 2001). Decompressive craniectomy was used as second line treatment in case of significant neurological deterioration and informed consent obtained from the patients' relatives.

Twenty-four patients in this study were divided into 2 groups. Group A ($n$=10) included patients who received decompressive craniectomy. Group B ($n$=14) underwent medical control. Patients whose relatives did not consent to surgery were also enrolled in Group B.

### Operative technique

Decompressive craniectomy was done by removing parts of the frontal, parietal, temporal, and occipital squama, resulting in a large bone flap (diameter>12 cm) (Schwab *et al*., 1998). A cruciate durotomy was then opened centered on the flap. In order to protect major vessels cross the dural margins from compression, we used absorbable gelatin with biomedical mucilage on its bone surface to create a "vascular tunnel". And this may provide free circulation to the herniated brain substance (Fig.1). Dural patch or homologous temporal fascia was placed into the incision for volume-enlarged dural repair. Cranioplasty was done about three months after discharge.

### Patient evaluation

Clinical status was rated on admission and at operation or surgical decision time using the National Institute of Health Stroke Scale (NIHSS) which was repeated one week after surgery. Conscious levels of two groups were checked one week after surgery. Month mortality was defined as patient death 30 d after onset. Patients were assed three months after surgery and the outcome was quantified on Barthel Index (BI) and the modified Rankin scale (RS).

### Statistical analysis

SPSS 11.5 for Windows package was used for statistical analysis. All values are expressed as means±*SD*. Paired *T* test was applied to analysis the difference of NIHSS change perioperation in Group A. Mann-Whitney U non-parametric tests and *T* tests were used to compare means between groups. Rates were compared by Fisher's exact test. Statistical significance was assigned to $P$ value of $<0.05$.

## RESULTS

Ten patients with malignant MCA infarction underwent decompressive craniectomy (for summarized data, Table 1); fourteen medically treated patients' characteristics are summarized in Table 2. There were no statistical differences between age, sex and initial NIHSS among patients in the two groups. One patient with decompressive craniectomy is described in Fig.1.

The age of the patients on operate was 58.7±17.3 years. Four patients had ischemic infarction restricted to the MCA territory; others had additional ACA

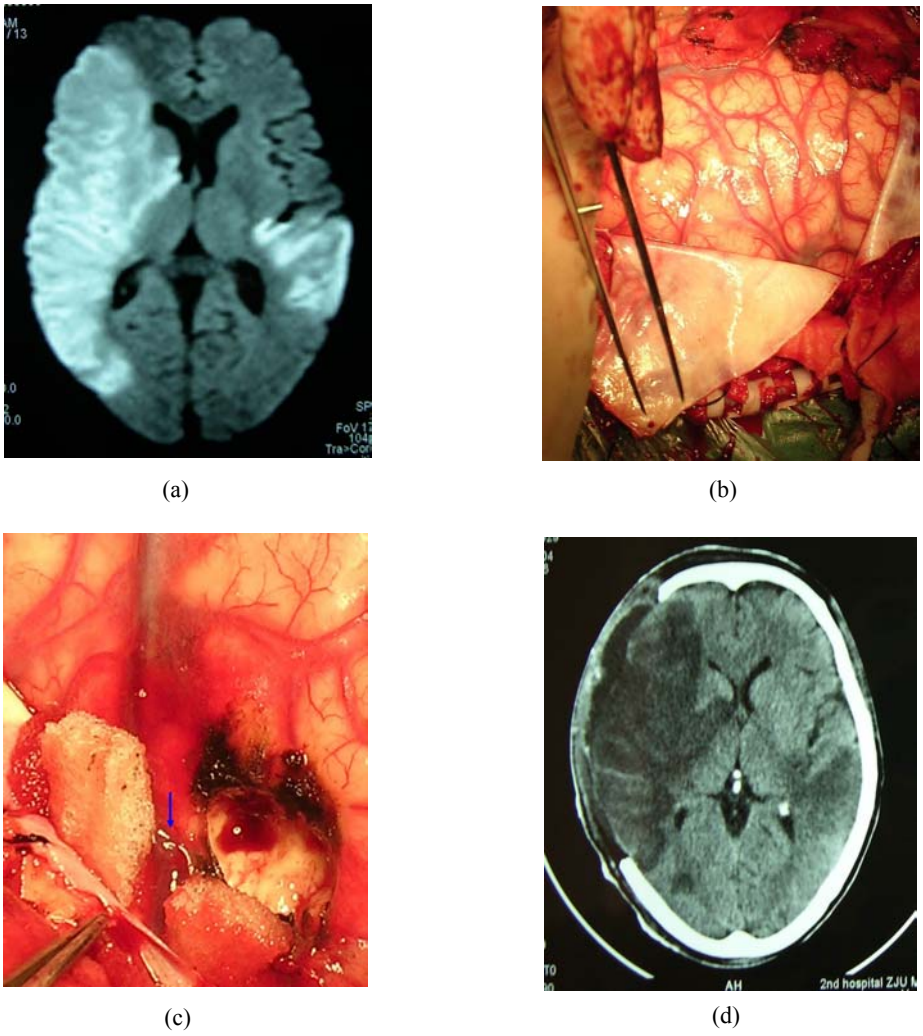

**Fig.1 CT and operative images of brain of one patient with sudden onset of left hemiplegia and coma. NIHSS at operation was 27 (a) The diffusion weighted imaging (DWI) showed a large hyperintensity area at right MCA and ACA territory; (b) During decompressive craniectomy, after a star-shaped durotomy, the infracted tissue bulged out; (c) Intraoperative picture of vascular tunnel (arrow) around the vein at dural margin, also shows excision of necrosis tissue in the base of temporal lobe; (d) Postoperative CT image of large MCA and ACA infarction**

(anterior cerebral artery) or PCA (posterior cerebral artery) infarction. The average interval between infarction and surgery was 62.1±37.0 h (range 31~140 h). NIHSS at operation was 26.0±3.3 (range 20~29) and 15.4±4.8 (9~25) at one week after surgery ($P$<0.001). One patient died after 111 h. She had delayed surgery because she was sent to our hospital late and her family members refused surgical treatment at first time. She died of heart failure at 8 d after surgery. The age of the 14 medically treated patients was 65.9±7.5 years. Nine of these patients (64.2%) died of transtentorial herniation in the first days after infarction.

The results for two groups are shown in Table 3. There were no statistical differences in NIHSS between the two groups during admission and surgical decision time ($P$>0.05). The mortality in Group A was 10.0% (1/10), while that in Group B was 64.2% (9/14) ($P$<0.001). The percent of patients able to obey commands at one week after surgery (or surgical decision) was 70.0% in Group A and 0% in Group B ($P$<0.001). At 3 months of onset, there were significant differences between Group A and Group B in both BI and RS ($P$<0.05).

**Table 1 Characteristics of patients in Group A**

| Patient No. | Sex/ age | Location of infarction | Infarction reason | *NIHSS* | | | Hours between onset and operation | Follow up | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | At admission | At operation* | After surgery | | Time | BI | RS |
| 1 | F/19 | L.MCA | Takayasu arteritis | 13 | 20 | 11 | 140 | 3 months | 90 | 1 |
| 2 | M/46 | R.MCA | Thrombosis | 16 | 28 | 9 | 54 | 3 months | 85 | 2 |
| 3 | M/50 | R.MCA+ACA | Thrombosis | 17 | 27 | 15 | 40 | 3 months | 75 | 3 |
| 4 | F/56 | L.MCA+ACA | Thrombosis | 17 | 27 | 15 | 71 | 3 months | 40 | 4 |
| 5 | M/59 | R.MCA | Embolism | 17 | 22 | 10 | 34 | 3 months | 70 | 3 |
| 6 | F/65 | R.MCA+ACA | Thrombosis | 15 | 29 | 25 | 111 | ↓8 d | NA | NA |
| 7 | F/70 | L.MCA | Embolism | 13 | 22 | 19 | 32 | 3 months | 40 | 4 |
| 8 | M/73 | R.MCA+ACA | Embolism | 19 | 29 | 16 | 39 | 3 months | 40 | 4 |
| 9 | F/74 | R.MCA+PCA | Embolism | 17 | 28 | 15 | 31 | 3 months | 40 | 4 |
| 10 | M/75 | R.MCA+PCA | Embolism | 17 | 28 | 19 | 69 | 3 months | 0 | 5 |

Note: L.MCA: left MCA; R.MCA: right MCA; NA: Not applicable; ↓ Patient died after *x* days; * $P<0.001$, between NIHSS at operation and NIHSS a week after surgery, paired *T* test

**Table 2 Characteristics of patients in Group B**

| Patient No. | Sex/age | Location of infarction | Infarction reason | NIHSS | | Follow up | | |
|---|---|---|---|---|---|---|---|---|
| | | | | At admission | At surgical decision time | Time | BI | RS |
| 1 | M/54 | R.MCA | Embolism | 10 | 24 | 3 months | 0 | 5 |
| 2 | M/54 | L.MCA+ACA | Thrombosis | 16 | 29 | ↓5 d | NA | NA |
| 3 | M/55 | L.MCA+ACA | Embolism | 17 | 27 | 3 months | 40 | 4 |
| 4 | F/61 | R.MCA+ACA | Embolism | 19 | 27 | ↓ 3 d | NA | NA |
| 5 | M/63 | L.MCA | Thrombosis | 18 | 28 | ↓ 3 d | NA | NA |
| 6 | F/64 | L.MCA+ACA | Thrombosis | 19 | 29 | ↓10d | NA | NA |
| 7 | M/68 | R.MCA+ACA | Thrombosis | 15 | 26 | 3 months | 0 | 5 |
| 8 | M/69 | R.MCA+PCA | Embolism | 17 | 29 | 3 months | 0 | 5 |
| 9 | M/70 | L.MCA | Embolism | 17 | 28 | ↓21 d | NA | NA |
| 10 | M/71 | R.MCA+ ACA | Thrombosis | 18 | 27 | ↓3 d | NA | NA |
| 11 | F/71 | R.MCA | Embolism | 13 | 27 | 3 months | 40 | 4 |
| 12 | M/74 | L.MCA | Embolism | 17 | 29 | ↓3 d | NA | NA |
| 13 | M/74 | R.MCA | Thrombosis | 18 | 28 | ↓9 d | NA | NA |
| 14 | F/75 | R.MCA+ACA | Thrombosis | 17 | 29 | ↓2 d | NA | NA |

Note: L.MCA: left MCA; R.MCA: right MCA; NA: Not applicable; ↓ Patient died after *x* days

**Table 3 Results of malignant MCA infarction in two groups**

| | Group A | Group B |
|---|---|---|
| Mortality | 10.0%* | 64.2% |
| Consciousness recovery | 70.0%** | 0% |
| NIHSS at admission | $16.1\pm1.9^{\Delta}$ (13~19) | 16.5±2.4 (10~19) |
| NIHSS at a week after surgery | $26.0\pm3.3^{\Delta\Delta}$ (20~29) | 27.6±1.4 (24~29) |
| BI | $53.3\pm28.8^{a}$ (0~90) | 16.0±21.9 (0~40) |
| RS | $3.3\pm1.2^{b}$ (1~5) | 4.6±0.5 (4~5) |

Note: *$P<0.01$, Group A versus Group B, Fisher's exact test; **$P<0.001$, Group A versus Group B, Fisher's exact test; $^{\Delta}P>0.05$, Group A versus Group B, *T* test; $^{\Delta\Delta}P>0.05$, Group A versus Group B, *T* test; $^{a}P<0.05$, between Group A and Group B, Mann-Whitney U non-parametric test; $^{b}P<0.05$, between Group A and Group B, Mann-Whitney U non-parametric test

## DISCUSSION

Decompressive craniectomy with durotomy is usually performed as a last resort in patients with malignant brain edema because of infarction or trauma (Mitchell *et al*., 2004; Messing-Junger *et al*., 2003; Schwab *et al*., 1998). However, in the setting of cerebral ischemia, the indications are still controversial, although it is clear that with even the best conservative therapy, the mortality rates are approximately 80% (Berrouschot *et al*., 1998; Heinsius *et al*., 1998; Wijdicks and Diringer, 1998). A more aggressive therapeutic method is therefore necessary to reduce mortality in these patients. Even though decompressive craniectomy for supratentorial infarction has been done in a few patients sporadically over four decades, it is only in the last two decades that the treatment has been studied systematically (Uhl *et al*., 2004; Morley *et al*., 2002; Steiner *et al*., 2001; Schwab *et al*., 1998). A recent review, however, revealed no evidence from the extant literature of solid basis for the use of this approach (Morley *et al*., 2002).

Our study, support previous findings that suggest mortality, may be better after surgery. In Group A, only one patient died (mortality 10.0%); 8 d after operation because of failure of heart function, not because of herniation. The mortality rates in previous reports varied from 0%~35% (Schwab *et al*., 1998; Rieke *et al*., 1995; Kalia and Yonas, 1993). In Group A, NIHSS significantly decreased with exciting consciousness recovery rate (70.0%). Patients who received decompressive craniectomy had mean BI of 53.3 and RS of 3.3. Our results accorded with data published by Pranesh *et al*.(2003) and Schwab *et al*.(1998). It was often thought that decompressive craniectomy must be offered only to patients with non-dominant hemispherical strokes because the speech function is unaffected and outcome would be better (Carter *et al*., 1997). In Group A, we found speech function improved most in patients with dominant hemispherical infarction. This is consistent with Schwab *et al*.(1998)'s report.

Decompressive surgery can reduce ICP, secondary ischemia, edema and can also significantly decrease infarction size. However, this procedure can allow bulge of brain tissue through the defect. This frequently results in compression of vessels by the dural margin with further congestion, edema, ischemia and hemorrhage in the bulged brain tissue. The method of creating a vascular tunnel was first reported by Csokay (2001) in traumatic brain swelling, here we newly applied this technique in malignant MCA infarction to help to prevent vascular compression and ischemia of brain bulging through the defect. Volume-enlarged dural repair can help to prevent cortex conglutination and possible shift of the brain after surgery.

We also found in Group A that patients more than 60 years had relative bad RS (4 patients' RS≥4 out of 5 patients). But "younger" patients achieved good RS (one patient's RS>3 among 5 patients). Therefore, the effectiveness of decompressive craniectomy in the elderly subpopulation remain questionable. Moreover, relatively small scale cases should be regarded cautiously. Randomized controlled trials are needed for carrying out proper evaluation of its role in the management of patients with malignant MCA infarction.

## CONCLUSION

Decompressive craniectomy confers clear survival benefit to patients presenting neurological deteriorated malignant MCA infarction and secondary edema. Current evidence indicates that majority of survivors have favorable outcome including speech function.

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