

Herniation despite Decompressive Hemicraniectomy in Large Hemispherical Ischemic Strokes

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Background: Despite decompressive hemicraniectomy (DHC), progressive herniation resulting in death has been reported following middle cerebral artery (MCA) strokes. We aimed to determine the surgical parameters measured on brain computed tomography (CT) scan that are associated with progressive herniation despite DHC in large MCA strokes. **Methods:** Retrospective chart review of medical records of patients with malignant hemispheric infarction who underwent DHC for cerebral edema was performed. Infarct volume was calculated on CT scans obtained within 24 hours of ictus. Radiological parameters of craniectomy bone flap size, brain volume protruding out of the skull, adequate centering of the craniectomy over the stroke bed, and the infarct volume outside the craniectomy bed (volume not centered [VNC]) were measured on the postoperative brain CT. **Results:** Of 41 patients who underwent DHC, 7 had progressive herniation leading to death. Radiographic parameters significantly associated with progressive herniation included insufficient centering of craniectomy bed on the stroke bed ($P = .03$), VNC ($P = .011$), additional anterior cerebral artery infarction ($P = .047$), and smaller craniectomy length ($P = .05$). Multivariate logistic regression analysis for progressive herniation using craniectomy length and VNC as independent variables demonstrated that a higher VNC was significantly associated with progressive herniation despite surgery ($P = .029$). **Conclusions:** In large MCA strokes, identification of large infarct volume outside the craniectomy bed was associated with progressive herniation despite surgery. These results will need to be verified in larger prospective studies. **Key Words:** Middle cerebral artery—ischemic stroke—herniation—decompressive hemicraniectomy—craniectomy. © 2017 National Stroke Association. Published by Elsevier Inc. All rights reserved.

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Received June 14, 2017; revision received August 4, 2017; accepted September 14, 2017.

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1052-3057/\$ - see front matter

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<https://doi.org/10.1016/j.jstrokecerebrovasdis.2017.09.016>

Introduction

Despite aggressive medical treatment, the prognosis of malignant middle cerebral artery (MCA) infarction is poor secondary to progressive cerebral herniation leading to death in approximately 80% of the cases.^{1,2} Decompressive hemicraniectomy (DHC) undertaken within 48 hours has demonstrated reduction in mortality and improvement in functional outcomes among patients with malignant MCA strokes.³ Timely decompressive surgery for malignant MCA stroke has demonstrated a marked reduction in mortality to approximately 30%.³⁻⁵

Despite DHC, lack of clinical improvement secondary to progressive herniation necessitating a second decompressive procedure and mortality has been reported.⁶⁻¹¹ Common causes of mortality in patients that underwent

DHC include various neurological factors (e.g., herniation, new infarctions, and hemorrhagic complications), non-neurological factors (e.g., pulmonary embolism, pneumonia, sepsis, and myocardial infarction), and withdrawal of care.^{3,9,12,13} Transtentorial herniation is the predominant cause of early mortality occurring within 0-14 days of onset of symptoms.^{10,12} Certain factors that have been associated with lack of clinical improvement and mortality following DHC included nonreactive pupil before surgery, large volume infarction, and lack of improvement in midline shift after surgical decompression.^{6,9,14} Recently, a more aggressive extended DH has demonstrated marked reduction in mortality when compared with the traditional DHC in patients with large hemispherical MCA strokes.¹⁵⁻¹⁷ Despite these reports, there is limited information specific to various mechanisms that lead to progressive herniation in patients with DHC.

The primary objective of this study was to explore various radiographic parameters measured on head computed tomography (CT) scan that are associated with progressive herniation and fatality from cerebral edema despite DHC in patients with large MCA strokes. We hypothesized that factors besides craniectomy length play a contributing role in progressive herniation in patients with MCA strokes that underwent DHC including the stroke volume outside the craniectomy bed (volume not centered [VNC]).

Materials and Methods

Patient Selection

Institutional Review Board at University of Arkansas for Medical Sciences approved the conduct of this study. A retrospective review of the medical records and imaging studies of all patients admitted to our tertiary care hospital with large MCA ischemic stroke who underwent a DHC for cerebral edema from 2010 to 2015 was performed. The inclusion criteria of this study were (1) large hemispheric strokes (>50% of MCA territory) as defined on head CT or magnetic resonance imaging from involvement of the MCA or internal cerebral artery (ICA) involvement or both; and (2) with or without signs of ipsilateral anterior cerebral artery (ACA) or posterior cerebral artery (PCA) infarction. Exclusion criteria were (1) strokes secondary to head trauma and associated arterial dissection, subarachnoid hemorrhage with vasospasm; (2) DHC for symptomatic hemorrhagic transformation of ischemic stroke (parenchymal hemorrhage grade 2); and (3) neurological decline from significant postoperative hemorrhage or infarction of the contralateral hemisphere.

All patients with large hemispheric strokes were admitted to the intensive care unit for aggressive medical care. Management of stroke and their associated comorbid conditions including blood pressure, hyperglycemia, and fever were performed according to the American Heart Association guidelines for stroke treatment.¹⁸ Endotracheal

intubation was usually performed for patients with respiratory failure or failure to protect their airways and to maintain normoxemia and normocapnea. Euvolemia was maintained using intravenous fluids in all patients. Osmotherapy, preferably using hypertonic saline over mannitol or both, was administered in patients with decreased level of consciousness, prior to performing DHC. The timing of DHC was in accordance to the American Heart Association guidelines.¹⁸ In general, DHC was performed within 48 hours of symptom onset in patients less than 60 years with two-thirds or complete MCA stroke from MCA or ICA occlusion. For patients older than 60 years or with poor premorbid conditions, DHC was performed if they had (1) progressive midline shift, and (2) decrease in level of consciousness; however, the timing for DHC was extended beyond 48 hours from symptom onset.¹⁸ The surgical technique involved removal of a large bone flap with a diameter of 12 cm that included the frontal, parietal, temporal, and parts of the occipital regions to provide decompression of the middle cranial fossa, with resection of the dura and placement of a dural patch.¹⁵

Data Collection

The following characteristics of patients were collected: age, gender, baseline medical comorbidities, presence or development of pupillary asymmetry, National Institutes of Health Stroke Scale, Glasgow Coma Score, and serum sodium levels upon presentation and prior to and after DHC. All preoperative and postoperative brain CT scan and magnetic resonance imaging when available were reviewed to determine the extent of MCA stroke, presence of additional ACA or PCA infarction, and degree of midline shift at the septum pellucidum at baseline, before and after DHC. All images were reviewed by a board-certified neuroradiologist who was blinded to the clinical outcomes.

Radiological Parameters

The following radiological parameters were collected:

1. Infarct volume was calculated using the ABC/2 volume estimation of an ellipsoid, where A is the largest diameter, B the largest perpendicular diameter, and C the vertical diameter (number of slices multiplied by thickness of each slice). These were measured on brain CT scans obtained within 24 hours of symptom onset (Fig 1, A).^{11,19}
2. The length, width, and area of the craniectomy bone flap were measured from the immediate postoperative brain CT scan. Similarly, the brain volume protruding out of the skull (height and volume) was measured. These measurements were defined as follows²⁰:
 - a) The area of bone flap was defined as $A = D \times d \times \pi$ (D is the anteroposterior diameter,

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