Neuroimaging in Deteriorating Patients With Cerebellar Infarcts and Mass Effect

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Background and Purpose—The decision to proceed with surgery in cerebellar infarct with mass effect (CIMASS) in deteriorating patients is based on clinical features. The potential role of neuroimaging in predicting deterioration has not been systematically studied. In this study we determine the role of neuroimaging in predicting deterioration in CIMASS.

Methods—We retrospectively reviewed the clinical and neuroimaging features in 90 patients with cerebellar infarcts. We selected for detailed analysis CIMASS in 35 patients.

Results—Eighteen patients remained stable and 17 deteriorated. Of these 17 patients, 8 were treated conservatively and 9 had surgery. Radiological features indicative of progression were more common in deteriorating patients compared with stable patients: fourth ventricular shift (82.3% versus 50%, \( P = 0.075, \text{OR} = 4.67 \)), hydrocephalus (76.5% versus 11.1%, \( P = 0.0001, \text{OR} = 26 \)), brain stem deformity (47% versus 5.6%, \( P = 0.0065, \text{OR} = 15.1 \)), and basal cistern compression (35.3% versus 0%, \( P = 0.0076, \text{OR} = 20.91 \)). Differences in upward displacement of the aqueduct and pontomesencephalic junction from Twining’s line, tonsillar descent on sagittal MRI, and infarct volumes between stable and deteriorating patients were not statistically significant.

Conclusions—Hydrocephalus, brain stem deformity, and basal cistern compression may herald deterioration in CIMASS. Admission to a neurological-neurosurgical intensive care unit and consideration of preemptive surgery are warranted in these patients. Vertical displacement of tonsils or aqueduct, demonstrated by MR imaging, did not predict deterioration.

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Key Words: brain edema ▪ cerebellar infarction ▪ magnetic resonance imaging ▪ outcome ▪ tomography, x-ray computed

Cerebellar infarcts may develop mass effect (CIMASS) in 10% to 25% of all cases.\(^1^-^3\) When the infarcted tissue and surrounding edema become space occupying within the posterior fossa, brain stem compression and fourth ventricular collapse occur, resulting in obstructive hydrocephalus. Previous authors\(^4^-^6\) have suggested that patients with CIMASS progress through 3 arbitrary clinical stages. The early clinical stage of CIMASS is characterized by signs from cerebellar dysfunction due to infarction, followed by a stage punctuated by brain stem compression in which the level of consciousness fluctuates, emerging into a final stage of coma. Both brain stem infarction and brain stem compression, caused by CIMASS, may have similar clinical presentation.\(^7^-^8\) This distinction remains important in the decision to proceed with occipital craniotomy.

It is not known whether correlation of the clinical stage of cerebellar infarct with neuroimaging offers any predictive value. Radiological features associated with CIMASS have been previously described; these include fourth ventricular shift or effacement, obstructive hydrocephalus, deformity of the brain stem, compression of the basal cisterns, aqueductal displacement, downward tonsillar herniation, and buckling of the quadrigeminal plate.\(^9^-^10\) There has been no previous study establishing the sequence in which these changes occur over time, nor if such a sequence, correlated with clinical findings, would aid in the decision to proceed with neurosurgical intervention. Furthermore, the role of MR imaging in predicting deterioration, judged by the extent of horizontal and vertical tissue displacement,\(^9^-^11,12\) is not known. We present a series of patients with CIMASS, propose the sequence of radiological features indicative of progression, and quantify the extent of vertical brain displacement.

Subjects and Methods

Subjects
The medical records, investigations, management, and outcomes of patients who presented with acute cerebellar infarct from 1990 to April 1999 were reviewed. The patients had been diagnosed clinically and the diagnoses confirmed by CT, MRI, or both. Patients with indeterminate onset of symptoms (\(n = 37\)) and those who were...
originally admitted elsewhere for treatment and referred for rehabilitation only (n=30) were excluded, as were patients with prior cerebellar infarcts (n=40). Patients with clinical or radiological evidence of acute brain stem infarction (n=38) or concurrent cerebral infarction (n=38) were also excluded. In patients with previous strokes, the original territory of these strokes, together with their extent of recovery and baseline level of function, were noted. In total, 90 patients with cerebellar infarction were selected by using the above criteria. The clinical stage of CIMASS and the Glasgow Coma Score (GCS) at presentation and during the hospital stay were recorded. Deterioration was determined if there was decrease of 2 points in the GCS. This was attributed to the mass effect of the cerebellar infarct if there was radiological evidence of mass effect and in the absence of brain stem infarct. Patients with a GCS of <12 on admission were considered to have deteriorated. The progress of all patients was carefully abstracted from the records. The postoperative courses of all patients were recorded until their discharge from the hospital. The modified Rankin Scale score was used to assess each patient at discharge and at follow-up. A good outcome at discharge was defined as having a modified Rankin score of 0 to 2, a moderate outcome as having a modified Rankin score of 3 to 4, a poor outcome as having a score of 5, and death as a score of 6.

### Neuroimaging

The CT imaging parameters were 10-mm contiguous slice thickness with additional contiguous fine cut of 3 to 5 mm through the posterior fossa. The field of view was 25 cm and the matrix 512. Patients underwent MR imaging on a 1.5-T scanner. The imaging parameters were 5-mm section thickness and 0- to 2.5-mm interslice gap. 256×192 matrix, 20-cm field of view for conventional spin-echo images, axial and sagittal T1-weighted (repetition time [TR] 500 ms/excitation time [TE] 20 ms), axial T2-weighted (TR 2200/TE 80), and proton density (TR 2200/TE 30).

The location and vascular territory of each infarct was determined by comparing the hard copy films with illustrations from the anatomic map of the brain stem and cerebellum. This was done by the authors, blinded to the clinical data, and the original radiological reports. All cases were then classified into 1 of 2 groups, CIMASS or non-CIMASS. Criteria for the diagnosis of CIMASS on CT and MRI were deformity or displacement of the fourth ventricle, obstructive hydrocephalus as evidenced by progressive dilatation of the lateral ventricles (with sulcal effacement of the frontal lobes and parieto-occipital lobes) and third ventricles on consecutive scans, effacement of the basal cisterns, and deformity or anterior bowing of the brain stem. Patients were diagnosed with CIMASS if 1 or more of the above criteria were met. When available, the remote effect of the CIMASS was further quantified by measuring vertical brain shift in midsagittal MRI planes.

### Technical Measurements

The midsagittal MRI plane includes the corpus callosum, the straight sinus, cerebral aqueduct, and internal occipital protuberance. Within this plane, 3 MRI reference lines may be found: the Twining’s line, the foramen magnum line, and the incisural line. Twining’s line (T) extends from the anterior tuberculum sellae to the internal occipital protuberance (Figure 1). The foramen magnum line is drawn from the inferior tip of the clivus to the posterior lip of the foramen magnum. The incisural line extends from the anterior tuberculum sellae to the confluence of the straight sinus, the great cerebral vein of Galen, and the inferior sagittal sinus; the iter is normally contained within this plane. The perpendicular distance of the pontomesencephalic junction and aqueduct opening to T (T-PMJ and T-A, respectively) were measured to assess rostral displacement of the brain stem, as previously described. The perpendicular distance of the caudal poles of the cerebellar tonsils with respect to their positions above or below the foramen magnum line (F-ton) was measured as previously described; positions below the reference line were recorded as negative values. The distance of the iter to the incisural line (iter-I) was also measured. All distances were converted to true dimensions by using the scales accompanying the images. The vertical measurements on the mid-sagittal MRI were compared between the CIMASS and non-CIMASS group. A control group consisting of patients with no intracranial mass or stroke was selected at random and matched with the patients in the CIMASS group for sex and age.

### Infarct Volumes

For calculating infarct volumes, a cursor was used to trace around the image of the infarct on each MR or CT slice as viewed on the computer screen, to obtain the area on each slice. This was multiplied with the slice thickness to calculate the volume for that level; the individual volumes of the slices and interslice gaps were then added to obtain an accurate infarct volume. Using these parameters, the volume of the infarct and the extent of vertical brain shift in the following groups were compared: age and sex-matched controls, patients with cerebellar infarcts without mass effect, patients with CIMASS who remained stable (group 1), and patients with CIMASS who deteriorated (group 2). Group 2 was further divided into 2 subgroups, patients who were treated conservatively (group 2A) and those who were treated surgically (group 2B). Controls were selected only for patients who had MRI to assess vertical shift.

### Statistical Analysis

Categorial data, such as the presence or absence of hydrocephalus, were analyzed with Fisher’s exact test for statistical significance. Continuous variables, such as the distance of the iter from the incisural line, were compared between the groups with Wilcoxon’s rank sum test.

### Mechanism of Infarction

The stroke mechanisms were categorized by using the results of cerebral angiography, transthoracic and transesophageal echocardiography, transcranial Doppler examination, and MR angiography. The TOAST classification was used to classify the mechanism of infarction.

### Results

**Patient Characteristics**

We studied 90 patients with isolated cerebellar infarct. There was no difference in the risk factors or clinical features at presentation between those who subsequently developed CIMASS and those who did not. Thirteen had cerebral
angiography, 19 had transthoracic echocardiography, 49 had transesophageal echocardiography, 12 had transcranial Doppler examination, and 35 had MR angiography. Of the 90 patients with cerebellar infarct, there were 35 patients (39%) who, on the basis of radiological criteria, had CIMASS. The brain stem signs associated with brain stem compression were: gaze palsy, cranial neuropathy and pupillary asymmetry, hemiplegia and quadriplegia, and decorticate and decerebrate posturing. The presence of brain stem signs (from brain stem compression) at presentation was associated with a higher risk of subsequent deterioration ($P<0.001$).

### Neuroimaging Findings

The radiological features are summarized in the Table and Figures 3 and 4. Twenty-one of 90 patients (23%) had a normal CT initially. In the CIMASS group, 9 of 35 patients (26%) had normal CT initially. The mean and median number of days that elapsed from ischemic stroke onset to the first radiological examination, and 35 had MR angiography. Of the 90 patients, 18 of 35 patients (26%) had normal CT initially. In the CIMASS group, 9 of 35 patients (26%) had normal CT initially. Non-CIMASS, n=55

<table>
<thead>
<tr>
<th>Group</th>
<th>Total CTs Reviewed, n</th>
<th>Total MRIs Reviewed, n</th>
<th>Deformity of Fourth Ventricle, n</th>
<th>Shift of Fourth Ventricle From Midline, n</th>
<th>Hydrocephalus, n</th>
<th>Anterior Displacement or Deformity of Brain Stem, n</th>
<th>Compression of Basal Cisterns, n</th>
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<tbody>
<tr>
<td>Non-CIMASS, n=55</td>
<td>42</td>
<td>45</td>
<td>0</td>
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<tr>
<td>(CIMASS with no deterioration, n=18)</td>
<td>24</td>
<td>12</td>
<td>18</td>
<td>9</td>
<td>2</td>
<td>1</td>
<td>0</td>
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<td>Group 2A</td>
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<tr>
<td>(CIMASS with deterioration, conservative, n=8)</td>
<td>12</td>
<td>5</td>
<td>8</td>
<td>7</td>
<td>4</td>
<td>1</td>
<td>1</td>
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<tr>
<td>Group 2B</td>
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<tr>
<td>(CIMASS with deterioration and surgery, n=9)</td>
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<td>8</td>
<td>9</td>
<td>7</td>
<td>9</td>
<td>7</td>
<td>5</td>
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<tr>
<td>Groups 2A and 2B</td>
<td>48</td>
<td>13</td>
<td>17</td>
<td>14</td>
<td>13</td>
<td>8</td>
<td>6</td>
</tr>
</tbody>
</table>

Radiological Features of 90 Patients With Cerebellar Infarcts, by Group

with no deterioration and 8 of 17 patients with deterioration. The sensitivity and specificity of brain stem deformity for predicting clinical deterioration were 47% and 94%, respectively ($P=0.007, OR=15.1 [2.26 to 303.93]$). Basal cistern compression was not present in any patient with no deterioration and was present in 6 of 17 patients with deterioration. The sensitivity and specificity of basal cistern for predicting clinical deterioration were 35% and 100%, respectively ($P=0.008, OR=20.9 [1.98 to 220.61]$).

### Vertical Brain Shift Parameters

The median perpendicular distance from the most inferior portion of the tonsil to the foramen magnum line (F-ton) for the control group was 1.4 mm (range −5.7 to 8.6 mm); non-CIMASS patients, 1.4 mm (range −10.0 to 11.4 mm); group 1, −0.7 mm (range −8.6 to 5.7 mm); group 2A, −2 mm (range −4.3 to 0 mm); group 2B, −12.3 mm (range −17.1 to 0 mm); and combined groups 2A and 2B, −9.0 mm (range −17.1 to 0 mm). The median perpendicular distance from the iter of the aqueduct to the incisural line (Iter-I) for the control group was 0 mm (range −5.7 to 5.7 mm); non-CIMASS patients, 0 mm (range −2.8 to 5.7 mm); group 1, 2.9 mm (range −2.9 to 5.9 mm); group 2A, 2.7 mm (range 0 to 5 mm); group 2B, 2.9 mm (range 0 to 8.6 mm); and combined groups 2A and 2B, 2.9 mm (range 0 to 8.6 mm). The median perpendicular distance from the iter of the aqueduct to Twining’s line (T-A) for the control group was 15.7 mm (range 11.4 to 20.0 mm); non-CIMASS patients, 17.1 mm (range 11.4 to 24.4 mm); group 1, 17.1 mm (range 11.7 to 22.9 mm); group 2A, 18.5 mm (range 16.0 to 20.0 mm); group 2B, 18.6 mm (range 10.0 to 22.9 mm); and combined groups 2A and 2B, 18.6 mm (range 10.0 to 22.9 mm). The median perpendicular distance from the pontomesencephalic junction to Twining’s line (T-PMJ) for the control group was 5.4 mm (range 1.4 to 8.6 mm); non-CIMASS patients, 5.7 mm (range 0 to 8.6 mm); group 1, 6.5 mm (range 5.7 to 9.1 mm); group 2A, 8.0 mm (range 7.0 to 8.6 mm); group 2B, 7.1 mm (range 0 to 12.9 mm); and combined groups 2A and 2B, 7.3 mm (range 0 to 12.9 mm). There was no statistically significant difference in the F-ton,
iter-I, T-A, or T-PMJ values between the control group and the non-CIMASS group. A larger extent of tonsillar descent was observed in CIMASS compared with non-CIMASS ($P<0.001$). Larger iter-I, T-A, and T-PMJ values were demonstrated in CIMASS compared with non-CIMASS ($P<0.05$, $P<0.05$, and $P<0.001$, respectively). Within the CIMASS group, there was no statistically significant difference in F-ton, iter-I, T-A, or T-PMJ values between those who deteriorated and those who remained stable.

Infarct Volumes, Territories and Mechanisms

The median infarct volumes were group 1, 24.2 cm$^3$ (range 6.6 to 49.0 cm$^3$); group 2A 23.4 cm$^3$ (range 17.5 to 37.4 cm$^3$); group 2B 42.0 cm$^3$ (range 15 to 55.7 cm$^3$); combined groups 2A and 2B 33.0 cm$^3$ (range 15 to 55.7 cm$^3$). Larger infarcts were demonstrated in CIMASS compared with non-CIMASS ($P<0.001$). There was no statistically significant difference in infarct volumes between groups 1 and 2 (Wilcoxon rank sum test).

The arterial distributions of patients with CIMASS were posterior inferior cerebellar artery (PICA), 21 patients (60%); full PICA territory and contralateral medial PICA branch, 1 patient (3%); bilateral medial branch of PICA, 3 patients

**Figure 3.** Axial CT scan showing shift and compression of the fourth with dilatation of the third ventricle and both temporal horns.

**Figure 4.** Midsagittal T1-weighted MR in a patient with cerebellar infarct and mass effect showing vertical brain shift.
(9%); superior cerebellar artery (SCA), 7 patients (20%); bilateral SCA infarcts, 2 patients (6%); and medial branch of SCA, 1 patient (3%). Of the 22 patients with full PICA territory infarction, 1 had infarction of the contralateral medial branch of PICA. Of the 9 patients with full SCA territory infarction, 1 had infarction of the contralateral medial branch of the SCA and another had contralateral full PICA territory infarction. There was no difference in the distribution of infarct territories between the CIMASS patients who deteriorated and those who did not.

The mechanisms of infarction in the CIMASS group were large-artery disease (13 patients), cardioembolism (12 patients), ischemic stroke of undetermined etiology (2 patients), and incomplete workup (8 patients). There were 3 patients who had vertebral artery dissection; they were included under the large-artery disease group. There were no cases of small-vessel occlusion among the CIMASS patients. Twenty-seven of 90 patients had incomplete workup as defined by TOAST criteria. 3 patients had 2 or more possible causes identified, and 1 patient had a negative evaluation.

Outcome
The clinical progress and outcome at discharge are summarized in Figure 2. The mean number of days that elapsed from the first onset of cerebellar stroke symptoms to the first onset of brain stem signs was 2 days (range 1 to 6 days). All patients who deteriorated were treated with mannitol, steroids, and/or hyperventilation. The 90 patients in this series were followed up for a median of 16 months (range 1 to 105 months). Most patients improved at follow-up. Seven of the patients without CIMASS (2 patients in group 1, 4 patients in group 2, and 3 patients in group 3) died of unrelated causes.

Discussion
In this study, downward displacement of tonsils or rostral displacement of the aqueduct/pontomesencephalic junction on midsagittal MRI did not predict deterioration. Mass effect from swelling of a cerebellar infarct occurred more commonly than previously reported, but clinical deterioration occurred in less than half of the patients. Clinical deterioration coincided with the development of hydrocephalus, brain stem deformity, and basal cistern compression.

Patients with CIMASS often have full territorial PICA or SCA infarcts, or the infarcts are confined to the medial vermis hemispheric branches of the PICA or SCA. Isolated lateral hemispheric branches of the PICA or SCA and any territorial infarct confined traditionally to AICA were not identified with CIMASS. Full territorial infarcts have been associated more frequently with the development of mass effect than small infarcts. Between the groups of CIMASS that deteriorated and the group that did not, there was no statistically significant difference in the distribution of the territories of the infarcts. These findings agree with the results of a recently published study.

Earlier definition of the involved territory would allow stratification of patients into those at risk and those at less risk, although the presence of an infarct in one of these territories does not always equate with deterioration. MR imaging with fluid-attenuated inversion recovery or diffusion-weighted imaging would improve early detection, but this technology was not applied uniformly through the study time frame and was therefore not included. Although other studies have not found a similar relationship, our study found that the presence of hemorrhagic infarct was associated with deterioration. This may be a reflection of a larger volume of infarction.

Of the CIMASS patients who deteriorated, obstructive hydrocephalus was linked to radiographic features of brain stem deformity and basal cistern compression. This observation can be explained by the cerebellar infarct exerting its mass effect in a posterior-to-anterior plane, leading to obliteration of the fourth ventricle and anterior displacement of the brain stem against the clivus. This is responsible for the dilatation of the third ventricle, temporal horns, and lateral ventricles. Based on the frequency of radiological signs found in patients with CIMASS, we propose that the order of progression of radiological signs was fourth ventricular deformity, fourth ventricular shift, obstructive hydrocephalus, brain stem deformity, and basal cistern compression.

Ropper reported that the vertical displacement of the PMJ on coronal films for patients with acute supratentorial masses did not differ from measurements in patients without masses. Our findings indicate statistically significant rostral displacement of the PMJ, aqueduct, and tonsillar descent in patients with CIMASS compared with non-CIMASS patients and controls. However, it was not possible to demonstrate a statistically significant difference between CIMASS patients who deteriorated and those who remained stable. These findings suggest that vertical brain displacement in CIMASS occurred, but they are not predictors of clinical deterioration. In addition, they support the hypothesis that the presence of hydrocephalus, brain stem compression, or both, but not vertical brain displacement, constitutes the main reason for neurological deterioration in patients with CIMASS. These findings are novel and should be taken into account when contemplating craniotomy.

Development of hydrocephalus in the setting of impaired alertness coincided with deterioration in CIMASS. Admission to a neurological intensive care unit and consideration of preemptive surgery may be warranted in these patients. In patients with CIMASS, the presence of vertical displacement may not predict clinical deterioration, and decision to proceed with surgery should not be based solely on this MR finding. Our results should be interpreted with caution, because the number of patients with CIMASS in our study was small. Further MR studies are needed to extend these observations.

References


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