Plasma α-Melanocyte Stimulating Hormone Predicts Outcome in Ischemic Stroke

Dannielle Zierath, BS; Pat Tanzi, BSN, RN, CCRC; Kevin Cain, PhD; Dean Shibata, MD; Kyra Becker, MD

Background and Purpose—α-Melanocyte stimulating hormone (α-MSH) is an endogenously produced neuropeptide derived from the same precursor as adrenocorticotrophic hormone. α-MSH has profound immunomodulatory properties and may also be neuroprotective. Nothing is known about α-MSH and changes in its plasma concentrations in patients with acute ischemic stroke.

Methods—in this prospective observational study, plasma concentrations of α-MSH, adrenocorticotrophic hormone, cortisol, and interleukin 6 were assessed longitudinally over the course of 1 year after stroke onset in 111 patients. Logistic regression was used to evaluate the effect of initial plasma α-MSH, adrenocorticotrophic hormone, cortisol, and interleukin 6 on long-term outcome.

Results—There was an early decrease in plasma α-MSH in patients with severe stroke (National Institutes of Health Stroke Scale ≥17) that normalized over the course of the year; these same patients evidenced elevations in plasma cortisol and interleukin 6. Higher initial plasma α-MSH, but not adrenocorticotrophic hormone, cortisol, or interleukin 6, was independently predictive of good long-term outcome.

Conclusions—This research is the first to study endogenous changes in plasma α-MSH after stroke. The independent effect of early plasma α-MSH on stroke outcome, as well as a growing body of experimental data demonstrating improved stroke outcome with exogenous α-MSH administration, suggests a potential therapeutic role for α-MSH in the treatment of stroke. (Stroke. 2011;42:3415-3420.)

Key Words: α-MSH ■ stroke ■ outcome

Alpha-melanocyte stimulating hormone (α-MSH) is a 13 amino acid neuropeptide derived from proopiomelanocortin (POMC), a prohormone polypeptide expressed in the brain, the pituitary gland, and in peripheral tissues, such as the immune system and skin.1 The processing of POMC depends on a series of prohormone convertases (PCs), and the tissue in which POMC is processed determines its eventual end products. In brain, POMC is expressed in the arcuate nucleus of the hypothalamus. Cleavage by PC1 leads to the production of adrenocorticotrophic hormone (ACTH) and β-lipotropin; further processing by PC2 leads to production of smaller peptides, including α-MSH and β-endorphin.1,2 α-MSH production is, thus, more robust in tissues that highly express PC2.

As part of the acute phase/stress response in stroke, cortisol is elevated, and these elevations correlate with both stroke severity and outcome.3-5 Cortisol production depends largely on the expression of ACTH, and given the common origin of ACTH and α-MSH, we hypothesized that there might be stroke-induced alterations in α-MSH. In patients with traumatic brain injury, for instance, decreases in plasma α-MSH occur and are associated with worse outcome.6 To our knowledge, no studies have evaluated endogenous changes in α-MSH after stroke. Based on experimental data, however, a decrease similar to that seen in patients with traumatic brain injury is anticipated.7 We, thus, sought to describe the time course of changes in plasma α-MSH after ischemic stroke in relation to ACTH and cortisol. Because not all cortisol production depends on ACTH, however, we also assessed plasma interleukin (IL) 6, a cytokine that activates the hypothalamic-pituitary-adrenal axis and can directly stimulate cortisol production by the adrenal gland.8

Methods

Research Subjects
This study was part of a larger prospective study that followed immune responses over the course of the year after stroke onset.9,10 The study was approved by the institutional review board, and all of the patients or their surrogates provided informed consent. Patients with ischemic stroke admitted to either Harborview Medical Center or the University of Washington Medical Center from September 2005 through May 2009 who were ≥18 years of age could be enrolled within 72 hours of symptom onset and were felt not likely to die from their stroke were eligible. Patients with ongoing therapy for malignancy, known history of HIV or hepatitis B or C, history of brain tumor, anemia (hematocrit <35 on admission), and those taking immunomodulatory drugs were excluded. Blood was drawn...
as soon as possible after stroke onset and at 3, 7, 30, 90, 180, and 365 days after stroke onset. Plasma was frozen at −80°C until use.

Clinical Data
Demographic and clinical data were collected on all of the patients. Stroke severity was determined by the National Institutes of Health Stroke Scale score and outcome by the modified Rankin Scale. Total infarct volume on initial diffusion-weighted MRI was calculated by the ABC/2 method by a single radiologist trained in the Cardiovascular Health Study and Atherosclerosis Risk in Communities protocols for infarct scoring. Information about therapeutic interventions for the treatment of stroke and stroke-related complications, such as infection, was collected. Infection was defined as clinical symptoms of an infection (fever and/or pyuria for urinary tract infection and fever and/or productive cough and radiographic evidence of consolidation for pneumonia) and positive culture data (for both pneumonia and urinary tract infection).

Laboratory Studies
Leukocyte counts, plasma cortisol, and ACTH concentrations were determined by the clinical laboratory. Plasma α-MSH concentrations were determined using a commercially available enzyme immunoassay kit (Phoenix Pharmaceuticals, Belmont, CA). Briefly, peptides were eluted from 0.5 mL of acidified plasma using C18-SEP columns containing 200 mg of C18 (Phoenix Pharmaceuticals, Belmont CA); the samples were evaporated by centrifugal vacuum concentration and reconstituted in 125 μL of buffered saline before enzyme immunoassay. The concentration of circulating IL-6 was measured with a cytometric bead-based system (Fluorokine MAP, R&D Systems); the lower limit of detection was 1.11 pg/mL. Values below the limit of detection are referred to as not detected and assigned the lowest limit of detection for statistical testing.

Statistics
Descriptive data are presented as median and interquartile range. Group comparisons were performed using the Mann-Whitney U test or Kruskal-Wallis H test. Data were normalized and associations tested using the Pearson correlation. Logistic regression was used to estimate the odds ratio and 95% CI for the effect of the highest initial α-MSH concentration (within 72 hours of stroke onset) on neurological outcome at 1, 3, 6, and 12 months after stroke onset. Given the relatively severe strokes seen in this study, good outcome was defined as independent ambulation (modified Rankin Scale ≤3). Significance was set at P≤0.05.

Results
A total of 114 patients were enrolled in the parent study; plasma α-MSH concentrations were determined in 111 of these patients,
who are the subject of this article. The characteristics of the overall study population have been described elsewhere. For the 111 patients in whom α-MSH was assessed early (by 72 hours), the median age was 57 years (range: 44–66 years), the median National Institutes of Health Stroke Scale score was 11 (range: 4–19), the median infarct volume was 12 mL (range: 1–80 mL), and 35% of the patients were women. As in other publications related to this study population, we divided patients on the onset (Figure A). There was still a trend toward decreased least severe strokes at both 24 hours and 72 hours after stroke (Table 1). Given the significant relationship between stroke severity and α-MSH, cortisol, and IL-6, correlations corrected for stroke severity (using the NIHSS score as a continuous variable) are also presented. Data are normalized and presented as Pearson’s r.

**Table 1. Correlations Among α-MSH, ACT, Cortisol, Infarct Volume, and Stroke Severity at 24 Hours and 72 Hours After Stroke Onset**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Infarct Volume*</th>
<th>NIHSS</th>
<th>ACTH</th>
<th>Cortisol</th>
<th>IL-6</th>
</tr>
</thead>
<tbody>
<tr>
<td>α-MSH</td>
<td>24 h: −0.148, NS</td>
<td>−0.479, P=0.010</td>
<td>−0.127, NS</td>
<td>−0.501, P=0.025</td>
<td>−0.238, NS</td>
</tr>
<tr>
<td></td>
<td>72 h: −0.247, P=0.014</td>
<td>−0.241, P=0.016</td>
<td>−0.139, P=0.181</td>
<td>−0.115, NS</td>
<td>−0.209, P=0.042</td>
</tr>
<tr>
<td>Cortisol</td>
<td>24 h: 0.498, P=0.007</td>
<td>0.728, P&lt;0.001</td>
<td>0.304, P=0.132</td>
<td>0.378, P=0.100</td>
<td>0.496, P=0.014</td>
</tr>
<tr>
<td></td>
<td>72 h: 0.420, P&lt;0.001</td>
<td>0.616, P&lt;0.001</td>
<td>0.346, P&lt;0.001</td>
<td>0.535, P&lt;0.001</td>
<td>0.358, P&lt;0.001</td>
</tr>
<tr>
<td>ACTH</td>
<td>24 h: 0.069, NS</td>
<td>−0.068, NS</td>
<td>...</td>
<td>...</td>
<td>−0.124, NS</td>
</tr>
<tr>
<td></td>
<td>72 h: 0.138, P=0.161</td>
<td>0.127, P=0.193</td>
<td>...</td>
<td>...</td>
<td>0.133, P=0.197</td>
</tr>
<tr>
<td>IL-6</td>
<td>24 h: 0.517, P=0.006</td>
<td>0.700, P&lt;0.001</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>72 h: 0.393, P&lt;0.001</td>
<td>0.566, P&lt;0.001</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

*Infarct volume is not available for 3 persons.

In this study we found early and sustained elevations in both plasma cortisol (to 1 month) and IL-6 (to 6 months) among patients with severe stroke, whereas ACTH concentrations were largely unchanged and α-MSH concentrations decreased early after stroke. That elevated plasma cortisol is seen in patients with severe strokes and is associated with worse outcome is well documented. Given the significant relationship between stroke severity and plasma α-MSH concentrations on outcome was not apparent until later time points after stroke and was independent of initial stroke severity, patient age, and infection status.

**Discussion**

In this study we found early and sustained elevations in both plasma cortisol (to 1 month) and IL-6 (to 6 months) among patients with severe stroke, whereas ACTH concentrations were largely unchanged and α-MSH concentrations decreased early after stroke. That elevated plasma cortisol is seen in patients with severe strokes and is associated with worse outcome is well documented. Given the significant relationship between stroke severity and plasma α-MSH concentrations on outcome was not apparent until later time points after stroke and was independent of initial stroke severity, patient age, and infection status.
IL-6. Given that the half-life of α-MSH in circulation is on the order of minutes, it is possible that more significant associations between α-MSH and stroke severity were missed because of timing of blood draws.

Despite the limitations of this study with regard to timing of blood draws, we were still able to demonstrate a decrease in plasma α-MSH among patients with severe strokes (National Institutes of Health Stroke Scale $\geq 17$) early after stroke onset. To our knowledge, this is the first study that addresses endogenous changes in plasma α-MSH after ischemic stroke, although we did find a similar decrease in plasma α-MSH in an animal study of severe stroke. Further, we found that higher plasma α-MSH was associated with an increased likelihood of experiencing a good clinical outcome, an effect that was most apparent at later time points after stroke and independent of stroke severity, patient age, and infection status. In contrast, the associations between cortisol and IL-6 on outcome were most robust at early time points after stroke and explained almost entirely by the fact that cortisol and IL-6 are markers of stroke severity. The lack of an independent association among cortisol, IL-6, and stroke outcome has been documented previously.5,18

Both the independent association of α-MSH with stroke outcome and the delay in this observed association suggest that the effect of early plasma α-MSH on outcome is more than a reflection of the stress response related to stroke severity and that maintenance of plasma α-MSH after stroke onset may be protective. Furthermore, a growing body of experimental data shows that exogenous administration of α-MSH decreases infarct volume and improves stroke outcome.7,19–22 There are numerous mechanisms by which α-MSH (and related neuropeptides) could improve stroke outcome, and these effects are mediated through 5 different melanocortin receptors (MCRs). Potent antipyretic properties of α-MSH, which could potentially be capitalized on in the treatment of stroke, are mediated through the MCR3/MCR4

### Table 2. Differences Between Initial Plasma α-MSH Concentrations (pg/mL) Based on Clinical and Demographic Variables

<table>
<thead>
<tr>
<th>Patient Characteristics</th>
<th>Variables</th>
<th>Unadjusted $P$</th>
<th>Adjusted for Stroke Severity $P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>Yes</td>
<td>12.2 (6.1–20.5), N=39</td>
<td>11.6 (1.8–17.6), N=72</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>Yes</td>
<td>11.6 (1.9–17.9), N=100</td>
<td>13.3 (10.5–45.8), N=11</td>
</tr>
<tr>
<td>Medical history</td>
<td>Yes</td>
<td>11.6 (4.7–23.5), N=26</td>
<td>11.4 (2.0–17.0), N=85</td>
</tr>
<tr>
<td>AF</td>
<td>Yes</td>
<td>16.1 (9.9–27.9), N=16</td>
<td>11.5 (1.9–15.8), N=95</td>
</tr>
<tr>
<td>CHD</td>
<td>No</td>
<td>12.3 (4.7–23.5), N=26</td>
<td>11.4 (2.0–17.0), N=85</td>
</tr>
<tr>
<td>DM</td>
<td>No</td>
<td>13.5 (0.6–22.8), N=27</td>
<td>11.6 (4.3–15.5), N=84</td>
</tr>
<tr>
<td>HTN</td>
<td>No</td>
<td>12.5 (0.5–21.4), N=59</td>
<td>11.0 (6.1–17.1), N=52</td>
</tr>
<tr>
<td>Smoker</td>
<td>No</td>
<td>10.2 (0.6–14.3), N=43</td>
<td>12.6 (4.7–18.7), N=68</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>No</td>
<td>13.0 (0.2–22.3), N=26</td>
<td>11.6 (3.7–18.0), N=82</td>
</tr>
</tbody>
</table>

Statistics are by Mann-Whitney $U$ test or Kruskal-Wallis $H$ test and are either unadjusted or adjusted for stroke severity (using the NIHSS score as a continuous variable).

AF indicates atrial fibrillation; CHD, coronary heart disease; DM, diabetes mellitus; HTN, hypertension; TACS, total anterior circulation stroke; PACS, partial anterior circulation stroke; LACS, lacunar stroke; POCS, posterior circulation stroke; PNA, pneumonia; NIHSS, National Institutes of Health Stroke Scale; NS, not significant; IV tPA, intravenous tissue-type plasminogen activator; MSH, melanocyte stimulating hormone. α-MSH values indicate the highest α-MSH concentration within the first 72 h after stroke onset.

*Three patients did not have MRI imaging and are not included in this analysis ($P=0.200$).
robust anti-inflammatory and immunomodulatory properties of the immune system and is responsible for mediating the autoimmune encephalomyelitis. We also found that inhalation. MSH has neurotrophic properties and can improve outcome in animal models of experimental allergy. It is not surprising that it has been shown to improve outcome in animal models of stroke. Consistent with the observation that early administration may still be of therapeutic value, the attractiveness of MSH as a therapeutic agent is further enhanced by its potential ease of administration; MSH-related neuropeptides are absorbed through the nasal mucosa rapidly after inhalation. In addition to exogenous administration of the neuropeptide, plasma MSH concentrations could be augmented by strategies that favor MSH processing from POMC/ACTH (ie, enhancing PC2 activity). The potent immunomodulatory properties of MSH, however, suggest the possibility that this peptide could predispose to infection, a complication that was seen in an animal model of stroke. In the current study, however, we did not find an independent association between MSH and infection risk. Furthermore, we did not see infectious complications related to MSH administration in our animal model of stroke.

Limitations of this study include the lack of tightly controlled timing of blood draws early after stroke onset. The median time from stroke onset to the “24-hour” blood draw was 28 hours (N=30), whereas the median time from stroke onset to the “72-hour” blood draw was 68 hours (N=101). It is certainly possible that rapid changes in plasma MSH were missed by this sampling protocol. For the logistic regression, we chose to use the highest MSH (ACTH, cortisol, and IL-6) within the first 72 h after stroke. MSH indicates melanocyte stimulating hormone; ACTH, adrenocorticotrophic hormone; IL, interleukin; NIHSS, National Institutes of Health Stroke Scale; NS, not significant (P>0.200); CI, confidence interval.

Data are presented as the odds ratio (95% CI) per 10-ng/mL increase in plasma MSH concentrations. The data show the highest plasma MSH indicates melanocyte stimulating hormone; ACTH, adrenocorticotrophic hormone; IL, interleukin; NIHSS, National Institutes of Health Stroke Scale; NS, not significant (P>0.200); CI, confidence interval.

Data show the highest plasma MSH within the first 72 h after stroke.

- MS-H is an attractive candidate for stroke therapy given its multiplicity of actions and the possibility that delayed administration may still be of therapeutic value. The attractiveness of MSH as a therapeutic agent is further enhanced by its potential ease of administration; MSH-related neuropeptides are absorbed through the nasal mucosa rapidly after inhalation. In addition to exogenous administration of the neuropeptide, plasma MSH concentrations could be augmented by strategies that favor MSH processing from POMC/ACTH (ie, enhancing PC2 activity). The potent immunomodulatory properties of MSH, however, suggest the possibility that this peptide could predispose to infection, a complication that was seen in an animal model of stroke. In the current study, however, we did not find an independent association between MSH and infection risk. Furthermore, we did not see infectious complications related to MSH administration in our animal model of stroke.

Limitations of this study include the lack of tightly controlled timing of blood draws early after stroke onset. The median time from stroke onset to the “24-hour” blood draw was 28 hours (N=30), whereas the median time from stroke onset to the “72-hour” blood draw was 68 hours (N=101). It is certainly possible that rapid changes in plasma MSH were missed by this sampling protocol. For the logistic regression, we chose to use the highest MSH (ACTH, cortisol, and IL-6) within the first 72 hours of stroke onset to increase statistical power (if only the 72-hour values are used, the results are similar but not quite as strong).

Table 3. Likelihood for a Good Outcome at Given Time Points Based on Initial Plasma MSH or Cortisol

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model</th>
<th>α-MSH mRS ≤3</th>
<th>P</th>
<th>ACTH mRS ≤3</th>
<th>P</th>
<th>Cortisol mRS ≤3</th>
<th>P</th>
<th>IL-6 mRS ≤3</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mo, N=102</td>
<td>Unadjusted</td>
<td>1.355 (0.938–1.957)</td>
<td>0.105</td>
<td>0.852 (0.676–1.075)</td>
<td>0.176</td>
<td>0.169 (0.075–0.381)</td>
<td>&lt;0.001</td>
<td>0.583 (0.389–0.874)</td>
<td>0.009</td>
</tr>
<tr>
<td></td>
<td>NIHSS</td>
<td>1.031 (0.694–1.531)</td>
<td>NS</td>
<td>0.921 (0.688–1.232)</td>
<td>NS</td>
<td>0.394 (0.158–0.986)</td>
<td>0.047</td>
<td>0.896 (0.633–1.269)</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>NIHSS+age</td>
<td>1.000 (0.667–1.499)</td>
<td>NS</td>
<td>0.931 (0.698–1.241)</td>
<td>NS</td>
<td>0.389 (0.153–0.988)</td>
<td>0.047</td>
<td>0.915 (0.643–1.302)</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>NIHSS+age+infection</td>
<td>0.991 (0.657–1.494)</td>
<td>NS</td>
<td>0.940 (0.693–1.274)</td>
<td>NS</td>
<td>0.392 (0.154–0.998)</td>
<td>0.049</td>
<td>0.940 (0.661–1.337)</td>
<td>NS</td>
</tr>
<tr>
<td>3 mo, N=100</td>
<td>Unadjusted</td>
<td>2.147 (1.159–3.978)</td>
<td>0.015</td>
<td>0.840 (0.659–1.070)</td>
<td>0.157</td>
<td>0.299 (0.154–0.581)</td>
<td>&lt;0.001</td>
<td>0.556 (0.375–0.825)</td>
<td>0.004</td>
</tr>
<tr>
<td></td>
<td>NIHSS</td>
<td>1.639 (0.883–3.043)</td>
<td>0.118</td>
<td>0.880 (0.657–1.180)</td>
<td>NS</td>
<td>0.657 (0.307–1.405)</td>
<td>NS</td>
<td>0.755 (0.518–1.102)</td>
<td>0.145</td>
</tr>
<tr>
<td></td>
<td>NIHSS+age</td>
<td>1.568 (0.844–2.911)</td>
<td>0.154</td>
<td>0.896 (0.678–1.183)</td>
<td>NS</td>
<td>0.664 (0.300–1.467)</td>
<td>NS</td>
<td>0.752 (0.503–1.123)</td>
<td>0.163</td>
</tr>
<tr>
<td></td>
<td>NIHSS+age+infection</td>
<td>1.688 (0.859–3.319)</td>
<td>0.129</td>
<td>0.923 (0.682–1.248)</td>
<td>NS</td>
<td>0.721 (0.311–1.669)</td>
<td>NS</td>
<td>0.792 (0.530–1.183)</td>
<td>NS</td>
</tr>
<tr>
<td>6 mo, N=97</td>
<td>Unadjusted</td>
<td>5.212 (1.614–16.834)</td>
<td>0.006</td>
<td>0.736 (0.560–0.969)</td>
<td>0.029</td>
<td>0.368 (0.178–0.759)</td>
<td>0.007</td>
<td>0.877 (0.738–1.042)</td>
<td>0.136</td>
</tr>
<tr>
<td></td>
<td>NIHSS</td>
<td>4.219 (1.225–14.530)</td>
<td>0.023</td>
<td>0.741 (0.533–1.032)</td>
<td>0.076</td>
<td>0.724 (0.300–1.747)</td>
<td>NS</td>
<td>1.033 (0.655–1.248)</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>NIHSS+age</td>
<td>4.236 (1.191–15.063)</td>
<td>0.026</td>
<td>0.729 (0.534–0.996)</td>
<td>0.047</td>
<td>0.662 (0.254–1.727)</td>
<td>NS</td>
<td>1.030 (0.652–1.246)</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>NIHSS+age+infection</td>
<td>5.763 (1.350–24.591)</td>
<td>0.018</td>
<td>0.737 (0.537–1.011)</td>
<td>0.059</td>
<td>0.677 (0.257–1.784)</td>
<td>NS</td>
<td>1.050 (0.863–1.278)</td>
<td>NS</td>
</tr>
<tr>
<td>12 mo, N=96</td>
<td>Unadjusted</td>
<td>4.444 (1.322–14.943)</td>
<td>0.016</td>
<td>0.874 (0.657–1.163)</td>
<td>NS</td>
<td>0.464 (0.215–1.000)</td>
<td>0.050</td>
<td>0.873 (0.734–1.039)</td>
<td>0.125</td>
</tr>
<tr>
<td></td>
<td>NIHSS</td>
<td>3.551 (1.034–12.201)</td>
<td>0.044</td>
<td>0.922 (0.683–1.245)</td>
<td>NS</td>
<td>0.937 (0.355–2.473)</td>
<td>NS</td>
<td>1.006 (0.631–1.218)</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>NIHSS+age</td>
<td>3.502 (0.991–12.370)</td>
<td>0.052</td>
<td>0.920 (0.625–1.354)</td>
<td>NS</td>
<td>0.859 (0.288–2.563)</td>
<td>NS</td>
<td>0.998 (0.820–1.215)</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>NIHSS+age+infection</td>
<td>4.612 (1.087–19.572)</td>
<td>0.038</td>
<td>0.933 (0.630–1.381)</td>
<td>NS</td>
<td>0.933 (0.294–2.955)</td>
<td>NS</td>
<td>1.012 (0.826–1.240)</td>
<td>NS</td>
</tr>
</tbody>
</table>
concentrations of plasma α-MSH are independently associated with better stroke outcome. These data, along with a robust body of experimental data, suggest that strategies to increase α-MSH may be a viable therapeutic intervention for the treatment of acute ischemic stroke and should be further investigated.

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**Disclosures**

None.

**References**

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血漿中α-メラニン細胞刺激ホルモン濃度は虚血性脳卒中の転帰を予測する

Plasma α-Melanocyte Stimulating Hormone Predicts Outcome in Ischemic Stroke

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Abstract

脳卒中後24時間の血漿中α-MSH濃度は予後を予測する可能性が示唆されている。虚血性脳卒中患者の脳損傷の程度と関連があるとされるα-MSH濃度を測定し、その予後に影響を与えるかを検討した。

結果: 脳卒中の患者において、血漿中α-MSH濃度は脳卒中後の予後を予測する可能性がある。α-MSH濃度が高いほど予後は良好であった。

結論: 血漿中α-MSH濃度は脳卒中の予後を予測する能力があると示された。