Increased Resting Energy Expenditure after Endovascular Coiling for Subarachnoid Hemorrhage

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Background: Appropriate nutritional care from the acute stage is essential for improved functional outcomes and reduced mortality in patients with subarachnoid hemorrhage (SAH). Although endovascular coiling is increasingly being used as an alternative to neurosurgical clipping and craniotomy for ruptured aneurysms, the resting energy expenditure (REE) of patients treated with this new technique has not been systemically evaluated. *Methods:* We measured REE values by indirect calorimetry in 12 SAH patients treated with endovascular coiling. We averaged the REE measurements obtained on days 1 and 7 after endovascular coiling, and then we statistically compared the mean REE values with those in 30 patients with acute cerebral infarction (ACI) by the Wilcoxon rank-sum test ($P < .05$). Next, we calculated the ratio of measured REE values to the values estimated using the Harris–Benedict equation to adjust for demographic differences in sex, weight, height, and age between the groups. *Results:* The ratios were significantly higher in SAH patients (median value, 1.12; interquartile range, 1.05-1.23) than in ACI patients (median value, 1.02; interquartile range, .97-1.09). *Conclusions:* Because endovascular coiling is less invasive than neurosurgical clipping, the observed increase in REE was attributed to metabolic changes after SAH. To provide optimal nutritional care to SAH patients from the acute stage, clinicians should be aware of this change in REE. **Key Words:** Diet—embolization—metabolism—nutrition—stroke.

Introduction

Appropriate nutritional care is critically important for stroke patients to achieve better functional outcome and reduced mortality.[1,2] Metabolic parameters such as resting energy expenditure (REE) differ depending upon the type of stroke and severity of symptoms.[3,4] Subarachnoid hemorrhage (SAH) is one of the most severe types of stroke and often requires surgical and/or endovascular intervention, and is associated with hypermetabolism during the acute stage.[5,6] However, previous studies included only patients who had undergone conventional procedures such as neurosurgical clipping and craniotomy, which often lead to hypermetabolism due to their invasiveness.

However, treatment for SAH has changed in the last decade. Endovascular coiling, a less invasive procedure than neurosurgical clipping or craniotomy, is increasingly being used as an alternative treatment for ruptured intracranial aneurysms.[7] However, to our knowledge, no studies have investigated REE in patients treated with endovascular coiling for SAH due to a ruptured aneurysm. The aim of this study was to evaluate REE in the acute care setting among patients with SAH treated with endovascular coiling.

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Materials and Methods

Patients

The present study included patients who were diagnosed as having SAH and who underwent endovascular coiling for ruptured intracranial aneurysm at Nishinomiya Kyoritsu Neurosurgical Hospital from April 2014 to February 2015. The diagnosis of SAH and the location of the aneurysm were confirmed by computed tomography, magnetic resonance angiography, and computed tomography angiography on admission. The severity of SAH was classified according to the World Federation of Neurological Surgeons (WFNS) Scale[8] and the Hunt and Kosnik scale.[9] On admission, weight was measured by using a patient lift scale and height was measured in the supine position. To minimize variability arising from differences in endovascular intervention technique, we included only patients who were treated by a single neurosurgeon (Y.Y., the second author of this article). Endovascular coiling was performed within 2 days of SAH onset in all patients. Exclusion criteria were as follows: (1) oxygen therapy or ventilator dependence; (2) conditions associated with altered energy metabolism (e.g., fever >38°C, poorly controlled diabetes mellitus, advanced cancer, sepsis); (3) coma; (4) restlessness; and (5) refusal of indirect calorimetry measurements. A total of 17 SAH patients were recruited. Among them, five were excluded from the analysis based on the exclusion criteria (3 patients were dependent on a ventilator and 2 patients were receiving oxygen therapy).

Controls

Thirty patients with acute cerebral infarction (ACI) who were hospitalized during the same period as those with SAH were used as a control group. The details of these 30 patients have been previously reported.[10] Briefly, the control group comprised patients who lived independently in their community before stroke onset, and whose severity of stroke symptoms on admission was between 4 and 25 according to the National Institutes of Health Stroke Scale. Patients who subsequently required acute medical services were excluded.

Metabolic Measurement

We assessed REE by indirect calorimetry using a portable metabolic analyzer designed to measure oxygen consumption and energy expenditure (FitMate Metabolic System, COSMED, Rome, Italy). Measurements were performed on days 1 and 7 after endovascular coiling, between 6 a.m. and 8 a.m. at least 9 hours after meal consumption.[11] The patients rested in the supine position for at least 30 minutes before each measurement and were instructed to breathe normally, but not to talk, move, or sleep during the measurement. The gas analyzer was automatically calibrated before each measurement. All measurements were conducted for 15 minutes, and the first 5 minutes of data were discarded.[12] Heart rate, respiratory rate, and blood pressure were monitored during the measurement process. Data collected on days 1 and 7 after endovascular coiling were averaged, and the mean was used in statistical analysis. The measurement procedures were nearly identical for the control subjects, as previously reported.[10]

REE is dependent on age, sex, weight, height, and other variables.[13,14] In the present study, in addition to indirect calorimetry measurements, we also estimated REE values derived from the Harris–Benedict equation. This equation is the most widely used predictive equation for estimating REE in a wide variety of clinical settings, including acute stroke care,[15] and it is used to calculate REE (kilocalorie) as follows:

$$\text{Men: } 13.75 \times \text{Weight (kg)} + 5 \times \text{Height (cm)} - 6.76 \times \text{Age (years)} + 66.47$$

$$\text{Women: } 9.56 \times \text{Weight (kg)} + 1.85 \times \text{Height (cm)} - 4.86 \times \text{Age (years)} + 655.1$$

The calculated values were then used to adjust REE values for differences in sex, weight, height, and age between the SAH and ACI groups.

Statistical Analysis

The aim of the present study was to evaluate REE among patients with SAH in the acute care setting relative to patients with ACI. To accomplish this, we adjusted for the differences in patient characteristics using the Harris–Benedict equation. In this procedure, the ratio of the measured REE value obtained using indirect calorimetry to the estimated value obtained using the Harris–Benedict equation was calculated for each patient to adjust for age, sex, weight, and height. This ratio was then used to compare the SAH and ACI groups using the Wilcoxon rank-sum test.

To compare the profiles of the patients in the SAH and ACI (control) groups, we used the Pearson chi-square test for categorical data and the Wilcoxon rank-sum test for interval data. A P value less than .05 was considered statistically significant.

Ethical Considerations

The study protocol was approved by the Institutional Review Board of Hyogo College of Medicine. Written informed consent for inclusion in the study was obtained from all patients or their family members.

Results

The profiles of the 12 patients (9 women, 3 men; median age, 54.5 years; age range, 35-84 years; median weight, 54.7 kg; weight range, 43.1-96.1 kg; mean height, 153 cm;