Loss of Consciousness at Onset of Subarachnoid Hemorrhage as an Important Marker of Early Brain Injury

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IMPORTANCE Loss of consciousness (LOC) is a common presenting symptom of subarachnoid hemorrhage (SAH) that is presumed to result from transient intracranial circulatory arrest.

OBJECTIVE To clarify the association between LOC at onset of SAH, complications while in the hospital, and long-term outcome after SAH.

DESIGN, SETTING, AND PARTICIPANTS A retrospective analysis was conducted of 1460 consecutively treated patients with spontaneous SAH who were part of a prospective observational cohort study at a large urban academic medical center (the Columbia University SAH Outcomes Project or SHOP). Patients were enrolled between August 6, 1996, and July 23, 2012. Analysis was conducted from December 1, 2013, to February 28, 2015.

EXPOSURES Loss of consciousness at onset was identified by structured interview of the patient and first responders. Patients (80.5%) were observed for up to 1 year to assess functional recovery.

MAIN OUTCOMES AND MEASURES Modified Rankin scale scores were assigned based on telephone or in-person interviews of the patient, family members, or caregivers. Complications while in the hospital were predefined and adjudicated by the study team.

RESULTS Five hundred ninety patients (40.4%) reported LOC at onset of SAH. Loss of consciousness was associated with poor clinical grade, more subarachnoid and intraventricular blood seen on admission computed tomographic scan, and a higher frequency of global cerebral edema (P < .001). Loss of consciousness was also associated with more prehospital tonic-clonic activity (22.7% vs 4.2%; P < .001) and cardiopulmonary arrest (9.7% vs 0.5%; P < .001) vs patients who did not experience LOC. In multivariable analysis, death or severe disability at 12 months was independently associated with LOC after adjusting for established risk factors for poor outcome, including poor admission clinical grade (adjusted odds ratio, 1.94; 95% CI, 1.38-2.72; P < .001). There was no association between LOC at onset and delayed cerebral ischemia or aneurysm rebleeding.

CONCLUSIONS AND RELEVANCE Loss of consciousness at symptom onset is an important manifestation of early brain injury after SAH and a predictor of death or poor functional outcome at 12 months.

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Loss of consciousness (LOC) is one of the most common presenting symptoms of subarachnoid hemorrhage (SAH). Subarachnoid hemorrhage is often accompanied by a dramatic increase in intracranial pressure and reduction in cerebral perfusion pressure, leading to transient cessation of cerebral blood flow, as documented by angiography and transcranial Doppler ultrasonography. Loss of consciousness at ictus also has been linked to global cerebral edema, a marker of early brain injury after SAH demonstrated on computed tomographic (CT) scan.

Initial clinical grade assessed with the Hunt and Hess scale or World Federation of Neurological Surgeons scale are well established strong prognostic indicators in SAH. There are data analyzing the long-term effect of symptoms at onset of SAH on survival and recovery. We sought to determine the effect of LOC at onset of SAH, complications during hospitalization, and 1-year outcome.

### Clinical Assessment

**History of present illness**, including symptoms of SAH within 6 hours of onset (headache, LOC, nausea, vomiting, change in mental status, seizures, cardiopulmonary resuscitation performed, and sentinel headache) were obtained by interview of the patient, family members, and first responders, such as emergency medical response personnel. Loss of consciousness was broadly defined as any sudden, abnormal alteration of alertness, awareness, or responsiveness to sensory stimuli at symptom onset during the prehospital phase of illness, regardless of duration. Starting in 2002, in addition to recording the presence or absence of LOC, we added an item to evaluate the duration of LOC (<10 minutes with subsequent recovery, 10-60 minutes with recovery, or >60 minutes with or without subsequent recovery). Prehospital tonic-clonic activity was recorded based on accounts from eyewitnesses, and cardio-pulmonary arrest was defined as any episode of apnea or lack of pulse treated with basic life support. Demographic data, including age, sex, race/ethnicity, and relevant medical history were recorded. Results from the initial neurologic examination on hospital admission were evaluated based on the Hunt and Hess Scale and Glasgow Coma Scale administered by emergency department health care professionals or a study neurointensivist. We also assessed Acute Physiology and Chronic Health Evaluation (APACHE) II diagnostic category and calculated a physiological subscore by subtracting the Glasgow Coma Scale score, age, and chronic health conditions (eg, severe organ system insufficiency, compromised immune system) from the total score.

### Radiographic and Laboratory Assessment

Modified Fisher scale score and presence of intracerebral hemorrhage, intraventricular hemorrhage, hydrocephalus, and infarction were recorded using the results of the initial CT scan. Presence and degree of hydrocephalus were measured by bicaudate index as previously described. Computed tomographic scanning was performed after all episodes of neurologic deterioration. We also recorded initial glucose level, cardiac troponin level, chest radiograph findings, echocardiographic findings, and all initial diagnostic and follow-up angiographic findings (location and size of ruptured aneurysm, presence of vasospasm, and so forth).

### Clinical Management

Surgical or neuroradiologic treatment for aneurysm was performed as soon as possible. Nimodipine, 60 mg, was given orally every 4 hours. Vasospasm was assessed by transcranial Doppler ultrasonography, CT angiography, or angiography. Patients received fluid and blood pressure management to avoid hypovolemia and to maintain euvoemia. Normal saline solution and supplemental albumin, 5%, were administered to maintain even fluid balance and a central venous pressure of 5 mm Hg or more. Symptomatic delayed cerebral ischemia (DCI) was treated with vasopressors to maintain systolic blood pressure between 180 and 220 mm Hg in most patients. Patients whose volume and pressure treatment failed were considered for inotrope administration and/or endovascular treatment.

### Outcome Assessment

Modified Rankin Scale (mRS) scores were prospectively assessed at 3 and 12 months. Hospital complications after SAH were diagnosed by the treating neurointensivist and adjudicated by the entire SHOP study team on a weekly basis. Global cerebral edema was diagnosed based on CT scan results, as previously described. Delayed neurologic deterioration from all causes was defined as a 2-point or more decrease in the Glasgow Coma Scale score or new focal finding within any 24-hour period, excluding postoperative (<48 hours) deterioration due to operative complications. Delayed cerebral ischemia was defined as otherwise unexplained clinical deterioration (such as a new focal deficit, decrease in level of consciousness, or both) or a new infarct shown on CT scan that was not visible on the admission or immediate postoperative CT scan, or both, after exclusion of other potential causes of clinical deterioration, such as hydrocephalus, rebleeding, or seizures. Aneurysm rebleeding was defined as an acute neurologic deterioration with a new hemorrhage apparent on CT scan.
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Statistical Analysis
Data are presented as mean (SD) or median (interquartile range) for continuous variables and as absolute numbers and percentages for categorical variables. All analyses were performed with R statistical software, version 2.12.2 (R Project), and STATA, version 14.0 (StataCorp LP). Owing to the multiple statistical tests performed, \( P < .01 \) was considered statistically significant. All variables were considered in univariate analysis. Logistic regression was used to test the association of the presence of LOC to functional outcome and complications using known predictors of poor outcome as covariates. Poor outcome was defined as death and moderate to severe disability at 1 year (mRS score, 4-6). To determine the prognostic significance of transient LOC without confounding by seizures, cardiac arrest, or sustained impairment of consciousness owing to poor clinical grade, we repeated the logistic regression analysis of outcome predictors after excluding patients who had experienced tonic-clonic activity, underwent cardiopulmonary resuscitation, or had poor clinical grade on admission (Hunt and Hess score of 4 or 5). According to methodological guidelines from the Patient-Centered Outcomes Research Institute, we performed multiple imputations using Bayesian methods to account for the 12-month mRS scores lost to follow-up. Procedures to create and analyze 5 imputed data sets were carried out using the mi package for R. Diagnostic plots were used to evaluate the fit of the imputed values produced by the marginal model.

Results

Admission Clinical Features
Among 1482 patients with SAH enrolled in SHOP, the presence or absence of LOC was recorded in 1460 patients. Mean (SD) patient age was 55 (15) years, 486 patients (33.3%) were men, and 669 patients (45.8%) were white. Five hundred ninety patients (40.4%) lost consciousness at onset of SAH. Among 443 of these patients whose duration of LOC was recorded (starting in 2002), 169 (38.1%) lost consciousness for less than 10 minutes, 93 (21.0%) for 10 to 60 minutes, and 181 (40.9%) for longer than 60 minutes. Longer duration of LOC was associated with worse Hunt and Hess scale scores on admission (Table 1). If LOC lasted less than 10 minutes, the risk of presenting with a Hunt and Hess score of 4 or 5 was only 16.0% and 6.5%, respectively; if LOC lasted more than 60 minutes, the risk was 21.5% and 67.4%, respectively.

Admission characteristics associated with LOC at onset of SAH included a history of hypertension, a discrete sentinel headache less than 2 weeks before the index bleeding event, persistent change in mental status, acute tonic-clonic activity, and cardiac arrest with successful cardiopulmonary resuscitation (Table 2). Loss of consciousness was also associated with poor clinical grade assessed with the Hunt and Hess scale and Glasgow Coma Scale, and higher median APACHE II physiological subcores, higher serum glucose and troponin levels, and a higher frequency of pulmonary edema and left ventricular dysfunction seen on echocardiography (Table 2). Headache and vomiting at onset occurred less frequently in patients with LOC. There were no differences with regard to age, sex, race/ethnicity, or systolic blood pressure on admission.

Admission Radiologic Features
Compared with patients who did not lose consciousness, patients with LOC had more cisternal and intraventricular blood on CT scan results, global cerebral edema, parenchymal hematoma, hydrocephalus, and acute infarction (Table 2). Admission angiography demonstrated a higher frequency of large aneurysms (>10 mm) and posterior circulation aneurysm location in patients with LOC but no difference in the frequency of acute or ultra-early vasospasm.

Aneurysm Treatment and Hospital Complications
Patients with LOC at onset of SAH were more likely to have their aneurysm be coiled than clipped and were treated a mean of one-half day earlier than patients who did not lose consciousness (Table 3). In univariate analysis, LOC was associated with an increased risk of all-cause neurologic deterioration, DCI, aneurysm rebleeding, and new infarction from any cause detected on follow-up CT scan (Table 3). Logistic regression revealed that after adjusting for age, admission Hunt and Hess grade, APACHE II physiological subcore, and aneurysm size, LOC was associated with global cerebral edema but not with DCI or rebleeding (Table 4).

Outcome at Discharge and 12 Months
We imputed 12-month mRS scores from discharge or 3-month outcomes for 20% (n = 228) of patients who were lost to follow-up. We found that 51.2% of patients (n = 154) with LOC were dead or severely disabled at 12 months (mRS score of 4-6) compared with 17.7% (n = 302) of those who did not lose consciousness (\( P < .001 \)) (Table 3). The overall pattern of recovery in both groups showed that between discharge and 12 months the relative proportion of patients with no disability (mRS score, 0 or 1) or death (mRS score of 6) increased whereas those who were bedbound or unable to walk without assistance (mRS score of 4 or 5) decreased substantially (Figure). Multivariable logistic regression analysis revealed that death or functional dependence at 12 months was significantly associated with LOC even after controlling for age, admission clinical grade, APACHE II physiological subcore, and aneurysm size (Table 4). After excluding patients with prehospital cardiac arrest, witnessed tonic-clonic activity at onset, or poor grade on admission (Hunt and Hess score of 4 or 5), LOC remained significantly related to functional outcome at 12 months (odds ratio, 2.0; \( P = .003 \)) (eTable in the Supplement).

Discussion
Our findings indicate that LOC at onset of SAH is a simple and robust indicator of a severe bleeding event. Patients with LOC had significantly larger SAHs, intraventricular hemorrhages, and parenchymal intracerebral hemorrhages on admission CT scan findings. Loss of consciousness was also associated with global cerebral edema, which is thought to be an important
Table 1. Hunt and Hess Scale Scores on Admission

<table>
<thead>
<tr>
<th>Duration of LOC, min</th>
<th>Admission Hunt and Hess Grade, No. (%)a</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 or 2 (Headache Only)</td>
</tr>
<tr>
<td>&lt;10</td>
<td>80 (47.3)</td>
</tr>
<tr>
<td>10-60</td>
<td>19 (20.4)</td>
</tr>
<tr>
<td>&gt;60</td>
<td>4 (2.2)</td>
</tr>
</tbody>
</table>

Abbreviation: LOC, loss of consciousness.

* Percentages are calculated across each row. Data are reported on a subset of 443 patients enrolled from July 2002 through July 2012. Patients with LOC less than 60 minutes were observed to have recovered wakefulness during the prehospital phase; if they were subsequently found to have a Hunt and Hess grade 4 or 5 on admission, they effectively experienced a “lucid interval.” All patients with LOC more than 60 minutes who presented with a Hunt and Hess grade 4 or 5 remained persistently unconscious from the onset of symptoms.

Table 2. Admission Characteristics of Patients With SAH With and Without Loss of Consciousness

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Loss of Consciousnessa</th>
<th>Present (n = 590)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent (n = 870)</td>
<td>55 (14)</td>
<td>55 (15)</td>
<td>.81</td>
</tr>
<tr>
<td>Present (n = 590)</td>
<td>55 (15)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male, No. (%)</td>
<td>295 (33.9)</td>
<td>191 (32.4)</td>
<td>.54</td>
</tr>
<tr>
<td>White race/ethnicity, No. (%)</td>
<td>412 (47.4)</td>
<td>257 (43.6)</td>
<td>.15</td>
</tr>
<tr>
<td>Medical history, No. (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>381 (44.5)</td>
<td>302 (53.9)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>67 (7.9)</td>
<td>50 (9.1)</td>
<td>.41</td>
</tr>
<tr>
<td>Atrial fibrillation or arrhythmia</td>
<td>22 (2.6)</td>
<td>18 (3.3)</td>
<td>.44</td>
</tr>
<tr>
<td>Prehospital symptoms, No. (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sentinel headache</td>
<td>150 (18.0)</td>
<td>133 (26.4)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Headache at onset</td>
<td>844 (97.5)</td>
<td>466 (87.3)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Vomiting</td>
<td>532 (61.4)</td>
<td>297 (51.9)</td>
<td>.005</td>
</tr>
<tr>
<td>Change in mental status</td>
<td>237 (27.4)</td>
<td>395 (69.7)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Tonic-clonic activity</td>
<td>36 (4.2)</td>
<td>128 (22.7)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Cardiac arrest with successful</td>
<td>2 (0.5)</td>
<td>26 (9.7)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>cardiopulmonary resuscitation</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Hunt and Hess grade, No. (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 (Mild headache)</td>
<td>299 (34.4)</td>
<td>61 (10.3)</td>
<td></td>
</tr>
<tr>
<td>2 (Moderate to severe headache)</td>
<td>232 (26.7)</td>
<td>52 (8.8)</td>
<td></td>
</tr>
<tr>
<td>3 (Lethargy, confusion, or mild focal signs)</td>
<td>222 (25.5)</td>
<td>150 (25.4)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>4 (Stupor)</td>
<td>87 (10.0)</td>
<td>116 (19.7)</td>
<td></td>
</tr>
<tr>
<td>5 (Coma)</td>
<td>30 (3.5)</td>
<td>211 (35.8)</td>
<td></td>
</tr>
<tr>
<td>Glasgow Coma Scale score, median (IQR)</td>
<td>15 (4-15)</td>
<td>8 (4-14)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>APACHE II physiological subscore, median (IQR)b</td>
<td>5 (3-7)</td>
<td>7 (5-11)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Systolic blood pressure, mean (SD), mm Hg</td>
<td>157 (33)</td>
<td>159 (393)</td>
<td>.49</td>
</tr>
<tr>
<td>Glucose, mean (SD), mg/dL</td>
<td>141 (44)</td>
<td>186 (82)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Troponin I, median (IQR), ng/mL</td>
<td>0.02 (0.02-0.50)</td>
<td>0.20 (0.02-0.70)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Pulmonary edema on chest radiograph, No. (%)</td>
<td>39 (4.6)</td>
<td>82 (14.3)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Ejection fraction &lt;50%, No. (%)</td>
<td>39 (8.7)</td>
<td>89 (22.4)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Abbreviations: APACHE, Acute Physiology and Chronic Health Evaluation; IQR, interquartile range; IVH, intraventricular hemorrhage; SAH, subarachnoid hemorrhage.

SI conversion factors: To convert glucose to millimoles per liter, multiply by 0.0555; troponin I to micrograms per liter, multiply by 1.0.

* Percentages reflect the number of patients with the complication divided by the number of patients with a completed response.

b Scale: 0, no physiological derangement; 51, maximal physiological derangement.

c Scale: 0, no SAH; 30, SAH completely filling all cisterns and fissures.

d Scale: 0, no IVH; 16, IVH completely filling all 4 ventricles.
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Five hundred ninety patients (40.4%) in our overall study population presented with LOC, which is within the range of previous reports.1,16 In univariate analysis, patients with LOC reported less headache and vomiting, which most likely reflects the inability to self-report an accurate history when the initial symptoms were not witnessed. Loss of consciousness was strongly associated with poor clinical grade: 327 (55.4%) of those who reported LOC at symptom onset were subsequently assessed as Hunt and Hess grade 4 or 5 (stuporous or comatose) on hospital admission, whereas only 117 (13.4%) patients who did not experience LOC were assessed as the same grades on admission. A previous report found that one-third of 70 patients with SAH who presented with acute confusion (Hunt and Hess grade 3) had experienced antecedent LOC.17 In our study, the proportion of patients assessed as Hunt and Hess grade 3 was nearly identical among those who did or did not experience LOC (25.4% and 25.5%, respectively), suggesting that LOC at symptom onset implies an increased risk of severe, as opposed to mild, brain injury. As a result of the association between LOC and poor clinical grade on presentation, these patients were treated earlier and treated more frequently with coils than was the cohort who did not experience LOC.

We confirmed the previously reported association between LOC and global cerebral edema.5,11 The cause of early global cerebral edema is thought to be rebound hyperemia associated with blood-brain barrier disruption in the setting of abnormal autoregulation after intracranial circulatory arrest.5 Our results did not demonstrate an independent association between LOC and subsequent DCI or rebleeding after controlling for other prognostic variables. By contrast, a prior study of 125 patients with SAH reported that 43% of patients lost consciousness at ictus for longer than 1 hour, and longer duration of unconsciousness predicted the subsequent occurrence of DCI.18 Another prospective study found that LOC was associated with rebleeding and death or severe disability at 3 months.19 One possible explanation for our failure to replicate these findings is reduced sensitivity owing to lower event rates in our study, as care has been modernized and outcomes have improved (DCI, 31%16 vs 19.6% in our study; and rebleeding, 23%16 vs 8.8% in our study).

The most common mechanisms underlying LOC at onset of SAH are likely to be reduced cerebral perfusion pressure in the setting of elevated intracranial pressure, seizures, or neurogenic cardiopulmonary dysfunction, manifesting as hypotension or frank cardiac arrest. In many cases, the cause of LOC may be multifactorial. A nearly instantaneous increase in intracranial pressure can occur in less than 1 minute after SAH.2 If the reduction in cerebral perfusion pressure is transient, presumably the patient will fully recover.
consciousness, as he or she would after a syncopal attack. In our study, nearly 40% of patients lost consciousness for less than 10 minutes, and another 21.0% lost consciousness for 10 to 60 minutes, with subsequent witnessed recovery of alertness during the prehospital phase. Many of these patients effectively experienced a “lucid interval” and were later found to have lapsed back into stupor or coma (Hunt and Hess grade 4 or 5) on hospital admission (Table 1). In these cases, the presumable cause of secondary worsening was either owing to a gradual increase in intracranial pressure from obstructive hydrocephalus or progressive brain edema, or aneurysm rebleeding. Regardless, we found that the association between LOC and poor outcome persisted even after excluding patients assessed as Hunt and Hess grade 4 or 5 and those with acute tonic-clonic activity or cardiac arrest, confirming that even syncope at onset with sustained recovery of consciousness thereafter confers a poor prognosis. It is well established that even with transient LOC, a corresponding episode of brief global hypoxia-ischemia is enough to trigger apoptosis, delayed neuronal death, and other negative downstream effects in selectively vulnerable cell populations.

In the 40% of patients with LOC who fail to recover consciousness within 1 hour, sudden LOC presages a state of prolonged unconsciousness, in which case the patient presents with a poor clinical grade owing to severe early brain injury. A primary reduction of cerebral perfusion pressure caused by severe intracranial hypertension is the most likely primary cause of early and prolonged LOC at onset of SAH. Autoregulatory failure, acute vasoconstriction, cortical spreading depolarization, and intraluminal platelet aggregation may also contribute to cerebral blood flow reduction in early brain injury. Acute physiological derangements, such as extremes of blood pressure, hyperglycemia, hypoxemia, and metabolic acidosis, have been shown to predict both poor admission grade and long-term outcome and may exacerbate these processes and serve as targets for intervention. Evidence of acute ischemic injury on diffusion-weighted imaging is present in approximately 70% of patients with SAH presenting with a poor clinical grade on admission.

Tonic-clonic activity was described in 128 of 563 patients (22.7%) with LOC in our study, as opposed to only 36 of 870 (4.2%) of those without LOC. Previous studies have reported seizure activity at onset in 8% to 27% of patients with SAH. Diagnostic uncertainty is likely to be common when bystanders describe tonic-clonic activity at onset of SAH since similar movements can result from epileptic activity, cerebral hypoperfusion, or motor posturing. Regardless, our data suggest that up to one-fifth of cases of LOC at onset of SAH may be directly caused by acute seizures. In a previous analysis of our study cohort, we found that tonic-clonic activity at onset is related to an increased risk of subsequent in-hospital seizures, pneumonia, and DCI but not with long-term disability or mortality after SAH.

Cardiac arrest treated successfully with prehospital cardiopulmonary resuscitation occurred in 9.7% of our patients who experienced LOC at onset of SAH. Subarachnoid hemorrhage is a well-known cause of sudden cardiac arrest. Among undifferentiated patients admitted to the hospital following out-of-hospital cardiac arrest, 6% to 16% have evidence of SAH on admission CT scans. In addition to cardiac arrest as a cause of LOC, our data suggest that more subtle neurocardiogenic disturbances may also play a contributing role. Troponin I elevation, pulmonary edema, and left ventricular dysfunction shown on the echocardiogram were associated with LOC in our study. Cardiac dysfunction has been associated with reduced global cerebral perfusion within the first 24 hours of SAH.

Our study has several limitations. Most important, LOC is a subjective phenomenon that we tracked from patient histories; the diagnosis may be prone to observer bias and
less-than-perfect interobserver reliability. In all likelihood, we have underestimated the frequency of LOC at onset of SAH because of the absence of a reliable witness in some cases. We broadly classified the duration of LOC pathway through the process of data collection. Future studies should more precisely track the timing of recovery of consciousness and obtain assessments of level of consciousness at multiple early time points. We did not record the presence of significant cardiac arrhythmia on admission, systematically perform early electroencephalograms to determine the frequency of epileptiform activity, or obtain admission magnetic resonance imaging to explore LOC as a potential risk factor for early ischemic injury. We assigned mRS scores without using a scripted interview and had to impute 1-year scores in 20% of our study population based on the best information available at 3 months.

Conclusions

With improvements in therapy for vasospasm and safer surgical techniques for aneurysm repair, early brain injury now poses the most important threat to survival with good recovery after SAH. Our study indicates that LOC at onset is associated with a 2.8-fold increase in the risk of poor outcome after SAH. Given its strong association with global cerebral edema and poor admission clinical grade, LOC should be considered a straightforward and clinically important marker of early brain injury after SAH, with ominous implications. In the future, the presence or absence of LOC may be useful for risk stratification and targeting therapy designed to minimize the effects of early brain injury after SAH.

REFERENCES


