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Correlation Between ED Symptoms and Clinical Outcomes in the Patient with Aneurysmal Subarachnoid Hemorrhage

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Abstract

Introduction—Aneurysmal subarachnoid hemorrhage (aSAH) is a devastating neurologic insult often presenting to the emergency department as a headache. Recognition and prompt treatment are important to good outcomes. The purpose of this analysis was to examine the presentation of aSAH patients to the emergency department and determine whether presentation predicts length of stay or death.

Methods—This is a retrospective review of data gathered from 2 existing studies. Data from patients diagnosed with acute aSAH were reviewed for symptoms, clinical presentation, history, demographics, and laboratory results. Statistical analysis was completed by use of χ^2 and regression analysis.

Results—This sample of 193 adult aSAH patients confirmed headache as well as meningeal signs as the most frequent symptom on presentation to the emergency department, and this was cited as the most common reason for seeking medical treatment. Symptom presentation did not appear to affect length of stay; however, survival analysis showed that patients who presented with a Hunt and Hess grade greater than 3 along with bradycardia were 15.6 times more likely to die within the first month of aSAH.

Discussion—Although aSAH presentation remains the same, this analysis did find a correlation between poor clinical grade and bradycardia to be a significant predictor of death at 30 days. Additional study may help to determine whether any intervention could lessen this effect. Although patient diagnosis and referral from the community emergency department to a tertiary center were relatively quick, there was a wide window of time between patient recognition of symptoms and seeking medical treatment.

Aneurysmal subarachnoid hemorrhage (aSAH), which affects roughly 30,000 Americans each year, is an overwhelming and devastating neurologic insult. What frequently begins as a headache may signal a complex neurologic cascade with high morbidity and mortality rates (approximately 45%).¹ aSAH (accounting for approximately 5% of all strokes) occurs when an aneurysm, typically located on one of the vessels in the circle of Willis, ruptures and spills blood into the subarachnoid space. This blood causes the patient to have the onset of a severe headache and signs of meningeal irritation (neck stiffness or nuchal rigidity, photophobia, and/or positive Kernig sign), as well as altered level of consciousness (LOC) or impaired cognition, seizure, nausea, vomiting, visual disturbances, and/or cranial nerve deficits.² Patients who report less severe symptoms may have a sentinel (warning) leak that may precede a larger hemorrhage by days to weeks. Risk factors for the development of aSAH include high blood pressure, smoking, and gender, with women aged over 55 years having a higher incidence of aSAH.^{3,4} Other factors associated with aSAH include alcohol and drug use (eg, sympathomimetic drugs such as cocaine) and family history of cerebrovascular disease.¹

Because the primary and most common symptom of aSAH is a headache, diagnosis is often made in the emergency department. After diagnosis, patients with aSAH require early intervention by experienced clinicians because delays in diagnosis and treatment have been associated with a higher risk of rebleeding and death.⁵ In turn, accurate and timely diagnosis, based on skilled and accurate assessments by nurses in the emergency department, are essential. Emergency nurses need to be alert to the presenting symptoms so that they can initiate protocols to heighten awareness of the possible diagnosis, provide frequent assessment, promote test completion, and facilitate disposition of the patient to the appropriate level of care.⁴ Because of the length and intensity of involvement that the nursing staff have with the patient, they are ideally positioned to coordinate multi-disciplinary care and facilitate interventions to improve outcomes in those at highest risk. The purpose of this study was to (1) identify key variables that are commonly associated with patients who present to the emergency department with aSAH and (2) determine the extent to which clinical presentation in the emergency department is associated with severity of injury and outcomes.

Methods

This study was a secondary analysis of data from 2 large prospective studies of aSAH patients (NIH R01HL074316 and NIH R01NR004339). Adult aSAH patients with a Fisher grade greater than 1 who were admitted to the neurovascular intensive care unit (ICU) of a level 1 trauma center from March 2003 through September 2008 were recruited for the studies. The Fisher grade is a 5-point scale specifically used to determine the amount of blood on initial computed tomography (CT) scan in patients with subarachnoid hemorrhage (0, no CT scan; 1, no blood detected on CT scan; 2, diffuse or vertical layers blood <1 mm thick; 3, localized clot and/or vertical layers blood >1 mm thick; and 4, diffuse or no subarachnoid hemorrhage but with intraventricular or intraparenchymal clot).^{6,7} Patients were excluded if they had a chronic degenerative neurologic disease (ie, multiple sclerosis or myasthenia gravis) or a hemorrhage resulting from trauma or mycotic aneurysm. Informed consent was obtained for all patients based on institutional review board protocol. All patients received standard nursing and medical care in the neurovascular ICU.

KEY VARIABLES

Patient demographics and comorbidities were obtained from the patients or their families or the medical records. Patients or families were queried regarding the patients' medical history, specifically regarding the presence or absence of cardiovascular disease, neurologic problems, pulmonary disease, hypertension, renal insufficiency, diabetes, cancer, drug and/

or alcohol use, and gastrointestinal problems. Smoking history was recorded as none (including those who quit) or in packs per day. Patient height and weight were obtained to calculate body mass index (BMI), which was used to evaluate obesity (underweight [BMI <18.5], overweight [BMI >25], obese [BMI >30]).⁸ Family history of cerebral aneurysm and menopausal status (for female patients) was also attained. Patients and their representatives were questioned or their records were reviewed regarding onset of symptoms, time of initial presentation to an emergency department, and arrival at the tertiary hospital. If the exact hour for symptom onset was not known (11% of patients described in this article), early morning was rounded to 6 am, midday to noon, early evening to 6 pm, and late night to midnight.

Symptoms were recorded in several ways. First, the presence or absence of a series of symptoms was recorded (headache, stiff neck; syncope, weakness, or confusion; nausea, vomiting; vision or speech deficits; seizure; unresponsive period; or other). Then, the patients or their representatives were asked to choose the one symptom that was the most significant in their seeking medical care. Medication taken to alleviate symptoms before arrival in the emergency department was documented as well as improvement or worsening of symptoms between facilities.

Clinical data including heart rate, blood pressure, temperature, and respiratory rate were gathered from the triage data in the ED record. Initial glucose and troponin I levels determined in the emergency department or upon admission to the tertiary center were documented.⁹ Initial electrocardiographic reports were reviewed and categorized into 3 specific variables by interpretation (within normal limits, arrhythmia, QT abnormalities, ST-segment abnormalities, or injury pattern), ischemia, and rhythm.

Severity of injury, according to Hunt and Hess (HH) criteria and Fisher score, were evaluated and graded by the admitting neurosurgeon. The HH score evaluates aSAH by severity of symptom findings (0, unruptured aneurysm; 1, symptomatic, mild headache or slight nuchal rigidity; 2, moderate to severe headache with nuchal rigidity and no neurologic deficit; 3, other than cranial nerve palsy, drowsy, confused; 4, stupor, moderate to severe hemiparesis, and early decerebrate posturing; and 5, deep coma with decerebrate posturing or moribund appearance).^{10,11} The Glasgow Coma Score (GCS) was obtained from the ED record before any intervention. Outcome measures including ICU and hospital length of stay (LOS) as well as death were collected from hospital and research records.

STATISTICS

By use of SPSS software, version 16.0 (SPSS, Chicago, IL), exploratory analyses including sociodemographic, clinical, and symptom characteristics were completed. Differences in demographic factors, clinical factors, physiologic responses, and symptom characteristics by HH score, Fisher grade, and responsiveness were assessed by use of the 2-sided Fisher exact test for categorical variables (or Pearson χ^2 exact test where appropriate). The Mann-Whitney *U* test (or Kruskal-Wallis test where appropriate) was used for continuous variables. Differences in LOS by symptoms were assessed by use of independent *t* tests after exclusion of deceased patients. All survival analyses were conducted by use of STATA/SE software, version 10.1 (StataCorp, College Station, TX). Because there was no significant difference in survival between 30 days after injury and 90 days after injury, survival time was defined as the time from the date of injury to date of death, with 30 days as the last observation period. To identify significant associations between covariates and survival, Kaplan-Meier curves, the log-rank test of equality across strata for all categorical predictors, and univariate Cox proportional hazard regression models for continuous covariates were used. A multivariate Cox proportional hazard regression model was built controlling for predictors with a *P* < .10 by use of a backward elimination procedure. Graphical plots of

Schoenfeld and scaled Schoenfeld residuals, as well as tests of proportionality for the overall model and individual covariates, were used to verify the proportional hazards assumption. Goodness of fit of the final model was assessed by use of Cox-Snell residuals and a graphical plot of the Nelson-Aalen cumulative hazard function.

Results

Findings are reported on a total of 193 adult aSAH patients aged 24 to 75 years (mean, 52.2 years; SD, 10.8). The majority of subjects were women (71.5% [n = 138]) and white (88% [n = 169]). Among the patients reporting a pre-existing history (71.5%), cardiovascular disease (43% [n = 83]) and hypertension (38% [n = 74]) were most common, and most women (47%) reported having gone through menopause. Patients in this group were also often overweight or obese by BMI (61% [n = 117]) and smokers at the time of diagnosis (60% [n = 116]) (Table 1).

Most of the sample was first examined at a community emergency department (89% [n = 169]) and referred to the tertiary care hospital. Patients were more likely to arrive at the community emergency department by ambulance (64% [n = 108]). Although there was a wide range of time (1.25-93.2 hours), 43% of patients (n = 73) arrived at the community emergency department between 10 and 15 hours after onset of symptoms. Once in the community emergency department, patients averaged 7 hours (SD, 8.3 hours) for diagnosis and disposition, with an overall time from the onset of symptoms to arrival at the tertiary hospital of 27.3 hours (SD, 18.8 hours) (Table 1).

Headache was the most common symptom reported (83% [n = 160]). Other frequently reported symptoms included nausea (42% [n = 81]), vomiting (43% [n = 83]), and changes in LOC (32% [n = 62]). The symptom most reported as the reason for seeking medical care was headache and stiff neck (50% [n = 96]), followed by a change in LOC (24% [n = 46]) (Table 2). Headache was often accompanied by other meningeal signs including stiff neck, nausea, and blurred vision ($r = 0.22$, $P < .01$). Few patients (26% [n = 51]) attempted to take any medications to alleviate their symptoms before seeking treatment. Interestingly, the majority of patients (58% [n = 112]) did not notice any change in symptoms between presentation to the emergency department and arrival at the tertiary care hospital; in fact, 31% (n = 59) experienced worsening of symptoms during this time period.

Most patients in this analysis arrived at the emergency department without significant neurologic deficits as manifested by GCS of 15 (n = 107 [55%]) and HH score lower than 3 (n = 36 [39%]). Because of the requirements for the parent study, all aSAH patients in this analysis had blood on CT scan; a Fisher grade of 3 was most common (n = 87 [45%]). Mean systolic blood pressure (mean, 154 mm Hg; SD, 27.3 mm Hg), glucose level (mean, 142 mg/dL; SD, 44.4 mg/dL), and troponin I level (mean, 0.74 ng/mL; SD, 3.4 ng/mL) were outside of accepted standards (Table 1). In addition, in patients with 12-lead electrocardiograms completed, readings were within normal limits in fewer than half (n = 63 [43%]) (Table 3).

Severity of injury was associated with both symptoms and presentation and was most strongly associated with a main complaint of change in LOC (HH, $P < .01$; Fisher, $P = .03$) as well as GCS (HH, $P < .01$; Fisher, $P < .01$). There was a significant association between HH score and complaints of headache ($P < .01$) and stiff neck ($P = .02$), with patients with lower HH scores (<3) reporting these symptoms more frequently (Table 2). Although vital signs did not differ based on severity of injury, patients with both higher HH scores and higher Fisher scores had significantly higher glucose levels ($P < .01$ and $P = .04$, respectively) than those with lower scores. In addition, higher HH scores were also associated with higher troponin I levels ($P < .01$) (Table 2).

Because 25% of these patients had a change in LOC or presented to the emergency department as unresponsive, a comparison between groups was completed. Overall, patients with a change in LOC had higher glucose levels ($P < .01$), troponin I levels ($P < .01$), and severity-of-injury scores ($P = .02$) compared with those without LOC. Physiologically, only heart rate was significantly different between groups; patients with a change in LOC had a significantly lower heart rate than those without a change in LOC ($P = .01$). Of interest, patients with a change in LOC were less likely to have any past medical conditions ($P = .04$) (Table 1).

The majority of these patients ($n = 170$ [88%]) were alive 1 month after diagnosis. Of the patients who were alive at discharge, the mean LOS in the ICU was 13.1 days (SD, 7.1) and the mean overall hospital stay was 16.8 days (SD, 8.5) (Table 1). The only presenting symptoms associated with ICU LOS were headache ($P = .03$) and weakness ($P = .01$). Patients without headache or weakness on arrival to the emergency department had longer ICU LOS than those with reports of headache; however, this did not remain significant in total hospital LOS. No other symptoms were associated with either ICU or hospital LOS.

To evaluate the impact of presentation on survival, a univariate analysis using Kaplan-Meier curves for all of the categorical predictors along with log-rank tests of equality showed that patients who reported a loss of consciousness as their reason for seeking medical attention had the lowest probability of survival over time ($P = .04$). Patients with neurologic deficit (HH score > 2) also had a higher probability of death ($P < .01$). For continuous predictors, a univariate Cox proportional hazards analysis was run, which did not show any significant findings. To build the model, we looked at significant predictors of symptom for seeking medical care, HH score, Fisher score, GCS, cardiac rhythm, and medical history of neurologic problem (other than the exclusion criteria), and we eliminated those that were not significant to the model. The final model showed that patients with an HH score greater than 2 who also had sinus bradycardia (hazard ratio [HR], 13.9; confidence interval [CI], 5-39.1), sinus tachycardia (HR, 5.1; CI, 1-19.53), or a history of neurologic complaints (HR, 5.3; CI, 1.5-19.1) were more likely to die. Finally, the interaction between neurologic deficits (HH) and cardiac rhythm was evaluated, which suggested that patients with both an HH score greater than 2 and sinus bradycardia (heart rate < 60 beats/min) were 15.6 times more likely to die within 30 days than those with no clinical deficits (HH score ≤ 2) and patients with normal sinus rhythm or other arrhythmia (Figure 1).

Discussion

These findings are consistent with the ED presentation of aSAH as a sudden, severe headache accompanied by other meningeal signs. Comparable to reports by Fontanarosa,¹² this analysis found the same top 3 symptoms, reporting an even higher percentage of patients with headache and slightly lower percentage with nausea or vomiting and change in LOC. Patients without neurologic deficit had more symptoms than those with neurologic deficit, potentially reflecting the patient's cognitive ability to recognize or remember his or her symptoms at presentation. This study did identify overall elevations in indicators of physiologic stress response such as blood pressure, troponin I, and glucose and also found that these correlated with severity of injury. In general, patients with aSAH present with relatively normal vital signs; however, a wide range of variations may be seen, as indicated by the minimum and maximum levels in this analysis. Guidelines have been written by a joint task force of the American Heart Association and the American Stroke Association to handle the needs of these complex patients and to guide the evaluation of patients with headaches and other symptoms of subarachnoid hemorrhage.¹ As expected, this study showed high levels of female patients and smokers and relatively few comorbidities because patients with aSAH are often relatively healthy individuals before their acute event.

It would appear that the majority of patients recognize the severity of the situation as demonstrated by the number of persons arriving at the emergency department by ambulance as opposed to private automobile; however, the relatively long period of time between onset of symptoms and presentation to the emergency department warrants additional investigation. Although there has been an improvement in the recognition of symptoms and time-sensitive treatment of cardiovascular problems such as acute chest pain, this analysis emphasizes the need for a similar effort to advance community awareness of the symptoms of neurologic compromise such as those seen in acute ischemic or hemorrhagic stroke. In addition, because patients with aSAH are more likely to present to a community emergency department, it is important for all emergency nurses to possess the ability to assess these patients and initiate treatment.

The number of symptoms on presentation did not impact LOS in this group; in fact, patients who reported more symptoms often had a shorter LOS. This may be attributed to the more alert patient's ability to recall symptoms at the time of onset. Physiologically, heart rate was significantly different in patients with and without LOC changes and was an indicator of significantly poorer outcomes at 30 days. Finally, in the survival analysis, patients in this study who exhibited heart rates lower than 60 beats/min and had clinically visible neurologic impairments were 15.6 times more likely to die within 1 month of diagnosis. Further investigation is needed to determine whether patient care in the emergency department or treatment during hospitalization could improve clinical outcomes.

Implications for Emergency Nurses

Although aSAH can be a devastating neurologic insult, the majority of patients may not have significant neurologic impairments during initial presentation in the emergency department. Emergency care personnel are often more attuned to patients with physical impairments such as those seen during more severe ischemic neurologic events, whereas general pain complaints such as headache and nausea may be overlooked or misdiagnosed. Failure to appreciate these neurologic symptoms by patients and their families may also lead to a delay in seeking care. As advocates to the community, emergency nurses are positioned to heighten awareness of hemorrhagic stroke as has been done for ischemic stroke.

Although there is a strong suggestion here that symptoms may predict outcome from aneurysm rupture, the overall goal of emergency care is diagnosis, stabilization, and disposition to the appropriate level of care. One-third of the patients in this report continued to worsen after presentation to the emergency department, which warrants frequent assessment and potential intervention by the emergency nurse. Working collaboratively with the rest of the ED health care team, the nurse is an excellent advocate for the patient with aSAH from the time of triage until disposition by recognizing the possible differential diagnosis, preventing or recognizing demise, and ultimately, improving time from symptoms to treatment.

Limitations

There is a bias in severity of injury because patients in this sample included only those with spontaneous aSAH who had blood on CT scan. In addition, the area from which these patients were recruited does not have a wide ethnic background and benefits from a relatively short mileage to a tertiary center and a well-developed air transport system, making it possible for patients to be moved to a higher level of care once the diagnosis has been determined. Finally, symptom reporting is subjective, and a variety of sources were used to gather these data, including patients, families, and records.

Conclusions

In a time when quick recognition and disposition are not only possible but preferable, it is important to note that there is evidence to support that patients who presented to a tertiary center with aSAH were first seen in the community ED setting and rapidly transported to a higher level of care. Additional effort is still needed to help patients and families to recognize the potential severity of symptoms at the time of onset and to promptly seek care. Because nurses have the most contact with patients and families, they are uniquely positioned to serve as caregivers and educators to facilitate rapid treatment of patients with aSAH.

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FIGURE 1.
Kaplan-Meier survival estimates by HH scale and rhythm.

TABLE 1

Sociodemographic and clinical characteristics

Characteristic	Total	Responsive	Unresponsive
Male [n (%)]	55 (28.5)	42 (28.6)	13 (28.3)
Female [n (%)]	138 (71.5)	105 (71.4)	33 (71.7)
Race [n (%)]			
White	169 (87.6)	130 (88.4)	39 (84.8)
Not white	24 (12.4)	17 (11.6)	7 (15.2)
HH score [n (%)]			
Low (1-2)	76 (39.4)	67 (45.6)	9 (19.6) ^a
High (3-5)	117 (60.6)	80 (54.4)	37 (80.4)
Fisher grade [n (%)]			
2	70 (36.3)	56 (38.1)	14 (30.4) ^b
3	87 (45.1)	70 (47.6)	17 (37.0)
4	36 (18.7)	21 (14.3)	15 (32.6)
Family history of aneurysm [n (%)]			
Yes	30 (19.2)	19 (15.8)	11 (30.6)
No	126 (80.8)	101 (84.2)	25 (69.4)
Smoking status [n (%)]			
Not regular smoker	77 (39.9)	56 (38.1)	21 (45.7)
Regular smoker	116 (60.1)	91 (61.9)	25 (54.3)
BMI [n (%)]			
Underweight	8 (4.1)	5 (3.4)	3 (6.5)
Normal	68 (35.2)	53 (36.1)	15 (32.6)
Overweight	63 (32.6)	47 (32.0)	16 (34.8)
Obese	54 (28.0)	42 (28.6)	12 (26.1)
Menopausal status [n (%)]			
Yes	65 (47.4)	46 (44.7)	19 (55.9)
No	61 (44.5)	50 (48.5)	11 (32.4)
Unknown	11 (8.0)	7 (6.8)	4 (11.8)
Medical history [n (%)]			
No medical history	55 (28.5)	36 (24.5)	19 (41.3) ^b
Cardiac	17 (8.8)	12 (8.2)	5 (10.9)
Hypertension	74 (38.3)	61 (41.5)	13 (28.3)
Pulmonary	18 (9.3)	10 (6.8)	8 (17.4)
Neurologic	9 (4.7)	6 (4.1)	3 (6.5)
Cancer	13 (6.7)	12 (8.2)	1 (2.2)
DM	10 (5.2)	6 (4.1)	4 (8.7)
Drug abuse	11 (5.7)	8 (5.4)	3 (6.5)
ETOH abuse	17 (8.8)	15 (10.2)	2 (4.3)
GI	13 (6.7)	9 (6.1)	4 (8.7)

Characteristic	Total	Responsive	Unresponsive
Other	52 (26.9)	44 (29.9)	8 (17.4)
Glasgow Coma Scale [n (%)]			
Severe disability (3-8)	24 (12.4)		
Moderate disability (9-12)	17 (8.8)		
Mild (13-15)	152 (78.8)		
Alive at 1 mo [n (%)]	170 (88.1)		
Age [mean (SD)] (y)	52.2 (10.8)	52.1 (10.3)	52.3 (12.3)
Comorbidity [mean (SD)]	1.4 (1.5)		
ICU LOS [mean (SD)] (d)	13.1 (7.1)		
Hospital LOS [mean (SD)] (d)	16.8 (8.5)		
Time from onset to emergency department [mean (SD)] (h) ^c	20.9 (15.4)		
Time from onset to tertiary [mean (SD)] (h) ^d	27.3 (18.8)		
Time from community emergency department to tertiary [mean (SD)] (h) ^d	7.0 (8.3)		
Physiologic responses [mean (SD)]			
Heart rate	81.59 (16.21)	79.9 (13.8)	85.1 (20.0) ^b
Systolic blood pressure	153.52 (27.28)	152.3 (26.3)	156.2 (29.2)
Diastolic blood pressure	86.35 (18.27)	85.1 (17.0)	89.1 (20.6)
Respiratory rate	18.13 (3.42)	17.7 (3.4)	19.1 (3.3)
Temperature (°C)	36.53 (0.67)	36.5 (0.7)	36.6 (0.7)
SaO ₂	97.53 (2.5)	97.5 (2.5)	97.6 (2.7)
Glucose (mg/dL)	142.3 (44.4)	140.2 (48.0)	146.8 (35.5) ^a
Troponin	0.7 (3.4)	0.7 (3.9)	0.8 (1.9) ^a

^a $P < .01$.

^b $P < .05$.

^c N = 193, includes 24 patients who did not go to an outside hospital.

^d n = 169, includes patients who went to outside hospital first.

TABLE 2

Symptoms and physiologic responses

Characteristics	Total response	HH score		Fisher grade			
		Low (1-2)	High (3-5)	2	3	4	
Symptoms [n (%)]							
Headache	160 (82.9)	71 (93.4)	89 (76.1) ^a	60 (85.7)	75 (86.2)	25 (69.4)	
Blurred/double vision	28 (14.5)	14 (18.4)	14 (12.1)	11 (15.9)	16 (18.4)	1 (2.8)	
Confusion	29 (15.0)	7 (9.2)	22 (19)	8 (11.4)	14 (16.3)	7 (19.4)	
Trouble speaking (dysarthria)	32 (16.6)	8 (10.5)	24 (20.5)	6 (8.6)	18 (20.7)	8 (22.2)	
Weakness	51 (26.4)	17 (22.4)	34 (29.1)	18 (25.7)	20 (23)	13 (36.1)	
Nausea	81 (42.0)	47 (61.8)	34 (29.1) ^a	33 (47.1)	36 (41.4)	12 (33.3)	
Vomiting	83 (43.0)	34 (44.7)	49 (41.9)	23 (32.9)	43 (49.4)	17 (47.2)	
Stiff neck	53 (27.5)	28 (36.8)	25 (21.4) ^b	22 (31.4)	24 (27.6)	7 (19.4)	
Seizure	24 (12.4)	5 (6.6)	19 (16.2)	7 (10.0)	11 (12.6)	6 (16.7)	
Unresponsive	62 (32.1)	16 (21.1)	46 (39.3) ^b	20 (28.6)	28 (32.2)	14 (38.9)	
Syncope (dizzy)	31 (16.1)	12 (15.8)	19 (16.2)	10 (14.3)	16 (18.4)	5 (13.9)	
Other	26 (13.5)	13 (17.1)	13 (11.1)	12 (17.1)	9 (10.3)	5 (13.9)	
Symptom choice [n (%)]							
Headache/stiff neck	96 (49.7)	56 (73.7)	40 (34.2)	40 (57.1)	47 (54)	9 (25)	
Syncope/weakness/confusion	29 (15.0)	8 (10.5)	21 (17.9)	9 (12.9)	13 (14.9)	7 (19.4)	
Nausea/vomiting	2 (1.0)	0 (0)	2 (1.7)	1 (1.4)	1 (1.1)	0 (0)	
Vision/speech	4 (2.1)	1 (1.3)	3 (2.6)	1 (1.4)	2 (2.3)	1 (2.8)	
Seizure	13 (6.7)	2 (2.6)	11 (9.4)	4 (5.7)	7 (8)	2 (5.6)	
Other	3 (1.6)	0 (0)	3 (2.6)	11.4	0 (0)	2 (5.6)	
Unresponsive	46 (23.8)	9 (11.8)	37 (31.6) ^a	14 (20)	17 (19.5)	15 (41.7)	
Physiologic responses [mean (SD)]							
Heart rate		79.0 (12.3)	83.3 (18.2)	82.2 (12.9)	81.3 (17.5)	81.0 (19.1)	
Blood pressure							
Systolic		152.5 (24.5)	154.2 (29.0)	151.4 (29.8)	155.3 (27.5)	153.3 (21.5)	
Diastolic		84.5 (16.5)	87.6 (19.3)	86.5 (16.3)	85.7 (20.0)	87.5 (17.9)	

Characteristics	Total response	HH score		Fisher grade			
		Low (1-2)	High (3-5)	2	3	4	
Respiratory rate		18.3 (2.6)	18.0 (3.9)	18.0 (3.2)	18.0 (3.3)	18.7 (4.3)	
Temperature (°C)		36.6 (0.5)	36.5 (0.8)	36.6 (0.7)	36.5 (0.7)	36.5 (0.6)	
SaO ₂		97.6 (2.1)	97.5 (2.8)	97.4 (2.7)	97.3 (2.6)	98.4 (1.8)	
Glucose (mg/dL)		128.3 (40.0)	151.4 (44.9) ^a	133.2 (42.2)	147.5 (46.1)	147.7 (42.6) ^b	
Troponin		0.2 (0.5)	1.1 (4.3) ^a	0.4 (0.8)	0.5 (1.4)	2.0 (7.3)	

^a $p < .01$.

^b $p < .05$.

TABLE 3

Initial electrocardiographic findings

Characteristic	N	%
Interpretation		
Within normal limits	63	42.86
Arrhythmia	3	2.04
QT changes	17	11.56
ST changes	12	8.16
Injury pattern	52	35.37
Ischemia		
None	80	74.1
Present	28	25.9
Rhythm		
Normal sinus	118	71.08
Sinus bradycardia	17	10.24
Sinus tachycardia	17	10.24
Arrhythmia	14	8.43