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Clinical Presentation to the Emergency Department Predicts Subarachnoid Hemorrhage-Associated Myocardial Injury

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Abstract

Introduction—Aneurysmal subarachnoid hemorrhage is frequently seen in emergency departments. Secondary injury such as subarachnoid hemorrhage-associated myocardial injury (SAHMI) affects one-third of survivors and contributes to poor outcomes. SAHMI is not attributed to ischemia from myocardial disease but can result in hypotension and arrhythmias. It is important that emergency nurses recognize which clinical presentation characteristics are predictive of SAHMI to initiate proper interventions. The aim of this study was to determine whether clinical presentation of aSAH patients to the emergency department predict SAHMI as defined by troponin I 0.3ng/ml.

Methods—This was a prospective descriptive study. SAHMI was defined as troponin I 0.3ng/ml. Predictors included: demographics and clinical characteristics, injury severity, admission 12-lead ECG, initial emergency department vital signs, and pre-hospital symptoms at time of aneurysm rupture.

Results—Of 449 patients, 126 (28%) had SAHMI. Patients with SAHMI were more likely to report seizures and unresponsiveness, with significantly lower Glasgow coma score, and higher proportion of Hunt and Hess grades 3-5 and Fisher grades III and IV (all $p < .05$). SAHMI patients had higher atrial and ventricular rates and longer QTc interval on initial ECG ($p < .05$). On multivariable logistic regression, poor Hunt and Hess grade, report of prehospital unresponsiveness, lower admission Glasgow coma score, and longer QTc interval were significantly and independently predictive of SAHMI ($p < .05$).

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Discussion—Components of the clinical presentation of subarachnoid hemorrhage to the emergency department predict SAHMI. Identifying SAHMI patients in the emergency department can be helpful in determining surveillance and care needs, and informing transfer unit care.

Keywords

subarachnoid hemorrhage; myocardial injury; neurocardiac injury; QTc interval

Introduction

Aneurysmal subarachnoid hemorrhage (aSAH) is a major form of hemorrhagic stroke that is commonly encountered in the emergency department. Up to 15% of patients die before they reach the emergency department.¹ Survivors of aSAH are left with functional and cognitive disability due to the initial bleeding as well as to secondary complications.^{2,3} While common neurologic complications include delayed cerebral ischemia,⁴ vasospasm,⁵ and hydrocephalus,⁶ patients also frequently display cardiovascular complications including hypotension with need for hemodynamic support,^{7,8} arrhythmias,⁹ myocardial wall motion abnormalities with depressed cardiac output and ejection fraction, and elevated cardiac troponin level.¹⁰ These neurologic and cardiac complications are not independent of each other. The brain-heart interaction is well recognized after neurological insults, particularly after aSAH, and is thought to be catecholamine mediated.¹¹ Subarachnoid hemorrhage-associated myocardial injury (SAHMI) is type of neurocardiac injury that involves myocardial dysfunction associated with subarachnoid bleed. Approximately, 1/3 of aSAH patients experience SAHMI.¹²⁻¹⁵ SAHMI is partly attributed to sudden sympathetic activation at the time of aneurysm rupture which causes a rapid and sustained increase in serum catecholamine levels directly damaging the heart and resulting in myocardial contraction band necrosis. Importantly it is not associated with myocardial ischemia.^{11,16} Nevertheless, the subsequent myocardial cellular damage is manifested by systemic hypoperfusion, electrocardiographic (ECG) changes, elevated cardiac enzymes, and wall motion abnormalities.¹¹ Although most of these abnormalities reverse over time, SAHMI has been associated with poor outcomes and death in these patients.¹⁷

Since SAHMI occurs as a consequence of the aneurysm rupture, early detection of symptoms in the emergency department is essential. The reasons for this are twofold. First, it must be recognized that these symptoms are attributed to aneurysm rupture, and not a primary acute coronary syndrome due to ischemic cause. Secondly, recognizing this complication in aSAH patients permits the emergency nurse to anticipate and apply supportive care due to the myocardial dysfunction, as well as alert the staff of the transfer unit to be vigilant for further SAHMI manifestations. The aim of this study was to determine which aspects of the initial clinical presentation of aSAH patients to the emergency department predicted SAHMI as defined by cardiac troponin I ≥ 0.3 ng/ml.

Methods

This was a prospective descriptive study (R01HL074316 and R01NR014221) of patients with spontaneous aSAH. The local institutional review board approved this study, and all

patients received a detailed verbal and written explanation about the study before they provided consent.

Patients and Setting

Eligible patients were those with age 21-75 years, spontaneous aneurysm rupture, Fisher grade >1 and/or Hunt and Hess (HH) grade >2, and available cardiac troponin I (cTnI) levels. Exclusion criteria included SAH from mycotic aneurysm or trauma, the history of myocardial injury (<1 year), or chronic debilitating neurological disease. The diagnosis of aSAH was confirmed by computerized tomography (CT) scan and/or digital subtraction angiography. Patients were admitted to the neurovascular intensive care unit in a regional comprehensive stroke and level I trauma center in Western Pennsylvania between March 2003 and August 2015.

Subarachnoid Hemorrhage Associated Myocardial Injury (SAHMI)

SAHMI was defined as peak cTnI ≥ 0.3 ng/ml. cTnI levels were obtained at least daily for the first 5 days post bleeding, and measured using the Beckman Coulter Access AccuTnI assay (Beckman Instruments Inc., Chaska, MN).

Clinical Presentation

Age, sex, race, and past cardiac medical history were collected from the patients, families, and/or the medical records. Aneurysm site was determined by the attending neurosurgeon using digital subtraction angiography, and further categorized as anterior or posterior circulation aneurysms for analysis. Injury severity was evaluated by Glasgow coma scale (GCS), Fisher grade, and Hunt and Hess (HH) grade. Initial GCS was extracted from the emergency department records. Fisher and HH grades were determined by the attending neurosurgeon based on the patient's initial clinical exam (HH grade) and the amount of cerebral blood on the first CT scan (Fisher grade). Admission HH score was further categorized as good (HH grade 0-2) or poor (HH grade 3-5) for analysis. Patients, surrogates and medical records were interrogated for pre-hospital symptoms including headache, unresponsiveness, and seizures.

The admission 12-lead ECG was evaluated for ventricular rate, atrial rate, PR interval, QRS duration, and QTc interval by a cardiologist. QT interval was corrected for heart rate using Bazett's equation (QTcB). Initial vital signs were extracted from emergency department records including heart rate, temperature, respiratory rate, pulse oximetry (SpO₂), and blood pressure (non-invasive).

Statistical Analyses

Statistical analyses were conducted using IBM SPSS 24. Sample description was completed using frequency distribution for categorical variables and mean $\pm$ standard deviation for continuous variables. Univariate group comparison was performed using chi square statistics for categorical variables and student's *t*-test or Mann-Whitney U statistics for continuous variables. Mann-Whitney U test was used when the assumptions of student's *t*-test were violated. When the results of Mann-Whitney U test were similar to *t*-test, *t*-test statistics were reported. Prediction analytics in the form of multivariable analyses were performed

using binary logistic regression as a function of the probability for a patient to have SAHMI after aSAH. Predictors with a p value <0.2 were entered in the multivariable logistic regression model. Predictors with the greatest p value were excluded one by one on a serial fashion until all the predictors in the model were statistically significant (p values $<.05$). Due to multicollinearity, HH grade, unresponsiveness, and admission GCS were used in different models.

Results

Of all 449 patients included in the study, the mean age was 53 ± 11 years, with a majority female (328, 73%), and Caucasian (393, 88%). The mean admission GCS was 13 ± 4 , with a poor HH grade (245, 55%). Most aneurysms were eventually obliterated with endovascular coiling (301, 67%). The most reported initial symptoms of patients in this analysis included headache 321 (86%), seizure 41 (11%), and unresponsiveness 125 (33%).

Of the total sample, 126 patients (28%) had SAHMI. The clinical presentation characteristics of patients with and without SAHMI are shown in Table 1. There were no between-group differences in age, sex, race, prior cardiac history, or aneurysm site or treatment. Patients with SAHMI had significantly lower GCS (11.0 ± 4.2 vs. 13.5 ± 2.8) score, greater proportion of poor HH grade (81% vs. 44%), and Fisher grade III (47% vs. 37%) and IV (24% vs. 13%); [all $p < .0001$] than those with no SAHMI. In addition, patients with SAHMI displayed a greater proportion of seizure (17% vs. 9%) and unresponsiveness (52% vs. 26%) and less report of headache (76% vs. 90%) compared to those with no SAHMI; all $p < .05$. On the admission 12-lead ECG (Table 2), SAHMI patients had significantly higher ventricular rate (81.2 ± 19.4 vs. 74.3 ± 17.1) and atrial rate (81.4 ± 19.5 vs. 75.5 ± 17.0), and longer QTcB interval (474.8 ± 44.5 vs. 453.2 ± 33.8), [all $p < .05$] as compared to no SAHMI patients respectively. SAHMI patients also presented to the emergency department with a higher heart rate (83.6 ± 18.7 vs. 77.5 ± 16.7), and slightly lower systolic blood pressure (median and minimum-maximum: SAHMI 144 mmHg (90-252) vs no SAHMI 148 mmHg (92-240), $p = 0.049$).

On binomial multivariable logistic regression (Table 3), poor HH grade and GCS, as well as a report of unresponsiveness and prolonged QTcB interval were significant and independent predictors of SAHMI. Patients with poor HH grade and unresponsiveness at the time of bleeding had a 4.01 and 3.04-fold (respectively) increased odds of SAHMI compared to those with good HH grade and those who did not have unresponsiveness at the time of bleeding ($p < .01$). For every one unit decrease in admission GCS, the odds of SAHMI increased by 17% ($p < .001$). For every 1 milliseconds increase in QTcB interval the odds of SAHMI increased by 1% ($p < .001$).

Discussion

This analysis sought to explore if the clinical characteristics of aSAH patients admitted to the emergency department predicted SAHMI for the purpose of informing emergency nurses of the likelihood of this post-aSAH complication. Our findings indicate that clinical presentation and neurological exam in the emergency department can be a useful

prognosticating tool for SAHMI. The severity of neurological symptoms/exam at the time of bleeding or on initial presentation are strong and independent predictors of SAHMI. The worse the symptoms observed at the time of bleed, the greater the odds for those patients to develop SAHMI. Patients whose prehospital symptoms included unresponsive at the time of bleeding, those with poorer HH grades and/or lower GCS score in the emergency department, or longer QTcB interval on the initial 12-lead ECG are at high risk for SAHMI. Our findings are useful for predicting as well as detecting SAHMI. While unresponsiveness, HH grade, and GCS score can be used to identify patients at high risk, prolonged QTcB can be used for detecting SAHMI at the bedside. Importantly, of all SAHMI patients, no one displayed any ECG changes consistent with ischemia. The absence of ischemic ECG changes is an important clinical clue to rule out a primary cardiac ischemic diagnosis as the cause for unresponsiveness. The prediction and early detection of SAHMI will potentially alert nurses to observe patients closely for other manifestations of SAHMI such as hypotension and arrhythmias, and prevent any delay in supportive treatment, which may in turn serve to improve outcomes in those patients. We recommend that bedside nurses vigilantly monitor aSAH patients with these characteristics.

Evidence suggests an association between SAHMI and World Federation of Neurological Societies (WFNS) score; which is a function of GCS score.¹¹ GCS, a commonly used neurologic grading criterion in Emergency Departments, and HH grade quantify the severity of symptoms that likely represents the degree of neurological damage. Evidence in the literature suggests that catecholamine levels are positively associated with the neurological status (WFNS) and poor clinical grade (HH) in patients with aSAH.^{11,18} The underlying mechanism is likely to be mediated through catecholamine release. The brainstem ischemia that is caused by elevated intracranial pressure at the time aneurysm rupture induces a sudden activation in the sympathetic nervous system which in turn leads to increased catecholamine levels.^{18,19} Our findings are also consistent with findings by others who demonstrated that HH grade and loss of consciousness at the time of bleeding are predictors of SAHMI,²⁰⁻²⁴ suggesting that patients with poor HH grade on admission and become unresponsive at the time of bleeding likely had higher levels neurologic stress and therefore greater catecholamine release, and thus greater susceptibility for SAHMI. Poor neurological exam on admission has been found to predict myocardial wall motion abnormalities.²⁵ These findings indicate that SAHMI is neurogenic in nature and is incrementally associated with the degree of neurological damage. Therefore, patients with low GCS, poor HH grade, a report of unconsciousness or seizure should be closely assessed and monitored for SAHMI. Interestingly, we found that the severity of symptoms measured by HH grade was a stronger SAHMI predictor than the amount of blood evident on the CT scan as measured by Fisher grade, and Malik et al. have reported similar findings.²¹

The finding that patients with SAHMI were more likely to display the symptom of unconsciousness or seizure rather than headache at the time of bleed was unexpected, since a description of “the worst headache ever” is taught as a clinical cue for aSAH. To our knowledge, this is the first study to report this relationship between headache and SAHMI as compared to other symptoms. We suspect that since SAHMI is associated with more severe injury, those patients were more likely to move directly to seizure or an unconscious state, precluding their ability to complain of a headache. Whereas patients without SAHMI had

less injury severity and lower GCS scores, thus were in a state of lighter consciousness and so able to notice and report a headache.

Prolonged QTcB interval and SAHMI has been reported in the literature; suggesting repolarization abnormalities in patients with elevated catecholamine and cTnI levels.²⁶ In this study, we found an association between SAHMI and prolonged QTcB independent of the initial neurological exam and severity of prehospital symptoms. We also identified a univariate association between SAHMI and a higher heart rate in the emergency department vital signs and the initial ECG. However, when controlling for HH grade this association was not maintained. Sustained elevated heart rate in the intensive care units was associated with SAHMI even after controlling for HH grade in other reports.²⁷.

Limitations

This study has several limitations in part due to patient recruitment over a 12-year period. This may have resulted in variations in standard care for this population over this time period including earlier and more appropriate treatment of aSAH and SAHMI, as well as changes assays measuring cTnI levels. HH grade is graded and assessed by members of the neurosurgery team and is subject to inter-rater variations in the given grade. Initial symptoms at time of bleed including headache, unresponsiveness, or seizure were obtained retrospectively from patient and family reports. While some symptoms may be easily noticed, or recalled by patients and communicated to their families, unresponsiveness and seizure may have not been precisely observed by the families, or the patients may have passed through a number a symptom phases before they became unresponsive. Lastly, the majority of our patients were Caucasian and female, this may threaten the external validity of this study but it represents the general demographic characteristics of the population in this area.

Implications to Emergency Nurses

Emergency nurses are often the first to encounter aSAH patients. It is important that emergency nurses understand the myocardial dysfunction associated with aSAH, and do not mistake its symptoms with other potential causes for unconsciousness, hypotension and arrhythmias such as myocardial ischemia, hypovolemia or electrolyte imbalances. Our findings emphasize that clinical presentation and neurological exam in the emergency department can be predictive of neurocardiac injury after aSAH. These findings can be very helpful for triaging aSAH patients in the emergency department as well as initiating proper and timely interventions.

Conclusion

This study confirms that select aspects of clinical presentation of aSAH in the emergency department can predict SAHMI. Patients who become unresponsive at the time of bleeding, those with poor HH grade, low GCS score, or long QTcB interval on admission should be vigilantly monitored for SAHMI through serial and frequent cTnI measurement as well as ECG and blood pressure surveillance. Further studies are warranted to test whether initiating serial troponin I measurement and continuous ECG surveillance for patients with poor

clinical presentation and neurological exam in the emergency department would result in early detection of SAHMI.

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References

1. Huang J, van Gelder JM. The probability of sudden death from rupture of intracranial aneurysms: a meta-analysis. *Neurosurgery*. 2002; 51(5):1101–1105. discussion 1105–1107. [PubMed: 12383354]
2. De Marchis GM, Pugin D, Meyers E, et al. Seizure burden in subarachnoid hemorrhage associated with functional and cognitive outcome. *Neurology*. 2015
3. Wong GK, Nung RC, Sitt JC, et al. Location, Infarct Load, and 3-Month Outcomes of Delayed Cerebral Infarction After Aneurysmal Subarachnoid Hemorrhage. *Stroke; a journal of cerebral circulation*. 2015; 46(11):3099–3104.
4. Yousef K, Crago E, Kuo CW, Horowitz M, Hravnak M. Predictors of delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage: a cardiac focus. *Neurocrit Care*. 2010; 13(3):366–372. [PubMed: 20645025]
5. Carlson AP, Yonas H. Radiographic assessment of vasospasm after aneurysmal subarachnoid hemorrhage: the physiological perspective. *Neurol Res*. 2009; 31(6):593–604. [PubMed: 19660189]
6. Bakker AM, Dorhout Mees SM, Algra A, Rinkel GJ. Extent of acute hydrocephalus after aneurysmal subarachnoid hemorrhage as a risk factor for delayed cerebral infarction. *Stroke*. 2007; 38(9):2496–2499. [PubMed: 17673710]
7. Marshman LA. Cushing's 'variant' response (acute hypotension) after subarachnoid hemorrhage. Association with moderate intracranial tensions and subacute cardiovascular collapse. *Stroke*. 1997; 28(7):1445–1450. [PubMed: 9227698]
8. Dankbaar JW, Slooter AJ, Rinkel GJ, Schaaf IC. Effect of different components of triple-H therapy on cerebral perfusion in patients with aneurysmal subarachnoid haemorrhage: a systematic review. *Critical care*. 2010; 14(1):R23. [PubMed: 20175912]
9. Frangiskakis JM, Hravnak M, Crago EA, et al. Ventricular arrhythmia risk after subarachnoid hemorrhage. *Neurocritical Care*. 2009; 10(3):287–294. [PubMed: 19184553]
10. Hravnak M, Frangiskakis JM, Crago EA, et al. Elevated cardiac troponin I and relationship to persistence of electrocardiographic and echocardiographic abnormalities after aneurysmal subarachnoid hemorrhage. *Stroke*. 2009; 40(11):3478–3484. [PubMed: 19713541]
11. Salem R, Vallee F, Depret F, et al. Subarachnoid hemorrhage induces an early and reversible cardiac injury associated with catecholamine release: one-week follow-up study. *Crit Care*. 2014; 18(5):558. [PubMed: 25358417]
12. Tanabe M, Crago EA, Suffoletto MS, et al. Relation of elevation in cardiac troponin I to clinical severity, cardiac dysfunction, and pulmonary congestion in patients with subarachnoid hemorrhage. *The American journal of cardiology*. 2008; 102(11):1545–1550. [PubMed: 19026312]
13. Frangiskakis JM, Hravnak M, Crago EA, et al. Ventricular arrhythmia risk after subarachnoid hemorrhage. *Neurocritical care*. 2009; 10(3):287–294. [PubMed: 19184553]
14. Yarlaga S, Rajendran P, Miss JC, et al. Cardiovascular predictors of in-patient mortality after subarachnoid hemorrhage. *Neurocritical care*. 2006; 5(2):102–107. [PubMed: 17099255]
15. Miketic JK, Hravnak M, Sereika SM, Crago EA. Elevated cardiac troponin I and functional recovery and disability in patients after aneurysmal subarachnoid hemorrhage. *American journal of critical care : an official publication, American Association of Critical-Care Nurses*. 2010; 19(6):522–528. quiz 529.
16. Kopelnik A, Zaroff JG. Neurocardiogenic injury in neurovascular disorders. *Critical care clinics*. 2006; 22(4):733–752. abstract ix-x. [PubMed: 17239752]

17. Hravnak M, Frangiskakis JM, Crago EA, et al. Elevated cardiac troponin I and relationship to persistence of electrocardiographic and echocardiographic abnormalities after aneurysmal subarachnoid hemorrhage. *Stroke*. 2009; 40(11):3478–3484. [PubMed: 19713541]
18. Ogura T, Satoh A, Ooigawa H, et al. Characteristics and prognostic value of acute catecholamine surge in patients with aneurysmal subarachnoid hemorrhage. *Neurol Res*. 2012; 34(5):484–490. [PubMed: 22664172]
19. van Loon J, Shivalkar B, Plets C, Goffin J, Tjandra-Maga TB, Flameng W. Catecholamine response to a gradual increase of intracranial pressure. *J Neurosurg*. 1993; 79(5):705–709. [PubMed: 8410249]
20. Tung P, Kopelnik A, Banki N, et al. Predictors of neurocardiogenic injury after subarachnoid hemorrhage. *Stroke; a journal of cerebral circulation*. 2004; 35(2):548–551.
21. Malik AN, Gross BA, Rosalind Lai PM, Moses ZB, Du R. Neurogenic Stress Cardiomyopathy After Aneurysmal Subarachnoid Hemorrhage. *World neurosurgery*. 2015; 83(6):880–885. [PubMed: 25655685]
22. Jeon IC, Chang CH, Choi BY, Kim MS, Kim SW, Kim SH. Cardiac troponin I elevation in patients with aneurysmal subarachnoid hemorrhage. *J Korean Neurosurg Soc*. 2009; 46(2):99–102. [PubMed: 19763210]
23. Naidech AM, Kreiter KT, Janjua N, et al. Cardiac troponin elevation, cardiovascular morbidity, and outcome after subarachnoid hemorrhage. *Circulation*. 2005; 112(18):2851–2856. [PubMed: 16267258]
24. Mavridis IN, Meliou M, Pyrgelis ES. Troponin elevation in subarachnoid hemorrhage. *Journal of Acute Disease*. 2015; 4(1):7–11.
25. van der Bilt IA, Hasan D, van den Brink RB, et al. Time course and risk factors for myocardial dysfunction after aneurysmal subarachnoid hemorrhage. *Neurosurgery*. 2015; 76(6):700–705. discussion 705-706. [PubMed: 25714519]
26. Sommargren CE, Zaroff JG, Banki N, Drew BJ. Electrocardiographic repolarization abnormalities in subarachnoid hemorrhage. *J Electrocardiol*. 2002; 35(Suppl):257–262. [PubMed: 12539136]
27. Schmidt JM, Crimmins M, Lantigua H, et al. Prolonged elevated heart rate is a risk factor for adverse cardiac events and poor outcome after subarachnoid hemorrhage. *Neurocritical care*. 2014; 20(3):390–398. [PubMed: 24043479]

Contribution to Emergency Nursing Practice

- We corroborate that unresponsiveness at the time of injury in the pre-hospital setting is associated with subarachnoid hemorrhage-associated myocardial injury.
- We report that poor Hunt and Hess grade, low Glasgow coma score and prolonged QTc interval in the emergency department can be predictive of subarachnoid hemorrhage-associated myocardial injury. These findings can be utilized in the emergency department to triage patients with subarachnoid hemorrhage.
- Proper management of subarachnoid hemorrhage-associated myocardial injury in the emergency department requires correct understanding of this phenomenon. We report that subarachnoid hemorrhage-associated myocardial injury is neurogenic in nature and is not related to primary cardiac pathology.

Table 1
Demographic and clinical presentation differences between patients with and without subarachnoid hemorrhage-associated myocardial injury (SAHMI)

	No SAHMI N=323	SAHMI N=126	<i>p</i> -value
Age (Mean years $\pm$ SD) *	53 $\pm$ 11	55 $\pm$ 11	.11
Sex [n (%)]			.82
Male	88 (27%)	33 (26%)	
Female	235 (73%)	93 (74%)	
Race [n (%)]			.24
White	279 (86%)	114 (91%)	
Other	44 (14%)	12 (10%)	
Past cardiac medical history [n (%)]	35 (11%)	15 (13%)	.66
Aneurysm Site [n (%)]			.58
Anterior Circulation	209 (65%)	78(62%)	
Posterior Circulation	114 (35%)	48(38%)	
Aneurysm treatment [n (%)]			.22
Surgical clipping	111 (34%)	35 (28%)	
Endovascular coiling	212 (66%)	89(72%)	
Admission Glasgow coma scale (Mean $\pm$ SD) *	13.5 $\pm$ 2.9	11.0 $\pm$ 4.2	<.0001
Hunt and Hess [n (%)]			<.0001
Good: Grade 0-2	180 (56%)	24 (19%)	
Poor: Grade 3-5	143 (44%)	102 (81%)	
Fisher Grade [n (%)]			<.0001
Grade II	162 (50%)	37 (29%)	
Grade III	120 (37%)	59 (47%)	
Grade IV	41 (13%)	30 (24%)	
Clinical presentation at time of bleeding	241(90%)	80(76%)	.001
Headache [n (%)]	23(9%)	18(17%)	.017
Seizure [n (%)]	70(26%)	55(52%)	<.001
Unresponsiveness [n (%)]			

Key:

* Student's *t*-test results are similar to Mann-Whitney U test

Table 2
Admission 12-lead electrocardiogram and vital signs in the emergency department.
Differences between patient with and without subarachnoid hemorrhage-associated myocardial injury (SAHMI)

	No SAHMI	SAHMI	<i>p</i> -value
Admission ECG (Mean±SD) *			
Ventricular rate (per minute)	N=274	N=118	.001
	74.3±17.1	81.2±19.4	
Atrial rate (per minute)	N=274	N=118	.001
	75.5±17.0	81.4±19.5	
PR interval (milliseconds)	N=267	N=114	.350
	153.9±26.3	151.2±24.0	
QRS duration (milliseconds)	N=273	N=118	.680
	88.6±11.4	89.2±15.2	
QTc (milliseconds)	N=273	N=118	<.001
	453.2±33.8	474.8±44.5	
Vital signs in the emergency department			
Heart rate (Mean±SD) *	N=319	N=119	.001
	77.5±16.7	83.6±18.7	
Respiratory rate (Mean±SD) *	N=319	N=119	.422
	18.1±3.3	18.1±4.0	
Temperature [Median(min-max)]	N=311	N=114	.055
	36.6 (33.9-38.5)	36.5(33.3-38.5)	
SpO ₂ (Mean±SD) *	N=308	N=118	.893
	97.8±2.5	97.5±3.1	
Systolic BP [Median(min-max)]	N=318	N=118	.049
	148(92-240)	144(90-252)	
Diastolic BP [Median(min-max)]	N=318	N=118	.723
	82(43-180)	81(42-148)	

Key:

* Student's *t*-test results are similar to Mann-Whitney U test

Table 3
Predictors of subarachnoid hemorrhage-associated myocardial injury (SAHMI) (n=329)

Variable	Odds ratio	95% confidence interval	P values
Poor Hunt and Hess grade ¹	4.01	2.37-6.78	<.001
Unresponsiveness ²	3.04	1.83-5.06	<.001
Admission Glasgow Coma Scale ³	.83	.78-.89	<.001
QTcB [*] interval	1.01	1.00-1.02	<.001

Due to multicollinearity, Hunt and Hess grade, unresponsiveness, and admission Glasgow coma scale were used in different models

¹ Specificity = 91%, sensitivity=29%

² Specificity = 89%, sensitivity=23%

³ Specificity = 91%, sensitivity=19%

^{*} electrocardiogram QT interval corrected for heart rate using Bazett's equation