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

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Contribution to Emergency Nursing Practice:

- Neurocardiac nursing assessment in the emergency department can be utilized to triage patients with subarachnoid hemorrhage.
- Emergency nurses need to be vigilant for cardiac complications in patient with unresponsiveness at the time of subarachnoid hemorrhage.
- Nurses are the first step in patient care. To provide patients with the best care possible, nurses need to be highly competent in recognizing alarming symptoms.

Absract

Introduction: Aneurysmal subarachnoid hemorrhage (aSAH) 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 patients who present to the emergency department with clinical aSAH are likely to develop SAHMI, as defined by troponin I ≥0.3 ng/mL.

Methods: This was a prospective descriptive study. SAHMI was defined as troponin I ≥0.3 ng/mL. Predictors included demographics and clinical characteristics, severity of injury, admission 12-lead electrogardiogram (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 to 5 and Fisher grades III and IV (all $P < .05$). Patients with SAHMI had higher atrial and ventricular rates and longer QTc intervals 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 ($P < .05$).

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

Key words: Subarachnoid hemorrhage; Myocardial injury; Neurocardiac injury; QTc interval

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Aneurysmal subarachnoid hemorrhage (aSAH) is a major form of hemorrhagic stroke 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] Although 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 neurologic 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 one third of patients with aSAH experience SAHMI.[9,12,13] 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.[11,14] Nevertheless, the subsequent myocardial cellular damage is manifested by systemic hypoperfusion, electrocardiographic 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.[10]

As 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 patients with aSAH permits the emergency nurse to anticipate and apply supportive care for the myocardial dysfunction as well as alert the staff of the transfer unit to be vigilant for further manifestations of SAHMI. The aim of this study was to determine which aspects of the initial clinical presentation of patients with aSAH 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 detailed verbal and written explanations about the study before they provided consent.

PATIENTS AND SETTING

Eligible patients were those aged 21 to 75 years, with 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 neurologic 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 at the University of Pittsburgh Medical Center 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 postbleeding 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. Severity of injury was evaluated by Glasgow coma scale (GCS), Fisher grade, and Hunt and Hess (HH) grade. Initial GCS was extracted from the ED records. Fisher and HH grades were determined by the attending neurosurgeon, based on the patient's initial clinical examination (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 prehospital 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 ED records including heart rate, temperature, respiratory rate, pulse oximetry (SpO_2), and blood pressure (noninvasive).

STATISTICAL ANALYSES

Statistical analyses were conducted using IBM SPSS 24 (SPSS Inc, Chicago, IL). Sample description was completed using frequency distribution for categorical variables and mean ± 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