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Vascular

Cardiac complications after aneurysmal subarachnoid hemorrhage

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Abstract

Background: Cardiac complications are frequently encountered by neurointensivists caring for patients with SAH. Our aim was to better characterize the natural history of various cardiac abnormalities in this population. We sought to determine the risk factors for cardiac abnormalities, patient outcome, and impact of treatment type on cardiac abnormalities.

Methods: We performed a single center retrospective review of admissions of patients with aneurysmal SAH to the neurosurgical ICU in a large university hospital. Patient demographics, pertinent history, cardiac tests, hospital LOS, intervention type, and discharge outcome were collected.

Results: Data from 266 patients were available for analysis. Of these patients, 50% (n = 133) demonstrated cardiac abnormalities as indicated by abnormal EKG, ECHO, or troponin I. Only age was determined to be an independent statistically significant predictor of cardiac abnormality (P = .01). There was no difference in mortality between the cardiac abnormality and control groups (P = .33). However, there was increased morbidity in the cardiac abnormality group as demonstrated by worse discharge disposition, in addition to increased length of hospital stay (22.6 vs 17.1 days, P < .01). The incidence of cardiac abnormalities was the same among surgical and endovascular treatment groups.

Conclusions: Cardiac abnormalities, including those that meet ACC criteria for MI, are common among patients with SAH. However, in contrast to cardiac events outside the context of SAH, these abnormalities do not increase mortality. They do, however, adversely affect discharge disposition and prolong hospital LOS. The type of aneurysm treatment does not affect the incidence or outcome of cardiac abnormalities.

Keywords: Subarachnoid hemorrhage; Intracranial aneurysm; Echocardiograph; Electrocardiograph; Left ventricular dysfunction; Myocardial infarction

1. Introduction

Subarachnoid hemorrhage affects more than 30,000 patients in the United States annually. Although neurologic morbidity is the prominent feature, this condition affects many organ systems. One of the most studied and common comorbidities of SAH involves the cardiovascular system [2,3]. It has been estimated that between 50% and 100% of patients with SAH will experience some EKG abnormalities, including T-wave inversions, peaked P waves, ST-segment elevations/depressions, QT prolongation, and, in some cases, Q waves [1,6,8,26,29,37-39,41]. In addition, ECHO findings such as regional wall motion abnormalities and global left ventricular impairment are often seen [9,23,30,36,49]. Elevations of troponin have also been observed [10,11,20,35]. These changes are often evident in patients without preexisting cardiac disease or electrolyte abnormality, and coronary angiography in this population has failed to demonstrate underlying CAD [4,12,16,19,23,24,28,36,41]. The cardiac changes after SAH appear to

Abbreviations: ACC, American College of Cardiology; CAD, coronary artery disease; CT, computed tomography; ECHO, echocardiograph; EKG, electrocardiograph; HH, Hunt and Hess grade; ICU, intensive care unit; LOS, length of stay; MHPG, 3-methoxy-4-hydroxyphenylglycol; MI, myocardial infarction; SAH, subarachnoid hemorrhage.

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resolve spontaneously, and the risk of death related to the apparent cardiac pathology appears to be low [28,36,41,42].

Although cardiac abnormality in the setting of SAH has been studied in the past, the current cardiac evaluation tools were not commonplace in many prior studies. Modern practice includes routine measurement of troponin levels and ECHO data. Several questions that are relevant to our practice remained unanswered in the existing literature. We reviewed our experience with 294 consecutive patients with SAH at our institution to determine the predictors of cardiac abnormalities in SAH, the effect of cardiac abnormalities on patient outcome, and the possible contribution of surgical or endovascular treatment to this phenomenon in the current era. For the first time, patients with cardiac events occurring before aneurysm treatment and those with cardiac events after intervention were separated and compared. Using these data, we aim to better characterize the cardiac abnormalities associated with SAH in these critically ill patients.

2. Methods

2.1. Patient selection

We reviewed 294 charts of consecutive patients with nontraumatic aneurysmal SAH admitted to the University of Illinois, at Chicago Neurosurgical Intensive Care Unit, Chicago, Ill, from July 9, 1999, to February 25, 2003. Approval for access to patient records and waiver of informed consent were obtained from our institutional review board. Patients with a diagnosis of SAH, either by CT scan or lumbar puncture, were included if they were at least 18 years old and had an aneurysm identified by cerebral angiography. Patients with evidence of concomitant arteriovenous malformation, subdural hematoma, epidural hematoma, intracerebral hemorrhage, or history of trauma were excluded. Data regarding demographic and clinical parameters (age, sex, grade, aneurysm location, magnesium level, history of smoking, cocaine use, CAD, hypertension, and diabetes mellitus) were analyzed, in addition to pertinent laboratory values and type of intervention for aneurysm treatment.

2.2. Determination of cardiac changes

Available EKG results, troponin I levels, and ECHO data were reviewed for all patients. The official cardiologist EKG and ECHO reports from the time the study was performed were used. Cardiac abnormality was considered to be present based upon at least one of the following criteria:

1. EKG—ST- or T-wave abnormalities, poor R progression, new bundle branch block, or Q wave
2. Troponin I levels—2.0 ng/mL or greater (laboratory normal value <2.0 ng/mL)
3. ECHO—regional wall motion abnormalities, global wall motion abnormalities, or left ventricular ejection fraction less than 40%

If any of these parameters were positive based upon the criteria outlined, the patient was classified as demonstrating a cardiac abnormality. Where possible, previous EKG and ECHO data were used to confirm that EKG and ECHO abnormalities represented new changes. No patients with elevated cardiac troponin I values had renal failure or sepsis, which is known to falsely elevate troponin I values. For many patients, records from subsequent hospital admissions were available and were reviewed to document the resolution of cardiac abnormalities. For patients with left ventricular hypertrophy or bundle branch block, EKG criteria were not used to include patients in the cardiac abnormality group unless abnormalities represented new changes verifiable by prior EKG—prior EKG data were mandatory for inclusion in the cardiac abnormality group for these patients. If none of these parameters were available for review, the patient was excluded from analysis. Thus, only patients who had undergone cardiac testing (EKG, troponin I, or ECHO) were included in the study population (either control or cardiac abnormality group).

2.3. Outcome endpoints

Outcome at discharge (dead vs alive) and discharge status (home, inpatient rehabilitation facility, long-term facility, or death) were noted. Total hospital LOS was recorded.

2.4. Statistical analysis

Demographic and clinical variables between patients with and without cardiac changes were compared using the unpaired 2-tailed Student t test or the χ^2 test where appropriate. Risk factors for having cardiac changes were assessed with multivariate logistic regression of any covariates showing P value of less than .10 on univariate analysis. Similar analyses were performed for outcomes, using ordinal logistic regression for discharge status. All analyses were performed with STATA (Intercooled version 6; Stata Corporation, College Station, Tex). P values less than .05 were considered significant.

3. Results

3.1. Patient demographics and clinical parameters

A total of 294 patients meeting our inclusion criteria were admitted during the study period. Parameters reflecting cardiac function (EKG, troponin I levels, or ECHO) were available for review in 266 patients, who formed the study group. No significant differences in demographic or clinical variables were evident between the excluded patients (n = 28) and the study group (n = 266).

Demographic and clinical features of the entire study group are shown in Table 1. The average age of patients was 53 years old, and there was a female preponderance as is typical for aneurysmal SAH. More than half the cohorts had a history of hypertension and were smokers, much higher than the 20% to 25% rate in the general population. No