Electrocadiographic Scoring Helps Predict Left Ventricular Wall Motion Abnormality Commonly Observed after Subarachnoid Hemorrhage

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Background: Cardiac wall motion abnormality (WMA) is a common complication in patients with subarachnoid hemorrhage (SAH) and is one determinant of their prognosis. The aim of this study was to examine whether the electrocardiography (ECG) findings at admission could predict WMA commonly observed after SAH. *Materials and methods:* We studied 161 SAH patients with SAH who were hospitalized in our institution between April 2007 and November 2010. We performed bedside 2-dimensional transthoracic echocardiography and 12-lead surface ECG within 24 hours of SAH onset. Each of the following ECG changes was scored as having 1 point: ST elevation, ST depression and T wave inversion. We summed up the points in every patient and compared with WMA evaluated by echocardiography. *Results:* The study subjects were classified into 2 groups based on the presence of WMA. Multivariate analysis revealed that ST elevation, ST depression and T wave inversion were strong independent predictors of WMA. Receiver operating characteristic curve determined that the threshold value to predict WMA was 4 points (sensitivity 86.5%, specificity 83.1%, AUC 0.94, $P < .0001$). *Conclusions:* In conclusion, a novel ECG score may well predict WMA after SAH which may associate with an increased risk of mortality.

Key Words: Subarachnoid hemorrhage—Left ventricular wall motion abnormality—ECG scoring—ST elevation—ST depression—T wave inversion.

Introduction

Patients with aneurysmal subarachnoid hemorrhage (SAH) frequently associate with cardiac abnormality such as electrocardiogram (ECG) changes including lethal arrhythmia, and cardiac wall motion abnormality (WMA).[1-6]

The incidence rates of abnormal ECG findings and WMA have varied in individual reports.[1,7-10] Landis et al. reported the most common ECG change was nonspecific ST-T wave abnormalities at 62%, followed by ST elevation at 13%.[6] In another report, 93% of the SAH patients showed abnormal ECG in the acute phase. In detail, prolonged QT interval was seen in 86%, U wave 62%, ST depression 69%, ST elevation 14% and T wave inversion 29%.[11] According to a systematic review and meta-analysis, 157 (4.3%) of 3581 patients admitted with SAH had findings of Takotsubo cardiomyopathy.[12] In this report, pulmonary edema, the most common clinical presentation, occurred in 42% of the patients. We previously reported that WMA provides significant prognostic information in patients with SAH.[7] Neurological status, rate-corrected QT interval, left ventricular

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ejection fraction (LVEF), and WMA were significant predictors of mortality. ECG abnormalities, LVEF <50%, and WMA were observed in 62%, 11%, and 28% of the patients, respectively.

It has been hypothesized that massive release of catecholamine into the systemic circulation after SAH results in nonischemic myocardial injury which is usually reversible.[13,14] We investigated the relationships among plasma catecholamine, serum estrogen and SAH-induced WMA, and showed that decreased estradiol and elevated norepinephrine levels were associated with WMA[15]. Left ventricular (LV) dysfunction is reported to be associated with higher refractory cerebral vasospasm and in-hospital mortality, while beta blocker therapy after SAH may be considered as a cardioprotective and cerebral vasospasm preventive therapy.[16] Angiographic cerebral artery vasospasm is an important cause of morbidity and mortality after SAH. In severe SAH patients, reduction in cardiac output may increase the risk of cerebral ischemia related to vasospasm.[15,17-19] Thus, it is beneficial to predict WMA in the early stage of SAH, thereby contributing to appropriate patient management. Whereas ECG is a routinely available diagnostic tool in almost any clinical setting, echocardiography is not as ubiquitous or as frequently performed as ECG. The aim of this study was to examine whether ECG was useful in predicting WMA after SAH.

Methods

Study Population

Between April 2007 and November 2010, a total of 213 patients with aneurysmal SAH were admitted to our institution, which is a tertiary referral center for stroke. A diagnosis of SAH was made based on the findings of computed tomography (CT), cerebral angiography, and/or lumbar puncture. If brain CT suspects SAH, 3-dimensional CT angiography was performed to search ruptured aneurysms. Forty-two patients were excluded from this analysis because of the lack of any of the following: blood sample collection, records of echocardiogram or ECG within 24 hours of SAH onset. Other exclusion criteria were cardiac pacemaker recipients, a history of myocardial infarction, cardiomyopathy and significant valvular heart disease, which excluded 10 more patients. As a result, 161 (76%) of the 213 patients with SAH were included in this retrospective study. Neurological status on admission was rated according to the World Federation of Neurological Surgeons (WFNS) grade.[20] Short-term mortality was searched up to 85 days. The study protocol was approved by the ethics committee of our institution. Informed consent was obtained from each patient or an appropriate designee.

Echocardiography

All 161 patients underwent bedside 2-dimensional echocardiogram using a GE Vivid 7 (GE Healthcare Japan, Tokyo, Japan) in the neurosurgical intensive care unit. Experienced ultrasound technicians performed the echocardiogram procedure, and the following images were routinely obtained: parasternal long axis; parasternal short axis at the levels of the mitral valve, papillary muscles, and apex; apical 2-, 3-, and 4-chamber; subcostal 4-chamber; and subcostal short axis. According to the guidelines of the American Society of Echocardiography, LV wall was divided into 17 segments.[21] A semiquantitative wall motion score was assigned to each segment visually with the scoring system as follows; 1-normal or hyperkinetic, 2-hypokinetic, 3-akinetic, 4-dyskinetic. LV wall motion score index (WMSI) was calculated by averaging the scores of all LV segments. While WMSI 1.0 showed LV wall motion was completely normal, WMA was defined if WMSI was more than 1.0. As a result, the subjects were classified into 2 groups: WMA (+) and WMA (−). All echocardiogram studies were interpreted by a board-certified cardiologist who was unaware of the clinical course and outcome.

ECG Evaluation

A 12-lead surface ECG was taken on the day of hospital admission at a paper speed of 25 mm/s. A single observer blinded to the clinical course and outcome of the patient performed the ECG analysis. A pathological Q-wave was considered significant when 0.04 seconds in duration or 25% of the height of the R wave for that lead. ST-segment depression >0.1 mV and ST segment elevation >0.25 mV were defined as significant. A T-wave 0.1 mV in depth was defined as inverted. Q-wave and T-wave abnormalities were assessed in leads I, II, aV_L, aV_F, and V_{2-6}. When an inverted U wave was more than 0.1 mV in depth, it was also considered abnormal. The QT interval was defined as the time between QRS onset and the point at which the isoelectric line intersected a line drawn tangentially to the maximal slope of the T-wave. Bifid T-waves exhibiting a time interval of 0.05 seconds between the first and second components were considered T-waves; otherwise, the second component was considered a U wave. The rate-corrected QT interval (QTc interval) was determined according to Bazett's formula: $QTc = QT/[RR]^{1/2}$. The mean QTc interval was calculated from all QTc intervals measured.

Statistical Analysis

For comparison of demographic variables between patients with and without WMA, Fisher exact test was used for categorical variables and unpaired t test for continuous variables. Univariate and multivariate regression analyses were used to identify clinical variables that were correlated with WMA. From the receiver operating characteristics curve (ROC) of ECG score, we derived optimal threshold values to distinguish patients with WMA from