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ORIGINAL RESEARCH

Abnormal ST segment in electrocardiograph predicts poor outcome in patients with acute subarachnoid hemorrhage

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Abstract

Background and purpose: There are high occurrences of abnormal electrocardiographic (ECG) in patients with acute subarachnoid hemorrhage (SAH). Thus, we want to determine whether any specific characteristics in ECG are associated with poor clinical outcomes in patients with SAH inhospital.

Methods: A total of 145 patient who selected from 270 cases with non-traumatic SAH was included in this study. A standard surface ECG was assessed for all patients within 72 hours of SAH onset. All patients were stratified into Good or Poor outcome groups according to the in-hospital mortality or neurological worsening (World Federation of Neurological Surgeons, WFNS class) when they discharge from our hospital.

Results: These patients in Poor outcome (n = 29) had significantly high heart rate (93.52 $\pm$ 22.23 bpm vs 78.42 $\pm$ 18 bpm, $P < 0.01$), prolonged QTc (458.17 $\pm$ 44.88 ms vs 436.89 $\pm$ 43.46 ms, $P = 0.027$) and corrected T_{peak}–T_{end} interval (cTp-e, 106.19 $\pm$ 22.22 ms vs 93.14 $\pm$ 21.04 ms, $P = 0.007$) and high occurrence of ECG abnormalities including ST segment (90% vs 44%, $P < 0.01$) and left ventricular high voltage (28% vs 10%, $P = 0.03$). Multivariable logistic regression identified independent variables indicating poor outcome in-hospital including abnormal ST (OR = 2.507, 95% CI, 1.051-5.941, $P = 0.037$) and WFNS class (OR = 2.280, 95% CI, 1.605-3.240, $P < 0.001$).

Conclusions: Abnormal ST segment of ECG is an independent indicator for poor inhospital outcomes regardless the severity of patients with SAH and warrant to further study their mechanism in the future.

Keywords

Subarachnoid hemorrhage; In-hospital prognosis; Electrocardiographic abnormalities; ST elevation and depression; QTc interval

1. Introductions

Immediately after acute subarachnoid hemorrhage (SAH), there were high chances of various electrocardiographic (ECG) changes (50–90%) [1]. These changes have not attracted attention from neurologist and cardiologist because severe cardiac arrhythmias are not very common [2]. However, these abnormal ECG characteristics may have important speculative values for patients' prognosis because they are associated with excessive adrenergic release, which may cause myocardial injury and cardiac arrhythmias [3]. Study had showed that over released plasma norepinephrine in heart after acute stroke in experimental models caused cardiac dysfunction [4, 5]. The cardiac sympathetic nerve over-activation including high adrenergic activities and norepinephrine releasing has a significant impact on cardiac electrophysiology, arrhythmogenesis and sudden death [6, 7]. Therefore, it is necessary to determine which ECG changes after SAH are clinically significance for predicting patient's outcome.

The most common alterations of ECG after SAH are repolarization abnormalities including the prolongation of QT interval and the deviation of ST segment, anomaly of T-wave morphology, and the presences of abnormal U and Q waves [8]. These ECG changes mimicking cardiac ischemic-like shape may complicate the diagnostic and therapeutic courses of SAH [9, 10]. Several studies reported that some abnormalities of ECG may predicted a poor clinical outcome at 3 months [11, 12]. Other studies also showed that these ECG changes indicates severe neurological injury although they did not means high all-cause mortality in SAH [13, 14]. A recent study reported that ST-T abnormalities and peak levels of troponin and NTproBNP within 72 h after the onset of SAH were associated with an increased risk of death within three months [15]. These studies infer that the interplay between brain and heart may cause poor clinical outcome.

We supposed that some ECG abnormalities may be associ-

ated with in-hospital death or poor clinical outcome after SAH. In the present study, we retrospectively analyzed the characteristics of ECG in patients with SAH and their correlations with mortality and worsening inhospital to find out ECG markers for poor prognosis of SAH.

2. Methods

2.1 Study populations

This study was approved by Ethics Committee of Yijishan Hospital under approval number 2015120024. All participants provided written informed consent for protecting their privacy and knowledge.

We reviewed retrospectively the medical records in detail of all patients with spontaneous SAH admission to our hospital from January 2016 to December 2018. The inclusion criteria in this study included all non-traumatic SAH patients according to clinical and imaging diagnostic criteria of computed tomography (CT) scan and magnetic resonance imaging (MRI), or combination with assaying cerebrospinal fluid in some difficult cases [16, 17]. The exclusion criteria in this study included SAH resulting from tumor; blood diseases like leukemia and aplastic anemia; recent head/neck operation; chronic renal insufficiency; has cardiac structure abnormal like congenital heart disease, valvular heart disease, acute myocardial infarction and infective endocarditis; first ECG collection or admission delay after clinical symptoms more than 72 hours; ECG with heart rhythm disturbances such as atrial fibrillation and bundle branch blocks; abnormal serum potassium and serum calcium.

2.2 Clinical assessment

All demographic information was collected including age, sex, and medical comorbidities like coronary heart disease (CHD), hypertension and diabetes mellitus (DM). The elderly patients ($\geq$ 65 years) classified by age grade. The initial clinical severity and prognosis of SAH when admission to hospital evaluated by using a clinical grading system such as the Hunt and Hess Scale or the World Federation of Neurosurgical Societies (WFNS) scale [16]. The details of grading standards are described as follows.

2.2.1 Hunt and HESS severity scale

class 1, asymptomatic or mild headache;
class 2, moderate or severe headache, nuchal rigidity, can have oculomotor palsy;
class 3, confused, drowsiness, or mild focal signs;
class 4, stupor or hemiparesis;
class 5, coma, moribund, and/or extensor posturing.

2.2.2 World Federation of Neurological Surgeons (WFNS) class

class 1, Glasgow coma scale (GCS), 15: no motor deficit;
class 2, GCS 13–14: no motor deficit;
class 3, GCS 13–14: presence of focal motor deficit;
class 4, GCS 7–12: presence of motor deficit;
class 5, GCS 3–6: presence of motor deficit.

Two neurologists who blind to the final groups assessed all patients using the above classification criteria based on medical records. According to previous reports, the poor clinical outcome of SAH was defined as death in-hospital or clinical worsening showed Glasgow Coma Scale (GCS) decline more than two points when discharge from our hospital which based on eye opening, verbal response and motor response [13, 18] (Fig. 1).

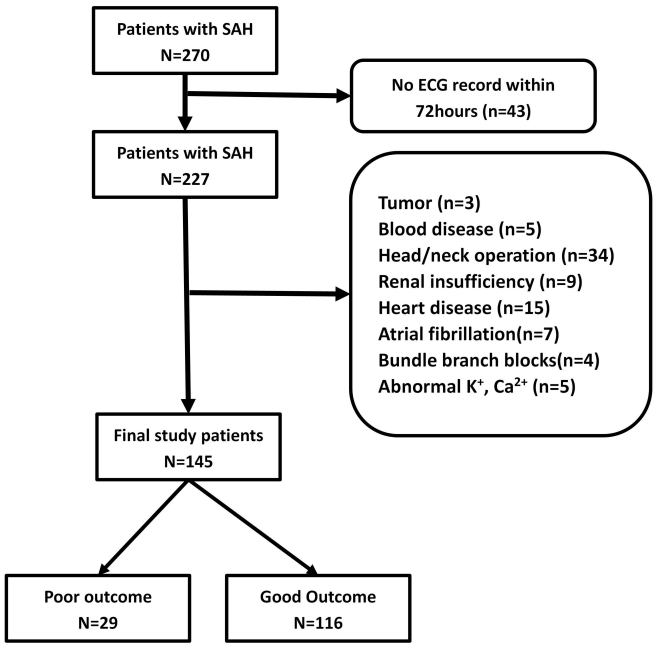

FIGURE 1. Flow chart of study design. SAH, subarachnoid hemorrhage; ECG, electrocardiogram.

2.3 Electrocardiography

All standard twelve-lead ECG from SAH patients were collected within 72 hours after SAH before clinical treatment. Some basic ECG parameters such as heart rate (HR), *P* wave duration, PR interval, QRS duration, and QRS axis were measured using a computerized automated measurement system (FCP-710, Beijing Futian Electronic Medical Instrument Co., Ltd.) and checked manually by the two cardiologists who were blinded to the clinical data of these patients. Specific ECG parameters such as QT interval (The QT interval was calculated as the time from the start of the QRS complex to the end of the T-wave from the longest QT interval of the 12 leads), ST segment changes, and T and U wave abnormalities defined as previous descriptions [19–21]. Corrected QT interval (QTc) was calculated with Bazett's formula as $QT/\sqrt{RR}$. The Tp-e interval was measured as duration from the peak to the end of T-wave point on the precordial V5 or lead 2 (if the V5 lead was not suitable for this measurement). The measured values were corrected according to the heart rate using Bazett's formula [22, 23].

Left ventricular hypertrophy in ECG (LVH) was diagnosed when Sokolow-Lyon criteria (the sum of the S wave in V1 and the R wave in V5 exceeded 3.5 mV in female and 4.0 mV in male) or Cornell voltage criterion (the sum of the amplitudes of the R wave in lead aVL and S wave in lead V3 exceeded 2.8

mV in men and 2.0 mV in women) was meet with. Abnormal heart rate was defined as the number of QRS complexes is less than 60 or more than 100 beats per minutes [19, 24, 25]. The inter-observer and intra-observer variability coefficients for the QTc interval were 3.3% and 3.6%, respectively, and those for the Tp-e interval were 3.1% and 3.4%, respectively.

The morphologic abnormalities of ECG were defined and analyzed as following criteria [21, 26]: (1) Abnormal QTc intervals: the maximal QTc intervals were more than 450 ms in male or 460 ms in female patients; (2) Abnormal ST segment: the ST segment elevation in limb leads is higher than 0.1 mV or 0.2 mV in chest leads or the ST segment depression is more than 0.05 mV; ST-segment score defined as following: class I: normal, class II: depression, and class III: evaluation; (3) Abnormal T-wave was defined as following: peaked, symmetrical, biphasic, flat, and inverted; (4) Abnormal Q wave: Q wave duration is more than 0.04 seconds or 25% of the height of the R wave for that lead.

2.4 Statistical analysis

Continuous variables were shown as mean ± standard deviation and compared with independent samples t test if variables were normal distribution or Mann-Whitney U test if variables were non-normal distribution. Categorical variables were compared with Fisher exact test or χ^2 test when appropriate. Kendall's tau-b correlation analysis was used to analyze the association between variables and poor outcome. Multivariate logistic regression was used to analyze clinical variables and abnormal ECG parameters which showed univariate comparison $P < 0.05$ between the 2 groups. Statistical analyses were performed using SPSS 19.0 statistical package (SPSS Inc, Chicago), and $P < 0.05$ was considered significant.

3. Results

A total of 270 spontaneous SAH patients who were admitted to our hospital. According to the above mentioned exclusion criteria, a total 145 cases from 270 patients were included in the present study for final analysis. According to the inhospital outcome, these patients were stratified into two different groups by good outcome (N = 116) or poor outcome (N = 29) (Fig. 1).

As shown in Table 1, clinical variables such as age, sex, comedication were similar between these 2 groups of patients. However, the WFNS class and Hunt-Hess scale, two variables indicating the initial clinical severity of these patients, were significantly higher in patients with poor outcome than in patients with good outcome.

As shown in Table 2, ECG parameters are significantly different between the two groups. Compared with good outcome, poor outcome group had significantly faster heart rate (P = 0.002), prolonged QTc intervals (P = 0.027), Tp-e (P = 0.007), higher left ventricular hypertrophy (P = 0.002) and higher frequency abnormal ST segment ($P < 0.001$). Other ECG parameters such as T-wave morphology, Tp-e, QRS wave axis, abnormal U wave and Q wave were equal between two groups (all P values were more than 0.05).

The correlation between ECG parameters and in-hospital poor outcome in patients with SAH was shown in Table 3. There were significantly positive correlations between in-hospital poor outcome and heat rate, QRS wave axis, abnormal ST segment, cTp-e and LV hypertrophy. There were marginal statistical significance correlations between in-hospital poor outcome and QTc intervals.

The were no significant correlation between age and ECG changes except LV hypertrophy (r = 0.180, P = 0.005) and cTp-e (r = 0.115, P = 0.042). There was significant correlation between WFNS grade and ECG changes including LV hypertrophy (r = 0.173, P = 0.027), abnormal ST segment (r = 0.313, $P < 0.001$), abnormal T-wave (r = 0.174, P = 0.027) and cTp-e (r = 0.154, P = 0.017).

As shown in Table 4, multivariable logistic regression was used to analyze the independent variables for poor outcome in-hospital including WFNS class, Age grade, Abnormal HR, and Abnormal ST segment. Among of them, WFNS class and abnormal ST segment were the independent variables which predicting poor outcome in-hospital for patients with nontraumatic SAH.

4. Discussion

At the acute phrase of SAH, abnormal changes of ECG are very common [1]. However, the clinical significance of these alterations of ECG in SAH patients is unclear. In the present study, our study demonstrates that abnormal ST segment including depression and elevation are independent predictors for poor clinical outcome beside clinical severity when they admission in-hospital patients with SAH.

The changes of ST-segment on surface ECG are associated with acute ischemia or infarction because of the existence of flow of "injury current" between the ischemic and nonischemic zones [27, 28]. A representative ECG change in SAH is ST elevation or depression which mimicking acute ischemic heart disease [10]. The ST changes are easy to be diagnosed mistakenly as acute ischemic heart disease especially when the occurrence of high cardiac troponin releasing [29, 30]. The misdiagnosis may cause fatal adverse consequence because some treatment strategy of ischemic heart disease including anticoagulants, antiplatelet therapy, and fibrinolytic agents are dangerous to SAH patients [31]. In the present study, SAH patients with poor outcome had more chances of abnormal ST changes. Correlation analysis also confirmed that there is a positive correlation between outcome and abnormal ST segment. Sugimoto *et al.* [20] using a novel ECG score system containing ST elevation, ST depression, QTc prolongation and T-wave inversion to predict cardiac wall motion abnormality and risk of mortality. Zhang *et al.* [32] also reported that ST-T changes in SAH patients were independently associated with an increased risk of adverse clinical outcomes. Our study highlighted the role of ST-segment deviation because we found that only abnormal ST segment is an independent predictor of in-hospital poor outcome in SAH patients. The ST-segment changes may be a marker of myocardial injury, which might furtherly result in poor prognosis for SAH patients.

Prolongation of QTc interval was the most prevalent ECG abnormalities at the early phrase of SAH [33, 34]. Previous studies had showed that the prolonged QTc interval was

TABLE 1. Demographic characteristics of the patients with SAH.

	Good outcome	Poor outcome	*P*
	N = 116	N = 29	
Gender (male, n, %)	57 (49)	11 (38)	0.305
Age (years)	58.91 ± 13.18	63.68 ± 13.87	0.102
Age grade (> 65 years, n,%)	11 (9.5)	18 (62.1)	< 0.001
Commodities, (%)			
hypertension	36 (31)	8 (28)	0.719
CAD	6 (5)	0 (0)	0.600
DM	10 (8.6)	2 (6.9)	1.000
WFNS class, n (%)			< 0.001
I	85 (73.2)	5 (17.2)	
II	5 (4.3)	1 (3.4)	
III	6 (5.2)	1 (3.4)	
IV	18 (15.5)	8 (27.6)	
V	2 (1.7)	14 (48.3)	
Hunt-Hess scale, n (%)			< 0.001
I	26 (22)	1 (3.4)	
II	65 (56)	6 (21)	
III	19 (16)	7 (24)	
IV	5 (4)	6 (21)	
V	1 (0.8)	9 (31)	
Serum K^+ (mMol/L)	3.73 ± 0.43	3.87 ± 2.39	0.545
Serum Ca^{2+} (mMol/L)	2.35 ± 0.38	2.45 ± 0.44	0.570

an independent predictor of in-hospital mortality and adverse clinical outcome [18, 35]. In the present study, patients with acute SAH had a high proportion of QTc interval prolongation regardless their severity (52% vs 32%). Although patients in poor outcome group had longer QTc interval, the correlation between outcome and QTc interval did not reach statistics significance (r = 0.162, *P* = 0.052). Moreover, logistic regression models revealed that abnormal QTc interval could not act as an independent predictor of in-hospital poor outcome in our SAH patients. We deduced that the difference between our results and previous studies may come from different sample size and grouping method.

T-wave morphology of surface ECG is affected by both transmural and apicobasal gradients in repolarization [36]. The incidence of T-wave abnormalities was also frequent among the patients with SAH and had a serious of fluctuating changes, which show a poor outcome [20, 37]. In this study, abnormal T-wave such as inversion, flat, biphasic and peaked upright is common in our SAH patients. However, neither the T-wave differences, nor the correlation of T-wave and in-hospital outcome reach statistics significance. Recent study had shown that T-wave alteration and QTc prolongation recovered immediately after clipping of the aneurysm [38]. Therefore, T-wave abnormalities may be non-specific changes caused by cerebral injury.

cTp-e is another electrocardiographic marker reflecting transmural dispersion of repolarization. It has been used as a risk stratification tool for predicting ventricular tachycardia/fibrillation and sudden cardiac death in different clinical settings [39]. Recent study had shown that Tp-e (including Tp-Te/QT, and Tp-Te/QTc) are associated with disease severity and clinical outcome in patients with SAH [40]. In this study, Poor outcome patients with SAH have significantly prolonged cTp-e interval compared with other SAH patients. However, cTp-e interval is not an independent risk factor for poor outcome of SAH. Therefore, T-wave morphologic abnormalities may be non-specific changes caused by cerebral injury.

We also found that SAH patients with poor outcome in-hospital have higher heart rate. However, heart rate was not independent risk factors for poor outcome. Although previous report showed that bradycardia and relative tachycardia are strongly and independently associated with 3-month mortality after subarachnoid hemorrhage [35], abnormal heart rate was not very common in the present study.

The mechanism leading ECG abnormalities is unclear. There are several evidences that SAH cause local cerebral arteriolar spasm and ischemic damage in the hypothalamus and surrounding area [41, 42]. These cerebral lesions may lead to overexcited sympathetic never activities and sympathetic neurotransmitters releasing to the heart. Therefore, high elevated plasma sympathetic neurotransmitters such as epinephrine and norepinephrine may cause coronary vasospasm and reversible ischemic ‘stunned myocardium’

TABLE 2. ECG findings of the patients.

	Good outcome	Poor outcome	*P*
	N = 116	N = 29	
HR (beats/min)	78.42 ± 18	93.52 ± 22.23	0.002
Abnormal HR, n (%)	22 (19)	15 (52)	< 0.001
P (ms)	87.47 ± 10.04	85.85 ± 20.42	0.691
QRS (ms)	92.91 ± 13.40	88.48 ± 13.53	0.122
PR (ms)	150.56 ± 19.3	148.15 ± 26.77	0.661
QTc (ms)	436.89 ± 43.46	458.17 ± 44.88	0.027
prolonged QTc, n (%)	37 (32)	15 (52)	0.042
QRS wave axis (°)	37.03 ± 38.63	48.75 ± 39.76	0.160
LV hypertrophy, n (%)	12 (10)	8 (28)	0.03
Tp-e (ms)	81.94 ± 17.82	86.72 ± 19.95	0.248
cTp-e (ms)	93.14 ± 21.04	106.19 ± 22.22	0.007
Morphology changes, n (%)			
ST segment score			< 0.001
I (normal, n, %)	65 (56)	3 (10)	
II (depression, n, %)	40 (34)	18 (62)	
III (elevation, n, %)	11 (10)	8 (28)	
T-wave			0.450
Normal (n, %)	50 (43)	8 (28)	
Flat (n, %)	26 (22)	9 (31)	
Inversion (n, %)	23 (20)	7 (24)	
Peaked Upright (n, %)	17 (15)	5 (17)	
Abnormal U wave (n, %)	49 (42)	16 (55)	0.436
Abnormal Q wave (n, %)	10 (8.6)	5 (17)	0.182

TABLE 3. ECG parameters are correlated with poor outcome in patients with SAH inhospital.

	r	*P*
HR (beats/min)	0.242	< 0.001
QRS wave axis (°)	0.143	0.037
LV hypertrophy (n, %)	0.200	0.016
Abnormal ST segment	0.359	< 0.001
cTp-e (ms)	0.216	0.017
QTc (ms)	0.162	0.052

[42]. Actually, human autopsy in patients with SAH who showing considerable normal coronary arteries suggested that these ECG abnormalities might be caused by transient vasoconstriction of microcirculation in the heart and cardiac toxic effects of sympathetic neurotransmitters, but not by actual myocardial infarction [43]. We speculate that severer myocardial ischemia in SAH may cause ST-segment deviation and product additional risk to patients. Further studies are needed to clarify the mechanism of abnormal ST-segment in SAH patients.

This study had some limitations. Firstly, the sample of population is not great enough to evaluate more variables of risk factors. Secondly, selection bias is existed in this study because we excluded some patients with brain operation. Actually, T-wave alteration and QTc prolongation recovered immediately after clipping of the aneurysm [38]. Thirdly, we did not get more ECG records including pre-SAH, acute phrase, chronic phrase of SAH. Thus, we cannot exclude pre-existed ECG abnormalities in some patients. Fourthly, T-wave morphologic changes other than inversion and peaked upright could not be evaluated in this study for the difficult to definition. A novel method like T-wave morphologic analysis should be performed in future study to clarify these nonspecific T-wave abnormalities [19]. Finally, it is necessary to design a strict prospective study to determine whether these ECG changes of SAH come from acute cerebral injury secondary or from myocardial injury primary.

5. Conclusions

This study demonstrated that abnormal ST segment such as depression or elevation within 72 hours is an independently predictor of poor clinical outcomes in inhospital patients with SAH regardless initial clinical severity. Compared to previous studies [18, 35, 40], our study abandon complicated description of ECG features and morphologic calculation. This finding highlight ST-segment deviation as the key points, simplifies

TABLE 4. Statistically independent variables associated with poor outcome in patients with SAH inhospital by multivariable logistic regression.

	B	SE	Wald	Sig	Exp (B)	95% CI	
WFNS class	0.824	0.179	21.143	0.000	2.280	1.605	3.240
Age grade	0.990	0.555	3.177	0.075	2.690	0.906	7.988
Abnormal HR	0.877	0.594	2.181	0.140	2.405	0.751	7.705
Abnormal ST segment	0.919	0.440	4.360	0.037	2.507	1.058	5.941

the identification of abnormal changes of ECG in SAH patients, and is easier to apply in clinical emergency.

AUTHOR CONTRIBUTIONS

DW and BL designed the study. YX, WX and YZ collected the data. YX and DW analyzed the data. DW analyzed the results and drafted the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by Ethics Committee of Yijishan Hospital under approval number 2015120024. All participants provided written informed consent for protecting their privacy and knowledge.

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CONFLICT OF INTEREST

We have no conflict of interest or the appearance of a conflict of interest.

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