TAJ December 2023; Volume 36 Number-2

ISSN 1019-8555
The Journal of
Teachers Association
RMC, Rajshahi

**Original Article**

# Relation of ECG Abnormalities to Clinical Severity and Troponin I Changes in Patients with Subarachnoid Hemorrhage

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## Abstract

**Background: Subarachnoid hemorrhage (SAH) is a devastating condition. Aside from its neurological morbidities, SAH is associated with significant medical complications. Subarachnoid hemorrhage frequently results in myocardial injury with electrocardiographic (ECG) abnormalities and release of cardiac troponin I. This myocardial injury after SAH is a neurally mediated process that is dependent on the severity of neurological injury.**

**Objectives: This study was designed to determine ECG changes in patients with SAH and these changes were compared with neurological severity as well as elevated troponin I levels.**

**Patients and methods: This cross-sectional descriptive study was conducted over 30 patients with SAH. Demographic (age, sex), hemodynamic (pulse, systolic and diastolic blood pressure) and neurological (GCS, WFNS score) information were recorded. We evaluated their on-admission ECG and Troponin I levels.**

**Results: Out of 30 patients, at least one morphological ECG abnormality was present in 17 patients (56.7%) and a total of 21 different abnormalities were present. There were no significant associations between the number or type of observed ECG abnormalities and WFNS grade. But there was a statistically significant correlation between ischemic like ECG changes and elevated troponin I (p = 0.035). The presence of T inversion also significantly explained an elevated Troponin I concentration (46.2%, p = 0.035).**

**Conclusion: ECG changes are prevalent in acute SAH. Ischemic like ECG changes are related to an increase in Troponin I, suggesting that these ECG changes may indicate neurocardiogenic cause of cardiac injury after SAH.**

**Keywords: Subarachnoid hemorrhage, ECG, Troponin I, GCS, WFNS score**

**TAJ 2023; 36: No-2: 17-23**

## Introduction

Subarachnoid hemorrhage (SAH) has both local intracranial effects (hydrocephalus and vasospasm) and global systemic effect that can affect pulmonary and cardiovascular system.[1,2] Cardiac abnormalities, as evidenced by release of cardiac enzymes, changes in ECG or clinical or echocardiographic evidence of left ventricular dysfunction, occurring in association with

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aneurysmal SAH, have been well described.[3,4] Patients with more severe SAH are more likely to develop both cardiac abnormalities [5,6] and poor neurological outcome.[7] A prevailing hypothesis is that a catecholamine surge [8] at aneurysm rupture from cardiac sympathetic nerve endings causes subendocardial contraction band necrosis.[9]

ECG abnormalities are frequently seen in patients following subarachnoid hemorrhage and are unexplained by preexisting cardiac conditions. The most common ECG abnormalities include T wave and ST segment abnormalities. Specific types of ECG abnormality have been associated with poor outcome. These ECG abnormalities are usually seen in patients with severe neurological injury and are not independently predictive of mortality.[5]

The phenomenon of ECG abnormalities in patients with SAH was first reported in 1947. [10] Hersh C [11] noted that in seven patients with SAH who had serial ECGs, there was an increased incidence of depressed ST segments in those who died. Cruickshank et al. [12] prospectively studied 40 patients with SAH; six patients died, of whom five had consistently abnormal ECGs.

Markers for cardiac damage and dysfunction are associated with an increased mortality, poor outcome and delayed cerebral ischemia after SAH. Two scales that are widely used to grade the severity of SAH are the Hunt and Hess and the World Federation of Neurological Surgeons (WFNS) scales.[7,13] In both scales, there is a useful correlation between higher scores and worse patient outcomes.

Cardiac troponin is a diagnostic biomarker for cardiac damage and it increases in patients of subarachnoid hemorrhage.[14] SAH frequently results in myocardial necrosis with release of cardiac enzymes.[15] Cardiac Troponin I release occurs frequently after SAH and has been associated with a neurogenic form of myocardial injury.[16]

The study was done to assess ECG changes after acute SAH. These changes were compared with neurological severity and elevated troponin level. The result of the study might be helpful for better understanding diagnostic and therapeutic implications of acute neurocardiogenic injury after SAH.

## Materials and Methods

This cross-sectional descriptive study was conducted in neuromedicine unit of Rajshahi Medical College Hospital during the period of January 2015 to December 2016. Patients who were detected as Subarachnoid Hemorrhage (within 24 hours of onset of symptoms) and who fulfill the inclusion criteria was enrolled in this study.

The World Federation for Neurosurgeons Score (WFNS) was calculated by observers who were blinded to patient's ECGs. WFNS grade $\geq 3$ was regarded as severe.[6]

A 12 lead ECG was recorded in all patients at admission before any surgical intervention or drug administration that could have affected the ECG. An observer blinded to the patient's clinical course and all other data analyzed the ECGs.

In addition, Troponin I (cTnI) was measured in all patients at admission. A cTnI level $\geq 0.12$ ng/ml was considered as an indicator of cardiac injury.

## Results

30 patients were included in this study.

**Table 1: Baseline characteristics of study population (n=30)**

| | Mean±SD | Frequency(%) | Minimum | Maximum |
|---|---|---|---|---|
| Age | 54.83±12.80 | | 35 | 80 |
| Sex | | | | |
| Male | | 15 (50%) | | |
| Female | | 15(50%) | | |

| | | | |
|---|---|---|---|
| Pulse | 75.13±16.98 | 52 | 124 |
| Systolic BP | 141.33±31.81 | 90 | 240 |
| Diastolic BP | 87.00±18.03 | 60 | 140 |
| GCS | 12.53±2.69 | 8 | 15 |
| WFNS (%) | | | |
| 1 | 13(43.3%) | | |
| 2 | 1(3.3%) | | |
| 3 | 3(10%) | | |
| 4 | 13(43.3%) | | |

The mean age of the patients in this study was 54.83±12.80 years (range 35-80 years). The gender distribution of the population was equal; 50% were male and 50% were female. On admission, the lowest and highest GCS score were 8 &15. The lowest and highest pulse rate were 52 and124 beats/minute. The highest and lowest recorded systolic blood pressure were 240 & 90 mm of Hg and diastolic blood pressure were 140 & 60 mm of Hg. The most frequently occurring WFNS grading were grade 1 and grade 4 (both were 43.3% of patients).

**Table 2: Percentage distribution of study patients by ECG abnormalities**

| | **No. of patients** | **%** |
|---|---|---|
| Normal ECG | 13 | 43.3 |
| Abnormal ECG | 17 | 56.7 |
| Total | 30 | |

At least one morphological ECG abnormality was present in 17 patients (56.7%) out of 30.

**Table 3: Types of ECG abnormalities among study patients (n = 17)**

| | **No. of patients** | **%** |
|---|---|---|
| **Repolarization abnormality** | | |
| ST elavation | 2 | 6.7 |
| ST depression | 2 | 6.7 |
| T inversion | 8 | 26.7 |
| **Conduction abnormality** | | |
| RBBB+LAHB | 1 | 3.3 |
| **Rhythm abnormality** | | |
| Sinus bradycardia | 5 | 16.7 |
| Sinus tachycardia | 2 | 6.7 |
| **Pathological Q** | 1 | 3.3 |

A total of 21 different abnormalities were present. Repolarization abnormalities accounted for 40.1% of ECG alterations; the most common being T wave inversion (26.7%), ST segment elevation (6.7%) and

ST segment depression (6.7%). Pathological Q wave was present in one patient (3.3%). Conduction abnormalities also occurred in one patient (3.3%). The commonest arrhythmia was sinus bradycardia (16.7%) followed by sinus tachycardia (6.7%).

**Table 4: Relationship between ECG changes and WFNS grade**

| ECG | WFNS (%) | | $\chi^2$, df |
|---|---|---|---|
| | Grade 1&2 | Grade 3,4&5 | p |
| ECG changes | 6 (42.9) | 11 (68.8) | 2.039, 1<br>0.153 |
| ST elevation | 0 (0) | 2 (12.5) | 1.875, 1<br>0.171 |
| ST depression | 0 (0) | 2 (12.5) | 1.875, 1<br>0.171 |
| T inversion | 2 (14.3) | 6 (37.5) | 2.058, 1<br>0.151 |
| RBBB+LAHB | 1 (7.1) | 0 (0) | 1.182, 1<br>0.277 |
| Pathological Q | 0(0) | 1 (6.2) | 0.905, 1<br>0.341 |
| S. bradycardia | 3 (21.4) | 2 (12.5) | 0.460, 2 |
| S. tachycardia | 1 (7.1) | 1 (6.2) | 0.795 |

*Values reach statistical significance.

There were no significant associations between the number or type of observed ECG abnormalities and WFNS grade.

**Table 5: Percentage distribution of study patients by Troponin I level**

| | N (%) | Mean±SD | Minimum | Maximum |
|---|---|---|---|---|
| Normal | 17 (56.7) | | | |
| Elevated | 13(43.3) | 0.325 ±0.640 | 0.001 | 2.700 |
| Total | 30 | | | |

Thirteen of the 30 patients demonstrated elevations of Troponin I (43.3%). Among the recorded Troponin I levels the lowest level was 0.001 ng/ml and highest level was 2.700 ng/ml.

**Table 6: Association of different ECG abnormalities with Troponin I changes**

| | Troponin I N(%) | | $\chi^2$, df p |
|---|---|---|---|
| | Elevated ≥0.12 ng/ml | Normal <0.12 ng/ml | |
| Ischemic changes | 8 (61.5) | 4 (23.5) | 4.434, 1 0.035* |
| ST elevation | 2 (15.4) | 0 (0) | 2.802, 1 0.094 |
| ST depression | 1 (7.7) | 1 (5.9) | 0.039, 1 0.844 |
| T inversion | 6 (46.2) | 2 (11.8) | 4.455, 1 0.035* |
| Pathological Q | 1 (7.7) | 0 (0) | 1.353, 1 0.245 |
| RBBB+LAHB | 0 (0) | 1 (5.9) | 0.791, 1 0.374 |

*Values reach statistical significance.

12 patients had ischemic like ECG changes. Among them 8 patients (61.5%) had elevated Troponin and 4 patients (23.5) had normal troponin. There was a statistically significant correlation between ischemic like ECG changes and elevated troponin (p = 0.035). The presence of T inversion also significantly explained an elevated Troponin I concentration (46.2%, p = 0.035).

## Discussion

Subarachnoid hemorrhage (SAH) is the bleeding in the subarachnoid space. It occurs explosively sudden resulting in intense vasospasm not only of the cerebral vessels but also of the extracerebral vessels specially the coronary vessels giving rise to their respective consequences.

The age of SAH varies in different parts of the world. In our study, the mean age was 54.83±12.80 years (range 35-80 years). Similar pattern of age distribution and mean age was observed by by Tung et al.[15], Parekh et al.[17] and Colkesen et al.[18]. But lower mean age group was observed by Sakr et al.[6], Horowitz et al.[19], Lorsheyd et al.[20] and Manninen et al.[21] Male-female ratio in our study was 1:1. Female predominance was observed by Sakr et al.[6], Tung et al.[15], Parekh et al.[17], Colkesen et al.[18], Horowitz et al.[19], Manninen et al.[21] and Ahmadian et al.[22] Most of the reviewed studies showed female predominance and maximum no. (87.4%) of female patients were included in the study by Ahmadian et al.[22] Male predominance was observed by Lorsheyd et al.[20] In our study, the lowest GCS score was 8 and highest score was 15. The mean GCS was 12.53±2.69. The median GCS was 13 in a study by Parekh et al.[17] The mean of pulse rate, systolic and diastolic blood pressure at admission were 75.13±16.98, 141.33±31.81 and 87.00±18.03 respectively in this study. Similar

mean heart rate was found in the studies by Tung et al.[15] and Parekh et al.[17] The most frequently occurring World Federation of Neurosurgical Societies (WFNS) grading in our study were grade 1 and grade 4 (both were 43.3% of patients). A study by Sakr et al.[6] showed that most frequently occurring WFNS grade was 1 (48.7%) followed by grade 4 (19.9%), grade 5 (13.5%), grade 2 (10.3%) and grade 3 (7.7%). This picture does not resemble our study. The possible explanation may be the difference of sample size between the studies.

ECG alterations have been described frequently in the course of SAH. In our study, 56.7% of patients with no history of ischemic heart disease had abnormal ECGs. Our study is also consistent with the study by Parekh et al.[17] (59%). Higher frequencies were found by Lorsheyd et al.[20] (73%), Naidech et al.[16] (69%), Horowitz et al.[19] (68.1%) and Sakr et al.[6] (66.7%). Frequencies lower than our study were reported by Manninen et al.[21] (43%) and Ahmadian et al.[22] (44.8%). In our study, repolarization abnormalities in the form of ST-T wave changes accounted for 40.1% of ECG alterations. The pattern of ECG abnormalities observed in our study are also consistent with previously published data (Sakr et al.[6], Parekh et al.[17], Lorsheyd et al.[20], Manninen et al.[21], Ahmadian et al.[22]). Andreoli et al.[23] studied 70 patients with SAH; 91% experienced cardiac arrhythmias and a prolonged QT interval on the ECG was observed in 29. We did not detect any malignant arrhythmias in our study.

There were no significant associations between the number or type of observed ECG abnormalities and WFNS grade in our study. A study by Sakr et al.[6] reported that only ST segment depression was related to higher WFNS score.

The frequency of cTnI elevation (≥0.12 ng/ml) was 43.3% among the patients we tested, a result that is higher than the following studies by Horowitz et al.[19] (17%), Parekh et al.[17] and Tung et al.[15] (20%).In other studies, the reported frequency of cTnI elevation was higher than our study; Naidech et al.[16] (68%), Ahmadian et al.[22] (71.6%). The disparity in the incidence of myocardial injury in SAH reported in our study and those of others could be attributed to the differing grades of SAH in the study population, differences in the timing and methodology of biochemical assays and the use of cTnI assay in our study.

The relation on ECG abnormalities including ischemic-like changes to cTnI elevation has been studied by some investigators. We found that ischemic like ECG changes (61.5%, $p = 0.035$) and the presence of T inversion (46.2%, $p = 0.035$) were significantly related to elevation in troponin I. Parekh et al.[17] noted that patients with elevated troponin had a statistically higher proportion of ST-T changes (39%, $p < 0.01$), similar correlation to what was observed in our trial. Horowitz et al.[19] prospectively studied 47 patients with SAH and found that there was no association between elevated cTnI and abnormal ECG ($p = 0.401$).

## Conclusion

In our study, at least one morphological ECG abnormality was present in 56.7% of patients with no history of ischemic heart disease and a total of 21 different abnormalities were present. There were no significant associations between the number or type of observed ECG abnormalities and WFNS grade. But there was a statistically significant correlation between ischemic like ECG changes and elevated troponin and the presence of T inversion also significantly explained an elevated Troponin I concentration. These findings support a neurocardiogenic cause of cardiac injury after SAH. So, ECG and Troponin I should be done for early detection of neurocardiogenic injury after Subarachnoid hemorrhage.

**Conflict of interest:** None declared

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