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# Incidence of Cardiac Dysfunction After Brain Injury

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## ABSTRACT

**Introduction:** Cardiovascular complications in patients with subarachnoid hemorrhage are considered to be a neurally mediated process rather than a manifestation of coronary artery disease. **Aim:** The aim of study is to show the incidence and type cardiac complications after traumatic and spontaneous SAH. **Patients and methods:** The study had prospective character in which included 104 patients, with diagnosed subarachnoid hemorrhage (SAH), in the period from 2014 to 2017. Two groups of patients were formed. Group I: patients with SAH caused by the rupture of a brain aneurysm. Group II: patients with SAH after traumatic brain injury. **Results:** Electrocardiogram (ECG) abnormalities was predominant after traumatic brain injury 74 %, with statistically significant difference atrial fibrillation 42.5 % ( $p = 0.043$ ) and sinus bradycardia 31.4 % ( $p = 0.05$ ). Hypertension are predominant in patients with spontaneous SAH with statistically significant difference (15 (27.7%) vs 36 (72%)  $p=0.034$ ) and hypotension in group II (10 (18.5%) vs 2 (4%)  $p = 0.021$ ) with traumatic SAH patients. The time in Intensive Care Unit (ICU) for traumatic SAH group was  $6.1 \pm 5.2$  days and  $3.9 \pm 1.16$  for spontaneous SAH group with statistical significance ( $p = 0.046$ ). Respiratory support time was longer in traumatic SAH group ( $39.4 \pm 23.44$  vs.  $15.66 \pm 22.78$ ) with  $p = 0.043$ . **Conclusion:** Cardiac dysfunction in patients with subarachnoid hemorrhage are considered to be a neurally mediated process rather than a manifestation of coronary artery disease. Early treatment of cerebral injury could be reduce incidence of cardiac complications after traumatic brain injury. Cardiac dysfunction in patients with SAH is still very high, despite substantial qualitative progress in their treatment.

**Keywords:** subarachnoid hemorrhage, cardiac dysfunction, brain injury.

## 1. INTRODUCTION

In the critically ill neurosurgical patients cardiac complication are prevalent (1). Mortality and morbidity from SAH are mostly secondary to rebleeding of unsecured aneurysms and the delayed ischemic deficit or infarction from arterial vasospasm (2). Neurocardiogenic injury or stress cardiomyopathy is associated with subarachnoid hemorrhage, intracranial hypertension and cerebral ischemia, present with mild troponin release (3), hemodynamically instability, hypertension and ECG abnormality with arrhythmias (4). The severity of the myocardial dysfunction and troponine release correlates with severity of the neurologic injury (5). ECG abnormalities are common after SAH. The most common cardiac abnormality are atrial and ventricular arrhythmias (6). Prolonged Q-T interval ( $>550$  msec) occurs frequently after SAH (7) and has been associated with an increased incidence of malignant ventricular rhythms including torsades de pointes (8). SAH can result in a reversible, “stun-

ning”-like myocardial injury. The severity of the dysfunction correlates best with the severity of the neurologic injury, it is thought to be catecholamine mediated (9). In addition to the classic “canyon T waves”, non specific T-wave changes, Q-T prolongation, ST-segment depression, and U waves have been described (10). Medical management cardiac dysfunction be required treatment before and after surgical (11).

## 2. AIM

The aim of study is to show the incidence and type cardiac complications after traumatic and spontaneous SAH.

## 3. PATIENTS AND METHODS

A total of 104 cases, 54 with traumatic subarachnoid hemorrhage (group I) and 50 patients with spontaneous rupture (group II), were observed during 4 Year study period, in neurosurgical ICU, University Clinical Center Tuzla, Bosnia and Herzegovina. All patients after hospital admission underwent CT scan of the brain

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and postoperative were admitted in ICU and depending on the severity of the injury. All complication we detected during ICU stay. Patients with age < 18 were exclude. Criteria for cardiac event included hyper/hypotension, tachy/bradycardia, arrhythmia and ECG abnormality analyzed. We investigated the duration of respiratory support.

**Statistical analysis**

The continues variables were expressed as mean ± standard deviation (SD). Non-parametric methods were used for all comparisons of meteorological parameters. Data were compared by the Wilcoxon signed–rank test so that value of p < 0.05 was considered significant in all tests.

**4. RESULTS**

Patient demographic. Fifty or 92.5% male and 4 (8%) female had traumatic subarachnoid hemorrhage. Twenty or 40% male and thirty female or 60 % had spontaneous hemorrhage.

Gender distribution	Group I	Group II	P value
Male	50 (92.5%)	20 (40%)	p=0.066
Female	4 (7.4%) p=0.0031	30 (60%) p=0.10	p=0.007
Age distribution	52.07±20.35	57.6±10.6	p=0.71

**Table 1. Patient demographic**

Result showed statistically significant between gender distribution in traumatic SAH patients, men/women (50 vs 4, p=0.0031), and between two groups in women distribution (4 vs 30, p=0.007) (Table 1).

Type of complication	Group I	Group II	P value
Hypertension	15 (27.7%)	36 (72%)	p=0.034
Arrhythmias	32 (59.25%)	32 (64%)	p=0.13
Pneumonia	11 (20.3%)	4 (8%)	p=0.06
Myocardial infarction	-	-	-
VAP	29 (53.7%)	7 (14%)	p=0.026
Pleural effusion	9 (16.6 %)	4 (8%)	p=0.12
Hipotension	10 (18.5%)	2 (4%)	p=0.021

**Table 2. Type of complication**

Comparative analyses of cardiac manifestations showed more frequent occurrence of hypertension with statistically significant difference in group II with spontaneous SAH (15 (27.7%) vs 36 (72%) p=0.034), and hypotension in group I (10 (18.5%) vs 2 (4%) p=0.021 ) with traumatic SAH patients (Table 2).

ECG abnormality	Group I/N54	Groupe II/N50	P value
Atrial fibrillation	23 (42.5%)	6 (12%)	p=0.043
Ventricular arrhythmias	11 (20.3%)	2 (4%)	p=0.11
LBBS	3 (5.5%)	-	-
Prolonged QT interval	11 (20.3%)	12 (24%)	p=0.21
Sinus bradycardia	17 (31.4%)	12 (24%)	p=0.05
ST segment elevation	-	-	-
ECG abnormality	40 (74.0%)	32 (64%)	p=0.066

**Table 3. Cardiac complication**

Cardiac complication analysis showed higher incidence of atrial fibrillation with statistically significant

difference (p = 0.043) and sinus bradycardia (p = 0.05) in group I compared to group II patients (Table 3).

	Group I	Group II	P value
Respiratory support (h)	39.4 ± 23.44	15.66 ± 22.78	p=0.043
ICU days	6.1 ± 5.2	3.9 ± 1.16	p=0.046

**Table 4. ICU stay and respiratory support**

The time in ICU for traumatic SAH group was 6.1 ± 5.2 days and 3.9 ± 1.16 for spontaneous SAH group with statistical significance (p = 0.046). Respiratory support time was longer in traumatic SAH group (39.4 ± 23.44 vs. 15.66 ± 22.78) with p = 0.043 (Table 4).

**5. RESULTS**

Results showed statistically significant between gender distribution in traumatic SAH patients, men/women (50 vs 4, p=0.0031), and between two groups in women distribution (4 vs 30, p=0.007). Comparative analyses of cardiac manifestations showed more frequent occurrence of hypertension in group II with spontaneous SAH (15 (27.7%) vs 36 (72%) p=0.034), and hypotension in group I (10 (18.5%) vs 2 (4%) p=0.021 ) with traumatic SAH patients. Cardiac complications analysis showed higher incidence of atrial fibrillation (p = 0.043) and sinus bradycardia (p = 0.05) in group I with statistically significant difference compared to group II patients. The time in ICU for traumatic SAH group was 6.1 ± 5.2 days and 3.9 ± 1.16 for spontaneous SAH group with statistical significance (p = 0.046). Respiratory support time was longer in traumatic SAH group (39.4 ± 23.44 vs. 15.66 ± 22.78) with p = 0.043.

**6. DISCUSSION**

Mortality in patients with SAH is still very high, despite substantial qualitative progress in their treatment. In our study the most frequent cardiac complications was hypertension, that prevailed in the group with spontaneous SAH (72%) and had a statistically significant difference (p = 0.034). Hypertension is also the most common cardiac complication in patients with SAH (73,6%) (9). Hypotension was rarely represented in percentages, but there was statistically significant difference comparing both groups (p=0,02). In our study, ECG abnormalities were found in 40 (74%) of patients with traumatic SAH and in 32 (64%) of patients with spontaneous SAH. Atrial fibrillation was present in 23 (42,5%) patients with traumatic SAH while in the second group, patients with spontaneous SAH was at 6 (12%) patients that had statistically significant difference (p=0,043%). Macrea and colleagues analyzed eight studies (386 patients), where they found ECG abnormalities in 32% of patients with minimal, 55% with moderate, and 58% with severe neurological deficit (p < 0.0001) in patients with spontaneous SAH (1). Zhang in his study included 834 patients with non traumatic SAH showed the following results. The frequency of ECG abnormalities for all enrolled patients was 65%. Neurogenic pulmonary edema (NPE) occurred in 192 patients (23%). The median delay from SAH onset to NPE was 3 days (interquartile range [IQR]: 5 days). Delayed cerebral

ischemia (DCI) occurred in 223 patients (27%; median delay to DCI, 4 days; IQR: 5 days). In total, 141 patients (17%) died in the hospital (12). Klaudia and colleagues analyzed study of 266 patients. 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=0.01). There was no difference in mortality between the cardiac abnormality and control groups (p=0.33) (13). Huang and colleagues studied prospectively a cohort of 222 adult patients with spontaneous SAH. Compared with the survivors (n=178), the non survivors (n=44) had significantly slower heart rate p=0.018 and more prolonged QTc (p=0.001). There were significantly higher frequency of occurrence of ECG morphologic abnormalities (66% vs 37%, p=0.001) and nonspecific ST- or T wave changes (NSSTTCs; 32% vs 12%, p=0.015) in the non survivors compared with those in the survivors (14). Our study showed that 29 (53,7%) patients with traumatic SAH and 7 (14%) patients with spontaneous SAH had ventilator-associated pneumonia. Between the two groups there was a statistically significant difference (p = 0.026). Study of Zygun and colleagues resulted that patients with polytrauma were at higher risk for development of VAP than patients with isolated head injury. VAP was not associated with increased hospital mortality, but VAP is associated with significant morbidity in patients with severe traumatic brain injury (15).

## 7. CONCLUSION

Most common cardiac complications after brain injury are hypertension, atrial fibrillation, ECG abnormalities and ventricular arrhythmias. Comparative analyses of cardiac manifestations showed more frequent occurrence in traumatic brain injury patients. Monitoring of cardiac function with 12-channel ECG, control serum troponin and correction of electrolytic disbalance play a major role in successful treatment of cardiac complications after brain injuries. Early treatment of cerebral injury could be reduce incidence of cardiac complications after traumatic brain injury.

- Authors' contributions: S.S., A.K., H.A. gave substantial contributions to the conception or design of the work in acquisition, analysis, or interpretation of data for the work. Each author had a part in article preparing for drafting or revising it critically for important intellectual content, and all authors gave final approval of the version to be published and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.
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