Changes in Electrocardiogram in Patients With Spontaneous Subarachnoid Hemorrhage: A Cross-Sectional Study

Binod Poudel [1] , Prasanna Karki [1] , Samjhana Panta [2] , Aastha Lamsal [3] , Parash Koirala [4] , Surya Devkota [5] , Gopal Sedain [6] , Mohan R. Sharma [6]

1. Internal Medicine, Tribhuvan University Institute of Medicine, Kathmandu, NPL 2. Intensive Care Unit, Tribhuvan University Institute of Medicine, Kathmandu, NPL 3. Internal Medicine, Kist Medical College, Lalitpur, NPL 4. Cardiology, National transplant centre, Kathmandu, NPL 5. Cardiology, Manmohan Cardiothoracic Vascular and Transplant Center, Institute of Medicine, Tribhuvan University Teaching Hospital, Kathmandu, NPL 6. Neurosurgery, Tribhuvan University Institute of Medicine, Kathmandu, NPL

Corresponding author: Binod Poudel, binod.poudel16@gmail.com

Abstract

Background

Electrocardiographic (ECG) changes are frequently reported findings in patients with subarachnoid hemorrhage (SAH). We conducted a retrospective descriptive study to assess the prevalence of electrocardiographic changes in patients with non-traumatic SAH.

Methods

In this single-center retrospective cross-sectional study, ECG recordings of 45 patients who presented to Tribhuvan University Teaching Hospital in the year 2019 with SAH were collected and analyzed to detect any abnormalities.

Results

In our study, 88.8% of patients had some form of ECG abnormality. The most common ECG abnormalities associated with SAH were QTc prolongation, T-wave abnormalities, and bradycardia, which were found, respectively, in 35.5%, 24.4%, and 24.4% of the patients. Other ECG changes we observed include ST depression, large U-waves, atrial fibrillation, and premature ventricular contractions.

Conclusion

Morphological and rhythm abnormalities are frequently observed in patients with SAH, which may cause diagnostic dilemmas and unnecessary workups. Further studies are required to evaluate their significance and correlate ECG changes with clinical outcomes.

Introduction

Intracranial hemorrhages (ICHs) are classified, depending on the cause, to be traumatic and non-traumatic, and the anatomical site further classifies ICHs into four main groups: epidural hemorrhage, subdural hemorrhage, subarachnoid hemorrhage (SAH), and intraparenchymal hemorrhage. SAH is defined as bleeding into the subarachnoid space, which is a potential space beneath the arachnoid membrane and above the pia mater. Subarachnoid hemorrhage is a worldwide health burden with high mortality and morbidity among survivors. The overall incidence of SAH is 7-11 per 100 000 inhabitants/year with a higher incidence in Japan and Finland [1]. Most SAHs are caused by ruptured saccular aneurysms. Other causes include trauma, arteriovenous malformations/fistulae, vasculitides, intracranial arterial dissections, amyloid angiopathy, bleeding diathesis, and illicit drug use. The aneurysmal rupture is most common in the age group of 50 to 55 years [2].

Acute stress on the nervous system has been known to affect the heart both structurally and functionally and is described in the literature as neurogenic stress cardiomyopathy (NSC). The occurrence of NSC is reported more frequently in some types of intracranial hemorrhage such as non-traumatic SAH than in others [3]. Despite tremendous advances in cardiac evaluations, an electrocardiograph (ECG) remains to be an essential tool to rule out cardiac illness, especially in emergency setups. The reported prevalence of ECG changes in patients with SAH ranges from 71% to 100% [3-5]. This might have major implications for the emergency management of the patient. Moreover, the presence of concomitant heart illness in this age

group cannot be denied.

ECG changes being that common, there have been both prospective and retrospective studies as well as holter monitoring of patients with SAH to find out its prevalence and significance. In previous large-sample studies, commonly detected abnormalities were large U wave, T wave abnormality, QTc prolongation, high R wave, ST depression, and bradycardia [4,5]. There have also been reports of cases where SAH has presented as a complete heart block and transient ST elevated myocardial infarction [6,7]. Some studies have tried to evaluate the significance of such ECG changes and there is evidence that the electrocardiographic abnormalities, especially abnormal Q or QS wave and nonspecific ST, T changes, may predict the development of neurogenic pulmonary edema within 24 hours in adult patients with spontaneous SAH [8]. This suggests a comprehensive knowledge of the ECG changes is pertinent in the management of SAH.

Evidence regarding the prevalence of ECG changes in SAH is lacking in our region. The aim of our study is to determine ECG changes in spontaneous SAH presenting over a period of one year in a tertiary hospital in Nepal.

Materials And Methods

Setting

This study was conducted at the Tribhuvan University Teaching Hospital, Kathmandu, Nepal, between Jan 2019 and Dec 2019. This hospital is the largest in Nepal, has a large patient flow, and has a department of neurosurgery. It was carefully selected for the review of such data.

Study design

This retrospective observational study was approved by the Institutional Review Committee of Institute of Medicine, Tribhuvan University, with reference number: 501(6-11)E2. Structured proforma was used to record the data retrospectively for the admitted patients. We reviewed the records of patients admitted between January to December 2019.

Inclusion criteria

Patients above the age of 18 years with the diagnosis of subarachnoid hemorrhage who were admitted to the neurosurgery ward of the hospital were enrolled in the study.

Exclusion criteria

Patients were excluded if they had any of the followings: 1. Had a history of cardiovascular disease; 2. Was under antiarrhythmic treatment in the prior 6 months including beta blockers or any other drugs that could alter ECG recording; 3. Electrolyte abnormalities including severe hypokalemia (Serum K+ >6.0mEq/L), hyperkalemia (Serum K+ <3mEq/L), and hypocalcemia (Serum Ca <8mEq/L); 4. Diuretic treatment in the past month.

Study parameter

The diagnosis of subarachnoid hemorrhage was considered in any patient with severe and sudden onset or rapidly escalating headache and was confirmed by findings suggestive on computed tomography (CT) scan. Clinically suspicious but those negative on CT scan were subjected to lumbar puncture and CT angiography. We included only spontaneous cases of subarachnoid hemorrhage in our study. Demographic characteristics including age, sex, and pertinent medical history (diabetes mellitus, hypertension) of the patients were collected. We also collected whether SAH was aneurysmal or non-aneurysmal and whether it was repaired during the active stage. The neurological status of the patient including the Hunt-Hess score, World Federation of Neurological Surgeons (WFNS) score, reactivity, and size of the pupil was noted from the admission file. Table *1* summarizes the neurological grading scale used during our study, and both Hunt-Hess and WFNS scales are well standardized and their association with clinical outcomes is well described.

World Federation of Neurological Surgeons Scale (WFNS)			Hunt and Hess Scale	
Grade	GCS	Neurologic Examination	Grade	Neurologic Examination
1	15	No motor deficit	1	Awake, alert, no cranial nerve or motor deficits, mild headache, minimal or no nuchal rigidity
2	13-14	No motor deficit	2	Awake, alert, moderate to severe headache, nuchal rigidity, no motor deficits,may have cranial nerve deficit
3	13-14	Motor deficit	3	Confusion or lethargy, with or without mild focal neurologic deficits
4	7-12	With or without motor deficit	4	Stuporous, more severe focal neurologic deficit
5	3-6	With or without motor deficit	5	Comatose, motor posturing or no motor response

TABLE 1: SAH clinical grading scales - Hunt-Hess and WFNS scoring

WFNS: World Federation of Neurological Surgeons; GCS: Glasgow Coma Scale; SAH: subarachnoid hemorrhage

[9,10]

ECG analysis

A 12-lead Electrocardiography (ECG) was performed for all patients at the time of admission on a calibrated device with a paper speed of 25 mm/sec and a standard amplitude of 10 mm/mV. The ECG performed closest to the time of presentation was used in case of multiple ECG readings. All ECGs were interpreted by two cardiologists who were blinded to the clinical data of the patient. The following parameters were evaluated for each ECG:

1. Rhythm abnormalities

1a. Heart rate (Bradycardia if HR <60 beats per minute (bpm) and Tachycardia if HR> 100 bpm)

1b. Arrhythmias including Atrial fibrillation, Premature Ventricular Contractions (PVCs)

2. Morphological ECG changes

2a. Poor progression of R waves

2b. ST Depression (>= 0.1 mV; 80 ms post J point)

2c. T-wave abnormalities (peaked T-waves, flat T-waves, and T-wave inversion)

2d. Corrected QTc interval (calculated as QT interval divided by the square root of RR interval in seconds): considered prolonged if more than 440 in males and 460 in females.

2e. Large U wave (amplitude >1mm)

2f. Axis deviation

Statistical analysis

Data were compiled, edited, and checked as per a proforma to maintain consistency. The data was recorded and analyzed in Microsoft Excel for Windows (Microsoft Corp., Redmond, USA).

Results

During the period of one year, a total of 52 adult patients with non-traumatic SAH were treated in this center, out of which only 45 were included in our study; the remaining seven were excluded because of coexisting electrolyte abnormalities. Demographic and clinical characteristics are summarized in Table 2. The mean age of patients was 54.3 ± 13.47 years. Of these patients, 30 (66.67%) were females. The average time of presentation was 44.35 ± 29.35 hours. Only two (4.4%) cases presented after a week while 39 (86.6%) cases presented within 72 hours. 12% of patients had some comorbidity - either diabetes or hypertension or both.

2023 Poudel et al. Cureus 15(6): e40045. DOI 10.7759/cureus.40045

3 of 9

Clinical details	Number	Percentage
Age	54.3±13.47	
Sex(F)	30	66.67
Time of presentation	44.35±29.35	
Hypertension	12	26.66
Diabetes	4	8.88
Localization of hemorrhage		
ACOM	16	35.55
PCOM	7	15.55
MCA	8	17.77
ICA	2	4.44
Perimesencephalic	4	8.88
Negative ct angiography	6	13.33
Multiple	2	4.44
Neurological status at presentation		
Hunt-Hess score		
Hunt Hess 1	1	2.22
Hunt Hess 2	35	77.77
Hunt Hess 3	9	20
Hunt Hess 4	0	0
WFNS		
WFNS 1	31	68.88
WFNS 2	9	20
WFNS 3	3	6.66
WFNS 4	2	4.44
Management		
Clipping	31	68.88
Coiling	2	4.44
Conservative	11	24.44
Deferred treatment	1	2.22
Death	4	8.88

TABLE 2: Baseline characteristics of patients with SAH

ACOM: Anterior Communicating Artery; PCOM: Posterior Communicating Artery; MCA: Middle Cerebral Artery; ICA: Internal Carotid Artery; WFNS: World Federation of Neurological Surgeons; SAH: Subarachnoid Hemorrhage

Characteristics of SAH

An initial CT scan localized the aneurysm in 39 (86.6%) patients out of which 16 (35.5%) localized in the anterior circulation. Six (13.3%) patients with spontaneous SAH were non-aneurysmal in origin. Most patients in our study had Hunt-Hess 2 and WFNS 1 scores at the time of presentation. Of these patients, 31 (68.8%) received clipping and 11 (24.2%) opted for conservative management. Only two patients underwent

coiling. We should acknowledge the fact that coiling was not available at the study site and patients were referred to another center if they preferred coiling.

Characteristics of ECG abnormalities

The prevalence of different ECG changes is summarized in Table 3. Out of the 45 patients enrolled in the study, 40 (88.8%) patients had some form of ECG abnormality. Isolated rhythm abnormality was present in six (13.3%) patients, all presenting as bradycardia. All other 34 (75.5%) patients had concomitant rhythm and morphological changes in ECG.

Abnormality	Number	Percent
Rhythm abnormality		
Mean heart rate	69.83±12.04	
Bradycardia	11	24.44
Atrial fibrillation	1	2.2
Sinus arrhythmia	1	2.2
Premature Ventricular contraction	1	2.2
Morphological abnormality		
Left Axis deviation	7	15.55
P mitrale	4	8.88
Poor progression of R wave	4	8.88
ST depression	4	8.88
Left Bundle Branch Block	1	2.22
T-wave abnormality		
Peaked T-wave	3	6.66
Flat T-wave	1	2.22
T- wave inversion	7	15.55
Corrected QT interval		
Prolonged	16	35.55
Shortened	2	4.44
U-wave	5	11.11

TABLE 3: Prevalence of ECG abnormality

Rhythm abnormality

The heart rate was found to be 69.8 ± 12.04. Isolated rhythm abnormality was present in six (13.3%) patients, all presenting as bradycardia. Bradycardia was present in a total of 11 (24.4%) of patients. We encountered single cases of atrial fibrillation (Figure 1), sinus arrhythmia, and premature ventricular contraction.
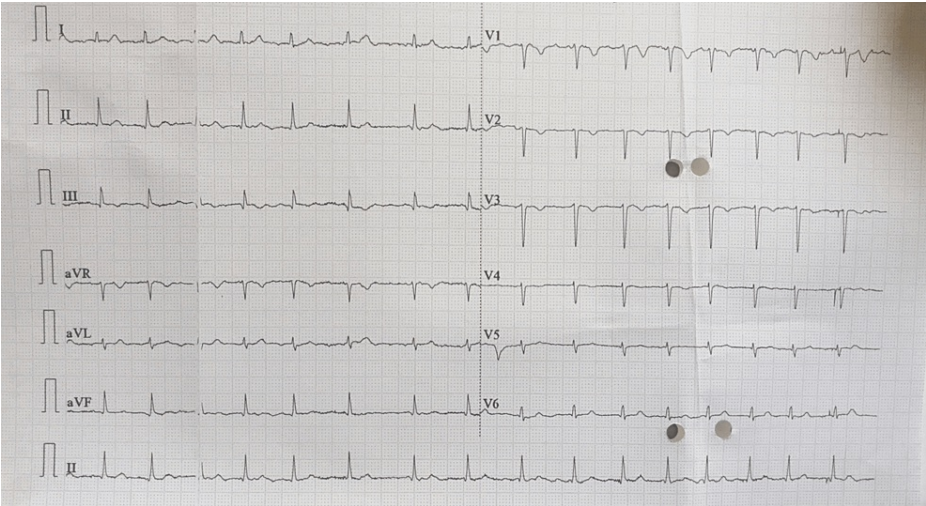
FIGURE 1: Representative image of rhythm abnormality with a 12 lead ECG of 75 years female with SAH

The ECG reveals an irregular rhythm with absent p waves (atrial fibrillation) with T-wave inversion in V1 to V3.

SAH: Subarachnoid hemorrhage

Morphological abnormality

Out of the 34 patients with some form of morphological abnormality, a total of 12 (26.6%) patients had morphological changes without rhythm abnormality. The most frequent of these was abnormal QTc. Sixteen (35.5%) patients had prolonged QTc interval. Figure 2 shows a representative image showing the most commonly encountered abnormality, i.e., prolonged QTc.
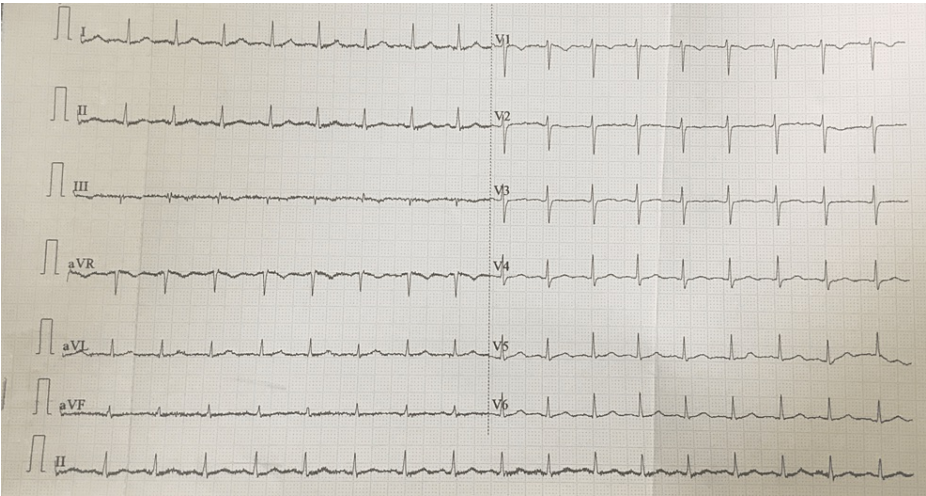

FIGURE 2: 12-lead ECG of a 34-year-old female with SAH

Recordings reveal prolonged QTc of 468 with nonspecific T- wave changes.

SAH: Subarachnoid Hemorrhage

T wave abnormality was also frequently encountered. A total of 11 (24.4%) patients had a T wave abnormality. Seven patients (15.5%) developed a T wave inversion.

Left axis deviation was present in seven (15.5%) cases. P mitrale was encountered in four (8.8%) and ST Depression in four (8.8%) cases. None of the patients had ST elevation. One patient presented as a left bundle branch block. U wave was detected in five (11.1%) patients.

Discussion

The mechanism by which ECG changes occur in SAH is not clear. It has been suggested that SAH has been associated with raised plasma norepinephrine and autonomic sympathetic activity which is primarily responsible for most of the ECG abnormality [11]. There have been animal studies where cardiac ischemic changes and arrhythmias were induced by hypothalamic stimulation [12]. There is an increased incidence of raised intracranial pressure in patients with SAH and raised intracranial pressure can mediate ECG abnormalities [13,14]. Transient coronary vasospasm following SAH may also mediate ECG changes [15].

Prevalence

The reported prevalence of ECG changes in SAH ranges from 71 to 100% [3-5]. In our study, 88.8% of patients had some form of ECG abnormalities. Reported prevalence is higher in studies in which ECG recordings were retrieved within 48 hours or serial ECG recording was done [5]. In our study, the most common ECG abnormalities were QTc prolongation, T-wave abnormality, and bradycardia, found in 35.5%, 24.4%, and 24.4% of patients, respectively. We also observed left axis deviation, ST depression, and U wave in a minority of patients.

In a previous study of 406 patients with SAH, Rudehill et al detected large U wave, T wave abnormality, QTc prolongation, high R wave, and ST depression in 47%, 32%, 24%, 19%, and 15%, respectively [4]. A similar study by Brouwers et al, who reviewed serial ECG prospectively in 61 patients, found T wave abnormality, bradycardia, ischemic ST changes, large U wave, and QTc prolongation in 59%, 50%, 50%, 44%, and 39%, respectively [5]. Compared to our study, Brouwers et al enrolled 77% of patients within 24 hours and remaining all patients within 72 hours and concluded that studies in which surveillance is started later in the course of illness may miss significant data. Excluding the two patients who presented to our center after weeks, 43 presented in 44.35 ± 29.35 hours. This delayed presentation may be attributable to fewer morphological changes than in the previous studies.

QTc changes

The most common ECG abnormality noted in our study is QTc prolongation, which is a measure of delayed ventricular repolarization. Patients with QTc prolongation are susceptible to arrhythmias including ventricular tachycardia and torsades de pointes [16]. QTc prolongation may be attributed to increased plasma catecholamines, corticosteroids, and potassium levels. In a multivariate analysis of risk factors for QT prolongation in 100 patients following SAH by Fukui et al., hypokalemia was found to be an independent risk factor for severe QTc prolongation. They recommended, a patient with SAH when hospitalized during the acute phase of SAH, as a rule, potassium-free intravenous fluids should not be used as initial fluid therapy, particularly in female patients. Avoidance of drugs that prolong ventricular repolarization (quinidine, procainamide, and disopyramide) is mandatory [17].

T wave changes

Among the T wave abnormalities, T wave inversion was the most common finding in our study. Typical transient, symmetric, and deep inverted electrocardiogram T waves in the setting of stroke are commonly referred to as cerebral T waves. Myocardial infarction is an important cardiac cause of T wave inversion [18]. There have been case reports where patients with T wave inversion, ST segment elevation, and increased troponin I were later found to have SAH in the CT scan [19]. T wave abnormalities can provide added evidence to support the clinical diagnosis but T wave must be considered along with QRS and ST segment abnormalities. T wave inversion along with prolonged QT interval is more likely to be associated with cerebrovascular accidents while that with normal QTc interval is likely associated with myocardial infarction. Inverted T waves produced by myocardial ischemia are classically narrow and symmetric [20].

ST depression

We observed four patients who had ST depression and all of them had ST depression in three or more precordial leads. But we did not encounter any cases of ST elevations. There has been a case report where a patient has had an off-hospital cardiac arrest and had ST elevation with increased cardiac biomarker but normal coronary angiography and was later found to have SAH. In a study of 15 cases of electrocardiographic abnormalities suggestive of recent myocardial infarction or ischemia in association with SAH four patients with such abnormalities died and had a normal heart autopsy [21]. They concluded without a good history or clinical findings of heart disease, an electrocardiogram showing changes suggestive of recent infarction or acute myocardial ischemia should be interpreted as indicative of intracranial rather than of heart disease. A minority of our patients had left axis deviation and U wave but their prevalence is reportedly higher in other studies [22]. Also, their clinical significance and implication are minimal.

Rhythm abnormalities

Cardiac arrhythmias in SAH are thought to occur secondary to acute central nervous dysfunction and sudden increased intracranial pressure. Rhythm abnormalities were relatively rare than morphological abnormalities

2023 Poudel et al. Cureus 15(6): e40045. DOI 10.7759/cureus.40045

7 of 9

in our study. Sinus bradycardia was observed in 24.4% of our cases with one case of atrial fibrillation and one case of premature ventricular contractions. In a prospective study of 580 spontaneous SAH patients by Frontera et al cardiac arrhythmias occurred in 4.3% of patients with atrial fibrillation and flutter being the most common finding [23]. They concluded that arrhythmias were associated with an excess ICU stay of five days and an increased risk of death or severe disability. Di Pasquale et al detected cardiac arrhythmias in 90% of the patients by Holter monitoring and the frequency and severity were higher in subjects studied within 48 hours [24]. There have been case reports where SAH has been associated with life-threatening arrhythmias [25]. So, all patients with SAH must be kept under continuous cardiac rhythm monitoring in Neuro-ICU, especially in the first 48 hours for early detection of life-threatening arrhythmias and prevention of their complications.

Implications

ECG changes, though common in SAH, are not an accurate predictor of myocardial dysfunction, rather myocardial dysfunction is related more closely to the severity of neurological dysfunction caused by SAH [26]. However, it should be acknowledged that ECG abnormalities, specifically corrected QT interval, are independently associated with an increased risk of neurogenic pulmonary edema and delayed cerebral ischemia, whereas ST depression is independently associated with an increased risk for in-hospital death [27]. The correct interpretation of ECG is required to prevent delays in treatment and wasteful investigations. Similarly, continuous cardiac rhythm monitoring of patients with SAH is essential for early detection and treatment of life-threatening cardiac arrhythmias. QT prolongation, a common finding in SAH, warrants avoidance of potassium-free fluid as initial fluid therapy and drugs prolonging ventricular repolarization. Proper knowledge about the pathophysiology of ECG changes in SAH patients may make heart donations possible in brain-dead patients of SAH with ECG abnormalities [22].

Conclusions

This study demonstrates that both morphological and rhythm abnormalities are frequently encountered in patients with SAH. Eighty-eight percent of patients had some form of ECG abnormality. QTc prolongation was most commonly encountered. Though ST-T changes and U wave were among the most frequent abnormalities in past studies, we observed few such cases. We recommend further prospective studies with serial ECG or Holter recording in study populations like ours.

Additional Information

Disclosures

Human subjects: Consent was obtained or waived by all participants in this study. Institutional Review Committee, Institute of Medicine (IOM), Tribhuvan University issued approval 501(6-11)E2. The research proposal was submitted to and approved by the Institutional Review Committee (IRC) of the Institute of Medicine (IOM), Tribhuvan University on April 20, 2023. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. García-Ballestas E, Durango-Espinosa Y, Mendoza-Flórez R, Moscote-Salazar LR, Keni R, Deora H, Agrawal A: The puzzle of spontaneous versus traumatic subarachnoid hemorrhage . Apollo Med. 2019, 16:141-47.
2. Shea AM, Reed SD, Curtis LH, Alexander MJ, Villani JJ, Schulman KA: Characteristics of nontraumatic subarachnoid hemorrhage in the United States in 2003. Neurosurgery. 2007, 61:1131-7; discussion 1137-8. 10.1227/01.neu.0000306090.30517.ae
3. Kreus KE, Kemilä SJ, Takala JK: Electrocardiographic changes in cerebrovascular accidents . Acta Med Scand. 1969, 185:327-34. 10.1111/j.0954-6820.1969.tb07342.x
4. Rudehill A, Olsson GL, Sundqvist K, Gordon E: ECG abnormalities in patients with subarachnoid haemorrhage and intracranial tumours. J Neurol Neurosurg Psychiatry. 1987, 50:1375-81. 10.1136/jnnp.50.10.1375
5. Brouwers PJ, Wijdicks EF, Hasan D, Vermeulen M, Wever EF, Frericks H, van Gijn J: Serial electrocardiographic recording in aneurysmal subarachnoid hemorrhage. Stroke. 1989, 20:1162-7. 10.1161/01.str.20.9.1162
6. Pepper J, Fenton C, Brydon H: Complete heart block as the presenting feature in subarachnoid haemorrhage. BMJ Case Rep. 2017, 2017: 10.1136/bcr-2017-222423
7. Lai CH, Juan YH, Chang SL, Lee WL, How CK, Hsu TF: Subarachnoid haemorrhage mimicking transient ST-segment elevation myocardial infarction. Acta Clin Belg. 2015, 70:304-6. 10.1179/2295333715Y.0000000019
8. Chen WL, Huang CH, Chen JH, Tai HC, Chang SH, Wang YC: ECG abnormalities predict neurogenic pulmonary edema in patients with subarachnoid hemorrhage. Am J Emerg Med. 2016, 34:79-82. 10.1016/j.ajem.2015.09.032
9. Hunt WE, Hess RM: Surgical risk as related to time of intervention in the repair of intracranial aneurysms . J

Neurosurg. 1968, 28:14-20. 10.3171/jns.1968.28.1.0014

10. Report of World Federation of Neurological Surgeons committee on a universal subarachnoid hemorrhage grading scale. J Neurosurg. 1988, 68:985-6. 10.3171/jns.1988.68.6.0985

11. Myers MG, Norris JW, Hachniski VC, Sole MJ: Plasma norepinephrine in stroke. Stroke. 1981, 12:200-4. 10.1161/01.str.12.2.200

12. Melville KI, Blum B, Shister HE, Silver MD: Cardiac ischemic changes and arrhythmias induced by hypothalamic stimulation. Am J Cardiol. 1963, 1:781-91. 10.1016/0002-9149(63)90281-9

13. Zoerle T, Lombardo A, Colombo A, Longhi L, Zanier ER, Rampini P, Stocchetti N: Intracranial pressure after subarachnoid hemorrhage. Crit Care Med. 2015, 43:168-76. 10.1097/CCM.0000000000000670

14. Jachuck SJ, Ramani PS, Clark F, Kalbag RM: Electrocardiographic abnormalities associated with raised intracranial pressure. Br Med J. 1975, 1:242-44. 10.1136%2Fbmj.1.5952.242

15. Yuki K, Kodama Y, Onda J, Emoto K, Morimoto T, Uozumi T: Coronary vasospasm following subarachnoid hemorrhage as a cause of stunned myocardium. Case report. J Neurosurg. 1991, 75:308-11. 10.3171/jns.1991.75.2.0308

16. Lanzino G, Kongable GL, Kassell NF: Electrocardiographic abnormalities after nontraumatic subarachnoid hemorrhage. J Neurosurg Anesthesiol. 1994, 6:156-62. 10.1097/00008506-199407000-00002

17. Fukui S, Katoh H, Tsuzuki N, et al.: Multivariate analysis of risk factors for QT prolongation following subarachnoid hemorrhage. Crit Care. 2003, 7:1-6. 10.1186%2Fcc2160

18. Stone J, Mor-Avi V, Ardelt A, Lang RM: Frequency of inverted electrocardiographic T waves (cerebral T waves) in patients with acute strokes and their relation to left ventricular wall motion abnormalities. Am J Cardiol. 2018, 121:120-4. 10.1016/j.amjcard.2017.09.025

19. Saritemur M, Akoz A, Kalkan K, Emet M: Intracranial hemorrhage with electrocardiographic abnormalities and troponin elevation. Am J Emerg Med. 2013, 31:271.e5-7. 10.1016/j.ajem.2012.05.007

20. Catanzaro JN, Meraj PM, Zheng S, Bloom G, Roethel M, Makaryus AN: Electrocardiographic T-wave changes underlying acute cardiac and cerebral events. Am J Emerg Med. 2008, 26:716-20. 10.1016/j.ajem.2007.10.017

21. Cropp GJ, Manning GW: Electrocardiographic changes simulating myocardial ischemia and infarction associated with spontaneous intracranial hemorrhage. Circulation. 1960, 22:25-38. 10.1161/01.cir.22.1.25

22. Chatterjee S: ECG changes in subarachnoid haemorrhage: a synopsis . Neth Heart J. 2011, 19:31-4. 10.1007%2Fs12471-010-0049-1

23. Frontera JA, Parra A, Shimbo D, et al.: Cardiac arrhythmias after subarachnoid hemorrhage: risk factors and impact on outcome. Cerebrovasc Dis. 2008, 26:71-8. 10.1159%2F000135711

24. Di Pasquale G, Pinelli G, Andreoli A, Manini G, Grazi P, Tognetti F: Holter detection of cardiac arrhythmias in intracranial subarachnoid hemorrhage. Am J Cardiol. 1987, 59:596-600. 10.1016/0002-9149(87)91176-3

25. Estanol BV, Marin OS: Cardiac arrhythmias and sudden death in subarachnoid hemorrhage . Stroke. 1975, 6:382-6. 10.1161/01.str.6.4.382

26. Davies KR, Gelb AW, Manninen PH, Boughner DR, Bisnaire D: Cardiac function in aneurysmal subarachnoid haemorrhage: a study of electrocardiographic and echocardiographic abnormalities. Br J Anaesth. 1991, 67:58-63. 10.1093/bja/67.1.58

27. Zhang L, Qi S: Electrocardiographic abnormalities predict adverse clinical outcomes in patients with subarachnoid hemorrhage. J Stroke Cerebrovasc Dis. 2016, 25:2653-9. 10.1016/j.jstrokecerebrovasdis.2016.07.011

2023 Poudel et al. Cureus 15(6): e40045. DOI 10.7759/cureus.40045

9 of 9