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Original Research Article

ELECTROCARDIOGRAPHIC CHANGES IN PATIENTS WITH SUBARACHNOID HEMORRHAGE - A DESCRIPTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Subarachnoid haemorrhage (SAH) is a serious form of cerebrovascular disease frequently associated with electrocardiographic (ECG) abnormalities that may mimic primary cardiac pathology and complicate clinical decision-making. These changes are believed to result from sympathetic overactivity and autonomic dysfunction following intracranial bleeding. The present study was undertaken to evaluate the pattern and frequency of ECG changes in patients with SAH.

Materials and Methods: This hospital-based observational study was conducted in the medicine ward of Government Medical College, Miraj, over 18 months. Fifty adult patients with radiologically confirmed SAH were included after obtaining written informed consent from close relatives. Detailed history, neurological and cardiovascular examinations, and non-contrast CT brain were performed in all cases. Routine laboratory investigations, including electrolytes were done to exclude electrolyte-related ECG changes. A 12-lead ECG was recorded within 24 hours of admission Two-dimensional echocardiography was performed in patients found to have ECG abnormalities. Patients with prior cardiac disease, congenital heart disease, previous ECG abnormalities, electrolyte imbalance, pregnancy, cerebral venous sinus thrombosis, and age below 18 years were excluded.

Results: The highest incidence of SAH was observed in the 51-60 year age group (28%), followed by 61-70 years (24%). Males constituted 60% of the study population. T-wave abnormalities were the most frequent ECG finding (34%), predominantly T-wave inversion (32%). Rhythm disturbances were present in 26%, with sinus tachycardia being the most common (22%). ST-segment abnormalities were noted in 16% of cases. QTc prolongation was seen in 8% and abnormal U waves were present in 4%. Electrolyte levels were usually normal in all affected patients.

Conclusion: ECG abnormalities are common in cases of SAH. T-wave inversion was the most frequent ECG finding in studied cases. This was followed by rhythm disturbances and ST-segment changes. Recognition of these neurogenic ECG changes is important to avoid misdiagnosis as primary cardiac disease and prevent inappropriate and unnecessary intervention.

Keywords: Subarachnoid Hemorrhage, Electrocardiography, Stroke, Cardiac Arrhythmias, Sympathetic Nervous System.

INTRODUCTION

Cerebrovascular disease includes some of the most common devastating disorders ischemic stroke, hemorrhagic stroke, cerebrovascular anomalies such as intracranial aneurysm and AV malformation. Intracranial hemorrhage encompasses four broad types of hemorrhage: epidural hemorrhage, subdural hemorrhage, subarachnoid hemorrhage, and intraparenchymal hemorrhage. Each type of

hemorrhage is different concerning aetiology, findings, prognosis, and outcome. Haemorrhagic stroke accounts for 10% of all stroke associated with 50% case of fatality rate. Hypertension, trauma, cerebral amyloid angiopathy cause major of the haemorrhage. Aneurysmal subarachnoid hemorrhage, hypertension intracranial hemorrhage is the important cause. Common sites are basal ganglia, (putamen), thalamus cerebellum and pons. In non-hypertensive patient hemorrhagic disorders, neoplasm, vascular malformation are the causes.[1]

In 1947, Byer and colleagues first described marked QT prolongation with large T and U waves on the ECG of four patients with stroke. Subsequently Burch and colleagues described an ECG pattern after stroke consisting of large inverted T waves, prolonged QT intervals and large septal U waves that has become distinctive of cerebral vascular injury.[2]

Subarachnoid hemorrhage (SAH) is bleeding into the subarachnoid space—the area between the arachnoid membrane and the pia mater surrounding the brain. SAH may occur as a result of a head injury or spontaneously, usually from a ruptured cerebral aneurysm. Risk factors for spontaneous cases include high blood pressure, smoking, family history, alcoholism, and cocaine use. Generally, the diagnosis can be determined by a CT scan of the head if done within six hours of symptom onset. Occasionally, a lumbar puncture is also required. After confirmation further tests are usually performed to determine the underlying cause. In 85 percent of spontaneous cases the cause is a cerebral aneurysm. In 15–20 percent of cases of spontaneous SAH, no aneurysm is detected on the first angiogram. About half of these are attributed to non-aneurysmal perimesencephalic hemorrhage, in which the blood is limited to the subarachnoid spaces around the midbrain (i.e., mesencephalon).[3]

The combination of intracerebral hemorrhage and raised intracranial pressure (if present) leads to a "sympathetic surge", i.e., over-activation of the sympathetic system. This is thought to occur through two mechanisms, a direct effect on the medulla that leads to activation of the descending sympathetic nervous system and a local release of inflammatory mediators that circulate to the peripheral circulation where they activate the sympathetic system. As a consequence of the sympathetic surge there is a sudden increase in blood pressure; mediated by increased contractility of the ventricle and increased vasoconstriction leading to increased systemic vascular resistance. The consequences of this sympathetic surge can be sudden, severe, and are frequently life-threatening. The high plasma concentrations of adrenaline also may cause cardiac arrhythmias (irregularities in the heart rate and rhythm), electrocardiographic changes (in 27 percent of cases) and cardiac arrest (in 3 percent of cases) may occur rapidly after the onset of hemorrhage. A further consequence of this process is neurogenic pulmonary edema where a process of increased pressure within the pulmonary circulation causes leaking of fluid from the pulmonary capillaries into the air spaces, the alveoli, of the lung.[4]

Hypothalamic stimulations and autonomic dysfunctions have been linked but not conclusively proven to be causative. These considerations may influence therapeutic interventions as infusions of large volumes of fluids or administrations of vasopressors may prove detrimental in patients with a compromised heart. ECG changes in SAH commonly reflect ischaemic heart disease and have been known to present with ST-segment elevation and T-wave inversion.[5] Therapeutic thrombolytic therapy and anticoagulation as well as withholding of life-saving neurosurgery in such cases may well endanger the life of the patient concerned. SAH has been associated with malignant ventricular arrhythmias, including ventricular tachycardia, torsades de pointes, and ventricular fibrillation, particularly if the corrected QT (QTc) interval is prolonged, often leading to compromise in a haemodynamically unstable patient.

MATERIALS AND METHODS

This hospital-based observational study was conducted in the medicine ward of a tertiary care centre over a period of 18 months. The study was carried out at Government Medical College, Miraj. All adult patients diagnosed with subarachnoid haemorrhage on the basis of radiological findings and whose close relatives were willing to give written informed consent were considered for inclusion in the study. A total of 50 patients were selected accordingly. It was an observational study conducted on eligible patients during the specified study period. After admission, a detailed history was obtained regarding the temporal profile of stroke, including history of risk factors such as hypertension, diabetes mellitus, smoking, trauma, history of ischemic heart disease, and rheumatic heart disease. Detailed neurological examination, including fundoscopy, as well as cardiovascular examination, was carried out in all cases. The diagnosis of cerebrovascular accident was made on the basis of the temporal profile of the clinical syndrome, clinical examination, and non-contrast computed tomography (NCCT) scan of the brain. CT brain was performed within 24 to 48 hours. Patients diagnosed with subarachnoid haemorrhage based on radiological findings were further studied. [Figure 1]

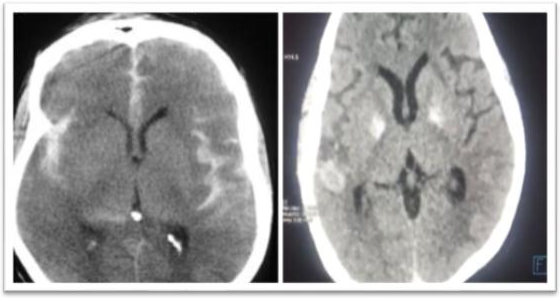

Figure 1: Left: Axial non-contrast CT brain showing acute subarachnoid hemorrhage with hyperdense blood in the bilateral sylvian fissures and adjacent cortical sulci. Right: Axial non-contrast CT brain showing subarachnoid hemorrhage in the basal cisterns/sylvian fissures with mild ventricular prominence suggestive of early hydrocephalus. [Figure 1]

Routine investigations included serum electrolytes, namely sodium, potassium, and magnesium, along with complete blood count, renal function tests, chest X-ray, and 12-lead ECG, which was taken and monitored for the next 72 hours. The reference values considered were serum sodium 135–145 mEq/L, potassium 3.5–5.0 mEq/L, and magnesium 0.70–0.95 mmol/L.

Patients was subjected to 12-lead ECG and 2D echocardiography within 24 hours of admission and was followed up for newly occurring changes. ECG was interpreted for rate, rhythm, ST-segment changes, QRS complex, T waves with their amplitude and morphology, and corrected QT interval. Heart rate less than 60/min was regarded as bradycardia and heart rate exceeding 100/min was regarded as tachycardia. Normal P wave duration was considered as less than or equal to 0.11 seconds. PR interval between 0.12 and 0.20 seconds was taken as normal, and QRS complex duration less than or equal to 0.12 seconds was considered normal. ST-segment depression of 0.5 mm or elevation of more than 1 mm was taken as abnormal. T wave was considered abnormal when inversion was present in leads in which it is normally upright, namely leads I, II, and V3–V6, while it may be variable in leads III, aVL, V1, and V2. QTc prolongation was defined when the corrected QT interval exceeded 0.44 seconds; QTc was calculated by dividing the actual QT interval by the square root of the RR interval, both measured in seconds. U wave was taken as significant when exaggeration of U wave voltage was noted in more than two leads or when it appeared in leads in which it is not normally seen, other than V3–V4. Left ventricular hypertrophy was diagnosed if the sum of the depth of the S wave in lead V1 and the height of the R wave in either lead V5 or V6 exceeded 35 mm, or if the R wave in lead aVL measured 11 to 13 mm or more. Right ventricular hypertrophy was diagnosed by the presence of tall R waves in right chest leads, with the R wave taller than the S wave in lead V1, and persistent S wave in leads V5–V6.

Two-dimensional echocardiographic screening was done in all patients with ECG changes. Left ventricular ejection fraction was used to assess left ventricular systolic function, while Doppler indices across the mitral valve (A>E) were used to assess left ventricular diastolic dysfunction. Mitral valve opening using planimetry was used to detect mitral stenosis; in addition, valve thickening, doming of the anterior mitral leaflet, and paradoxical motion of the posterior mitral leaflet were also assessed. Flow across the aortic valve was used to look for aortic stenosis, including valve opening, and severe aortic stenosis was considered if aortic valve opening was less than 8 mm. Colour imaging and Doppler were utilized for detecting any regurgitation. Patients showing abnormal chest X-rays with cardiomegaly were excluded from the study. Patients with previous history of ischemic heart disease, known congenital cardiac anomalies, previously documented ECG abnormalities, and diagnosed electrolyte abnormalities were also excluded from the study.

Inclusion Criteria

- Subarachnoid haemorrhage patient with close relatives willing to give written informed consent
- Age of subjects more than 18 years belonging to both gender with diagnosed on imaging

Exclusion Criteria

- Age less than 18 years.
- Pregnant females.
- Patient with Cerebral Venous Sinus Thrombosis.
- Patients with electrolyte abnormalities.
- Patients with cardiac diseases diagnosed 2 d echo.
- Patients with congenital cardiac anomalies.
- Patients with previous abnormalities in ECG

RESULTS

A total of 50 patients diagnosed with subarachnoid hemorrhage who met the inclusion and exclusion criteria were analysed for electrocardiographic changes during the study period. All patients included in the study were observed for ECG changes and a detailed analysis of the ECG of each patient was performed.

Analysis of age and gender distribution revealed that the highest incidence of subarachnoid hemorrhage was in the 51-60 year age group (28%) followed by the 61-70 year age group (24%), with over half of all cases occurring in the 5th, 6th, and 7th decades of life. [Table 1]

Table 1: Age distribution of the study population (n=50)

Age (years)	Males	Females	Total	Percentage
21-30	3	0	3	6%
31-40	3	2	5	10%
41-50	5	3	8	16%
51-60	9	5	14	28%
61-70	6	6	12	24%
71-80	4	4	8	16%
Total	**30**	**20**	**50**	**100%**

Analysis of depolarization parameters revealed that P wave morphology, PR interval, and QRS complex duration were within normal limits in all 50 patients in the study group.

Rhythm disturbances were present in 13 patients (26%) while 37 patients (74%) maintained normal sinus rhythm throughout the monitoring period. [Table 2]

Table 2: Incidence of rhythm disturbances in the study group (n=50)

Sr. No.	Category	No. of patients (n=50)	Percentage
1	Rhythm abnormality	13	26%
2	Normal rhythm	37	74%
	Total	**50**	**100%**

ST segment abnormalities were observed in 8 patients (16%) while 42 patients (84%) had normal ST segments. Among patients with ST segment changes, ST segment depression (14%) was more frequent than ST segment elevation (2%). [Table 3]

Table 3: Incidence and distribution of ST segment changes in the study group (n=50)

Category	Sub-category	No. of patients (n=50)	Percentage
ST segment changes	ST segment elevation	1	2%
	ST segment depression	7	14%
	Total	**8**	**16%**
No ST segment changes	-	42	84%
Total		**50**	**100%**

T wave abnormalities were the most common ECG finding, present in 17 patients (34%). Among these, T wave inversions constituted the vast majority observed in 16 patients (32%), while tall T waves were observed in only 1 patient (2%). [Table 4]

Table 4: Incidence and distribution of T wave changes in the study population (n=50)

Category	Sub-category	No. of patients (n=50)	Percentage
T wave abnormality	T wave inversions	16	32%
	Tall T waves	1	2%
	Total	**17**	**34%**
Normal T wave	-	33	66%
Total		**50**	**100%**

Prolonged QTc interval was observed in 4 patients (8%). No cases of shortened QTc interval were recorded in the study group. [Table 5]

Table 5: Incidence of QTc interval abnormalities in the study group (n=50)

Sr. No.	Category	No. of patients (n=50)	Percentage
1	Prolonged QTc	4	8%
2	Shortened QTc	0	0%
	Total	**4**	**8%**

Abnormal U waves were present in 2 patients (4%) while 48 patients (96%) had no abnormal U waves. [Table 6]

Table 6: Incidence of abnormal U waves in the study group (n=50)

Sr. No.	Category	No. of patients (n=50)	Percentage
1	Abnormal U waves present	2	4%
2	No abnormal U waves	48	96%
	Total	**50**	**100%**

Overall, the most frequently observed ECG abnormality was abnormal T waves (34%), followed by rhythm abnormalities (26%), ST segment changes (16%), prolonged QTc interval (8%), and abnormal U waves (4%). Some patients had more than one ECG abnormality. [Table 7]

Table 7: Overall ECG findings in the study group (n=50)

Sr. No.	ECG Abnormality	No. of patients	Percentage
1	Abnormal T waves	17	34%
2	Rhythm abnormality	13	26%
3	ST segment changes	8	16%
4	Abnormal QTc	4	8%
5	Abnormal U waves	2	4%

DISCUSSION

The present study was carried out in medicine ward at Government Medical College Miraj, over a period of 18 months. A total of 50 patients diagnosed with subarachnoid hemorrhage based on radiological findings were studied for electrocardiographic changes.[12] lead ECG was taken for all the patients at admission and they were monitored for the next 72 hours. CT brain scan was taken within 24-48 hours. Patients with previous ECG abnormalities, known cardiac disease, and electrolyte disturbances were excluded from the study. In our study, a considerable number of patients had ECG changes.

The most common ECG finding observed in cases of subarachnoid hemorrhage was abnormal T waves which were observed in 34% of cases. Among the T wave abnormalities, T wave inversion was the most common. This was consistent with the study of Cruickshank et al, who observed tall T waves and short PR interval in their study on cerebrovascular accidents.[6] The study of Hugenholtz showed extremely inverted and wide T waves, prominent U waves and prolongation of QTc interval.7 Burch and colleagues described abnormal T waves, prominent U waves, and prolonged QTc in patients with CVA. 8 Crop and Manning analysed ECG in 29 patients with SAH and found flat or negative T waves in 15 patients, prolonged QTc in 14, and ischemic ST segment changes in 11 patients.[9] Miller and Abildskov studied 50 patients and found that most had non-specific ST-T wave abnormalities with a high incidence of notched T waves.[11]

The second most common abnormality among the patients of subarachnoid hemorrhage was rhythm abnormalities which were observed among 26% of the cases. Among the rhythm abnormalities, sinus tachycardia was the most common finding observed in 22% of the cases. Other uncommon abnormalities that were observed included sinus bradycardia and atrial fibrillation. Schuster had observed prolonged QTc and bradycardia as characteristic of SAH.[12] Di Pasquale et al obtained a 12-lead ECG at the time of admission from a sample of 120 patients with SAH. Holter monitoring was started on the same day and cardiac arrhythmias were detected in 90% of the 107 adequate Holter recordings. Torsades de pointes occurred in four patients and progressed to ventricular fibrillation and asystole in one of the four.[13]

The third most common abnormality observed among the patients of subarachnoid hemorrhage were ST segment abnormalities. ST segment abnormalities were observed among 16% of the cases, with the most common being ST segment depression. This finding was consistent with the study of Frentz and Gormsen who observed depression of ST segment as the most common abnormality in 11 out of 15 patients with intracerebral hemorrhage.[18] Kreus and his coworkers observed a high frequency of ECG abnormalities in CNS lesions, with 25 out of 35 patients with SAH showing ECG changes.[14]

None of the patients with ECG abnormalities had altered electrolyte values, showing that these ECG changes were not associated with electrolyte disturbance and their pathogenesis is different. The patient with stroke and with an abnormal ECG represents a common diagnostic challenge to the clinician, because ECG changes in stroke mimic the findings of myocardial ischemia, rhythm disorders, and other cardiac disorders. Bozuluolcay M et al reported that the third day mortality was 14.8% in patients with ECG abnormalities whereas it was 8.5% in the patients without ECG changes.[15] Understanding that these ECG changes occurring in patients with CVA is important because it may lead to erroneous judgment of assigning these patients as having coronary artery disease. These patients should be evaluated for cardiac injury and treated only if necessary. Therapeutic thrombolytic therapy and anticoagulation as well as withholding of life-saving neurosurgery in such cases may well endanger the life of the patient concerned.

CONCLUSION

Subarachnoid hemorrhage was most common in the 6th and 7th decade with male preponderance. ECG changes were frequently associated with subarachnoid hemorrhage. The most common ECG abnormality was T wave inversion, followed by rhythm disturbances and ST segment changes. All patients with ECG abnormalities had normal electrolyte levels, confirming a neurogenic rather than metabolic origin. Recognizing these neurogenic ECG changes is critical to avoid misdiagnosis as primary coronary artery disease and prevent inappropriate therapeutic interventions.

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