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Original Research Article

A Study of Analysis of Different Electrocardiographic Changes in Acute Cerebrovascular Accidents

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Abstract:

Introduction: A wide number of studies have demonstrated the fact that primary neurologic abnormalities may produce ECG changes without any myocardial lesion. Objectives of the study were to evaluate pattern of ECG changes resulting from cerebrovascular accidents to correlate pathophysiology of lesion with ECG changes in patients with cerebrovascular accident; and to correlate localization of lesion with ECG changes in cerebrovascular accident.

Materials and Methods: This observational correlational study, with 100 CVA patients admitted within 72 hours of onset, was conducted in SBLS Civil Hospital, Jalandhar, Punjab, from 2021to 2022. CT scan, ECG and blood investigations were done, and findings recorded.

Results: Rhythm Disturbances- seen in 35% patients. Sinus tachycardia (ST) was noted in 15% patients, sinus bradycardia in 11% patients, atrial fibrillation (AF) in 5% patients and supra-ventricular tachycardia (SVT) in 4% patients. Presence of LVH- Left Ventricular Hypertrophy was seen in 25% patients. T wave abnormality- was noted in 45% cases. 38% cases showed inverted T- wave and 7% cases showed tall T-wave. In the maximum number of cases (30%), lesions were localized in basal ganglion, followed by 15% cases with capsuloganglionic location and 12% lesions localized in frontal region. Out of 29 cases with cerebral haemorrhage, 17 cases had abnormal ECG and out of 7 cases with SAH, 6 cases had abnormal ECG.

Conclusion: patients with SAH had maximum Percentage of ECG changes and minimum with cerebral haemorrhage. Lesions localized to basal ganglia and capsuloganglionic were associated with most abnormal ECG changes. But further studies with greater sample size are required to clarify the causal connection more precisely between the ECG abnormalities and the intracranial lesion.

Keywords: ECG, Cerebral haemorrhage, cerebral infarction, subarachnoid haemorrhage.

Introduction

Abnormalities of the electrocardiogram (ECG) are enormously useful in the recognition of heart disease, but abnormal ECG can also occur in a variety of states in which the primary pathology is non-cardiac. ECG abnormalities have been described with various CNS diseases like seizures, trauma, ischemic stroke, intra-cerebral brain hemorrhage (ICH), and less commonly with tumors, electroconvulsive therapy, and meningitis. Recently, emotion and stress-induced cardiomyopathy have also been described. [1-3] Electrical stimulation experiments propose an anterior parasympathetic control region and a posteriorly located area of cardiovascular sympathetic control. [4] In 1938 first time association between central nervous system (CNS) disease and ECG changes was described. [5] Cerebrovascular accident (CVA), also known as stroke is the second most common cause of death worldwide, after ischemic heart disease. The World Health Organization (WHO) defined stroke as a "neurological deficit of cerebrovascular cause that persists beyond 24 hours or is interrupted by death

within 24 hours". [6] Cerebrovascular accidents (CVA) can be classified into two major categories: ischemic and haemorrhagic. [7] Ischaemic strokes are caused by the stoppage of the blood supply to the brain, while haemorrhagic strokes result from the rupture of a blood vessel or an abnormal vascular structure. About 87% of strokes are ischemic, the rest 13% are haemorrhagic. Bleeding can also develop inside areas of ischemia, which is known as "haemorrhagic transformation." It is unknown how many haemorrhagic strokes start as ischemic strokes. [8] It has been estimated that globally the incidence of acute stroke is around 13.7 million in 2016. [9] The prevalence of stroke in India is estimated as 203 per 100,000 populations above 20 years and it comes around to a total of about 1 million cases. The male-to-female ratio is 1.7:1. [10] It is estimated that stroke represents 1.2% of the total deaths in the country when all ages are included. [11]

A wide number of studies have demonstrated the fact that primary neurologic abnormalities may produce ECG changes without any myocardial lesion. These ECG changes include effect on T wave, U wave, S-T segment, QT interval and even arrhythmias. These changes may resemble those of myocardial ischemia and acute 2 myocardial infarction, leading to misinterpretation and delay in operative management of sub-arachnoid haemorrhage. [12]

ECG abnormalities occur in 60-90% of patients with intra-parenchymal or subarachnoid bleed and in about 5-20% of patients with acute ischaemic stroke. [13] There are shreds of evidence suggesting, those patients who had ECG changes following cerebrovascular accidents have poorer prognosis compared to those who did not show any ECG changes. Approximately 2- 6% of all stroke patients die from cardiac causes in the first three months after ischemic stroke. [14]

There are only a few studies showing the correlation between electrocardiogram (ECG) changes and CVA. Hence, the present study was planned to determine the electrocardiographic changes in patients with acute cerebrovascular accident.

We aimed to evaluate the different electrocardiographic patterns associated with pathophysiological categories of acute cerebrovascular accidents among patients without any known cardiovascular disease.

Objectives of the study

1. To evaluate pattern of ECG changes resulting from cerebrovascular accident.
2. To correlate pathophysiology of lesion with ECG changes in patients with cerebrovascular accident.
3. To correlate localization of lesion with ECG changes in cerebrovascular accident.

Materials and methods: This observational correlational study was conducted in SBLS Civil Hospital, Jalandhar, Punjab, from April 2021 to April 2022. Study participants were patients suffering from acute CVA and admitted within 72 hours of onset.

Study Design:

Prospective observational study. The study included a total of 100 patients suffering from acute CVA admitted within 72 hours of onset.

Inclusion criteria

1) All the patients, male and female above the age of 18 years, who have been diagnosed with CVA and admitted within 72 hours of onset.
2) Those who were willing to give written informed consent.

Exclusion criteria

1) Traumatic brain injury within a week prior to CVA. 26
2) History of subdural hematoma.
3) Artificial pacemaker.
4) CNS conditions which mimic stroke (Hypoglycaemia, brain tumours, Todd's palsy)
5) Known cardiac disease. Patient information sheet was provided, and written informed consent was taken from all the patients included in the study.

Detailed history and physical examination were done in all the patients included in the study and necessary investigations were carried out. In patients who were clinically diagnosed with stroke, CT scan brain was done to determine whether the stroke was ischemic or haemorrhagic. Based on CT scan findings patient were categorised into 3 different types: cerebral infarction, cerebral haemorrhage and subarachnoid haemorrhage.

Electrocardiograph- a 12 lead (ECG) was recorded with sensitivity of 10 mm/mV and a paper speed of 25 mm/s of all patients. ECG was recorded at the time of admission, at 6-hour, 12-hour, 24-hour and lastly 72-hours after the admission. ECG measurements were done manually. Rate, rhythm, P wave, PR interval, QRS complex, QRS axis, LVH, ST segment, T wave, QTc interval, U wave were recorded carefully, and changes were noted. All patients included in the study were followed up during their course in the hospital.

They were treated according to the standard institutional protocol for management of CVA. Any association of ECG changes with pathophysiology and localisation of CVA was noted.

ECG changes and abnormalities were recorded according to the following criteria

- Heart rate above 100 beats/min (BPM) was considered tachycardia and heart rate below 60 beats/min was considered bradycardia. [27]
- Impulse originating from SA (sinoatrial) node which was regular in nature was considered as normal rhythm.
- P wave with amplitude of 0.25mV and duration 10 mm.
- ST segment depression of 0.5mm or elevation of more than 1 mm (but elevation of 2 mm for V1 and V2) was considered abnormal.
- Normally T wave is upright. T wave less than 0.1mV in depth was defined as inverted & T wave of height more than 10mm was considered as tall T wave.
- Corrected QT interval (QTc) = QT interval in sec/√R-R interval in sec
- QTc prolongation was considered when it was >0.45 sec in males and >0.46 sec in females.

• Abnormal U wave was considered as negative U wave with more than 0.1mv depth or positive U wave higher than 25% of T wave.

U wave was considered significant if it was visible in more than 2 leads. Apart from CT scan brain and ECG some other investigations were also carried out in these patients 28 Other investigations 1) CBC 2) LFT Profile 3) RFT Profile 4) Blood sugar 5) Lipid profile 6) Serum electrolytes 7) Serum Calcium 8) HIV/HCV/HBsAg 9) 2D echocardiogram (if required) 10)Chest X ray (if required) 11) Urine routine microscopy (if required) 12) Prothrombin time (if required) 13)Cardiac Enzyme (Trop I) 14) Other Specific radiological investigations relevant to disease Statistical analysis Statistical analyses was performed using SPSS Statistics for Windows, IBM, Version 25.0, IBM Corp. Results were presented as Mean ± SD (Min-Max) and frequency (Percentage). Inferential statistics like Chi-square test, Fischer Exact test, and independent t test was applied. The significance level adopted was 5%.

Results

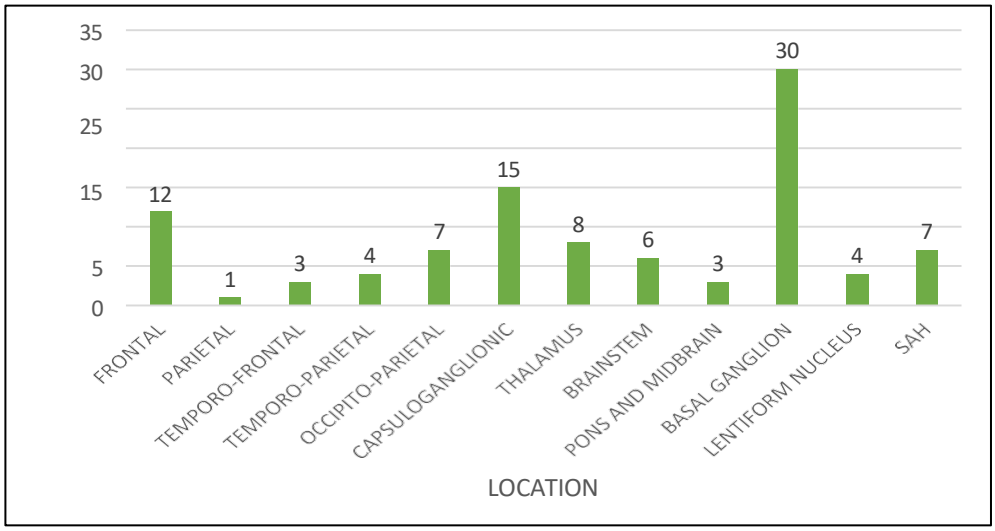
Table 1 shows the Distribution of study participants based on location of cerebral lesion. Graph 1 shows Distribution of study participants based on location of cerebral lesion. Table 2 shows ECG abnormalities by types of CVA. The maximum number of cases with rhythm disturbance were localized in capsuloganglionic region (10), followed by frontal (7), thalamus (7), sub-arachnoid (6), brainstem (2). There was 1 case each in parietal lobe, temporo-parietal region, and basal ganglion. On statistical analysis, this was not found to be significant ($p>0.05$). The maximum number of cases with LVH were localized in basal ganglion (11), followed by capsuloganglionic region (5). There were 3 cases each of temporo- parietal and occipito-parietal region, 2 cases in pons and midbrain and one case of SAH. On statistical analysis, this was not found to be significant ($p>0.05$). The maximum number of cases with abnormal T wave were localized in basal ganglion (15), followed by frontal region (9), capsuloganglionic region (8), brainstem (5). There were 3 cases each of temporo-parietal and pons and midbrain region, 1 case each of parietal region and SAH. On statistical analysis, this was not found to be significant ($p>0.05$). The maximum number of cases with abnormal ST segment were localized in basal ganglion (11), followed by frontal region (9), brainstem (5), SAH (4), temporo- parietal region (3). There was 1 case each of parietal region and capsuloganglionic region. On statistical analysis, this was not found to be significant ($p>0.05$). The cases which showed the presence of U wave were localized in thalamus (3) and basal ganglion (3). On statistical analysis, this was not found to be significant ($p>0.05$).

The maximum number of cases with prolonged QTc Interval were localized in basal ganglion (8), followed by 6 cases each of SAH and brainstem region. There were 4 cases each of frontal region and capsuloganglionic region. 3 cases were localized in thalamus and 1 in temporo-parietal region. On statistical analysis, this was not found to be significant ($p>0.05$). The maximum number of cases with prolonged PR Interval was localized in capsuloganglionic region (2). There was 1 lesion each in thalamus, basal ganglion, lentiform nucleus and sub-arachnoid region. On statistical analysis, this was not found to be significant ($p>0.05$). Rhythm disturbances were noted in 35 cases on admission, which gradually showed resolution to 32 cases at 6 hours, 28 cases at 12 hours and 24 hours, 27 cases at 48 hours and 23 cases at 72 hours. LVH was seen to be consistent in 25 cases from time of admission till 72 hours. T wave abnormality, ST segment abnormality, presence of U wave and prolonged QTc interval showed decrease in number of cases with time. Prolonged PR interval was seen to be consistent in 6 cases from time of admission till 72 hours.

Table 1: Distribution of study participants by various parameters (n=100)

Parameter			Parameter			Parameter		
age group (years)	**n**	**%**	**Risk factor present**	**n**	**%**	**T-Wave**	**n**	**%**
31-40	12	12	Hypertension	63	63	Normal	55	55
41-50	17	17	Diabetes	40	40	Inverted	38	38

51-60	31	31	Smoking	6	6	Tall	7	7
61-70	24	24	Alcoholism	23	23	**ST segment**	**n**	**%**
71-80	11	11	Previous MI	0	0	Normal	66	66
81-90	5	5	Previous stroke	10	10	Elevation	10	10
Gender	**n**	%	Lipid profile deranged	48	48	Depression	24	24
Male	63	63	**LVH present**	25	25	**U wave present**	**6**	**6**
Female	37	37	**Rhythm**	**n**	**%**	**QTc interval prolonged**	**32**	**32**
BMI	**n**	%	SR	65	65	**PR interval prolonged**	**6**	**6**
Underweight	5	5	ST	15	15	**Type of CVA**	**n**	**%**
Normal	51	51	SB	11	11	Cerebral infarction	64	64
Overweight	21	21	AF	5	5	Cerebral hemorrhage	29	29
Obese	23	23	SVT	4	4	SAH	7	7
CVA	**n (with abnormal ECG)**	%						
Cerebral infarction (64)	45	66.2						
Cerebral hemorrhage (29)	17	25						
SAH (7)	6	8.8						



Graph 1: Distribution of study participants based on location of cerebral lesion (n=100).

Table 2: ECG abnormalities by types of CVA (n=100).

ECG parameter	Total		Cerebral infarction		Cerebral haemorrhage		SAH	
Rhythm	n	%	n	%	n	%	n	%
SR	65	65	43	67.2	21	72.4	1	14.3
ST	15	15	10	15.6	4	13.8	1	14.3
SB	11	11	6	9.4	0	0	5	71.4
AF	5	5	5	7.8	0	0	0	0
SVT	4	4	0	0	4	13.8	0	0
LVH Absent	75	75	50	78.1	19	65.5	6	85.7
LVH Present	25	25	14	21.9	10	34.5	1	14.3
T wave Normal	55	53	34	53.1	15	51.7	6	85.7
T wave Inverted	38	36	23	35.9	14	48.3	1	14.3
T wave Tall	7	11	7	11	0	0	0	0
ST segment normal	66	66	45	70.3	18	62.1	3	42.9
ST elevation	10	10	7	10.9	3	10.3	0	0

ST depression	24	24	12	18.8	8	27.6	4	57.1
U wave Absent	94	94	58	90.6	29	100	7	100
U wave Present	6	6	6	9.4	0	0	0	0
QTc interval Normal	68	68	46	71.9	21	72.4	1	14.3
QTc Prolonged	32	32	18	28.1	8	27.6	6	85.7
PR interval Normal	94	94	61	95.3	27	93.1	6	85.7
PR interval prolonged	6	6	3	4.7	2	6.9	1	14.3

Discussion:

The mean age of study participants in the present study was 58.42 ± 13.07 years. This is comparable to the findings of Garg SM et al., [15] The mean age in their study was 53.00 ± 14.96 years. We observed that majority of patients 16 were in the age group 51-60 years, followed by 24 patients in age group 61-70 years, 17 patients in 41-50 years, 12 patients in 31-40 years and 11 patients in 71-80 years. There were only 5 patients in age group of 81-90 years. Our findings are consistent with Dutt R et al., [17] who also observed that majority cases in their study were in age group 51-70 years. The study participants comprised of 63% males and 37% females. This finding is in conformity with the findings of Kumar HNH et al., [18] who found 67.8% males and 32.2% females in their study. Similar findings were observed by Jaikar BSK et al., [19] who found 60 % males and 40% females. The lower incidence of stroke seen in women than men is attributable to a variety of factors which include estrogenic effects on the cerebral circulation, genetic susceptibility, and reduced blood pressure values. About 51% patients had normal BMI. Out of remaining 49%, 23% were obese, 21% were overweight and only 5% were underweight. In study done by Kumar HNH et al., [18], about 48.6% were overweight.

In our study hypertension and diabetes were reported as risk factors in 63% and 40% patients respectively. It was observed that 6% patients were smokers, and 23% patients were alcoholic. No patient had history of previous myocardial infarction. 10% patients reported history of previous stroke. 52% patients had normal lipid profile and remaining 48% had deranged lipid profile. In the study done by Kumar HNH et al., [18] there were 69.7% smokers, 48.6% alcoholics, 54.1% diabetics and 72.5% hypertensives. Stroke and heart attack both are caused by diseases of blood vessels. They share same risk factors and by modifying these risk factors may reduce the possibility of stroke. [20]

ECG Changes: The most common abnormality observed in the present study was T-wave abnormality (45%), followed by rhythm disturbances (35%), ST segment abnormality (34%), prolonged QTc interval (32%), LVH (25%), presence of U wave (6%) and prolonged PR interval (6%). Our findings are consistent with study done by Togha M et al., [21] who found that most common ECG abnormalities associated with stroke were T-wave abnormalities, prolonged QTc interval and arrhythmias in 28.9%, 30.7%, and 16.2% cases respectively. In the present study, rhythm disturbances were seen in 35% patients. Sinus tachycardia (ST) was noted in 15% patients, sinus bradycardia in 11% patients, atrial fibrillation (AF) in 5% patients and supra-ventricular tachycardia (SVT) in 4% patients. The rhythm disturbances in study by Garg SM et al., [15] included sinus tachycardia (10%), sinus bradycardia (8%), sinus arrhythmias (4%), and atrial fibrillation (4%). On the other hand, Goldstein DS [22] observed greater percentage of rhythm disturbances in their study population, sinus tachycardia (28%), sinus bradycardia (8%), sinus arrhythmias (7%), atrial fibrillation (14%). LVH was noted in 25% cases in our study. In study by Garg SM et al, [15] LVH was observed in 16% cases. T wave inversion was seen in 38% cases in the present study. Goldstein DS [22] and Purushothaman S et al., [23] observed T wave inversion in 29% and 34% cases respectively.

ST segment abnormality was observed in 34% cases, including 24% cases of depression and 10% cases of elevation in ST-segment. In studies done by Goldstein DS22 and Purushothaman S et al., [23] ST-segment depression was found in 27% and 29% cases respectively, which is consistent with our study. Goldstein DS22 observed presence of U-waves in 13 % of stroke patients. Arruda WO24 observed 12.7% patients with acute cerebrovascular hemorrhage had U-wave. QTc prolongation was observed in 32% patients in our study. In studies done by Garg SM et al., [15], it was observed in 36% cases, and this was the commonest abnormality in their study. Goldstein DS22 found prolonged QTc interval in 45% patients with stroke. Studies by Goldstein DS [22] and Purushothaman S et al., [23] found prolonged PR interval in 9% and 11% patients respectively. We found prolonged PR interval in 6% cases in our study. The studies done by Byer E [25] and Burch GE [26] stated a pattern of QT prolongation, abnormal T waves, and U waves are considered unique of acute cerebrovascular accident. Studies by Kumar HNH et al., [18] and Kuruvilla T et al., [27] also showed that the number of cases with cerebral infarction were greater than the number of cases with cerebral haemorrhage, like our study. Hemorrhagic strokes are less common than ischemic stroke but cause a remarkable number of deaths worldwide. Hemorrhagic stroke causes

severe, morbid damage to cerebral tissue that can leave individuals paralyzed or weak, with difficulty in motor activities and cognitive abilities. [28]

The present study highlights that the patients with cerebrovascular accidents often have abnormal ECG in the absence of known organic heart disease. The understanding of these ECG changes which are occurring in patients with CVA is important because it may lead to erroneous judgment of assigning these patients as CAD. The most common abnormality observed in the present study were T-wave abnormality, rhythm disturbances, ST segment abnormality and prolonged QTc interval.

Continuous ECG monitoring should be advisable to patients with acute stroke for detection of these changes and treated immediately with accurate interventions. We also attempted to determine relation of ECG changes with pathophysiology and observed that patients with SAH had maximum Percentage of ECG changes and minimum with cerebral haemorrhage. Lesions localized to basal ganglia and capsuloganglionic were associated with most abnormal ECG changes. But further studies with greater sample size are required to clarify the causal connection more precisely between the ECG abnormalities and the intracranial lesion.

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