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Original Research Article

STUDY OF ELECTROCARDIOGRAPHIC CHANGES IN ACUTE ISCHAEMIC STROKE

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ABSTRACT

Background: CAD and ischaemic cerebrovascular disease are the leading causes of morbidity and mortality. CAD often coexists with asymptomatic carotid artery atherosclerosis, TIA, or ischaemic stroke. Thus, there is increasing interest in identifying CAD in patients with cerebrovascular disease, including those without clinical manifestations of heart disease. The objective is to study the incidence and pattern of ECG changes in patient with acute ischaemic stroke. To assess the relation of ECG changes in acute ischaemic stroke to the location of cerebral lesion and to assess the treatment outcome of the acute ischaemic stroke patients while they are admitted in the hospital.

Materials and Methods: This cross-sectional observational study was conducted in Department of Internal Medicine, Rohilkhand Medical College & Hospital Bareilly, Uttar Pradesh, India. The subjects attending emergency, outdoor and indoor clinics of the Department of Internal Medicine, RMCH Bareilly, Uttar Pradesh, India suffering from Acute Ischaemic Stroke and satisfying the inclusion and exclusion criteria of study were included in the study. A total of 50 patients suffering from Acute Ischaemic Stroke were included in the study. The study was conducted from 2023 – 2025.

Results: Ischaemic stroke is a major cause of morbidity and mortality. Incidence of ischaemic stroke increases with the advancement of age. Demographic variables like age, gender; modifiable factors like HT, DM, smoking, and dyslipidemia are risk factors for the development of stroke ECG changes occur very commonly in acute ischaemic strokes. The major ECG changes were T wave inversion, Rhythm disturbance, ST segment change and QTc prolongation. The incidence of mortality is higher in stroke patients with ECG abnormalities.

Conclusion: To increase the survival period and reduce the morbidity and mortality in patients of ischaemic stroke, the prognostic factors should be identified and attempts should be done to control DM and HT. 24hours Holter monitoring should be done to all patients admitted with stroke to look for any ECG abnormalities.

Keywords: Acute Ischaemic Stroke, ischaemic cerebrovascular disease

INTRODUCTION

The World Health Organization (WHO) defines the Stroke in as “rapidly developing clinical symptoms and signs of focal disturbance of cerebral function, lasting for more than 24 hours or leading to death with no apparent cause other than that of vascular origin”.[1] On evaluation recent data has shown that about 87% of strokes are ischaemic and 13% are due to haemorrhage (intracerebral or subarachnoid).[2]

Ischaemic stroke can be classified on the basis of following various etiology: Large vessel atherosclerosis; Cardioembolic; Small vessel atherosclerosis; Other determined etiology; Undetermined etiology. Stroke is a major public health concern because of its high morbidity and

disability. It is the second commonest cause of mortality and the most common cause of morbidity.
The incidence of stroke population varies from 116-163/100,000 population.[3]
The Clinical features of the ischaemic stroke depend upon the extent and severity of involvement of specific arteries. Time and Mode of onset of the stroke might help us to determine the etiology. A large number of risk factors for stroke have been described.
Neuroimaging, especially CT (Computed Tomographic) Scan Brain helps to confirm the ischaemic strokes, rule out the stroke mimics, and find out the involved arterial territory and the extent & pattern of involvement.
Another novelty is that stroke is no more considered as unavoidable and untreatable. There is now a clear consensus that stroke is an emergency and that specialized units and teams will improve the outcome and may lower the costs.
Therapy of acute phase of ischaemic central stroke is based on immediate actions to suppress the severity of damage with the earliest possible initiation of reperfusion therapy together with the initiation and maintenance of adequate therapy to prevent further cerebral reinfarctions. The key factor for adequate and effective secondary prevention is elucidation of the etiology of ischaemic stroke because the risk of brain reinfarction is highest in the first few weeks after the episode of primary event. The exclusion of potential cardiac or vascular sources of embolization into the cerebrovascular system is very important in choosing adequate secondary prevention. Origins of embolization are very important to identify because they represent different thromboembolic risks.
Electrocardiography is frequently used as a diagnostic method after ischaemic stroke. Its routine use to elucidate the causes of stroke has role in patient groups, especially in young patients who present with cryptogenic stroke and no cardiovascular risk factors, as well as in the setting of a deep venous thrombosis, and older patients with a suspicion for structural heart disease or left ventricular or left atrial thrombus.
The essential pre-requisite for an adequate and effective secondary prevention is recognition of a cause of ischaemic stroke because the highest risk of repetition of cerebral ischaemic event is during the first few weeks after the primary CVA (Cardio vascular accident).
Therapy of an acute phase of an ischaemic stroke is focused on immediate actions to limit further brain damage with the earliest possible initiation of reperfusion therapy. The next immediate step is to initiate secondary prevention. It is to establish an adequate pharmacotherapy to prevent further stroke.
A search for possible origins of thromboembolism is an integral part of management of ischaemic stroke.[4,5]
It is also known as CVA or apoplexy but, stroke is the popular term now a days.[6]
The incidences of stroke increase exponentially with the increase in the age. Brain majorly influences the cardiac structure and its function. As the history reveals, the contemporary clinical observation and animal experimentation, there are evidences that cardiac lesions could develop as a result of any nervous system disease. Ivan Pavlov has first explained about the dysfunction of visceral organ as a result of a neurological stimulus.[7]

MATERIALS AND METHODS

This cross-sectional observational study was conducted in Department of Internal Medicine, Rohilkhand Medical College & Hospital Bareilly, Uttar Pradesh, India. The subjects attending emergency, outdoor and indoor clinics of the Department of Internal Medicine, RMCH Bareilly, Uttar Pradesh, India suffering from Acute Ischaemic Stroke and satisfying the inclusion and exclusion criteria of study were included in the study. A total of 50 patients suffering from Acute Ischaemic Stroke were included in the study. The study was conducted from 2023–2025.

Inclusion Criteria

1. All documented patients suffering from Acute Ischaemic Stroke proven by neuroimaging brain.

Exclusion Criteria

1. Patient on medication that could alter cardiac functions.
2. Patients with known cardiac illness.
3. Patients with haemorrhagic stroke.

Protocol of the study: For all the patients who fulfilled the inclusion and exclusion criteria,

- A detailed history regarding the clinical profile of Acute Ischaemic Stroke including risk factors like hypertension, DM, dyslipidemia, history of cardiac diseases and smoking was taken.
- Detailed neurological examination was done.
- investigations were done: Complete blood count, Fasting blood sugar level, Fasting lipid profile, Serum Electrolytes,

CT scan of the brain: it was done within 24 hours of admission ECG recording was done within 24 hours of admission.

Procedure for ECG recording:

- The ECG machine was calibrated so that 1 mV of signal causes a deflection of 1 cm on the ECG paper. ECG recording speed was 25mm/sec.
- The procedure was explained to patient/patient's bystanders.
- Patient was placed in supine position.
- Limb leads and chest leads were attached to the electrodes which were then placed on the patient after application of electrode gel in the following positions.

➢ Electrode on the Right Leg marked RL, function as an electrical ground.
➢ Electrode marked LL is placed on the Left Leg. Both LL and RL are placed just above the ankles.

- Electrode marked LA is attached to the Left Arm and RA to Right Arm just above the wrist.
- V1 – electrode in the 4th intercostal space just to the right of the sternum.
- V2 – electrode on the 4th intercostal space just to the left of the sternum.
- V3 – electrode placed midway between V2 and V4.
- V4 – electrode in the midclavicular line in the 5th intercostal space.
- V5 – electrode in the anterior axillary line at the same level as V4.
- V6 – electrode in the mid axillary line at the same level as V_4.

12 lead ECG recording was done. In all cases, rhythm strip (lead II) was taken. ECG printout was detached on which patient's name, inpatient number, date and time were written.

After the procedure, electrodes were removed and the sites were cleaned.

The following ECG criteria were applied for analysis.

Heart rate: Sinus bradycardia <60 beats/min Sinus tachycardia >100 beats/min

PR interval >0.2 second considered as prolonged QRS width >0.1 second: considered as wide

ST segment depression of 0.5 mm or elevation of 1 mm: considered abnormal Rate corrected QT (QTc) >0.44 second: considered as prolonged

T wave negative in lead II and V_4–V_6: considered as abnormal

Right ventricular hypertrophy (RVH):

- A tall R wave in lead V_1, equal to or larger than the S wave in that lead
- Right axis deviation
- T wave inversion in V1-V4 chest leads Left ventricular hypertrophy (LVH):
- The voltage of the S wave in lead V1 plus the voltage of the R wave in lead V5 or V6 often exceeds 35mm
- A high voltage R wave (11 to 13 mm or more) is seen in lead aVL when the QRS axis is horizontal
- Inverted T waves in leads with tall R waves
- Left axis deviation

The data thus obtained from the study was statistically analyzed.

Statistical Analysis: Data was collected, compiled and analyzed by using SPSS software version 22. Kolmogorov-Smirnov test was done to know whether the data sets were different from normal distribution or not. Normally distributed data was analyzed using ANOVA and non-normally distributed data was analyzed using non-parametric test. Descriptive statistics was calculated for quantitative variables. Frequency alongwith percentages were calculated for qualitative and categorical variables. Graphical representation of the variable was shown to understand the results clearly and categorical data was analyzed using chi- square test. The level of significance is usually denoted as alpha has the following criteria as: if $p<0.05$ then hypothesis is said to be significant.

RESULTS

Out of the total of 50 patients: 36 were male i.e 72% and 14 were female i.e 28%. Out of the total 50 patients: 26 (52%) were in the age group <60years. 24 (48%) were in the age group ≥60years.

Table 1: Baseline Characteristics

Variables			Number	Percentage
Total Patients			50	100%
Sex		Male	36	72%
		Female	14	28%
Age	<60 years	Male	19	38%
		Female	07	14%
	>60 years	Male	17	34%
		Female	07	14%
Diabetes			14	28%
Hypertension			27	54%
Smoking			23	46%
Dyslipidemia			09	18%
Prior Stroke			06	12%

Table 2: Age and Sex Distribution

Age	<60 years	Percentage	≥60 years	Percentage	P value
Males	19	73%	17	70%	$\chi 2 = 0.3116$ df=1 p=0.8599
Females	7	27%	7	30%	

[Table 2] shows that:

- 73% of the total patients are males below 60 years of age while 27% are females.
- 70% of the total patients are males above 60 years of age while 30% are females.
- Chi square value is 0.3116; degree of freedom is 1 and p-value calculated is 0.8599.

Table 3: Risk Factors in Stroke Patients

Risk Factors	<60years (N=26)		≥60 years (N=24)		Z Value (two tailed)	P value
	Male	Female	Male	Female		
Diabetes	0	1	9	4	3.96	.00008*
Hypertension	4	1	17	5	5.13	< .00001*
Smoking	4	1	15	3	3.96	.00008*
Dyslipidemia	2	2	3	2	2.40	.0164*
Prior Stroke	0	1	2	3	1.85	.06432
*The result is significant at 0.05 Significance level						

[Table 3] shows the risk factor among 50 patients: diabetes in <60years is 1 and in >60years is 13; of hypertension in <60years is 5 and in >60years is 22; of smoking in <60years is 5 and in >60years is 18; of dyslipidemia in <60years is 4 and in >60years is 5; of prior stroke in <60years is 1 and in >60years is 5 out of which all were found statistically significant except prior stroke.

Table 4: Clinical Features of Study Group

Clinical Feature	<60years (N=26)		≥60 years (N=24)	
	Male	Female	Male	Female
Lt Sided Hemiplegia	10	4	7	4
Rt Sided Hemiplegia	9	3	10	3
Headache	2	0	3	1
Vomiting	1	0	1	0
Seizure	1	0	2	0
Drowsy	0	0	2	2

[Table 4] shows that out of 50 patients, in <60years- 14 and in >60years 11 had left side hemiplegia; in <60 years 12 and in >60years 13 patients had right side hemiplegia; in <60years 2 and in >60years 4 had headache; in <60years 1 and in >60years 1 had vomiting; in <60years 1 and in >60 years 2 had seizures; and in <60years 0 and in >60years 4 had drowsiness at the time of admission.

Table 5: ECG Changes in Stroke Patients

ECG Changes	<60years (N=26)		≥60 years (N=24)		Z value (two tailed)	P value
	Number	Percentage	Number	Percentage		
Tachycardia	5	19%	8	33%	1.135	.25428
Bradycardia	4	15%	2	8%	0.767	.4413
P wave abnormality	1	4%	1	4%	0.057	.9521
QTc prolongation	2	8%	7	29%	1.974*	.04884*
ST segment elevation	1	4%	6	25%	2.1537*	.03156*
ST segment depression	0	0	5	21%	2.453*	.01428*
T wave inversion	12	46%	4	17%	2.233*	.02574*
PR interval abnormality	3	11%	2	8%	0.6532	0.5157
Arrhythmia	3	11%	10	42%	2.426*	.0151*
*The result is significant at 0.05 Significance level						

[Table 5] shows the ECG changes in the stroke patients: in <60years- 5 and in >60 years- 8 had tachycardia; in <60years- 4 and in >60years- 2 had bradycardia; in <60years- 1 and in >60years- 1 had P wave abnormality; in <60years- 2 and in >60years- 7 had QTc prolongation; in <60years- 1 and in >60years- 6 had ST segment elevation; in <60years- none and in >60years- 5 had ST segment depression; in <60years- 12 and in >60years- 4 had T wave inversion; in <60years- 13 and in >60years- 7 had PR interval abnormality (out of which 3 had short PR interval and 2 had long PR interval); in <60years- 3 and in >60years- 10 had arrhythmias (out of which 7 had Atrial Fibrillation, 3 had Brady-arrythmia and 3 had Tachy-arrythmia). Out of all ECG changes found- QTc prolongation, ST segment elevation, ST segment depression, T wave inversion and arrhythmias were found to be statistically significant.

Table 6: Electrolytes in Stroke Patients

	<60years (N=26)		≥60 years (N=24)	
	Number	Percentage	Number	Percentage
Hypernatremia	1	3.8%	0	0
Hyponatremia	0	0	0	0
Hyperkalemia	0	0	1	4.1%
Hypokalemia	0	0	0	0

[Table 6] shows that out of 50 patients suffering with stroke, in <60years- 1 (4%) and in >60years- 00 had hypernatremia; none had hyponatremia; in <60years- 00 and in >60years- 1 (4%) had hyperkalemia; none had hypokalemia.

Table 7: Location and Type of Cerebro Vascular Lesions

Cerebral Lesion	Total No. of Patients	Percentage
Basal ganglia	3	6%
Thalamus	3	6%
Gangliocapsular	16	32%
Temporo-parietal	2	4%
Fronto-parietal	4	8%
Frontal	9	18%
Parietal	5	10%
Parieto-occipital	6	12%
Occipital	2	4%

[Table 7] shows that maximum (32%) had cerebral lesion in the gangliocapsular area, followed by frontal area (18%), parieto-occipital area (12%), parietal area (10%), fronto-parietal area (8%), basal-ganglia and thalamus areas (each 6%) and temporo-parietal and occipital areas (each 4%). The mean values of- age in <60years is 45.76 ± 15.65 and in >60years is 57.9 ± 15.61; and of pulse rate in <60years is 83.26 ± 26.47 and in >60 years is 90.12 ± 27.81.

DISCUSSION

Researches have now shown that there is existence of a bidirectional interaction between the brain and the heart. Acute Ischaemic Stroke patients are extremely vulnerable to severe cardiac complications. Sympathetic hyperactivity, hypothalamic-pituitary-adrenal axis, the immune and inflammatory responses have been identified as the main pathological mechanisms involved in brain-heart axis dysregulation after Acute Ischaemic Stroke.[8]

Acute Ischaemic Stroke can contribute to impaired cerebellar autoregulation, thus making cerebral blood flow directly dependent on cardiac function.[9]

Central pathways regulating autonomic responses from the brain to the heart involves structures implicated in physiological, pathological and emotional responses.[10] When brain damage occurs, each central regulatory region triggers different pathways that depend on the injured area involved and on the extent of injury. Stimulation of the orbital surface of the frontal lobe and cingulate gyrus, for instance, alters BP and heart rate control; ischaemic lesions of the insular cortex affects BP control and trigger serious cardiac complications, such as arrhythmias and autonomic dysfunctions.[11,12]

Moreover, left hemisphere brain infarction is associated with the greater risk of adverse cardiac outcomes and increase long term mortality.[13]

Enhanced sympathetic activity and release of catecholamines after hypothalamus-pituitary-adrenal axis and autonomic activation act as a potential new molecular target for cardiac dysfunction and are associated with increased risk of MI.[14] The consequences of catecholamine surge are cardiomyocyte necrosis, hypertrophy, fibrosis and cardiac arrhythmias.

Parasympathetic connections include noradrenergic pre-ganglionic neurons in the medulla oblongata, nucleus ambiguus, vagus nerve and reticular formation. These nuclei connect with the epicardial ganglionated plexus, communicating through post-ganglionic fibers that release acetylcholine and vasoactive intestinal peptide. By binding type II muscarinic receptors, acetylcholine reduces intracellular cyclic adenosine monophosphate levels, thus slowing the speed of depolarization. Activation of this pathway results in lengthening of atrioventricular conduction time and reduces ventricular contractility.[10]

The local and systemic inflammatory response to Acute Ischaemic Stroke crosses the damaged blood brain barrier and reaches to the systemic circulation and causes possible secondary cardiac damage.

Both ischaemic and arrhythmic ECG changes are common after Acute Ischaemic Stroke within the first 24 hours.[12] Additionally, acute MI or MI like abnormalities are very common after Acute Ischaemic Stroke,[15] and are associated with an increased risk of unfavorable functional neurological outcomes and major cardiovascular events.[16]

Electrocardiographic changes occurs in 60 – 90% of Acute Ischaemic Stroke patients.[17,18] Common features include T wave inversion (35%), ST depression (33%), prolonged QTc interval (29%), and U waves (28%).[19] AF, supraventricular tachycardia, ventricular ectopic beats, ventricular tachycardia and sinus tachycardia are the most common arrhythmias after Acute Ischaemic Stroke.[20] Conduction abnormalities are usually coupled with acute hemodynamic instability, which is associated with increased morbidity and mortality after Acute Ischaemic Stroke.[21] Furthermore, a history of heart failure, Acute Ischaemic Stroke severity, QTc interval and ventricular extrasystoles are independent risk factors for serious cardiac events after Acute Ischaemic Stroke.[22]

Study population consisted of 50 cases of which 36 patients (72%) were male and 14 patients (28%) were female. 24 patients belonged to age group >60years and 26 patients belonged to age <60years.

Patients with previous cardiac abnormalities were excluded from the study. 12 lead ECG and Neuroimaging (NCCT head/ MRI brain) was taken within 24-48 hours and analyzed and the patients were categorized as Acute Ischaemic Stroke. The male: female ratio was 2.5:1 which is comparable to

other studies done by Anand et al. (1.7:1) and Nagaraja et al. (2:1). The lower incidence of stroke in women may be attributed to genetic factors, positive effects of estrogen on the cerebral circulation or to lower blood pressure values compared to men.[23.24] Moreover, ischaemic heart disease, peripheral artery disease and cigarette smoking are more prevalent among men.[25]

In our study patients had number of ECG changes. The most common abnormality noted was T wave inversion seen in 32% of the patients. This is consistent with the study of Koochaki Ebrahim et al. where they studied 262 patients in which 112 (42%) were females and 150 (57.2%) were males.[26] The mean age was 67.5 ± 11.9. The frequency of the ECG changes observed in 179 (68.3%) of patients with acute ischaemic stroke. ECG changes observed were mostly, but not at all related to myocardial ischaemia. Inversion of T wave was the most common finding observed in 98 (37.4%) patients.

Similar study by Mansoureh Togha studied ECG changes in 361 patients of stroke and observed that 39.9% of patients had T wave inversion and concluded that ischaemia like ECG changes and arrhythmias are frequently seen in stroke patients, even in those with no history or signs of primary heart disease, which supports a central nervous system origin of these ECG abnormalities.[27]

Rhythm disturbance was observed in 26% patients. This is consistent with study of Doungporn Ruthirago et al.who studied 501 patients of acute stroke in whom 92% had ischaemic stroke and found arrhythmias in 25.1% of the patients. AF was most common arrhythmia found in the study accounting for 16%. New, AF was more common in cardioembolic than non-embolic stroke.[28]

Jine Wu et al. studied 273 patients of acute ischaemic stroke with mean age of 61.13±13.38 years and reported QTc prolongation in 23% to 45% of patients during acute stroke.[29]

Risk factors play a very important role in increasing the burden of ischaemic stroke worldwide. Findings from the study showed that the following risk factors accounted for more than 80% of the global risk of all strokes: HT, smoking, DM, abdominal obesity, diet and low physical activity.[30]

DM was found in 28% of the patients. This was found consistent with the study of Rasha H. Soliman et al. where they observed diabetes in 34.7% of the patients.[31]

We also tried to correlate the ECG changes with any specific area of cerebral lesions but couldn't find any significant relationship.

Regarding the relationship between the locations of acute ischaemic stroke lesions and ECG abnormalities Naveen P. et al. briefly noted that ECG changes appeared to have no relationship to arteriographic findings.[32]

There are some hypothesis about cardiac abnormalities during cerebral injury. The ECG changes and cardiac arrythmias frequently encountered after stroke are not solely explicable by concomitant ischaemic cardiac disease.

Excessive sympathoadrenal tone is contributary. Specifically, it is now believed that augmentation of intracardiac sympathetic nerve activity occurs, producing cardiac myocyte damage and depolarizing ionic shifts, resulting in electrocardiogram repolarization changes and arrhythmias. Experimental and clinical evidences now implicate the insular cortex and its subcortical connections in the generation of cardiac arrhythmias under stress and following hemispheric stroke. Lateralization studies indicate that destruction of areas adjacent to the right insular cortex, or involving non-cardioactive zones within this region have especially marked cardiac effects. This very likely contributes to the cardiac mortality which is the principle long term cause of death in stroke patients.[33]

CONCLUSION

Ischaemic stroke is a major cause of morbidity and mortality. Incidence of ischaemic stroke increases with the advancement of age. Demographic variables like age, gender; modifiable factors like HT, DM, smoking, and dyslipidemia are risk factors for the development of stroke ECG changes occur very commonly in acute ischaemic strokes. The major ECG changes were T wave inversion, Rhythm disturbance, ST segment change and QTc prolongation. The incidence of mortality is higher in stroke patients with ECG abnormalities. Hence, to increase the survival period and reduce the morbidity and mortality in patients of ischaemic stroke, the prognostic factors should be identified and attempts should be done to control DM and HT. 24hours Holter monitoring should be done to all patients admitted with stroke to look for any ECG abnormalities. Of all the ischaemic stroke patients assessed in this study, ECG changes of all forms were noted in the patients. T wave inversion was the most common abnormality noted in our study (32%). Next common abnormality noted was the rhythm disturbance (26%). ST segment change was observed in 24% of patients. QTc prolongation was observed in 18% of the patients. These ECG changes were not associated with any particular site of cerebral lesions. Cardiac disturbances are diverse and frequent in the setting of acute neurological injury. More importantly the presence of cardiac abnormalities has significant impact on clinical management and affects cardiac and neurological outcome.

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