

Original article :

Incidence and Pattern of ECG Changes in Patient with Cerebrovascular Accidents: An Observational Study

Arohi Kumar

Associate Professor, Department of General Medicine, Narayan Medical College & Hospital, Sasaram, Bihar, India.

Corresponding Author:

Dr. Arohi Kumar, Associate Professor, Department of General Medicine, Narayan Medical College & Hospital, Sasaram, Bihar, India.

Abstract

Background: CNS regulates the blood pressure, heart rate, vasomotor tone, and cardiac output and plays a major role in myocardial metabolism and cardiac contraction. The actual mechanism for electrocardiographic changes in acute stroke patients is unclear. An observational study of stroke patients indicated an increased incidence of sudden death among patients with right insular strokes.

Material & Methods: This is a observational study was conducted on 50 CVA patients admitted under the Department of General Medicine, Narayan Medical College & Hospital, Sasaram, Bihar, India. Patients were categorized based on the CT finding as cerebral infarction, cerebral hemorrhage and sub- arachnoid hemorrhage. ECG was then interpreted with rate, rhythm, ST segment, QRS complex, T wave amplitude and morphology and QT interval was calculated. QTC interval was calculated based on Bazetts formulae.

Results: In our study showed that the 78% of all stroke patients had some form of ECG changes, 89% of patients with hemorrhages had abnormal ECG changes, 70 % of patients with infarct had changes and 100 % of patients with SAH had changes. Maximum no. of patients with (N=31) capsuloganglion & majority of changes in ECG was seen.

Conclusion: Understanding that 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. These patients should be evaluated for cardiac injury and treated only if necessary.

KeyWords: CVA, ECG, Cerebral infarct, Cerebral hemorrhage.

INTRODUCTION

Cardiac abnormalities were described with various CNS diseases including seizures, trauma, ischemic stroke, ICH and less commonly tumors, electroconvulsive therapy and meningitis.

In every year patients suffering from acute cerebrovascular disease almost half a million people in the world. This cerebrovascular events, including ischemic stroke, intracerebral and subarachnoid hemorrhage, giving a nearly 20% of mortality rate.¹

The incidence of stroke in India was estimated as 203 per 100,000 populations above 20 years, amounting to a total of about 1 million cases. Approx 12% of all stroke appear in population below 40 years.² It is estimated that stroke present in all ages, 1.2% of mortality occurred in the country. The proportion of stroke death increases with age, 2.4% of all deaths in old age (>70 years of age).³

The physiological & anatomical pathways involved

in brain-heart interaction have been explained in both animal and human studies. The ability to propagate the arrhythmia by activation of the sympathetic nervous system represented a neurogenic mechanism.⁴ The medulla oblongata has been described as the principal site of vagal sympathetic and parasympathetic region involved in cardiac control.⁵ In addition both anatomical and physiological evidences implicate the hypothalamus in cardiac control.⁶ Electrical stimulation experiments represented a cardiovascular sympathetic control located posteriorly and parasympathetic control region located anteriorly.⁷

In fact, the CNS regulates the blood pressure, heart rate, vasomotor tone, and cardiac output and plays a major role in myocardial metabolism and cardiac contraction. The actual mechanism for electrocardiographic changes in acute stroke patients is unclear. An acute stroke event can cause catecholamine mediated cardiac changes.⁸

An observational study of stroke patients indicated an increased incidence of sudden death among patients with right insular strokes.

MATERIAL & METHODS

This is a observational study was conducted on 50 CVA patients admitted under the Department of General Medicine, Narayan Medical College & Hospital, Sasaram, Bihar, India.

Inclusion Criteria

All patients with acute cerebrovascular accidents.

Exclusion Criteria

- Patients with underlying heart diseases.
- Patients on drugs.
- Previously diagnosed patients with electrolyte abnormalities.

- Patient with hepatic or renal diseases.

12 lead ECG was taken and monitored on the day of admission. CT scan was taken within 24-48 hrs. All patients with acute cerebrovascular accidents were studied. They were evaluated with X-ray, serum electrolytes, and sugar and blood urea. Patients were categorized based on the CT finding as cerebral infarction, cerebral hemorrhage and sub- arachnoid hemorrhage. ECG was then interpreted with rate, rhythm, ST segment, QRS complex, T wave amplitude and morphology and QT interval was calculated. QTC interval was calculated based on Bazetts formulae.

RESULTS

In our study showed that the 78% of all stroke patients had some form of ECG changes, 89% of patients with hemorrhages had abnormal ECG changes, 70 % of patients with infarct had changes and 100 % of patients with SAH had changes (table 1). Maximum no. of patients with (N=31) capsuloganglion & majority of changes in ECG was seen (table 2 & 3).

DISCUSSION

Patients with previous abnormalities were excluded from the study. 12 lead ECG taken for all the patients admitted and were monitored. CT scan was taken within 24-48 hrs and analyzed and patients were categorized as cerebral infarct and intracerebral hemorrhage and SAH. In our study considerable no of patients had ECG changes. The most common abnormality noted was ST segment changes in patient with cerebral hemorrhage. 60 percent of patients had the above changes. Of which 50% had ST segment elevation and 10% had ST segment depression. These findings consistent with study of Frenzt and Gorsmen⁹ who reported an incidence of 71 % with ICH.

QTC prolongation was the next common abnormality noted in our study. 50 percent of patients with intracerebral hemorrhage had QTc prolongation. This is consistent with study of Arruda and Lacerda¹⁰ which showed 67% of patients with ICH and also study of Keller and Williams¹¹ in patients with stroke.

The next common abnormality noted was tall T waves, which was observed in 40% Of patients with intracerebral hemorrhage. This was observed in the study of Cruickshank et al,¹² who observed Tall T waves, short PR interval in their study on CVA.

T wave inversion was observed in 10% of patients with intracerebral hemorrhage and 20% patients with cerebral infarction. Rhythm disturbance was observed in 15% Of patients with cerebral infarction and 40% of patients with ICH AND 100% of patients with SAH.

Regarding the relationship between the locations of CVA lesions and ECG abnormalities, Frenz and Gormsen⁹, and Kreuz et al.¹³ briefly noted that ECG changes appeared to bear no relationship to arteriographic findings.

Recently, however Yamour et al.¹⁴, using the computerized tomographic (CT) scan, suggested that frontal lobe hemorrhages were associated especially with the ECG abnormalities of corrected QT interval (QTC) prolongation and neurogenic T waves. However in our study there

no specific correlation of ECG changes with site of cerebral lesion.

All these patients with ECG abnormalities, a screening echo were performed to rule out cardiac abnormalities associated. Sites which contribute to regulation of the cardiovascular function are known to be the anterior half of the cerebral cortex which includes the top of the frontal lobe, the motor and premotor cortex, and anterior part of the temporal lobe, hypothalamus, the limbic system, and the cerebellar hemisphere. The intimate functional connections between the hypothalamus, and posterior orbital and anterior insula and those between the hypothalamus and peripheral sympathetic nerves have also been demonstrated.

These findings suggest that the structures related to cardiovascular function are widely distributed within the central nervous system. Therefore, it is likely that CVA lesions not only in the frontal lobe, but also in the temporo-parietal lobe and basal ganglia can destroy or irritate such widely spread neurons or pathways regulating the cardiovascular system, resulting in ECG changes.

CONCLUSION

Understanding that 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. These patients should be evaluated for cardiac injury and treated only if necessary.

REFERENCES

1. Alter M, Zhang ZX, Sobel E, Fisher M, Davanipour Z, Friday G. Standardized incidence ratios of stroke: A worldwide review. *Neuroepidemiology* 1986;5:148-58.
2. Anand K, Chowdhury D, Singh KB, Pandav CS, Kapoor SK. Estimation of mortality and morbidity due to stroke in India. *Neuroepidemiology*. 2001;20:208-11.
3. Cropp D, Manning GW. Electrocardiographic changes simulating myocardial ischemia and infarction associated with spontaneous intracerebral hemorrhage. *Circulation*. 1980;12:25.
4. Levy A: The exciting causes of ventricular fibrillation in animals under chloroform anaesthesia. *Heart* 1913;4:319.

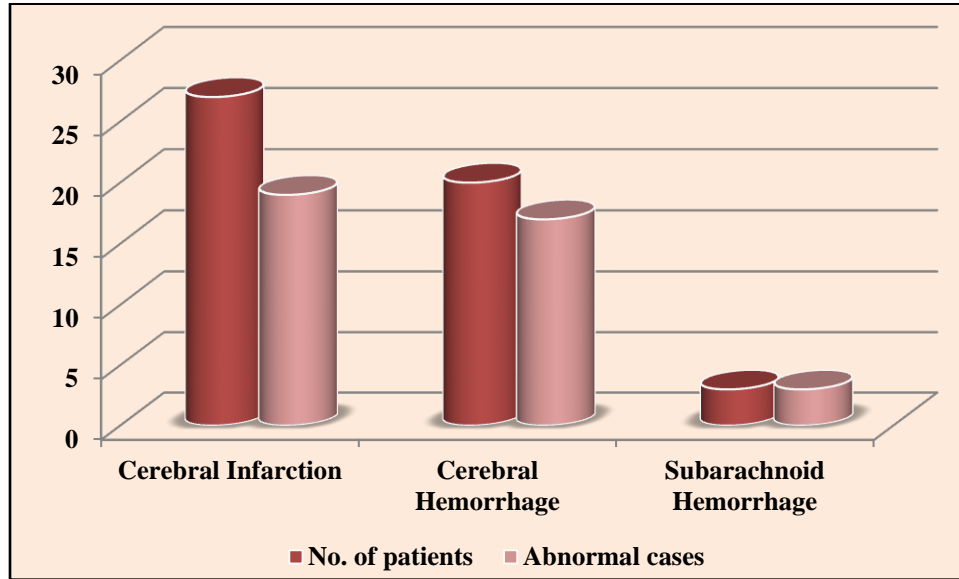
5. Housley GD, Martin-Body RL, Dawson NJ, Sinclair JD: Brain stem projections of the glossopharyngeal nerve and its carotid sinus branch in the rat. *Neuroscience*.1987; 22:237.
6. Calaresu FR, Ciriello J: Projections to the hypothalamus from buffer nerves and nucleus tractus solitarius in the cat. *Am J Physiol*.1980; 239:R130.
7. Melville KI, Blum B, Shister HE, Silver MD: cardiac ischemic changes and arrhythmias induced by hypothalamic stimulation. *Am J Cardiol*.1963; 12:781.
8. Arab, D., Yahia, AM., Qureshi, AI. Cardiovascular manifestations of acute intracranial lesions: Pathophysiology, manifestations, and treatment. *J Intensive Care Med*; 2003;18:119-29.
9. Frentz v, GormsenJ; Electrocardiographic pattern with cerebrovascular accidents, circulation. 1962; 25:22.
10. Arrudo and Lacerda; ECG findings in acute cerebrovascular hemorrhage-Arq neuropsiquiatr. 1992 september ;50(3): 269-74.
11. Keller and Williams; Cardiac dysarrhythmias associated with CNS dysfunction; *Journal of Neuroscience*. 1993;25:349-55.
12. Cruickshnok, Neil Dcoyer and J. Price ECG changes and their prognostic significance in CVA –*Journal of Neurology*-1974;37:735-759.
13. Kreuz Ke, Kemilla SJ, Takala JK;Electrocardiographic changes in cerebrovascular accidents, *Actamed Scand*. 1969; 185:327.
14. Yamour BJ, Sridharan MR, Rice JF, et alElectrocardiographic changes in cerebrovascular hemorrhage. *Am Heart J*. 1980; 99:294.

Table 1: Incidence Of Abnormal ECG's In The Study Group

Study Group	No. of patients	Abnormal cases	Percentage
Cerebral Infarction	27	19	70%
Cerebral Hemorrhage	20	17	89%
Subarachnoid Hemorrhage	3	3	100%
Total	50	39	78%

Table 2: Location And Type Of Cerebro Vascular Lesion With No Of Patients

Cerebral Lesion	Total No of Patients	No of Patients with Hemorrhage	No on Patients with Infarct
Basal Ganglia	1	0	1
Thalamus	5	2	3
Capsuloganglion	31	13	18
Frontal	3	1	2
Parietal	7	3	4
Temporo-Parietal	2	2	0
Occipital	1	1	0



Graph 1: Incidence Of Abnormal ECG's In The Study Group

Table 3: Percent and No Of Patients With Specific ECG Changes

Cerebral Lesion	Rhythm Disturbance	ST Segment Changes	QTC Prolongaton	T Wave Changes
Basal Ganglia	1 (100%)	1 (100%)	1 (100%)	0 (0%)
Thalamus	2 (40%)	1 (20%)	2 (40%)	2 (40%)
Capsuloganglion	6 (31%)	11 (36%)	6 (31%)	11 (36%)
Frontal	2 (66%)	1 (33%)	2 (66%)	2 (66%)
Parietal	2 (100%)	1 (50%)	0 (0%)	0 (0%)
Temporo-Parietal	2 (28%)	2 (28%)	2 (28%)	1 (14%)
Occipital	1(100%)	0 (0%)	0 (0%)	0 (0%)