

ELECTROCARDIOGRAPHIC CHANGES OBSERVED IN HAEMORRHAGIC AND ISCHAEMIC CEREBROVASCULAR DISEASES

K. C. Channappa¹, K. S. Yogeesh², Prakash Harishchandra³

¹Professor, Department of General Medicine, Kanachur Institute of Medical Sciences, Kanachur, Mangalore.

²Associate Professor, Department of General Medicine, Kanachur Institute of Medical Sciences, Kanachur, Mangalore.

³Senior Resident, Department of General Medicine, Kanachur Institute of Medical Sciences, Kanachur, Mangalore.

ABSTRACT

INTRODUCTION

Cardiac abnormalities are relatively common after acute neurologic injury. Disturbances can vary in severity from transient ECG abnormalities to profound myocardial injury and dysfunction. CNS is involved in the generation of cardiac arrhythmias and dysfunction even in an otherwise normal myocardium.

AIM

To find out proportion of ECG changes observed in ischaemic and haemorrhagic stroke.

MATERIALS AND METHODS

The Electrocardiographs of 100 patients with acute stroke were studied to find out the types of ECG abnormalities among different types of stroke.

RESULTS

In our study, the most common ECG abnormalities associated with stroke were prolonged QTc interval, ST-T segment abnormalities, prominent U wave and arrhythmias. Trop-I was positive in 12.8% patients with ECG changes. Statistical significance was found in association with Trop-I positivity and ST depression.

CONCLUSION

Usually patients with heart disease present with arrhythmias and Ischaemic like ECG changes. But these changes are also seen most often in the patients with presenting with stroke who didn't have any past history of heart disease. This shows that arrhythmias and ischaemic ECG abnormalities are primarily evolved due to central nervous system disorders.

KEYWORDS

Electrocardiogram (ECG), Cerebrovascular disease (CVA), ST-T wave, Trop-I.

HOW TO CITE THIS ARTICLE: Channappa KC, Yogeesh K S, Harishchandra P. Electrocardiographic changes observed in haemorrhagic and ischaemic cerebrovascular diseases. J. Evid. Based Med. Healthc. 2016; 3(23), 1025-1028.

DOI: 10.18410/jebmh/2016/235

INTRODUCTION: DEFINITION: According to WHO, stroke is defined as "rapidly developing clinical signs of focal or global disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular origin". This includes subarachnoid haemorrhage but excludes transient ischemic attacks, subdural hematoma and haemorrhage or infarction caused by infection or tumour.¹

Completed Stroke: It means the deficit has become maximal, usually within 6 hours.

Stroke-In-Evolution: It means progression during the first 24 hours.

Minor Stroke: Patient recovers without significant deficit, usually within a week.

Transient Ischemic Attack (TIA): It means a focal deficit, such as a weak limb, aphasia or loss of vision lasting from few seconds to 24 hours. There is complete recovery. The attack is usually sudden. TIAs have a tendency to recur and may herald thromboembolic stroke.²

Risk Factors for Stroke:

1. Non Modifiable Factors: Age, gender, race/ethnicity, family history, genetics.

2. Modifiable Factors: Arterial hypertension, TIAs, prior stroke, asymptomatic carotid bruit/stenosis, cardiac disease, aortic arch atheromatosis, diabetes mellitus, dyslipidaemia, cigarette smoking, alcohol consumption, increased fibrinogen, elevated homocysteine, low serum folate, elevated anticardiolipin antibodies, oral contraceptive use, obesity.

Submission 19-02-2016, Peer Review 04-03-2016,
Acceptance 11-03-2016, Published 21-03-2016.

Corresponding Author:

Dr. K. C. Channappa,

Professor, Department of General Medicine,
Kanachur Institute of Medical Sciences,
Kanachur, Mangalore.

E-mail: giridharkc2589@gmail.com

DOI: 10.18410/jebmh/2016/235

Cardiac abnormalities are relatively common after acute neurologic injury. Disturbances can vary in severity from transient ECG abnormalities to profound myocardial injury and dysfunction. Evidence from animal models and clinical observation indicates that CNS is involved in the generation of cardiac arrhythmias and dysfunction even in an otherwise normal myocardium. The medulla has been described as the principal site of vagal parasympathetic and sympathetic areas involved in cardiac control. In addition, anatomical and physiological evidence exists to implicate the hypothalamus in cardiac control.³ Beattie and colleagues first described cardiac arrhythmias after hypothalamic stimulation.

The insular cortex has wide spread connectivity with other areas of brain which are involved in autonomic control. Pathologically cardiac myocytolysis was demonstrated only if insular cortex was involved. This evidence collectively support the stroke can alter cardiovascular tone by directly damaging the insular cortex or other interrelated areas. In addition, right middle cerebellar artery stroke was associated with significantly increased incidence of supraventricular tachyarrhythmias. The most common disturbances include ECG abnormalities, cardiac arrhythmias and myocardial injury and dysfunction. In 1947, Byer and colleagues first described QT prolongation with large T and U waves in ECG of four patients with acute stroke.

Subsequently Burch and colleagues described an ECG pattern after an acute stroke consisting of large inverted T waves, prolonged QT interval, large septal U waves that has become distinctive of cerebrovascular injury.⁴

The most common stroke related ECG abnormalities is QT prolongation, a myocardial repolarisation abnormality associated with an increased risk of a characteristic life threatening cardiac arrhythmias, known as Torsade de pointes. Among acute stroke patients, prolonged QT interval is more frequently observed after haemorrhagic strokes.⁵ Ventricular tachyarrhythmias including sudden death and torsade de pointes are often preceded by QT prolongation in patients with SAH.

ST segment changes (including ST elevations) occur in 22-35% of patients in ischemic stroke, but Interpretation of such findings is complicated by the increase prevalence of cardiac disease in this sub group of stroke patients. However new T wave abnormalities appear in approximately in 15% of patients with acute stroke even in the absence of electrolyte disturbances or primary ischemic heart disease. Inverted or flat T wave have also been reported in up to 55% of patients with SAH, the stroke subgroup with the lowest prevalence of co existent cardiac disease. Kono and colleagues performed detailed cardiac assessments of twelve patients with acute SAH and ST elevations in ECG.

New U waves occur in isolation or with T waves and QT abnormalities in approximately 13-15% of patients with acute ischemic stroke and SAH.⁶

Nearly every type of cardiac arrhythmia has been reported after acute stroke, including bradycardia, supraventricular tachycardia, atrial flutter, atrial fibrillation, ectopic ventricular beats, multifocal ventricular tachycardia, ventricular flutter and ventricular fibrillation. Most

arrhythmias occur within 1st week after stroke, occurring in 25 to 40% of patients of ischemic stroke or ICH. Atrial fibrillation was the most common cardiac arrhythmia, accounting for 60% of events.⁷

Troponin is a highly sensitive and specific marker of myocardial necrosis. The quoted prevalence of a positive troponin level in acute stroke varies, but has been reported to be as high as 34%.⁸ It has been suggested that some of the myocardial damage observed in acute stroke is due to patchy myocyte damage (myocytolysis) due to activation of sympatho-adrenal system that may be linked to insular damage.

AIMS AND OBJECTIVES:

1. To find out proportion of ECG changes observed in ischaemic and haemorrhagic stroke.
2. To find out proportion of myocardial ischemic components responsible for these ECG changes through Troponin-I assay.

MATERIALS AND METHODS: A prospective analytical study was conducted at the department of General Medicine, Kanachur Institute of Medical Sciences, Kanachur, Mangalore from January 2015 to January 2016.

Inclusion Criteria: 100 Cerebrovascular accident patients diagnosed through either CT or MRI of age less than 75 years were included in the study.

Exclusion Criteria:

1. Known case of IHD, congenital, valvular or cardiomyopathic heart diseases.
2. Recurrent strokes.
3. Electrolyte imbalance.

Data Collection: All patients underwent detailed clinical examination including CVS and CNS examination. Routine blood and urine investigations along with lipid profile, ECG (on admission and after 3-5 days), 2D echo, CT brain, TROP-I.

Analysis: Qualitative variables were summarized using percentages and proportions. Quantitative variables were summarized using mean with Standard deviation/Median. Chi-square test was used to compare the proportion of various ECG changes. ($p < 0.05$).

RESULTS: The incidence of stroke in this study was more common in the age group of 55-64 years (35%) followed by 45-54 years (33%). The incidence of stroke in 64-75 years was 32%.

Among patients in the study group 43% were hypertensives, 40% diabetic, 16% had elevated cholesterol level, 20% consumed alcohol, 22% used tobacco.

Out of 100 patients, 71 were males and 29 were females. Male-Female ratio was 2.4:1.

In our study, 58% of patients had ischemic stroke and 42% had haemorrhagic stroke.

The CVA patients were divided into 2 categories, ischemic and haemorrhagic group (including SAH). On symptom analysis, the commonest presentation of stroke was weakness. Right hemiplegia was present in 37 patients (60.34%) in case of ischemic stroke and 14 patients (33.3%) in haemorrhagic stroke which was statistically significant ($p < 0.05$). Left sided hemiplegia was present in 15 patients (25.8%) of ischemic stroke, whereas 12 patients (28.57%) in haemorrhagic stroke.

Case of ischemia 5 patients (8.6%) and 24 patients (57.14%) in haemorrhage presented with headache. Vomiting was present in 3 patients (5.17%) in infarction group, while 18 patients (37.5%) in haemorrhagic group. Loss of consciousness was present in 4 patients (6.8%) in ischemic group while 10 patients (23.8%) in haemorrhagic group. Seizure was present in 3 patients (5.17%) in ischemic group and 4 patients (9.52%) in haemorrhagic group. Aphasia was present in 5 patients (8.6%) in ischemic group.

The ECG changes are evenly distributed among the various type of stroke. Sinus tachycardia occurred in 12 patients (20.68%) with ischemia, 6 patients with ICH and 1 with SAH (14.28%). Sinus bradycardia was seen in 3 patients (5.17%) with ischemia and 7 patients (14.5%) with ICH. None of SAH patients had bradycardia. T wave change was seen in 5 patients (8.6%) with ischemia and 3 patients (7.14%) with haemorrhagic stroke. QTc prolongation was seen in 17 ischemic patients (29.31%) and 8 haemorrhagic patients (19.05%). Among the ischemic group, T wave inversion (34.48%) and ST segment depression (32.76%) were the most common abnormalities, followed by QTc prolongation (29.31%) and presence of U waves (27.59%). In case of haemorrhagic group, T wave inversion (33.33%) and arrhythmias (33.33%) occurred in equal incidence followed by U waves (30.95%) and ST segment depression (23.81%). Ventricular premature contractions were seen in 9 cases and all were hypertensive and 6/9 had ST-T changes. 3 patients had atrial ectopic beats, 8 had atrial fibrillation and 10 had RBBB.

On statistical analysis there was significant relationship between ST depression and Trop I positivity. Echo was done in all 100 patients of which regional wall motion abnormality was found only in 4 patients (5.7%) and all of these patients had ST-T type of ECG abnormality and elevated Trop I value.

ECG CHANGES	ISCHEMIC STROKE(NO.)	ISCHEMIC STROKE(%)	HAEMORRHAGIC STROKE(NO.)	HAEMORRHAGIC STROKE(%)
Tachycardia	12	20.68	6	14.28
Bradycardia	3	5.17	7	14.5
P wave	5	8.6	3	7.14
QTc prolongation	17	29.3	8	19.05
ST elevation	6	10.34	4	9.52
ST depression	19	32.76	10	23.81
Tall T wave	5	8.7	2	4.76
T wave inversion	20	34.58	14	33.3
PR interval abnormality	5	8.62	6	14.29
U wave abnormality	16	27.5	13	30.59
Arrhythmias	12	20.69	14	33.3

Table 1

DISCUSSION: Incidence of stroke was highest in the age group of 55-64 years accounting for 35% of patients followed by 45-54 years (33%). Indians develop stroke at younger age compared to western population due to high prevalence of risk factors for stroke like hypertension, diabetes, hyperlipidaemia, smoking.⁹

Out of 100 patients, 71 were males and 29 females. The male: female ratio was 2.4:1. This ratio is comparable to other studies done by Nagaraja et al¹⁰ (2:1). The lower incidence of stroke in women may be attributed to genetic factors, positive effects of oestrogen on cerebral circulation and moreover ischemic heart disease, peripheral artery disease and cigarette smoking are more common among men. In the present study, hypertension was present in majority of cases i.e. 43%, which is comparable with that found in the studies of Pandiyan et al¹¹ and Watila et al.¹² Next commonest risk factors were smoking (22%) and diabetes (40%) which was comparable that found in Pandiyan et al,¹¹ being 23.6% and 49.8%. Hyperlipidaemia followed which was present in 16% of cases which can be compared with the study of Watila et al¹² which was 10%.

In this study, 58% of patients had ischemic stroke, which is comparable with that found in the studies of Bharucha et al¹³ and Harsha Kumar et al¹³ i.e., 57.3% and 56% respectively. Among the patients, 42% had haemorrhagic stroke in the present study that is comparable with 37.9% and 44% in Bharucha et al¹⁴ and Harsha Kumar et al¹³ study group respectively. The most common cause for ischemic stroke is atherosclerosis of the arteries, large and small that supply the brain. Haemorrhagic stroke are less common than ischemic but causes significant number of deaths worldwide. Bradycardia, T wave inversion, ST segment depression, U wave were seen in 8%, 15%, 13%, 28% respectively in Goldstein et al, while in the present study it is 10%, 34%, 29%, 29% respectively. Increased QTc was seen in 32% by Goldstein et al,⁶ were as 25% in this study. A similar study was done by Familoni et al⁸ in 2006 where QTc prolongation was seen in 28% cases, T wave inversion in 21.8%, ST segment depression in 29.7%, U wave in 9.3% and arrhythmias in 34.4% of the cases in the group.

In the present study, 10 out of 78 patients had elevated Trop I, having ECG abnormalities. These Trop-I changes are more with ST-T changes suggestive of myocardial ischemia. Among TROP-I association with ECG changes there was a statistical significance with ST depression (p value-0.027).

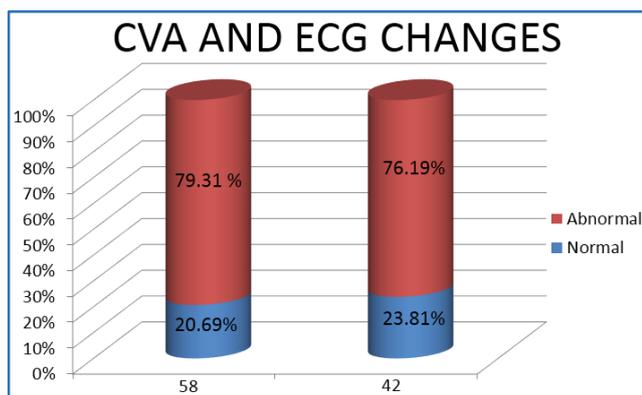


Fig. 1

So we have to suspect an MI component when there are non-specific ECG changes of CVA. So workup with TROP-I and Echo may help to rule out myocardial ischemic components in CVA patients.

CONCLUSION: CVA is an important cause of morbidity and mortality in health care system. Incidence of CVA increases with advancement of age. Risk factors like age, gender, modifiable factors like hypertension, diabetes, smoking, alcohol and hyperlipidaemia are important contributing factors for the development of stroke. ECG changes occur very commonly in acute CVA. The ECG changes occur independent of nature of stroke i.e. they are equally seen in ischemic and haemorrhagic group. The major ECG abnormalities were ST-T changes, QTc prolongation and positive U waves. Trop-I was found to be elevated in 10 out of 78 patients (12.8%) with ECG abnormalities. Statistical significance was found in association with Trop-I positivity and ST depression.

REFERENCES:

1. Bonita R. Epidemiology of Stroke. *Lancet* 1992;339(8789):343-4.
2. Clarke CRA. Cerebrovascular disease and stroke. In: Kumar P, Clark M editors. *Clinical Medicine*. Edinbergh Elsevier 2005;6th ed:1209-19.
3. Calaresu FR, Ciriello J. Projections to the hypothalamus from buffer nerves and nucleus tractus solitarius in the cat. *Am J Physiol* 1980;239(1):130-136.
4. Byer E, Ashman R, and Toth LA. Electrocardiograms with large, upright T waves and QT intervals. *Am Heart J* 1947;33(6):796-806.
5. Burch GE, Myers R, Abildskov JA. A new ECG pattern observed in cerebrovascular accidents. *Circulation* 1954;9:719-23.
6. Goldstein DS. The electrocardiogram in stroke: relationship to pathophysiological type and comparison with prior tracings. *Stroke* 1979;10(3):253-259.
7. Familoni OB, Odusan O, Ogun SA. The pattern and prognostic features of QT intervals and dispersion in patients with acute ischemic stroke. *J Natl Med Assoc* 2006;98(11):1758-62.
8. Jensen JK, Kristensen RS, Bak S, et al. Frequency and significance of troponin T elevation in acute ischemic stroke. *Am J Cardiol* 2007;99(1):108-112.
9. Sridharan SE, Unnikrishnan JP, Sukumaran S, et al. Incidence, types, risk factors, and outcome of stroke in a developing country: the Trivandrum stroke registry. *Stroke* 2009;40(4):1212-8.
10. Nagaraja D, Christopher R, Tripathi M, et al. Preceding infection as a risk factor of stroke in the young. *J Assoc Physicians India* 1999;47(7):673-5.
11. Pandiyan U, Arjundas G, Arjundas D. Risk factors and stroke outcome-an Indian Study. *IJPMR* 2005;16(2):29-33.
12. Watila MM, Nyandaiti YM, Bwala SA, et al. Gender variation in risk factors and clinical presentation of acute stroke, Northeastern Nigeria. *J Neurosci Behav Health* 2011;3(3):38-43.
13. Kumar HNH, Kalra B, Goyal N. A study on stroke and its outcome in young adults (15-45 Years) from coastal South India. *Indian J Community Med* 2011;36(1):62-65.
14. Kuruvilla T, Bharucha NE. Epidemiology of stroke in india. *Neurol J Southeast Asia* 1998;3:5-8.