Original Article

Relationship of electrocardiographic changes with mortality in cerebrovascular accidents

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Abstract

Background: A variety of cardiovascular events like cardiac arrest, arrhythmias, and severe hypotension can be seen in stroke victims. Electrocardiographic abnormalities are known to occur in such patients. Stroke cases with more ECG abnormalities has poor prognosis. the present study was undertaken to find out relationship of the electrocardiographic changes with mortality in CVA. **Material and Methods:** The study consisted of 109 cases with history and clinical manifestation suggestive of acute stroke and confirmed by CT head and admitted in medical wards of the hospital. Age and sex matched 109 controls with history and clinical manifestation not suggestive of heart disease or neurological disease were also included in this study. **Results:** Amongst 109 cases studied, 67(61.47%) had cerebral thrombosis and 42(38.53%) had cerebral haemorrhage. Amongst 67 cases of cerebral thrombosis group, 21 (31.34%) had various electrocardiography abnormalities and amongst 42 cases of haemorrhage group, 32 (76%) had electrocardiography abnormalities. The mortality rate in cerebral thrombosis group was 10.44% (7 deaths out of 67 cases).The mortality rate in cerebral haemorrhage group was 47.61% (20 deaths out of 42 cases) in the present study. **Discussion:** ECG abnormalities have clear relationship with mortality. More the ECG abnormalities worse the prognosis. This study suggests that ECG abnormalities are commonly seen in CVA patients and the mortality was higher in stroke group with abnormal ECG.

Key Words: Electrocardiographic abnormalities, cerebrovascular accidents, mortality.

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INTRODUCTION

The term stroke or cerebrovascular accident (CVA) has been defined as sudden, non-convulsive focal neurological deficit of vascular aetiology[1]. The clinical manifestations of stroke ranges from transient ischemic attack so trivial as to escape attention to its severest form of patient being hemiplegic and comatose, the occurrence being so dramatic and frightening that it has been designated as apoplexy. Stroke is one of the leading cause of death throughout the world including both developed and developing countries and is a major cause of morbidity and mortality imposing considerable challenge to rehabilitative programmes. In India, it is the third most common cause of mortality among non-communicative disease[2]. A variety of cardiovascular events like cardiac arrest, arrhythmias, and severe hypotension can be seen in stroke victims. Electrocardiographic abnormalities are known to occur in variety of central nervous system lesions like cerebrovascular accident, head trauma, intracranial space occupying lesion, meningitis, etc.[3-4] Many of the patients of cerebrovascular accident require urgent surgical intervention but have be treated conservatively because of their associated ECG changes, some have died and the post-mortem examination has shown an apparently normal heart[5]. Now, there is sufficient evidence to suggest that certain electrocardiographic changes occur in patients with cerebrovascular accident in the absence of any demonstrable cardiac pathology[6]. There is clear correlation between the electrocardiographic alterations and the prognosis. Cases with more ECG abnormalities has poor prognosis[7-8]. The unexpected event of sudden death has its correlation to cerebral and coronary arteriosclerotic disease. The need for ECG observation in stroke patients has to be increased to reduce the excess risk of mortality in CVA. Hence, the present study was

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undertaken to find out relationship of the electrocardiographic changes with mortality in CVA.

MATERIAL AND METHODS

The study consisted of 109 cases with history and clinical manifestation suggestive of acute stroke and confirmed by CT head and admitted in medical wards of the hospital. The study consisted of age, sex matched 109 controls with history and clinical manifestation not suggestive of heart disease or neurological disease, and attended medicine OPD for other health problems were in included in this study. Patients were excluded if they had a history or signs of heart disease like valvular stenosis or insufficiency, verified angina pectoris, myocardial infarction, heart surgery, had been treated with antiarrhythmic drugs, calcium antagonists, digitalis, antiepileptics, lithium and levodopa, have cerebral infarction due to cardiac embolism as evaluated by clinical or echo cardiographic examination, had signs of electrolyte disturbances, had transient ischemic attack, meningitis with CVA, brain tumor, head injury, had EGG changes indicating old Q wave infarction or ST elevation more than two millimetres in chest leads and one millimetre in limb leads and 2-D Echo suggestive of wall hypokinesia and more than there fold rise in CPK-MB levels. A detailed general physical and neurological examination including fundus examination was carried out for definite neurological localization. The report of CT-scan was read by, expert radiologist who was unaware of the clinical details and diagnosis. The scan were evaluated for locations of the intracranial haemorrhage and infarction, presence of intraventricular or displacement of intracranial structures. The non-haemorrhagic cerebral infarction was diagnosed when sharply marginated homogenous, speckled low-density lesion confined to a vascular territory. Twelve lead electrocardiograms were taken in all the cases of cerebrovascular accident at the time of admission. It was repeated after 48 hrs and after seven days, and whenever it was possible.

RESULTS

In our study the age of the cases ranged from 35 years to 80 years of age with mean age of 56.99 years. Maximum incidence was in the age group of 41 to 70 years of age i.e., 88 cases (80.73%).Age of the controls ranged from 32 years to 80 years of age with mean age of 54.94 years. It was observed that amongst 109 cases studied, 62 (56.88%) were male and 47 (43.12%) were females. The male to female ratio was 1.32:1.In our study, 109 controls with 62 males and 47 females were studied and matched.

Table 1: Pattern of cerebrovascular accident

Type	Male		Female		Total	%
	No. of cases	%	No. of cases	%		
Thrombosis	44	70.97	23	48.94	67	61.47
Haemorrhage	18	29.03	24	51.06	42	38.53
Total	**62**	**100**	**47**	**100**	**109**	**100**

It was observed that amongst 109 cases studied, 67 (61.47%) had cerebral thrombosis and 42 (38.53%) had cerebral haemorrhage. Thus, cerebral thrombosis was more common in males in our study. Fundus examination showed evidence of hypertensive retinopathy in 42 (38.53%) cases; these included 20 cases of cerebral thrombosis and 22 cases of cerebral haemorrhage group. Diabetic retinopathy was noticed in 1 case from cerebral thrombosis group. Fundus examination in control group showed hypertensive retinopathy in 12 (11%) controls.

Table 2: ECG changes in cases with acute cerebrovascular accident and controls

ECG	No. of Cases	%	No. of Controls	%
Normal	56	51.38	103	94.50
Abnormal	53	48.62	6	5.50
Total	**109**	**100**	**109**	**100**

X^2=51.53; df=1; p<0.001

In present study amongst 109 cases studied, various electrocardiographic abnormalities were noticed in 53 (48.62%) cases, whereas 6 (5.50%) controls had abnormal electrocardiogram. The electrocardiographic changes observed in acute cerebrovascular accident patients were statistically more frequent when compared with controls (p <0.001). Amongst 67 cases of cerebral thrombosis group, 21 (31.34%) had various electrocardiography abnormalities and amongst 42 cases of haemorrhage group, 32 (76%) had electrocardiography abnormalities. The common abnormalities noticed were T wave inversion in lateral (I,aVL, V3 to V6) and inferior (II, III, aVF) leads.

Table 3: Various ECG changes observed in controls

ECG changes	No. of Controls	%
Normal	103	94.50
Sinus tachycardia	2	1.83
Ventricular premature complex	2	1.83
Flat T(V4-V6)	1	0.92
Sinus Bradycardia	1	0.92
Total	**109**	**100**

In present study electrocardiographic changes observed in 6 controls were, sinus tachycardia in two, ventricular premature complexes in two, flat T wave in one and sinus bradycardia in one control. It was noticed that in cerebral thrombosis group, out of 67 cases, 21 showed electrocardiographic abnormalities. Amongst these, in 9 cases electrocardiographic changes reverted to normal and nobody died in this group. In cerebral haemorrhage group, out of 32 cases with electrocardiographic abnormalities, in 7 cases electrocardiographic changes reverted to normal. It was observed that ECG changes reverted to normal in 16 cases out of 53 cases and persisted in 37(69.81%) cases. Out of 37 cases with persistent ECG changes 26 cases died (70.27%). Commonest ECG abnormality noticed in patients who died was T wave abnormalities in 9 cases (34.62%) which included tall T wave in 5 cases (19.24%) and T wave inversion in 4 cases (15.38%). Rhythm abnormalities were observed in 9(34.62%) cases who died. Atrial fibrillation in 2(7.69%), supraventricular tachycardia in 2(7.69%) sinus tachycardia in 2(11.54%), sinus bradycardia in 7.69% cases. Persistently changing ECG was observed in 6(23.07%) cases who died other ECG changes observed in patients who died was q wave in 1 case (3.85%)and prolong QTc in 1 case (3.85%). The mortality rate in the present study was noticed to be 24.77% (27 deaths out of 109 cases). The mortality rate in cerebral thrombosis group was 10.44% (7 deaths out of 67 cases).The mortality rate in cerebral haemorrhage group was 47.61% (20 deaths out of 42 cases) in the present study. The mortality was more in cerebral haemorrhage group ($p<0.001$).

Table 4: Mortality in relation to ECG abnormalities

ECG pattern	No. of cases	survival	Death	%
Normal	56	55	1	1.79
Abnormal	53	27	26	49.06

Z=6.67; SE=7.093; p<0.001

In present study, mortality in relations to electrocardiographic abnormalities were studied. Out of 109 cases 53 had abnormal electrocardiogram and out of this 27 survived and 26 (49.06%) died. The normal electrocardiograms were observed in 56 cases, of which 55 survived and one died (1.79%).Thus, it was noticed that mortality was high in patients with electrocardiographic abnormalities ($p<0.001$).

DISCUSSION

The incidence of stroke is higher in India due to increased prevalence of risk factors like hypertension and diabetes[2]. In present study, the age of the cases ranged from 35 to 80 years with mean age 56.99 years. The maximum 88 (80.73%) cases were in the age group 41-70 years. The study also included age, sex matched 109 controls with mean age 54.94 years. The age distribution in present study was similar with studies of Foulkdes *et al*[9] observed maximum incidence in 5th to 7th decade, Thacker *et al*[10] noticed mean age (54.5 years) in their study. There was a slight male preponderance in our study with male to female ratio 1.3:1, which is comparable to other studies done by Anand *et al*[11] (1.7:1) and Nagaraja *et al*[12] (2:1). Men showed an increased prevalence of ischemic heart disease due to risk factors such as tobacco chewing, smoking and alcohol drinking. The lower incidence of stroke seen in women is attributable to genetic susceptibility, estrogenic effects on the cerebral circulation, and to reduced blood pressure values compared to me n. In present study amongst 109 cases studied, 67 (61.47%) had cerebral thrombosis, while 42 (38.53%) had cerebral haemorrhage. Cerebral thrombosis was more common than cerebral haemorrhage in our study. Hemorrhagic strokes are less common than ischemic stroke, but cause a significant number of deaths. Hemorrhagic stroke causes severe, morbid damage to cerebral tissue that can leave individuals paralyzed or weak, with difficulty in motor activities and cognitive abilities[13]. In present work, amongst 109 cases, various electrocardiographic abnormalities were noticed in 53 (48.62%) cases, the incidence of electrocardiographic abnormalities in present study was similar with Lindgren *et al*[14] (50%) and Bozluolcay *et al*[15], where ECG changes were demonstrated in 62.1% of patients. This can be explained on basis of inclusion and exclusion criteria in present study. Out of 109 controls, 6 had electrocardiographic changes. The electrocardiographic changes in cerebrovascular accident were statistically more frequent when compared with controls ($p<0.001$). In our study the incidence of electrocardiographic abnormalities was more in cerebral haemorrhage group compared to cerebral thrombosis group. In cerebral haemorrhage group out of 42 cases, 32(76%) had abnormal electrocardiogram and in cerebral thrombosis group, out of 67 cases, 21(31.34%) had various electrocardiographic abnormalities. Similar findings were observed by, Togha *et al*[16] noticed that in cerebral thrombosis group, 15% cases had abnormal ECG and in cerebral haemorrhage group, 71% had abnormal electrocardiogram. Kocan[17] noted ECG changes were more common in hemorrhagic stroke. In present study, T wave abnormalities were observed in 15 cases (13.76%). Jesper *et al*[18] evaluated characteristics and prevalence of ST-segment depression and/or T-wave inversion in the resting electrocardiogram of 244 consecutive patients with acute ischemic stroke, but without ischemic heart disease. The prevalence of ST-T changes ranged from 13% to 16%. In present study, U wave was observed in 3

cases (2.75%). Similar findings were observed by Yamour *et al*[4] (4.62%). The pathological q wave was observed in 2 cases (1.83%). The Q-wave observed in any studies appear to be due to old changes, not appear to be because of acute stroke. Prolonged QTc was observed in 2 cases (1.83%). Rhythm abnormalities commonly noticed were sinus bradycardia, sinus tachycardia, atrial fibrillations, ventricular premature complex, and supraventricular tachycardia. Similar findings were noted by Yamour *et al*[4] (6.15%), Goldstein *et al*[19] 9.84% and Ramani *et al*[20] 10%. Sinus tachycardia was noticed in 8 cases (7.3%). Atrial fibrillation was noticed in 2 cases (1.83%) in present study, similar with studies by Romani *et al*[20] 1.25% and Yamour *et al*[4] 3.57%.Ventricular premature complex in present study was noticed in 2 cases (1.83%) similar with other studies by Ramani *et al*[20] 2.5% and Yamour *et al*[4] 3%.Supraventricular tachycardia in present study was noticed in 2 cases (1.83%) similar with study by Lindgren *et al*[14] who noticed in 1 (3.5%) case. Commonest ECG abnormalities noticed In patients who died was T wave abnormalities in 34.62% cases, which include tall T wave in 5 cases (19.24%) and T wave inversion in 4 cases (15.38%). Rhythm abnormalities in 9 cases (34.62%). Which includes atrial fibrillation in 2(7.69%). Sinus tachycardia in 3 (11.54%) and sinus bradycardia in 2 (7.68%) cases. Persistently changing pattern ECG was observed in six cases (23.07%) other ECG changes observed were q-wave in one case (3.85%) and prolonged QTc in one case (3.85%). Similar findings were observed in studies by Lindgren *et al*[14]. A similar study was done by Familoni *et al*[21] in 2006 where QTc prolongation was seen in 28% of the cases, T wave inversion in 21.8%, ST segment depression in 29.7%, U wave in 9.3%, and arrhythmia in 34.4% of the cases in study group. In present study, mortality rate in acute cerebrovascular accident was (24.77%).Mortality in cerebral thrombosis group and cerebral haemorrhage group was (10.45%) and (47.61%) respectively. Similar findings were observed by Purushothaman *et al*[22] who noticed mortality was (13%) and (33.33%) in cerebral thrombosis group and cerebral haemorrhage group respectively. In present study mortality was more in patients with ECG abnormalities (49.06%) whereas mortality was less in patients with normal ECG. Thus, ECG abnormalities have clear relationship with mortality. More the ECG abnormalities worse the prognosis. Similar findings were noted by William ES and Martin AS[23] and Davis AM[24]. This study suggests that ECG abnormalities are commonly seen in CVA patients and the mortality was higher in stroke group with abnormal ECG.

REFERENCES

1. Victor M, Ropper AH, Adams RD. Cerebrovascular diseases. In: Adams and Victor's Principles of Neurology. 7th ed. McGraw-Hill Professional. pp.406.
2. Kuruvilla T, Bharucha NE. Epidemiology of stroke in India. Neurol J Southeast Asia. 1998; 3:5–8.
3. Dimant J, Grob D. Electrocardiographic changes and myocardial damage in patients with acute cerebrovascular accidents. Stroke1977; 8:448–55.
4. Yamour BJ, Sridharan MR, Rice JF, et al: Electrocardiographic changes in cerebrovascular hemorrhage. Am Heart J 99:294-300, 1980.
5. Cuadrado-Godia E, Ois A, Roquer J. Heart Failure in Acute Ischemic Stroke. Current Cardiology Reviews. 2010; 6(3):202-213.
6. Chua HC, Sen S, Cosgriff RF,Gerstenblith G, Beauchamp NJ Jr, Oppenheimer SM. Neurogenic ST depression in stroke. ClinNeurolNeurosurg1999; 101(1):44-48.
7. Main K, Von Arbin M, Britton M. Prognosis in acute stroke with special reference to some cardiac factors. J Chron Dis 1983; (36):279-288.
8. Maiuri F, Bonvenuti D, Maddalena G. Electrocardiographic changes and their prognostic significance in patients with subarachnoid hemorrhage. ActaNeurol 1984; 612:111-116.
9. Foulkes MA, Wolf PA, Price TR. The stroke data bank, designs, methods and baseline characteristics. Stroke 1988;19:547-554.
10. Thacker AK, Radhakrishnan K, Malco JC. Clinical and computed tomographic analysis of intracerebral haemorrhage. J AssoPhy India 1991 ;(39):317-319.
11. Anand K, Chowdhury D, Singh KB, Pandav CS, Kapoor SK. Estimation of mortality and morbidity due to strokes in India. Neuroepidemiology. 2001; 20:208–11.
12. Nagaraja D, Gururaj G, Girish N, Panda S, Roy AK, Sarma GR, et al. Feasibility study of stroke surveillance: Data from Bangalore, India. Indian J Med Res. 2009; 130:396–403.
13. Torpy JM, Burke AE, Glass RM. Hemorrhagic stroke. JAMA. 2010; 303:2312.
14. LindgrenA, Wotilfart B, Patilms O, Johanasson BB. Electrocardiographic changes in stroke patients without heart disease. Clinical physiology 1994 ;(14):223-231.
15. Bozluolcay M, Ince B, Celik Y, Harmanci H, Ilerigelen B, Pelin Z. Electrocardiographic findings and prognosis in ischemic stroke. Neurol India. 2003; 51:500–2.
16. Togha M, Sharifpour A, Ashraf H, Moghadam M, Sahraian MA. Electrocardiographic abnormalities in acute cerebrovascular events in patients with/without cardiovascular disease. Annals of Indian Academy of Neurology 2013; 16(1):66-71.
17. Kocan MJ. The brain heart connection: cardiac effects of acute ischemic stroke. J CardiovascNurs 1998; 13:57-68.
18. Jensena JK, Bakb S, Flemming P, Høilund-Carlsenc,Mickleya H.Prevalence of electrocardiographic ST-T changes duringacute ischemic stroke in patients without known ischemic heart disease. International Journal of Cardiology 2008; 128:137-138.
19. Goldstein DS. The electrocardiogram in stroke: Relationship to pathophysiological type and comparison with prior tracings. Stroke 1979; 10:253-9.

20. Ramani A, Sheety U, Kundaje GN. Electrocardiographic abnormalities in CVA. Angiology 1990; 41:681-686.
21. 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 Assoc2006; 98:1758–62.
22. Purushothaman S, Salmani D, Prarthana KG, Bandelkar SMG, Varghese S. Study of ECG changes and its relation to mortality in cases of cerebrovascular accidents. Journal of Natural Science, Biology, and Medicine2014; 5(2):434-436.
23. William ES, Martin AS. Electrocardiographic changes associated with neurologic events. Chest 1994; 1316-17.
24. Davis AM, Natelson BH. Brain interactions: the neurocardiology of arrhythmia and sudden cardiac death. Text heart Inst J 1993; (20):158-69.

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