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Electrocardiographic Abnormalities in Acute Stroke Cases Compared With Controls

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Abstract:
Background: *Stroke or cerebrovascular accident according to World Health Organization (WHO) is a focal or global neurological deficit of vascular origin lasting more than twenty-four hours or resulting in death before twenty-four hours. In sub-Saharan Africa as well as around the globe, stroke is the second leading cause of death worldwide after ischaemic heart disease with the victims mainly in the productive age ranges.*
Notably, strokes are preceded by modifiable risk factors such as hypertension, diabetes mellitus, obesity, smoking and alcohol ingestion. The use of electrocardiography (ECG) in addition to controlling these risk factors would reduce the occurrence, morbidity and mortality of stroke in at-risk patients as ECG is both a preventive and prognostic tool. The knowledge of data regarding abnormalities present in this group of patients will offer knowledge that will enhance physicians' patient management and provide data for future research.
Objectives: *To evaluate the prevalence of ECG abnormalities in stroke patients in University of Benin Teaching Hospital (UBTH); to determine if any difference exists between the ECG of stroke patients and controls*
Methods: *This was a cross sectional analytical study carried out in University of Benin Teaching Hospital (UBTH), Benin between January 2010 and 2013. Study subjects consisted of consecutive one hundred and twenty admitted stroke patients and one hundred and twenty admitted non-stroke patients who met the inclusion criteria.*
History and physical examination were carried out for all patients with laboratory investigations and electrocardiographic examinations also performed on all patients. The data was analyzed using SPSS version 21 software with a p–value of less than 0.05 considered significant for all comparisons.
Results: *Some risk factors for stroke in this study were hypertension (76.67%), male sex (46.67%), age older than sixty years (43.33%), diabetes mellitus (23.33%) and obesity (10.00%). Diabetes mellitus was the most common disease condition among the controls in this study (43.33%) while 76.67% of the stroke patients and 7.50% of the controls had abnormalities on their ECG on presentation.*
The most common ECG abnormality among the stroke patients was Left axis deviation (52.17%) and left ventricular hypertrophy (44.44%) among the controls. ECG abnormalities were more in stroke patients (stroke - 76.67% vs. controls - 7.50%).
Conclusion: *This study found a high prevalence of ECG abnormalities in stroke patients (76.67%) compared to controls in UBTH. Hypertension, an easily identifiable condition was common it also tends to cluster with other risk factors. Stroke risk factors can be identified early by history, physical examination and work up which should include ECG. Myriad types of ECG abnormalities were seen in stroke patients in this study. As there were high incidences of modifiable stroke risk factors for example hypertension and diabetes mellitus in this study. Controlling these risk factors would reduce the occurrence of stroke. Therefore, early diagnosis and treatment of stroke risk factors will significantly reduce morbidity, mortality and prolong life in these patients.*
Keyword: *stroke, cerebrovascular accident, acute*

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I. Introduction

Stroke according to World Health Organization (WHO) is an acute neurological deficit of cerebrovascular origin that persists beyond twenty-four hours or is interrupted by death within twenty-four hours. It has been projected that stroke could soon be the most common cause of death worldwide as it is currently the second leading cause of death in the world, ranking after heart disease.[1-9]

Globally there is an increasing trend in the burden of non-communicable diseases especially cardiovascular and cerebrovascular diseases particularly in developing countries. Across Africa and in Nigeria the prevalence of stroke is also increasing. This transition imposes more constraints in dealing with the double burden of communicable and non-communicable diseases in a poor economy characterized by inadequate health systems.

This has been attributed to more people now living up to and beyond middle age because of improvement in sanitation and reduction in prevalence rate of infectious diseases coupled with increasing use of tobacco, westernized lifestyle and urbanization with reduced physical activity, increased caloric consumption and psychosocial stress also been implicated.

These acts synergistically to cause increased cardiovascular and cerebrovascular risk via weight gain, hypertension, dyslipidaemia, dysglycaemia and hyperuricaemia. By year two thousand and twenty it is predicted that non-communicable diseases will cause seven out of every ten deaths in developing countries compared with less than half that is obtained today.[2-9]

About eight hundred thousand (800,000) people in the United States, have stroke each year, one hundred and thirty thousand (130,000) of them die each year. One American dies from stroke every four minutes on average. Stroke cost the United States, an estimated $36.5 billion each year. Worldwide, stroke is the second leading cause of death after ischaemic heart disease, and is followed by lower respiratory tract infections, chronic obstructive lung disease, diarrhea and HIV/AIDS, as the leading six killers worldwide as at 2013.[10,11]

The incidence of stroke increases exponentially from thirty years of age, and the etiology varies with age. Advanced age is one of the most significant stroke risk factors. Ninety five percent of stroke occurs in people aged forty-five and above, two-thirds of stroke occurs in those over the age of sixty five. [3,12]

Disability affects seventy five percent of stroke survivors enough to decrease their employability and stroke can affect patients physically, mentally, emotionally, or its combination. The result of stroke varies widely depending on size and location of lesion. Thirty to fifty percent of stroke survivors suffer post stroke depression, which is characterized by lethargy, irritability, sleep disturbance, lowered self-esteem and withdrawal while up to ten percent of all stroke patients develop seizures most commonly in the weeks subsequent to the stroke event and the severity of the stroke increases the likelihood of a seizure.[8, 13-15]

Stroke can be classified into 2 major categories - ischaemic stroke and haemorrhagic stroke[2]

Ischaemic stroke occurs as a result of an obstruction within a blood vessel supplying blood to the brain. It accounts for about eighty seven percent of all stroke cases. The underlying condition for this type of obstruction is the development of fatty deposits lining the vessel wall. This condition is called atherosclerosis. These fatty deposits can cause obstruction mainly as shown below;

Types of Ischaemic Stroke

(a)Cerebral thrombosis. Refers to a thrombus that develops at the clogged part of the vessel. (b)Cerebral embolism. Refers generally to a blood clot in the cerebrovascular system from another location in the circulatory system, usually the heart and large arteries of upper chest and neck, these tend to be associated with atrial fibrillation and other heart diseases. (c)Systemic Hypo-perfusion. This is a general decrease in blood supply for example in shock. (d)Venous thrombosis. This leads to stroke due to locally increased venous pressures which exceeds the pressure generated by the arteries. These infarcts are more likely to undergo haemorrhagic transformation (leaking of blood into the damaged area) than other types of ischaemic stroke. (e) Cryptogenic stroke. This is stroke of unknown origin. Constitutes thirty to forty percent of all ischaemic stroke.[9, 16-19]

Less frequently used though stroke can also be classified based on the Oxford classification into 4 types;

Total Anterior Circulation Infarct (TACI). (b) Partial Anterior Circulation Infarct (PACI). (c) Lacunar infarct (LAC). (d) Posterior Circulation Infarct (POCI). These four entities predict the extent of the stroke, the area of the brain affected, the underlying cause and the prognosis.[20, 21]

Haemorrhagic stroke arises from bleeding within the brain parenchyma or intra ventricular spaces. They constitute about fifteen percent of stroke. They result in tissue injury by causing compression of tissue from expanding haematoma or haematomas. This can distort and injure tissues. In addition, the pressure may lead to a loss of blood supply to the affected tissues with resulting infarction, and the blood released by brain haemorrhage appears to have direct toxic effects on brain tissue and vasculature. Inflammation also contributes to the secondary brain injury after haemorrhage [19, 22, 23].

Physicians have known for centuries that primary cardiac disorders can lead to stroke but the realization that stroke may produce cardiac abnormalities is more recent. In 1947 Byer, Ashman and Toth described a patient with intra cerebral haemorrhage whose ECG showed marked QT prolongation with large T and U waves. In 1954 Burch, Myers and Abildskov reported a pattern of QT prolongation, abnormal T and U waves which they considered distinctive of acute stroke [25, 26].

Since Burch pointed out the association of electrocardiographic (ECG) abnormalities with cerebrovascular accidents (CVA) many reports have appeared in literature. Most of which dealt with the incidence and kinds of ECG abnormalities in CVA[27-34].

Regarding the relationship between the location of CVA lesions and ECG abnormalities, Fentz and Kreus briefly noted that ECG changes appeared to bear no association to arteriography findings[27, 29]. More

recently however Yamour using the computerized tomography scan (CT scan) suggested that frontal lobe haemorrhages are associated especially with ECG abnormalities of corrected QT interval prolongation and neurogenic T waves[35].

Miura T using the CT scan attempted to clarify the relationship of ECG abnormalities to the location of CVA in the brain and found that there were no ECG changes specific to a particular CVA location and suggested that the widely distributed neurons and pathways within the CNS probably influence ECG changes in stroke. Also suggested that CVA lesions produce ECG changes by destroying or irritating the neurons or pathways in the brain especially the top of frontal lobe, motor and premotor cortex, anterior part of temporal lobe, hypothalamus,the limbic system and cerebellum. The intimate functional connections between the hypothalamus, posterior orbitofrontal, the medial forebrain bundle, anterior insular, the hypothalamus and peripheral sympathetic nerves have been demonstrated[36].

Kocan in USA found that patients at highest risk for development of ECG changes after stroke include those with hemorrhagic stroke, those with stroke involving the right cerebral hemisphere and elderly patients with stroke. Khechinashvili and Asplund in Sweden found a prevalence of ECG abnormalities in stroke to be ninety percent[4, 37].

Oppenheimer stated that stroke whether ischaemic or hemorrhagic induces cardiac damage by non ischaemic mechanisms. The evidence was derived from autopsy studies and investigation of ECG, cardiac enzymes changes and plasma catecholamine changes after stroke which showed that increased sympathoadrenal tone resulting from damage to cortical areas involved in cardiac and autonomic control is the likely cause. Recent experimental evidence indicate that the insular cortex plays a principal role in stroke – related cardiac damage.[38]

Oppenheimer demonstrated that micro stimulation of the rat posterior insular cortex in phase with the ECG R-wave elicit pure cardiac effects unaccompanied by change in blood pressure or respiration. This successfully demonstrates cardiac chronoscopic organization and arrhythmogenesis within the insular. He also stated that pathways exist linking the insular cortex with the lateral hypothalamic Area (LHA) and also stated that the LHA has been shown to mediate the sympathetic and blood pressure effects of insular cortex stimulation.[39]

Meyer found right hemispheric stroke involving the insular cortex are most susceptible to develop cardiac autonomic dysfunction in German patients.[100]

Sander and klingelhofer in a German study investigated the effects of left and right-sided hemispheric brain infarction on variability in circadian blood pressure and cardiovascular measures in thirty-five (35) patients to test for asymmetry of the sympathetic consequences of stroke. No significant difference regarding age, size of infarct, extent and frequency of damage to the insular cortex could be detected between the two groups.

Patients with right–sided stroke showed significantly reduced circadian blood pressure variability, a higher frequency of nocturnal blood pressure increase, higher serum noradrenaline concentrations and ECG more frequently showed QT prolongation and cardiac arrhythmias. However irrespective of the hemisphere damaged patients with insular infarction showed the most pronounced changes of these parameters. Those finding suggest lateralization of sympathetic activation with right-sided dominance for sympathetic effects following hemispheric stroke.[41]

Goldstein reviewed electrocardiographic records of one hundred and fifty patients with acute stroke along with one hundred and fifty age and sex matched controls. To assess the relative frequency of ECG abnormalities among the pathophysiologic categories of stroke and to distinguish new abnormalities at the time of stroke from those noted on prior tracings. Of the one hundred and fifty patients with stroke ninety two percent showed ECG abnormalities. The most common abnormalities were QT prolongation (45%), ischaemic changes (35%), U- waves (28%), tachycardia (28%) and arrhythmia (27%). Cerebral embolism patients had a significant increased frequency of atrial fibrillation (47%). While those with Subarachnoid Heamorrhage showed an increased frequency of QT prolongation (71%) and sinus arrhythmia (18%). Familoni in Nigeria studied 64 acute ischaemic stroke patients and found prolonged QT_{cmax} in 43.8%, ST- depression in 29.7%, T wave inversion in 21.8%, U wave in 9.3%. Fure in Norway studied two hundred and seventy-nine acute ischemic stroke patients and found ECG change of prolonged QT in 36%, ST depression in 24.5%, atrial fibrillation in 19.9% and T wave inversion in 17.8% of patients [5 ,6, 42].

Lane in Tucson USA, in a retrospective study sought to determine whether the nature and severity of cardiac arrhythmias in the context of an acute stroke vary in relation to where the stroke is located (the left or right hemisphere). He found that all four patients in their study with supraventricular tachycardia had right hemispheric stroke. There was a non-significant trend for left hemispheric stroke patients to have more severe ventricular arrhythmias [42]

Arboix and Alio in Barcelona, Spain carried out a prospective clinical study on one thousand patients with cerebral infarction (CI) (956 lacunars and 44 non lacunars infarctions) and in a control group (CG) (n=

1000) without organic cerebrovascular diseases to evaluate the ECG abnormalities. 72% of CI and 38% of CG had ECG abnormalities. These abnormalities were significantly more common at the beginning of the disease (72%) then three weeks after the development of focal neurological symptoms (54%). The major ECG findings were abnormal ventricular repolarization (changes in the ST segment and the T-wave), prolonged QTc interval and U-waves. Atrial fibrillations were significantly more common in non-lacunar infarctions (18%) than lacunar infarctions (2%) and in the CG (5%). Regarding the topography of the lesions, abnormalities of ECG were found in 80% of hemispheric infarctions, in 66% of the infarcts of basal ganglia and in 73% of brainstem infarctions. In cerebral infarction (CI) ECG abnormalities are common. They may be reversible, some of them are usually correlated with the type of CI and with the involved brain topography[43].

Liao in Ontario, Canada evaluated Holter ECG monitoring in five hundred and eighty-eight participants, new atrial fibrillation and flutter was detected in 4.6% of consecutive patients with ischaemic stroke. Duration of monitoring ranged from 24-72 hours. Liao concluded that screening consecutive patients with ishaemic stroke with routine Holter monitoring will identify new atrial fibrillation and flutter in approximately one in twenty patients[44].

Dogan in Turkey, studied two hundred and twenty-two stroke patients and compared ECG abnormalities in both ischaemic and heamorrhagic strokes. Sixty five percent of ischaemic stroke patients had ischaemic-like ECG changes and also fifty seven percent of haemorrhagic stroke patient. Atrial fibrillation was more frequent in ischaemic than haemorrhagic stroke (34% vs. 13%, $p = 0.01$) other ECG changes were not different in both groups. [45]

Ogun in Shagamu and Imarhiagbe in Benin both in Nigeria found that when CT scan is not available or its use is limited by distance the WHO criteria for acute stroke syndrome and the Benin Stroke Score are respectively useful.[46,47]

Aim and objectives:

1. To determine the prevalence of electrocardiographic abnormalities in stroke patients.
2. To determine the difference in ECG abnormalities between acute stroke cases and controls.

II. Materials And Methods

Study area and design: This study was carried in the University of Benin Teaching Hospital (UBTH) which is one of the six first generation hospitals in Nigeria that offers secondary and tertiary care to patients in Edo and neighbouring states. The was a descriptive study that assessed the prevalence of electrocardiographic abnormalities in stroke patients in the University of Benin Teaching Hospital (UBTH)

Sampling method: A simple non-randomized sampling method was used in selecting patients recruited for this study. One hundred and twenty patients presenting for the first time with clinical features and imaging findings of stroke (CT brain scan was performed in all cases) and were admitted into the UBTH medical wards along with one hundred and twenty age and sex matched controls (non-stroke patients) presenting to UBTH and were admitted into the medical wards. They had a detailed history and physical examination finding entered into the data acquisition sheet (copy attached).

Investigations carried out includes HIV screening, serum electrolyte, urea and creatinine and random blood sugar. ECG was performed on the stroke patients within the first twenty-four hours of presentation. ECG was done within the first twenty-four hours of admission in the controls

Inclusion criteria:

A. Patients that have first ever occurrence of stroke.
B. Patients that are eighteen (18) years old and above.
C. The patients that remain alive for at least 7 days post presentation (ECG was performed on presentation, third day and seventh day post presentation)

Exclusion criteria:

Patients excluded from this study were:

A. Patients that have two or more occurrence of stroke (recurrent stroke).
B. Patients less than eighteen (18) years of age.
C. Stroke resolved within twenty-four (24) hours, as evidenced by resolution of presenting complaints.
D. Patients that died within 7 days of presentation.
E. HIV positive patient.
F. Patients with malignancies.
G. Patients on immunosuppressive therapy.
H. Patients with electrolyte abnormalities.

Non-stroke patients (controls):
Controls in this study are:
A. Admitted non stroke patients that are eighteen (18) years and above.
Patients excluded from this study are:
A. Non stroke patients less than eighteen (18) years of age
B. HIV infected patients;
C. Patients with malignancies.
D. Patients on immunosuppressive drugs.
E. Patients with overt heart disease.

Data analysis: Anthropometric measurement and data collected using the preformat were collated and analyzed using the International Business Machines Statistical Product and Service Solutions (IBM- SPSS) version 22. Data were presented using tables and charts. Frequencies and percentages were used to present categorical data while continuous data were expressed as mean (Standard Deviation). Frequencies were compared using the Pearson's Chi-square test while means were compared using the independent t-test. Where the data was skewed, continuous data were expressed as mean (inter-quartile range) and compared using the Mann Whitney U test. Significant chi-square comparisons were further tested using a binomial logistic regression where applicable. A p value less than 0.05 were considered significant for all statistical comparisons.

ETHICAL CLEARANCE. Ethical clearance was obtained from the Research and Ethics Committee of the University of Benin Teaching Hospital, Benin City, Edo State. Informed consent was obtained from patients before participation in the study.Autonomy: Respect for respondents and confidentiality was maintained throughout the process of extracting the data.

III. Results

Table 1: Disease conditions in controls.

Disease condition	Frequency (N=120)	Percent (%)
Diabetes Mellitus	52	43.3
Koch Disease	16	13.3
Upper GI bleeding	16	13.3
Severe Hypoglycemia	8	6.7
Lobar pneumonia	8	6.7
Urinary tract infection	6	5.0
Chronic Gastro-enteritis	6	5.0
Cellulitis of the leg	4	3.3
Acute Poisoning	4	3.3

Diabetes mellitus comprising of uncontrolled type 2 diabetes mellitus, diabetic ketoacidosis and diabetic foot syndrome made up fifty-two (43.33%) of the controls. This was followed by Koch disease and upper gastro intestinal bleeding.

Table 2: ECG Observation in CVA and Control Cases

Finding	CVA n (%)	CONTROL n (%)	p value
NO ABN	28 (23.3)	111 (92.5)	<0.001
ABN	92 (76.7)	9 (7.5)	<0.001
SINUS TACHY	12 (13.0)	0 (0.0)	0.539
ATRIA FIB	12 (13.0)	0 (0.0)	0.539
LAD	48 (52.2)	0 (0.0)	0.008
RAD	0 (0.0)	0 (0.0)	
T INVERSION	4 (4.3)	0 (0.0)	>0.05
PROLONG QT	8 (8.7)	0 (0.0)	0,783
ST DEPRESSION	40 (43.5)	3 (33.3)	0.557
LAE	16 (17.4)	1 (11.1)	0.631
BAE	4 (4.4)	0 (0.0)	>0.05
LVH	28 (30.4)	4 (44.4)	0.389
RVH	0 (0.0)	1 (11.1)	0.147
LAFB	8 (8.7)	1 (11.1)	0.808

LBBB	0 (0.0)*	3 (33.3)	<0.001
PAC	4 (4.4)	1 (11.1)	0.372
PVC	4 (4.4)	0 (0.0)	>0.05
LLLV	4 (4.4)	0 (0.0)	>0.05
NSIB	4 (4.4)	0 (0.0)	>0.05
ROL	0 (0.0)	1 (11.1)	0.147

As in table 2 above, of the one hundred and twenty cases and controls, no ECG abnormalities were present in twenty-eight (23.33%) and one hundred and eleven (92.50%) of stroke cases and controls respectively. ECG abnormalities were present in ninety-two (76.67%) and nine (7.50%) of cases and controls. This difference was statistically significant, $p < 0.001$, OR 40.52, CI = 18.20 - 90.21

Rate: Twelve (13.00%) of cases and none of controls had sinus tachycardia on their ECG. There was no statistically significant difference between cases and control $p = 0.539$.

Rhythm: Twelve (13.00%) of cases and none of the controls had atrial fibrillation on their ECG. There was no statistically significant difference between cases and control $p = 0.539$.

Axis: Forty-eight (52.17%) had Left axis deviation and none of controls had Left axis deviation on their ECG. There was significant difference between cases and control $p = 0.008$

P wave: The p wave duration ranges from seventy-eight to one hundred and thirty four milliseconds for cases on day one and seventy eight to one hundred and sixteen milliseconds for controls. The mean p wave duration was 109.01 +/- 12.12ms for cases and 106.14 +/- 6.12ms for controls. The difference was not significant p greater than 0.05.

QRS complex: The QRS duration range from seventy-eight to one hundred and forty milliseconds with mean of 100.85 +/- 18.78ms on cases. The mean is within normal limit. The QRS duration range from seventy-eight to one hundred and eighteen milliseconds for controls with mean of 100.01 +/- 9.08ms. No significant difference between cases and control as $p > 0.05$

T wave inversion: Four (4.40%) and none of the control had this ECG abnormality. This difference was not significant, $p > 0.05$

PR interval: The mean PR interval was 154.04+/-19.18ms for the cases ranging between one hundred and fourteen to one hundred and ninety-four milliseconds. This was within normal range. The PR interval range from one hundred and two to one hundred and seventy-eight milliseconds with mean of 148.04 +/- 10.02ms. The difference was not significant $p>0.05$.

QT interval: Eight (8.67%) of cases and none of control had prolong QT interval. This difference between cases and control was not significant as $p = 0.783$

ST segment depression. Forty (43.50%) of cases and three (33.33%) of control had this ECG abnormality: This difference was not significant, $p = 0.557$.

Other observations. Left atrial enlargement was present in sixteen (17.39%) of cases and one (11.11%) of control. The difference was not statistically significant, $p = 0.631$. Four (4.44%) of cases had bi atrial enlargement on their ECG and none of controls. The difference between cases and controls was not significant, $p > 0.05$.

Left ventricular hypertrophy was present in ECG of twenty-eight (30.44%) cases and four (44.44%) of control. The difference was not statistically significant with $p = 0.389$. Right ventricular hypertrophy was absent on the ECG of cases but present in one (11.11%) of control. The difference was not significant, $p = 0.147$

Left anterior fascicular block was present in ECG of eight (8.70%) of cases and one (11.11%) of controls. This difference was not significant $p = 0.808$. Left bundle branch block was absent on ECG of cases. Among the controls, three (33.33%) had Left bundle branch block. The difference between cases and control was significant, $p < 0.001$.

Premature atrial complex was seen in four (4.44%) of the cases and one (11.1%) of control had premature atrial complex. The difference was not significant with $p = 0.372$. Premature ventricular complex was present in four (4.44%) of cases. This was not present on ECG of control. There was no statistical significance, $p > 0.05$.

Low limb lead voltage was present in four (4.44%) of cases and none of the controls. There was no statistically significant difference, $p > 0.05$. Non-specific intra-ventricular block was present in four (4.44%) of cases and none of the controls. Difference was not significant, $p > 0.05$. Reversal of leads was absent on ECG of cases, while present in one (11.11%) of control, $p = 0.147$.

IV. Discussion

One fourth of the stroke patients studied had no abnormality on their ECG on presentation. And three fourth had abnormalities on their ECG on presentation. The prevalence of ECG abnormalities in stroke cases in this study is 76. 7percent.The incidence of ECG abnormalities in stroke patients in the literature ranges from forty-nine to a hundred percent.[48] Left axis deviation was the most common ECG abnormality in this study.

Followed by ischaemic changes (comprising ST depression and T wave inversion), Left ventricular hypertrophy and left atrial enlargement respectively.

Though ECG abnormalities are generally more in stroke cases than control only left axis deviation was statistically significant among cases over control. And the reverse is the case with left bundle branch block among control over stroke cases.

ECG abnormalities have been noted to result from abnormal adrenergic activity with increased levels of plasma cathecolamines and dopamine. Furthermore, this tends to account for the arrhythmic, ischaemic and repolarization changes noted in stroke patients. An arrhythmogenic centre in the insular cortex may play a role in addition to possible hypothalamic and cardiac stimulation plus the sudden rise in intracranial pressure that occur in stroke patients, accumulation of these result in damage to the myocardium accounting for the changes described in all groups of patients in this study. [49-52]

V. Conclusion

The prevalence of ECG abnormalities in stroke patients in this study was 76.7%. Myriad types of ECG abnormalities are seen in stroke case but only left axis deviation was significant and left bundle branch block was significant among control controls when compared

Recommendation:

1. There is need to do similar studies using multicenter, larger number of patients and for longer duration to look at the ECG in stroke patients.
2. Many, if not any, abnormalities are possible on the ECG of stroke patients.
3. If ECG is found to be fairly or more accurately usable in strokediagnosis and or management, this could significantly reduce the cost burden of stroke management on the patients. (For example, CT brain scan is about twenty times more expensive than ECG test to the patients). In addition, the ECG machine is smaller, cheaper and easier to handle than the CT brain scan and require less expertise.

Limitations of this study: Diabetic patients were among controls. This is a single center study thus the sample size though adequate can be improved upon. A larger sample size would involve a large multicenter study which will take more time and resources beyond that available for this research

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