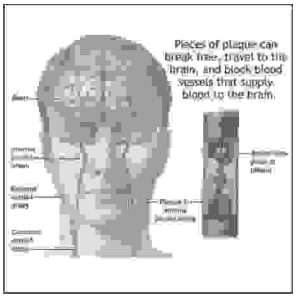
ORIGINAL PROF-1261

STROKE;
ELECTOCARDIOGRAPHIC CHANGES



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ABSTRACT... Objective: (1) To finding the frequency of various electrocardiographic abnormalities in sufferers of acute stroke (2) Comparing the ischemic and hemorrhagic stroke in this perspective. **Study design**: Observational. **Place and duration of study**: Emergency department of Nishtar Hospital, Multan from April 2003 to August 2005. **Patients and methods**: Two hundred patients of acute stroke who presented within 48 hours of the onset of neurological symptoms, were included in the study, irrespective of the comorbid conditions like hypertension, diabetes mellitus, valvular heart disease, coronary artery disease and cardiomyopathy. **Results**: Majority of the patients had ischemic stroke. ECG changes due to variation in heart rate, rhythm ischemia, QTc prolongation and left ventricular hypertrophy were frequent findings in sufferes of acute stroke. Except atrial fibrillation and left ventricular hypertrophy these changes were more or less similar in two types of stroke. **Conclusion**: A variety of electrocardiographic abnormalities can be found in sufferers of acute stroke. Except atrial fibrillation and left ventricular hypertrophy, there frequency is more or less similar whatever the type of stroke may be.

Key words: ECG, CT scan, QTc interval.

INTRODUCTION

Primary cardiac disorders can lead to stroke[1], but the idea that CNS disorder such as stroke may produce ECG changes and arrhythmia if fairly recent. For the first time in 1944, Byer and colleagues described the ECG changes in sufferers of subarachnoid hemorrhage[2]. ECG changes are present in anywhere from 60-90% of patients with intra-parenchymal or subarachnoid bleed and in about 5-20% of patients with acute ischemic stroke[3]. The underlying basis is disordered repolarization process[4]. There is a relation between these changes and sudden death in sufferers of stroke[5]. The possible mechanism is through disturbances in autonomic regulation and massive stimulation of the sympathetic nervous system[6]. Moreover the studies have shown that the frontal lobe, insular cortex and amygdale play an important role in regulating the heart rate via autonomic nerves[7,8].

Experimental studies even reveal that the left stellate

ganglion influences the posterior wall of the ventricles and the right stellate ganglion has influence over the anterior ventricular walls[7,8]. The ECG abnormalities most frequently noted are ischemic changes 35%, prolongation of QT interval 45% and disturbances in rate and rhythm 25%, which include atrial fibrillation, premature atrial and ventricular complexes, supra-ventricular and ventricular tachycardias (SVT and VT), torasede de pointis or polymorphic ventricular tachycardias[9,10].

Stroke induced ECG changes are evanescent, resolving over a period of days to months. However, the frequency and severity of ECG changes is highest within 48 hours of the onset of stroke which explains the importance of continuous ECG monitoring for these patients[11]. The objectives of this article are to know the frequency of various ECG changes in stroke patients. The article also compares the ischemic and hemorrhagic strokes in these parameters.

PATIENTS AND METHODS

This observational study was conducted from April 2003 to August 2005 at the Emergency department of Nishtar Hospital, Multan. It included 200 patients with acute stroke, admitted within 48 hours of the onset of neurological symptoms. A non-contrast CT scan of brain was used to define the stroke type. A 12 lead ECG was recorded at the time of admission. The first ECG was analyzed for estimation of various abnormalities like disturbance of heart rate, abnormalities of rhythm, bundle branch block (left or right), left ventricular hypertrophy, left ventricular hypertrophy and ischemia type changes were determined by measuring the ST/T deviation. Leads-III and V_6 were used for the estimation of QT interval, due to their higher importance in this aspect as compared to other leads[12]. Rate corrected QT interval (QTc) in these lead was calculated by using the formula[13]: Acute cut-off value of >=430 m sec was used for labeling a prolonged QTc[12,14,15]. While assessing the ECG abnormalities, pathologies like hypertension, coronary artery disease, valvular abnormalities and cardiomyopathies were not considered. Similarly, pre-stroke ECG changes were also not taken into account.

$$QTc = \frac{QT}{\sqrt{RR}}$$

RESULTS

A total of 200 patients were studied. Majority of the patients suffered ischemic stroke (58%) and were relatively older (mean age 62±12 years) to sufferers of hemorrhagic stroke (mean age 55±14 years) as shown in table-I.

Table-I. Demographic Data

Stroke type	No of pts	Mean age (Yrs)	Male	Female
Ischemic stroke	116(58%)	62±12	82(70.68%)	34(29.3%)
Hemorrhagic stroke	84(42%)	55±14	56(66.6%)	28(33.33%)

Most of the patients of hemorrhagic stroke had ECG evidence of left ventricular hypertrophy. Their proportion was quite large when compared with sufferers of cerebral infarction (69% vs 15.51%). Bundle branch block and premature ventricular complexes, although less frequently detected changes, were slightly more common in patients of ischemic stroke than the hemorrhagic variety (5.17% vs 15.5% each) as clear from table-II.

Table-II.

Stroke type	No of pts	Sinus tachycardia >=100/min	Sinus tachycardia >=120/min	AF	LVH	BBB	PVCs >=6/min
Ischemic stroke	116	63.8%	25.8%	17.24%	15.51%	5.17%	5.17%
Hemorrhagic stroke	84	30.95%	23.8%	-	69%	02.38%	02.38%

St-depression and T-inversion were more common manifestations of ischemic change than ST-elevation (46.66% vs 15.5%). This was true irrespective of the stroke type (Table-III).

Table-III.

Stroke type	No of pts	↓ ST	↑ ST	T-inversion	QTc III>=430m sec	QTcV_6>=430m sec
Ischemic stroke	88	46.66%	15.5%	46.6%	52.27%	63.63%
Hemorrhagic stroke	82	39%	17.07%	46.34%	63.4%	68.29

DISCUSSION

Cardiac autonomic imbalance generated by acute cerebral lesion plays an important role, not only in producing electrocardiographic abnormalities, but also in predisposing the patients towards early mortality[16]. Massive autonomic discharge along the sympathetic outflow tracts of the nervous system produces tachyarrhythmias[17]. Sinus tachycardia is a manifestation of such hypercatecholaminc state. Sinus tachycardia of higher magnitude may reflect a proportionately higher degree of autonomic discharge[18].

Our study demonstrates a frequency of 17.24% for atrial fibrillation, detected exclusively in patients with ischemic stroke[19]. This correlates with the results given by Bozluoclay, who detected the same ECG variable in 19.5% sufferers of ischemic stroke[17]. Due to unavailability of ECG record of many patients, it was not possible to calculate exactly the frequency of stroke induced atrial fibrillation. Atrial fibrillation is a parameter of early mortality due to stroke[20]. Chesebro has labeled atrial fibrillation as marker of multiple abnormalities of cardiovascular system in general[21]. It necessitates more sophisticated and sensitive methods of its detection, to be take into consideration. Observations of Jabaudon and colleagues have shown that 5% sufferer of acute stroke can have atrial fibrillation by Holter monitoring, despite negative surface ECG. Similarly 5.7% have atrial fibrillation detected by event loop recorder (ELR), with a normal standard ECG and Holter[22]. Such a study clearly concludes that surface ECG under diagnoses atrial fibrillation. From diagnostic and therapeutic point of view therefore, cardiac monitoring following acute stroke, with equipments like Holter and event loop recorder, should be mandatory. Left ventricular hypertrophy is detected more frequently in sufferers of hemorrhagic stroke (69%) than cerebral infarction (15.5%). A higher rate of occurrence of hypertension in patients with intra-cerebral hemorrhage could explain such difference. Goldstein gave a figure of 26% for this ECG change[9]. But he gave an overall estimate of this illness, rather than acute stroke, forms the basis for left ventricular hypertrophy and Bundle branch blocks. The underlying mechanism is volume/pressure overload[19]. Prolonged QRS complex decreases the stability of the myocardium[17]. Hence ectopic beats are precursors of ventricular tachy-arrhythmias. Premature ventricular complexes readily degenerate into condition like torasede de pointis and ventricular fibrillation[11]. Our study utilizes only a single surface electrocardiogram for detecting such arrhythmias, which might have underestimated the importance of this ECG variable. Its detection can be improved by Holter monitoring.

The frequency of ischemic change given in our study (46.6%) is close to the result calculated by Goldstein (35%)[9] and Bozloucly (37.9%)[19]. More frequent occurrence of ST-depression in sufferers of ischemic stroke than hemorrhagic stroke (46.6% vs 39%) is also given in another study[23]. The probable reason is sharing of same risk factors for coronary heart disease and ischemic stroke, which may co-exist in the same patient[24]. Our study does not adjust the frequency of ischemic changes for coronary artery disease. But regardless of origin, ST-segment changes are more or less similar in the two types of stroke[23.25]. Repolarization abnormality, manifesting as prolonged QTc comes as one of the most frequent ECG abnormalities in our study, irrespective of the stroke type (63.63% vs 68-29%). Our results correspond to the results of various international

studies (76% vs 71%)[14,20]. A prolonged QTc forms the basis for generation of torasede de pointis, hence its detection becomes important from prognostic point of view.

CONCLUSION

QTc prolongation, sinus tachycardia ischemic changes and left ventricular hypertrophy are the most frequent ECG abnormalities in sufferers of acute stroke. Atrial fibrillation, bundle branch blocks and premature ventricular complexes rank later. The two varieties of stroke manifest more or less similar patterns of ECG changes. Sufferers of acute stroke should receive continuous ECG monitoring for detection of these changes.

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CORRECTION

The amendment of the Professional Vol:14, No.02 (Prof-1036) page 193 are as under;

INCORRECT

ORIGINAL **PROF-1036**

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