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Electrocardiographic changes in patients with acute cerebrovascular stroke and their prognostic importance

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Abstract: Cerebrovascular stroke is a major public health concern and a leading cause of death worldwide. Complications after CVS can worsen neurologic and patient outcomes. Patients with CVS may have a variety of electrocardiographic changes which may be disturbance of rate, rhythm, P wave, QRS complex, PR interval, T wave, ST segment, QT interval or ischemic like changes. Early ECG may serve as an inexpensive test to screen for cardiac dysfunction prior to ordering more expensive and potentially more invasive testing. So, ECG should be a mandatory investigation for all patients with CVS. We were aiming in our study To determine if there are ECG changes with CVS and if these changes are related prognosis. In our study we found that ECG changes on admission are statistically significant but certain types of ECG changes were not correlated with outcome.
Further studies are required to more clarify the connection between these ECG abnormalities and CVS [Nermin [Sadek Nasr, Ass. Prof. Heba ahaa El Din El Serwi Ahmed Ezzet Mahmoud Bayoumy. **Electrocardiographic changes in patients with acute cerebrovascular stroke and their prognostic importance.** *Nat Sci* 2020;18(1):96-103]. ISSN 1545-0740 (print); ISSN 2375-7167 (online). http://www.sciencepub.net/nature. 13. doi:10.7537/marsnsj180120.13.

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1. Introduction

Cerebrovascular accidents, the second most frequent cause of death world-wide, accounts for 6.2 million deaths. Between 1990 and 2010 the number of stroke patients has decreased by approximately 10% in the developed countries and increased by 10% in the developing countries **(Feigin et al., 2012).**

Stroke is the term used to describe episodes of focal brain dysfunction due to focal ischemia or hemorrhage. The incidence of first stroke is around 1.5/1000/year, and of transient ischemic attack 0.6/1000/year. About 22% of all strokes are recurrent events **(Hamel, 2006).**

Cardiovascular abnormalities are common after a stroke. Studies have evaluated electrocardiogram changes and rhythm disturbances in ischemic stroke patients without primary heart disease to distinguish abnormalities specifically associated with acute stroke **(Aro et al., 2014)**.

Disorders of the central nervous system cause a wide array of cardiovascular system dysfunction ranging from electrocardiogram changes and transient myocardial dysfunction to sudden cardiac death. Electrocardiogram changes are present in 60-90% of patients with intra-parenchymal or subarachnoid bleeding and in about 5-20% of patients with acute ischemic stroke **(Toghaet al., 2013)**.

The underlying basis is disordered repolarization process. The possible mechanism is through disturbances in autonomic regulation and massive stimulation of the sympathetic nervous system. Moreover studies have shown that the frontal lobe, insular cortex and amygdale play an important role in regulating the heart rate via autonomic nerves **(Xavier et al., 2012).**

Aim of the Work

The aim of this study is to determine the incidence of electrocardiographic changes associated with isolated acute cerebrovascular stroke, and to study if these changes are related to prognosis.

2. Patients and Methods

Prospective cross sectional study on 50 patients admitted to Ain Shams university critical care units and Al Helal Hospital critical care units within 48 hours of onset of acute cerebrovascular stroke.

Inclusion criteria

1. Age: 18 - 60 years.
2. Sex: both sexes.
3. Acute spontaneous cerebrovascular strokes (either hemorrhagic or infarction).

Exclusion criteria

1. Patients with past history of cardiovascular disease.
2. Patient with chest trauma with high troponin level.
3. Patients with prior stroke, stroke related to trauma and patients with subarachnoid hemorrhage.
4. Patient with electrolyte disturbance.
5. Patients who underwent neurosurgery.

Technique

Diagnosis of acute stroke will be established in all patients by NCCT scan either on admission or 48 hrs after or MRI scan acute cerebrovascular stroke.

Then in all patients the following data will be recorded:

1) Demographic data including age, sex, date of ICU admission and preexisting underlying disease.

2) Clinical examination determining level of consciousness and signs of lateralization.

3) 12 lead electrocardiogram record at the time of admission and during the next 48 hours as regard (ST segment elevations or depressions, T wave changes, U waves and dysrhythmias).

4) Troponin I will be measured on admission to exclude cardiac disease, and if there is any ECG changes recorded.

5) The outcome of the patient conditions as regard mortality or discharge from ICU and length of ICU stay.

Statistics and Results

Agreement statistics, sensitivity, specificity and predictive changes will be calculated.

3. Results

Thecurrentstudyaimedtodeterminetheincidenceof electrocardiographicchanges associated with isolated acute cerebrovascular stroke, and to study if these changes are related to prognosis. A prospective cross-sectional study was conducted on 50 patients admitted to Ain Shams university critical care units and Al Helal Hospital critical care units within 48 hours of onset of acute cerebrovascular stroke.

The mean age of the study participants was 52.7±5.1 ranging 41-60. Males were slightly more than females (58.0% and 42.0% respectively). Diabetes mellitus was found in 16% of participants while 38% were hypertensive (Table 1).

Table (1): Basic characteristics of the study participants

Parameters	No.	%
Age (mean ± SD, range)	52.7±5.1 41-60	
Gender		
Males	29	58.0
Females	21	42.0
	comorbidities	
DM	8	16.0
HTN	19	38.0

As presented in Table 2, it was found that 64% of the study participants showed signs of lateralization on admission. Hemorrhage was found in 58% of them and 42% had infarction. ECG was abnormal in 80% of the participants. The most common ECG abnormality was QT prolongation (24%) followed by T-wave inversion (18%).

Table3 shows that ECG abnormalities decreased to 76% after 48hours of admission when 2 of the participants admitted with T-wave inversion changed to normal. The mean duration of ICU stay was 6.4±5.4 ranging 2-20. Out of the study participants 64% died and 36% stayed alive on discharge.

Table (2): Clinical data of the study participants on admission

Parameters	No.	%
Clinical examination		
Low GCS less than 12/15	18	36.0
Lateralization	32	64.0
Type of stroke		
Hemorrhage	29	58.0
Infarction	21	42.0
ECG changes on admission		
Normal	10	20.0
Abnormal	40	80.0
Type of ECG changes on admission		
QT prolongation	12	30.0
T wave inversion	9	22.5
ST depression	7	17.5
Sin tach	6	15.0
U wave	3	7.5
Brad	2	5.0
Other	1	2.5

Table (3): Clinical data of the study participants after 48 hours

Parameters	No.	%
ECG changes after 48 hours		
Normal	12	24.0
Abnormal	38	76.0
Type of ECG changes 48 hours in all patients		
QT prolongation	12	24.0
T wave inversion	7	14.0
ST depression	7	14.0
Sin tach	6	12.0
U wave	3	6.0
Brad	2	4.0
Other	1	2.0
Duration of ICU stay (mean ± SD, range) in all patients	6.4±5.4	2-20
Outcome		
Dead	32	64
Alive	18	36

As shown in Table 4, there was a statistically significant relation between outcome on discharge and gender where 61.1% of dead were females and 68.8% of discharged alive were males.

Table 5 shows that there was a statistically significant relation between outcome on discharge and low GCS (p<0.001) were 77% of dead patients had low GCS. On the other hand, signs of lateralization were significantly higher among patients discharged alive than dead ones (87.5% vs. 22.2% respectively) (p<0.001). Hemorrhage was found in 77.8% of dead while infarction was present in 22.2% (p=0.034). Abnormal ECG changes on admission was significantly higher among dead (100%) than those discharged alive (68.8%) (p=0.009). Regarding the type ECG changes, QT prolongation was significantly higher among dead patients (50%) than discharged alive (9.4%) (p=0.004).

Table (4): Relation between outcome on discharge and basic characteristics of the study participants

Parameters	Alive (32)	Dead (18)	P value	OR (95% C.I)
Age (mean ± SD)	52.5±5.0	53.2±5.3	0.641	-
Gender				
Males	22(68.8%)	7(38.9%)	0.040*¥	For female gender 3.5(1.0-11.6)
Females	10(31.3%)	11(61.1%)		
DM	4(12.5%)	4(22.2%)	0.436$	2.0(0.4-9.2)
HTN	9(28.1%)	10(55.6%)	0.055¥	3.2(1.0-10.7)

¶ Mann-Whitney U test * Statistically significant at p<0.05 ¥ Chi-square test $ Fisher's exact test

Table (5): Relation between outcome on discharge and clinical data of the study participants on admission

Parameters	Alive (32)	Dead (18)	P value	OR (95% C.I)
Clinical examination				
Low GCS less than 12/15	4(12.5%)	14(77.8%)	<0.001*¥	24(5.3-112.8)
Lateralization	28(87.5%)	4(22.2%)	<0.001*¥	0.0(0.0-0.2)
Type of stroke				
Hemorrhage	15(46.9%)	14(77.8%)	0.034*¥	For hemorrhage 4.0(1.1-14.7)
Infarction	17(53.1%)	4(22.2%)		
ECG changes on admission				
Normal	10(31.3%)	0(0.0%)	0.009*$	-
Abnormal	22(68.8%)	18(31.2%)		
Type of ECG changes on admission				
QT prolongation	3(9.4%)	9(50.0%)	0.004*$	9.7(2.1-43.7)
T wave inversion	4(12.5%)	3(16.7%)	0.692$	1.4(0.3-7.1)
ST depression	4(12.5%)	3(16.7%)	0.692$	1.4(0.3-7.1)
Sinus tachycardia	5(15.6%)	1(5.6%)	0.399$	0.3(0.0-3.0)
U wave	3(9.4%)	0(0.0%)	0.544$	-
Bradycardia	2(6.3%)	0(0.0%)	0.530$	-
Other	1(3.1%)	0(0.0%)	1.000$	-

* Statistically significant at p<0.05 ¥ Chi-square test $ Fisher's exact test

Table (6): Relation between outcome on discharge and clinical data of the study participants after 48 hours

Parameters	Alive (32)	Dead (18)	P value	OR (95% C.I)
ECG changes after 48 hours				
Normal	10(31.3%)	2(11.1%)	0.170$	3.6 (0.7-18.9)
Abnormal	22(68.8%)	16(88.9%)		
Type of ECG changes 48 hours				
QT prolongation	3(9.4%)	9(50.0%)	0.004*$	9.7 (2.1-43.7)
T wave inversion	4(12.5%)	5(27.8%)	0.253$	2.7 (0.7-11.7)
ST depression	4(12.5%)	3(16.7%)	0.692$	1.4 (0.3-7.1)
Sinus tachycardia	5(15.6%)	1(5.6%)	0.399$	0.3 (0.0-3.0)
U wave	3(9.4%)	0(0.0%)	0.544$	-
Bradycardia	2(6.3%)	0(0.0%)	0.530$	-
Other	1(3.1%)	0(0.0%)	1.000$	-
Duration of ICU stay (mean ± SD)	4.1±3.8	10.4±5.4	<0.001*¶	-

* Statistically significant at p<0.05 $ Fisher's exact test ¶ Mann-Whitney U test

As presented in table 6, the length of ICU stay was significantly higher among dead patients

(10.4±5.4) than patients discharged alive (4.1±3.8). (p<0.001).

A backward conditional logistic regression analysis was done for predictors of outcome on discharge among study subjects. Factors entered into the model were sex, GCS, stroke type, duration of ICU stay and ECG changes on admission. As presented in table 7, predictors of outcome on discharge were female gender, low GCS on admission and ECG changes on admission although were statistically insignificant.

Table (7): Predictors of outcome on discharge among the studied patients.

	Beta	Standard error	P value.	Odds ratio	95% C.I. for OR	
					Lower	Upper
Female gender	2.318	1.131	0.040	10.160	1.108	93.180
Low GCS	3.557	1.130	0.002	35.056	3.827	321.129
ECG changes on admission	19.406	11829.271	0.999	267917710.980	0.000	.
Constant	-22.380	11829.271	0.998	0.000		

4. Discussion

The mechanisms by which acute cerebrovascular events cause ECG changes are unsettled. It has been suggested that changes in the autonomic nervous system activity can be primarily responsible for these ischemic, arrhythmic, and repolarization changes. **(Myers et al., 1981 and Bozluolcay et al., 2003).**

The current study aimed to determine the incidence of electrocardiographic changes associated with isolated acute cerebrovascular stroke, and to study if these changes are related to prognosis.

The demographic and historical data is very important in patients with acute cerebrovascular stroke as it helps in its diagnosis and treatments, as the electrocardiographic spectrum seems to be related to the type of cerebrovascular disease and its localization. (Khechinashvili and Asplund, **2002).**

The autonomic and cardiovascular effects of stroke; however, are modulated by concomitant factors such as pre-existent cardiac diseases and electrolyte disorders. Although many subsequent reports have described ECG abnormalities and rhythm disturbances in stroke, especially subarachnoid hemorrhage (SAH), few have included an adequate number of patients to statistically assess the relative frequencies of these abnormalities among the pathophysiologic categories of stroke. Furthermore, few previous studies have evaluated ECG changes and rhythm disturbances in ischemic stroke patients without primary heart disease to distinguish abnormalities specifically associated with acute stroke. In view of the varied explanations for the ECG abnormalities in acute CVA. **(**Togha **et al., 2013).**

Our results on historical and demographic aspects of the patients cleared that, the mean age of the study participants was 52.7±5.1 ranging from 41-60. Males were slightly more than females (58.0% and 42.0% respectively). Diabetes mellitus was found in 16% of participants while 38% were hypertensive.

Ibrahim et al. (2016) reported that, the mean QT dispersion (QTD) was higher in hypertensive patients (65 ± 22 versus 49 ± 19 ms and p = 0.029) and was not affected by dyslipidemia, diabetes mellitus, age or gender.

Also, Khechinashvili and Asplund. (2002) observed that, repolarization and ischemic-like electrocardiographic (ECG) changes that were observed during acute phase of stroke may cause misdiagnostic and management problems for the clinician. The information which is taken from the history can help with the results obtained from ECG changes and QT prolongation during the acute phase of stroke and their coexistence with other abnormal cardiac findings obtained, that includes ischemic-like ECG changes and/or QT prolongation, that in their study were found in 76% of the examined patients with subarachnoid hemorrhage. Such ECG changes were present in more than 90% of unselected patients with ischemic stroke and intracerebral hemorrhage, but the prevalence was much lower after exclusion of patients with preexisting heart disease. In comparison with other abnormal cardiac findings (cardiac wall motion abnormality detected by echocardiography, elevated levels of biochemical markers of myocardial injury, autopsy findings, thallium scintigraphy), these ECG changes were characterized by a high sensitivity but a very low specificity. Thus, in patients with subarachnoid hemorrhage, repolarization and ischemic-like ECG changes are mainly direct consequences of the cerebral condition and their absence essentially rules out cardiac abnormalities. In patients with ischemic stroke and intracerebral hemorrhage, these ECG abnormalities (and QT prolongation) most often represent preexisting coronary artery disease. The specificity of ECG changes to diagnose acute myocardial infarction is low in the acute phase of stroke.

Meanwhile, our results on **clinical data of the study participants on admission** cleared that, about 64% of the study participants showed signs of lateralization on admission. Hemorrhage was found in 58% of them and 42% had infarction. ECG was

abnormal in 80% of the participants. The most common ECG abnormality was QT prolongation (30%) followed by T-wave inversion (22%).

This is in agreement with **Lazar et al. (2003),** who studied QTD in 140 patients with acute stroke, and transient ischemic attacks (TIA). They found that QTD was higher in patients with intracerebral hemorrhage (P = 0.03) as compared to infarction stroke & TIA. QT interval on ECG represents total duration of ventricular depolarization and repolarization. QT-dispersion represents increased variation or heterogeneity of ventricular repolarization and has been used as a prognostic marker for ventricular arrhythmias in patients with cardiac diseases. As the heart has important and pronounced autonomic innervations, it is to be expected that neurovascular disturbances might result in a wide spectrum of cardiac function disorders **(Oppeheimer et al., 1994 and Pinto et al., 2006).**

These results agreed with those of **(Alter et al., 1986)** where they cleared that, people suffer from acute cerebrovascular events, including ischemic stroke, intracerebral and subarachnoid hemorrhage, giving a mortality of nearly 20%.

Also, agreed with those of**, (Goldstein, 1979; Villa et al., 2001; Sommargren, 2002; Dogan et al., 2004; van den Bergh et al., 2004; Catanzaro et al., 2008),** where they reported that, acute strokes, especially subarachnoid hemorrhage is frequently accompanied by a variety of electrocardiographic (ECG) abnormalities, some of which may be indistinguishable from those seen in association with an episode of severe myocardial ischemia and/or infarction.

Meanwhile, the **clinical data of the study participants** showed that ECG abnormalities decreased to 76% after 48 hours of admission when 2 of the participants admitted with T-wave inversion changed to normal. The mean duration of ICU stay was 6.4±5.4 ranging 2-20. Out of the study participants 64% died and 36% stayed alive on discharge.

The appearance of these symptoms and signs attributed to, Sustained sympathetic stimulation results in structural damages to the myocardium, which may be mediated by a sudden increase in intracranial pressure, **(Shanlin et al., 1988)**, hypothalamic, **(Melville et al., 1963 and Pinto et al., 2006)** and cardiac nerve stimulation or through an arrhythmogenic center in the insular cortex. **(Hirashima et al., 2001).** Moreover, direct damage to the cardiac innervations or imbalance between the left and right sympathetic outflows to the heart, underlying atherosclerotic or hypertensive cardiovascular disease, or asymptomatic/undetectable primary heart disease are among the suggested causes. **(Greenhoot and Reichenbach , 1969; Natelson, 1985 and Pinto et al., 2006).**

Our results agreed with those of **(**Togha**, et al., 2013)** where they reported that,. Electrocardiographic (ECG) changes are frequently after acute strokes. These results were attributed to that cardiovascular effects of strokes are modulated by concomitant or pre-existent cardiac diseases, and are also related to the type of cerebrovascular disease and its localization. Also, the pattern of ECG changes associated with pathophysiologic categories of acute stroke among patients with/without cardiovascular disease and also, the specific ECG changes are related to the location of the lesion.

Meanwhile, our results on the **Relation between outcome on discharge and basic characteristics of the study participants showed that,** there was a statistically significant relation between outcome on discharge and gender where 61.1% of dead were females and 68.8% of discharged alive were males.

Meanwhile, our results on the **relation between outcome on discharge and clinical data of the study participants on admission, cleared that,** there was a statistically significant relation between outcome on discharge and low GCS ($p<0.001$) were 77% of dead patients had low GCS. On the other hand, signs of lateralization were significantly higher among patients discharged alive than dead ones (87.5% vs. 22.2% respectively) ($p<0.001$). Hemorrhage was found in 77.8% of dead while infarction was present in 22.2% ($p=0.034$).

Abnormal ECG changes on admission was significantly higher among dead (100%) than those discharged alive (68.8%) ($p=0.009$). Regarding the type ECG changes, QT prolongation was significantly higher among dead patients (50%) than discharged alive (9.4%) ($p=0.004$).

Our results agreed with those of, **(Familoniet al., 2006),** where they found that, thirty-five (54.7%) of the patients had ischemic-like ECG changes made up of ST depression (29.7%), T-wave inversion (21.8%) and U wave (9.3%). Twenty-eight (43.8%) had QTcmax prolongation. Twenty-four (37.5%) of the patients had no pre-existing heart disease. The QT was similar when compared with the total cohort except in QTcmax, where there was significant difference (447.3+/-72.2 vs. 408.6+/-40.3 msecs). Mortality rate of the total cohort at 28.1% was significantly higher than in those without pre-existing heart disease at 8.3%, suggesting that presence of pre-existing heart disease contributed to mortality. QTcmax ($r=0.293$ $p=0.045$) and days on admission ($r=-0.543$ $p=0.001$) were the other variables that correlated with mortality in the total cohorts. They concluded that: Ischemic-like and repolarisation ECG changes are common in patients with acute ischemic

stroke. These changes tend to be due to pre-existing heart disease rather than the stroke state.

The abnormalities of ECG that appear in the examined patients of this study attributed to SAH causes a decrease in serum magnesium mediated possibly by free fatty acids, sympathetic stimulation, and coronary vasospasm (decreasing the serum magnesium also causes vasospasm**). (van den Bergh et al., 2004).** Although widely reported, the prolonged length of the QTc interval was debated by **Shuster. (1960) and Tümüklü et al. (2019)**, they cautioned against inadvertently including the U-wave in the QTc interval. The U-wave, which is often 1 mm or greater in amplitude in patients with SAH, can be mistakenly interpreted as a part of a notched T-wave if the U wave occurs early during repolarization.

Abnormal T-wave was the most abnormal ECG findings in patients with ischemic stroke. However, the higher incidence of abnormal T-wave in ischemic stroke patients with cardiovascular diseases implies the influences of other factors on the T-wave than the intracranial disorder in this group. The accurate interpretation of T-wave changes can assist the clinician toward an accurate diagnosis. Low-amplitude and abnormally inverted T-waves may be the result of non-cardiac pathophysiology; while T-wave inversions produced by MI are classically narrow and symmetric. ST segment depression is another ECG change often reported in SAH patients**. (Catanzaro et al., 2008).** It has been reported that advanced age itself, which is the time of occurrence of stroke, **(Goldstein et al., 1979; Lindgren et al., 1994 and McDermott et al., 1994 and Tümüklü et al., 2019),** is associated with the presence of ST-segment change. In the population based-study, ischemia-like ECG changes were observed in 27% of men and 31% of women in the population aged 65–74 years. New pathologic Q-waves, in contrast to ischemic changes, rarely occur in stroke. Only 2 cases in which acute strokes produced Q-waves without an autopsy evidence of infarction have been reported, yet. **(Goldstein , 1979).**

The high frequency of ECG abnormalities in our study population is in line with the frequency reported in the literature. **(Goldstein, 1979; Mayer et al., 1999; Villa et al., 2001; Sommargren, 2002; Dogan et al., 2004; van den Bergh et al., 2004; Catanzaro et al., 2008; Frontera et al., 2008; Liman and Endres, 2008),** According to the results of these studies, the ischemia-like ECG abnormalities and QT interval prolongation can occur in more than 90% of the patients with ischemic or hemorrhagic stroke, and this rate will decrease by 8–40% if patients with a history of known heart disease and treatment by cardiac drugs are excluded. In an early study of 150 patients with acute stroke, **Goldstein , (1979) and** Damhoff and Huecker, 2018**).,** detected QT prolongation in 45%, ischemic changes in 39%, arrhythmias in 27%, and U-waves in 28% of their patients. In a study on patients with ischemic stroke but without history of primary heart disease, **Dogan *et al*. (2004)** found ischemia-like ECG changes in 65% of patients, QTc interval prolongation in 26%, and arrhythmias in 44% of them. In the study of **(Lindgren *et al*., 1994)** transient ST-T changes were found in 54% of patients with ischemic stroke with no primary heart disease. Prominent U-wave, QT interval prolongation, and arrhythmia were observed in 17%, 13%, and 4% of them, respectively. Compared with our study, however, that study included only 24 patients with cerebral infarction. On the other hand, in the study of **McDermott *et al*., (1994)** ST-segment depression was noted in only 8% of the selected patients without a history of coronary heart disease, whereas it was observed in 60% of all patients. However, the number of study patients was also limited to 51 cases.

Most of the ECG abnormalities described after an acute cerebral event were linked to SAH. In SAH, the incidence of ECG abnormalities ranges from 49% to 100%, including changes in ST segment (15% to 51% of patients), T-waves (12% to 92%), prominent U-waves (4% to 47%), QT prolongation (11% to 66%), pathological Q waves, and sinus dysrhythmias**. (Frontera et al., 2008 and** Damhoff and Huecker, 2018**).**

This results agreed with tose of **(**Togha **et al., 2013)** where they observed that, Ischemia-like ECG changes and arrhythmias are frequently seen in stroke patients, even in those with no history or signs of primary heart disease, which support a central nervous system origin of these ECG abnormalities. Further study is necessary to better define the brain-heart interaction.

Also, **(Stone et al., 2018)** reported that, ECGs were examined for cerebral T waves, defined as T-wave inversion of ≥5 mm depth in ≥4 contiguous precordial leads. Echocardiograms of those meeting these criteria were examined for the presence of left ventricular (LV) wall motion abnormalities. Follow-up evaluation included both ECG and echocardiogram. Of the 800 patients, 17 had cerebral T waves on ECG (2.1%). All 17 patients had ischemic strokes, of which 11 were in the middle cerebral artery distribution (65%), and 2 were cerebellar (12%), whereas the remaining 4 involved other locations. Follow-up ECG showed resolution of the T-wave changes in all 17 patients. Of these patients, 14 (82%) had normal wall motion, and 3 had transient wall motion abnormalities (18%). Two of these patients had Takotsubo-like cardiomyopathy with apical ballooning, and the third had globally reduced LV

function. Coronary angiography showed no significant disease to explain the LV dysfunction. In summary, in our study of patients with acute stroke, cerebral T waves were rare and occurred only in ischemic stroke. Eighteen percent of patients with cerebral T waves had significant transient wall motion abnormalities. Patients with stroke with cerebral T waves, especially in those with ischemic strokes, should be assessed for cardiac dysfunction.

Our results on the r**elation between outcome on discharge and clinical data of the study participants after 48 hours showed that,** the length of ICU stay was significantly higher among dead patients (10.4±5.4) than patients discharged alive (4.1±3.8). ($p<0.001$).

While, our results on the **Predictors of outcome on discharge among the studied patients, cleared that, a** backward conditional logistic regression analysis was done for predictors of outcome on discharge among study subjects. Factors entered into the model were sex, low GCS less than12/15, stroke type, duration of ICU stay and ECG changes on admission. As presented in table 7, predictors of outcome on discharge were female gender, low GCS on admission and ECG changes on admission.

Our results agreed with **(**Togha **et al., 2013)** where they attempts to correlate the ECG abnormalities with the location of the brain lesion that, have been made previously by several authors, but the reported results are divergent. The direct stimulation of many areas of the CNS is known to result in abnormal ECG patterns; while no relationship of the ECG changes, to the site of the bleeding aneurysm, was found by **Cropp and Manning, (1960), Shuster (1960) and Hunt *et al.* (1969) and** Damhoff and Huecker (2018)**.,** In studies focused on the intracranial vascular spasm after SAH and the appearance of ECG abnormalities, **Wilkins *et al.* (1968)** showed no relationship, while **Stober and Kunze (1982) and Tümüklü et al. (2019)** were able to find a correlation between cerebral arteries spasms of the left hemisphere, T-wave inversion, and QT prolongation. A correlation between arrhythmias and T-wave abnormalities and hemorrhages in the anterior cerebral circulation has been reported in patients with cerebral hemorrhages. **(Yamour et al., 1980).** Also, **Dogan *et al.* (2004)** observed that lesion of insular cortex, which lies beneath frontoparietal and superior temporal opercula, leads to several cardiac abnormalities such as ischemic ECG changes, arrhythmias, and myocytolysis. **(Oppenheimer et al., 1992 and Tümüklü et al., 2019).**

Our results agreed with those of (Ibrahim, et al., 2016) where they observed that, out of 40 patients, 25% died within the first 3 months of stroke. Increased QTD and lower GCS on admission were the only predictors of high mortality. The hemorrhagic stroke has high mortality of 15–30%. Our study showed 7 patients (35% of hemorrhagic strokes and 17% of all strokes) died of hemorrhagic stroke. The size of infarction or hemorrhage was not evaluated as prognosticator. The possibility that QTD in our patients with acute stroke is due to humoral change in the level of catecholamines is speculative (depending on previous studies) as we did not measure it in our study population as well as that the number of our patients did not allow for subgroup analysis or studying lesion size effect on QTD. Despite these limitations, it was concluded that in patients presenting with acute neurological events and increased QTD on admission ECG was significantly related to functional outcomes. Furthermore, increased QTD was related to the type of neurological event as QTD values were greater in patients with subarachnoid hemorrhage.

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