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Characterization of electrocardiographic findings and their relationship with mortality in acute cerebrovascular disease

Julio O. Cabrera-Rego[1], MD, PhD; Abdel del Busto Mesa[2], MD; Jorge L. Munguía Rodríguez[3], MD; and Alberto A. Yanes Quintana[1]✉, MD

[1] Intensive Coronary Care Unit, Hospital Docente Manuel Fajardo. Havana, Cuba.
[2] Department of Endocrinology. Hospital Docente Miguel Enríquez. Havana, Cuba.
[3] Facultad de Ciencias Médicas Finlay-Albarrán, Hospital Docente Joaquín Albarrán Domínguez. Havana, Cuba.

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ARTICLE INFORMATION

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Abbreviations
CVD: cerebrovascular diseases
ECG: electrocardiogram
IPH: intraparenchymal hemorrhage
SAH: subarachnoid hemorrhage

✉ *AA Yanes Quintana*
Unidad de Cuidados Coronarios Intensivos. Hospital Docente "Manuel Fajardo". Zapata y 29 El Vedado. La Habana, Cuba.
E-mail address:
npfm@infomed.sld.cu

ABSTRACT

Introduction: In Cuba, descriptive and experimental studies that explore cardiovascular disorders secondary to neurological diseases are scarce, both in the clinical and neurocardiology fields.
Objectives: To characterize the electrocardiographic findings in the first 72 hours of evolution of the cerebrovascular disease and its relation to mortality.
Method: An observational, descriptive, longitudinal prospective study was carried out in 166 patients admitted to the Hospital Clínico-Quirúrgico Joaquín Albarrán, with the diagnosis of cerebrovascular disease of any etiology and form of presentation, during the period of January 2015 to December 2016.
Results: Electrocardiographic findings were present in 32.5% of patients, mainly sinus tachycardia (27.7%), T wave inversion and premature atrial contractions (13.3% each). A significantly higher frequency of electrocardiographic changes was found in patients with subarachnoid hemorrhage (33.3% vs. 5.4%), lower score on the Glasgow coma scale (29.7% vs. 5.4%) and location at the level of the basal ganglia (50.0%). The presence of new electrocardiographic findings was related to a 7.2 times greater probability of in-hospital death (40.7% vs. 7.1%).
Conclusions: The presence of new electrocardiographic alterations in patients with cerebrovascular disease can be used as a marker of risk of in-hospital mortality.
Keywords: Stroke, Electrocardiography, Cardiac arrhythmias

Caracterización de los hallazgos electrocardiográficos y su relación con la mortalidad en la enfermedad cerebrovascular aguda

RESUMEN

Introducción: En Cuba, los estudios descriptivos y experimentales que exploran los trastornos cardiovasculares secundarios a enfermedades neurológicas son escasos, tanto en el campo de la clínica como de la neurocardiología.
Objetivo: Caracterizar los hallazgos electrocardiográficos en las primeras 72 horas de evolución de la enfermedad cerebrovascular y su relación con la mortalidad.
Método: Se realizó un estudio observacional, descriptivo, de corte longitudinal prospectivo, en 166 pacientes ingresados en el Hospital Clínico-Quirúrgico Joaquín Albarrán, con el diagnóstico de enfermedad cerebrovascular de cualquier etiología y forma de presentación, durante el período de enero de 2015 a diciembre de 2016.

Resultados: Los hallazgos electrocardiográficos estuvieron presentes en el 32,5% de los pacientes, fundamentalmente la taquicardia sinusal (27,7 %), la inversión de la onda T y las extrasístoles auriculares (13,3% cada uno). Se encontró una frecuencia significativamente mayor de cambios electrocardiográficos en los pacientes con hemorragia subaracnoidea (33,3% frente a 5,4%), menor puntuación en la escala de coma de Glasgow (29,7% frente a 5,4%) y localización a nivel de los ganglios basales (50,0%). La presencia de nuevos hallazgos electrocardiográficos se relacionó con una probabilidad 7,2 veces mayor de muerte intrahospitalaria (40,7% frente a 7,1%).

Conclusiones: La presencia de nuevas alteraciones electrocardiográficas en pacientes con enfermedad cerebrovascular puede ser empleado como un marcador de riesgo de mortalidad intrahospitalaria.

Palabras clave: Accidente cerebrovascular, Electrocardiografía, Arritmias cardíacas

INTRODUCTION

In Cuba, cerebrovascular diseases (CVD) represent the third cause of death, second only to cardiovascular diseases and cancer. They contribute to more than 50% of admissions from acute neurological diseases in secondary care centers in the country, with hospital mortality rates ranging from 10 to 64% for different etiologic subtypes. In the year 1970 there were 5155 deaths, in 2005 there were 8787 and in 2016, 9011 deaths were reported due to this disease, what evidences the increase in the number of deaths due to this cause[1].

It is known for centuries that cardiac diseases can cause secondary involvement of the central nervous system. The most frequent example is the cardioembolic ischemic stroke, secondary to atrial fibrillation. However, this fact seen in reverse, *i.e.*, lesions of the central nervous system that cause secondary cardiac alterations, was raised only half a century ago, by Byres, in 1947, who described it in the subarachnoid hemorrhage. It was not until 1954 that Burch reported that patients with stroke could also have electrocardiographic alterations similar to cases of hemorrhagic stroke, although it is in the latter where it has been identified more often[2].

In Cuba, in 1980, doctors Manuel and Orlando Hernandez Meilán conducted extensive work on the electrocardiographic manifestations found in patients suffering from ischemic and hemorrhagic stroke, and they obtained as a result a prevalence of disorders of the rhythm rather than morphological, as well as an incidence of these alterations similar to studies in later years, at the *Instituto de Neurología y Neurocirugía Dr. Rafael Estrada González*, when corroborating what had been researched in other universities and specialized centers of the world[3].

Nevertheless, in Cuba, descriptive and experimental studies that explore cardiovascular disorders secondary to neurological diseases are scarce, both in the clinical and neurocardiology fields[3,4]. Identifying possible early complications in the course of an acute stroke, and being able to be evaluated by means of an electrocardiographic tracing, represents an extremely important matter from the clinical point of view, and a fundamental challenge in the increasingly successful treatment of these diseases for the medical community. For this reason, it was established as the objective of this work.

METHOD

An observational, descriptive, longitudinal prospective study was conducted in 166 patients admitted to the *Hospital Clínico-Quirúrgico Joaquín Albarrán* during the period from January 2015 to December 2016, with the diagnosis of CVD of any etiology and form of presentation, who came in the first 24 hours of the evolution of clinical symptomatology.

Exclusion criteria

The patients excluded were the ones with: 1) craniocencephalic or thoracic trauma, 2) hidroelectrolyte and acid-basic unbalances, 3) previous CVD, 4) cardioactive drugs history (beta-blockers, non-dihydropyridine calcium antagonists, digoxin, vasoactive amines and amiodarone), 5) hemodynamic instability (sustained hypotension or requiring support with amines to maintain a stable mean blood pressure) and other concomitant neurological pathology.

Procedure

A structured interview was conducted where variables such as age, sex, clinical data and personal pathological history were researched. The patients underwent a general physical, neurological and cardiovascular examination at the time of admission to the unit, as well as a conventional 12-lead electrocardiogram upon arrival of the patient and daily in the first 72 hours of evolution. All patients underwent a computed tomography in the first 72 hours of admission to the center.

Variables

The following variables were evaluated:

- Demographical: age and sex.
- Clinical: Glasgow coma scale, etiologic diagnosis (ischemic: atherothrombotic cerebral, cardioembolic, lacunar, inhabitual, undetermined infarction; and hemorrhagic: intraparenchymal hemorrhage [IPH] and subarachnoid hemorrhage [SAH]), as well as the state upon discharge (alive or deceased).
- Tomographic: topographic diagnosis for the ischemic and hemorrhagic stroke.
- Electrocardiographic: flat or inverted T wave, ST-segment elevation or depression, presence of pathological Q wave, long QT interval (determined by the Bazett formula), and rhythm disorders (sinus tachycardia, premature atrial and ventricular contractions, atrial fibrillation, atrial flutter and new branch block).

Statistical analysis

All the variables included in the study were qualitative and were grouped in absolute numbers and percentage. There were held cross tabulations of qualitative variables, regarding the presence of electrocardiographic changes, by using the Chi square test. There was significant association among variables if $p < 0.05$. For the statistical analysis, the SPSS version 20.0 (SPSS Inc., Chicago, IL, USA) was used.

RESULTS

During the study period, a total of 166 patients with diagnosis of CVD were included, of which 54 showed new electrocardiographic changes, for a 32.5% of the total. Among the alterations found, the most frequent was sinus tachycardia in 46 patients (27.7%), followed in order of frequency by premature atrial contractions and flat or inverted T wave (**Table 1**). Other less frequent findings were the premature ventricular contractions, the ST-segment depression and atrial fibrillation, while the ST-segment elevation was found only in two patients.

Table 1. Distribution of the patients under study according to electrocardiographic findings (n=166).

Electrocardiographic findings	Nº	%
Morphological alterations		
Flat or inverted T wave	22	13.3
ST-segment depression	10	6.0
ST-segment elevation	4	2.4
Q wave presence	2	1.2
Long QT interval	12	6.0
Rhythm disorders		
Sinus tachycardia	46	27.7
Premature atrial contractions	22	13.3
Premature ventricular contractions	10	6.0
Atrial fibrillation	10	6.0
Atrial flutter	2	1.2
New bundle branch lock	0	0.0

The atherothrombotic cerebral infarction was the most frequent CVD, from the point of view of the etiological diagnosis, present in 78 patients (47.0%). The cardioembolic CVD followed in order of frequency with 15.7%, while the SAH was present in 14.5% of the patients (**Table 2**). The IPH and lacunar infarction were the least frequent. Regarding the etiological distribution according to electrocardiographic alterations, the group with changes in the electrocardiogram (ECG) was significantly more frequent in those who had a diagnosis of SAH (33.3% *vs.* 5.4%) and IPH (22.2% *vs.* 7.1%).

In **table 3A** can be observed that the ECG alterations were more frequent in patients with affected vertebrobasilar territory (71.4% *vs.* 43.7%), although the differences were not significant (p=0.179). According to the topographic diagnosis of IPH, the lobar location predominated, followed by the basal ganglia and the thalamus, in that order. In patients with IPH with no electrocardiographic changes, the 87.5% had lobar, whereas this was not present in any of the patients with new ECG changes, those in

which predominated the topography of basal ganglia and thalamus. These differences were significant (p=0.019) (**Table 3B**).

In relation to age, ECG alterations were present in a greater percentage in the group of 70 years and over (48.1% *vs.* 25.0%), although the differences were not significant (p=0.108). Likewise, in the group with electrocardiographic alterations, a slightly higher percentage of men were found compared to those who did not present new changes in the ECG (63.0% *vs.* 55.4%), although the differences were not significant (p=0.509) (**Table 4**). In this same table is shown that, in the group with electrocardiographic abnormalities, a significantly higher percentage of patients with a Glasgow scale less than 8 points was found compared to those without changes in the ECG (29.7% *vs.* 5.4%, p=0.0026).

Thirty patients died, representing 18.1% of the total patients included (**Table 5**). In the group with electrocardiographic alterations, a significantly higher percentage of deceased was found, compared to that which showed no further changes in the ECG (40.7% *vs.* 7.1%; p=0.0003) with a probability 7.5 (95% CI 2.13-26.79) times greater of in-hospital death for those patients with CVD, with new electrocardiographic alterations.

Table 2. Distribution of patients according to electrocardiographic alterations and etiological diagnosis.

Etiological diagnosis	Alterations in the ECG		Total (n=83)
	Yes (n=27)	No (n=56)	
Ischemic			
ACI	14 (26.0%)	64 (57.1%)	78 (47.0%)
Cardioembolic	10 (18.5%)	16 (14.3%)	26 (15.7%)
Lacunar	0 (0.0%)	18 (16.1%)	18 (10.8%)
Hemorrhagic			
IPH	12 (22.2%)	8 (7.1%)	20 (12.0%)
SAH	18 (33.3%)	6 (5.4%)	24 (14.5%)

p<0.001
Values are expressed in n (%)
ACI, atherothrombotic cerebral infarction; ECG, electrocardiogram; IPH, intraparenchymal hemorrhage; SAH, subarachnoid hemorrhage.

Table 3A. Distribution of patients according to electrocardiographic alterations and topographic diagnosis in the group with ischemic stroke.

Topographic diagnosis	Alterations in the ECG		Total (n=78)	p
	Yes (n=14)	No (n=64)		
Carotid	4 (28.6%)	36 (56.3%)	40 (51.3%)	0.179
Vertebrobasilar	10 (71.4%)	28 (43.7%)	38 (48.7%)	

Values are expressed in n (%)
ECG, electrocardiogram

Table 3B. Distribution of patients according to electrocardiographic alterations and topographic diagnosis in the group with intraparenchymal hemorrhage.

Topographic diagnosis	Alterations in the ECG		Total (n=20)	p
	Yes (n=12)	No (n=8)		
Lobar	0 (0.0 %)	7 (87.5 %)	7 (40.0 %)	0.019
Thalamus	4 (33.3 %)	0 (0.0 %)	4 (20.0 %)	
Basal ganglia	6 (50.0 %)	0 (0.0 %)	6 (30.0 %)	
Cerebellar	0 (0.0 %)	1 (12.5 %)	1 (0.0 %)	
Brainstem	2 (16.7 %)	0 (0.0 %)	2 (10.0 %)	

Values are expressed in n (%)
ECG, electrocardiogram

DISCUSSION

In the present study, electrocardiographic changes were detected in 32.5% of the patients included, less than what was recorded by van Bree[5], Hasegawa[6] and Hjalmarsson[7], and in Cuba by Sánchez[8] and Pérez[4]; in the first four studies it ranged between 70-90%, a difference that is probably due to the fact that patients with previous cardiovascular diseases or taking cardioactive medications were not excluded, as it was done in the present research.

However, in the study by Pérez *et al*[4], the same criteria than ours were adopted and, despite this, the prevalence of ECG changes remained somewhat higher. Similarly, Sánchez *et al*[3], in a recent study described, in 200 patients, a 58.5% with altered electrocardiographic changes, which may also be related to the evolution time during which the ECG were

Tabla 4. Distribution of patients according to electrocardiographic alterations and age.

Variables	Alterations in the ECG		Total (n=166)	p
	Yes (n=54)	No (n=112)		
Age (years)				
<50 years	1 (1.8%)	6 (5.4%)	7 (4.2%)	0.108
50-59 years	5 (9.3%)	24 (21.4%)	29 (17.5%)	
60-69 years	22 (40.7%)	54 (48.2%)	76 (45.8%)	
≥70 years	26 (48.1%)	28 (25.0%)	54 (32.5%)	
Sex				
Male	34 (63.0%)	62 (55.4%)	96 (57.8%)	0.509
Female	20 (37.0%)	50 (44.6%)	70 (42.2%)	
Glasgow coma scale				
3 to 7 points	16 (29.7%)	6 (5.4%)	22 (13.3%)	0.0026
8 to 11 points	20 (37.0%)	36 (32.1%)	56 (33.7%)	
12 to 15 points	18 (33.3%)	70 (62.5%)	88 (53.0%)	

Values are expressed in n (%)
ECG, electrocardiogram

Tabla 5. Distribución de los pacientes según alteraciones electrocardiográficas y estado al egreso.

State at discharge	Alterations in the ECG		Total (n=166)	p
	Yes (n=54)	No (n=112)		
Alive	32 (59.3%)	104 (92.9%)	136 (81.9%)	0.0003
Deceased	22 (40.7%)	8 (7.1%)	30 (18.1%)	

Values are expressed in n (%)
ECG, electrocardiogram

continued, that in our study was only in the first 72 hours.

In relation to the type of electrocardiographic alterations, in the study by Kumar *et al*[9] the main findings were rhythm disorders, followed by long QT interval, inversion of the T waves, sinus tachycardia and, in a similar percentage to our study, ST-segment depression. On the other hand, in patients with IPH[5] the main findings were the long QT interval (36%), followed by changes in the ST-T-segment (23%), sinus bradycardia and T-wave inversion in 16%, and sinus tachycardia in 13%.

Moreover, Somasundaran *et al*[10] reported a percentage quite similar to our study, of patients with T wave inversion (20.6%), as well as ST-segment depression (15.5%), although the percentage of sinus tachycardia was lower (6.3%).

In the study by Sánchez *et al*[3] this type of tachycardia was the most frequent ECG alteration, followed by premature contractions and flat or inverted T wave, which coincides with our results. In 25.2% were also identified with ST elevation and 19.0% with long QT interval.

In relation to the etiological diagnosis, in our study a greater probability of alterations in the ECG was verified for patients with hemorrhagic CVD. Somasundaran *et al*[10] reported, more frequently, T wave inversion in patients with hemorrhagic stroke compared to ischemic (27.5% *vs.* 15.5%). Similarly, Saxena *et al*[11] found ST-T changes more frequently in those with hemorrhagic stroke (54.2% *vs.* 45.8%). And in 60 patients with SAH[12], the main finding was the inversion of T waves (35.0%), although the ST-segment depression and long QT interval were less frequent.

The carotid vascular territory irrigates the lobe of the insula (5th lobe), center top rector of the cardiovascular function, that, when damaged, its repercussion on the brain-heart axis is well-noticed. However, it should be noted that in the vertebrobasilar territory, the blood flow is turbulent and irrigates primary control structures of the cardiac function, hence, after its affectation, also some complications were registered[3]. In relation to the topography of patients with ischemic stroke, alterations in the ECG of patients with vertebrobasilar territory involvement were found more frequently in this study.

Regarding the topography of patients with IPH in a study conducted in Spain[13] with 380 patients with a first spontaneous IPH, it was found a predominance of lobar location (38.4%), followed by the location at the level of the basal ganglia (21.3%) and, less frequently, at the level of the thalamus (12.4%), cerebellum (7.4%) and brainstem (5.0%).

In a research conducted in Cuba, in Cienfuegos,

Vergara *et al*[14] also found a predominance of the lobar location (38.9%), followed by the thalamic (30.5%), while the basal ganglia, cerebellum and stem were the least frequent, in that order.

Regarding age, Sánchez *et al*[3] found a proportional increase in the percentage of patients with electrocardiographic alterations with increasing age. In a population study conducted Prineas *et al*[15] the percentage of individuals with electrocardiographic alterations was significantly higher in those older than 65 years, and slightly higher in men than in women, although these showed, more frequently, a long QT interval. Similar to our results, Saxena *et al*[11] found no age or gender differences in terms of a higher prevalence of electrocardiographic alterations in patients with stroke.

Regarding the Glasgow coma scale, a relationship between lower scores and the greater presence of electrocardiographic alterations was also identified in our study. This aspect is important if one considers that in the research by Suárez *et al*[16] the Glasgow coma scale was the variable with the highest predictive power of mortality, with a probability of death 10.74 times higher for patients with a score ≤10.

Regarding the association of electrocardiographic alterations with mortality, Kumar *et al*[17], in 321 patients with SAH, found a probability of in-hospital death 5.74 times higher in those with ST-segment depression; 4.44 times greater with sinus bradycardia; 3.56 times greater with long QT interval, and 3.06 times greater with ST-segment elevation.

In the study by Van Bree *et al*[5], patients with long QT interval showed a probability 10.8 times of blood in the intraventricular space; 3.3 times greater displacement of the midline and 2.3 times greater than having a bruise with volume > 30 ml, variables that are directly related to a worse prognosis.

In a research where 834 patients with SAH[18] were studied, those who had ST-segment depression showed a 3.0 times greater probability of in-hospital death and those with long QT had a 1.5 had a greater probability of neurogenic pulmonary edema. In addition, nonspecific repolarization disorders showed a 2.7 times greater probability of this type of edema and 2.2 times greater of in-hospital death.

Other studies report a similar outcome[19-21]. These evidences support the fact that the presence of electrocardiographic alterations, mainly the changes of the ST-T and the long QT interval in patients with stroke, identify a subgroup of individuals with an increased risk of complications and in-hospital death.

CONCLUSIONS

The presence of new electrocardiographic changes in patients with cerebrovascular diseases is related to a smaller Glasgow scale, with the etiological diagnosis of the disease, mainly the SAH, and a greater probability of in-hospital death. Using routinely the interpretation of the electrocardiogram in the first 72 hours of presenting a cerebrovascular disease, given its relationship with a worse prognosis in these patients, will allow a more complete risk stratification and prevent future complications.

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