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ELECTROCARDIOGRAPHIC FINDINGS IN ACUTE CEREBROVASCULAR HEMORRHAGE

A PROSPECTIVE STUDY OF 70 PATIENTS

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SUMMARY — Seventy patients with hemorrhagic stroke were prospectively evaluated regarding the electrocardiographic abnormalities observed within the first 48 hours of the ictus. Group I comprised 55 patients with spontaneous cerebral hemorrhage, and group II 15 patients with subarachnoid hemorrhage. Patients taking cardiac drugs (beta blockers, calcium-channel blockers, inotropic drugs) or with severe metabolic/electrolyte disturbances were excluded. The most common ECG abnormality was a prolonged Q-Tc interval: group I, 37 (67.2%); group II, 8 (53.3%). Only 4 (7.2% patients of group I and no patient of group II had a normal ECG. No relation was found between the site of the intracerebral hematoma and the occurrence of any particular ECG change. A prolonged Q-Tc may be related to the development of severe cardiac arrhythmias observed in some patients with acute cerebral hemorrhage.

KEY WORDS: cerebral hemorrhage, electrocardiogram, Q-Tc prolongation.

Achados eletrocardiográficos em hemorragia cerebrovascular aguda: estudo prospectivo de 70 casos.

RESUMO — Foram analisados os achados eletrocardiográficos obtidos dentro das primeiras 48 horas de instalação de acidente vascular cerebral hemorrágico em 70 pacientes. O grupo I foi composto de 55 pacientes com hematoma intracerebral espontâneo e o grupo II, de 15 pacientes com hemorragia subaracnóidea. Pacientes em uso de drogas cardíacas (beta bloqueadores, bloqueadores de cálcio, drogas inotrópicas) e/ou distúrbios metabólico/eletrolíticos graves foram excluídos. A alteração eletrocardiográfica mais comum em ambos os grupos foi o prolongamento do intervalo Q-Tc: grupo I, 37 (67,2%); grupo II, 8 (53,3%). Os eletrocardiogramas foram normais em 4 (7,2%) pacientes do grupo I e em nenhum paciente do grupo II. Não houve correlação significativa entre o local do hematoma cerebral e a ocorrência de alguma alteração específica do eletrocardiograma. O prolongamento do intervalo Q-Tc pode representar fator de risco potencial para o desenvolvimento súbito de arritmias cardíacas graves, observado em alguns pacientes com acidentes vasculares cerebrais hemorrágicos.

PALAVAS-CHAVE: hemorragia cerebral, eletrocardiograma, prolongamento do intervalo Q-Tc.

In 1938, Aschenbrenner and Bodechtel [3] stated that intracranial lesions may be responsible for electrocardiographic abnormalities, but the first report of ECG changes in patients with cerebrovascular accidents was given by Byer, Ashman and Toth, in 1947 [6]. In 1954, Burch, Meyers and Abildskov [5] described a pattern of QT prolongation, abnormal T waves, and U waves which they considered distinctive of acute stroke. Since then, several reports about the prevalence and pathophysiology of ECG findings in different cerebrovascular events has accumulated [4,5,7,9,11,12,14-16,19,22,25]. With the exception of few works [9,16,18,19], those studies

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were undertaken prior to the advent of brain CT scan and have employed different clinical and laboratory criteria for the classification of cerebrovascular diseases. There seems to be no prospective and systematic study involving a large number of patients with spontaneous cerebral hemorrhage with the use of computed tomography scanning[17].

The objective of this study is to describe the prevalence of ECG abnormalities in patients with intracerebral hemorrhage, and to compare these data with a group of patients with subarachnoid hemorrhage.

PATIENTS AND METHODS

A total of 70 patients were prospectively evaluated from January 1986 to August 1990. They were divided in two groups: Group 1, patients with nontraumatic intracerebral hemorrhage (ICH) (n=55); and Group II, patients with subarachnoid hemorrhage (SAH) (n=15). Only patients whose brain CT-scans, electrocardiograms (ECG), chest X-ray, serum biochemical/electrolyte profiles and blood gas analysis were obtained within 48 hours of onset of the cerebrovascular event were included. Patients taking beta-blockers, calcium channel blockers, inotropic drugs (dopamine, amrinone), and antiarrhythmic drugs were excluded. Other criteria of exclusion were shock or hypotension, abnormal electrolyte/biochemical serum levels (K, Ca, glucose, creatinine), hypoxemia (p02 less than 60mmHg). Blood pressure was measured and recorded at the time of the ECG recording in each patient. No patient was taking anticoagulants or antiaggregant drugs. Patients with valve heart disease were excluded.

ECGs were evaluated on the basis of all current criteria[8] by one of the authors (FSL), who was not aware of the diagnosis of the patients. Corrected Q-T intervals (Q-Tc) were calculated, by Bazett's formula (Q-Tc=Q-T/ /R-R'), and the normal limits were 0.420 for male patients and 0.430 for female patients. Regarding the site, the intracerebral hematomas were grouped by location as follows: deep cerebral (basal ganglia and/or thalamus), superficial cerebral (lobar), deep and superficial cerebral, and posterior fossa (brainstem or cerebellum). All patients of both groups, with the exception of one patient with SAH, underwent cerebral angiography. Hunt and Hess scale was applied to the SAH patients and recorded at the time of the ECG recording. The Group I patients were subdivided into patients with and without frontal lobe extension, and their ECG findings were compared. We also evaluated the possible correlation of some specific ECG changes (e.g., P wave changes[22]), with the presence or not of intraventricular bleeding. Neurogenic T waves were defined as T waves of large amplitude and duration, as originally described by Byer et al.[6]. Chi square test was used for random samples greater than 10, and Fisher's exact test for smaller samples. The significance level was defined as 5%.

RESULTS

The age, sex distribution, and the sites of the cerebral hemorrhage of the Group I (ICH) patients are shown in Table 1. Of the 55 patients, only 10 had a previous history of arterial hypertension. The age, sex distribution, and location of the cerebral aneurysm of the Group II (SAH) patients are shown in Table 2. Regarding Hunt and Hess scale, the patients were classified as follows: grade 1 five, grade 2 four, grade 3 four, grade 4 one, and grade 5 one. In one patient of this group, cerebral angiography was not performed due to rapid clinical deterioration and death. Three other patients had a negative angiogram. The mean age of both groups did not differ significantly (two-tailed t Student test). A higher proportion of hypertensive patients in Group I is evident ($\chi^2 = 10.999$, $p < 0.01$). In 51 patients of group I, the side of the lesion was determined: 31 were right-sided and 20 left-sided. No significant differences of the ECG changes were observed between right-sided and left-sided lesions.

ECG abnormalities observed in both groups are depicted in Table 3. The two groups did not differ significantly regarding the prevalence of any ECG change. In Group I, a significant higher proportion of patients with increased Q-Tc interval has concomitant sinus tachycardia ($\chi^2 = 11.475$, $p < 0.01$). This observation was not present in Group II (Fisher's exact test, $p = 0.4308$). Of the 20 ICH patients with sinus tachycardia, 19 (95%) had an increased Q-Tc interval, while 17 (50%) patients without tachycardia had an increased Q-Tc interval. Furthermore, the frequency of Q-Tc prolongation was strongly related to high blood pressure ($\chi^2 = 5.0850$, $p < 0.05$). Among the patients with prolonged Q-Tc (n=37), 31 (83.8%) had arterial

Table 1. Group I, spontaneous intracerebral hemorrhage.

Number of patients	55
Sex (M/F)	38/17
Sex ratio	2.2 : 1.0*
Mean age (years)	51.4 ± 13.8
Age range (years)	16 - 82
Hypertension (%)	43 (78.2)
Hematoma location (%)	
1) Hemispheral	27 (49)
2) Basal ganglia/thalamus	18 (32.7)
3) 1+2	6 (10.9)
4) Posterior fossa	4 (7.2)

* $p<0.05$.

Table 2. Group II, subarachnoid hemorrhage.

Number of patients	15
Sex (M/F)	3/12
Sex ratio	1 : 4 *
Mean age (years)	51.9 ± 11.3
Age range	29 - 65
Hypertension (%)	5 (33.3)
Aneurysm location	
Anterior communicating artery	6
Internal carotid artery	4
Middle cerebral artery	1
Unknown	4

* $p<0.05$.

hypertension, while 6 (50%) patients with normal Q-Tc were hypertensive. Thirty-one (72%) hypertensive patients had prolonged Q-Tc, while only 6 (50%) normotensive patients had an abnormal Q-Tc.

There was no correlation between the site of the cerebral hemorrhage and ECG abnormality. Intraventricular bleeding occurred in 21 patients, and its presence was not related to any particular ECG change. Of four group I patients with normal ECG, two presented intraventricular bleeding. There was no significant correlation between the frequency of ECG ischemic changes with high blood pressure. The combination of prolonged Q-Tc and U waves was found in only 2 (3.6%) patients of Group I, and in no patients of Group II. The only patient of the SAH group with U waves had a normal Q-Tc interval. In the 5 Group I patients with neurogenic T waves, no specific correlation with the side or site of the hemorrhage, as well as with the presence or not of intraventricular bleeding, was aparrent. Only one patient of the SAH developed neurogenic pulmonary edema 2.

Table 3. Electrocardiographic findings (%).

	Group I	Group II
Prolonged Q-Tc	37 (67.2)	8 (53.3)
Sinus tachycardia	20 (36.3)	4 (26.6)
Ischemic T and S-T changes	19 (34.5)	3 (20.0)
Left ventricular hypertrophy	12 (21.8)	1 (6.6)
Nonspecific T and S-T changes	11 (20.0)	2 (13.3)
U wave	7 (12.7)	1 (6.6)
Neurogenic T wave	6 (10.9)	4 (26.6)
Sinus bradycardia	5 (9.0)	2 (13.3)
Electrical inactive area	5 (9.0)	1 (6.6)
Right atrial hypertrophy	4 (7.2)	1 (6.6)
Left atrial hypertrophy	3 (5.4)	—
Premature atrial complexes	3 (5.4)	—
Left anterior fascicular block	3 (5.4)	—
Atrial fibrillation	1 (1.8)	—
First degree atrioventricular block	1 (1.8)	—
Junctional rythm	1 (1.8)	—
Normal	4 (7.2)	—

COMMENTS

Repolarization changes were the most common findings in both groups. The observation of prolonged Q-Tc in 67.2% of the ICH patients and in 53.3% of the SAH patients is similar to some previous studies of patients with ICH[9,15,18] and SAH[15,18,21]. Yamour et al.[25] found this abnormality in only 10% of 50 cases of spontaneous cerebral hemorrhage. However, these authors only included patients without previous hypertension and whose earlier ECG were normal. Moreover, they found a higher incidence of ECG abnormalities in hemorrhages located in the frontal lobes. This observation was not confirmed neither by other authors [9,15] nor by our study. In fact, no correlation seems to exist between the site or side of the cerebral lesion and any particular ECG change. One may expect that almost any acute expanding lesion within the central nervous system may lead to an autonomic deregulation, as both sympathetic and parasympathetic neurons receive extensive connections from several other central neurons which themselves receive afferents from peripheral mechanoreceptors and chemoreceptors and/or other central nuclei[23].

Stolar et al.[22] suggest a possible relationship between the presence of intraventricular bleeding and the occurrence of tall P waves (greater than 2.5mm) in leads II III and AvF. This relationship was not observed in our group of ICH patients.

The observation of left ventricular hyperthophy (LVH), bundle branch blocks, and pathologic Q waves may not be considered new findigs in all patients with ICH [9]. Some of those changes may reflect long-term effects of chronic arterial hypertension. We observed a higher prevalence of LVH in the ICH group (21.8%) in comparison with the SAH group (6.6%), probably reflecting a higher prevalence of systemic hypertension in the former group. Nevertheless, it should be kept in mind that ECG changes simulating myocardial infarction may occasionally occur in patients with intracranial hemorrhage and subarachnoid hemorraghe [2,11].

The mechanism of ECG abnormalities in patients with cerebral hemorrhage remains obscure. Raised cathecolamine levels in patients with ischemic and hemorrhagic strokes leading to myocardial injury have been reported by some authors [10,16]. Any acute cerebrovascular event with raised intracranial pressure may cause an acute increase of cathecolamine levels in the heart via the autonomic nervous system, with subsequent myocardial injury [25].

Neural mechanisms are importantly involved in the regulation of ventricular repolarization. Different branches of the cardiac sympathetic nerves supply different areas of the ventricular myocardium [1]. Imbalances in the activity of these various branches produce uneven cardiac depolarization and repolarization, which may result in Q-T interval prolongation and the appearance of irritable foci, reentrant circuits and ventricular ectopies [1,19]. It is well established that nonuniform repolarization properties enhance vulnerability to fibrillation and other reentrant arrhythmias [1]. The parasympathetic role in ventricular repolarization is much less important than sympathetic effecs [1,24], but some authors have observed a higher frequency of ventricular arrhythmias in SAH patients with previous vagally-induced arrhythmias [21]. The use of amine blockers (e.g. beta-blockers) seems to be effective in treating ventricular arrhythmias and preventing subendocardial lesions in SAH patients [15], however, its use in patients with non-traumatic intracerebral hemorrhage remais unsettled.

A prolonged Q-T interval is frequenty observed in patients with SAH who develop serious ventricular arrhythmias [15,20,25]. It is conceivable that patients with ICH and prolonged Q-Tc have a higher risk to develop potentially fatal ventricular arrhythmias. Sen et al.[20] described 2 patients out of 72 cases of intracranial hemorrhage who had marked Q-Tc prolongation and developed torsade de pointes and fatal ventricular fibrillation.

In conclusion, patients with ICH and SAH have a high prevalence of ECG changes in the first 48 hours of the cerebral event. Prolongation of Q-Tc interval is the most common abnormality and may indicate a higher risk to develop potentially fatal cardiac arrhythmias in this group of patients.

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