Cardiac injury in traumatic subarachnoid hemorrhagea: Prospective study in 35 patients

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Atteinte cardiaque au cours de l'hémorragie sous archnoidienne post traumatique : Etude propective de 35 cas

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RÉSUMÉ

Prérequis : L'association d'anomalies électrocardiographiques (ECG), en particulier les troubles de la repolarisation, à une hémorragie sous arachnoidienne post traumatique (HSA) est décrite depuis plus de 50 ans. Très peu d'études se sont intéressées à ces anomalies cardiaques au cours de l'HSA post traumatique (HSA-t).
But : Relever l'incidence des complications coronariennes au cours de l'HSA-t et étudier la valeur prédictive de mortalité des anomalies ECG au cours de l'HSA-t
Méthodes : Cette étude prospective menée entre 2001 et 2009 au service de réanimation de l'institut national de Neurologie de Tunis concerne 35 patients consécutifs traumatisés crâniens avec HSA-t à l'admission. Un électrocardiogramme a été pratiqué quotidiennement pendant les six premiers jours ainsi qu'un dosage de la Troponine Ic au 1er jour post traumatisme puis au 3ème et au 5ème jour. L'analyse statistique était basée sur le test de variance non paramétrique de Kruskal-Wallis pour comparer les moyennes; et les tests chi 2 et Fisher pour comparer les pourcentages, avec un seuil de significativité 0.05. Des liaisons entre 2 variables quantitatives ont été étudiées par le coefficient de corrélation de Pearson. Nous avons calculé l'Odds ratio pour l'étude de la mortalité.
Résultats : L'âge des patients est de 39 ± 17 ans (14-70). Le sex ratio est égal à 4 en faveur des hommes. On constate des anomalies de la repolarisation à type de modifications de l'onde T et du segment ST chez 20 patients (57%). Les anomalies de l'onde T sont les plus fréquentes et observées chez 13 patients. La majorité de ces anomalies surviennent au 3ème jour. La Troponine Ic est élevée chez 12 patients soit 34%. Le maximum d'élévation a eu lieu au 3ème jour. L'élévation de la Troponine Ic est associée à un mauvais grade scannographique de FISHER (p=0.01) et aux anomalies de l'onde T(p=0.002). L'élévation de la Troponine Ic et les anomalies de l'onde T ressortent comme facteurs indépendants de mortalité d'après une analyse bi variée.
Conclusion : Nous déduisons que l'incidence des modifications ECG n'est pas rare au cours de l'HSA-t. Les troubles de la repolarisation et l'élévation de la la troponine IC constituent des facteurs prédictifs indépendants de mortalité au cours de l'HSA-t.

Mots-clés
HSA-t, Troponines Ic, ECG, mortalité

SUMMARY

Background : Various electrocardiographic abnormalities have been noted since 1954 in patients with head trauma complicated by subarachnoid hemorrhage (SAH). However, very few studies have interested to these ECG modifications in the case of post traumatic SAH (t-SAH)
Aim: To assess the incidence of ECG abnormalities during the first five days after admission and the predictive value of these cardiac complications on the mortality in t-SAH.
Methods: This prospective study included 35 patients out of 125 with traumatic SAH diagnosed in the emergency unit in Rabta's hospital (2001-2009). Patients with cardio vascular history, thoracic trauma, non neurological coma and vascular-related neurological coma were excluded. An electrocardiogram monitoring was performed. A brain CT scan was performed in admission, 48 h after and case of neurological aggravation. Serum cardiac troponin IC levels were determined on hospital admission and then on the third and fifth days of hospitalization. The statistical analysis was based on the non-parametric variance test of Kruskal-Wallis to compare the means; on the chi 2 and Fisher tests to compare percentage, with a significant result at 0.05 percentile and on the Odds ratio non-parametric factors for death. Association between 2 quantitative variables have been analyzed by Pearson coefficient of correlation.
Results: Mean age of the 35 patients was 39 ± 17 years. Sex ratio was 4 in favor of men. The prevalence of electrocardiographic changes was of 57% (20 patients). Serum Troponin I level showed a peak on the 3rd day then it decreased. The majority of electrical abnormalities occurred during the third after admission and are associated to a markedly increased Troponin I plasma level and to the highest rate of mortality. Statistical analysis showed a significant correlation between T wave changes and the increase of serum Tn IC level (p= 0; 0002). The relative risk of mortality was higher than 7.2 times in cases with increase serum TnIc level.
Conclusion: We demonstrated that ECG changes were common in patients with t SAH and the major predictive factors of mortality were the increase of serum TnIC and T wave changes.

Key-words
HAS –t, Troponin Ic, ECG, Mortality.

Subarachnoid hemorrhage (SAH) is a complication of head trauma inducing frequently cerebral vasospasm and even cerebral infarct. Various electrocardiographic abnormalities have been noted in patients with head trauma complicated by subarachnoid hemorrhage (1-5). They are considered to be secondary to the massive catecholamine discharge in systemic circulation (6, -9). Serum cardiac troponin I (TnI) was considered a highly sensitive and specific marker of myocardial cell lesion and might be regularly performed in these patients to detect early myocardial ischemia. We carried out a prospective study in 35 patients with traumatic SAH (tSAH) in order to assess the incidence of coronary complications during the first five days after admission and to demonstrate the interest of troponin le blood assay in the diagnosis of coronary abnormalities.

PATIENTS AND METHODS

Patients:

Our prospective study included 35 patients out of 125 patients with tSAH diagnosed in the emergency unit in the Rabat's Hospital over a period of 15 months : from January 2008 to March 2009. Patients with any cause of cardiovascular injury were excluded: history of cardiovascular injury; thoracic trauma and vascular-related neurological coma. The selected patients are hospitalized in the intensive care and anesthesia department of the National Institute of Neurology.

Methods:

The following scoring Systems were used: Acute Physiology and Chronic Health Evaluation II (APACHE II), Injury Severity Score (ISS), the scanographic grade of Fisher and the Glasgow Coma Scale (GCS) after correction of hemodynamic and respirator troubles.

A continuous electrocardioscope and a daily electrocardiogram were performed. Brain CT scan was performed on admission, 48 hours after hospitalization and case of neurological aggravation. All patients had tracheal intubation and mechanical ventilation with sedative drugs (midazolam and fentanyl) in order to avoid secondary brain insults and to optimize cerebral oxygenation by maintaining mean arterial pressure >90 mmHg, Pa02= 100 mmHg, PaC02 = 30-35 mmHg, a normal osmolarity, and avoiding hyperthermia, hyperglycemia and hypoglycemia..Serum cardiac troponin I, creatine kinase (CK) and its MB fraction (CK-MB) levels were determined on hospital admission and then on the third and the fifth days of hospitalization. This strategy was due to economic reasons and mainly because all blood samples were analyzed in a laboratory out of our hospital.

The statistical analysis was based on the non-parametric variance test of Kruskal-Wallis to compare the means; on the chi 2 and Fisher tests to compare percentage, with a significant result at 0.05 percentile and on the Odds ratio non-parametric factors for death. Association between two quantitative variables have been analyzed by Pearson coefficient of correlation.

RESULTS

The mean age of the 35 patients was 39 ± 17 years. Five patients were treated for hypertension, 3 patients for diabetes mellitus, 2 patients had a chronic obstructive pneumopathy, and one patient had liver cirrhosis. Twenty-three patients had an isolated cranial trauma; the others had associated trauma such as facial trauma (4 patients), limb trauma (7 patients) and cervical trauma (1 patient). The mean delay to admission in the intensive care unit was 14 ± 4 hours. Fourteen patients required surgery for different indications: long bone fracture (8 patients), extra-dural hematoma (4 patients), compressive sub-dural hematoma (1 patient) and cerebral contusion (1 patient).

The means ISS, APACHE II, and Glasgow scores were respectively 27 ± 14, 12 ± 6 and 6 ± 3. Thirty patients had a GCS at admission < 8. Twenty patients developed cardiac arythmia during the first five-day : 8 with tachycardia, 4 with bradycardia, 3 with premature ventricular complexes, 2 with premature atrial complexes and 3 with left or right bundle branch blocks. Seventeen patients developed cardiac ischemia with ST-segment elevation (1 patient), ST-segment depression (3 patients), and T-wave changes (20 patients). The majority of electrocardiographic abnormalities appeared on the third day and their mean duration was 4 ± 1 days. Six patients died before the fifth day after admission. Three of them presented ST-segment depression or T-wave changes.

Blood Tn Ic level was increased in 12 patients. CK-MB was elevated in 23 patients and CPK was increased in 31 patients. Four patients died before Tn Ic blood level was measured on the third day. Three of them had a normal TnIc blood level on the first day and the other one had TnIc increased at that time. Two patients died before TnIc was determined on the fifth day: one of them had an increased TnIc level on the first day then it get normal on the 3rd day, the second patient had a normal TnIc level on the first day then an increased level on the following days. Serum Tn Ic level showed a peak on the 3rd day then it decreased. Both groups of patients with and without increased serum TnIc level were comparable concerning mean ISS (respectively 26 vs 26) and mean APACHE II scores (respectively 12 ± 8 vs 12 ± 7).

There was no correlation between the increase of troponin I level and the severity of initial neurological state in patients assessed by the Glasgow score but this increase was correlated to the scanographic grade of Fisher with a p value of 0.01.

A significant correlation was found between T-wave changes and the increase of serum TnIc level (p= 0.002), but no correlation was observed between the latter and ST-segment shifts (p=0.5).

In this study, five factors were found as predictive of mortality in the univariate Statistical analysis: troponin, T-wave changes, ISS, the scanographic grade of Fisher at the 48th hour of hospitalization and the Glasgow Coma Scale (Table I).

Patients with increased troponin had a likelihood rate of death of 66.7%, while the latter is of 21% in patients with normal range of Tn Ic. Relative risk of death was 7.2 higher in the case

of elevation of troponinIc (p= 0.02) and 4.8 higher in patients with T-wave changes (p= 0.03).

Table 1 : Predictive factors of early mortality

	Probability of death if factor present	Probability of death if factor absent	OR (odds ratio)	p
Troponine IC	66.7	21.7	7.2	0.02
T wave	56.3	21.1	4.8	0.03
ISS	100	26.7	No definite	0.03
Fisher 48	54.5	7.7	14.4	0.009
Glasgow	43	0	No definite	0.1

DISCUSSION

According to the current study, the prevalence of electrocardiographic abnormalities during SAH-t was 57%. It seems to be a common complication seen in case of SAH-t. Electrocardiographic abnormalities during SAH have been previously reported by Burch's in 1954 (1) who frequently found T-wave inversion and QT segment enlargement in patients with SAH due to aneurysm rupture. Despite the large number of studies interested in myocardial lesions after SAH secondary to aneurysm rupture (2-6), few clinical studies focused on this specific affection in traumatic SAH.

Physiopathological mechanisms responsible of these electrocardiographic abnormalities remain far from being well-known. Many hypotheses were discussed: coronary organic lesions, massive catecholamine discharge, tachycardia and hypertension secondary to cerebral hemorrhage and coronary vasospasm. The catecholamine discharge hypothesis is the most supported one (7, 8, 9).The incidence of electrocardiographic changes associated to SAH varied in literature. It was of 98% in two prospective studies: The first was carried out by Mayer and al (10) whose study included 57 patients daily monitored by a 12 leads ECG during the first three days after admission. The second was carried out by Browers and al (3) who studied 61 patients with traumatic SAH daily monitored by a 12 leads ECG throughout the 12 first days. They found that the majority of electrocardiographic changes occurred during the first three days. In our study, all patients had at least one ECG during the first five days of hospitalization. The incidence of electrocardiographic changes was about 57 % and most of them appeared during the third day. Repolarization troubles, especially the T-wave inversion were reported to be the most common abnormalities (1, 10-13). These electrical abnormalities were frequently associated to myocardial lesions and to increased serum TnIc level which is specific to the myocardium and may confirm the diagnosis of myocardial lesion especially in trauma patients with lesions of skeletal muscles (6, 14). Elevated levels of troponin have been reported in patients with acute ischemic stroke. Parekh and al (15) had daily measured serum TnIc level in 39 patients with subarachnoid hemorrhage during seven consecutive days. They found that the peak of serum TnIc level occurred on day 1 for 6 patients and on day 2 for one patient. The others had normal range of troponin during the 7 days of the study. In the current study, serum Tn Ic level was quantitatively analyzed on day 1, day 3 and day 5 in all our patients. Both peaks of serum TnIc level and repolarization abnormalities were noted during the third day.

Masaki et al. (16) prospectively evaluated one hundred three patients with SAH in order to determine the relations of TnI to clinical severity, systolic and diastolic cardiac function, pulmonary congestion, and length of intensive care unit stay. Highly positive cTnI wit SAH was associated with clinical neurologic severity, systolic and diastolic cardiac dysfunction, pulmonary congestion, and longer intensive care unit stay. Even mild increases in cTnI were associated with diastolic dysfunction and pulmonary congestion.

The severity of neurological status was found to be correlated to a higher incidence of myocardial troubles as diagnosed by biological parameters or echocardiograms (4, 13, 15, 16). We found in the current study that the increase of serum TnIc level was independent from physiologic scores and Glasgow coma scale, but it has a statistical significant correlation with the Fisher scanographic scale at the 48th hour as it was reported by Parekh and al (15).

CONCLUSION

Our study demonstrates first of all that electrocardiographic changes in patients with traumatic subarachnoid hemorrhage were common. Secondly, both peaks of blood serum TnIc level and main repolarization abnormalities were detected on the third day and finally that the major predictive factors of mortality were the increase of serum TnIc level and T-wave changes, suggesting that daily assaying of this myocardial enzyme must be systematically performed in all patients with tSAH during at least the seven first days.

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