Aneurysmal subarachnoid hemorrhage with electrocardiographic abnormalities: A case report

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Abstract

Background: The interaction between cardiovascular system and cerebral damage in patients with subarachnoid hemorrhage (SAH) have been described frequently. Electrocardiographic (ECG) alterations occurring during the course of SAH have been previously reported.

Case report: we report a case of woman who had electrocardiographic abnormalities. Computer tomography (CT) angiography revealed subarachnoid hemorrhage (SAH) with cerebral artery aneurysm. ST-elevation regressed later after intubation.

Conclusion: This case illustrates the importance of neurological injury as one of the differential diagnoses while managing ECG changes in cardiovascular disease.

Key-words: *Aneurysm, Electrocardiography, Subarachnoid hemorrhage*

INTRODUCTION

Subarachnoid hemorrhage (SAH) is a relatively rare pathology, it mainly affects healthy young adults. More than 50% of patients hospitalized for SAH are under 50 years old and are mostly women (about 60%). SAH is responsible for a mortality rate of around 50%, of which 10% before arrival at the hospital and 25% in the first 24 hours. Among the survivors, 60% have more or less disabling sequelae [1, 2].

SAH neurological complications are, there,

rebleeding, vasospasm and delayed cerebral ischemia, hydrocephalus, increased intracranial pressure and seizures. However, there are non-neurological complications that can worsen the prognosis for these patients. These extra neurological complications are mainly, respiratory, metabolic and cardiovascular such as electrocardiographic changes, left ventricular dysfunction, and troponin elevations mimicking myocardial ischemia can also occur [3].

CASE PRESENTATION

A 58-year-old woman was referred by the emergency medical service team to the emergency department for a witnessed loss of consciousness on the beach. The patient was with a medical history of controlled hypertension. No illicit or recreational drug use was known. When we arrived at the scene, a physical examination revealed a reduced level of consciousness (Glasgow Coma Scale 7/15) and, high blood pressure of 220/100. Heart sounds were regular with no murmurs and the lungs were clear to auscultation bilaterally. pupils were dilated with minimal pupillary response. The electrocardiogram (ECG) showed normal sinus rhythm, with a heart rate of 140 bpm and extended ST-elevation in the anterior territory with systematized ventricular bigeminy (Figure 1).

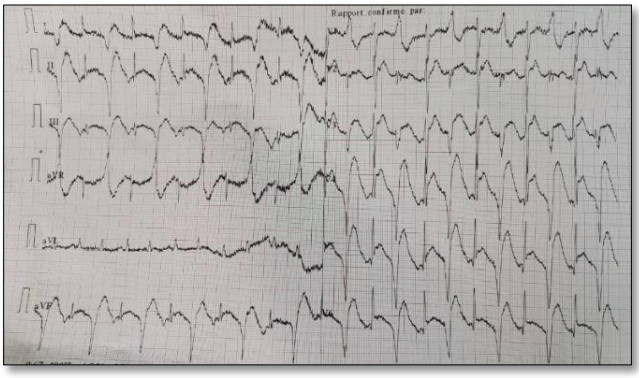

Figure 1: Electrocardiogram showing normal sinus rhythm, with extended ST-elevation in the anterior territory and ventricular bigeminy.

The patient was transported to the emergency department (ED) after having been stabilized (he was intubated, ventilated, and sedated on scene).

After intubation, ECG showed normal sinus rhythm, with 100bpm heart rate with extended ST-elevation in the anterior territory, however, there was ventricular extrasystoles disappeared without any administered antiarrhythmic medication (Figure 2).

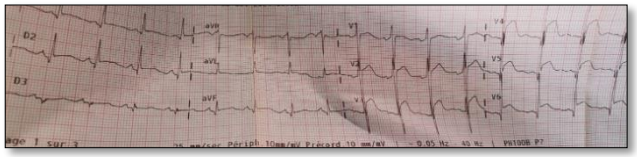

Figure 2: Electrocardiogram showing normal sinus rhythm, with 100bpm heart rate and extended ST-elevation in the anterior territory.

In the ED, Laboratory exams showed increased levels of high sensitive cardiac troponin T-sampled 2 times in an interval of 4 hours (526ng/l then 1200ng/l). Acute coronary angiography was performed, which revealed normal coronary arteries. For further diagnostic workup, cerebral computed tomography with angiography was performed showing subarachnoid hemorrhage (SAH) with cerebral artery aneurysm (Figure 3).

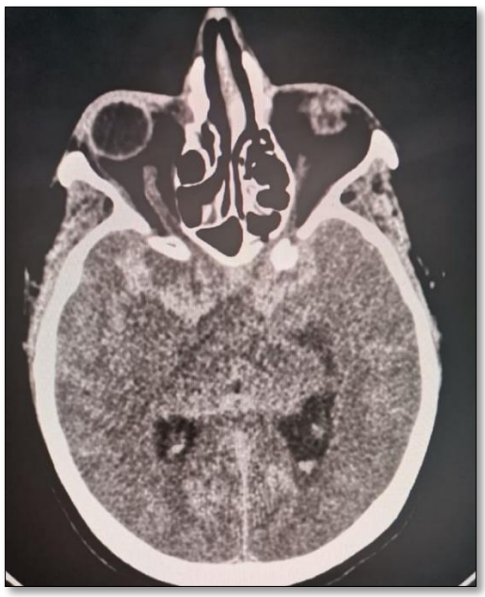

Figure 3: Cerebral computed tomography with angiography showing SAH with cerebral artery aneurysm.

Due to severe subarachnoid hemorrhage and loss of brainstem reflexes, no additional intervention was recommended by neurosurgery. For further care, the patient was transferred to the intensive care unit. On follow-up, the patient presented Staphylococcus aureus ventilator-associated pneumonia with hemodynamic instability but quickly resolved by optimizing the hemodynamic status with secondarily adapted empirical antibiotic therapy.

Neurologically, without any sedation, the patient kept a minimal state of consciousness (an outline of opening and closing of the eyes but very invariable with facial expressions) with tetraplegia.

The patient has been discharged tracheostomized under ventilation at home.

DISCUSSION

The interaction between the cardiovascular system and cerebral damage could explain the existence of arrhythmias, or even cardiac arrest during the occurrence of significant psychological stress. Electrocardiographic (ECG) alterations occurring during the course of SAH have been previously reported. Clinically, the neurocardiogenic effects of subarachnoid hemorrhage may present with electrocardiographic changes, elevations of troponin and/or brain-type natriuretic peptide, as well as regional wall motion abnormalities, including Takotsubo cardiomyopathy.

SAH-induced ECG abnormalities can be transitory, and the frequency of ECG abnormalities in prospective SAH studies increases to near 100% when serial ECG monitoring is used [4, 5]. These ECG abnormalities are nonspecific and tend to change with time;

Several mechanisms for the occurrence of cardiac complications after SAH have been suggested, but none has been proven. However, a generally accepted hypothesis is that sympathetic stimulation induces catecholamine release in the myocardium, which may lead to impaired systolic and diastolic function, repolarization abnormalities, and myocardial damage. The first descriptions concerned electrocardiographic changes (changes: T wave, ST and QT segments, arrhythmias) as well as an elevation of myocardial biomarkers ([6]. These abnormalities are very frequently found (25 to 100% depending on the studies), but they seem, fortunately, rarely linked to myocardial damage [7].

Repolarization disorders are present in 30% of patients [8], QT prolongation is rarer, but favors the appearance of arrhythmias (4% of patients) [9]. ECG abnormalities are diffuse and more frequent in cases of severe neurological impairment.

Several studies have not found a correlation between the sudden rise in catecholamine levels and the electrical changes [10, 11].

BNP is also released in the acute phase of SAH and its elevation is linked to that of troponin [12]. Elevated troponin and BNP are both associated with mortality, but only troponin is associated with poor neurological outcomes.

Echocardiography is strongly recommended in the acute phase [13]. Contraction abnormalities are observed in 13 to 38% of patients and a decrease in ejection fraction < 50% in 15 to 32% of them [14, 15]. This complication is not limited to a single coronary territory. These abnormalities most often recover within a few days but could be predictive of delayed cerebral ischemia. Coronary angiography is normal, confirming that myocardial cell necrosis is related to elevated catecholamines [16].

In case of myocardial dysfunction, cardiac output monitoring should be considered [13]. Similarly, blood volume must be monitored. The treatment of serious cardiac complications is that of acute heart failure, favoring inotropic agents. Hemodynamic control can delay the treatment of the aneurysmal sac, without exceeding a few hours.

CONCLUSION

Assessing ECG changes during cerebral damage is important. SAH studies show that catecholamine toxicity is the most probable cause of cardiac injury temporally related to an explosive increase in intracranial pressure. Evaluation and management of patients with SAH and cardiac dysfunction should be aimed at reducing the risk for symptomatic cerebral vasospasm. Newer therapeutic approaches should incorporate this evolving understanding of the pathophysiology.

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