Case Report
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Subarachnoid Hemorrhage with Type 2 Myocardial Infarction: A Case Report

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Abstract: Acute myocardial infarction (AMI), stroke and subarachnoid hemorrhage (SAH) are all associated with high mortality following hospitalization. Electrocardiographic (ECG) changes occurring during SAH have been described frequently. To the best of our knowledge, there is no reported case with SAH and existing Q waves, ST elevations on anterior and lateral walls, and Atrial fibrillation on ECG.

We describe a 47-year-old female patient with a SAH that presented electrocardiographic evidence of MI. By reporting this case, we share our uncommon experience, the increasing reports will allow identifying the clinical features useful for differentiating diagnosis from myocardial infarction in order to avoid treatment with anticoagulants and antiplatelets, potentially dangerous particularly in the group of patients with a hemorrhagic cerebral accident.

Keywords: Subarachnoid hemorrhage, SAH, Type 2 MI, Myocardial infarction.

Introduction

Acute myocardial infarction (MI), stroke and subarachnoid hemorrhage (SAH) are all associated with high mortality following hospitalization [1]. In instances of myocardial injury with necrosis, where a condition other than coronary artery disease (CAD) contributes to an imbalance between myocardial oxygen supply and/or demand, the term "MI type 2" is employed [2]. In critically ill patients, or in patients undergoing major (non-cardiac) surgery, elevated values of cardiac biomarkers may appear, due to the direct toxic effects of endogenous or exogenous high circulating catecholamine levels. Also, coronary vasospasm and/or endothelial dysfunction have the potential to cause MI [2-5]. Electrocardiographic (ECG) alterations occurring during the course of subarachnoid hemorrhage (SAH) have been described frequently. In patients with acute aneurysmal SAH, repolarization abnormalities are the commonest ECG alterations, and ST depression is more common in patients with poor outcome. (6). Transient left ventricular dysfunction with an akinetic or dyskinetic apex has also been described in SAH patients without significant coronary heart disease [7-9]. We describe a patient with a SAH that presented electrocardiographic evidence of MI.

Case Presentation

A 47-year-old female patient with background of neurofibroma in bowels and underwent several surgeries due to bowel obstruction. She had ileostomy and was receiving TPN through Hickman catheter. The patient hospitalized several times due to pneumonia and sepsis and was received two times successfully CPR during hospitalizations. She had no any cardiac history. Her last hospitalization was due to leukocytosis, diagnosed with sepsis. During hospitalization she complained with facial numbness and consulted to neurology. She had nystagmus, anisocoric pupils and positive Babinski reflex. Computerized Tomography (CT) findings was diffuse acute SAH within resultant effacement of the basal cisterns and also acute intraventricular hemorrhage

(Fig. 1). Meanwhile she had positive troponin and CPK level (Tr 8,83 ng/ml, CPK 3284 U/l) and ECG changes (Q waves and ST elevations on anterior and lateral walls, atrial fibrillation (Fig. 2). She was diagnosed with type 2 MI but did not receive antiplatelet therapy because of the SAH. After positive blood cultures (*Staph. epidermidis*) the patient transferred to ICU with hemodynamically unstable profile and suspicion of endocarditis. In ICU the patient was intubated, sedated and mechanically ventilated. Started inotropes (noradrenaline and adrenaline drips) and antibiotics (meropenem, vancomycin and tazocin). Poor global function, thickened, pathological MV and AV was seen on bed side echocardiography. Neurosurgeons were waiting to perform any procedure because of the unstable hemodynamically condition and poor prognosis of the procedure for SAH. Decided to perform TEE by cardiologist but unfortunately the patient died on 4th ICU hospitalization due to severe sepsis and multiorgan dysfunction.

Discussion

Myocardial damage occurring in association with SAH is a well-described phenomenon [10-13]. It has been reported that abnormalities of electrocardiogrphy, echocardiography and serum cardiac specific markers are associated with cerebrovascular disease [14]. The most common cause is subarachnoid hemorrhage, but additional causes include head injury, meningitis and brain tumor [15-17]. The real pathophysiology remains unclear till now, coronary artery spasm, coronary thrombosis, and catecholamine induced oxygen supply-demand mismatch had been mentioned [18, 19].

Unconscious cerebral hemorrhage patients with electrocardiograms showing ST segment elevation, we have a tendency to misdiagnose as acute myocardial infarction and treat with multiple antiplatelet and anticoagulate agents, which can cause harmful effects. Furthermore, delaying accurate diagnosis may result in catastrophic outcome.

Conclusion

In critically ill patients with SAH, elevated cardiac biomarkers may appear, due to the direct toxic effects of endogenous or exogenous high circulating catecholamine levels. Also coronary vasospasm and/or endothelial dysfunction have the potential to cause MI. By reporting this case, we wish to share our uncommon experience and hope that it may be helpful in future cases. We hope that the increasing reports will allow to identify the clinical features useful for differentiate diagnosis from myocardial infarction in order to avoid treatment with anticoagulants and antiplatelets, potentially dangerous particularly in the group of patients with hemorrhagic cerebral accident.

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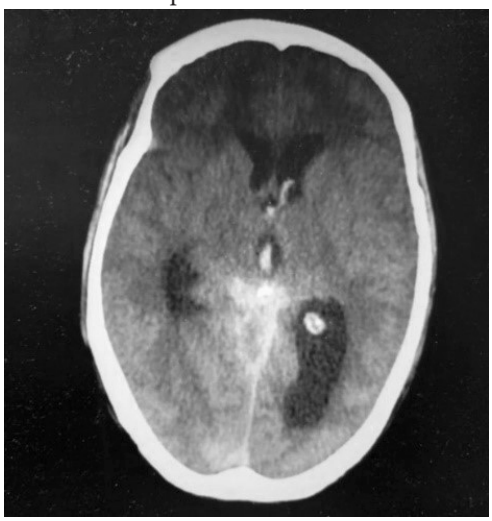

Figure 1. Cranial CT scan of the patient
There is hyperdense material is seen filling the subarachnoid space.

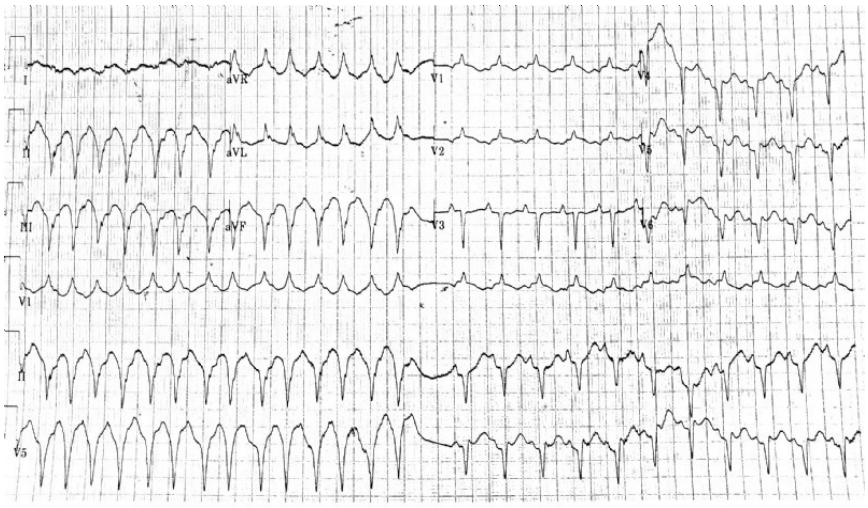

Figure 2. Electrocardiography (ECG) of the Patient
Q waves and ST elevations on anterior and lateral walls, atrial fibrillation.
Diagnose: Type 2 MI

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