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Case Report

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Subarachnoid hemorrhage mimicking ST-segment elevation myocardial infarction after return of spontaneous circulation

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Electrocardiogram changes in subarachnoid hemorrhage (SAH) have been described as ST-T changes that mimic acute coronary syndrome and even acute ST-segment elevation myocardial infarction. Elevation of cardiac enzymes and abnormality of regional myocardial wall motion have been reported frequently for SAH. We report a case of an out-of-hospital cardiac arrest survivor with high suspicion of ST-segment elevation myocardial infarction based on the electrocardiogram and bedside echocardiography, who had normal coronary arteries on emergent coronary angiography. The patient was ultimately diagnosed with SAH as a cause of out-of-hospital cardiac arrest.

Keywords Electrocardiography; Subarachnoid hemorrhage; Heart arrest

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Capsule Summary

What is already known

Subarachnoid hemorrhage patients may present with electrocardiogram changes, cardiac enzyme elevations, and echocardiographic abnormalities.

What is new in the current study

In the comatose resuscitated patient, subarachnoid hemorrhage should be considered as a possible cause of cardiac arrest, even with signs of acute myocardial infarction.

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INTRODUCTION

It is well known that acute coronary syndrome (ACS) is the most common cause of cardiac arrest.[1] However, spontaneous subarachnoid hemorrhage (SAH) is also a common neurological disorder that leads to out-of-hospital cardiac arrest.[2,3] Typical electrocardiogram (ECG) changes, such as ST-segment elevation or depression and abnormal T wave morphologies, may mimic myocardial infarction or ischemia. This may cause physicians to be confused between ACS and SAH.[4,5] Furthermore, SAH patients also have elevated troponin levels and myocardial regional wall motion abnormalities due to neurogenic stunned myocardium.[6] These nonspecific findings can lead to misdiagnosis and incorrect therapeutic decisions in these cardiac arrest patients, such as thrombolytic therapy and percutaneous coronary intervention. In this article, we report a case of an out-of-hospital cardiac arrest survivor whose initial ECG indicated a ST-segment elevation myocardial infarction (STEMI); however, she was finally diagnosed with SAH. Compared with previous reports of cardiac arrest survivors with SAH mimicking acute myocardial infarction (AMI), the ECG of this case was more distinct because of its ST-segment elevations in the lateral leads and the reciprocal ST-segment depression in the inferior leads indicating a typical STEMI. We suggest that, even with signs of AMI, clinicians should consider the possibility of SAH as a cause of cardiac arrest in comatose resuscitated patients.

CASE REPORT

A 48-year-old woman without any known history of disease suddenly collapsed and lost consciousness. The emergency medical service (EMS) providers responded within 5 minutes. She had no palpable pulse, and the initial ECG rhythm, determined by an automated external defibrillator, was asystole. EMS providers continues cardiopulmonary resuscitation (CPR) and arrived at the emergency department about 30 minutes after the sudden collapse.

In ED, CPR was performed in accordance with the current Advanced Cardiac Life Support guidelines of 2010. After 4 minutes of resuscitation with a total of 2 mg epinephrine, she recovered spontaneous circulation. Her vitals were as follows: blood pressure, 122/99 mmHg; pulse rate, 144/min; respiratory rate, 20/min. She was still unconscious, and both pupils were dilated and non-reactive. Her Glasgow Coma Scale Score was 3; endotracheal intubation was performed for airway protection by an emergency physician. The initial 12-lead ECG showed sinus tachycardia and ST-segment elevation in leads I, aVL, V5, and V6 with reciprocal ST-segment depression in leads III, aVF, and V1 to V3 (Fig. 1). Results of the initial biochemical tests revealed followings: sodium 142 mmol/L, potassium 3.7 mmol/L, glucose 321 mg/dL, serum creatine kinase MB 4.5 U/L, and troponin-I 0.63 μg/L.

Within 10 minutes after recovery of spontaneous circulation, a low blood pressure was documented (83/43 mmHg), and norepinephrine infusion was started in the mean time a cardiologist performed an emergent bedside echocardiography and decided to do an emergent coronary angiography because of regional wall abnormalities that suggested an ischemic insult of the left circumflex artery.

At 30 minutes after recovery of spontaneous circulation, she underwent coronary angiography, which revealed normal coronary arteries without any evidence of stenosis. After admission into the cardiac care unit, a computed tomography scan of the brain was performed. The brain computed tomography scan demonstrated diffuse SAH with brain swelling (Fig. 2). Surgical inter-

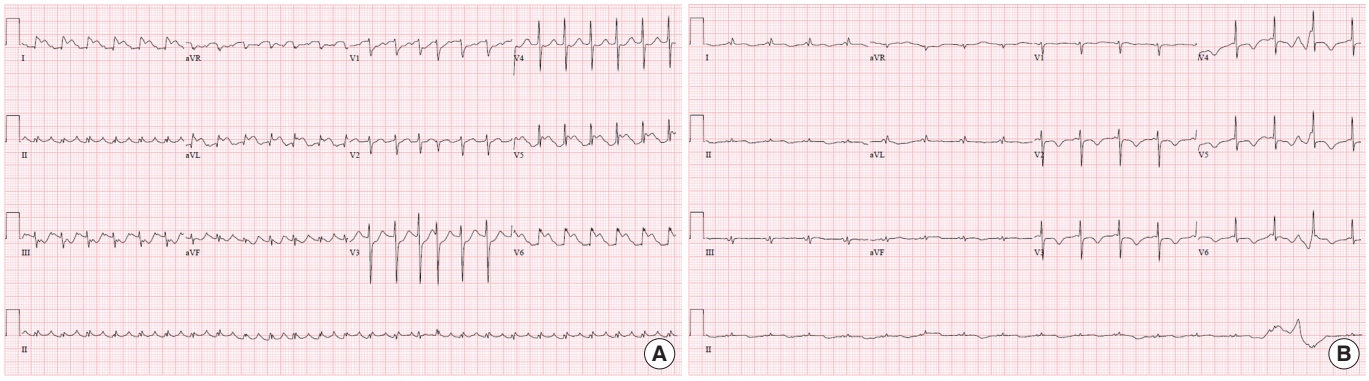

Fig. 1. (A) Initial electrocardiogram showing sinus tachycardia, and ST-segment elevation in leads I, aVL, V5, and V6, with reciprocal ST-segment depression in leads III, aVF, and V1 to V3. (B) Twenty hours later, electrocardiogram showed that ST-segment abnormalities disappeared and T wave inversion were noted.

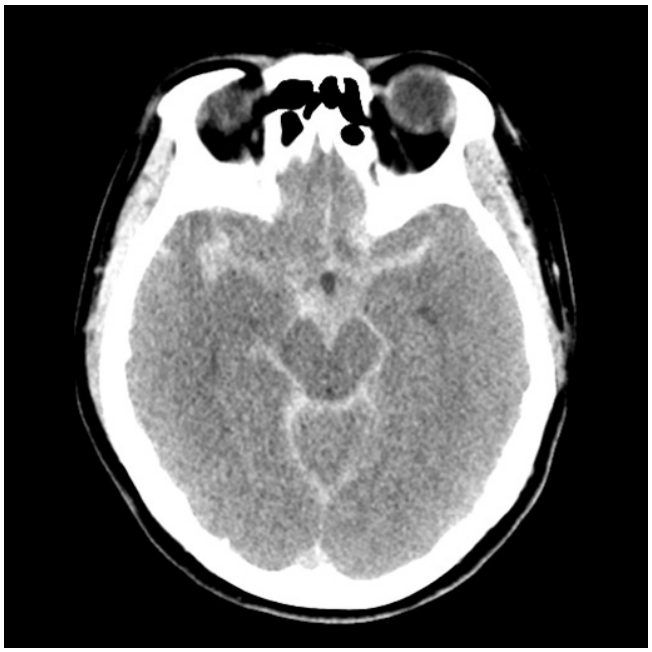

Fig. 2. Computed tomography scan of brain demonstrating diffuse subarachnoid hemorrhage with brain swelling.

vention was not considered because of her unstable condition.

Twenty hours after the onset of the symptoms, the ST-segment abnormalities disappeared and T-wave inversion was noted (Fig. 1B). Serial values of the serum cardiac enzymes increased within 24 hours, and the peak values of creatine kinase MB and troponin-I were 28.3 U/L and 7.25 μg/L, respectively. On the third day of hospitalization, the echocardiography showed akinesia of the left ventricle (LV) mid-walls with moderate to severe LV dysfunction (ejection fraction, 36%), which implied a variant form of stress-induced cardiomyopathy. Despite the administration of a vasopressor, her blood pressure was not maintained. The shock progressed gradually, and on the 13th day of hospitalization, she was pronounced dead.

DISCUSSION

Many cases have been reported with cardiac manifestations following SAH including ECG changes such as ST-segment deviations, T-wave inversion, and QTc prolongation;[5] cardiac enzyme elevations; and echocardiographic abnormalities. Despite many reports, it is still a challenge for clinicians to distinguish intracranial hemorrhage from ACS, especially among post-cardiac arrest survivors.

A previous studies on cardiac manifestations of SAH showed that cardiac abnormalities were found in 14.1% of patients with non-traumatic SAH. The most common ECG finding was T-wave inversion and ST-segment depression, suggesting subendocardial ischemia.[4] Mitsuma et al.[5] first demonstrated the frequency of cardiac abnormalities in SAH patients who experienced cardiac arrest. They reported that 8 out of 10 patients had ST-segment elevation in aVR with ST-segment depression in widespread leads, and three of these patients showed Takotsubo-like regional LV dysfunction.

To our knowledge, this is the first report describing an out-of-hospital cardiac arrest survivor whose initial ECG changes and echocardiographic findings strongly suggested STEMI, but who was finally diagnosed with SAH after the emergent coronary angiography revealed entirely normal coronary arteries. The ECG changes of lateral wall myocardial infarction typically present as ST-segment elevation in leads I, aVL, V5, and V6 with reciprocal ST-segment depression in leads II, aVF, V1, and V2. However, lateral wall infarction usually occurs in other territories of the myocardium such as the anterolateral and inferolateral areas. In our case, the ST-segment elevation in the lateral leads was obvious, but the reciprocal changes of the ST-segment depression were relatively obscure. In addition, there was no contiguous ST-segment depression in the precordial leads and the upsloping ST-segment depression was an unusual finding for ischemic myocardial injury.

Many interventions have been emphasized for improving post-cardiac arrest outcomes, including immediate thrombolysis and percutaneous coronary intervention.[7] According to the current guidelines, regardless of the level of consciousness in cardiac arrest survivors, aggressive treatment such as percutaneous coronary intervention was recommended for STEMI. Emergent coronary angiography was suggested even patients without evidence of STEMI.[7] Emergency physicians may also feel pressured to promptly initiate revascularization treatment due to the importance of door-to-balloon interval. Thus, further studies will be needed to clarify the differences in the ECG findings of ACS and SAH.

We believe that observations reported here have potentially significant implications for physicians making decisions of post-arrest care, for once successfully resuscitated patients. Clinicians should consider an immediate brain computed tomography to exclude SAH as a cause of cardiac arrest, even with ST-segment elevation.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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