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When subarachnoid hemorrhage mimics acute coronary syndrome in out-of-hospital cardiac arrest: a diagnostic challenge

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Abstract

Background Out-of-hospital cardiac arrest (OHCA) is most often due to acute coronary syndrome (ACS), but less frequent causes like subarachnoid hemorrhage (SAH) may mimic cardiac disease through ECG changes and biomarker elevation, risking misdiagnosis and inappropriate interventions. The aim of this case was to present a case of a patient who experienced OHCA due to subarachnoid hemorrhage mimicking ACS leading to catastrophic results.

Case presentation A 42-year-old woman with no cardiovascular risk factors suffered sudden cardiac arrest during exercise. Initial ECG and troponin levels suggested SCA, but coronary angiography was normal. Subsequent head CT revealed a massive subarachnoid hemorrhage. Despite supportive care, the patient died on day 5 from cerebral edema.

Conclusion SAH can cause cardiac arrest with ECG changes mimicking ACS, highlighting the importance of cranial CT in comatose post–cardiac arrest patients.

Keywords Out-of-hospital cardiac arrest, Like subarachnoid hemorrhage, Acute coronary syndrome, Computed tomography

Background

Out-of-hospital cardiac arrest (OHCA) is most frequently caused by acute coronary syndrome (ACS) [1]. However, non-cardiac causes, such as acute cerebrovascular events like subarachnoid hemorrhage (SAH), are less common [2]. SAH can be accompanied by electrocardiogram abnormalities, myocardial injury, and elevated levels of cardiac biomarkers in some patients leading to misdiagnosis and incorrect therapeutic decisions in these cardiac arrest patients, such as thrombolytic therapy and percutaneous coronary intervention [3].

Case presentation

A woman 42-year-old without history of cardiovascular risk factor presented in her home a sudden cardiac arrest. She was found lying on the floor before being taken to the Emergency Department. According to the family, the patient collapsed at her home when she was training. His husband denied any chest pains, palpitations or seizure like activities before the collapse. Cardiopulmonary resuscitation (CPR) was initiated which was continued by emergency medical services for 15 min following current resuscitation guidelines. Return of spontaneous circulation was achieved. She remained unconscious, with both

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pupils dilated and non-reactive, and her Glasgow Coma Scale (GCS) score was 3. Endotracheal intubation was performed for airway protection by an emergency physician and sedated with midazolam and fentanyl.

On initial evaluation in the Emergency Department the patient was afebrile, a blood sugar level of 94 mg/dl, her vitals were as follows: blood pressure at 108/59 mmHg; pulse rate at 114 beats/min;

The initial 12-lead ECG showed sinus tachycardia and ST-segment elevation in aVR and V1 with ST-segment depression in all other leads (Fig. 1). Results of the initial biochemical tests revealed the following: sodium 142 mmol/L, potassium 3.7 mmol/L.

Transthoracic echocardiography findings were all normal, but troponin I level was high at 0.63 µg (reference range: 0-0.04 ng/mL). The initial diagnosis was Acute coronary syndrome (Very High-Risk NSTEMI). Therefore, the patient was admitted to the Department of Cardiology. No loading dose of acetyl salicylic acid and clopidogrel has been given and heparin was not administered.

The patient was immediately taken to the angiography laboratory to perform primary percutaneous coronary intervention coronary angiography, which revealed normal coronary arteries without any evidence of stenosis (Fig. 2). A computed tomography (CT) scan of the head without contrast (Fig. 3) revealed massive subarachnoid hemorrhage (SAH), classified as grade IV according to the Fisher scale, with crowding of the basilar cisterns and a mass effect on the ventricles. The patient was deemed ineligible for neurosurgical intervention, given the patient's clinical status. On the 5th day until the admission, the patient died because of acute cerebral oedema with encephalomalacia.

Discussion

Sudden cardiac arrest (SCA) results from abrupt disturbances in the electrical or mechanical activity of the heart, or from a lack of synchrony between these two components [4].

The underlying mechanisms include ventricular fibrillation (VF), pulseless ventricular tachycardia (VT), asystole, and pulseless electrical activity (PEA). Among out-of-hospital cardiac arrests (OHCA), the most common initial rhythms are VF and VT [1].

While acute coronary syndrome (ACS) has traditionally been considered the predominant etiology of SCA, recent studies using advanced imaging modalities, particularly computed tomography (CT), have revealed a higher proportion of non-cardiac causes than previously recognized [2].

The diagnosis of acute coronary syndrome (ACS) after sudden cardiac arrest is based on evidence of myocardial

Fig. 1 Initial electrocardiogram showing sinus tachycardia, and ST-segment elevation in aVR with ST-segment depression in V3 to V6, DI and aVL and inferior leads

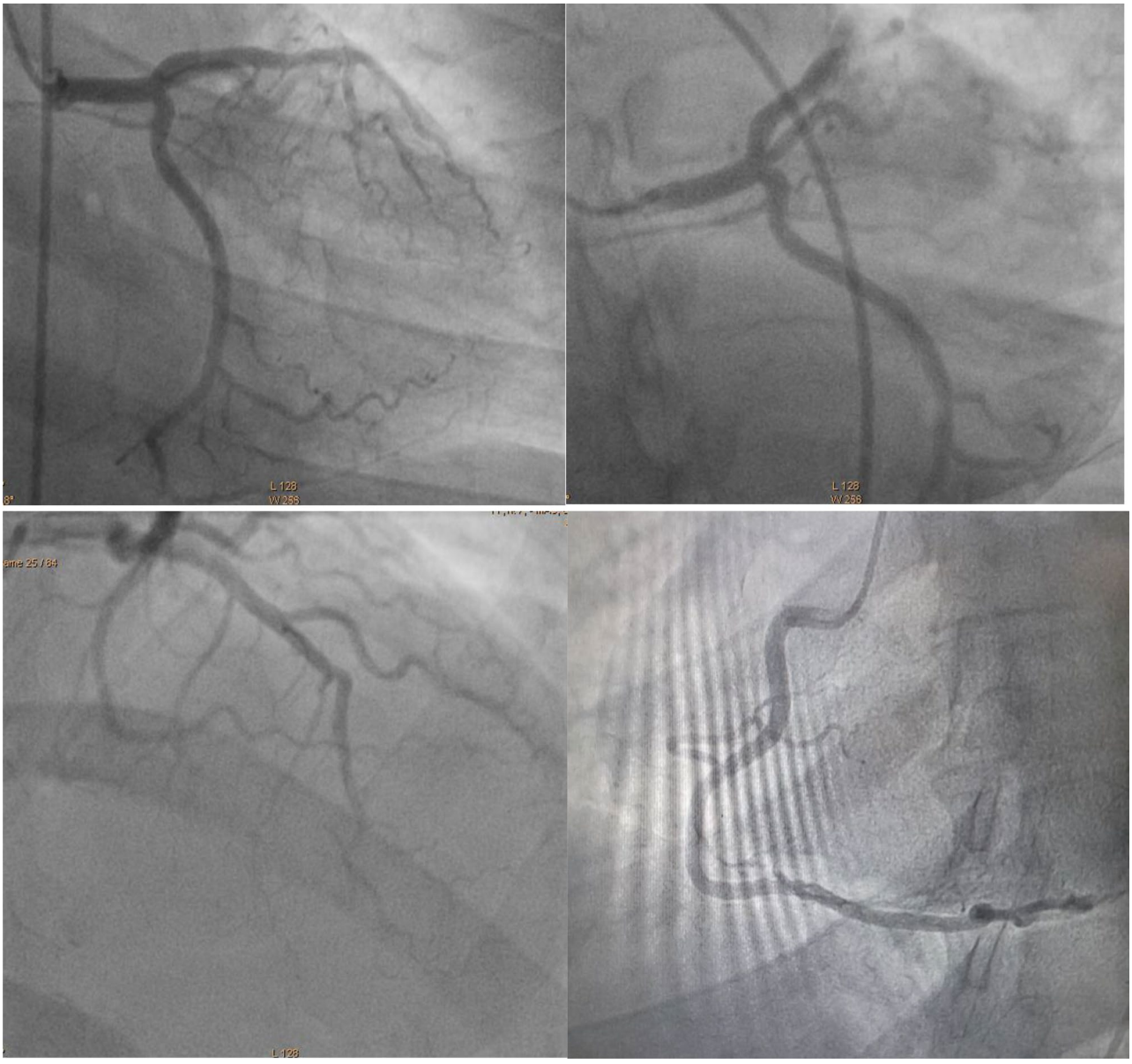

Fig. 2 Coronary angiography revealed normal coronary arteries without any evidence of stenosis

ischemia—such as characteristic ECG changes, elevated cardiac troponin, or imaging findings [5]. In patients successfully resuscitated from SCA who demonstrate ST-segment elevation or equivalent (like our patient) urgent coronary angiography is strongly recommended [5].

However, several secondary causes of OHCA must also be considered, including subarachnoid hemorrhage (SAH), pulmonary embolism, and severe respiratory failure. SAH, most often due to rupture of a cerebral aneurysm, may mimic ACS by producing ECG abnormalities, elevated troponin levels, and echocardiographic findings suggestive of myocardial dysfunction [3]. These abnormalities—commonly including repolarization disturbances and stress-induced (Takotsubo) cardiomyopathy—can lead to diagnostic confusion and inappropriate management, particularly the administration of anticoagulants, which are contraindicated in SAH [6].

Previous studies have demonstrated that cardiac manifestations occur in approximately 14.1% of patients with non-traumatic SAH. The most frequent ECG findings include T-wave inversions and ST-segment depression, consistent with subendocardial ischemia [7].

Mitsuma et al. were the first to describe the prevalence of cardiac abnormalities among SAH patients who suffered cardiac arrest, reporting that 8 out of 10 exhibited ST-segment elevation in lead aVR accompanied by diffuse ST-segment depression, while three of these patients

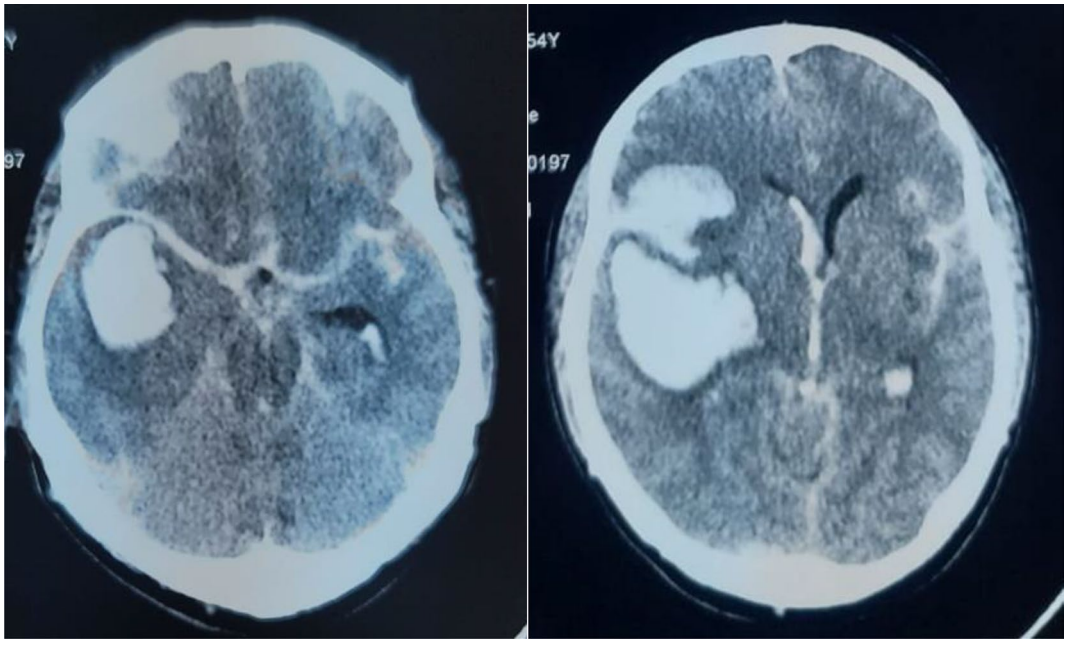

Fig. 3 A computed tomography (CT) scan of the head revealed massive subarachnoid hemorrhage (grade IV according to the Fisher scale) with crowding of the basilar cisterns and a mass effect on the ventricles

developed Takotsubo-like regional left ventricular dysfunction [8].

The pathophysiological mechanism linking SAH to cardiac abnormalities involves a massive sympathetic discharge mediated by the central neuroendocrine axis, leading to excessive catecholamine release from both adrenal glands and cardiac nerve terminals [9].

This catecholaminergic storm can trigger ventricular arrhythmias such as VF and cause ECG abnormalities. Furthermore, SAH may induce coronary vasoconstriction, resulting in subendocardial ischemia and direct catecholamine-induced myocardial injury, which may culminate in myocardial infarction [10]. The combined effects of catecholamine toxicity, transient coronary spasm, inadequate perfusion during VF, and electrical injury from defibrillation contribute to elevated troponin levels frequently observed after resuscitation.

To differentiate SAH from ACS, clinicians should carefully evaluate troponin dynamics, echocardiographic patterns, and clinical context, particularly the presence of headache or neurological symptoms preceding cardiac arrest [11]. In cases where SAH is suspected, prompt brain CT imaging is essential. Failure to recognize SAH and its misclassification as ACS may delay appropriate neurosurgical management and lead to clinical deterioration.

Point-of-care ultrasound (POCUS) is an invaluable bedside tool in the early post-return of spontaneous circulation (ROSC) phase. Focused echocardiography can help differentiate global left ventricular dysfunction seen in subarachnoid hemorrhage or stress cardiomyopathy from regional wall motion abnormalities suggestive of coronary occlusion [12]. Additionally, assessment of the optic nerve sheath diameter and transcranial Doppler may provide indirect evidence of raised intracranial pressure or vasospasm, supporting a neurogenic cause [13]. Thus, POCUS can guide diagnostic prioritization between coronary and neuroimaging investigations in post-ROSC patients with unclear etiology [14].

Conclusion

SAH can cause cardiac arrest and sometimes the ECG modifications may mimic that of the acute coronary syndrome. CT scan should be considered to exclude intracranial pathology in a comatose post cardiac arrest with myocardial Infarction.

Abbreviations

OHCA	Out-of-hospital cardiac arrest
SCA	Sudden cardiac arrest
ACS	Acute coronary syndrome
SAH	Subarachnoid hemorrhage
CPR	Cardiopulmonary resuscitation
VF	Ventricular Fibrillation
VT	Ventricular Tachycardia
CT	Computed tomography

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Author contributions

Lahmouz youssef and Jaouad Nguadi contributed to the clinical management of the patient and drafting of the manuscript.Hanae El Ghiati contributed to data collection and preparation of the clinical documentation.Jihane Fagouri contributed to the literature review and drafting of the discussion section. Meryem Bennani contributed to patient evaluation and revision of the manuscript.Abdelilah Ben El Makki contributed to diagnostic interpretation

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Consent to publish
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Competing interests
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