Indicators of Subarachnoid Hemorrhage as a Cause of Sudden Cardiac Arrest

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Subarachnoid hemorrhage (SAH) may present with cardiac arrest (SAH-CA). We report a case of SAH-CA to assist providers in distinguishing SAH as an etiology of cardiac arrest despite electrocardiogram findings that may be suggestive of a cardiac etiology. SAH-CA is associated with high rates of return of spontaneous circulation, but overall poor outcome. An initially non-shockable cardiac rhythm and the absence of brainstem reflexes are important clues in identifying SAH-CA. [Clin Pract Cases Emerg Med. 2017;1(2):132–135.]

INTRODUCTION

Subarachnoid hemorrhage (SAH) as a cause of out-of-hospital cardiac arrest (OHCA) is rare.1-3 Most patients will present with pulseless electrical activity or asystole. SAH-CA is not typically due to intrinsic cardiac disease although numerous changes can be seen on electrocardiography.4,5 We report a case of SAH-CA to review the pathophysiology and remind clinicians to consider intracranial hemorrhage as a cause of death in any patient presenting with a non-shockable rhythm and lack of brainstem reflexes.

CASE REPORT

A 55-year-old man with no cardiac history collapsed at work. Bystander cardiopulmonary resuscitation (CPR) was initiated promptly, and initial rhythm on emergency medical services (EMS) personnel arrival was asystole. Return of spontaneous circulation (ROSC) was achieved within four minutes of CPR and epinephrine administration. Endotracheal intubation was performed without sedative or paralytic agents and the patient was transported to the emergency department (ED). A prehospitalization 12-lead electrocardiogram (ECG) demonstrated diffuse anterolateral ST segment depression and aVR ST segment elevation (Image 1a). Upon arrival to the ED, the patient was found to be pulseless with electrical activity and required an additional seven minutes of CPR and multiple doses of epinephrine before ROSC was achieved. Point-of-care ultrasound demonstrated depressed left ventricular function without signs of right ventricular strain, aortic enlargement, or apical ballooning syndrome. The patient had fixed and dilated pupils, no spontaneous respiratory efforts, no response to painful stimuli, and required continuous epinephrine to maintain his blood pressure.

Based on the prehospital ECG, a posterior ST segment elevation myocardial infarction was considered as a cause of his arrest, although a repeat ECG demonstrated substantial resolution of the prehospital electrocardiographic abnormalities (Image 1b).

The case was discussed with the critical care cardiologist, and the cardiac catheterization laboratory was activated. The absence of an initial shockable rhythm raised diagnostic uncertainty and a computed tomography (CT) of the head was obtained prior to catheterization. CT of the head demonstrated diffuse SAH with intraventricular extension and mild hydrocephalus. Follow-up CT angiogram revealed a small anterior communicating artery aneurysm. The plan for coronary angiography was cancelled. Soon after admission to the ED, the patient was found to be pulseless with electrical activity and required an additional seven minutes of CPR and multiple doses of epinephrine before ROSC was achieved.
the neurosciences intensive care unit the patient was declared brain dead and became an organ donor.

**DISCUSSION**

It is estimated that only 2-8%\(^1\) of OHCA are caused by SAH. More than half of SAH-CA patients collapse without preceding headache and 12% may never reach medical attention.\(^6\)-\(^9\) Non-cardiac causes of OHCA more often present with a non-shockable rhythm, like pulseless electrical activity (PEA) and asystole. Almost all patients with SAH have been shown to have ECG changes, and nearly two-thirds had changes suggestive of myocardial infarction or ischemia.\(^10\)-\(^14\) ECG changes in the setting of SAH can include sinus tachycardia, atrial fibrillation, ST segment elevation or depression, QT prolongation, and rarely torsades de pointe, ventricular tachycardia and ventricular fibrillation (VF).\(^10\),\(^11\) VF after an initially non-shockable rhythm is likely secondary to CPR and epinephrine administration.\(^14\) VF and torsades de pointe in patients with OHCA secondary to SAH are uncommon.\(^4\),\(^10\),\(^11\),\(^14\)

Anoxic ischemic brain injury from primary cardiac causes of sudden cardiac arrest preferentially affect the cortex and diencephalon and spare the brainstem.\(^15\)-\(^17\) Absent pupillary, corneal, oculocephalic responses, spontaneous breathing or a Full Outline of UnResponsiveness (FOUR) score of 0 is highly unusual after CPR and ROSC unless associated with extremely prolonged resuscitation or exsanguination.\(^18\),\(^19\) A FOUR score is a 20-point clinical grading scale (with scores of 0 to 16) designed to assess patients with an impaired level of consciousness.\(^19\) The FOUR score assesses eye responses, motor responses, brainstem reflexes, and breathing pattern. It can be used in place of the Glasgow Coma Scale in intubated patients with traumatic or nontraumatic brain injuries and has been validated in the ED.\(^20\) The FOUR score would test several brainstem reflexes and breathing drive not identified with a GCS of 3.

Why does massive SAH lead to cardiac arrest? Why do patients with SAH-CA present with asystole or PEA? The cause of SAH-CA is not due to intrinsic cardiac disease, as
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