

Cerebellar Haemorrhage Leading to Sudden Cardiac Arrest

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ABSTRACT

Introduction: Intracranial haemorrhage (ICH) is a known, but a rare cause of out of hospital cardiac arrest (OHCA). It results in the development of non-shockable rhythms such as asystole or pulseless electrical activity (PEA).

Case Report: A 77- years old male had an OHCA without any prodrome. An emergency medical services (EMS) team responded to an emergency call and intubated the patient at the site before transporting him to the Acute Care Hospital, New Brunswick, New Jersey, USA. On admission, a non-contrast computed tomography scan of the head revealed a large cerebellar haemorrhage. Non-traumatic ICH is a rare cause of OHCA. Although subarachnoid haemorrhage causing cardiac arrest has been described in the literature, cerebellar haemorrhage leading to cardiac arrest is rare. The mechanism by which ICH patients develop cardiac arrest is likely explained by a massive catecholamine surge leading to cardiac stunning. **Conclusion:** A non-shockable rhythm in the setting of a sudden cardiac arrest should raise alarms for a primary non-cardiac ethology, especially a primary cerebrovascular event. The absence of brainstem reflexes increases the likelihood of an intracranial process.

Keywords: cardiac arrest, intracranial haemorrhage, asystole, cerebellar haemorrhage

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INTRODUCTION

Intracranial haemorrhage (ICH) is a known, but a rare cause of out of hospital cardiac arrest (OHCA) leading to the development of non-shockable rhythms such as asystole or pulseless electrical activity (PEA). The incidence of ICH causing OHCA is unclear but 4-18% of OHCA are caused by subarachnoid haemorrhage (SAH) [1]. Fatal cerebellar haemorrhage leading to a cardiac arrest is very rare compared to SAH.

CASE REPORT

A 77-years old Caucasian male with a history of coronary artery triple bypass surgery, atrial fibrillation and aortic valve replacement with no history of hypertension, was found unresponsive at home by his wife. An emergency medical services (EMS) team responded to an emergency call. They were informed that he was on

apixaban (ELIQUIS®) [Bristol-Myers Squibb Company, Princeton, NJ, USA] 5 mg two times a day. There was no palpable pulse upon their arrival. The heart rhythm was asystolic. Prompt cardiopulmonary resuscitation (CPR) was initiated and endotracheal intubation was performed. Three rounds of epinephrine [HOSPIRA®, Inc., Wake Forest, IL, USA] were administered through an intraosseous access and return of spontaneous circulation (ROSC) was achieved after ten minutes. No defibrillation was given as the rhythm was non-shockable throughout the event. He was transported to the emergency department (ED) from where he was admitted to the coronary care unit (CCU). On arrival, his blood pressure was 52/28 mmHg, heart rate was 57 beats per minute, oxygen saturation was 92% on ventilator settings of pressure regulated volume control (PRVC) mode, tidal volume of 500 mL, positive end expiratory pressure (PEEP) of 5 mm Hg, fraction of inspired oxygen (FiO₂) of 100%, respiratory rate of 12

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breaths per minute and temperature of 93.6 F. His pupils were fixed and dilated and there was no response to painful stimuli or any spontaneous respiratory efforts.

A 12-lead EKG showed an atrial fibrillation with a right bundle branch block. An arterial blood gas (ABG) showed a pH of 6.9, pCO₂ of 68 mm Hg, pO₂ of 94 mm Hg, oxygen saturation of 88% and bicarbonate of 13 mmol/L. Lactic acid was reported to be 9.6 mmol/L. Immediate central venous access was obtained and the patient was given three litres of intravenous normal saline boluses for intravascular volume expansion. Bicarbonate infusion was initiated. He was also started on norepinephrine, dopamine and epinephrine infusions for hemodynamic support. An emergent non-contrast computed tomography (CT) scan of the head was obtained which revealed a large cerebellar haemorrhage with extension into the ventricles and upper cervical region with resultant hydrocephalus (Figure 1). A consultant neurosurgeon was consulted and a decision was made that no acute surgical intervention was to be undertaken at this point. The patient was not a good candidate for therapeutic hypothermia, given the haemodynamic instability and intracranial haemorrhage. The family declined any further interventions or any escalation of care and unfortunately, the patient died after 22 hours of admission.

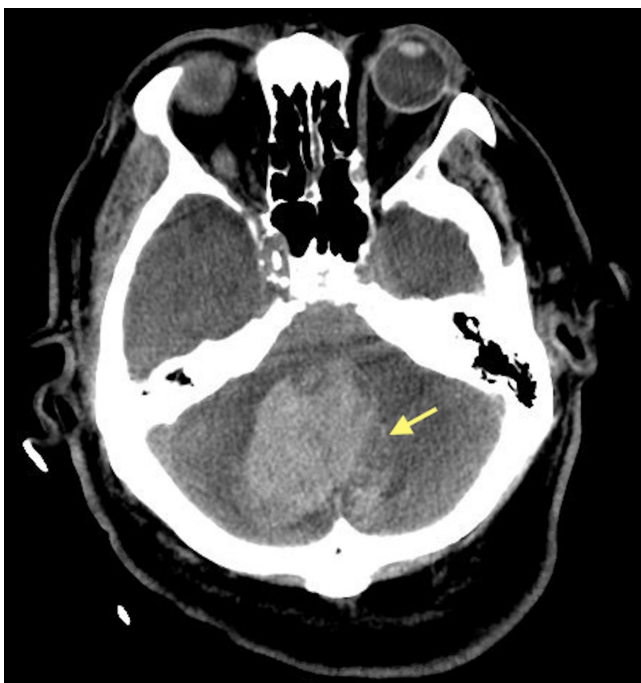


Fig. 1. Non-contrast CT scan of the head showing a large cerebellar hemorrhage (yellow arrow).

■ DISCUSSION

The true incidence of cardiac arrest from non-cardiac origin, especially neurological aetiologies, is unclear. Some reports suggest that cardiac arrest of non-cardiac aetiology comprises one third of the total number of cases of cardiac arrest and usually presents as non-shockable rhythms like asystole or PEA [2]. Although subarachnoid haemorrhage (SAH) has been described as a cause of cardiac arrest, non-traumatic ICH is an uncommon cause of OHCA. Only 2-8% of OHCA is caused by SAH [2]. In these patients, examination of the patient often reveals ST segment changes and other abnormal findings, which may falsely suggest a coronary event as the cause of sudden cardiac death [3]. In 2015, Arnaout et al. [4] showed that in comparison to a non-neurological cardiac arrest group, female gender, onset of a neurological prodrome, initial non-shockable rhythm and unspecific EKG repolarization abnormalities were independent predictive factors of a primary cerebrovascular event.

There is a growing body of evidence supporting SAH as a primary cause of OHCA, but reports of cerebellar bleed causing OHCA are extremely rare. In a study conducted by Inamasu et al. [5] in 2009, out of 124 patients who had a witnessed OHCA, six patients complained of sudden headache prior to collapse. Of these patients, four were found to have SAH and only two were found to have cerebellar haemorrhage [5]. The present case is unique because the patient was an elderly male presenting with no neurological prodrome. Nonetheless, the prognosis of OHCA associated with SAH or other intracranial haemorrhage is grave, but ROSC can be achieved temporarily after initial resuscitative efforts.

The mechanism by which ICH patients develop cardiac arrest is likely explained by a massive catecholamine surge leading to cardiac stunning. Also, a sudden rise in the intracranial pressure from the haemorrhage leads to brainstem dysfunction causing respiratory arrest and hypoxia. Severe hypoxia, in turn, triggers the release of adenosine, thus decreasing cardiac contractility, atrioventricular conduction and automaticity [2]. ICH as the cause of OHCA can be suspected in patients with evidence of brainstem dysfunction following ROSC. Anoxic ischemic brain injury from cardiac arrest secondary to primary cardiac aetiologies affects the cortex and spares the brainstem. It can involve the brainstem in cases of extremely prolonged resuscitation. The absence of spontaneous respiratory efforts,

pupillary reflex, oculoccephalic response and corneal reflex heightens the possibility of brainstem involvement from an intracranial pathology.

■ CONCLUSION

A non-shockable rhythm in the setting of a sudden cardiac arrest should raise alarms for a primary non-cardiac aetiology, especially a primary cerebrovascular event. The absence of brainstem reflexes increases the likelihood of an intracranial process. Potentially treatable intracranial lesions should also be considered to avoid further cardiac arrest.

In conclusion, the present case highlights the need to consider intracranial haemorrhage as a rare but important cause of OHCA.

■ CONFLICT OF INTEREST

Authors declare no conflict of interests.

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