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Case Report

Complete neurologic recovery in an out-of-hospital cardiac arrest facilitated by initiation of therapeutic hypothermia in a young athlete with an anomalous right coronary artery

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Abstract Therapeutic hypothermia is an accepted modality for improving neurologic outcome in patients who have sustained cardiac arrest with return of spontaneous circulation. Despite postresuscitative neuroprotection, it is uncommon to have patients who have undergone prolonged cardiopulmonary resuscitation to recover full neurologic function. An unusual case of sudden cardiac arrest in a young athlete with an anomolous right coronary artery is presented.

1. Case report

A 21 year old man experienced a witnessed collapse after an intense training session. Cardiopulmonary resuscitation (CPR) was immediately initiated and the patient received one time defibrillation with an automated external defibrillator (AED) for a ventricular tachycardia/ventricular fibrillation (VT/VF) arrest. The patient continued to receive CPR after defibrillation for 23 minutes before return of spontaneous circulation (ROSC). He had an organized rhythm and palpable pulse during the 5-minute transport to a medical facility. His pulse was 120 beats per minute, respiratory rate 18 breaths per minute, and blood pressure 135/78 mmHg; he was afebrile on arrival at the emergency department. He soon exhibited generalized tonic-clonic seizures, for which he was given intravenous (IV) lorazepam and phenytoin, but he continued to be in status epilepticus. He then experienced decorticate posturing with fixed and unresponsive pupils, and was intubated and sedated. His initial electrocardiogram (ECG) showed sinus tachycardia with nonspecific T wave abnormalities. Admission laboratory data showed a white blood cell count of 10.6 K/cm^2, hemoglobin 16.2 g/dL, platelet count 279 K/cm^2, Na^+ 144 mEq/L, K^+ 4.0 mEq/L, Cl^- 98 mEq/L, CO_2 16 mEq/L, sGOT 60 IU/L, sGPT 46 IU/L, creatinine 0.77 mg/dL, glucose 106 mg/dL, lactate 6.4 mml/L, CK-MB 3.3 ng/mL, Trop T < 0.010 ng/mL (negative at < 0.040 ng/mL), and INR 1.1. Total creatine kinase was not measured. An arterial blood gas showed a pH of 7.18, PaO_2 390 mmHg, and $PaCO_2$ 54 mmHg on FIO_2 100%. Urine toxin screen was positive for cannabis. Cranial computed tomographic (CT) scan was negative for intracranial bleeding, and his chest radiograph was within normal limits.

His past medical history was relatively unremarkable with the exception of traumatic injury to his left forearm and

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abdomen, from which he recovered completely without complications. He was an elite athlete with an otherwise unremarkable past medical and family history.

The patient was admitted to the intensive care unit (ICU) where therapeutic hypothermia for neuroprotection was initiated. Hypothermia was achieved via an IV femoral cooling catheter (ICY® catheter; Zoll Medical Corp., Chelmsford, MA, USA) with a computerized cooling system (Alsius CoolGard 3000®; Zoll Medical Corp.). A controlled rate of cooling at less than 1.5° C/hr was used to achieve a goal core temperature of 33° C. During the cooling process, a vecuronium infusion and intermittent IV meperidine were administered to prevent shivering. The patient was given amiodarone and lorazepam infusions since admission, and IV regular insulin was infused to maintain serum glucose between 120 and 160 mg/dL.

The patient achieved the desired core temperature within 12 hours without experiencing arrhythmias or hypotension. His temperature remained at 33° C for 18 hours, after which active rewarming was initiated at 0.5° C/hr. The rewarming goal temperature of 37.5° C was reached within 10 hours. Lorazepam and vecuronium infusions were then discontinued and the patient was given a propofol infusion. By the third inpatient day, the patient had spontaneous facial movements and he responded to painful stimuli. He was extubated on the seventh hospital day. On the following day, the patient was awake, alert, and ambulating without complaints. Neurologic examination showed no deficits except loss of memory of the time leading up to cardiac arrest.

Subsequent electroencephalogram (EEG) and electrophysiology studies were inconclusive. Lab data were unremarkable and he had negative cultures. Cardiac catheterization and chest CT angiography showed an anomalous origin of the right coronary artery (RCA) (Fig. 1), which was felt to be the primary cause of his cardiac arrest. The origin was superior to the level of the left coronary sinus, with 90° angulation of the vessel as it turned and coursed between the right ventricle and the pulmonary outflow tract. The patient then underwent open repair and reimplantation of the anomalous RCA and was discharged home a week after surgery. He is actively competing as an elite athlete without symptoms or recurrence of the arrhythmia.

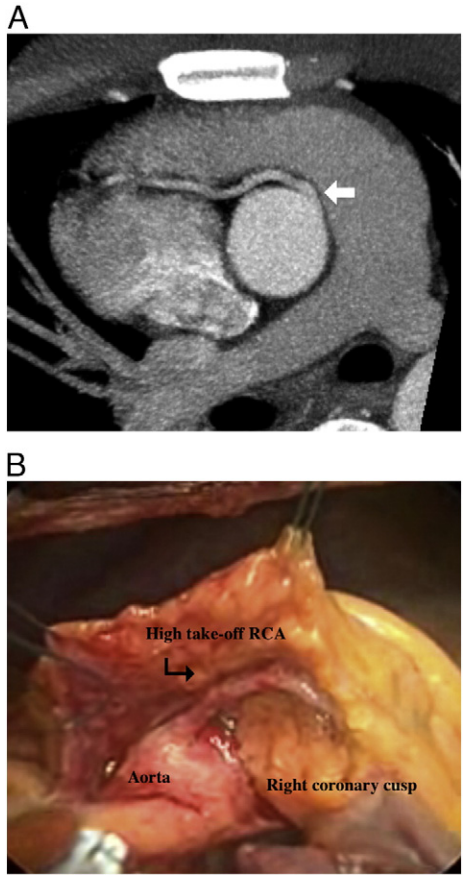

Fig. 1 (A) CT-angiography showing the right coronary artery (RCA) ostium (arrow) with a clockwise rotation arising from the tubular portion of the ascending aorta. (B) Intraoperative finding of a high-takeoff RCA.

2. Discussion

2.1. Anomalous coronary artery

As a group, coronary artery disease (CAD) and cardiac anatomic anomalies account for up to 80% of sudden cardiac deaths. The prevalence of an anomalous origin of a coronary artery range from 0.2% to 1.4% of the general population, with the majority of these anomalies involving the left coronary artery [1,2]. Though uncommon, it is the second most frequently encountered pathology after hypertrophic cardiomyopathy that is associated with sudden death.

Sudden death due to an anomalous RCA is rare. The anomalous artery often courses between the pulmonary artery and aorta, where it may be compressed by the great vessels or develop an acute angled kinking at its origin. Other abnormalities such as a slit-like ostium may also result in ischemia and sudden death [3]. Curiously, pathologic analyses of deceased individuals with distinct anomalous coronary artery pathology of the right or left side have shown no relation to sudden death rates with regard to ostium size, angle of take-off, or length of intramural course of the anomalous artery. It was noted also that age greater than 30 years is associated with a lower incidence of sudden death from anatomic anomalies [4].

The management of an anomalous coronary artery is primarily symptom-directed. Surgical repair is usually indicated for the young and for individuals with cardiac arrhythmias, syncope, or sudden death. Medical treatment is reserved for those who experience dyspnea or angina, and it usually entails administration of beta blockers [5]. Since this patient experienced a cardiac arrest, he underwent surgical correction and did well postoperatively.