**CASE REPORT**

Full Recovery Two Months After Therapeutic Hypothermia Following Cardiopulmonary Resuscitation in a Patient with Out-of-hospital Cardiac Arrest

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**ABSTRACT**

Neurologic impairments are very common among patients who get a recovery of spontaneous circulation after suffering from out-of-hospital cardiac arrest. Therapeutic hypothermia is established as a standardized therapeutic strategy for those patients in whom it decreases mortality rate and improves neurologic outcome. Herein, we report a case of patient who experienced out-of-hospital cardiac arrest with ischaemic heart disease and ventricular arrhythmia and got a full recovery without any neurologic impediments 2 months after being managed with therapeutic hypothermia.

**Key words:** Hypothermia. Cardiopulmonary resuscitation. Heart arrest.

**INTRODUCTION**

The prevalence of out-of-hospital cardiac arrest (OHCA) is increasing because of increasing cardiovascular diseases or rapid aging. More patients get a recovery of spontaneous circulation (ROSC) after successful cardiopulmonary resuscitation (CPR) due to established emergency medical system and disseminated CPR training. Among these patients, only 3 – 7% of patients are able to return to their previous level of functioning, whereas almost all patients are in a poor prognosis as being in a deep comatose or persistent vegetative state.1 Based on the report that therapeutic hypothermia (TH) in comatose survivors of OHCA has been associated with decreased mortality rate and improved functional neurologic outcome in 2002,2,3 American Heart Association takes it as a major part of standard treatment in the Advanced Cardiac Life Support.4 Here, we report a rare case with OHCA due to ischaemic heart disease and ventricular arrhythmia, who showed full recovery without any neurologic impairment 2 months after receiving TH following CPR.

**CASE REPORT**

A 56-year-old man was alleged to fall backward following losing his consciousness 10 minutes after he started to run in a marathon race. Although he was soon treated by an escort ambulance, generalized cyanosis was seen in 2 minutes. After 4 minutes from a ride he arrived at a nearby hospital. There was no spontaneous respiration, no light reflex in either pupils, and no palpable pulse. Five minutes after initiating CPR, the patient got a ROSC with sinus bradycardia (30 beats/minute) and was referred to our hospital in semi-comatose state for post-resuscitation advanced care. He was a hypertensive and active smoker (40 pack-years). There was no family history of sudden cardiac death. Initial vital sign at our hospital revealed blood pressure of 100/60 mmHg, heart rate of 103 beats/minute, body temperature (BT) of 36°C, respiratory rate of 17 breaths/minute, and oxygen saturation of 90% with oxygen 7 L/min supplied via endotracheal tube. Initial electrocardiogram (ECG) showed sinus tachycardia (heart rate of 114 beats/minute) and right axis deviation (+103 degree). Cardiac profiles revealed Creatine kinase (CK) of 387 U/L, CK-MB of 4.01 µg/L, Troponin I of 0.14 ng/mL, and Myoglobin of 160.3 ng/mL. In an arterial blood gas analysis, pH was 7.333, oxygen saturation 95.2%, bicarbonate 22.9 mM, partial pressure of carbon dioxide 42.7 mmHg, and oxygen 84.3 mmHg. Computerized tomography (CT) of brain, scanned at the previous hospital, showed no abnormalities. Chest X-ray and CT at our hospital revealed atelectasis and aspiration pneumonia on both the lower lobes.

The patient was admitted to intensive care unit (ICU) immediately and got a TH using a surface cooling technique (The Arctic Sun®, Medivance Co., Louisville). Cooling pads were applied per the manufacturer’s instructions to the back, chest and thighs. The automatic mode was set to a target temperature of 33°C, and the maximum cooling rate was used. No ice bags were used. The patient was paralyzed and sedated using Vecuronium bromide (0.1 mg/kg every 2 hours) and Midazolam (0.125 mg/kg/hr initially and adjusted as needed) during the induction and maintenance period.

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The patient was maintained in the target range between 32 and 34°C. Twenty-four hours after the initiation of cooling, the maintenance period ended and rewarming was initiated. Rewarming was begun by setting the target temperature at 36 degrees and the rewarming rate at 0.3°C per hour. Once the patient’s temperature reached 36°C, the device was turned off. Cooling system was used for another 24 hours to maintain normothermia (BT between 36.5 – 37°C) and then the cooling system was removed from the patient. Due to aspiration pneumonia and profuse bronchial secretion, the patient was kept intubated for another 2 weeks and then tracheostomy was done.

At that time electroencephalogram (EEG) showed diffuse cerebral dysfunction (Figure 1A). However, overall course was favourable and, on 19th day after arrest, mechanical ventilation and sedatives were weaned successfully. One week later, he got a recovery without any neurologic impairment and moved to general ward in an alert mentality.

On the 38th day after the cardiac arrest, tracheostomy was removed and thereafter he has stable vital signs. The EEG was done twice and the last one showed normal findings (Figure 1B). Thereafter, we investigated to reveal the cause of cardiac arrest in this patient. Coronary angiography and 99m-Tc MIBI scan showed total occlusion of right coronary artery (Figure 2A) and hypoperfusion of inferobasal wall, (Figure 2B) respectively. In addition, electrophysiologic study provoked ventricular tachycardia and fibrillation (Figure 3A and 3B) which is considered as the cause. On 64 days after the event, implantable cardioverter/defibrillator was implanted. He was discharged with no neurologic complications 69 days after the incident.

**DISCUSSION**

This case implies that the optimization of all links in the chain of survival, early bystander CPR and defibrillation followed by immediate application of TH, can achieve a good-quality neurological outcome. Efforts to improve survival should focus on timely and effective delivery of interventions by bystanders and emergency medical services (EMS) personnel and immediate application of TH.

The majority (70 – 85%) of OHCA have a cardiac cause, whereas such events can occur from non-cardiac causes i.e., trauma, drowning, overdose, asphyxia, electrocution, primary respiratory arrests, and other non-cardiac etiologies. A recent meta-analysis of 79 studies involving 142,740 patients indicates that following OHCA, 7.6% of patients will survive to hospital discharge. Survival to hospital discharge was more likely among those: witnessed by a bystander, witnessed by EMS, who received bystander CPR, were found in ventricular fibrillation/ventricular tachycardia, or achieved return of spontaneous circulation. Although 53% of events were witnessed by a bystander, only 32% received bystander CPR. This means that the majority of persons who experience the OHCA event, irrespective of etiology, do not receive bystander-assisted CPR or other timely interventions that are known to improve the likelihood of survival to hospital discharge (e.g., defibrillation), although nearly half of the events are witnessed.

One of the most significant advances in CPR treatment in the past decade is TH. Post-ROSC cooling has been shown to improve neurological outcome for patients with...
OHCA. Due to its protective effect on the brain and the myocardium, TH has been extensively studied in cardiac arrest patients with coma as well as in patients presenting with acute myocardial infarction. In the setting of cardiac arrest, randomized studies have shown that TH decreases mortality and improves neurological outcomes. Guidelines have, therefore, recommended cooling (32°C – 34°C) for 12 – 24 hours in unconscious adult patients with spontaneous circulation after OHCA due to ventricular fibrillation. Recently, intra-arrest cooling during CPR is likely to protect the myocardium from reperfusion injury and enhance neurological benefits.

White et al. reported 2 patients who had a near-normal neurologic recovery after resuscitation from extreme-duration pulseless cardiac arrest followed by induced TH. One patient had pulseless cardiac arrest for 96 minutes and he not only survived but also awakened and recovered without measurable downstream neurologic deficits. The patient has returned to employment. Second patient was pulseless for 63 minutes. He collapsed at home where he was alone with his wife, who called 911 emergency services. It is highly likely that the patient was, for all practical purposes, devoid of meaningful circulation and oxygen delivery to the brain for more than 9 minutes before emergency responders arrived and escalated the resuscitation effort. After an eventful hospital course, he was discharged to home to live independently. Mild memory loss is his only measurable neurologic deficit. Clearly these 2 cases challenge the old axiom that humans cannot be expected to survive a cardiac arrest lasting more than 5 or 6 minutes, especially when one considers that the second patient of White et al. experienced either frank circulatory arrest or a combination of profoundly inadequate circulation intermixed with arrest for more than 9 minutes before the arrival of emergency personnel. In the not-too-distant past, emergency responders would not have attempted resuscitation of the second patient because of concern that, although it may have been possible to resuscitate the heart, the patient would eventually die in the hospital or be discharged from the hospital with a profound neurologic injury.

Education of public officials and community members about the importance of increasing rate of bystander CPR and promoting the use of early defibrillation by lay and professional rescuers is critical to increase survival rate of OHCA events. A longitudinal tracking of the performance allows to better understand which elements of their care are working well and which elements need improvement.

REFERENCES