

Therapeutic Hypothermia after Prolonged Cardiac Arrest: Case Report with Review of Literature

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ABSTRACT

Patients who survive cardiac arrest often develop severe neurological dysfunction due to the hypoxic brain injury and reperfusion induced cell death. Therapeutic hypothermia (TH) has become a standard therapy of cerebral protection following the successful return of spontaneous circulation in patients of out-of-hospital cardiac arrest, according to American heart association guidelines. This is a case report of a 30-year-old patient who developed in-hospital cardiac arrest and was revived after prolonged cardiopulmonary resuscitation (CPR) and also required primary angioplasty. TH was then established with local measures for 24 hours for cerebral protection. The patient was gradually and successfully weaned off from ventilator with no neurological impairment. There is an increasing evidence of TH and its protective mechanisms in patients with non-shockable arrest rhythms with particular emphasis on neurological outcomes. This article emphasizes the role of TH in every successful CPR irrespective of the cardiac rhythm.

Keywords: Asystole, Cardiopulmonary resuscitation, Neurological outcome, Return of spontaneous circulation

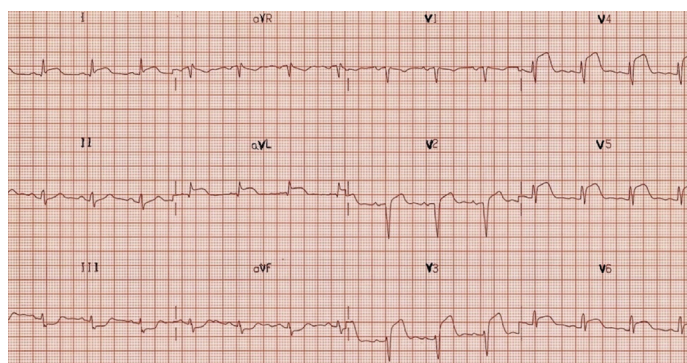
CASE REPORT

A 30-year-old male was presented to the emergency with the complaint of severe epigastric pain. In the emergency department the patient suddenly collapsed and went into an asystolic cardiac arrest. Code blue was announced and the patient was revived after a prolonged CPR for 50 minutes, without any shockable rhythm. The patient was unresponsive and had fixed dilated pupils after return of spontaneous circulation (ROSC). His electrocardiogram (ECG) showed ST elevation myocardial infarction in anterolateral leads [Table/Fig-1]. He was immediately shifted to the Cath lab for primary percutaneous transluminal coronary angioplasty (PTCA) and found to have significant (90%) occlusion of left anterior descending (LAD) coronary artery and a stent was inserted and then shifted to medical ICU. TH was started in the ICU without delay with cold saline infusion and surface cooling with water, ice packs in axilla and groin and fan to target temperature of 32°C to 34°C. With the start of TH, arterial blood pressure dropped to 90/50mmHg with heart rate 90 per minute and as per protocol adrenaline infusion was started to aim a mean arterial pressure of 80mmHg. The patient was also started on sedation with intravenous midazolam and fentanyl. TH was continued for 24 hours and then gradually re-warming was done at a rate of 0.5°C per hour. There were no major electrolyte changes, shivering or arrhythmias during TH and re-warming. Sedation was discontinued and the patient regained his consciousness with good neurological recovery. The patient was successfully weaned off from the ventilator the next day without any neurological deficit.

DISCUSSION

Therapeutic hypothermia (TH) refers to the targeted temperature management of the patient surviving cardiac arrest, in which the body temperature is lowered so as to reduce the risk of tissue and neuronal injury due to lack of blood flow initially and then from reperfusion [1,2].

The benefits and knowledge of TH in post cardiac arrest survivor are proven in the setting of critical care [1-6]. The practice of TH has remained limited despite this, especially in the developing countries, including India, where there are no established guidelines following CPR. This may be also partly due to the lack of awareness



[Table/Fig-1]: ECG showing ST elevation myocardial infarction in anterolateral leads (I, aVL, V2-6)

among the clinicians that this practice is still underutilized. Good neurological outcomes are desired, but difficult to achieve post cardiac arrest. The few hours post ROSC and also during the CPR itself is aimed at achieving this goal of cerebral preservation. The aim of presenting this case is to emphasize and encourage the timely use of the potential benefits of TH which may be applied also in non-shockable cardiac arrest with prolonged arrest period and not only in Ventricular Fibrillation (VF)/Ventricular Tachycardia (VT) [1-6]. Neurologic injury is the most common cause of death in patients with out-of-hospital cardiac arrest and contributes to the high mortality and morbidity of in-patients with cardiac arrest who regain spontaneous circulation [7,8]. When combined with a standard post-cardiac arrest care, lowering core body temperature to the range of 32°C to 34°C during the first hours after cardiac arrest improves neurologic outcome compared to not controlling body temperature [1]. A large randomized trial reports similar improvements in outcome whether the temperature is maintained at 33°C or 36°C [9]. An increase in mortality of about 20% has been associated with every hour delay in initiation of hypothermia [2,6].

The American heart association (AHA) guidelines 2010 for post cardiac arrest care has emphasized on the use of therapeutic hypothermia as a neuro-protective therapy in adult post-cardiac arrest victims whose initial cardiac rhythm is (VF/VT) and who remain comatose (GCS<8) after ROSC (class I; Level B) [10,11]. The

evidence of TH effectiveness after asystole or PEA is considered doubtful (class IIb; level C) [10-12]. Hyperthermia following cardiac arrest has been found to be associated with worse neurologic outcome [13].

There are four phases during the post-cardiac arrest: phase one includes the period immediately after ROSC up to 20 minutes. It is characterized by cardiovascular dysfunction, with a corresponding 63% mortality. The second or the intermediate phase- 20 minutes to 6-12 hours, post ROSC there is neurological and other organ damage and being the cause of morbidity at discharge. The third phase (from 6-12 hours to 72 hours) requires aggressive management as the post ROSC injury pathways are still active. Finally, the recovery phase-post 72 hours accounts for the mortality from infectious complications, and multiple organ failure.

The neuro-protective mechanism of TH is attributed to [3,4,14]:

- 6–10% reduction in cerebral metabolism per every degree Celsius of temperature drop;
- prevention from the cytotoxic cascade caused by free oxygen radicals;
- decreased apoptosis between 48 and 72 after the arrest;
- decreases cerebral inflammatory response;
- blood–brain barrier protection.

Hypothermia can be achieved by either internal or external methods. Internal methods include intravenous fluids like normal saline cooled to 4°C or the endovascular cooling catheters. The external cooling methods include ice packs placed in the groin, armpits, neck or the use of cooling blankets [3,8]. Shivering is a common response to hypothermia (most commonly occurs during induction of TH) and may cause disruption of the therapy due to heat production. Shivering may be abolished by using sedatives with or without neuromuscular blockade [3].

Hypothermia can produce several detrimental effects which can adversely affect the outcome of the patient [1,2]. TH may lead to increased blood viscosity causing deep vein thrombosis, impaired coagulation leading to bleeding diathesis, reduced cardiac output or arrhythmias, increased susceptibility to infection (especially pulmonary), cold induced diuresis leading to dyselectrolytemias (hypokalemia) and metabolic disturbances like hyperglycaemia.

Continuous temperature monitoring should be done to avoid fluctuations in temperature. Re-warming should be done slowly at a rate of about 0.5°C per hour. Research has shown that the TH is not only helpful in post-cardiac arrest, but may have a crucial role in various other cerebral insults like traumatic brain injury or stroke, where the mechanism of neuronal injury is essentially similar [15].

CONCLUSION

This case of cardiac arrest followed by therapeutic hypothermia emphasizes the advantages of TH on patient's neurological outcome. The ease of its administration and positive clinical outcome should encourage everyone to avail this modality by incorporating it in their institutional guidelines so that it may be initiated in a timely manner.

The authors also suggest that TH should be considered and initiated in every successful cardiopulmonary resuscitation irrespective of the type of arrest.

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