

# Two cases of asystolic cardiac arrests managed with therapeutic hypothermia

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## Abstract

Poor neurological outcome is a common sequel of prolonged cardiac arrest. Although Therapeutic Hypothermia (TH) for neuroprotection has been a subject for research for over Half a century, its use has been limited because of many controversies and lack of clear guidelines. However for over two decades there has been a revival of interest in mild therapeutic hypothermia (32-34°C) for neuroprotection. However its use after primary asystolic cardiac arrest has been questioned.

Herein presenting two cases of prolonged asystolic arrest (39 minutes and 25 minutes); where therapeutic hypothermia was successfully used in following prolonged cardio pulmonary resuscitation. On patients who were in deep coma after resuscitation, TH was applied for 24 hours as per institutional protocol with full neurological recovery in both the cases. Therapeutic hypothermia might have a potential role in even in non-shockable arrests and should be considered in every successful cardiopulmonary resuscitation with poor neurological status.

**Keywords:** Asystole, prolonged cardiac arrest, return of spontaneous circulation, therapeutic hypothermia, and neurological outcome

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## Introduction

Despite the reported benefit of therapeutic hypothermia (TH),<sup>[1-6]</sup> its practice in the critical care world remains limited especially in developing countries unlike the western world where it is an established intervention following Cardio-Pulmonary Resuscitation (CPR). Asian countries including Middle East and India are yet to adopt this procedure with a number needed to treat (NNT) of 6 for beneficial effects.<sup>[5,6]</sup>

Our aim of presenting these two cases is to highlight the potential benefits of TH not only in Ventricular Fibrillation (VF)/Ventricular Tachycardia (VT) but also in non-shockable cardiac arrest with prolonged arrest period.

## Case Report

### Case 1

68 years female on being discharged from ward following medical treatment for acute cholecystitis. While awaiting her discharge, she was found to be in cardiac arrest and a code blue was activated. CPR was started and the monitor showed asystole. After 6 to 7 minutes of CPR, rhythm started to change to VF with recurrent episodes of VT/VF and Torsade de pointes. Eleven DC shocks of 200 joules (Biphasic) each were given before Return of Spontaneous Circulation (ROSC) at about 39 minutes. Patient was shifted to the ICU and TH protocol was instituted. Noradrenaline infusion was used to counteract hypotension that accompanied start of the TH. In the meantime patient had two bouts of Torsade de pontes, which were reverted with magnesium loading. TH was continued for 24 hours and the patient was re-warmed slowly. There were no major electrolytes changes, shivering or arrhythmias during re-warming. Sedation was stopped; the patient opened eyes to verbal command and was successfully liberated from the ventilator with-out any neurological deficit.

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## Case 2

A 39 years male was brought to the emergency by paramedics with the complaint of severe epigastric pain and high blood pressure. In the Emergency, the patient suddenly collapsed and went into asystolic arrest. Patient was revived after 25 minutes of CPR without any shock-able rhythm. Patient was unresponsive and had fixed dilated pupils after ROSC. TH was started in the ICU without delay. With the start of TH, arterial blood pressure dropped to 100/60mmHg. As per protocol nor-adrenaline infusion was started, but further drop in blood pressure was noted. Systemic Vascular Resistance (SVR) was monitored using Vigileo-Flotrac technique that showed a high value of 3000dyne/sec/cm<sup>5</sup>. Trans Esophageal Echocardiography (TEE) revealed an enlarged but hypo-contractile heart, an abdominal ultrasound revealed a non-collapsible Inferior Vena Cava (IVC) and CVP was 20 cm. On immediately starting an inotrope (Dobutamine), dramatic improvement in hemodynamics was noticed. Further course of events were similar to the previous case with total recovery of neurological status.

## Discussion

Therapeutic hypothermia is usually indicated 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), whereas the evidence of its effectiveness after asystole or PEA is doubtful (class IIb; level C)<sup>[7]</sup> Similar findings were noted in a recent study where TH was independently associated with an improved outcome at hospital discharge in patients presenting with VF/VT with no clear effectiveness in nonshockable (Asystole/PEA) rhythm.<sup>[8]</sup>

Various methods have been in practice for TH, including external or internal cooling techniques. External cooling can be done with ice packs, ice cold saline, a cooling blanket, or a more advanced system where cool air is circulated through a tent or water filled pads.<sup>[7]</sup> Internal cooling devices are inserted into large vessels. But these advanced devices are not only costly, but also may be more time consuming for the nursing staff and might result in more temperature fluctuations.<sup>[9]</sup>

Protocol used for TH in these two cases was simple and involved the infusion of ice cold crystalloid solution at 4-degree Celsius through a rapid infusion system capable of delivering 100ml per minute. Large bore peripheral intravenous catheters were used for induction of TH (30 minutes) with a target volume of 30 ml/kg. Ice packs were applied on axilla, groin, and abdomen

along with a cooling blanket around the body. Multiple boluses of cold fluids (Ringer lactate) at a rate of 250 ml over 10 minutes were used to maintain the temperature. Standard monitoring included esophageal temperature, ECG, pulse oximetry, CVP, Cardiac output by Vigileo-Flotrac, bedside echocardiography and ultrasonographic evaluation of IVC, urine out put, blood glucose, lactate, blood gas analysis and electrolytes. Induction of TH with target temperature of 32 to 34 degree Celsius was attained within 30 min to 1 hour and the target temperature was maintained for 24 hrs. The process of rewarming was aimed at a target of 0.5 degree Celsius/hour. The target temperature was achieved at approximately 8 hrs. As per our protocol, hypotension after TH was treated with Noradrenaline infusion (unless pump failure). Sedation and neuromuscular paralysis were used to prevent shivering and continued throughout the procedure. Intravenous paracetamol 1 gm every 6hrly was used as antipyretic. Various mechanisms have been hypothesized including decreasing cerebral metabolic rate of oxygen and preventing free radical injury imposed by hypothermia. TH is not only helpful in post-cardiac arrest, but it might also have a role in various cerebral insults including traumatic brain injury or stroke, where the mechanism of neuronal injury might be essentially similar.<sup>[10]</sup>

## Conclusion

Therapeutic hypothermia for non VT/VF has not been subject to a formal randomized controlled trial. However, benefits from clinical trials of therapeutic hypothermia for various forms of neuronal insult indicate that it might also have a role in any form of cardiac arrest, since the mechanism of neurological damage is likely to be similar. We suggest that this treatment should be considered in every successful cardiopulmonary resuscitation irrespective of the type of arrest.

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