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CASE REPORT

Protective hypothermia: An old therapy with a new prospective

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ABSTRACT

Therapeutic hypothermia (protective hypothermia) has been known to have beneficial effects since ancient times but interest was renewed after two land mark publication a decade ago. The survival as well as quality of life of post cardiac arrest patients depends on neurological outcome. Mild induced hypothermia is recommended for improving the neurological status of these patients. All acute care physician, nurses and emergency medical services personals should be aware of this approach. We report a case of post cardiac arrest that displayed improved neurological status with mild therapeutic hypothermia.

Case: A young, female patient experienced perioperative cardiac arrest. Immediate resuscitation lead to return of spontaneous circulation in six minutes. Her post resuscitation Glasgow Coma score (GCS) was five. We induced therapeutic hypothermia—the patient required sedation and a chemical muscle relaxant. After 24 h we began slow rewarming. On day four, her GCS improved to 14, and she was extubated on day 6. She had mild cognitive disorder but was functionally independent. She was transferred to the ward on day 11 and subsequently discharged home.

Conclusion: Mild induced therapeutic hypothermia improves neurological status of post cardiac arrest patients; however, it had adverse effect of increased risk for infection, arrhythmia and electrolyte disorders.

Keywords: post cardiac arrest, mild induced hypothermia, infection, rewarming

INTRODUCTION

The majority of the cardiac arrest patients who experience the return of spontaneous circulation after admission to the intensive care unit die within a month due to cerebral hypoxic brain injury. Now-a-days induced mild hypothermia (protective hypothermia) is recommended therapy for these patients.

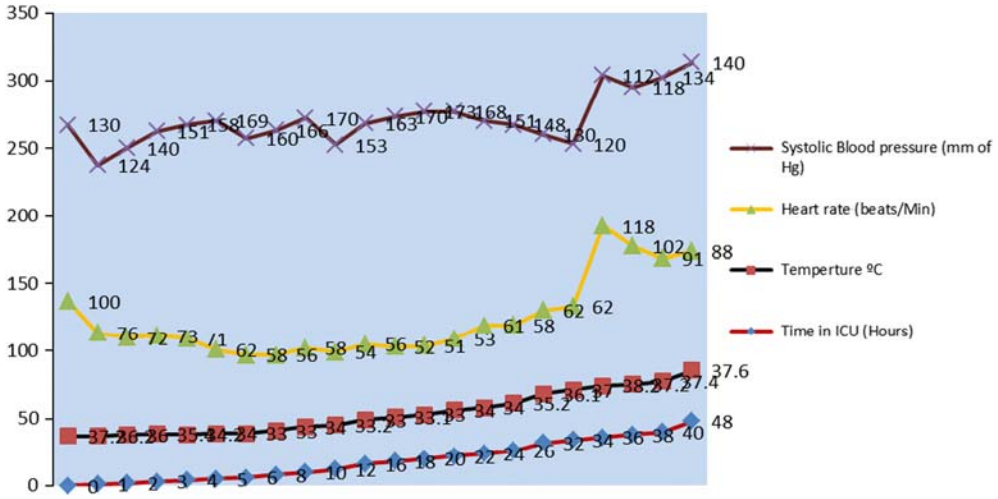

Figure 1. Induced hypothermia and hemodynamic parameters.

Beneficial effects of hypothermia have been known since the age of Hippocrates, but there has been increased interest and awareness over the last decade. One pivotal example of this kind of therapy was based on the survival of a medical resident suspended with her skies between rock and ice for 90 min. She was rescued with a core temperature of 13.7°C—cardiopulmonary bypass was used for resuscitation. She recovered fully and was able to complete her residency and practice as radiologist.[1]

According to a metanalysis by Seupeual et al. it was found that therapeutic hypothermia with the conventional cooling methods improves neurological outcome and survival of cardiac arrest patients.[2] Dumas et al. concluded that induced therapeutic hypothermia was not associated with a better outcome in cardiac asystole patients.[3] Here, we report a case of perioperative cardiac arrest had improved neurologically with therapeutic hypothermia therapy.

ILLUSTRATED CASE

A 25 year-old Sudanese female patient was scheduled for hemithryroidectomy under general anesthesia on 1st April, 2012, for follicular carcinoma of the thyroid gland. Induction of anesthesia and intubation was smooth and uneventful. After positioning patient for surgery, end-tidal carbon dioxide (Etco2) waves disappeared. By the time we discovered the nature of the problem, she experienced cardiac asystole. We immediately started cardiopulmonary resuscitation (CPR) and the surgical drapes were removed—it was found that the endotracheal tube was dislodged. Reintubated and continued CPR were performed as per acute cardiac life support guidelines. Within six minutes of CPR, spontaneous circulation returned (ROSC). Heart rate (HR) was 110/mint; blood pressure (Bp) 124/90, and oxygen saturation (Spo2) 99-100%. She was ventilated and shifted to the surgical intensive care unit (SICU) within 90 min.

On admission to SICU, her pupils were equal and reacting to light and she was hemodynamically stable without any support (heart rate 100/min; Bp 130/89 mm of Hg, and Spo2 100%). She had a cough reflex and experienced respiratory challenges resulting in pain, and her Glasgow coma score (GCS) was 5. Upon chest examination, we discovered there was bilateral equal air entry with no added sounds and her heart sounds were normal, without murmur. Her abdomen was soft with sluggish bowel sounds. She was sedated and we inserted a central venous catheter and PiCCO® arterial line. Her 12 lead ECG was normal but serum lactate levels were 5.5 mmol/Liters. It was decided to induce therapeutic hypothermia. Two liters of cold normal saline (4°C), a cooling mattress and a Haggar® cooling blanket were used. She required a chemical paralysis agent. Core body temperature was target between 33 and 35°C.
A core temperature of 33°C was achieved in four hours (Figure 1). She had slight bradycardia (HR 60/min) and hypertension (160 – 180 mm of Hg systolic) controlled by remifentanil infusion.

Induced therapeutic hypothermia was continued for 24 h, muscle relaxants were stopped, but sedation was continued and self-warming was allowed. She had a fever (38.2°C) and new infiltrates showed up on her chest X-ray. We started her on intravenous Agumentin® and paracetamol on day two. She was afebrile, hemodynaemically stable and her chest X-ray was better on day 3. Neurologically, she was trying to open eyes on verbal command and localizing to pain. Electroencephalogram (EEG) revealed hypoxic encephalopathy. She remained stable. ECG was normal, and we maintained oxygen saturation and started to wean her from the ventilator on day four. On day five, she had a brain MRI, which did not show any significant findings. She was

awake, moving all limbs but confused. She was extubated on day six. She then started on a normal diet, and physiotherapy continued. Her echocardiogram showed mild left ventricular systolic dysfunction with ejection fraction of 48% but no cardiac wall movement abnormality. She was started on ACE inhibitors. Apart from cognitive disorder, she was awake, hemodynaemiccally stable and transferred to the female surgical ward on day 11, to be followed by a neurologist, cardiologist, physiotherapist and surgeons. By day 15 she had further improved neurologically with no more cognitive dysfunction and was able to walk with support. On day 18 she was performing day-to-day activity independently, walking freely, able to read and write. The rehabilitation specialist advised her discharge on day 19.

DISCUSSION

The beneficial effects of hypothermia are known since the days of Hippocrates and Napoleon Bonaparte.[4] The first article in modern times was published in 1945, and hypothermia was extensively used during the 1950's for cerebral aneurysm surgeries. These days, severe hypothermia is used (20 – 25°C), which invites a battery of adverse effects leading to poor outcome.[4]

The Swedish medical resident's story of survival after prolonged cardiac arrest in a state of hypothermia—with improved neurological outcomes[1]—and two landmark studies showing better outcomes of the cardiac arrest patients with therapeutic mild hypothermia, has renewed interest in mild induced therapeutic hypothermia (32 – 35°C) therapy to the extent that the American heart association recommends therapeutic hypothermia as standard of care.[5] Mild therapeutic hypothermia (protective hypothermia) has been found to protect the brain and heart through various mechanisms. Hypothermia decreases the cellular metabolism—with each 1°C decrease in body temperature cellular metabolism will be reduced by 5 to 7%. Thus, mild therapeutic hypothermia causes heart and brain protection by decreasing metabolism. In post cardiac arrest patients, apoptosis is one of the prominent causes of cell death. Hypothermia helps by interrupting the apoptosis pathway.

Hypothermia also encourages the cell wall stability during ischemia thus preventing calcium influx into the cell and protecting cellular death. Therapeutic hypothermia significantly reduces the reperfusion injury and thus maintains the organ functions.[4]

There are mainly two methods of inducing therapeutic hypothermia: one is noninvasive and the other is invasive. Noninvasive methods involve body surface cooling by using cooling mattress/blankets combined with ice packs in the axilla and groin areas as well as gel pads covering the major portion of the body (Artic Sun®) and cooling cubs or cool caps. This method is more convenient and the most studied method of inducing hypothermia. However, cooling by this method is slow, involving increased incidences of skin burns, shivering and difficult assessment.[6]

Invasive cooling is administered through a catheter in the femoral vein. Cold saline is circulated through the catheter leading to rapid decrease in core body temperature. This method provides faster, precise cooling; it doesn't cause shivering and hence a chemical muscle relaxant is not required.[6]

Therapeutic induced hypothermia is not free from complications. It causes increased risk of infection, hyperglycemia, cardiac arrhythmia, electrolyte disturbance and coagulation disorders.[6] Therapeutic mild hypothermia is continued for 12 to 24 h, then the patient is allowed to slowly self rewarm. During warming, one has to be careful as, in this period, a patient can develop hypotension and electrolyte disturbance. Thus, at this stage, patients should be meticulously monitored.

Our patient had in-hospital hypoxic cardiac arrest. She was immediately resuscitated, and ROSC occurred within 6 min time. She was promptly shifted to our intensive care unit. We decided to induce therapeutic mild hypothermia, and as more potent invasive and surface devices were not available, 30 ml/kg of cold (4°C) saline infusion was started in combination with surface cooling: cooling blanket, mattress and ice packs in the axilla and groin region. Bernard et al. published that the ice cold saline is an inexpensive but significantly effective method of inducing mild therapeutic hypothermia.[7] As expected, we have to use chemical muscle relaxants to control shivering. Our patient was allowed to self warm and developed chest infection, which responded to antibiotics. Her neurological status improved significantly (from GCS 5 to 14).

Only a small number cardiac arrest patients recover from hypoxic brain insult (up to 9%).[8] Recent medical research recommends mild therapeutic hypothermia to improve neurological status of surviving cardiac arrest patients.[8]

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