Therapeutic hypothermia in PEA cardiac arrest for global and local cerebral protection: a case report and mini-review

C. Smith, A. Coleman, Y. Al-Baghdadi, M. Orlewicz

Department of Anesthesiology Wayne State/Detroit Medical Center, Harper Hospital, Detroit, Michigan 48201

Abstract

Therapeutic hypothermia (TH) has received increased attention in the last decade, although this treatment modality has been around since the 1940’s. TH is used as an adjuvant therapy for patients with refractory arrhythmias, cardiac arrest and traumatic brain injuries. We discuss the use of TH for CNS protection following pulseless electrical activity (PEA) cardiac arrest secondary to hypoxemia for a patient who had recently undergone three-vessel coronary bypass graft (CABG) surgery. The patient remained unresponsive after successful resuscitation and a head CT exhibited a new, acute left cerebral infarct. Initiation of TH was undertaken for both global protection and to minimize reperfusion injury to the new, local left infarct. Six hours after cardiac arrest the body temperature was lowered to 33° C for 24 hours and then the patient was re-warmed over the next 12 hours back to 36-37° C. Return of baseline mental status and continued improvement of hemiparesis was observed at the 36th hour. This case demonstrates the utility of TH to protect the brain both globally and locally after a PEA cardiac arrest in a patient that has undergone major cardiovascular surgery.

Keywords: therapeutic hypothermia, coronary artery bypass graft, pulseless electrical activity, ventilator associated pneumonia, cerebral protection

Case report

A 62-year-old male status post three-vessel CABG who had a complicated intensive care course, developed ventilator associated pneumonia (VAP) with difficulty managing secretions. On postoperative day 11 the patient developed a PEA cardiac arrest secondary to hypoxemia from mucus plugs. The patient was intubated and resuscitated for 5 minutes prior to restoration of a cardiac rhythm and perfusion. He was estimated to be pulseless for 5 min and had continued level of consciousness (GCS 3) following return of circulation. He was sent for a head CT scan that revealed an acute infarction of the left cerebral hemisphere with mild focal edema without herniation. For cerebral protection both globally and locally the ICU team initiated TH within six hours of arrest (Artic Sun cooling system, Medivance Inc. Louisville, CO). This consisted of cooling the patient to 33° C over 6 hours while maintaining MAP of 80-90 mmHg, and management of shivering with acetaminophen 650 mg elixir q4h, buspirone 30 mg q8h, and 0.5 mg/h lorazepam infusion. The patient was kept cool for 24 hours before being re-warmed (0.5° C/h) over the following 6 hours to a core temperature of 36-37° C. Temperature was measured both rectally and through the indwelling Foley catheter. Pupillary reflexes were checked q2h, and labs q6h (electrolytes, CBC, amylase, lipase, blood glucose, ABG, PT/INR/PTT), with continuous temperature monitoring. Initially the patient remained unresponsive upon re-warming but after 24 hours he was able to follow simple commands and tolerated weaning from the ventilator. At 36 hours post re-warming the patient was extubated. Initially he had significant right-sided weakness that dramatically improved over the next two days as he regained more function. Mental status continued to show improvement
back to baseline, and the patient was discharged home 2 weeks later.

Discussion

Although, there have been published reports where hypothermia was utilized in cardiac arrest with PEA or acute stroke [1, 2], this case describes for the first time a patient who recently had undergone major cardiac revascularization surgery and subsequently suffered an acute ischemic stroke and was managed successfully using TH to protect the brain both globally and to minimize reperfusion injury to the new, local infarcted lesion. Since the 1950s, TH has been shown to decrease mortality but when TH is used clinically, it is vital to understand the mechanisms involved [3]. There are four different stages of hypothermia: mild (34-35.9°C), moderate (32-33.9°C), moderately deep (30-31.9°C), and profound (below 30°C) [4]. Two landmark trials published in 2002 concerning hospital cardiac arrest patients who received moderate hypothermia (32-34°C) had improved functional recovery at discharge and a lower six month mortality compared to those patients who were not cooled [5, 6]. Cerebral protection is theorized to be secondary to reduced oxidative stress secondary to a decrease in excitatory neurotransmitter activity [7, 8].

Shivering, a thermoregulatory defense mechanism must be effectively managed because of the counter-productive increased metabolic rate. Common drugs to reduce thermoregulatory sensitivity include acetaminophen [9], benzodiazepines [10], propofol [11], meperidine [12], clonidine [13], muscle relaxants [14], dexametomidine [15], and buspirone [16]. Usually combinations are necessary to lower the shivering threshold [17]. The use of a benzodiazepine infusion for thermoregulation or sedation should be chosen cautiously because of the resulting accumulation and potential delayed awakening. If a benzodiazepine is utilized, lorazepam is both inexpensive and has a more predictable half-life than midazolam or diazepam, and is probably the better choice [18].

Initiation of TH is relatively contraindicated in patients with systemic infection/sepsis or who have recently underwent major surgery secondary to reduced immune function and coagulopathy [19]. Hypothermia was recently utilized in patients presenting with ventricular fibrillation who had undergone either percutaneous coronary revascularization or were hemodynamically unstable and required intra-aortic counter pulsation balloon pump and remained comatose. Using a graded neurological analysis, both groups had high grade (> 60%) neurological outcomes and an over 80% survival rate at 6 months [20].

Because our patient suffered an ischemia stroke, and his pneumonia was currently under control having been treated for approximately one week, we chose to proceed with cooling despite these theoretical risks in an effort to preserve brain function.

The hypothermic stress response mandates careful monitoring of serum glucose, which can increase [19] and potassium which may decrease with cooling [21]. Continuous ECG is necessary as arrhythmias or more commonly bradycardia may develop in temperatures at or below 33°C. PR and QT interval prolongation with widening of the QRS segment is commonly encountered as temperatures reach 33°C [22]. Patients are also at increased risk for bleeding secondary to attenuated clotting response and a decline in platelet counts with cooling [23, 24]. Increased MAP (80-100 mm Hg), especially following a stroke is important to provide cerebral perfusion. Blood pressure typically elevates with hypothermia secondary to peripheral vasoconstriction and elevated catecholamine levels and hypotension is a risk during the re-warming phase [25]. Nutrition is withheld during the cooling phase as glucose metabolism is inhibited with cooling but can resume upon completion of re-warming [26]. Occasionally, elevations in amylase or lipase are observed, but not felt to be clinically relevant unless persisting upon re-warming [27].

Commonly patients who qualify for TH have successfully been resuscitated after cardiac arrest (ventricular tachycardia or ventricular fibrillation) with return of spontaneous circulation within 1 hour, yet remain non-responsive [28]. Our case shows that patients with longer than one week post cardiac arrest ischemic stroke after major surgery with systemic infection and a non-cardiac cause for arrest may benefit from therapeutic hypothermia.

References

15. Talke P, Tayefeh F, Sessler DI, Jeffrey R, Noursalehi M, Richardson C. Dexmedetomidine does not alter the sweating threshold, but comparably and linearly decreases the vasoconstriction and shivering thresholds. Anesthesiology 1997; 87: 835-841

Hipoptermia terapeutica în stopul cardiac prin asistolie pentru protecția cerebrală globală și locală: prezentare de caz și mini-review

Rezumat

Hipoptermia terapeutica (HT) a căstigat în interes în cursul ultimei decenii, deși acest tratament a fost aplicat încă din 1940. Hipoptermia terapeutica este utilizată ca tratament adjuvant a pacienților cu aritmi refractare, oprire cardiacă și leziuni cerebrale post-traumative. Discutăm utilizarea HT pentru protecția sistemului nervos central după un stop cardiac cu asistolie cauzată de hipotermie la un pacient care a suferit un triplu by-pass coronarian. Resuscitarea cardiacă a fost eficientă, dar pacientul a rămas inconştient, iar CTcranian a evidențiat un infarcț recent la nivelul emisferii stângi a cerebelului. A fost inițiată HT atât pentru protecția globală, cât și pentru a minimaliza leziunile de reperfuzie la nivelul infarcțului cerebral. La şase ore de la stopul cardiac, temperatura corpului a fost coborâtă la 33°C pentru 24 de ore, apoi pacientul a fost reîncălzit, în următoarele 12 ore, până la 36-37°C. Restabilirea stării de conștiență și o continuă ameliorare a hemiparezei a fost observată la 36 de ore. Acest caz demonstrează eficacitatea HT pentru protecția creierului atât global cât și local după un stop cardiac prin asistolie la un pacient supus unei intervenții cardiovasculare majore.

Cuvinte cheie: hipotermie terapeutică, by-pass artro-coronarian, asistolie, pneumonie de ventilator, protecție cerebrală