

Asystolic Cardiac Arrest from Near Drowning Managed with Therapeutic Hypothermia

Daniel M. Aronovich, DO
Kirsten L. Ritchie, DO, MS
Jeffrey L. Mesuk, MD

Mount Sinai Medical Center, Department of Emergency Medicine, Miami Beach, Florida

Supervising Section Editor: Juan F. Acosta, DO, MS

Submission history: Submitted October 17, 2013; Revision received January 6, 2014; Accepted January 10, 2014

Electronically published April 4, 2014

Full text available through open access at http://escholarship.org/uc/uciem_westjem

DOI: 10.5811/westjem.2014.1.20043

[West J Emerg Med. 2014;15(4):369–371.]

An estimated 200,000 cardiac arrests occur out-of-hospital annually in the United States.^{1,2} The survival rates are 0-11% depending on the presenting rhythm.^{3,4} Following cardiac arrest, the brain can tolerate anoxia up to 2-4 minutes, upon which irreversible neuronal damage commences in the absence of re-establishment of circulation.⁵ Brain re-oxygenation with successful return of spontaneous circulation (ROSC) begins a deleterious chemical cascade that generates free radicals and other inflammatory mediators leading to devastating neurological outcomes termed post-resuscitation syndrome.⁶ The harmful effects of reperfusion injury can be mitigated with the use of therapeutic hypothermia (TH) as demonstrated in case reports and dog models from as early as the 1950s.^{7,8}

Physiological benefits of post-resuscitation therapeutic hypothermia include reduction in cerebral metabolic demands, reduction in intracranial pressure, and attenuation of an array of temperature dependent deleterious biochemical processes.⁹ Therapeutic hypothermia may be neuroprotective in many causes of brain injury. There have been limited published data using therapeutic hypothermia to treat patients resuscitated from cardiac arrest after near drowning. To date, results from available studies enrolling patients resuscitated from asystolic cardiac arrest have failed to show statistically significant treatment benefit. We describe the management of a patient with asystolic cardiac arrest after drowning in whom therapeutic hypothermia was used.

CASE REPORT

A 44 year-old Caucasian male was found floating in the sea for an unknown duration of time and was pulled out by fire rescue paramedics. He was unconscious, pulseless, and in asystole as determined by emergency medical services. Cardio-pulmonary resuscitation (CPR) was commenced immediately. Advanced cardiac life support recommendations were followed and continued for the next 25 minutes.

A total of 2 mg of epinephrine and 2 ampules of sodium bicarbonate given thru an intraosseus line resulting in return of spontaneous circulation, though with limited respiratory efforts. Subsequently, a supraglottic airway was inserted to facilitate ventilation. After a 15 minute ground transport the patient arrived in the emergency department in sinus tachycardia with a heart rate of 102 beats per minute, blood pressure of 140/90 mmHg, and oxygen saturation of 99% via supraglottic airway device with manual breaths every 6 to 8 seconds, rectal temperature was 37.1°C.

Despite return of spontaneous circulation, the patient was unresponsive with a Glasgow Coma Scale (GCS) of 3. Pupils were fixed and dilated measuring 3 mm. The supraglottic airway was replaced with an endotracheal tube. Chest radiograph demonstrated patchy opacities bilaterally, and computed tomography of the brain and c-spine were negative for acute pathology. Bloodwork was unremarkable, with the exception of a blood alcohol level of 427. At this point, a decision was made to initiate therapeutic hypothermia following the hospital protocol.

Patient was transferred to the intensive care unit (ICU) with a diagnosis of hypoxic brain injury after a near drowning. We followed a three category treatment approach to our post cardiac arrest patient. This included airway management, circulatory management, and neuroprotection. We utilized a low tidal volume, lung protective strategy for mechanical ventilation with parameters in place to maintain a PaO₂ of 60-100 mmHg and EtCO₂ of 35-40 mmHg or PaCO₂ of 40-45 mmHg. In the emergency department, we utilized end tital CO₂, however in the ICU routine arterial blood gases were utilized. From a circulatory standpoint, a mean arterial pressure of over 70 mmHg was maintained via intravenous fluids with a plan for vasopressor use on a as needed basis. The neuroprotective measures that were begun in the emergency department continued in the ICU. This included our hospital's therapeutic hypothermia protocol

which includes a sedation and shivering prevention protocol via fentanyl, versed, and cisatracurium besilate as necessary. Patient was cooled rapidly using an external, commercially available cooling device to a target temperature of 34°C. Active cooling was stopped after 24 hours and the patient was allowed to passively re-warm. Pupils became responsive to light within 8 hours and reached normal size and reaction within 40 hours. GCS improved by day 3 to the point of spontaneous eye opening and obeying commands. A transthoracic echocardiogram showed an estimated ejection fraction of 55 percent without any structural heart disease. Patient continued to improve significantly over the course of his hospital stay. Patient was subsequently transferred on day 28 of his hospitalization to a rehabilitation facility with minimal cognitive deficits and mild upper extremity weakness bilaterally with a cerebral performance category of 1.

DISCUSSION

The International Liaison Committee for Resuscitation (ILCOR) has suggested that cooling may be beneficial for patients suffering cardiac arrest due to reasons other than an initial rhythm of ventricular fibrillation.¹²

Following ILCOR advisory statement of 2002, American Heart Association (AHA) incorporated therapeutic hypothermia in its 2005 recommendations for cardiac arrest patients as a class 2A recommendation. The AHA has since made therapeutic hypothermia a class 1 recommendation in cardiac arrest with initial rhythm of ventricular fibrillation and pulseless ventricular tachycardia and a class 2B recommendation for nonshockable rhythms. However, therapeutic hypodumas

thermia for nonshockable rhythms has not been subject to a formal randomized controlled trial, and we cannot plausibly prove that hypothermia contributed to the good outcome in this case. However, clinical trials of the use of therapeutic hypothermia for shockable rhythms in cardiac arrest, where the mechanism of neurological damage is likely to be similar, suggest that this treatment may have a role.^{10,11} Dumas F et al¹³ conducted an observational study to assess the effectiveness of therapeutic hypothermia in nonshockable rhythms, however failed to show any difference in outcome. Utilizing the available literature on the subject, we determined it reasonable to apply this intervention in our patient. Our goal was to start this therapy as soon as possible, given the results shown by Mooney et al¹⁴, which demonstrated an increase in mortality of 20% for each hour that therapeutic hypothermia was delayed. In addition to therapeutic hypothermia, a more global approach to the management of our post cardiac arrest patient was followed. Stub D et al¹⁵ recommended a three-category approach to the post cardiac arrest patient and condition known as post cardiac arrest syndrome. The three treatment categories include oxygenation/ventilation, circulatory support, and neuroprotection. These recommendations were followed and included avoidance of hyperoxia and hyperventilation, a lung protective ventilation strategy, a target mean arterial pressure

between 70-100 mmHg as well as therapeutic hypothermia. Our patient was relatively young, without significant comorbidities and he was resuscitated after near drowning while severely intoxicated with alcohol. Either of these factors may actually have played a survival benefit role while he was resuscitated from asystolic cardiac arrest. Alcohol in particular is an interesting potential confounder as a neuroprotectant although a literature review revealed only data from rat models in ischemic stroke and traumatic brain injury.^{15,16}

CONCLUSION

Given the outcome of this case, our experience supports the role of therapeutic hypothermia in the management strategy for patients subjected to hypoxic brain injury following resuscitation from asystolic cardiac arrest. In particular, this case supports the application of TH for patients resuscitated from asystole after a near drowning event as well as a more global approach to the management of the post cardiac arrest patient focusing on oxygenation/ventilation, circulatory support and neuroprotection.

Address for Correspondence: Daniel M. Aronovich, DO. Mount Sinai Medical Center, Department of Emergency Medicine, 4300 Alton Rd., Miami Beach, FL 33140. Email: danaronuf@gmail.com.

Conflicts of Interest: By the WestJEM article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

REFERENCES

1. Rea TD, Pearce RM, Raghunathan TE, et al. Incidence of out-of-hospital cardiac arrest. *AM J Cardiol* 2004;93:1455-1460.
2. De Vreede-Swagmakers JJ, Gorgels AP, Dubois-Arbouw WI, et al. Out-of-hospital cardiac arrest in the 1990s; a population-based study in the Maastricht area on incidence, characteristics and survival. *J Am Coll Cardiol* 1997;30:1500-1505.
3. Engdahl J, Bang A, Lindquist J, et al. Can we define patients with no and those with some chance of survival when found in asystole out of hospital? *Am J Cardiol* 2000;86:610-614.
4. Gray WA, Capone RJ, Most AS. Unsuccessful emergency medical resuscitation-are continued efforts in the emergency department justified? *N Engl J Med* 1991;325:1393-1398.
5. Cole SL, Cordal E. Four-minute limit for cardiac resuscitation. *J Am Med Assoc* 1956;161:1454-1458.
6. Nolan JP, Neumar RW, Adrie C, et al. Post-cardiac arrest syndrome: epidemiology, pathophysiology, treatment, and prognostication: a scientific statement from the International Liaison Committee on Resuscitation: the American Heart Association Emergency Cardiovascular Care Committee: the Council on Cardiovascular

- Surgery and Anesthesia; the Council on Cardiopulmonary, Perioperative and Critical Care; the Council on Clinical Cardiology; the Council on Stroke (Part 1). *Int Emerg Nurs* 2009;17:203-225.
7. Safar P. Resuscitation from clinical death: pathophysiologic limits and therapeutic potentials. *Crit Care med* 1988;16:923-941.
 8. Bigelow WG, Lindsay K, Greenwood WF. Hypothermia; Its possible role in cardiac surgery: an investigation of factors governing survival in dogs at low body temperatures. *Ann Surg* 1950;132:849-866.
 9. Polderman KH. Induced hypothermia for neuroprotection: understanding the underlying mechanisms. In *Yearbook of Intensive Care and Emergency Medicine*. Springer; 2006.
 10. The hypothermia after cardiac arrest study group: Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. *N Eng J Med*. 2002;(346):549-556
 11. Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Eng J Med*. 2002;(346):557-563.
 12. Nolan JP, Morley PT, Hoek TL, et al. Advancement Life Support Task Force of the International Liaison Committee on Resuscitation. Therapeutic hypothermia after cardiac arrest: An advisory statement by the Advanced Life Support Task Force of the international Liaison Committee on Resuscitation. *Resuscitation* 2003;(57):231-235.
 13. Dumas F, Grimaldi D, Zuber B, et al. Is hypothermia after cardiac arrest effective in both shockable and nonshockable patients? *Circulation*, 2011;123(8):877-886.
 14. Mooney MR, Unger BT, Boland LL. Therapeutic hypothermia after out-of-hospital cardiac arrest: evaluation of a regional system to increase access to cooling. *Circulation*. 124 2011:206-214.
 15. Zeng X, Asmaro K, Ren C, et al. Acute ethanol treatment reduces blood-brain barrier dysfunction following ischemia/reperfusion injury. *Brain Res*. 2012:127-133.
 16. Wang T, Chou DY, Ding JY, et al. Reduction of brain edema and expression of aquaporins with acute ethanol treatment after traumatic brain injury: Laboratory investigation. *J Neuro* 118.2 2013:390-396.
 17. Stub D, Bernard S, Duffy SJ, et al. Post cardiac arrest syndrome: a review of therapeutic strategies. *Circulation*. 123 2011:1428-1435.