An estimated 200,000 cardiac arrests occur out-of-hospital annually in the United States. The survival rates are 0-11% depending on the presenting rhythm. 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. 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. 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.

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. 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.
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. 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 hypothermia 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. Dumas F et al 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 al, 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 al 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.

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.

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