

Full neurological recovery following a hypothermic, near-drowning cardiac arrest with a 34 minute submersion time: A case report

Daniel Eden¹

Abstract

Hypothermia increases the ischemic tolerance of the brain and can lead to a remarkable recovery in prolonged cardiac arrest. Neurological outcomes can be very favorable, particularly if cardiac arrest precedes hypothermia or if the onset of hypothermia following cardiac arrest is rapid. Case reports of good neurological recovery following up to 7 h submersion and core temperatures as low as 13.7°C can be found. Our case involved a 43-year-old male who presented following a collapse and out of hospital cardiac arrest. A 34 min period of face-down submersion in a canal on a cold February evening was followed by a brief period of cardiopulmonary resuscitation on extrication. He was transferred to our emergency department with a core temperature of 26°C. He went onto make a full neurological recovery. The UK's temperate climate means that case reports of prolonged hypothermic cardiac arrest are rare. While this gentleman had many favorable circumstances to his predicament, including age, cardiovascular fitness and rapid cooling, prompt and effective resuscitation, and a controlled warming regime would also have contributed.

Keywords: Hypothermia cardiac arrest, neurological recovery

Introduction

Accidental hypothermia is defined as an unintentional drop in core body temperature below 35°C, with a hypothermic cardiac arrest being the cessation of circulation as a result of hypothermia [1]. Treatment options can broadly be classified as external passive or active, or internal active. Choice of rewarming therapy is usually based on severity and equipment availability, including forced warmed air blankets, administration of warm IV fluids, and warm water immersion. For the critically unwell, more invasive methods such as pleural/peritoneal lavage or extra-corporeal circuits may also be considered. It is common knowledge that hypothermia is neuroprotective and increases the ischemic tolerance of the brain [2]. Neurological outcomes in hypothermic patients with witnessed cardiac arrest can be very favorable [3]. Literature is scattered with

remarkable outcomes in severely hypothermic patients undergoing prolonged submersion, with reports of up to 7 h and with core body temperatures as low as 13.7°C [4, 5, 6]. Due to the moderate climate usually afforded to the UK, cases here are conspicuous by their absence. February 2018 saw a period of unseasonably cold weather across much of the UK with many parts of The Midlands experiencing sub-zero temperatures.

Case Report

Our case is a 43-year-old male who presented to our emergency department following an out of hospital cardiac arrest. A keen runner with no significant medical history, he was seen running alongside a canal towpath in Stourport before sitting on a bench holding his head. Witnesses heard him falling into the canal and was found floating face down in the water. It was a cold February night with sub-zero temperatures

across most of the Midlands. Due to difficulty in extrication, it took an estimated 34 min before medical services could perform an initial assessment. He was found to be in pulseless electrical activity cardiac arrest and received 2 cycles of cardiopulmonary resuscitation with 1 dose of adrenaline before the return of spontaneous circulation. He was intubated at the scene and transferred to our emergency department. On arrival, he had a heart rate of 20–30 with frequent ventricular ectopics, an adequate blood pressure, a GCS of 3 and a core temperature of 26°C. His admission arterial blood gas is shown in Fig. 1:

pH	6.88
pCO ₂	10.9 kPa
pO ₂	52.5 kPa (FiO ₂ 1.0)
Lactate	10.2 mmol/l
K ⁺	0.9 mmol/l
Na ⁺	128 mmol/l

Figure 1: Admission arterial blood gas. Initial management included suctioning of copious endotracheal secretions (a combination of pulmonary edema and canal water), administration of glycopyrrolate, fluid resuscitation, and commencement of propofol sedation. Warming was

¹Department of Speciality Registrar (ST5 Anaesthetics) - Worcestershire Royal Hospital, Worcester, United Kingdom

Address of Correspondence

Dr. Daniel Eden,
Specialty Registrar (ST5 Anaesthetics) - Worcestershire Royal Hospital,
Worcester, United Kingdom.
Email: daniel.eden@doctors.net.uk



Dr. Daniel Eden

© 2018 by Journal of Anaesthesia and Critical Care Case Reports | Available on www.jaccr.com |

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

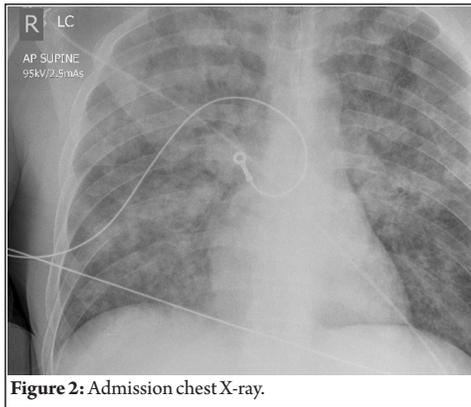


Figure 2: Admission chest X-ray.

commenced with heated intravenous fluids, bladder irrigation, and a forced air blanket. There was no suggestion on the preliminary electrocardiography (ECG) to suggest a primary cardiac event and no indication for a primary percutaneous coronary intervention. An urgent computed tomography (CT) head was requested, which was unremarkable. The only other evident injuries were the loss of two upper incisors. The patient was, therefore, transferred to the critical care unit for post-resuscitation care. Here standard neuroprotective measures and controlled active warming at a target rate of 1–2°C per hour were instigated. This was continued for around 8 h until a temperature of 35°C was achieved. A loading dose of levetiracetam was administered. No treatment was required for the severe hypokalemia as this increased along with core body temperature. Our target PaCO₂ was 4.5–5 kPa, however, despite administering neuromuscular blockade and optimizing ventilator settings, gas exchange became increasingly challenging over the first night. A chest X-ray showed widespread pulmonary infiltrates in both lung fields, and CT neck imaging reported consolidation in both lung apices. Bronchoscopy findings supported a diagnosis of acute respiratory distress syndrome and pulmonary edema. A representative arterial blood gas from this period showed a pH of 7.03, pCO₂ of 11.3 kPa, and a pO₂ of 15.3 kPa (on 100% oxygen). Referral to our local ECMO center was considered, but the unknown neurological outcome at the time contraindicated this. During the first 12 h, we were unable to achieve a pCO₂ of <9 kPa. Gas exchange eventually improved through a combination of airway pressure release ventilation, furosemide, and antibiotics. A bedside echocardiogram was unremarkable as 12 lead ECGs, laboratory

cardiac markers, and an angiogram. We extubated on day 7. Initial neurological examination was grossly normal with only moderate global weakness, reduced bilateral coordination, and a mild degree of delirium. These improved over the next few days and the patient went on to make a full neurological recovery. On day 19, he was discharged to his local hospital in Cornwall.

Discussion

Cause

The cause of our patient's collapse and subsequent cardiac arrest is uncertain. A primary cardiac event or arrhythmia is most likely; however, all subsequent cardiac investigations were normal. A primary neurological event is less likely given a normal CT head, but an epileptogenic cause cannot be ruled out. Furthermore, unclear is whether he suffered a cardiac arrest and then subsequent immersion, or arrested as a result of submersion hypoxia following a collapse. On balance hypoxia secondary to drowning is less likely as cardiac arrest preceded by asphyxiation is generally associated with a poor outcome (hypothermia does not have the same protective effect [7]). Therefore, we can probably conclude that cardiac arrest was followed by subsequent severe accidental hypothermia associated with immersion.

Blood gas analysis

Blood gas analysis in the hypothermic patient is complicated by the temperature correction employed by most blood gas analysis machines, where the sample is warmed to 37°C. While this allows a standardized comparison between different samples, it does not reflect the *in vivo* values. At lower temperatures gas solubility increases and while recorection to values at actual body temperature occurs in most blood gas analysis machines, in reality, we do not know what normal values at these low temperatures should be. Hypothermic hypokalemia is usually linked to an intracellular shift rather than a net loss [8]. A systematic review of hypokalemia associated with hypothermia by Buse et al. [8] found that this was likely multi-factorial as a combination of Na/K/ATPase upregulation, β-adrenergic stimulation, membrane stabilization, and pH.

Freshwater versus saltwater submersion

The pathophysiology of fresh water

drowning differs significantly from saltwater drowning. Freshwater is hypotonic and therefore enters the bloodstream through unprotected cells to attempt ionic equilibrium. As skin is keratinized, exposed lung capillaries act as this conduit diluting the blood with the potential for significant hemolysis and hyponatremia. This sodium dilution may also cause significant renal damage in the following hours. These features were not seen in this case. This differs from saltwater submersion since the material is hypertonic and therefore does not cross into the lung tissue. Instead, it forms a physical barrier to gas exchange, which will not improve until that water is removed from the lungs.

Pulmonary injury

An acute lung injury commonly follows drowning regardless of whether the inhaled liquid is fresh or salt water. Pathophysiology includes direct surfactant washout leading to alveolar collapse and a direct hypotonic toxic effect (in cases of fresh water) leading to interstitial and alveolar edema [9]. This may be exacerbated by bronchospasm and alveolar rupture causing acute emphysema, particularly if ventilation is proving difficult. Negative pressure pulmonary edema may contribute if airway obstruction occurred and particulate matter may cause bronchiole obstruction. Clinically, these injuries may manifest as hypoxia due to V/Q mismatch or hypercarbia due to poor ventilation and reduced lung compliance. Further down the line infective complications may occur as a result of contaminated water and aspiration.

Positive predictors

This case has several features that contribute to a remarkable recovery. His pre-morbid state was very favorable being young, previously fit, and well and having an excellent baseline cardiovascular fitness (being a regular runner). Being cooled by very cold water at high cooling rates is also believed to be beneficial [10]. Freshwater submersion increases this cooling rate as hypotonic water enters the bloodstream through lung capillaries. If this occurred in salt water, the temperature effects would be limited to exposed surfaces. If asphyxia is caused by immersion before cardiac arrest, aspiration of cold water may induce rapid cerebral hypothermia and therefore be more protective than other forms of asphyxia [1]. Likewise quality of resuscitation, in-hospital

management and limiting the rewarming rate may all help reduce complications [11, 12]. Bradycardia may contribute to improved outcomes in pediatric patients [7], and this is likely to apply to adults as well. Low potassium is a good outcome predictor [13].

Negative predictors

Conversely, excessive hyperkalemia and preceding hypoxia are known predictors of high mortality [10]. A high presenting K⁺ is indicative of a poor prognosis [13] and is likely a reflection of underlying cell damage. Low initial arterial oxygen tension and indoor cooling are associated with worse

outcomes [14], as is major trauma, severe underlying disease, increased submersion time, increased duration of resuscitation, and higher water temperatures [15]. Other physiological parameters such as plasma pH and core temperature will also contribute. While hypothermia has a protective effect on vital organs; it can also cause complications in itself such as coagulopathy, renal impairment, and metabolic derangements.

Conclusion

Hypothermia increases the ischemic tolerance of the brain and can lead to remarkable outcomes in prolonged cardiac

arrest. Outcome can be improved by rapid hypothermia, avoiding asphyxia before cardiac arrest, quality of resuscitation, and steady rewarming rates. Despite an extremely low admitting potassium, this did not require treatment and normalized of its own accord as the patient was warmed. Our case experienced submersion for 34 min with a core temperature of 26°C on extraction, going on to make a full neurological recovery.

References

- Hilmo J, Naesheim T, Gilbert M. Nobody is dead until warm and dead: Prolonged resuscitation is warranted in arrested hypothermic victims also in remote areas—a retrospective study from Northern Norway. *Resuscitation* 2014;85:1204-11.
- Tahsili-Fahadan P, Farrokhi S, Geocadin RG. Hypothermia and brain inflammation after cardiac arrest. *Brain Circ* 2018;4:1-3.
- Wollenek G, Honarwar N, Golej J, Marx M. Cold water submersion and cardiac arrest in treatment of severe hypothermia with cardiopulmonary bypass. *Resuscitation* 2002;52:255-63.
- Gilbert M, Busund R, Skagseth A, Nilsen PA, Solbø JP. Resuscitation from accidental hypothermia of 13.7 degrees C with circulatory arrest. *Lancet* 2000;355:375-6.
- Perk L, Borger van de Burg F, Berendsen HH, van't Wout JW. Full recovery after 45 min accidental submersion. *Intensive Care Med* 2002;28:524.
- Friberg H, Rundgren M. Submersion, accidental hypothermia and cardiac arrest, mechanical chest compressions as a bridge to final treatment: A case report. *Scand J Trauma Resusc Emerg Med* 2009;17:7.
- Eich C, Bräuer A, Timmermann A, Schwarz SK, Russo SG, Neubert K, et al. Outcome of 12 drowned children with attempted resuscitation on cardiopulmonary bypass: An analysis of variables based on the "Utstein style for drowning". *Resuscitation* 2007;75:42-52.
- Buse S, Blancher M, Viglino D, Pasquier M, Maignan M, Bouzat P, et al. The impact of hypothermia on serum potassium concentration: A systematic review. *Resuscitation* 2017;118:35-42.
- Carter E, Sinclair R. Drowning. Continuing education in anaesthesia. *Crit Care Pain* 2011;11:210-3.
- Mair P, Kornberger E, Furtwaengler W, Balogh D, Antretter H. Prognostic markers in patients with severe accidental hypothermia and cardiocirculatory arrest. *Resuscitation* 1994;27:47-54.
- Semple-Hess J, Campwala R. Pediatric submersion injuries: Emergency care and resuscitation. *Pediatr Emerg Med Pract* 2014;11:1-21.
- Zuckerbraun N, Saladino R. Pediatric drowning: Current management strategies for immediate care. *Clin Paediatr Emerg Med* 2005;6:49-56.
- Bender PR, Debehne DJ, Swart GL, Hall KN. Serum potassium concentration as a predictor of resuscitation outcome in hypothermic cardiac arrest. *Wilderness Environ Med* 1995;6:273-82.
- Farstad M, Andersen KS, Koller ME, Grong K, Segadal L, Husby P, et al. Rewarming from accidental hypothermia by extracorporeal circulation. A retrospective study. *Eur J Cardiothorac Surg* 2001;20:58-64.
- Kriz D, Piantino J, Fields D, Williams C. Pediatric hypothermic submersion injury and protective factors associated with optimal outcome: A case report and literature review. *Children (Basel)* 2017;5:4.

Conflict of Interest: Nil
Source of Support: None

How to Cite this Article

Eden D. Full Neurological Recovery Following a Hypothermic, Near-drowning, and Cardiac Arrest with a 34-min Submersion Time: A Case Report. *Journal of Anaesthesia and Critical Care Case Reports* Sep-Dec 2018;4(3):30-32.