CASE REPORT

Accidental hypothermic cardiac arrest and rapid mediastinal warming with pleural lavage: a survivor after 3.5 hours of manual CPR

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SUMMARY

A 30-year-old man suffered post-traumatic hypothermic cardiac arrest. On arrival in the emergency department, rectal core temperature was 23°C. Manual cardiopulmonary resuscitation (CPR) was continued as no mechanical chest compression device was available, and active and passive rewarming was undertaken. Bilateral thoracostomies confirmed good lung inflation. Defibrillation and intravenous epinephrine were discontinued until core temperature was elevated above 30°C. Extracorporeal rewarming was unavailable. When no increase in rectal temperature was achieved after 90 min, an alternative oesophageal probe confirmed mediastinal temperature as 23°C. Bilateral chest drain insertion, followed by microwave-heated saline pleural lavage, rapidly raised the oesophageal temperature above 30°C with subsequent successful defibrillation, initially to pulseless electrical activity and finally return of spontaneous circulation 3.5 hours after the commencement of CPR. The patient recovered fully and was discharged without neurological deficit. Rapid mediastinal warming with pleural lavage should be considered in units with no access to extracorporeal rewarming service.

BACKGROUND

This is a case report highlighting the efficacy of bilateral pleural lavage, as part of active rewarming, in the successful resuscitation of a 30-year-old man with ventricular fibrillation (VF) cardiac arrest, secondary to severe accidental hypothermia, after 3.5 hours of manual cardiopulmonary resuscitation (CPR). The case emphasises that medical teams in peripheral small hospitals, with no access to mechanical chest compression devices or extracorporeal rewarming equipment and expertise, can successfully resuscitate patients with severe accidental hypothermic cardiac arrest if they have no option to transfer the patient to a higher level of care.

CASE PRESENTATION

A 30-year-old male was brought to the emergency department (ED) by ambulance in VF cardiac arrest. CPR was in progress with a supraglottic airway in situ, and the patient had received multiple direct current (DC) shocks and intraosseous (IO) doses of epinephrine and amiodarone, from the ambulance service.

The patient was found early in the morning, unconscious/unresponsive in a 3 m deep road-side ditch, semisubmerged up to his waist in a stream with no upper body clothing, a short distance (30 m) from the scene of a single vehicle crash, in a rural location. It appeared that the patient had self-extricated from the vehicle and fallen into the ditch/stream in the dark. He may have been trapped in the ditch for some hours as the estimated time of the vehicle crash was up to 3 hours prior to the patient's discovery. A passer-by heard noises, which led him and police officers to the patient's location. Police officers commenced rescue efforts, and extrication from the ditch by emergency services was complex. The time of onset of cardiac arrest is unknown but appears to have occurred during the extrication process. VF cardiac arrest was confirmed and CPR commenced at the scene.

On arrival in ED, initial examination demonstrated multiple abrasions and contusions over the patient's torso and limbs and initial impressions were that the patient had possibly sustained a traumatic cardiac arrest. Spinal precautions were maintained. He was cold and initial rectal core temperature was recorded at 23°C. Manual CPR was continued along with regular DC shocks as active rewarming measures were instituted.

INVESTIGATIONS

Routine biochemistry and haematology tests were performed, and multiple venous blood gas samples monitored changes in pH, electrolytes and lactate, among others. Initial serum potassium on venous blood gas analysis was 4.4 mmol. There was no access to Focused Assessment with Sonography for Trauma (FAST scan) during the resuscitation to more definitively rule out cardiac tamponade or internal haemorrhage. Gantry and portable X-ray were not available, and therefore, initial trauma series X-rays of chest, pelvis and cervical spine were performed after return of spontaneous circulation (ROSC), when moving the patient to radiology was an option. Post-ROSC CT brain, spine, thorax, abdomen and pelvis were also performed along with plain X-rays of limbs.

DIFFERENTIAL DIAGNOSIS

Our differential diagnoses included, principally, hypothermic cardiac arrest and/or traumatic hypovolaemic cardiac arrest, and we focused our

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treatment in these areas. Other reversible causes of cardiac arrest were addressed by working through the '4Hs (hypoxia, hypovolaemia, hypothermia, hypokalaemia, etc) and 4Ts (tension pneumothorax, cardiac tamponade, drug toxicity, thromboembolic event)' while maintaining good-quality continuous CPR and rewarming the patient's core.

TREATMENT

The patient's oxygenation was maximised with endotracheal intubation and warmed humidified oxygen positive-pressure ventilation. Bilateral thoracostomies were performed to treat potential tension pneumothorax, to ensure good lung inflation and to exclude haemothorax; however, chest tubes were not yet inserted. There had been no penetrating chest injury. There was no external haemorrhage. There was no clinical evidence of significant long bone fractures or fractured pelvis, and abdominal examination showed no distension or other signs of overt intra-abdominal bleeding. FAST scan was not available. Hypovolaemia and hypothermia were vigorously addressed. Bilateral upper limb intravenous cannulae were inserted along with a second tibial IO needle. Warmed IO and intravenous fluid infusions were commenced. Due to the potential for internal haemorrhage, 4 units of warmed packed red cells were infused through a rapid infuser. Right subclavian vein access and femoral artery access were performed when resuscitation activities allowed. A 3-way urethral catheter was inserted, and warmed bladder lavage was commenced. An external warm air heating blanket was applied, along with 'heat packs' (zip-top freezer bags containing warm-water-soaked gauze) applied to the patient's head/neck, axillae and groins. Approximately 45 min after arrival, the rectal core temperature remained at 23°C, and DC shocks and intravenous epinephrine were stopped until the core temperature could be elevated above 30°C. Manual CPR and positive-pressure ventilation continued. There was no mechanical chest compression device available. Thoracotomy and open cardiac massage were not considered as closed CPR appeared effective with good femoral artery compression impulses palpable. Venous blood gas analysis ruled out major electrolyte and glycaemic disturbances.

Potential drug toxicity was addressed with intravenous naloxone and flumazenil. The presenting history and collateral medical history from family members suggested that a thromboembolic event was unlikely. The patient received intravenous magnesium sulfate along with intravenous antibiotics and sodium bicarbonate. Discussions with the regional specialist centre about extracorporeal rewarming culminated in a decision not to do the 30 km road transfer and to continue resuscitation locally.

Despite ongoing CPR and rewarming, the rectal temperature probe never registered much above 23°C. A second temperature probe was placed in the patient's oesophagus, which confirmed the core temperature at 23°C. Approximately 1.5 hours after arrival, bilateral chest drains were inserted through the existing thoracotomy sites and pleural lavage/mediastinal rewarming was commenced with microwave-heated normal saline (approximately 40°C). Multi-lumen intercostal drains were not available. Heated saline was flushed into the pleural spaces bilaterally using 60 mL catheter syringes and allowed to drain freely after every 300–400 mL, so that a constant flow of warm saline was maintained. Core temperature on the oesophageal probe rose rapidly from 23°C to 30°C over approximately 30 min. The patient was then defibrillated successfully into a slow bradycardic pulseless electrical activity rhythm initially. This alternated with intermittent VF rhythm requiring continued CPR and DC shocks over

a 30 min period. This was followed by an intermittent return of a 'brittle' bradycardic spontaneous circulation over a further 30 min, requiring intravenous atropine boluses and norepinephrine infusion. Consistent stabilised ROSC was achieved approximately 3 hours after arrival in ED, and no further chest compressions were required. Overall time from start of CPR prehospital to consistent/stabilised ROSC and end of CPR totalled approximately 3.5 hours.

Pleural lavage was stopped, and the chest drains were allowed to drain to underwater seal devices. Other ongoing rewarming measures were continued.

OUTCOME AND FOLLOW-UP

The patient underwent CT imaging of head, which demonstrated a right temporal lobe haemorrhagic contusion, and CT spine/thorax/abdomen and pelvis showed no significant injuries. Plain film radiology demonstrated no abnormalities. He was transferred to the intensive care unit (ICU). Active rewarming was stopped at core temperature of 35°C. Rhabdomyolysis developed with renal failure over the first 24 hours, requiring transfer to a regional ICU to access haemo-dialysis. The patient spent 6 days on the regional ICU. He was extubated on the third day postinjury. He continued on intermittent dialysis following transfer to the ward. Urine output returned approximately 2 weeks after initial presentation, and renal function improved steadily. Multiple occupational therapy and physiotherapy assessments detected no cognitive or neuromuscular limitations apart from those caused by soft tissue injury to limbs. The patient was discharged from hospital 3 weeks after the original resuscitation. He was initially reviewed frequently in the renal day unit. Renal function normalised over 6–8 weeks; however, night sweats prompted two further short admissions at 3 and 4 weeks postdischarge when microbiology and transoesophageal echocardiogram results prompted treatment for suspected infective endocarditis involving the aortic valve. The patient underwent 6 weeks of outpatient intravenous antibiotic treatment, and follow-up clinical and ECHO assessments confirmed resolution. He was last reviewed in outpatients, 16 weeks postincident. He returned to his life and family with no apparent cognitive or neuromuscular deficits. He recommenced his preincident job 24 weeks after the initial resuscitation. The patient has healed residual burn scars at the site of the left chest defibrillation pad and the adjacent left triceps region.

DISCUSSION

The European Resuscitation Council (ERC) Guidelines on the management of patients with accidental hypothermic cardiac arrest are clear and comprehensive, and the 2010 document was current and guided this resuscitation.^{1 2} The initial treatment phase/clinical decisions in ED were influenced by the strong suspicion of a traumatic cardiac arrest from the prehospital mechanism/history of events and the clinical findings on arrival. Passive and active rewarming techniques are well described, and the literature records many cases of survival following prolonged CPR for accidental hypothermic cardiac arrest.^{3–18} Extracorporeal rewarming and extracorporeal membrane oxygenation have been particularly successful when available to resuscitation teams.^{3–11} The absence of prearrest hypoxia improves likelihood of survival.^{3 5 6} Timing of the cardiac arrest in relation to rescue attempts in such cases appears important.^{6 9} It is possible that the subject of this case report suffered cardiac arrest during rescue movements, and this may have been in his favour. Initial serum potassium levels may also have a bearing on survival, and this

patient's normal potassium (4.4 mmol), on arrival, may also have been a pointer to possible ultimate success in his resuscitation.^{3 10}

The author believes that, in this case, there were three key factors that led to the successful outcome.

First, adherence to the traditional principle, that in these circumstances, 'no one is dead until they are warm and dead', guided the team's decisions and kept everyone focused on the main goals of maintaining good-quality CPR while trying everything possible to raise the patient's core temperature above 30°C.^{1 2 10} This was in spite of not having a mechanical chest compression device and being unable to access extracorporeal rewarming.

Second, a large team of staff members from varied disciplines, apart from the ED/cardiac arrest team, came together in the resuscitation room to help in the initial 3-hour prolonged in-hospital resuscitation efforts. High-quality teamwork in these circumstances is important.¹¹ The team included medical and nursing staff, a medical student, attendants and hospital management team members doing chest compressions and kitchen and attendant staff microwaving saline for the pleural lavage. Rotation of chest compressors was paramount to maintain continuous uninterrupted CPR. We had no need to employ intermittent CPR as a result.¹³ Microwave ovens at three locations in the hospital and runners were needed to keep a steady flow of warmed saline for the pleural lavage. Heating fluids for parenteral use using microwave ovens is previously described.^{19 20}

Thirdly, and the decisive 'game-changer' in this case, pleural lavage with warmed saline rapidly rose the temperature of the patient's mediastinum from 23°C to over 30°C (oesophageal temperature probe) over a short time frame and allowed successful defibrillation and eventual ROSC. Pleural lavage is well documented as a successful rewarming technique in severe accidental hypothermic resuscitation.^{14–17} Despite all of the other modalities of passive and active external/internal rewarming used, we did not see any elevation in core temperature until pleural lavage was commenced. The decision to place an oesophageal temperature probe to check a second core temperature site was prompted as the rectal probe was showing no appreciable increase in core temperature despite significant active rewarming attempts. Of note, the ERC 2015 Guidelines de-emphasise the use of rectal temperature probes in these circumstances.² The temperature of the lower third of the oesophagus correlates well with the temperature of the heart.¹ Placement of the oesophageal probe in this case was followed by the decision to use pleural lavage for rewarming, and the positive feedback from seeing immediate and steady mediastinal temperature rise was key in maintaining the team's focus on continuing resuscitation efforts.

Direct rewarming of the chest cavity, mediastinum and most importantly the heart with this simple procedure may be the single most important procedure to be undertaken, after all basic and advanced life support measures are instituted, in patients

with hypothermic cardiac arrest in medical units that have no access to extracorporeal rewarming techniques. The author will establish immediate oesophageal temperature monitoring and early pleural lavage rewarming in any future such patient presentations. A mechanical chest compression device was procured for ED following this case. Consistent access to FAST scan may aid diagnosis and treatment decisions in such cases, in particular by potential early identification of pericardial tamponade and intra-abdominal bleeding.^{21–24} More consistent access to FAST scan has also been achieved in ED.

Contributors Sole author.

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Learning points

- In the setting of hypothermic cardiac arrest 'the patient is not dead, until warm and dead'.
- Oesophageal temperature monitoring and early bilateral heated pleural lavage is effective.
- Successful resuscitation of patients with hypothermic cardiac arrest is possible by adhering to European Resuscitation Guidelines and in the absence of 'hi-tech' extracorporeal rewarming techniques.

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