Reminder of important clinical lesson

Therapeutic hypothermia after cardiac arrest in a 66-year-old man

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Hypoxic-ischaemic brain injury is an important cause of morbidity and mortality following both in- and out-of-hospital cardiac arrest. Despite significant advances in critical care the only intervention proven to increase survival rates after cardiac arrest is mild hypothermia. The authors present a case describing the use of therapeutic hypothermia after ventricular fibrillation cardiac arrest, including its indications and contraindications, and the techniques that can be used to induce it.

BACKGROUND

Therapeutic hypothermia is now the standard of care for adult patients with return of spontaneous circulation (ROSC) who remain comatose following out-of-hospital ventricular fibrillation (VF) cardiac arrest and might also be of benefit after non-shockable cardiac arrest, in-hospital cardiac arrest and in children. This case is a relatively common scenario that contains important messages about an emerging therapy that will become increasingly relevant to non-intensivists. It also serves as a useful summary of recently updated International Liaison Committee on Resuscitation (ILCOR) guidelines.

CASE PRESENTATION

A 64-year-old man presented to the emergency department with dyspnoea at rest. He denied chest pain. He had stage IV chronic renal impairment secondary to hypertension and was awaiting consultation for renal replacement therapy. On examination, he was alert with a respiratory rate of 35 breaths per min and oxygen saturation of 82% on 100% oxygen via a non-rebreather mask. He had a sinus tachycardia and a blood pressure of 140/85. Auscultation demonstrated bilateral end-inspiratory crackles confirmed by urgent chest radiograph to be pulmonary oedema. ECG showed voltage criteria for left ventricular hypertrophy and widespread T-wave inversion. Initial management with diuretic and intravenous nitrate failed to improve his symptoms so he was commenced on non-invasive ventilation. His symptoms transiently improved though he later deteriorated, becoming acutely hypoxic, and suffering VF cardiac arrest.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses of the pulmonary oedema include fluid overload due to deteriorating renal function, cardiac ischaemia, valvular heart disease, arrhythmia, pericardial constriction or acute pulmonary embolism. Urgent echocardiography and cardiac enzymes were inconsistent with acute pulmonary embolism, acute myocardial

infarction, or valvular cause. Serum potassium was normal and there were no monitored arrhythmias. It is likely that his cardiac arrest resulted from hypoxia secondary to worsening pulmonary oedema.

TREATMENT

Cardiopulmonary resuscitation was started immediately and ROSC was achieved after two cycles. He remained comatose, so sedation, intubation and mechanical ventilation were instituted. Therapeutic hypothermia was commenced using externally applied ice packs and 2000 ml 4°C 0.9% saline intravenously. He was transferred to the intensive therapy unit (ITU) where general intensive care measures and monitoring were initiated. He was haemodynamically optimised using intravenous fluids and norepinephrine. Blood glucose was controlled, and a core body temperature of 34°C maintained using an intravascular cooling catheter. He was haemofiltered for pulmonary oedema.

OUTCOME AND FOLLOW-UP

After 24 h of therapeutic hypothermia, he was rewarmed slowly over 12 h and once normothermic the sedation was stopped. He was initially confused but after a further 48 h full neurological recovery was evident. He was transferred to the dialysis unit where renal replacement therapy with peritoneal dialysis was established.

DISCUSSION

Patients who remain comatose following ROSC will typically require ITU support for monitoring and organ support. Despite significant advances in critical care the only intervention proven to increase survival rates after cardiac arrest is mild hypothermia. Prior to the introduction of therapeutic hypothermia hypoxic-ischaemic brain injury was the cause of death in 68% of patients who survived to ITU admission but subsequently died in hospital after out-of-hospital cardiac arrest and in 23% after in-hospital cardiac arrest.¹ Brain injury can cause cognitive dysfunction,

seizures, coma, persistent vegetative state and brain death. It can be exacerbated by fever, hyperglycaemia and seizures. In the un-cooled patient temperatures $>39^{\circ}\text{C}$ are common in the first 72 h after out-of-hospital cardiac arrest, and have been associated with a significantly increased risk of brain death.

A recent meta-analysis demonstrated 6-month survival and favourable functional neurological recovery in adults who remained comatose after ROSC following out-of-hospital VF cardiac arrest was more likely in the hypothermia group (risk ratio, 1.44; 95% CI, 1.11–1.76).² The ILCOR published updated guidelines in 2010 recommending that ‘adult patients who are comatose (not responding in a meaningful way to verbal commands) with spontaneous circulation after out-of hospital VF cardiac arrest should be cooled to 32°C to 34°C for 12 to 24 h.’³ They suggest that therapeutic hypothermia might also be of benefit after non-shockable cardiac arrest, in-hospital cardiac arrest and in children.³

Therapeutic hypothermia is generally divided into three stages: induction, maintenance and rewarming (a fourth, postrewarming, is sometimes added). For the general physician the most relevant of these is induction. Current consensus is that this should be started as soon as possible.³ Induction of hypothermia can be achieved using cold intravenous fluids (e.g. 30 ml/kg 4°C 0.9% saline or lactated Ringer’s) or ice packs applied to the groin, armpits, neck and head, facilitated by the normal drop in body temperature following ROSC.³ Although giving fluid in this way in the presence of postcardiac arrest myocardial injury has a theoretical risk of precipitating pulmonary oedema, the rapid infusion of cold intravenous fluids to induce hypothermia is well-tolerated as the systemic ischaemia/reperfusion response after ROSC causes vasodilatation and relative intravascular volume depletion.³ In this respect, postcardiac arrest syndrome has more in common with septic than cardiogenic shock such that maintaining an adequate mean arterial pressure usually requires fluid and/or vasopressor rather than inotropic support.

After induction with cold intravenous fluids, additional methods will be required to maintain hypothermia.³ Endovascular and external cooling devices are available for this, some of which incorporate temperature feedback mechanisms.⁴ The optimal temperature, induction and maintenance techniques, time of initiation, duration and rewarming rate and therapeutic window for therapeutic hypothermia all remain to be resolved.³ Sedation will usually prevent shivering during therapeutic hypothermia. Neuromuscular blockade is avoided where possible due to the risk of masking seizure activity, but if unavoidable continuous electroencephalogram monitoring should be considered. Percutaneous coronary intervention during

therapeutic hypothermia may be feasible, safe and associated with improved outcome.³

Hypothermia has a range of possible side-effects.⁴ They are particularly prevalent during the initiation and rewarming phases and include:

- ▶ Hypovolaemia and hypotension
- ▶ Reduced cardiac output, mainly due to a decrease in heart rate
- ▶ Bradycardia, increased PR, QT and QRS intervals and arrhythmias
- ▶ Fluctuations in plasma electrolyte concentrations
- ▶ Impaired coagulation cascade and increased bleeding risk
- ▶ Shivering
- ▶ Increased infection risk
- ▶ Insulin resistance and hyperglycaemia
- ▶ Bedsores
- ▶ Increased amylase, liver enzymes and lactate and reduced white cell count, platelets and pH
- ▶ Reduced drug clearance.

Learning points

- ▶ Therapeutic hypothermia is now the standard of care for adult patients with ROSC who remain comatose following out-of-hospital VF cardiac arrest.
- ▶ Its use is widespread in the UK with 86% of ITUs using it as part of cardiac arrest care.⁵
- ▶ Physicians should be familiar with the indications and contra-indications for this emerging therapy, and the techniques that can be used to induce it.

Competing interests None.

Patient consent Obtained.

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