Case report
Failure of normal glycaemic regulation in a patient with severe hypothermia

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Abstract
This report describes the case of an 88-year-old non-diabetic female who was presented to the emergency department following a presumed hypoglycaemic collapse due to self-neglect. Subsequent rewarming and resuscitation demonstrated a number of the significant consequences of severe hypothermia, including apparent secondary impairment of glycaemic autoregulation. The phenomenon of reversible inhibition of insulin secretion due to severe hypothermia has been documented previously in the field of cardiac surgery. The hyperglycaemia was not treated with any antihyperglycaemic agent, and her recovery was uneventful. Subsequent blood sugar level monitoring was normal. If insulin is administered to the hypothermic patient, intensive monitoring of blood glucose is essential due to the increase in endogenous insulin secretion on rewarming.

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1. Introduction
Accidental hypothermia is a potentially life-threatening condition of particular concern during the winter months, although it may occur in any season. Hypothermia may be classified according to core body temperature as mild (35–32 °C), moderate (32–30 °C) or severe (less than 30 °C) [1], and predisposing factors may include impairment of thermoregulation, a cold external environment, neglect (including self-neglect), hypothyroidism and hypoglycaemia [1–3]. Systemic effects of hypothermia may include cardiac arrhythmias, loss of normal glycaemic control and depressed level of consciousness [1–4–6]. The Advanced Life Support Course Provider Manual [1] offers guidance regarding many aspects of rewarming the hypothermic patient but does not specifically address glycaemic control. The case described here illustrates that significant derangements of blood sugar level may be encountered in severe hypothermia, even in the non-diabetic patient.

2. Case report
An 88-year-old lady seemed confused and dysarthric when telephoned by her daughter. The daughter entered her home with police assistance, and found her lying on the floor partially clad and unable to communicate. Examination of the house showed that she had not been to bed the night before. She was last seen by her daughter 20 h previously, at which time she had appeared fit and well. Her only pre-existing medical conditions were high blood pressure and treated hypothyroidism, though her appetite had been poor for some time. At the scene, the paramedics found her blood sugar level (BSL) to be 1.2 mmol l\(^{-1}\) and administered intramuscular glucagon. On arrival in hospital, it had risen to only 1.9 mmol l\(^{-1}\). Her Glasgow Coma Scale (GCS) score was noted to be 11 (Eyes 4, Verbal 2, Motor 5) and her temperature measured by a tympanic thermometer was 27.8 °C. She was maintaining her own airway, breathing spontaneously and had an irregular femoral pulse of 60 bpm. A brachial blood pressure reading could not be obtained. Secondary survey revealed only a minor skin tear to the left elbow. Pupils were noted to be unreactive but the daughter related a history of cataract surgery.

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Active rewarming was commenced. As the time of initial onset of hypothermia was unknown it was decided to rewarm at a rate of 1 °C per hour, giving a total rewarming time of 10 h. This was achieved by means of a warm air blanket and warmed intravenous fluids. She was catheterised to allow monitoring of urine output and offer the option of warm bladder lavage if her core temperature failed to rise. Fifty millilitres of 50% dextrose was administered intravenously for hypoglycaemia, resulting in a rapid increase in BSL to 20.4 mmol l⁻¹. BSL then remained persistently elevated, falling gradually to 10.9 mmol l⁻¹ at 10 h and 7.7 mmol l⁻¹ after 14 h (see Fig. 1).

Arterial blood gases showed her to have severe metabolic acidosis due to lactic acidosis, with an uncorrected pH of 7.05, a pCO₂ of 2.4 kPa and a blood lactate of 15.0 mmol/L (see Table 1). This was attributed to poor tissue perfusion resulting from her hypothermia. Her blood pressure became measurable after half an hour at 94/26 mmHg and after 1 h, with a core temperature by tympanic probe of 28.7 °C, her GCS had risen to 14 (Eyes 4, Verbal 4, Motor 6). She remained dysarthric and confused. Neurological examination at this time was otherwise normal. Cardiac monitoring showed initial slow atrial fibrillation (AF) interrupted by spells of ventricular fibrillation; however, the latter were consistently of less than a second in duration and resolved spontaneously. Towards the end of the first hour these spells ceased and she remained in slow AF. She was then transferred to HDU for continued rewarming, and received a single 200 mg bolus of intravenous hydrocortisone on suspicion of a possible autoimmune process targeting thyroid, pancreas and adrenal glands.

The following day she was both normothermic and consistently normoglycaemic, with a blood pressure of 147/66 mmHg, a pulse of 78 bpm in sinus rhythm and both the confusion and the dysarthria had resolved. She was therefore transferred to a medical ward. On examination she appeared neurologically intact. The remainder of her stay was uneventful.
The patient did not receive exogenous insulin but a review of the literature suggests this may be beneficial. Good glycaemic control in surgical patients is associated with a reduced risk of post-operative infection [8] and a significant reduction in morbidity and mortality in the intensive care unit [9]. In particular, significant reductions were demonstrated in rates of sepsicaemia and mortality due to septic foci, and also of acute renal failure and critical illness polyneuropathy. Whether the benefit also extends to medical patients remains unproven but as intravenous insulin therapy is well tolerated, tight glycaemic control offers considerable potential benefits at relatively low risk.

4. Conclusion

Severe hypothermia may cause a reversible failure of insulin secretion and peripheral insulin resistance. In the hyperglycaemic hypothermic patient who is not a known diabetic, while tight blood glucose control may potentially confer a significant mortality benefit, the care team should be aware that the patient may exhibit rebound hyperglycaemia when secretion of endogenous insulin resumes, and intensive monitoring of blood glucose levels is therefore necessary. As the secretion of endogenous insulin may at first be excessive, intensive monitoring should continue even after return of spontaneous normoglycaemia.

Conflict of interest

None.

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References

and insulin levels in man. Hormone levels during open heart surgery.

