CASE REPORT

Hypothermia as a forgotten sign of prolonged severe hypoglycaemia

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SUMMARY

Hypoglycaemia is a rare, although known cause of hypothermia. It appears to be a forgotten cause as well. Only one retrospective study has been done to look at this association and the last case published on this was over four decades ago. This case will remind physicians that hypoglycaemia is a possible primary cause of hypothermia. Interestingly, hypothermia in this case appears to be the body’s response for neuroprotection which is not dissimilar to the proven therapeutic induced hypothermia employed in patients with cardiac arrest.

BACKGROUND

Hypoglycaemia is a rare, although known cause of hypothermia. It appears to be a forgotten cause as well.1 Only one retrospective study has been done to look at this association and the last case published on this was over four decades ago.1,2 This case will remind physicians that hypoglycaemia is a possible primary cause of hypothermia. Interestingly, hypothermia in this case appears to be the body’s response for neuroprotection which is not dissimilar to the proven therapeutic induced hypothermia employed in patients with cardiac arrest.

INVESTIGATIONS

On admission, the patient had an ECG which showed some premature ventricular contractions and some non-specific ST changes. His troponin I was positive at 2.218 ng/mL (<0.049) with the next one being 2.013. His brain natriuretic peptide was 7741 pg/mL. All other laboratory tests were within normal range. His serum glucose was 108 mg/dL. His chest X-ray demonstrated bilateral pleural effusions and some infiltrates consistent with vascular congestion.

After the patient was persistently hypothermic, he had further workup performed which were all negative. He had a cortisol of 14 µg/dL (normal 13–22), thyroid stimulating hormone of 1.26 µU/mL (normal 0.5–4.9), and lactic acid 1.64 mmol/L (normal <2.2). Urinalysis excluded a urinary tract infection, and blood cultures remained negative. Moreover, the patient had unremarkable CT scans of the chest, abdomen and pelvis.

Prior to discharge, he had a left heart catheterisation which did not show any occlusive coronary artery disease. He had a two-dimensional echocardiogram and was diagnosed with systolic congestive heart failure with an left ventricular ejection fraction of 10%–15%.

DIFFERENTIAL DIAGNOSIS

The patient was diagnosed with non-ST-elevation myocardial infarction and new-onset heart failure on admission. For his hypothermia, severe additional differentials were considered. These included overwhelming sepsis, hypothyroidism, hypocortisolism or adrenal insufficiency. While some form of relative hypocortisolism is possible, given the fact that the patient did not have any other corresponding signs or symptoms prior to this event, adrenal insufficiency as a primary aetiology was
Hypoglycaemia can lead to encephalopathy. In such a scenario, the patient may present with nervousness, diaphoresis and confusion. Quite often a low core temperature is associated with this. The drop in temperature with hypoglycaemia might be transient but at times can be more persistent. The latter was observed in our case where the patient who became hypoglycaemic, remained hypothermic for about 12 hours, despite external rewarming measures.

The exact mechanism of how hypoglycaemia causes hypothermia is unclear but is believed to involve hypothalamic thermoregulatory system. Freinkel et al published a study in 1972 which provided some insight into the mechanism. They infused 2-deoxy-D-glucose (2-DG) in healthy young subjects and in mice. 2-DG inhibits intracellular glucose utilisation. Both groups exhibited a drop in rectal temperature which persisted for 6 hours despite adequate plasma glucose concentration. It was also noted that the hypothermia was at least five times more pronounced with intracranial drops in glucose levels as opposed to a drop in the plasma. This reaffirms the triggering site of hypothermia from hypoglycaemia to be in the CNS. A study conducted in 1981 by Gale et al had interesting observations on the effects of hypoglycaemia by infusing insulin. They found that hypoglycaemia led to increased sweating and inhibited shivering, both of which led to hypothermia.

The treatment for hypoglycaemia is standard with intravenous dextrose in severe cases. For more persistent hypoglycaemia, 10% dextrose is usually employed. Interestingly however, one study found that the spontaneous improvement in blood glucose was impaired at lower core temperatures. This makes it important to directly address the hypothermia with active and passive rewarming. Warming blankets, warm intravenous fluid infusion and airway warming are some of the measures that can be employed. We used active warming with a warming blanket and continuous infusion of 10% dextrose.

Hypothermia may be beneficial in severe hypoglycaemia. The benefits of therapeutic hypothermia in patients with cardiac arrest is well established. Hypothermia in such a scenario is neuroprotective and hence improves outcomes. It is quite possible that the hypothermia that results from hypoglycaemia is in fact a natural protective response by the body for neuroprotection. By lowering basal metabolic rate and the rate of glucose consumption by the brain, hypothermia may protect the brain from the adverse effects of glucose deprivation. The hypothermia seen with hypoglycaemia thus, not only provides a clue to the diagnosis, but may also have evolved as the body’s mechanism to survive the extreme stresses of severe hypoglycaemia.

**Learning points**

- Hypoglycaemia must be considered as the differential in any patient with hypothermia.
- Spontaneous improvement in blood glucose may be impaired at lower core temperatures.
- The hypothermia seen in hypoglycaemia may not be transient and in fact may last several hours after the blood glucose normalises.
- Hypothermia from hypoglycaemia might be a protective mechanism of the body.

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REFERENCES