

## Hypothermia: an under-estimated risk

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### Case presentation

**Prof. Bruno:** A 80-year-old woman was found unconscious in the street from a passer-by. It was a cold January morning, and the patient had slept outdoor. She was a homeless known in the Emergency Department (ED) as an alcohol abuser.

When she was admitted to the ED, she presented with stupor and apathy. She spoke very slowly, and her speech patterns were vague and slurred. Physical examination revealed that the skin was very cold, even on the torso; and there was marked pallor. The rectal body temperature was 29°C.

She had an abnormally slow rate of breathing (8 breaths/min), and the non-invasive  $SO_2$  was 86% on room air, and 96% after the administration of Oxygen by mask (6 L  $min^{-1}$ ). Blood pressure was 85/50 mmHg, and the pulse was 40 beats/min, and very weak. The 12-lead ECG showed sinus bradycardia at about 40 beats/min, and there

were prominent J waves (Osborn Wave). Hemogasanalytic values were normal. A Glasgow Coma Scale (GCS) score was 10. The patient underwent oro-tracheal intubation to provide airway protection. The finger-stick glucose was normal (108 mg %). The laboratory tests were within normal ranges except for a hyponatremia (120 mmol/L). We hypothesize the hyponatremia was due to volume depletion.

Our working diagnosis was accidental hypothermia. We immediately removed her wet cold clothing, and we covered the patient with an electric blanket. She was given warm saline fluids intravenously. Because she was classified as severe hypothermia with a high risk for cardiac arrest, she underwent extra-corporeal treatment by emergency haemodialysis to actively rewarm her blood rapidly outside the body. In this way it was possible to restore normal body temperature rapidly. This invasive method of rewarming permitted us to restore a normal core temperature providing safe treatment.

The patient left the hospital after several; days in good health. We advised her to avoid sleeping outside, to adequately cover her body including extremities and particularly the head.

### Differential diagnosis

**Dr. Caroselli, Dr. Pisani:** There are many causes of hypothermia. Often decreased thermogenesis is secondary to an endocrinologic failure such as hypopituitarism, hypoadrenalism, or myxedema, it may be due to alcohol or drug intoxication.

Among metabolic disorders, a frequent cause of hypothermia includes hypoglycaemia that may occur from a glycogen store depletion. Blood glucose determination is

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mandatory in each patient with hypothermia, and hypoglycaemia should be monitored and treated accurately to avoid cerebral injury.

Drugs commonly implicated in the development of hypothermia include sedative–hypnotics, phenothiazines and insulin.

Sepsis may alter the hypothalamic temperature set point, and it is a well-known cause of hypothermia as in diabetic ketoacidosis, as well as those diseases that cause immobilization.

Increased heat loss occur in patients with an increased peripheral blood flow (for example: burns, erythroderma, psoriasis, eczema), as well as in patients with massive cold infusions or overcooling in therapeutic hypothermia [1].

In trauma victims, hypotension and hypovolemia place thermostability at risk, and there is not a compensatory shivering thermogenesis.

Not least, hypothermia may also be induced by resuscitation with room-temperature fluids or cold blood transfusions. This is a particular iatrogenic risk in patients undergoing massive volume replacement, such as trauma and shock patients [2].

### Preliminary diagnosis

**Dr. Caroselli, Dr. Pisani:** In our clinical case, the rectal body temperature, and signs and symptoms permitted us to immediately recognise that patient was hypothermic. She was known to be an alcohol abuser, and she was found outdoors, but even if in appearance it seems easy to understand what has occurred, after a preliminary diagnosis, it is important to do a differential diagnosis. In fact sometimes hypothermia can mask a cerebral injury, for example after a head trauma that is very common in drunk patients.

### Further investigations

**Dr. Gabrieli:** Further laboratory findings to be considered are measurement of amylase and lipase (that were normal in our case) because pancreatitis is common in these patients. Clotting studies are useful because hypothermic patients sometimes show a coagulopathy and DIC. These were normal in this patient.

Cervical spine X-ray studies showed neither recent fracture nor post-traumatic signs. A head computed tomography (CT scan) was performed that did not show haemorrhage or other post-traumatic lesions. Initial echocardiography revealed a depression of cardiac output.

### Follow-up

**Prof. Bruno:** After the resolution of the hypothermia, we assisted improving the patient's clinical condition, and a second echocardiography revealed an improved cardiac performance.

At present the patient lives in a community, she stopped to drinking alcohol, and now lives indoors in a comfortable place especially during the winter!

### Discussion

**Dr. Caroselli, Dr. Gabrieli:** Hypothermia is a temperature-related disorder defined as a core temperature less than 35°C.

Normal heat loss occurs through five mechanism: radiation to the environment (55–65% body's heat loss), conduction—transfer of heat by direct contact—(2–3%), convection—transfer of heat by the actual movement of heated material—(12–13%), respiration (2–9%), evaporation (20–27%).

The hypothalamus controls two body mechanisms of heat conservation: shivering thermogenesis and non-shivering thermogenesis. The first is the heat gain through shivering, the second consists of an increase in metabolic rate [3].

The very young and the elderly are commonly at the greatest risk. The baby has a large surface area-to-mass-ratio, a relatively deficiency of the subcutaneous tissue layer and an inefficient shivering mechanism. For the elderly, the homeostatic capability progressively decreases with aging.

In the developed countries, the majority of hypothermic patients are intoxicated with ethanol or other drugs. Ethanol is a vasodilator that can be a cause of hypothermia by increasing heat loss via vasodilatation; it has also central nervous system (CNS) depressant effects; the intoxicated patients neither feel the cold nor react to it appropriately. However, there are publications that state a high blood alcohol level is a protection against the risk of ventricular fibrillation for a hypothermic heart at temperatures below 28°C [4]. Ethanol intake is also thought to protect the blood–brain barrier (BBB) against the effects of hypothermia [5].

Significant burns or severe exfoliative dermatitis prevent cutaneous vasoconstriction and increase transcutaneous water loss, predisposing to the development of hypothermia.

Patients with severe infections, diabetic ketoacidosis, immobilizing injuries, and various other conditions may have impaired thermoregulatory function. The final

common pathway seems to be a centrally mediated vasodilatation.

Hypothermia can also develop at mild temperature, especially among elderly non-ambulatory patients and those with significant co-morbidities, in patients without social or family support who cannot help themselves, or who have mental or motor skill deterioration, or in patients forced to the ground who cannot move for a clinical or traumatic problem, such as an elderly patient with a spontaneous hip fracture.

In the initial phase, heart rate, cardiac output and blood pressure rise, but with decreasing temperature, these all decline. Cardiac output and blood pressure may be markedly depressed by the negative inotropic and chronotropic effects of hypothermia and by concomitant hypovolemia.

Patients are at risk for dysrhythmias at body temperatures below 30°C (86°F); the risk increases as body temperature decreases. Although various dysrhythmias may occur at any time, the typical sequence is a progression from sinus bradycardia to atrial fibrillation with a slow ventricular response, to ventricular fibrillation and finally to asystole. The hypothermic myocardium is extremely irritable, and ventricular fibrillation may be induced by a variety of manipulations and interventions, including the mere handling of the patient [6].

Hypothermia causes a leftward shift of the oxyhemoglobin dissociation curve, potentially impairing oxygen release to tissues. However, the acidosis and decreased rate of tissue metabolism due to hypothermia compensate for this effect [7].

Hypothermia impairs renal concentrating abilities and induces a “cold diuresis,” leading to significant volume losses. Because of this concentrating defect, urine flow and specific gravity are unreliable indicators of intravascular volume and circulatory status. The immobile hypothermic patient is prone to rhabdomyolysis, and acute tubular necrosis may occur because of myoglobinuria and renal hypoperfusion.

The EKG in moderate or severe hypothermia may show a J wave or Osborn wave, which represents a positive deflection at the ST segment [2, 7]. These changes are best seen in the lateral precordial leads with increasing amplitude as hypothermia worsens. This wave is not pathognomonic because it may also be seen in patients with subarachnoid haemorrhage and intracranial injuries as well as in myocardial ischemia. Other EKG changes include PR, QRS and QT prolongation and T-wave inversion [8].

Although EKG changes can persist for days after rewarming, and reversal of the EKG changes may be delayed [9] and they do not require specific therapy. Moreover dysrhythmias in a hypothermic patients may be refractory to treatment.

In trauma patients, hypothermia is associated with coagulopathy [10], which is a result of impaired platelet function and thrombocytopenia caused by extravascular sequestration of platelets. Furthermore, as body temperature decreases, coagulation cascade enzymes tend to lose their function.

In severe hypothermia, paradoxical self disrobing by the patient may be observed. Although the cause of this phenomenon is unknown, it is hypothesized that cutaneous vascular disturbances result in a false sensation of warmth. Besides, in severe hypothermia, the level of consciousness progressively deteriorates to coma and loss of electroencephalographic activity, which may lead to terminal primitive behaviors (the hide-and-die syndrome). This concept may explain the reason that some patients who die of hypothermia are found in a hidden position [11].

Managing hypothermic patients is often a challenge because of the absence of a reliable history in most cases. Treatment includes both general supportive measures and specific rewarming techniques [12]. Oxygen and intravenous fluids should be warmed, and patients should have constant monitoring of their core temperature, cardiac rhythm, oxygen saturation and glucose levels. Sometimes a careful intubation after oxygenation can warrant the safety of the patient.

In patients with hypothermia, the decision to use passive or active rewarming techniques depends on clinical signs and symptoms, and on the degree of hypothermia. Passive rewarming is non-invasive, and should be used as the treatment modality for patients with mild hypothermia with a body temperature >32°C. It is achieved by using warm blankets that permit endogenous heat production. The patients must have intact thermoregulatory mechanisms, normal endocrine function, and adequate energy stores [13].

Active rewarming is necessary if the core temperature is <32°C, in hemodynamically unstable patients, or in respiratory failure. It is achieved by giving warm active supplementation. The most common methods are: warm forced-air blankets, infusing warm intravenous fluids (for example normal saline with 5% dextrose at 40°C), lavage of body cavities with warm fluids, and haemodialysis [14].

If central venous lines are placed, care should be taken to avoid entering and irritating the heart.

With proper diagnosis and management of hypothermia a good outcome can be achieved.

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