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Alpine cross-country skier with energy depletion and reduced consciousness

CASE REPORTS

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Impaired consciousness may be caused by a variety of reasons, and sometimes the simple explanations are overlooked. We describe here the case of a young cross-country skier who developed a serious medical condition with dramatic symptoms. Considerable pre-hospital resources were expended and hospital examinations carried out before it became evident that the condition could be easily treated.

A male in his late 20s was enjoying a day of alpine cross-country skiing with friends. The weather was good, but the snow was wet and heavy, and the 7-hour hike turned out to be more strenuous than originally envisaged. Just before they reached their destination, the man's behaviour suddenly changed – he sat down in the snow and said he did not have the energy to carry on. He was enthusiastically encouraged by his friends to walk the last little stretch, but he explained that he was utterly exhausted and could not continue.
After a short while he also became confused, unsettled, motorically agitated and aggressive. He was not very cooperative, and his friends worried that he would become hypothermic, since he was lying in the snow, flailing around. Due to poor mobile phone reception in the area, some of his friends skied down the slopes to the nearest habitation and phoned the hospital emergency department for help. The emergency department requested air ambulance assistance from the Rescue Coordination Centre.
The rescue helicopter's duty anaesthetist considered the situation to be indeterminate, with no definite diagnosis. Because the early stages of hypothermia were suspected, it was decided to have the man picked up by rescue helicopter.

Flying conditions were good – daylight, hardly any wind, and a temperature around 0 °C. The distance to the patient's location was approx. 30 nautical miles as the crow flies, which suggests a flying time of approx. 15 minutes by Sea King rescue helicopter.
Whilst in the air, the personnel were informed by the rescue control centre that the patient had previously been in good health and had been on a 7-hour ski touring hike with friends that day. He had been drinking a considerable amount of vodka, but had not had anything to eat.
His friends explained that he was lying in the snow, unable to carry on; they assumed that this was caused by exhaustion.
The man was soon found after a quick search. From aboard the helicopter the man was seen lying on his back, flailing in the snow. It was impossible to land nearby, so the rescuer was hoisted down to pick him up in a triangular harness. When the rescuer encountered the patient on the ground, he was

highly agitated and aggressive. However, after a while the rescuer managed to get him sufficiently under control to allow the two of them to be hoisted up to the helicopter together.

The first exploratory examination undertaken by the doctor aboard the air ambulance did not identify any major anomalies, but it was commented that the patient smelt strongly of alcohol. The Glasgow Coma Scale (GCS) score was reduced from the normal 15 points to 10. The part scores were 4-2-4 (normally 4-5-6), the first indicating that the patient opened his eyes when spoken to, the second that he did not communicate beyond grunting and groaning, and the last that he failed to localise stimuli other than by withdrawal. His pupils were of equal size and responded to light in the same way. The patient was appropriately dressed in warm clothing, and despite having spent 1 – 2 hours in the snow, the oxygen saturation meter showed a normal value of 97 %, without extra oxygen. The heart rate was 60/min, and the pulse amplitude was good. The patients' friends explained that he was in good health, that he was not on any medication and that he did not suffer from any allergies.

The air ambulance doctor consequently considered the peripheral circulation to be good. Although the patient refused to let the rescue crew measure his temperature, hypothermia was considered unlikely. The patient's cerebral symptoms and aggressive behaviour were therefore ascribed to alcohol intoxication.

It would not take long to reach the hospital by air, so because they were in transit with a less than cooperative patient, no further examinations were carried out. Also, due to the patient's agitation, no venous access was carried out.

On arrival at the emergency department, further examinations were carried out. The rectal temperature was 36.4 °C (36 – 38 °C), and hypothermia was therefore ruled out. Blood pressure and pulse readings were normal – 108/60 mm Hg and 80/min. It was difficult to establish the patient's respiration frequency due to his unsettled and agitated behaviour, but he suffered no obvious problems with his airways or respiration. The patient appeared to be heavily intoxicated by alcohol. This was later confirmed by a blood test result which showed an ethanol level of 2.1 ‰.

Prior to arrival, the emergency department's Trauma Leader had been told to expect a hypothermic and inebriated patient. However, hypothermia was ruled out once the rectal temperature had been measured. Initially, ethanol intoxication was assumed to be the main cause of the patient's uncooperative and abnormal behaviour. He kept waving his arms around, pulling at the staff and the monitoring equipment.

His respiration was however effortless, and it was decided to await sedation and intubation.

The patient's blood sugar level was measured at the emergency department; it proved to be as low as 0.8 mmol/l (3.5 – 7 mmol/l). Several 20 ml bolus doses of 50 % glucose (500 mg/ml) were therefore administered intravenously. After four bolus doses of glucose, bringing the total to 40 g, and before 30 minutes had elapsed, the patient had calmed down, and was behaving

appropriately and communicating normally. After a while, he turned over on his side and fell asleep; he was then admitted to the intensive care unit for monitoring until the following day.
The artery was tapped. Blood gas readings showed severe acidosis, with a pH of 7.15 (7.35 – 7.45), pCO_2 of 7.6 kPa (4.7 – 6 kPa), base excess –9 mmol/l (–3 to +3 mmol/l) and a lactate level of 6.6 mmol/l (0.7 – 1.8 mmol/l).
This indicated combined respiratory and metabolic acidosis. The hypercapnia was interpreted as a result of the alcohol intoxication gradually having led to hypoventilation. The metabolic acidosis was generally found to stem from lactate, even though there was no obvious source of lactate. There was no sign of tissue hypoperfusion as the blood pressure was normal and there was good circulation in the extremities. Furthermore, no symptoms gave reason to suspect anything pathological in the abdomen.

The doctors were surprised by the low blood sugar level. The patient had no known diabetes mellitus. This information had been provided by his friends and it was later confirmed by the patient himself that he was not on insulin or any other medication. Because the treatment normalised the cerebral symptoms, it was assumed that hypoglycaemia was the main cause of all the pathological events. It was discussed what had caused the hypoglycaemia, but alcohol-induced hypoglycaemia was not considered at that point.

Once the patient had received treatment for his low blood sugar level, his clinical situation improved relatively quickly. After 3.5 hours, the patient's blood gas readings were more or less normal, with a pH of 7.31 (7.35 – 7.45), pCO_2 of 5.3 kPa (4.7 – 6.0 kPa), and base excess – 6 mmol/l (–3 to +3 mmol/l).
Thanks to the speedy recovery it was not considered necessary to correct the acidosis medicinally, and the patient had no need for ventilation support. The blood gas levels normalised over the next eight hours.
The cause of the temporary lactic acidosis remains unknown.

All the patient's blood test results were normal before he was discharged the following morning.

Discussion

Ethanol can cause hypoglycaemia when taken in combination with fasting or physical exercise (). If no insulin has been administered, other causes may be endogen hyperinsulinemia, peroral antidiabetics, trimethoprim sulfa, beta blockers, antiarrhythmics, adrenal gland failure, sepsis and liver failure ().

One study found that 4 % of ethanol intoxicated patients suffered from hypoglycaemia (). The same study referred to another American study which suggested that 14 % of 1,418 drug and alcohol-induced episodes of hypoglycaemia were caused by ethanol (). These percentages may well be too high. Others have found an incidence of 0.9 % (). Of 2,348 patients treated for acute poisoning in Oslo, 21 suffered from hypoglycaemia. Ethanol was the agent in 1,018 of these cases, but no potential correlation was discussed (). A

large university hospital in New York found that ethanol was the cause in 45 of 125 patients admitted with hypoglycaemia. Hospitalisation was necessary in 31 of the 45 cases ().

However, another study has shown that the blood alcohol concentration of approximately half the patients with ethanol-induced hypoglycaemia may be so low at the time of testing that many are excluded from the statistics; the incidence is therefore similarly uncertain (). The same authors maintain that alcohol-induced hypoglycaemia has received much less attention in Europe than in the USA ().

In children, even small amounts of unintentionally administered or ingested ethanol will result in hypoglycaemia. Hypoglycaemia has been described after the washing of feverous skin with alcohol, and after ingestion of alcohol-containing mouthwash (). In small case report studies, the hypoglycaemia incidence in children intoxicated by ethanol has been stipulated to between 10 % and 24 % (), while another retrospective study found an incidence of 3.4 % (). Again, it is difficult to compare these studies, since by the time the patients have been admitted to hospital their ethanol concentration may have fallen to a level below that which would suggest ethanol as the cause of the hypoglycaemia.

In adults, the dominating combination is one of depleted glycogen stores and ethanol ingestion (). It is probable that this combination also features prominently in young children, whose glycogen stores empty much faster than in adults.

In adults, glycogen stores can be depleted after approx. 12 hours of fasting, but considerably sooner with increased consumption, as in the case of strenuous physical exercise. Our patient had been cross-country skiing for seven hours, a physically demanding hike involving a lot of up-hill climbing and down-hill snowboarding.

The mechanism has been known for a long time. Ethanol inhibits the gluconeogenesis (). For the alcohol dehydrogenase enzyme in the liver cells to be able to convert ethanol, an array of electron transport molecules is required, as well as the coenzyme nicotinamide adenine dinucleotide (NAD^+). As long as there is ethanol to be metabolised, the reduced NADH (nicotinamide adenine dinucleotide)/NAD^+ ratio will be considerably raised. Consequently, there is insufficient NAD^+ available for other reactions, such as in the gluconeogenesis.

One of the roles of NAD^+ is to be used by the lactate dehydrogenase enzyme for the conversion of lactate to pyruvate. If the NAD^+ level is low, the pyruvate concentration will fall and the formation of sugar from pyruvate will be impaired to the extent that the blood glucose level falls. The use of fat and amino acids in the gluconeogenesis also requires NAD^+, and this process becomes less effective when the ethanol conversion appropriates so much of this coenzyme (). This mechanism works even at low ethanol concentrations (fig 1). In animal studies, 47 % of the gluconeogenesis is inhibited at a blood ethanol concentration of 0.1 (). This matches the findings of a more recent study of five healthy male volunteers, whose gluconeogenesis was reduced by 45 % ().

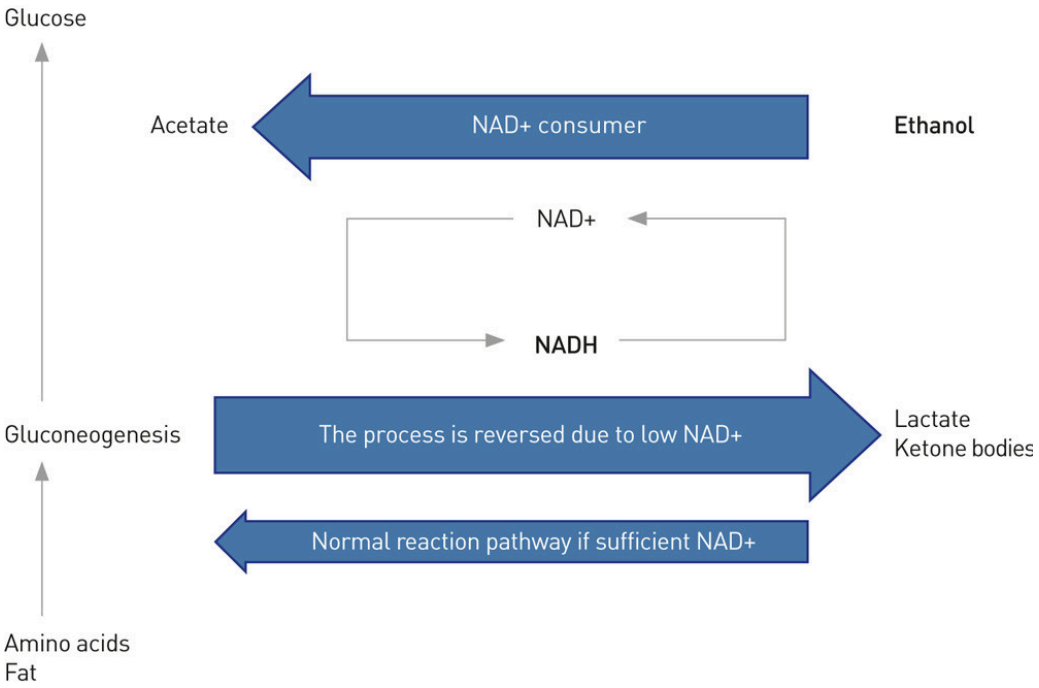

Figure 1 Greatly simplified presentation of the gluconeogenesis that takes place while under the influence of ethanol. For amino acids and fat to be converted into glucose through gluconeogenesis, NAD+ is required. Ethanol intoxication causes large amounts of NAD+ to be consumed as the ethanol is metabolised. This means that less NAD+ is available for the gluconeogenesis, thus causing hypoglycaemia. Rather than lactate being converted into glucose, the process is reversed, producing increased levels of lactate. NAD+ deficiency also increases the number of ketone bodies

Another mechanism that may result in hypoglycaemia is that ethanol also increases the level of a substance that inhibits the reading of a number of genes that are of particular importance to the gluconeogenesis (). A third cause is that ethanol increases the circulation of blood through the islets of Langerhans in the pancreas, at the expense of the exocrine part, which may lead to increased insulin secretion (). We are not aware of anyone having attempted to identify which of these factors is the greatest contributor to alcohol-induced hypoglycaemia.

Our patient's lactic acidosis was striking with readings that are normally encountered only in cases of serious septic shock or other serious circulation failure. Such patients generally suffer from tissue hypoperfusion, which results in type A lactic acidosis. Any lactic acidosis which is not caused by tissue hypoperfusion is referred to as type B lactic acidosis, and is often caused by deficient intracellular conversion of lactate. Type B lactic acidosis also occurs in connection with Metformin treatment, vitamin B_1 deficiency and malign hematologic diseases (). Alcohol-induced hypoglycaemia is yet another example of a type B lactic acidosis. If there is insufficient NAD^+, the lactate dehydrogenase enzyme will not be able to convert lactate to pyruvate fast enough, thus resulting in lactate accumulation ().

Because the lactate levels were measured on a small acid-base apparatus, it may have been the glycolic acid level that was measured, because these devices incorrectly interpret glycolic acid as lactate. Although we have previously

discussed this «lactic gap», ethylene glycol poisoning did not form part of this patient's assessment (). Methanol poisoning should also be considered in metabolic acidosis patients intoxicated by alcohol.

So far, we have been talking exclusively about ethanol-induced hypoglycaemia. It is an interesting question whether intoxication from other types of alcohol, e.g. ethylene glycol or methanol, will also appropriate NAD^+, inhibit the gluconeogenesis and induce hypoglycaemia while causing lactic acidosis. If so, this is yet another matter for consideration when exploring metabolic acid-base imbalance, which is often difficult ().

Several of the American articles on alcohol-induced hypoglycaemia assume that the condition is often not acknowledged, and may therefore be underreported (,). Although our patient's combination of fasting, vodka and a long cross-country skiing hike cannot be considered a common combination, it is nevertheless not uncommon for alcohol consumption to be accompanied by minimal or no consumption of food. Perhaps hypoglycaemia is the root cause of more instances of atypical intoxication, aggression and impaired consciousness than reported?

The ABC acronym of emergency medicine is well known. For some, its continuation – DEFG – **D**ont **E**ver **F**orget **G**lucose – has been an equally important mnemonic, ensuring that blood sugar measurements are never forgotten about for patients with reduced consciousness. It is easy to overlook this simple test, particularly when there are obvious factors that more readily present themselves as probable causes. We now know that the combination of fasting and alcohol may cause serious hypoglycaemia with cerebral symptoms, which should never be confused with alcohol intoxication.

It is therefore always important to establish whether hypoglycaemia is the cause of any consciousness impairment.

The patient has consented to the publication of this article.

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