Recovery from pH 6.38: lactic acidosis complicated by hypothermia

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Survival after extreme arterial acidosis is uncommon. A case of metformin induced lactic acidosis is described where the presenting pH was 6.38 exacerbated by hypothermia (29°C). Increased anion gap acidosis, its varied aetiology, potential reversibility, and the role of hypothermia are discussed. Early liaison with a medical toxicology unit is recommended when this rare condition is suspected.

CASE REPORT

A 62 year old Hungarian born truck driver arrived at the emergency department having been phoned through as an “unconscious male”. A few hours earlier his wife had put him to bed after he had complained of feeling “unwell”. He was known to suffer from diabetes, which was diet and “tablet” controlled. There was no other relevant past medical history. The patient had been found in an unheated bedroom on a day where the ambient temperature was 13°C. His core body temperature was 29°C, respiratory rate 12/min, oxygen saturation 96%, blood pressure 115/64, heart rate 52/min and blood glucose reagent stix 12 mmol/l (Boehringer Mannheim). Further examination revealed no evidence of foetor (alcohol or acetone), head injury, neck stiffness, rash, murmurs, abdominal tenderness or masses. The patient was able to open his eyes to speech, utter incomprehensible sounds and flex to pain. (Glasgow Coma Score 9). Fundoscopy and pupillary examination were unremarkable. Tendon reflexes were symmetrically reduced with downgoing plantars. A 12 lead electrocardiogram showed a sinus bradycardia and chest radiograph was normal. Initial arterial blood gas (ABG) analysis revealed a profound uncompensated respiratory and metabolic acidosis with a pH of 6.38 (temperature corrected pH 6.49) and base deficit of 38. Initially, it was assumed that the extreme acidosis represented an error in analysis, however factors that may potentiate the lactacidaemic effect are strenuous exercise, alcohol, renal and toxic compounds, or as a result of an inherited metabolic defect that results in lactate accumulation.

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impairment, liver and cardiovascular disease. In cases of metformin induced lactic acidosis a pH of <6.9 with a raised urea and lactate greater than 18 mmol/l is associated with a poor outcome. There is no clear relation between age, sex, dose and duration of treatment and development of lactic acidosis. Therapeutic plasma concentrations are between 1–2 mg/l, but irreversible cause of acidosis were present. These three cases suggest extreme acidosis is a poor independent factor for survival if hypothermia or metformin induced acidosis, or both, are present.

Vasodilatation secondary to hypercapnoea, heat loss from radiation and confusion preventing him from putting clothes on in an unheated room may all have contributed to him becoming cold. Moderately severe hypothermia offers a cerebral protective role in situations of cardiorespiratory arrest principally by reducing cerebral metabolic rate and hence oxygen demand. The mechanism by which hypothermia may be protective after return of spontaneous circulation is unclear, however reduced susceptibility to oxidant injury has been proposed. It could be postulated, by a similar mechanism our patient was less susceptible to oxidant injury with cardiorespiratory arrest, hypothermia and a temperature (33°C) uncorrected arterial pH of 6.33. The lowest recorded arterial pH survived is 6.30 occurring in an 84 year old man after metformin ingestion, although further details are unavailable. Our case is unusual in that hypothermia and a reversible cause of acidosis were present. These three cases suggest extreme acidosis is a poor independent factor for survival if hypothermia or metformin induced acidosis, or both, are present.

Figure 1  (left) Emergency department arterial blood gas sample on arrival (temperature corrected pH 6.494) and (right) ITU arterial blood gas sample after intensive care (uncorrected for temperature).
should raise the suspicion of type B, potentially reversible lactic acidosis, and the need for discussion with a medical toxicology unit, for both advise and specialist drug assay. Hypothermia is known to increase cerebral tolerance to hypoxia, however it may also offer a protective role in severe acidosis. This case shows that extreme acidosis in the presence of hypothermia may respond to treatment and that a full recovery is possible.

Contributors
S Ahmad initiated the original idea, carried out the literature search and wrote the paper. M Beckett wrote and edited the paper. S Ahmad acts as guarantor.

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