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A complicated hyperglycaemic emergency

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Case presentation

A 67-year-old woman of Jamaican origin was admitted to the emergency department with acute confusion. She had been found at home by police, who were notified by neighbours that she had not been seen for some days. No further history was available from the patient or family, and no past medical history was known. The primary care surgery where she was registered was not contactable, and there were no family contacts. On examination she was dehydrated (capillary refill six seconds, dry mucous membranes, poor urine output on catheterisation), confused, Glasgow Coma Score (GCS) 11 (eyes 4, movement 4, speech 3), blood pressure (BP) lying 95/65 mmHg, pulse 110 irregularly irregular, temperature 37.3°C. Cardiac examination was normal, and chest examination revealed coarse crackles at the left lung base. Neurological examination appeared normal, although was difficult due to confusion, but she was moving all four limbs and had no abnormalities on funduscopy. Baseline investigations at admission are shown in Box 1.

What is the differential diagnosis and likely diagnosis?

It is unclear whether this patient has a known diagnosis of type 1 or type 2 diabetes mellitus. The most likely reason for her acute problem is that of hypersomolar hyperglycaemic syndrome (HHS), formerly called hyperosmolar non-ketotic coma (HONK).[1] Diabetic ketoacidosis (DKA) is unlikely due to the patient's age and relative lack of acidosis. Ketosis is mild and frequent in a hypersomolar state due to lack of nutrient, although ketosis prone type 2 diabetes is well recognised. The patient also has atrial fibrillation (AF), although whether this is new or old is unclear. Renal impairment is likely to be due to dehydration, although acute on chronic renal impairment is also possible. Chest radiograph suggests a left basal pneumonia.

What is the initial management?

Clearly the patient needs meticulous attention to fluid resuscitation. Despite the hypernatraemia, normal saline should be used as it is likely to be hypotonic in this situation, and rapid infusion of a litre of 0.9% saline is appropriate initially, followed by one litre over two, four, then six hours according to blood pressure response, fluid status and urine output. The patient should be managed in a level 2 or 3 bed (high-dependency unit or intensive care unit) if possible.

Careful monitoring of all indices, including blood pressure, pulse, urine output and electrolytes should be undertaken. Full anti-coagulation should be commenced with low molecular weight heparin in view of the AF and high risk of thrombotic events in HHS, and broad spectrum antibiotics should be prescribed for the pneumonia.

Intravenous insulin should be commenced, but it is imperative to start at a low dose (around 1 unit per hour). Rehydration with fluid is likely to reduce glucose levels by 5–11 mmol/l. There is good evidence that a slow decline in glucose (2–3 mmol/l per hour) should be achieved in this situation, as more rapid correction in glucose will lead to rapid shifts in osmolality, with a high risk of pontine myelinolysis.

Box 1. Results of baseline investigations.

Sodium	148 mmol/l	(136–146 mmol/l)
Potassium	3.6 mmol/l	(3.5–5.1 mmol/l)
Urea	17.6 mmol/l	(2.5–6.4 mmol/l)
Creatinine	253 μmol/l	(62–106 μmol/l)
Serum osmolality	349 mOsm/kg	(280–295 mOsm/kg)
Haemoglobin	13 g/dl	(11.5–16.6)
White cell count	10.1 × 10^9/l	(4–11)
Platelets	186 × 10^9/l	(150–400)
Plasma glucose	34.5 mmol/l	(4–7 mmol/l)
pH	7.35	(7.35–7.45)
Bicarbonate	23 mmol/l	(22–26 mmol/l)
Base excess	−2 mmol/l	(−3–+3 mmol/l)
Chest radiograph	Left basal patchy shadowing	
ECG	Atrial fibrillation, otherwise normal	
Urinalysis	Protein +	
	Ketones +	
	Blood negative	
	Leucocytes negative	
	Nitrites negative	

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Case progression

The patient initially responded well to fluid resuscitation. Over 24 hours she received six litres of 0.9% saline, her urine output improved to 60–90 ml per hour, BP was 120/75 mmHg and pulse 90 irregular. She remained confused, but was more orientated and able to talk more coherently, with GCS ranging from 11–13, temperature 37.1°C. Capillary glucose levels improved, and were stable at around 10 mmol/l on one unit of insulin per hour. Electrolytes initially improved within eight hours of admission.

At around 36 hours after admission, her confusion deteriorated and electrolytes were noted to be deteriorating again, with marked hypernatraemia (Box 2). She appeared well hydrated, but GCS had dropped to 8, BP 130/75 mmHg, pulse 90 irregular, afebrile, urine output 70–120 ml/hour, capillary glucose 10–14 mmol/l.

What is the likely diagnosis and how should this be managed?

While the hyperglycaemia seems to have been corrected appropriately the deterioration in sodium and GCS is a concern. She has been well fluid hydrated. An intracerebral event is a possibility, as she is at high risk of stroke, and hence a computed tomography brain scan should be undertaken. This was reported as normal. Over treatment with 0.9% saline was also considered, although fluid replacement with hypotonic solutions was seen as too dangerous. As the urine output was noted to be high, consideration was given to the possibility of diabetes insipidus complicating HHS. This was supported by the finding of a low urine osmolality (103 mOsm/kg) compared to serum osmolality (373 mOsm/kg). Thyroid function and serum cortisol were normal. She was treated with 10 mcg of desmopressin intranasally twice daily, and her sodium corrected to normal with 72 hours, with her confusion completely resolving. The desmopressin was stopped within 48 hours, with no recurrence of her metabolic abnormalities.

She was stepped down to a medical ward, and commenced on oral hypoglycaemic agents, with reasonable control of hyperglycaemia prior to discharge.

Discussion

HHS is a common acute medical emergency, and it is important to distinguish the condition from DKA, as the management of the two differs significantly. Patients with HHS tend to be extremely dehydrated, ketone negative or weakly positive, older, often Afro-Caribbean and the history is frequently more subacute. Mortality rate is over 30%. The depth of unconsciousness correlates most closely with severity of plasma osmolality, and while the management principles are the same as with DKA, DKA requires high-dose intravenous fluids and insulin to rapidly correct the hyperglycaemia and ketogenesis, whereas HHS requires low-dose insulin therapy. Insulin should be infused at one unit/hour initially, aiming to lower blood glucose by no more than two to three mmol/l/hour, meaning that normoglycaemia may not be reached for 48 hours. Deaths occur in HHS due to thrombosis and sequelae of metabolic derangements caused by inappropriately rapid treatment of glucose.

Diabetes insipidus complicating HHS has been previously reported, although this has been associated with lithium therapy in some.[2–4] Diabetes insipidus may occur due to acute swelling of the hypothalamo–pituitary axis, leading to relative lack of vasopressin release. Alternatively, the renal tubules may become insensitive to the effects of vasopressin in poorly controlled diabetes, leading to a mild nephrogenic diabetes insipidus.[5]

In summary, therefore, we present a patient newly presenting with type 2 diabetes and HHS which was complicated by transient diabetes insipidus, leading to marked hypernatraemia. Her metabolic problem resolved rapidly with treatment with desmopressin and correction of the marked dehydration.

Box 2. Progress of electrolytes.

	Baseline	8 hrs	24 hrs	36 hrs
Sodium (mmol/l)	148	149	167	178
Potassium (mmol/l)	3.6	4.1	4.5	4.6
Urea (mmol/l)	17.6	11.3	10.3	7.8
Creatinine (μmol/l)	253	164	135	143

Key learning points

- Hyperosmolar hyperglycaemic syndrome (HSS) is a common hyperglycaemic crisis, and differentiation between HHS and diabetic ketoacidosis is important, as management differs considerably
- Patients with HHS tend to be older, with less acidosis and ketosis, and higher glucose levels
- Treatment with 0.9% saline and low-dose insulin therapy should be instituted. High-dose insulin therapy should be avoided
- Patients with HHS can frequently subsequently be managed with diet and oral hypoglycaemic therapy
- Persistent hypernatraemia despite adequate fluid resuscitation should suggest the possibility of diabetes insipidus complicating HHS

References

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