Hyperglycemia

Hyperglycemia crises

Diabetic ketoacidosis (DKA) is caused by absolute or relative low insulin levels.

Plasma glucose high causes an osmotic diuresis, with Na+ and water loss (up to 8–10L), hypotension, hypo perfusion, and shock.

Hyperglycemia crises

Normal compensatory hormonal mechanisms are overwhelmed and lead to high lipolysis.

In the absence of insulin, this results in the production of non-esterified fatty acids, which are oxidized in the liver to ketones.

Younger undiagnosed patients with diabetes often present with *DKA* developing over 1–3 days. Plasma glucose levels may not be grossly high; euglycaemic ketoacidosis can occur.

Urinalysis demonstrates ketonuria.

Hyperosmolar hyperglycaemic state (HHS) is caused by intercurrent illness, inadequate diabetic therapy, and dehydration. It develops over days/weeks and is more common in the elderly.

HHS is characterized by high glucose levels (>30mmol/L), high blood osmolality, and a lack of urinary ketones.

Mortality is 75–10% but may be even higher in the elderly.

Causes

Think of the four '*I*'s separately or (often) in combination:

- *Infection:* common primary foci are the urinary tract, respiratory tract, and skin.

- *Infarction:* myocardial, stroke, GI tract, peripheral vasculature.

- *Insuffificient insulin*.

- *Intercurrent illness*: many underlying conditions precipitate or aggravate DKA and HHS.

Clinical features

Hyperglycaemic crisis may present in various ways. Some of the following are usually present:

- *Signs of dehydration:* thirst, polydipsia, polyuria, d skin turgor, dry mouth, hypotension, tachycardia.

- *GI symptoms:* are common in DKA, with nausea, vomiting, and abdominal pain. This can be severe and mimic an 'acute surgical abdomen'.

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Clinical features

- *Hyperventilation* with deep rapid breathing (Kussmaul respiration) and the smell of acetone on the breath, is pathognomonic of DKA.

- *True coma* is uncommon, but altered conscious states and/or focal neurological deficits are seen particularly in older patients with HHS.

Investigations

Aim to confifirm the diagnosis and search for possible underlying cause(s):

- Check BMG and test the urine for glucose and ketones.

- Send blood for U&E, blood glucose, creatinine, and osmolality (or calculate it): mOsm/L= (2 × Na+) + glucose (mmol/L) + urea (mmol/L).

- Check ABG (look for metabolic acidosis ± respiratory compensation).

Investigations

- FBC and CXR (to search for pneumonia).
- ECG and cardiac monitoring (look for evidence of hyper-/hypokalaemia).
- Blood cultures and, if appropriate, throat or wound swabs.
- Urine/sputum microscopy and culture.

Management

If altered consciousness/coma, open/maintain a patent airway.

• Give O2 by mask, as required. Consider the possible need for GA and IPPV for coma ± severe shock.

• Commence *IVI* with 0.9% saline. Give 1000mL of 0.9% saline over 0.5–1hr, then 500mL/hr for the next 2–3hr. Persistent hypotension may require high in infusion rate and/or colloid administration.

Avoid over rapid infusion with the risks of pulmonary oedema and ARDS, especially in the elderly and patients with IHD.

Management

- *Insulin*: start an infusion of soluble insulin after IV fluids have started using an IV pump or a paediatric burette at 0.1U/kg/hr (typically 6U/ hr).
- Check blood glucose and ketone levels every hour initially—aim for blood glucose to drop by at least 3mmol/L/hr, and blood ketone by at least 0.5mmol/L/hr. Continue insulin infusion until blood ketone is <0.3mmol/L and blood pH is >7.3.

Management

- When plasma glucose is <14mmol/L, add 10% glucose IVI at a rate of 125mL/hr (through a large vein) to help ketone clearance and acid–base state.

- *Electrolyte balance*: although total body K+ is low, plasma K+ may be normal, high, or low. With treatment, K+ enters cells and plasma levels low—therefore, unless initial K+ levels are >5.5mmol/L, give 20mmol/ hr of potassium chloride (KCl), monitor ECG, and check K+ levels hourly.

- Despite the presence of metabolic acidosis, do not give sodium bicarbonate. Other electrolytes such as Ca2+, Mg2+, and phosphate (PO4 2–) are commonly disturbed but rarely need emergency correction.

Management

- Consider an NG tube to d the risk of gastric dilation and aspiration.

- Monitor urine output (most accurate with a urinary catheter).

- Consider a central venous catheter to monitor CVP to guide treatment in the elderly or severe illness.

- Arrange admission to ICU, HDU, or acute medical admissions unit.

Other aspects of management

1. Treat co-existing infection.
2. Prevent over rapid fluid administration.
3. Prophylactic anticoagulation
4. Treat HHS with 0.9% saline. Do not start insulin unless significant ketonaemia, or glucose does not fall with fluid therapy.