Clinical Practice Guidelines:
Medical/Diabetic emergency: Hyperglycaemia

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Date	February, 2023
Purpose	To ensure consistent management of patients with hyperglycaemia.
Scope	Applies to Queensland Ambulance Service (QAS) clinical staff.
Health care setting	Pre-hospital assessment and treatment.
Population	Applies to all ages unless stated otherwise.
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Diabetic emergency: Hyperglycaemia

February, 2023

A normal fasting blood glucose target for a person without diabetes is 3.9–5.6 mmol/L. In the diabetic patient hyperglycaemia can present as Diabetic Ketoacidosis (DKA), Hyperosmolar Hyperglycaemic Syndrome (HHS), or be asymptomatic.

DKA and HHS are predominantly caused by:[1]

- Acute illness (e.g. infection, ACS, CVA)
- Non-compliance with medication.

DKA is a life-threatening complication usually seen in patients with Type 1 Diabetes Mellitus, that is characterised by:[2]

- Hyperglycaemia
- Ketosis
- Metabolic acidosis.

It is caused by an ***absolute*** insulin deficiency, precipitating a number of physiological changes:

- High BGL increases blood osmolarity drawing water out of cells resulting in cellular dehydration.
- High BGL in the kidney filtrate results in osmotic diuresis and polyuria leading to severe dehydration & hypovolaemia. (fluid deficits typically range from 5–8 litres)[3]
- Alternative fuel sources, including fatty acids, are used, producing organic acids known as ketones. Accumulation of these result in a metabolic acidosis.[3]
- Dehydration leading to polydipsia.
- Loss of potasssium from the body.

HHS is a life-threatening complication of Type 2 Diabetes Mellitus (T2 DM) that is characterised by:

- Hyperglycaemia
- Hyperosmolarity
- Severe dehydration.

NOTE: *HHS has been known by numerous names, most notably Hyperosmolar Hyperglycaemic Nonketotic Syndrome (HHNS) and Hyperglycaemic Hyperosmolar Nonketotic Coma (HONK), however HHS is more widely accepted now, as coma is not a prerequisite and patients may present with some degree of ketosis.*

HHS is caused by a ***relative*** insulin deficiency, whereby there is sufficient insulin to limit ketone production thus preventing metabolic acidosis. It most commonly presents in patients > 60 and may be the primary presentation of T2 DM.[4–6] Grossly elevated BGLs still initiate the triad of polyuria, polydipsia and polyphagia, and fluid deficits typically range from 8–10 litres.[4] HHS has a greater rate of mortality due to the severity of underlying illness, typically sepsis.

Clinical features

The clinical features of DKA and HHS are similar:

Neurological:

- Lethargy
- ALOC
- Seizure
- Coma.

Cardiovascular:

- Signs of hypovolaemia (hypotension, tachycardia)
- Pale, cool or clammy or
- Flushed, hot if febrile.

The exceptions are:

- BGL: DKA (> 10 mmol/L) AND/OR HHS (> 40 mmol/L)
- Kussmaul respiration is due to the severe metabolic acidosis. This is not usually seen in HHS.

Risk Assessment

- Not applicable

Additional information

Mandatory transport criteria

If the patient has ANY of the following, they must be transported to hospital.

- Newly diagnosed diabetes
- Suspected cause of hyperglycaemia is due to the patient being affected by an illness that requires further investigation
- Inability to keep oral fluids down
- Moderate to severe dehydration
- Ketones > 1.5 mmol/L
- Pregnant

Note – Although treatment for severe dehydration may be required, correcting fluid deficits too quickly can cause cerebral oedema – especially in children.[7–9]