

[HOSPITAL / HEALTH AUTHORITY NAME]

DIABETIC AND ACUTE GLYCAEMIC EMERGENCIES PATHWAY

Protocol 27: Rapid Glucose Rescue, DKA / HHS Recognition, Fluid and Insulin Safety, Electrolyte Control, Complication Prevention, Transition, and Safe Disposition

DRAFT FOR EMERGENCY MEDICINE, INTERNAL MEDICINE, ENDOCRINOLOGY / DIABETES, PAEDIATRICS, OBSTETRICS, CRITICAL CARE, NEPHROLOGY, TOXICOLOGY, PHARMACY, LABORATORY, NURSING, EMS, TRANSFER, AND CLINICAL-GOVERNANCE REVIEW

STATUS: This is a draft clinical-governance document. Exact glucose thresholds, dextrose and glucagon products, fluid choices and rates, insulin infusion and subcutaneous regimens, potassium and phosphate replacement, laboratory intervals, paediatric and pregnancy pathways, cerebral-injury rescue treatment, intensive-care criteria, SGLT2-inhibitor policy, pump and continuous-glucose-monitoring procedures, transition regimens, discharge supplies, and transfer arrangements must be reconciled with current national guidance, local formulary, laboratory capability, specialist availability, and approved linked protocols before implementation.

IMMEDIATE SAFETY RULE: Measure point-of-care glucose immediately in every patient with altered mental status, seizure, collapse, focal neurological symptoms, behavioural change, shock, or unexplained critical illness. Treat hypoglycaemia without waiting for laboratory confirmation. DKA and HHS are not simply high-glucose states: safe treatment requires coordinated fluids, insulin, potassium, osmolality control, precipitant treatment, and repeated reassessment. Euglycaemic DKA may occur, especially with SGLT2 inhibitors, pregnancy, starvation, or reduced carbohydrate intake.

Document control	Details
Document owner	Emergency Department / Medical Services Directorate / Nursing Services / Clinical Governance
Clinical leads	Emergency Medicine; Internal Medicine; Endocrinology / Diabetes; Paediatrics; Critical Care
Supporting departments	Obstetrics; Nephrology; Cardiology; Infectious Diseases / Microbiology; Toxicology; Pharmacy; Laboratory; Dietetics; EMS; Transfer Coordination
Applies to	Adults, adolescents, children, and pregnant or postpartum patients presenting with hypoglycaemia, severe hyperglycaemia, ketosis, diabetic ketoacidosis, hyperosmolar hyperglycaemic state, or treatment-related glycaemic instability
Linked protocols	Altered Mental Status; Seizures; Stroke; Shock; Sepsis; Vomiting / Dehydration / Electrolytes; Acute Kidney Injury; Acute Coronary Syndrome; Poisoning / Overdose; Pregnancy Emergencies; Paediatric Emergency Assessment; Transfer
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1. Purpose

To provide a standardized emergency-department pathway for rapid recognition and treatment of hypoglycaemia, diabetic ketoacidosis (DKA), hyperosmolar hyperglycaemic state (HHS), mixed DKA / HHS, euglycaemic DKA, severe hyperglycaemia without crisis, and treatment-related glycaemic instability. The protocol prioritizes immediate glucose rescue, accurate metabolic classification, measured fluid and insulin therapy, potassium and osmolality safety, identification of the precipitating cause, prevention of neurological and cardiovascular complications, safe transition to subcutaneous treatment, and recurrence-prevention planning.

2. Scope

This protocol covers acute glycaemic emergencies in patients with known or previously undiagnosed diabetes, including patients using insulin injections, insulin pumps, automated insulin-delivery systems, continuous glucose monitoring, oral glucose-lowering medicines, GLP-1 receptor agonists, or SGLT2 inhibitors. It does not replace dedicated toxicology pathways for deliberate insulin or sulfonylurea overdose, paediatric DKA / HHS guidance, obstetric critical-care pathways, adrenal crisis protocols, starvation or alcoholic ketoacidosis pathways, or local inpatient diabetes policies, although these conditions may overlap and require parallel management.

3. Core policy statements

- Point-of-care glucose is a core component of the ABCDE assessment. A normal sensor reading does not exclude hypoglycaemia or hyperglycaemic crisis when perfusion is poor or symptoms are discordant; confirm with a meter or laboratory sample.
- Treat any clinically important hypoglycaemia immediately. Do not give oral carbohydrate to a patient with impaired consciousness, active seizure, unsafe swallowing, or aspiration risk.
- Use blood beta-hydroxybutyrate, when available, for DKA diagnosis and monitoring. Urine ketones may support diagnosis when blood ketone testing is unavailable but should not be used alone to declare resolution.
- DKA requires diabetes or hyperglycaemia plus significant ketonaemia and metabolic acidosis. HHS requires severe hyperglycaemia, hyperosmolality, minimal ketonaemia, and no substantial acidosis. Mixed presentations are common and should be treated according to the more dangerous component.
- Begin isotonic crystalloid promptly in DKA / HHS, but individualize rate and bolus size in frailty, pregnancy, heart failure, renal failure, dialysis, or risk of cerebral injury. Every fluid order requires a documented reassessment time.
- Check potassium before insulin whenever this can be done without unsafe delay. If potassium is below 3.5 mmol/L, replace potassium and defer insulin until the concentration is above the locally approved threshold.
- Insulin must continue until ketoacidosis has resolved, not merely until glucose normalizes. Add dextrose when glucose falls to the protocol threshold so that insulin can safely suppress ketogenesis.
- HHS requires slower correction than DKA. Track effective or total osmolality, sodium, glucose, fluid balance, and neurological status; avoid abrupt osmotic shifts.
- Routine bicarbonate and phosphate replacement are not indicated. Use only for defined severe indications under senior or critical-care oversight.
- In type 1 diabetes, avoid an unplanned period without basal insulin. Coordinate continuation or replacement of basal insulin during IV treatment and ensure overlap before stopping IV insulin.
- Suspect euglycaemic DKA in a symptomatic patient with acidosis and ketonaemia despite glucose below the usual DKA range, particularly with SGLT2 inhibitors, pregnancy, starvation, vomiting, reduced carbohydrate intake, or partial insulin treatment.
- Children and adolescents must be treated using the approved paediatric DKA / HHS pathway. Do not simply scale down the adult fluid or insulin regimen.
- Pregnancy-associated DKA can develop rapidly and at lower glucose concentrations. Maternal resuscitation, obstetric involvement, and fetal assessment must occur in parallel.
- Every episode requires a precipitant and recurrence-prevention review, including medication access, insulin delivery failure, infection, cardiovascular disease, psychosocial stress, food insecurity, cognitive impairment, substance use, and diabetes education.
- Safe discharge requires resolution of the acute emergency, a workable insulin and monitoring plan, access to medicines and supplies, sick-day and hypoglycaemia education, written return precautions, and named follow-up responsibility.

4. Definitions and diagnostic framework

Term	Operational definition
Level 1 hypoglycaemia	Glucose below 3.9 mmol/L (70 mg/dL) and at or above 3.0 mmol/L (54 mg/dL). Clinically important and requires action.
Level 2 hypoglycaemia	Glucose below 3.0 mmol/L (54 mg/dL). Requires immediate treatment whether or not symptoms are present.
Level 3 hypoglycaemia	A severe event with altered mental and/or physical function requiring assistance from another person, irrespective of the measured glucose.
Diabetic ketoacidosis	All three: diabetes or glucose at least 11.1 mmol/L (200 mg/dL); beta-hydroxybutyrate at least 3.0 mmol/L or urine ketones at least 2+; and venous pH below 7.3 and/or bicarbonate below 18 mmol/L.
Hyperosmolar hyperglycaemic state	All four: glucose at least 33.3 mmol/L (600 mg/dL); effective osmolality above 300 mOsm/kg or total osmolality above 320 mOsm/kg; beta-hydroxybutyrate below 3.0 mmol/L or urine ketones 2+ or less; and pH at least 7.3 with bicarbonate at least 15 mmol/L.
Mixed DKA / HHS	Hyperosmolality with significant ketonaemia and metabolic acidosis. Manage with DKA-dose insulin plus careful HHS-style osmolality monitoring.
Euglycaemic DKA	DKA-level ketonaemia and acidosis with glucose below 11.1 mmol/L (200 mg/dL), commonly associated with SGLT2 inhibitors, pregnancy, starvation, vomiting, or partial insulin treatment.
Ketosis without acidosis	Raised blood or urine ketones without DKA-level acidosis. May represent early DKA, starvation, illness, or inadequate insulin and requires cause-directed treatment and reassessment.
Effective osmolality	Calculated in SI units as $2 \times \text{sodium (mmol/L)} + \text{glucose (mmol/L)}$. Urea is excluded because it is not an effective osmole.

Term	Operational definition
Total calculated osmolality	Calculated in SI units as 2 x sodium + glucose + urea, all in mmol/L. Use the same formula consistently throughout treatment.

5. Roles and accountability

Role	Minimum responsibility
Triage / first-contact clinician	Obtain immediate glucose; identify severe hypoglycaemia, shock, Kussmaul breathing, dehydration, altered consciousness, pregnancy, insulin-pump use, SGLT2 exposure, and need for immediate resuscitation.
Lead ED clinician	Direct ABCDE care; classify hypoglycaemia, DKA, HHS, mixed crisis, or non-crisis hyperglycaemia; prescribe fluids, insulin, dextrose, and electrolyte monitoring; identify precipitants; activate specialist and transfer pathways.
Nursing team	Provide continuous monitoring, administer glucose rescue and infusions, record fluid balance and neurological status, obtain scheduled bedside and laboratory tests, use double checks for insulin and concentrated electrolytes, and escalate deviation from targets.
Endocrinology / diabetes team	Support crisis classification, insulin strategy, pump / technology management, transition, education, medication review, and recurrence prevention.
Paediatrics / obstetrics	Direct age-specific or pregnancy-specific pathways, fetal or neurological monitoring, and disposition.
Critical care / nephrology	Support severe acidosis, shock, altered consciousness, respiratory failure, potassium emergency, cerebral injury, renal failure, dialysis, or difficult fluid balance.
Pharmacy / laboratory	Maintain rapid glucose, ketone, blood-gas, electrolyte, and osmolality testing; support insulin and electrolyte safety, antidotal or toxicology treatment, and formulary-approved preparations.
Receiving / transfer team	Accept responsibility explicitly and ensure uninterrupted insulin, dextrose, potassium, monitoring, and fluid therapy during transfer.

6. Pathway activation and triage

Category	Operational criteria
RED / immediate resuscitation	Glucose-related seizure or coma; severe hypoglycaemia; shock; severe DKA; HHS with altered consciousness; potassium-related ECG change; pH below 7.0; respiratory fatigue; cerebral-injury concern; pregnancy with suspected DKA; child with suspected DKA / HHS.
ORANGE / very urgent	Moderate DKA; mixed DKA / HHS; persistent vomiting with ketonaemia; recurrent hypoglycaemia; sulfonylurea or long-acting insulin exposure; pump failure with ketones; new type 1 diabetes; significant renal or cardiac comorbidity.
YELLOW / urgent	Stable severe hyperglycaemia without acidosis; mild DKA in a monitored pathway; ketosis without acidosis; corrected hypoglycaemia requiring cause assessment or observation.
GREEN / lower acuity only after screening	Mild asymptomatic hyperglycaemia without ketones, acidosis, dehydration, precipitating emergency, pregnancy, or high-risk social / treatment concern. Clinician assessment and follow-up remain required.

7. First 10 minutes: parallel action

1. Begin ABCDE assessment, cardiac monitoring, pulse oximetry, temperature, repeat vital signs, and immediate point-of-care glucose.
2. If glucose is low or severe hypoglycaemia is clinically suspected, treat immediately while obtaining vascular access and a confirmatory sample; do not delay for laboratory results.
3. Establish one or two IV lines; use IO access if critically ill and IV access is not promptly available. Obtain an accurate weight or best documented recent weight for dosing.
4. Obtain venous blood gas, electrolytes, urea / creatinine, glucose, beta-hydroxybutyrate, CBC, and ECG. Add measured or calculated osmolality, magnesium, phosphate, pregnancy test, cultures, troponin, lipase, toxicology, or imaging according to presentation.

5. If DKA / HHS is suspected, begin isotonic crystalloid after initial samples without delaying resuscitation. Use smaller aliquots and more frequent reassessment in frailty, heart failure, renal failure, dialysis, or pregnancy.
6. Check potassium before starting insulin. Activate the local potassium-replacement pathway if below 3.5 mmol/L.
7. Identify and treat immediate precipitants in parallel: sepsis, acute coronary syndrome, stroke, pancreatitis, pregnancy complication, medication omission, pump failure, overdose, or SGLT2-associated euglycaemic DKA.
8. Escalate early to senior ED, endocrinology / diabetes, paediatrics, obstetrics, critical care, nephrology, toxicology, or transfer coordination as indicated.

8. Immediate stabilization: ABCDE

8.1 Airway and breathing

- Protect the airway in coma, recurrent seizure, vomiting with impaired protective reflexes, or respiratory fatigue. Prepare for aspiration risk.
- Kussmaul respiration is compensatory. Do not suppress it with sedation. Intubation in severe metabolic acidosis is high risk and requires senior airway planning, preoxygenation, haemodynamic preparation, and maintenance of adequate minute ventilation.
- Give oxygen for hypoxaemia or shock, not routinely for normal saturation. Search for pneumonia, pulmonary oedema, pulmonary embolism, or aspiration when clinically indicated.

8.2 Circulation

- Assess pulse, blood pressure, capillary refill, JVP, lung fields, peripheral oedema, urine output, and signs of shock. Continuous ECG monitoring is required for severe potassium disturbance, significant acidosis, or insulin infusion.
- Use isotonic saline or a balanced crystalloid according to the approved local pathway. Reassess after each bolus or defined fluid phase for perfusion, pulmonary congestion, sodium, osmolality, and urine output.
- Treat shock, sepsis, myocardial infarction, haemorrhage, or another cause of circulatory failure in parallel. Hyperglycaemia does not explain all hypotension.

8.3 Disability and exposure

- Record GCS or AVPU, pupils, focal deficit, seizure activity, behaviour, and serial neurological status. Persistent or worsening alteration despite improving osmolality requires investigation for stroke, infection, intoxication, hypoxia, or cerebral injury.
- Examine for dehydration, infection, insulin injection or pump sites, abdominal tenderness, pancreatitis, foot infection, pressure injury, trauma, pregnancy, and signs of endocrine or toxicological disease.

9. Hypoglycaemia: immediate rescue and recurrence prevention

Clinical situation	Immediate action and reassessment
Conscious, cooperative, safe swallow	Give 15-20 g rapid-acting glucose or carbohydrate. Recheck glucose after 10-15 minutes and repeat if still below 4.0 mmol/L. Once recovered, give a meal or longer-acting carbohydrate unless contraindicated, and review the cause and next insulin / medicine dose.
Impaired consciousness, seizure, unsafe swallow, or severe agitation	No oral treatment. Stop any running insulin infusion. Give formulary-approved IV glucose; if IV / IO access is unavailable, give approved glucagon preparation. Recheck every 10-15 minutes, repeat rescue as needed, protect the airway, and consider a dextrose infusion.
Sulfonylurea or meglitinide exposure	Expect delayed or recurrent hypoglycaemia, especially with renal impairment or older age. Discuss prolonged observation, serial glucose, octreotide, toxicology advice, and admission. Do not rely on a single corrected glucose.
Long-acting insulin overdose or dosing error	Anticipate prolonged recurrence. Admit or observe with frequent glucose, carbohydrate / dextrose support, potassium monitoring, and specialist or toxicology review.
Acarbose use	Use pure glucose / dextrose rather than sucrose because acarbose impairs sucrose breakdown.
Alcohol use, malnutrition, liver disease, or prolonged fasting	Glucagon may be less effective because glycogen stores are depleted. Prefer IV glucose when access is available and assess thiamine, nutrition, liver disease, sepsis, and adrenal insufficiency as clinically indicated.
Insulin pump / automated delivery	Confirm with a bedside meter. Suspend automated insulin delivery during severe hypoglycaemia if the patient cannot self-manage, but establish a safe basal-insulin plan promptly once recovered, especially in type 1 diabetes.

HYPOGLYCAEMIA SAFETY: A glucose value below 4.0 mmol/L in a patient treated with insulin or a secretagogue should be treated in the ED. Level 2 or level 3 episodes, recurrent events, impaired awareness, renal failure, pregnancy, injury, social vulnerability, or uncertain medication access require senior review and a prevention plan before discharge.

10. DKA, HHS, and mixed-crisis diagnosis

Feature	DKA	HHS
Glucose / diabetes	Known diabetes or glucose at least 11.1 mmol/L (200 mg/dL). Glucose may be lower in euglycaemic DKA.	Glucose at least 33.3 mmol/L (600 mg/dL).
Ketones	Beta-hydroxybutyrate at least 3.0 mmol/L or urine ketones at least 2+.	Beta-hydroxybutyrate below 3.0 mmol/L or urine ketones no more than 2+.
Acid-base	Venous pH below 7.3 and/or bicarbonate below 18 mmol/L.	pH at least 7.3 and bicarbonate at least 15 mmol/L.
Osmolality	May be normal or raised; calculate when glucose is very high, mental status is altered, or overlap is suspected.	Effective osmolality above 300 mOsm/kg or total osmolality above 320 mOsm/kg.
Typical tempo	Hours to one or two days; nausea, vomiting, abdominal pain, Kussmaul breathing, dehydration.	Days to weeks; profound dehydration, neurological change, weakness, infection, thrombosis.
Overlap	If hyperosmolality is present with DKA-level ketonaemia / acidosis, classify as mixed DKA / HHS.	Mixed presentations require DKA-dose insulin and HHS osmolality precautions.

11. Adult DKA severity and location of care

Severity	Suggested biochemical / clinical features	Minimum care level
Mild	Beta-hydroxybutyrate 3.0-6.0 mmol/L; pH above 7.25 to below 7.30 or bicarbonate 15-18 mmol/L; alert; no shock, major comorbidity, or high-risk social concern.	Monitored ED / observation or ward pathway with trained staff and rapid laboratory support. Selected uncomplicated cases may use a locally approved subcutaneous rapid-acting insulin pathway.
Moderate	Beta-hydroxybutyrate 3.0-6.0 mmol/L; pH 7.0-7.25 or bicarbonate 10 to below 15 mmol/L; alert or drowsy.	High-observation area, step-down, or ICU according to staffing, comorbidity, and response.
Severe	Beta-hydroxybutyrate above 6.0 mmol/L; pH below 7.0 or bicarbonate below 10 mmol/L; stupor / coma, shock, respiratory fatigue, or major precipitant.	ICU / critical care with senior endocrine and emergency oversight.

Severity components need not align perfectly. Use the most severe biochemical, neurological, haemodynamic, and contextual feature to determine care level.

12. Focused history and examination

Domain	Key questions / findings
Diabetes and treatment	Type and duration; insulin and non-insulin medicines; last doses; basal insulin; recent changes; access or affordability; pump / CGM model; infusion-set failure; recent hypoglycaemia; prior DKA / HHS; usual control.
Symptoms and timing	Polyuria, polydipsia, weight loss, nausea, vomiting, abdominal pain, dyspnoea, weakness, confusion, focal symptoms, seizure, fever, chest pain, diarrhoea, reduced intake, pregnancy symptoms.
Precipitants	Infection; missed insulin; new diabetes; myocardial infarction; stroke; pancreatitis; trauma; surgery; glucocorticoids; antipsychotics; SGLT2 inhibitor; alcohol or stimulant use; insulin / sulfonylurea overdose; pregnancy; psychosocial crisis.
Volume and renal status	Fluid intake and losses, urine output, CKD, dialysis, heart failure, cirrhosis, diuretics, weight change.
Examination	ABCDE; hydration and perfusion; Kussmaul breathing; mental status; focal neurology; infection source; abdomen; feet and skin; pump / injection sites; pregnancy; signs of fluid overload.

13. Investigations

Investigation	Purpose / interpretation
Immediate bedside	Capillary glucose, blood beta-hydroxybutyrate if available, ECG, pulse oximetry, temperature, weight, neurological score, urine output. Confirm extreme or discordant bedside values with laboratory testing.
Core laboratory	Venous blood gas, sodium, potassium, chloride, bicarbonate, urea, creatinine, laboratory glucose, magnesium, phosphate, CBC. Calculate anion gap and osmolality but do not use anion gap alone to diagnose or declare DKA resolution.
Cause-directed	Pregnancy test; blood / urine cultures; urinalysis; chest imaging; viral testing; troponin; lipase; liver tests; lactate; toxicology; cortisol; thyroid tests; CK; CT or other imaging as indicated.
Monitoring	Bedside glucose every 1-2 hours during crisis; electrolytes, renal function, beta-hydroxybutyrate, and venous pH approximately every 4 hours or more often if unstable. In HHS, calculate or measure osmolality at least every 4 hours and track sodium and fluid balance.

14. Identify and treat the precipitating cause

Common precipitant	Required action
Insulin omission, access problem, or pump failure	Restore reliable insulin delivery; inspect device and site; address affordability, supplies, education, cognition, mental health, and safeguarding. Do not label as non-adherence without investigating barriers.
Infection / sepsis	Obtain targeted cultures and imaging, give timely antimicrobials when indicated, and apply sepsis source-control principles. Leukocytosis alone may reflect stress and does not prove infection.
Acute coronary syndrome / stroke / thrombosis	Obtain ECG and cause-directed tests. Activate linked cardiovascular or neurological pathways. HHS carries substantial thrombotic risk.
Pancreatitis / abdominal disease	Use clinical findings and lipase selectively; abdominal pain may be caused by DKA but persistent or focal pain after correction requires further evaluation.
Medication-related	Stop SGLT2 inhibitor during suspected DKA. Review glucocorticoids, diuretics, antipsychotics, immunotherapy, enteral / parenteral nutrition, and recent insulin or sulfonylurea changes.
Pregnancy / postpartum	Activate obstetric and endocrine review; assess fetal status after maternal stabilization; investigate hyperemesis, infection, steroid exposure, and insulin-delivery interruption.
Psychosocial / intentional harm	Assess depression, self-harm, eating disorder, substance use, domestic safety, cognitive impairment, and access to food, refrigeration, housing, and care.

15. Adult DKA management

15.1 Fluids

- In adults without cardiac or renal compromise, begin isotonic saline or a balanced crystalloid at approximately 500-1,000 mL/hour during the first 2-4 hours, guided by perfusion, sodium, urine output, and clinical response.
- Use smaller boluses, such as 250 mL with frequent reassessment, in older or frail adults, heart failure, advanced kidney disease, dialysis, pregnancy, or risk of fluid overload. Do not force a standard volume when the response indicates harm.
- After initial restoration of perfusion, select fluid composition and rate according to haemodynamics, sodium trend, osmolality, urine output, oral intake, and cumulative balance.

15.2 Insulin and dextrose

- After initial fluids and confirmation that potassium is at least 3.5 mmol/L, start a fixed-rate IV insulin infusion at 0.1 units/kg/hour using an approved double-check and pump process. Routine IV insulin bolus is not required when infusion can start promptly.
- For selected uncomplicated mild or moderate DKA, a locally approved protocol may use subcutaneous rapid-acting insulin every 1-2 hours in a closely monitored area with trained staff and reliable testing. Do not use this approach for severe DKA, HHS, pregnancy, shock, altered consciousness, or major comorbidity.
- When glucose falls below approximately 13.9 mmol/L (250 mg/dL), add 5% or 10% dextrose according to the local pathway and reduce IV insulin to approximately 0.05 units/kg/hour. Continue insulin until ketonaemia and acidosis resolve.
- Do not stop insulin because glucose has normalized while ketones or acidosis persist. Adjust dextrose rather than prematurely stopping insulin.

15.3 Potassium, bicarbonate, and phosphate

- Follow the potassium algorithm in Section 19. Insulin, correction of acidosis, and improved perfusion may rapidly lower serum potassium despite a large total-body deficit.
- Do not give bicarbonate routinely. Consider only for severe acidemia, generally pH below 7.0, under senior and critical-care direction with potassium and sodium monitoring.
- Do not replace phosphate routinely. Consider replacement for severe hypophosphataemia with muscle weakness, respiratory compromise, cardiac dysfunction, or another defined indication, while monitoring calcium.

16. Adult HHS management

- HHS usually involves a larger water deficit and a more vulnerable patient than DKA. Start isotonic crystalloid, but titrate to haemodynamics, comorbidity, sodium, osmolality, urine output, and neurological status.
- Aim for gradual correction. A commonly used target is an osmolality fall of approximately 3-8 mOsm/kg/hour, a sodium fall no greater than 10 mmol/L in 24 hours, and a glucose fall of approximately 2.8-5.0 mmol/L/hour (50-90 mg/dL/hour). Use the locally approved range consistently.
- An initial rise in measured sodium as glucose falls may be expected and is not by itself an indication for hypotonic fluid if osmolality is falling appropriately. If osmolality is not falling despite adequate positive fluid balance, obtain senior advice before changing fluid tonicity.
- After initial fluid resuscitation and potassium assessment, use IV insulin at approximately 0.05 units/kg/hour when glucose is no longer falling adequately with fluids alone or when mild ketonaemia is present. If DKA-level ketonaemia or acidosis is present, treat as mixed DKA / HHS with 0.1 units/kg/hour.
- Add dextrose when glucose approaches approximately 14-17 mmol/L (250-300 mg/dL), while continuing the insulin and fluid strategy needed to resolve hyperosmolality.
- Provide pharmacological venous-thromboembolism prophylaxis unless contraindicated and investigate arterial or venous thrombosis when clinically suspected.
- HHS commonly requires high-dependency or intensive-care monitoring, especially with altered consciousness, very high osmolality, major comorbidity, shock, renal failure, or poor response.

17. Mixed DKA / HHS and euglycaemic DKA

Condition	Management priorities
Mixed DKA / HHS	Treat with DKA-dose insulin, potassium safeguards, and enough dextrose to continue insulin until ketosis resolves. At the same time, track osmolality and sodium like HHS and avoid rapid fluid or osmotic shifts.
SGLT2-associated euglycaemic DKA	Stop the SGLT2 inhibitor. Confirm ketonaemia and acidosis even if glucose is normal or modestly raised. Start insulin with early dextrose, replace fluids and potassium, identify the trigger, and obtain endocrine review. Do not restart the drug during the acute episode.
Pregnancy-associated euglycaemic DKA	Activate obstetric, endocrine, and critical-care teams immediately. Use maternal physiology and ketones, not glucose alone, to guide treatment. Add dextrose early enough to continue insulin and protect against hypoglycaemia.
Starvation / alcoholic ketoacidosis	Consider when glucose is low or normal and the history fits. Give thiamine when indicated, dextrose, fluids, and electrolytes; use insulin only when true DKA or significant hyperglycaemia coexists. Follow the toxicology / metabolic pathway.

18. Potassium and other electrolyte safety

Serum potassium	Action before and during insulin
Below 3.5 mmol/L	Defer insulin. Begin monitored potassium replacement, commonly around 10 mmol/hour or according to the approved local high-risk electrolyte pathway, until potassium is above 3.5 mmol/L. Obtain senior / critical-care input for severe hypokalaemia, ECG change, renal impairment, or high replacement requirement.
3.5-5.0 mmol/L	Begin insulin and usually add 20-30 mmol potassium per litre of IV fluid, adjusted to urine output and renal function, to maintain potassium around 4.0-5.0 mmol/L.
Above 5.0 mmol/L	Do not add potassium initially. Start insulin if otherwise safe and recheck potassium frequently, generally within 2 hours, because the concentration may fall rapidly.
Anuria, dialysis, or severe renal failure	Do not use routine potassium-containing fluids. Obtain nephrology / critical-care advice and use ECG, repeated potassium, volume status, and dialysis access to guide therapy.

- Replace magnesium when clinically important, especially with refractory hypokalaemia, arrhythmia, torsades, or documented deficiency.
- Correct sodium and osmolality gradually. Use one approved calculation method and trend it consistently rather than reacting to isolated values.
- Monitor phosphate, calcium, and magnesium in severe or prolonged crises, malnutrition, respiratory weakness, cardiac dysfunction, or refeeding risk.

19. Neurological deterioration and cerebral injury

NEUROLOGICAL EMERGENCY: New headache, irritability, slowing heart rate, rising blood pressure, recurrent vomiting, reduced consciousness, cranial-nerve abnormality, incontinence, focal deficit, seizure, or respiratory change during treatment may indicate cerebral injury or another intracranial process. Stop nonessential transport, obtain immediate senior / critical-care review, and treat suspected cerebral injury without waiting for imaging when the clinical picture is convincing.

- Cerebral injury is more common in children but may occur in adults, particularly younger adults or during rapid osmotic correction.
- Follow the approved paediatric or adult rescue protocol for hypertonic saline or mannitol. Elevate the head, avoid hypotension and hypoxia, reassess fluids and osmotic trends, and arrange urgent neuroimaging when stable enough.
- Consider stroke, venous thrombosis, infection, hypoglycaemia, sodium disorder, intoxication, and seizure as alternative or concurrent causes.

20. Paediatric DKA and HHS

- Activate the paediatric DKA / HHS pathway immediately. Record weight, GCS, perfusion, dehydration, glucose, ketones, venous pH, bicarbonate, sodium, potassium, and urine output.
- Do not give an insulin bolus. Begin IV insulin only after initial fluid therapy and according to the approved weight-based paediatric regimen, commonly 0.05-0.1 units/kg/hour.
- Replace estimated fluid deficit and maintenance in a controlled manner using the paediatric protocol. Reassess frequently for cerebral injury, fluid overload, potassium shift, and falling glucose.
- Add dextrose while insulin continues until ketonaemia and acidosis resolve. Use blood beta-hydroxybutyrate where available.
- Suspected cerebral injury requires immediate hyperosmolar treatment according to the paediatric rescue protocol; do not wait for CT.
- Paediatric HHS requires PICU and paediatric endocrine involvement, slower osmolality correction, aggressive but carefully monitored fluid replacement, and lower / delayed insulin dosing than DKA.

21. Pregnancy and postpartum considerations

- DKA may develop at lower glucose concentrations and progress rapidly. Check ketones and venous blood gas early in pregnant patients with diabetes who have vomiting, abdominal pain, dyspnoea, infection, reduced intake, hyperglycaemia, or fetal concern.
- Prioritize maternal ABCDE stabilization, fluids, insulin, potassium, and treatment of the precipitant. Involve obstetrics, endocrinology, anaesthesia / critical care, and neonatology as appropriate.
- Begin fetal assessment after maternal resuscitation when gestation is viable and resources allow. Fetal abnormalities often improve as maternal acidosis and perfusion improve; avoid delivery solely for transient fetal distress unless a separate obstetric indication exists.
- Consider euglycaemic DKA with SGLT2 exposure, starvation, hyperemesis, infection, corticosteroids, beta-agonists, or interrupted insulin. Use dextrose early enough to continue insulin.
- Hypoglycaemia in pregnancy requires prompt rescue and medication review, with a lower threshold for observation when recurrent or unexplained.

22. Insulin pumps, CGM, and automated insulin delivery

Issue	ED action
CGM reading discordant with symptoms	Check capillary or venous glucose. Poor perfusion, compression, sensor lag, medication interference, and device failure may cause misleading readings.
DKA in pump user	Assume insulin delivery failure until proven otherwise. Stop and remove or disconnect the pump according to local policy, inspect the cannula / reservoir / tubing, start standard IV insulin, and document the device and settings.
Severe hypoglycaemia in pump user	Suspend automated insulin delivery if the patient cannot self-manage, give rescue glucose, and involve the diabetes team before resumption. Prevent prolonged interruption of basal insulin in type 1 diabetes.
Restarting device	Restart only after the crisis has resolved, the patient is alert and competent, supplies are available, the infusion site is replaced when appropriate, settings are verified, and an overlap plan prevents insulin interruption.

Issue	ED action
Device during critical illness	Do not rely on patient-managed technology when consciousness, perfusion, cognition, staffing, imaging, procedures, or medication exposure makes it unsafe. Use hospital-approved monitoring and insulin systems.

23. Severe hyperglycaemia without DKA or HHS

- Confirm the result, assess hydration, ketones, acid-base status, osmolality when indicated, and the precipitating cause. Do not diagnose DKA or HHS from glucose alone.
- Provide oral or IV fluids according to clinical volume status. Treat infection, steroid effect, medication interruption, nutrition-related hyperglycaemia, or another cause.
- Use subcutaneous correction and basal insulin according to the approved local regimen. Routine IV insulin is not required for stable hyperglycaemia without crisis.
- New suspected type 1 diabetes, significant ketonaemia, weight loss, pregnancy, inability to self-manage, unreliable follow-up, or marked symptoms usually requires diabetes-team review and admission or structured urgent follow-up.
- Avoid rapid overcorrection and hypoglycaemia. A high glucose reading without symptoms is rarely an indication for repeated unmonitored correction doses.

24. Monitoring and treatment targets

Parameter	Minimum monitoring expectation
Glucose	Every 10-15 minutes during active hypoglycaemia rescue until stable; every 1-2 hours during IV insulin or acute DKA / HHS treatment.
Vital signs and neurological status	At least hourly in crisis, more frequently when unstable. Record GCS / AVPU and any new focal or cerebral-injury signs.
Electrolytes, renal function, pH, bicarbonate, beta-hydroxybutyrate	Approximately every 4 hours in stable treatment; every 2 hours or more frequently when potassium is abnormal, renal function is changing, severe DKA is present, or response is off target.
Osmolality and sodium in HHS	At least every 4 hours, with calculation after each relevant laboratory panel. Track rate of change rather than isolated results.
Fluid balance	Hourly intake, urine output, losses, cumulative balance, weight when feasible, and signs of congestion or hypoperfusion.
Insulin / dextrose / potassium infusions	Hourly verification of correct patient, concentration, line, pump rate, compatibility, and planned laboratory review. Use independent double checks according to medication-safety policy.

25. Resolution criteria and transition from IV insulin

Emergency	Resolution criteria
DKA	Beta-hydroxybutyrate below 0.6 mmol/L and venous pH at least 7.3 or bicarbonate at least 18 mmol/L. Glucose should be controlled, but do not use glucose or anion gap alone to declare resolution.
HHS	Calculated or measured osmolality below 300 mOsm/kg, glucose below 13.9 mmol/L (250 mg/dL), urine output above 0.5 mL/kg/hour, and cognitive status improved toward baseline.
Hypoglycaemia	Glucose remains above the treatment threshold without repeated rescue, symptoms have resolved, a sustaining meal / infusion plan is in place, and the cause and next medication dose have been addressed.

- Give scheduled subcutaneous basal insulin 1-2 hours before stopping IV insulin, or use the locally approved overlap interval. Longer overlap may be required for some basal preparations.
- Base the transition regimen on prior treatment, weight, renal function, nutritional intake, pregnancy, steroid exposure, and hypoglycaemia risk. Avoid discharge using correction-only insulin for a patient who requires basal insulin.
- Ensure the patient can eat or has a defined nutrition plan, and that glucose, potassium, renal function, and the precipitating illness are stable.
- If the home regimen was unsafe or ineffective, do not simply restart it without review.

26. Disposition

Disposition	Minimum criteria
ICU / high dependency	Severe DKA; HHS with neurological change; shock; respiratory failure; pH below 7.0; severe potassium disturbance; cerebral-injury concern; pregnancy with DKA; mixed crisis; major precipitant; dialysis; need for very frequent monitoring or vasopressors.
Inpatient ward / observation	Mild or moderate DKA in an approved pathway; recurrent or prolonged hypoglycaemia; ketonaemia with ongoing vomiting; severe hyperglycaemia with dehydration or new insulin requirement; unstable comorbidity; unresolved cause; social or treatment-safety concern.
Discharge after hypoglycaemia	Sustained recovery, no recurrent low during an appropriate observation period, cause identified and corrected, safe medication plan, meal access, supervision, supplies and glucagon when indicated, and timely follow-up.
Discharge after uncomplicated hyperglycaemia	No DKA / HHS, hydration restored, clinically stable, no dangerous precipitant, workable insulin / medicine plan, ability to monitor, education completed, supplies available, and named follow-up.

27. Discharge education and recurrence prevention

- Provide written sick-day rules: continue basal insulin in type 1 diabetes, monitor glucose more often, check blood or urine ketones during illness or persistent hyperglycaemia, maintain carbohydrate and fluid intake, and know when to seek urgent care.
- Explain hypoglycaemia recognition and treatment, the 15-g / 15-minute approach when safe, use of glucagon, driving and occupational restrictions after severe events, and the need to review insulin / secretagogue therapy.
- Review SGLT2-inhibitor sick-day interruption and peri-procedural policy. Patients with SGLT2-associated DKA require prescriber review before any restart decision.
- Confirm access to insulin, needles, pump supplies, glucose meter and strips, ketone testing, CGM supplies, glucagon, food, refrigeration, transport, and communication.
- Use teach-back. Document who will follow the patient, when, and how pending tests or culture results will be communicated. Arrange diabetes-team contact within an appropriate timeframe, commonly 24-72 hours after a crisis or major regimen change.

28. Transfer and handover

- Stabilize as far as practicable before transfer, but do not delay definitive care when local monitoring, ICU, paediatric, obstetric, dialysis, or specialist capability is inadequate.
- Continue insulin, dextrose, fluids, and potassium during transport using compatible pumps and trained personnel. Avoid interruption of insulin in unresolved DKA.
- Communicate diagnosis and severity, precipitant, weight, glucose / ketone / pH / bicarbonate / potassium / sodium / osmolality trends, fluids and balance, insulin / dextrose / potassium rates, urine output, neurological status, device information, pregnancy status, and outstanding risks.
- Confirm receiving clinician, destination, transport monitoring, infusion supply, repeat-test timing, and contingency actions for hypoglycaemia, potassium change, neurological deterioration, or pump failure.

29. Documentation and handover

- Record the initial glucose and time, diagnostic criteria, severity, weight, comorbidity, pregnancy status, pump / CGM use, SGLT2 exposure, and suspected precipitant.
- Document every fluid bolus and phase, insulin rate, dextrose start, potassium decision, laboratory trend, osmolality trend, urine output, neurological reassessment, and response to treatment.
- Record resolution criteria explicitly before transition, the overlap between IV and subcutaneous insulin, the final medication plan, education, supplies, follow-up, and pending-result ownership.
- Use closed-loop verbal and written handover at every transfer of care. The receiving clinician must know the next glucose and laboratory due times.

30. Quality indicators and audit

Indicator	Suggested standard
Glucose documented promptly in altered mental status, seizure, collapse, or critical illness	At least 95%
Hypoglycaemia treated without avoidable delay and rechecked within 15 minutes	At least 95%
DKA / HHS diagnostic criteria and severity documented	At least 90%
Potassium checked and acted on before insulin	At least 95%

Indicator	Suggested standard
Hourly glucose and required laboratory monitoring completed during IV insulin	At least 90%
HHS osmolality trend documented with safe correction rate	At least 90%
Precipitant identified or actively investigated	At least 90%
Subcutaneous insulin overlap documented before IV insulin stopped	At least 95%
Discharge sick-day / hypoglycaemia education and follow-up documented	At least 90%
Recurrent crisis, severe hypoglycaemia, cerebral injury, ICU transfer, or death reviewed	100% case review

31. Training and implementation

- Annual competency should include bedside glucose rescue, insulin infusion and pump safety, potassium replacement, DKA / HHS classification, osmolality calculation, cerebral-injury recognition, paediatric escalation, and transition planning.
- Use standardized order sets, flow sheets, infusion labels, double-checks, and bedside algorithms. Ensure 24-hour access to glucose, beta-hydroxybutyrate or urine ketones, blood gas, electrolytes, ECG, dextrose, glucagon, insulin, potassium, and transfer support.
- Run multidisciplinary simulations for severe hypoglycaemia, adult DKA, HHS, paediatric DKA with cerebral injury, pregnancy-associated euglycaemic DKA, and insulin-pump failure.
- Review recurrent presentations for structural barriers, including medicine cost, clinic access, food insecurity, health literacy, mental health, and supply-chain failure.

ANNEX A. One-page workflow

Step	Action
1. Recognize	Immediate glucose in altered, collapsed, seizing, focal-neurological, shocked, or critically ill patients. Look for dehydration, Kussmaul breathing, vomiting, polyuria, confusion, pump failure, pregnancy, and SGLT2 exposure.
2. Rescue low glucose	If unsafe swallow: IV glucose or glucagon. If safe swallow: 15-20 g rapid glucose. Recheck in 10-15 minutes and repeat until stable.
3. Classify hyperglycaemia	Obtain beta-hydroxybutyrate, venous pH / bicarbonate, electrolytes, renal function, and osmolality. Distinguish DKA, HHS, mixed crisis, euglycaemic DKA, ketosis without acidosis, and non-crisis hyperglycaemia.
4. Stabilize	Isotonic fluid, cardiac monitoring, urine output, potassium check, and precipitant treatment. Use smaller fluid aliquots in frailty, cardiac / renal disease, dialysis, and pregnancy.
5. Insulin safely	DKA / mixed: 0.1 units/kg/hour. HHS without acidosis: approximately 0.05 units/kg/hour after fluids / potassium assessment. Delay insulin if potassium below 3.5 mmol/L.
6. Add dextrose	Add dextrose when glucose reaches the protocol threshold so insulin can continue until ketones / osmolality resolve.
7. Monitor	Glucose every 1-2 hours; electrolytes, pH, ketones approximately every 4 hours; osmolality in HHS; hourly neurological and fluid-balance review.
8. Resolve and transition	Meet biochemical and clinical resolution criteria; overlap basal subcutaneous insulin before stopping IV insulin; address precipitant, education, supplies, and follow-up.

ANNEX B. Diagnostic and calculation card

Item	Formula / threshold
DKA	Diabetes or glucose at least 11.1 mmol/L + beta-hydroxybutyrate at least 3.0 mmol/L / urine ketones at least 2+ + pH below 7.3 and/or bicarbonate below 18 mmol/L.
HHS	Glucose at least 33.3 mmol/L + effective osmolality above 300 or total osmolality above 320 + beta-hydroxybutyrate below 3.0 + pH at least 7.3 and bicarbonate at least 15.
Effective osmolality, SI	$2 \times \text{Na (mmol/L)} + \text{glucose (mmol/L)}$.

Item	Formula / threshold
Total osmolality, SI	$2 \times \text{Na} + \text{glucose} + \text{urea}$, all mmol/L.
Anion gap	$\text{Na} - (\text{Cl} + \text{bicarbonate})$. Useful for context but not the preferred sole marker of DKA diagnosis or resolution.
DKA resolution	Beta-hydroxybutyrate below 0.6 and pH at least 7.3 or bicarbonate at least 18.
HHS resolution	Osmolality below 300, glucose below 13.9, urine output above 0.5 mL/kg/hour, and cognition improved.

ANNEX C. Hypoglycaemia rescue card

- [] Check ABCDE and bedside glucose; stop any insulin infusion.
- [] Safe swallow: give 15-20 g rapid-acting glucose / carbohydrate.
- [] Unsafe swallow, seizure, or unconscious: no oral treatment; give local IV glucose or glucagon if no vascular access.
- [] Recheck glucose after 10-15 minutes; repeat treatment until above 4.0 mmol/L and recovered.
- [] Give sustaining carbohydrate / meal or dextrose infusion as appropriate.
- [] Identify cause: insulin timing / dose, secretagogue, renal failure, reduced intake, alcohol, infection, exercise, pregnancy, overdose.
- [] Observe longer for sulfonylurea, long-acting insulin, renal failure, recurrent event, impaired awareness, or intentional exposure.
- [] Before discharge: medication change, glucagon and supplies, supervision, driving / occupational advice, follow-up.

ANNEX D. Adult DKA first-six-hour card

Time	Required actions
0-10 min	ABCDE, monitor, weight, glucose, ketones, venous gas, electrolytes, renal function, ECG, precipitant screen, IV access, isotonic fluid.
Within 1 hour	Review potassium; if at least 3.5, start IV insulin 0.1 units/kg/hour. Continue or plan basal insulin. Escalate severe DKA / high-risk patient.
Hourly	Glucose, observations, neurological status, urine output, infusion checks, cumulative fluid balance.
Approximately 2-4 hourly	Electrolytes, renal function, beta-hydroxybutyrate, venous pH / bicarbonate; adjust potassium and fluids.
Glucose below 13.9 mmol/L	Add 5% or 10% dextrose and reduce insulin to approximately 0.05 units/kg/hour; continue until DKA resolves.
At resolution	Confirm ketone and acid-base criteria, give / confirm subcutaneous basal insulin with overlap, establish meal and follow-up plan.

ANNEX E. Potassium and insulin safety card

Potassium	Insulin	Potassium replacement
Below 3.5	HOLD	Begin monitored replacement per local protocol; recheck frequently; start insulin only after threshold reached.
3.5-5.0	START / CONTINUE	Usually 20-30 mmol/L in IV fluid, adjusted for renal function and urine output.
Above 5.0	START / CONTINUE if otherwise safe	None initially; recheck within about 2 hours and add when concentration falls.
Anuric / dialysis	Specialist plan	No routine potassium-containing fluid; nephrology / critical-care guidance.

ANNEX F. HHS osmolality safety card

- [] Calculate effective or total osmolality with every relevant laboratory panel using the same formula.
- [] Target gradual fall, commonly 3-8 mOsm/kg/hour under the local approved pathway.
- [] Avoid sodium fall above 10 mmol/L in 24 hours and excessive glucose fall.
- [] An increasing sodium with falling osmolality may be expected; assess the osmolality trend and fluid balance before changing fluid tonicity.
- [] Use 0.05 units/kg/hour insulin after initial fluids / potassium assessment when indicated; use 0.1 units/kg/hour for mixed DKA / HHS.

- [] Add dextrose at approximately 14-17 mmol/L glucose while continuing resolution treatment.
- [] Monitor cognition, urine output, thrombosis, pressure injury, aspiration, and fluid overload.

ANNEX G. Euglycaemic DKA and SGLT2 card

- [] Do not exclude DKA because glucose is below 11.1 mmol/L.
- [] Check beta-hydroxybutyrate and venous pH / bicarbonate in symptomatic high-risk patients.
- [] Stop the SGLT2 inhibitor during suspected or confirmed DKA.
- [] Give insulin to suppress ketogenesis and start dextrose early enough to avoid hypoglycaemia.
- [] Investigate infection, fasting, reduced carbohydrate intake, vomiting, surgery, pregnancy, and insulin omission.
- [] Document sick-day and peri-procedural medicine advice; restart only after specialist review.

ANNEX H. Paediatric safety card

- [] Use the approved paediatric DKA / HHS pathway and involve paediatrics early.
- [] No insulin bolus. Start weight-based IV insulin after initial fluid therapy.
- [] Controlled fluid replacement; frequent weight-based reassessment and neurological observations.
- [] Add dextrose while insulin continues until ketones and acidosis resolve.
- [] Suspected cerebral injury: immediate hypertonic saline or mannitol per local protocol; do not wait for CT.
- [] Paediatric HHS: PICU / endocrine care, slower osmolality correction, lower or delayed insulin.

ANNEX I. Pregnancy DKA card

- [] Check ketones and venous gas early; DKA may occur with only modest hyperglycaemia.
- [] Maternal ABCDE, fluids, insulin, and potassium take priority.
- [] Activate obstetrics, endocrinology, anaesthesia / critical care, and neonatal support as appropriate.
- [] Begin fetal monitoring after maternal stabilization when viable and feasible.
- [] Add dextrose early for euglycaemic DKA so insulin can continue.
- [] Search for hyperemesis, infection, steroids, beta-agonists, SGLT2 exposure, and insulin-delivery failure.

ANNEX J. Pump / CGM device card

- [] Confirm sensor values with bedside or laboratory glucose when symptoms or perfusion are discordant.
- [] DKA: stop / remove pump per local policy, inspect system, and use standard IV insulin.
- [] Severe hypoglycaemia: suspend automated delivery if patient cannot self-manage; treat and reassess.
- [] Record device make, settings, last set change, site condition, remaining insulin, alarms, and downloaded data if available.
- [] Restart only after clinical recovery, competent self-management, verified settings, fresh supplies / site, and insulin-overlap plan.

ANNEX K. Precipitant checklist

- [] Insulin omitted, unaffordable, unavailable, expired, improperly stored, or delivered through a failed pump / set.
- [] New diabetes or progressive insulin deficiency.
- [] Infection / sepsis, including respiratory, urinary, skin / foot, dental, abdominal, and device-related sources.
- [] Myocardial infarction, stroke, thrombosis, pancreatitis, surgery, trauma, or pregnancy complication.
- [] SGLT2 inhibitor, glucocorticoid, antipsychotic, immunotherapy, stimulant, alcohol, or other medication / substance.
- [] Vomiting, starvation, eating disorder, reduced carbohydrate intake, enteral / parenteral nutrition problem.
- [] Cognitive impairment, depression, self-harm, substance use, domestic risk, food or housing insecurity, poor follow-up access.

ANNEX L. Monitoring chart

Time	Glucose	Ketones	pH / HCO ₃	K	Na / Osm	Fluid balance	Neuro	Insulin / dextrose / K rates
Arrival								
1 h								
2 h								
4 h								
6 h								
8 h								

Time	Glucose	Ketones	pH / HCO ₃	K	Na / Osm	Fluid balance	Neuro	Insulin / dextrose / K rates
12 h								

ANNEX M. Transition and discharge checklist

- ☐ DKA or HHS resolution criteria documented.
- ☐ Precipitating illness treated or plan established.
- ☐ Basal subcutaneous insulin given with required overlap before IV insulin stopped.
- ☐ Patient eating or has a safe nutrition plan; glucose and potassium stable.
- ☐ Medication reconciliation completed, including SGLT2 and secretagogue decisions.
- ☐ Glucose meter / CGM, strips, ketone testing, insulin, needles / pump supplies, and glucagon available.
- ☐ Hypoglycaemia and sick-day education completed with teach-back.
- ☐ Written return precautions and follow-up clinician / date documented.
- ☐ Driving, work, safeguarding, mental-health, and social barriers addressed.

ANNEX N. Transfer and handover minimum dataset

- ☐ Diagnosis: hypoglycaemia / DKA / HHS / mixed / euglycaemic DKA / non-crisis hyperglycaemia; severity and precipitant.
- ☐ Age, weight, pregnancy, diabetes type, comorbidity, pump / CGM, SGLT2 exposure.
- ☐ Glucose, beta-hydroxybutyrate, pH, bicarbonate, potassium, sodium, creatinine, osmolality trends and next tests due.
- ☐ Fluids given, cumulative balance, urine output, signs of overload or shock.
- ☐ Current insulin, dextrose, potassium, and other infusion concentrations and rates.
- ☐ Neurological status, ECG changes, airway / oxygen needs, infection / thrombosis / cerebral-injury concerns.
- ☐ Receiving clinician, destination, monitoring capability, transport staff, infusion supply, and contingency plan.

ANNEX O. Audit tool

Audit item	Met / not met / N/A
Immediate glucose obtained in relevant emergency presentation	
Hypoglycaemia treated and rechecked within 15 minutes	
DKA / HHS criteria and severity recorded	
Potassium reviewed before insulin	
Fluid, insulin, dextrose, and potassium orders had reassessment times	
Required hourly glucose and serial laboratory monitoring completed	
HHS osmolality rate monitored and within local target	
Precipitant investigated and treated	
IV-to-subcutaneous insulin overlap documented	
Education, supplies, and follow-up completed	
Adverse event / recurrent presentation / ICU transfer / death reviewed	

ANNEX P. Local configuration checklist

- ☐ Approved adult DKA, HHS, mixed-crisis, euglycaemic DKA, and severe-hyperglycaemia order sets.
- ☐ Approved adult hypoglycaemia algorithm with available oral glucose, IV glucose concentrations, glucagon products, and observation periods.
- ☐ Paediatric DKA / HHS and cerebral-injury rescue pathways, with PICU and transfer contacts.
- ☐ Pregnancy DKA pathway, obstetric / fetal-monitoring access, and critical-care escalation.
- ☐ Insulin infusion concentration, pump library, independent double-check, line-labeling, and transition standards.
- ☐ Potassium, magnesium, phosphate, bicarbonate, and hyperkalaemia protocols; renal and dialysis modifications.
- ☐ Blood beta-hydroxybutyrate availability, critical laboratory turnaround, osmolality calculation, and result-escalation process.
- ☐ Insulin pump / CGM / automated-delivery policy, device storage, documentation, and specialist contacts.
- ☐ SGLT2 sick-day / peri-procedural policy, diabetes education materials, discharge supplies, social-work and medication-access support.

[] Endocrinology, paediatrics, obstetrics, critical care, nephrology, toxicology, EMS, and interfacility transfer directory.

ANNEX Q. References and source tools

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3. American Diabetes Association Professional Practice Committee for Diabetes. Diabetes Care in the Hospital: Standards of Care in Diabetes-2026. *Diabetes Care*. 2026;49(Suppl 1):S339-S355. doi:10.2337/dc26-S016.
4. Glaser N, Fritsch M, Priyambada L, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State. *Pediatric Diabetes*. 2022;23:835-856. Current ISPAD resource version accessed June 2026.
5. Abraham MB, Karges B, Dovc K, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Assessment and Management of Hypoglycemia in Children and Adolescents With Diabetes. *Pediatric Diabetes*. 2022;23:1322-1340.
6. Joint British Diabetes Societies for Inpatient Care. The Management of Diabetic Ketoacidosis in Adults. Revised March 2023.
7. Joint British Diabetes Societies for Inpatient Care. The Management of the Hyperosmolar Hyperglycaemic State in Adults With Diabetes. February 2022.
8. Joint British Diabetes Societies for Inpatient Care. The Hospital Management of Hypoglycaemia in Adults With Diabetes Mellitus. January 2023.
9. World Health Organization and International Committee of the Red Cross. Basic Emergency Care: Approach to the Acutely Ill and Injured. WHO; 2018.
10. Local source tools to attach before approval: adult DKA / HHS order sets; paediatric DKA / HHS pathway; pregnancy DKA pathway; hypoglycaemia rescue chart; insulin and electrolyte infusion monographs; cerebral-injury rescue protocol; SGLT2 sick-day guidance; pump / CGM policy; transition and discharge forms; transfer directory.