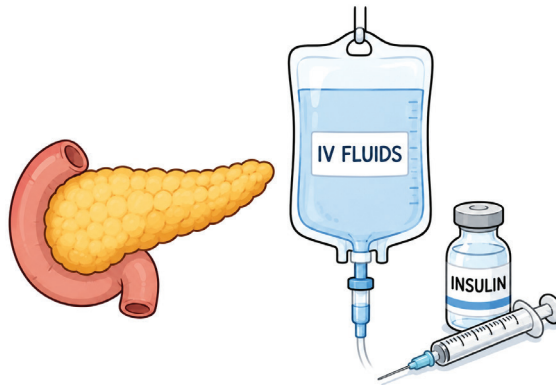




Diabetic Ketoacidosis: Diagnosis & Management

STG-005

Standard Treatment Guideline (STG)



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Standard Treatment Guidelines (STGs) for paediatric critical care are essential to ensure safe, evidence-based, ethical, and standardized management of critically ill children in Nepal. Given the wide variation in healthcare infrastructure, human resources, and availability of critical care services across the country, uniform clinical guidance is necessary to minimize unnecessary variations in practice and improve patient outcomes. These guidelines assist paediatricians, medical officers, residents, and other healthcare professionals in making timely, consistent, and appropriate clinical decisions, particularly in resource-limited settings. STGs also reinforce adherence to the Nepal Medical Council's Code of Ethics and Professional Conduct by promoting professional competence, patient safety, informed consent, appropriate documentation, and accountability. Furthermore, adherence to nationally accepted guidelines helps establish the expected standard of care, thereby reducing the risk of preventable errors, medico-legal disputes, and litigation while supporting defensible clinical practice. As practical bedside references, STGs facilitate rapid decision-making, enhance resident training, improve communication among multidisciplinary teams, and promote rational utilization of available resources. They also provide a framework for quality assurance, clinical audit, and continuous professional development. The adoption and implementation of National Paediatric Critical Care and Paediatric Endocrinology STGs will strengthen clinical governance, promote equitable delivery of critical care services, and contribute to improved survival and health outcomes for critically ill children throughout Nepal.

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Diabetic Ketoacidosis: Diagnosis & Management

STG-005

Introduction and Definition

Diabetic Ketoacidosis (DKA) is a medical emergency and the leading cause of morbidity and mortality in children with type 1 diabetes mellitus (DM). It is caused by lack of insulin production leading to decreased glucose utilization, release of counterregulatory hormones, and efflux of intracellular electrolytes into the blood stream. This results in ketosis and hyperglycemia and ultimately, dehydration, high anion gap metabolic acidosis, and electrolyte (mainly potassium) loss. DKA may be precipitated by infection, stress, or poor compliance with insulin therapy in children with type 1 DM.

Clinical Features and Diagnostic Approach

The diagnosis of DKA involves clinical and biochemical criteria:

- Clinical: History of polyuria, polydipsia, weight loss (mainly at initial presentation of DM); signs of dehydration, deep sighing respirations (Kussmaul's breathing), abdominal pain, vomiting, altered mental status and progressive obtundation leading to coma.
- Biochemical:
 - Hyperglycemia (blood glucose >200 mg/dl or >11 mmol/L)
 - Serum pH <7.3 and/or bicarbonate <18 mEq/L (High anion gap acidosis)
 - Moderate to large ketonuria and ketonemia (serum beta-hydroxybutyrate >3 mmol/L).

Severity of DKA is based on the degree of acidosis:

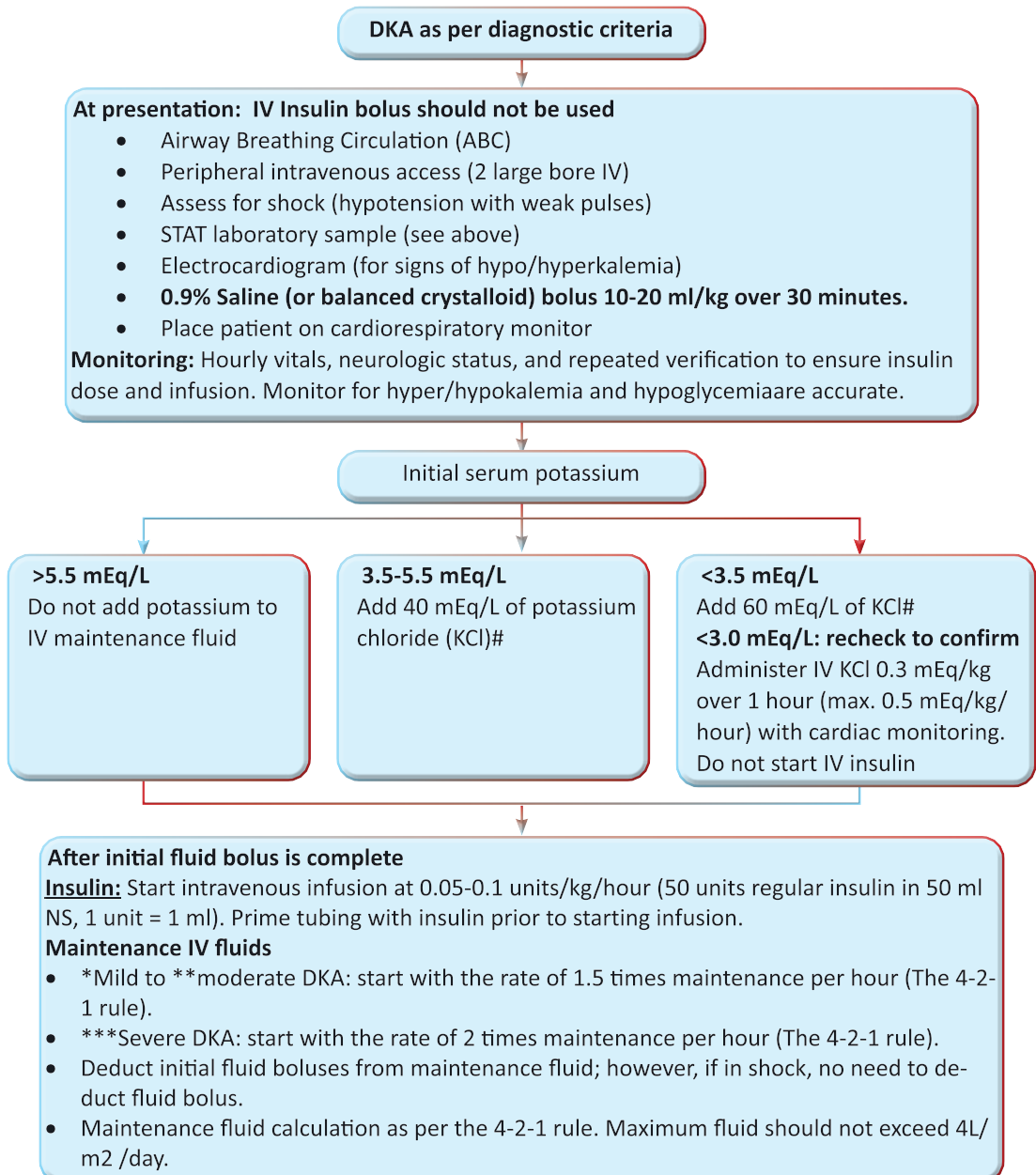
- *Mild: venous pH 7.2-7.29 or bicarbonate concentration <18 mEq/L, fluid deficit 5%
- **Moderate: pH 7.1-7.19 or bicarbonate <10 mEq/L, fluid deficit 7%
- ***Severe: pH <7.1 or bicarbonate <5 mEq/L, fluid deficit 10%

Investigations

- **STAT sample collection at presentation** for serum glucose (BG), venous blood gas (VBG for pH and bicarbonate), serum sodium, serum potassium, urine and/or serum ketones
- Other initial laboratory sample for HbA1C, renal function panel, magnesium, calcium, phosphorus; CBC; urine/ blood /wound cultures if suspicion for infection (e.g., fever)

- Routine laboratory sample collection:
 - Hourly: bedside serum glucose (BG)
 - 4 hourly: bedside point of care venous blood gas (pH, bicarbonate, chloride, anion gap)
 - 4-6 hourly: renal function test (sodium, potassium, urea, creatinine)
 - Urine Ketones daily

Management Flow Chart



Use either method for fluid deficit correction, maintain BG at 150-200 mg/dl to avoid sudden hypoglycemia. The type of fluid is 0.45% Saline or 0.9% Saline and add dextrose as per BG level as mentioned in the box below.

OR

Two Bag Method: Order 2 bags

Fluid 'A': Normal Saline with KCl #

Fluid 'B': 0.45% Saline with 10% Dextrose## and KCl #

Adjust 2 bags IVF rates according to BG

- BG >300: Fluid A at 100% of IVF
- BG 250 – 300: Fluid A at 75% + Fluid B at 25%
- BG 200 -250: Fluid A at 50% +Fluid B at 50%
- BG 150 -200: Fluid A at 25% + Fluid B at 75%
- BG <150: Fluid B at 100%

If BG <120, increase dextrose concentration to 12.5%.and/or decrease insulin by 20% or hold insulin until BG is above 100

One Bag Method

Change fluids according to hourly BG

- >300 mg/dl: Normal Saline with KCl #
- 250-300 mg/dl or decline of blood sugar >90 mg/dl/hour: DNS (5% Dextrose) with KCL#
- <150 mg/dl: 10% DNS## with KCL#
- If BG <120, increase dextrose concentration to 12.5%.and/or decrease insulin by 20% or hold insulin until BG is above 100

Resolution of DKA

Serum bicarbonate ≥ 18 mEq/L

Normal anion gap (10 ± 2)

- Administer home dose insulin glargine or long-acting insulin
- Once oral intake is tolerated: Order meal; administer SQ insulin; discontinue dextrose containing IV fluid, and after 30 minutes of SQ insulin discontinue insulin infusion
- Check a serum renal function or VBG after 4-6 hours to ensure patient is out of DKA
- Space bedside BG checks: before meals, before bedtime, at 2 am
- Administer NS or 0.45 NS with 20 mEq/L KCL at one maintenance for 12 hours OR until patient is drinking well.

##Method of preparation of 0.45% Saline with 10% Dextrose for one or two bag method

Take 500 ml of 0.45% Saline with 5% Dextrose

Remove 50 ml of above fluid following sterile technique

Then add 50% of 50 ml Dextrose to the above fluid

SPECIAL MANAGEMENT CONSIDERATIONS

GOALS OF MANAGEMENT

- Rehydration [by fluid administration]
- Correction of acidosis [by insulin and fluid infusions]
- Identify and treat precipitating event
- Identify and manage complications, if present:
 - Hypoglycemia (<80 mg/dL)
 - Electrolyte imbalance, mainly hypokalemia,
 - Cerebral edema

PERSISTENT HIGH ANION GAP ACIDOSIS

If high anion gap acidosis does not resolve

- Consider increasing IV fluid to 2x maintenance **and/or**
- Increasing insulin infusion to 0.1 units per kg per hour
- Infection/sepsis may be a rare cause

MANAGEMENT OF CEREBRAL EDEMA

- Neurologic checks every hour.
- Suspect clinical cerebral edema if sudden deterioration or worsening neurological examination:
 - Sudden severe headache
 - New onset vomiting
 - Change in neuro examination from previous examinations
 - Age-inappropriate incontinence
- Management:
 - 3% sodium chloride bolus 2.5 to 5 ml/kg, administer over 15 minutes x1
 - OR 25% (or 20%) Mannitol 0.5 g/kg IV administer over 30 minutes x1
- Consider CT head if necessary and feasible only after patient is stable.

AVOID SODIUM BICARBONATE

- Sodium bicarbonate is **not indicated** even though the serum pH may be below 7.
- It may be considered only if the patient is in shock (septic) requiring 2 or more vasopressors.
- It can cause paradoxical cerebral acidosis worsening cerebral edema; increase serum sodium load; and decrease serum potassium.

INVASIVE MECHANICAL VENTILATION SHOULD BE AVOIDED IF POSSIBLE

- Only indication for invasive mechanical ventilation is if the patient **does not** have a ventilatory drive.
- In DKA, patients may have obtundation and low GCS. However, they can continue to maintain ventilatory drive and manage their own pH by lowering PCO₂: invasive mechanical ventilation can take this away leading to increased PCO₂ resulting in life threatening further decrease in serum pH. This may contribute to worsening cerebral edema.

SHOCK

- **Hypotension in addition to weak pulses**
- Consider additional fluid bolus (NS or LR); vasopressors as indicated.
- Consider work-up for infection and start antibiotics.
- May require positive pressure ventilation support (non-invasive or invasive) and supplemental oxygen

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