

## An update on emergency management of diabetic ketoacidosis (DKA) among children and adolescent

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### Abstract

The narrative review was planned to evaluate general guidelines for the management of diabetic ketoacidosis in children presenting to the Emergency Department that must be identified timely and correctly, and its management may be adjusted based on individual patient needs and institutional protocols. It is always advisable to consult a senior doctor on call or the endocrinology team, if available, as soon as one suspects diabetic ketoacidosis, ideally at a health centre with experienced professionals in managing the condition.

**Keywords:** Diabetic, Diabetic ketoacidosis, Paediatric emergency medicine, Management.

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### Introduction

Children with diabetic ketoacidosis (DKA) often present early with symptoms such as abdominal pain, nausea, vomiting, shortness of breath, polydipsia, polyuria, weight loss, dehydration, drowsiness or confusion, and acidic breath. In contrast, hyperosmolar hyperglycaemic state (HHS) typically presents later and may be due to hyperosmolality, along with high blood glucose on a finger-prick test, and elevated levels of glucose and ketones in the urine. DKA is a potentially life-threatening complication of type I diabetes mellitus (T1DM), with increasing frequency.<sup>1,2</sup> Approximately 30% of patients with new-onset diabetes may be present with DKA, and an additional 6-8% may develop it annually.<sup>3</sup> DKA can be prevented through meticulous education, insulin compliance and early identification. Treatment involves careful administration of fluids and insulin to prevent recurrence. Previously, fluid management was restricted due to the fear of cerebral oedema, but recent trials, such as the Fluid Therapies Under Investigation in Diabetic

Ketoacidosis (FLUID trial by Paediatric Emergency Care Applied Research Network (PECARN), which found no significant difference in the development of cerebral oedema between slower and more rapid fluid administration (0.9% vs 0.45% saline).<sup>4</sup>

### Diagnostic Criteria

#### Biochemical:<sup>5</sup>

- \* Hyperglycaemia, blood glucose >11mmol/L (>200mg/dl).
- \* Venous potential of hydrogen (pH) <7.3 or sodium bicarbonate (NaHCO<sub>3</sub>) <18mmol/L (110mg/dl).
- \* Ketonaemia (serum beta [β] hydroxybutyrate [BOHB] >3mmol/L (≥32mg/dl), or moderate to large urine ketonuria (≥2+).]

**Clinical:** Polyuria and polydipsia are the most common presentations of DKA, though they may not be present in all cases. Signs of dehydration, tachypnea with acidotic breathing, tachycardia, vomiting, abdominal pain with or without diarrhoea, and increasing drowsiness may also serve as initial symptoms.

#### DKA Severity of DKA:<sup>6,7</sup>

The severity of DKA is categorized by the degree of acidosis.

**Mild:** Venous pH <7.3 or NaHCO<sub>3</sub><18mmol/L (110mg/dl).

**Moderate:** Venous pH <7.2 or NaHCO<sub>3</sub><10mmol/L (61mg/dl).

**Severe:** Venous pH <7.1 or NaHCO<sub>3</sub><05mmol/L (31mg/dl).

**Epidemiology:** DKA occurs at the rate of 6-8 % per year.<sup>1,3</sup> Almost 30% of new-onset diabetes may present with DKA. Though most cases of DKA are because of poor compliance, missed insulin and incorrect dose along with intercurrent illness, like fever and growth spurt.<sup>5</sup>

**Precipitating Factors:** DKA is one of the most common complications of T1DM in children, with several factors increasing the risk, such as low socioeconomic status (SES), delayed initial diagnosis, younger age, limited access to medical care, and living in regions with low T1DM

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prevalence.<sup>4</sup> Other contributing factors include pre-pubertal girls, acute illnesses, like vomiting and diarrhoea, and psychiatric disorders. Poor compliance and mismanagement remain the primary causes of DKA episodes globally, particularly among teenagers and adolescents.<sup>4</sup> Diabetes itself stresses the body's hormonal balance, and, when combined with acute illnesses, like infection, vomiting and diarrhoea, it can lead to increased ketolysis. Certain drugs and alcohol consumption can also worsen the condition.

**Pathophysiology:** Several hormones in the human body regulate glucose homeostasis, most of which are catabolic, such as glucagon, catecholamines and androgens. In contrast, insulin is the primary anabolic hormone. When blood glucose levels rise (postprandial), glucose stimulates the pancreatic beta cells, triggering a series of events that lead to insulin release (Figure-1).<sup>6</sup>

This released insulin has multiple effects on glucose metabolism. It reduces hepatic glucose production by minimising glycogenolysis and gluconeogenesis while enhancing glucose uptake in muscle and adipose tissues where it is stored as glycogen. Insulin also inhibits glucagon secretion. An imbalance between insulin and glucagon can lead to the development of DKA or HHS. Additionally, counter-regulatory hormones, like cortisol, catecholamines and growth hormone, which oppose insulin, contribute to increased glucose and ketoacid production, especially during stressors, such as sepsis, trauma or febrile illness.<sup>6,7</sup>

With increasing insulin deficiency or resistance, cellular glucose becomes depleted, leading to enhanced lipolysis of fat stores. This increases the oxidation of fatty acids and glycerol, which are then delivered in larger amounts to hepatic cells. In the liver, fatty acids are converted to acyl

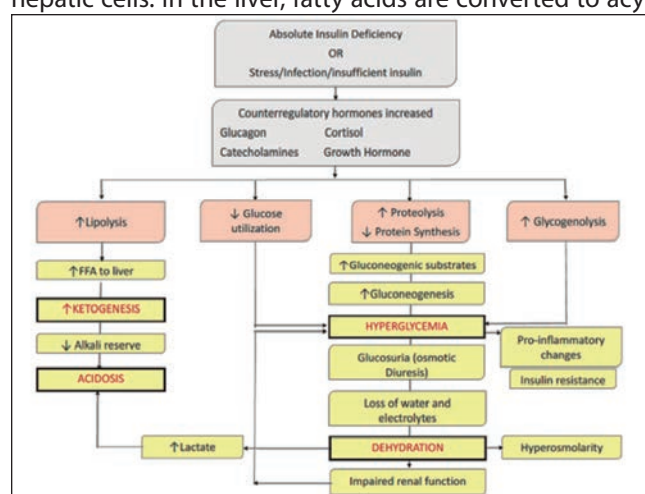
coenzyme A (acyl-CoA) in mitochondria, where they are used to produce adenosine triphosphate (ATP). This process results in the production of large amounts of acetoacetic acid, beta-hydroxybutyric acid and acetone. (Figure-1).

**Clinical Manifestation:** The early symptoms of diabetes, such as polydipsia, polyphagia and weight-loss, are often overlooked until the child presents with DKA, characterised by acidotic breathing, constant abdominal pain, vomiting and dehydration, which can mimic acute abdomen in many cases. These symptoms are more frequent in adolescent girls and harder to identify in younger children, especially in those with poor insulin compliance or concurrent illnesses.<sup>8</sup> Assessing the degree of dehydration can be challenging, though most patients exhibit tachypnoea with deep, laboured Kussmaul respiration, tachycardia and normal blood pressure despite significant dehydration. Central nervous system (CNS) manifestations, such as irritability, drowsiness and confusion, vary based on the severity of both DKA and dehydration.<sup>9</sup>

**Initial Laboratory Parameters:** Once DKA is clinically suspected and confirmed through blood glucose, venous blood gas and urinary ketones, several initial laboratory parameters should be documented to guide management. These include serum electrolytes, with calcium, magnesium and phosphorus, blood urea nitrogen (BUN), creatinine, albumin, corrected sodium (adjusted by 1.6mmol/L per 100mg/dl rise in glucose), and serum osmolality. Additional essential baseline investigations include a complete blood count (CBC), blood and/or urine culture, chest X-ray (CXR), glycated haemoglobin (HbA1c), and C-reactive protein (CRP), as well as blood and urine cultures.<sup>10</sup>

A few non-specific but relevant tests, such as thyroid peroxidase antibodies, thyroid-stimulating hormone (TSH), thyroglobulin antibodies, C-peptide, immunoglobulin A (IgA) levels, and tissue transglutaminase IgA, may aid in future management planning.

**Clinical Assessment and Initial Observation:** Physicians should assess and document clinical status and initial laboratory parameters for future comparison. They should create a diabetic chart to include weight, blood glucose levels, laboratory results, insulin administration, fluids, Glasgow Coma Scale (GCS), vitals and urine output. They should also monitor dehydration status and level of consciousness, conducting hourly neurological assessments, especially if the patient is drowsy at admission. GCS should be recorded and physicians should be vigilant for signs of cerebral oedema, infection or ileus. If the child's recent weight is unavailable, Broselow's chart should be used for guidance.



**Figure-1:** Pathophysiology of diabetic ketoacidosis (DKA). Copyright © American Diabetes Association. Adapted from Diabetes Care, Vol.29, 2006:1150-1159.<sup>36</sup>

## DKA Management

**Emergency Assessment and Stabilisation:** The airway has to be patent, and if the child is comatose, an oral airway should be inserted, and a nasogastric (NG) tube must be placed to aspirate and left on open drainage.

The patient status must remain nothing-per-oral (NPO).

Physicians should administer 100% oxygen via facemask and initiate continuous cardiac and pulse oximetry monitoring.

A Foley catheter must be inserted.

Two large-bore intravenous (IV) cannulas must be inserted for access, and an electrocardiogram (ECG) must be conducted, while blood samples must also be taken.

Using a central venous catheter must be avoided.

**Fluids:** The choice of fluids for managing DKA may include 0.9% saline, Ringer's lactate, Hartmann's solution, or Plasmalyte. Shock is rare in DKA; most children present with about 7% deficit of body fluids. Dehydration can be classified as mild (5%), moderate (7%), or severe (10%). Elevated anion gap, low pH, and high urea nitrogen levels are associated with severe dehydration.

For children in shock, a bolus of 0.9% saline at a rate of 10-20ml/kg should be administered as early as possible, with crystalloid solutions preferred over colloids.<sup>5</sup> However, those not in shock, may start with same fluids at the rate of 10-20ml/kg over 20-30 minutes or even slower.<sup>5</sup> Additionally, children with GCS of 14-15 who exhibit persistent tachycardia, or any signs of poor perfusion, may require an additional bolus of normal saline.<sup>3</sup>

All fluids administered should be documented meticulously, especially those given in other hospitals, in the emergency room (ER), and enroute to the ward. Fluid calculations should be based on age or body surface area rather than solely on age. Deficient and desired fluids are typically administered for at least 36-48 hours and should continue until the acidosis has resolved, and the child can tolerate oral intake. Additionally, the transition from IV to subcutaneous (SC) insulin should occur during this 48-hour period.

**Fluid Volume Calculation:** For moderate DKA, 1.5 times the total maintenance fluids for 48 hours should be calculated based on the child's body weight, and this amount should be administered over the first 48 hours. In the case of severe DKA, 2 times the total maintenance fluids should be calculated. For example, a 10kg child in moderate DKA would require a total fluid volume of 3,000ml for 48 hours, which translates to an infusion rate

of approximately 62ml/hour. (Table 1)

Another method of fluid calculation is more subjective and clinically challenging because, in DKA, there is typically more intravascular fluid compared to intra- and intercellular or interstitial fluids due to the high intravascular solute load. For most patients, with an average degree of dehydration estimated at approximately 7-9% of body weight, it is important to never exceed a dehydration estimate of 10%.<sup>5</sup>

**Potassium:** Severe hypokalaemia (<2.5mmol/L) is an independent marker of poor treatment outcomes and increased mortality. If a child presents with hypokalaemia, potassium replacement should begin during the initial volume expansion and prior to initiating insulin therapy. Once the child has been adequately resuscitated, potassium should be included in the rehydration fluid unless anuria is suspected. Continuous cardiac monitoring is essential, with frequent observations for any T-wave changes.

The initial potassium concentration may be set at 40mmol/L, consisting of 20mmol/L from potassium chloride (KCl) and 20mmol/L from either potassium phosphate (KPhos) or potassium acetate (KAcetate). In cases of severe hypokalaemia, the maximum IV infusion rate of potassium (diluted) should not exceed 0.5mmol/kg/hr. (Table 2).

If hypokalaemia persists despite maximum potassium replacement, the rate of insulin infusion should be reduced, and an endocrinology consult should be obtained immediately.

If an urgent serum potassium level is unavailable, an ECG can help determine whether the child has hyperkalaemia

**Table-1:** Calculation of fluid requirement as per the Holliday-Segar formula.<sup>9,10</sup>

### If the weight is Known

\*100 ml/kg for 1st 10 kg (04 ml/kg/hr)

\*50 ml/kg for 2nd 10 kg (02 ml/kg/hr), (1000 + 50 ml/kg/24 hours for each kgs)

\*20 ml/kg for the subsequent kgs (01 ml/kg/hr) for each Kg >20, (1500 + 20 ml/kg/24 hours for each kgs > 20)

### If the weight is Not Known

Age 0 - 2 years 80 ml/kg/24 hrs

3 - 5 years 70 ml/kg/24 hrs

6 - 9 years 60 ml/kg/24 hrs

10-14 years 50 ml/kg/24 hrs

>15 years 30 ml/kg/24 hrs

Hourly rate =  $\frac{24 \text{ hours maintenance} + \text{deficit} - \text{resuscitation fluid (already given)}}{24}$

e.g.: 10 kg children for 24 hours fluid calculation

5% dehydration:  $1000 + 500 = 1500 \text{ ml} / 24 = 63 \text{ ml/hour}$ . (Moderate DKA)

10% dehydration:  $1000 + 1000 = 2000 \text{ ml} / 24 = 83 \text{ ml/hour}$ . (Severe DKA)

**Do not include continuing urinary losses in the calculations**

DKA: Diabetic ketoacidosis.

**Table-2:** Calculation of electrolytes and requirement.<sup>5</sup>

| Electrolytes                 | Average losses/kg                           | 24 hours maintenance |
|------------------------------|---------------------------------------------|----------------------|
| Sodium                       | 6mmol (range 5–13)                          | 2–4mmol              |
| Potassium                    | 5mmol (range 3–6)                           | 2–3mmol              |
| Chloride                     | 4mmol (range 3–9)                           | 2–3mmol              |
| Phosphate                    | 0.5–2.5mmol                                 | 1–2mmol              |
| Serum Osmolality             | (2 [Na + K] + glucose) (285-295)            |                      |
| Corrected Na                 | Measured Na + 1.6 x (Glucose (mg/dl) – 100) |                      |
|                              | 100                                         |                      |
| Anion Gap                    | Na – (Cl + HCO <sub>3</sub> )               |                      |
| Normal range is 12 ± 2mmol/L |                                             |                      |

Use of hypotonic versus isotonic as replacement fluid therapy depends on physician judgment, hydration status, S. Na concentration and the osmolality. The PECARN (Paediatric Emergency Care Applied Research Network) study suggests a lower frequency of altered mental status in children rehydrated more rapidly with 0.45% saline, but these differences did not reach statistical significance. (4) Use of 0.45% normal saline is limited to infant or children less than five years of age or with initial S. Na of 145 or above, continuation of normal saline (NS) instead of 0.45% as rehydration or maintenance as the changes made in 2015.<sup>(4)</sup>

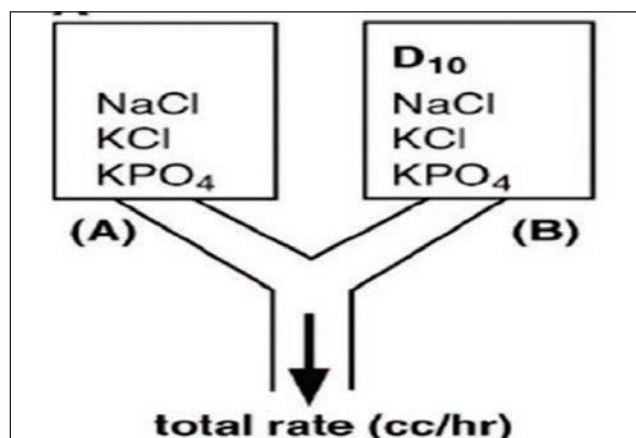
Generally, once the blood glucose level has fallen to  $\leq 200$  or 225 mg/dl, a two-bag system is implemented. This system allows for more independent manipulation of glucose levels and total fluid volume.

The two-bag system consists of 0.45% (or 0.9%) NS in one bag and 0.45% (or 0.9%) NS with 10% dextrose water (DW) added along with KCl (20mEq/L) and KPh (20mEq/L) in the second bag, each containing 1 litre. This setup ensures that the electrolyte content is identical in both bags, with one bag containing 10% DW, while the other bag being devoid of glucose.

or hypokalaemia. Findings such as prolonged PR interval, T wave flattening or inversion, ST depression, prominent U waves, and an apparent long QT interval may indicate hypokalaemia. Tall, peaked, symmetrical T waves and a short QT interval are signs of hyperkalaemia.

**Sodium:** Treatment protocols for DKA in children recommend frequent monitoring of serum sodium levels, typically every 2–4 hours, alongside glucose levels, to allow for timely adjustments in fluid therapy based on these changes.<sup>5,11</sup> Rapid fluctuations in sodium and glucose can adversely affect patient outcomes.<sup>5</sup> Disturbances in sodium metabolism are common in DKA, making their careful management essential for achieving optimal results (Table 2).<sup>12</sup>

DKA typically presents with low or low-normal plasma sodium concentrations.<sup>12</sup> However, hyponatraemia in DKA is often not true hyponatraemia, but rather pseudo-hyponatraemia caused by hyperglycaemia. This phenomenon occurs because hyperglycaemia increases serum osmolality, prompting intracellular fluid to shift to the extracellular compartment, which leads to dilutional hyponatraemia. The production of ketone bodies — resulting from a metabolic shift from glucose to fatty acids due to insulin deficiency — further elevates osmolality, exacerbating fluid shifts and the dilutional effect on serum sodium concentrations. This increased osmolality also stimulates the thirst centre, triggering the release of antidiuretic hormone (ADH), which promotes free fluid reabsorption in the renal tubules.<sup>13</sup> Additionally, the use of

**Figure-2:** The two-bag method.

hypotonic fluids during treatment can further dilute serum sodium levels.<sup>14</sup> Consequently, whenever hyponatraemia is encountered in a child with DKA, it is essential to calculate the corrected serum sodium for the degree of hyperglycaemia. A simple formula to calculate the corrected sodium level is:

Corrected  $[Na^+] = \text{Measured } [Na^+] + 0.016 \times (\text{Serum } [Glucose] - 100)$  (Figure-2).<sup>5</sup>

In many cases, no treatment is necessary for hyponatraemia in children with DKA. It is anticipated that serum sodium levels will rise with the initiation of treatment, with an estimated increase of approximately 1.6 mmol/L for every 100 mg/dl decline in serum glucose level.<sup>11,14</sup> While some retrospective studies indicated a higher incidence of cerebral oedema in patients whose serum sodium did not increase in response to treatment, this association was rejected by recent large-scale prospective studies.<sup>15–17</sup>

Hypernatraemia is rarely encountered in children with DKA, typically resulting from water deficit due to decreased oral intake or excessive losses that exceed sodium loss, such as from vomiting and diarrhoea.<sup>18</sup> Osmotic diuresis caused by glucosuria can lead to volume depletion and contribute to hypernatraemia.<sup>19</sup> Additionally, excessive consumption of carbonated beverages and herbal medications may also cause hypernatraemia in these patients.<sup>11</sup> Although there are no clear guidelines for treating hypernatraemia in DKA, literature suggests that the preferred fluid choice should be half-strength normal saline (0.45% saline) rather than 0.9% normal saline.<sup>18</sup>

**Phosphate:** In DKA, there is often a depletion of total serum phosphate, although plasma phosphate levels may appear normal initially. This is due to the hyperosmolar state during DKA, which causes an extracellular shift of intracellular phosphate.<sup>20,21</sup> Osmotic diuresis from

glucosuria and decreased reabsorption by the renal tubules contribute to the net loss of phosphate. Hypophosphataemia can worsen after initiating fluid and insulin therapy, as insulin promotes the cellular uptake of phosphate, exacerbating the deficiency.<sup>20</sup> Occasionally, hypophosphataemia may also occur in children with DKA when transcellular shifts compensate for or even exceed the net urinary loss of phosphate.<sup>21</sup> Reduced phosphate levels can lead to decreased ATP production, along with impaired levels and activity of 2,3-diphosphoglycerate (2,3-DPG), resulting in increased affinity of oxygen for haemoglobin (Hb) and reduced oxygen release to tissues.<sup>22</sup>

Hypophosphataemia generally does not cause clinically significant symptoms and is often acute and self-resolving. Consequently, the necessity for phosphate replacement during DKA treatment remains a topic of debate, with prospective studies indicating a lack of clinical benefit.<sup>22</sup> Current guidelines do not recommend routine phosphate replacement.<sup>22,23</sup> Phosphate administration can lead to hypocalcaemia; thus, it is advised only in cases of severe hypophosphataemia associated with metabolic encephalopathy, reduced myocardial contractility, myopathy, dysphagia, or ileus.<sup>23,24</sup> However, with careful monitoring to avoid hypocalcaemia, potassium phosphate (K Phos) can be used alongside KCl in equal amounts to provide 40mmol/L of potassium.<sup>25</sup> The lack of resolution of acidosis is likely due to the development of hyperchloraemic acidosis, a benign condition that should not delay the transition to SC insulin.

Severe hypophosphatemia (<1mg/dl), whether symptomatic or not, should be managed promptly. In such cases, the insulin infusion may be reduced or temporarily stopped. KCl and KPhos can be administered in combination; however, monitoring of calcium and magnesium levels is essential.

**Bicarbonate:** In paediatric and adolescent DKA cases, bicarbonate therapy is generally not recommended. IV NaHCO<sub>3</sub> should only be administered in scenarios involving life-threatening hyperkalaemia or severe acidosis (pH <6.9 with accompanying symptoms) that compromises myocardial contractility. This intervention should only be undertaken after consulting with an intensivist and is typically reserved for critical situations, such as peri-arrest or cardiac arrest.<sup>5,12,26</sup>

Research, including systematic reviews and randomised controlled trials (RCTs), has demonstrated that administering HCO<sub>3</sub> does not significantly improve hospitalisation duration, mortality rates, resolution of ketosis or acidosis, electrolyte imbalances, tissue oxygenation, or cerebrospinal fluid (CSF) acidosis.<sup>27</sup>

Conversely, the literature suggests that HCO<sub>3</sub> administration in children and adolescents with DKA can have multiple adverse effects. Rapid correction of acidosis with HCO<sub>3</sub> infusion can lead to hypokalemia.<sup>28</sup> Additionally, it may cause a paradoxical worsening of CNS acidosis, as the increased partial pressure of carbon dioxide (CO<sub>2</sub>) can cross the blood-brain barrier more readily than arterial bicarbonate, resulting in a decrease in cerebral pH and subsequent clinical and neurological deterioration. Administering HCO<sub>3</sub> to rapidly correct acidosis has also been associated with worsened tissue oxygenation. While acidosis promotes the Bohr effect, reducing Hb oxygen affinity, it also decreases 2,3-DPG levels in red blood cells, which paradoxically increases haemoglobin oxygen affinity. In the early stages of DKA, this balance initially favoured the Bohr effect. However, the introduction of HCO<sub>3</sub> and the sudden correction of acidemia may disrupt this equilibrium, potentially lowering 2,3-DPG levels further.<sup>27</sup>

**Antibiotics:** Infections, such as urinary tract infections or respiratory infections, combined with the discontinuation of insulin, are the most common triggers of DKA.<sup>29</sup> Diagnosing infections in patients with DKA can be challenging, as it primarily relies on clinical examination and culture results. Early signs of infection may be subtle, and culture results typically take 24–48 hours to obtain.<sup>30</sup> Patients with DKA may present with symptoms like fever, elevated procalcitonin and CRP levels, increased white blood cell (WBC) counts, and higher neutrophil percentages, even in the absence of a confirmed infection, complicating early detection further.<sup>31</sup>

In DKA, bacterial infections are linked to higher mortality rates and prolonged hospital stays.<sup>32</sup> Therefore, timely identification and appropriate antibiotic therapy are essential for improving patient outcomes. When selecting antibiotics for a patient with DKA, it is important to recognise that these children are already at risk for acute kidney injury (AKI) due to hypovolaemia. The use of nephrotoxic antibiotics can worsen renal injury, complicate management and potentially jeopardise outcomes.<sup>33</sup> Thus, careful selection of antibiotics is critical to avoid increasing the risk of AKI while ensuring effective treatment. Empiric antibiotics are typically initiated based on clinical suspicion and later adjusted to specific antibiotics once culture results are available.<sup>5,30</sup>

**Insulin:** DKA is caused by a reduced circulating level or insensitivity to insulin, along with increased levels of counter-regulatory hormones. Rehydration alone may reduce blood glucose concentration, but insulin therapy is essential to restore normal cellular metabolism and combat the excessive catabolism caused by lipolysis and

ketogenesis.<sup>33</sup> It is contraindicated to administer an initial insulin bolus or SC insulin at the beginning of treatment for moderate to severe DKA, but mild DKA may be managed with regular short-acting SC insulin, with the consensus of the endocrinology team.<sup>33</sup>

Regular insulin is recommended and should be administered via continuous IV infusion at a rate of 0.05 to 0.1 U/kg/hour. A lower dose of 0.05 U/kg/hour may be used in children with pH >7.15. The infusion should not be discontinued until the acidosis has resolved.<sup>33</sup>

**Insulin Preparation:** The common method of insulin preparation was recommended by Glaser N. et al. in 2022<sup>5</sup> and involves diluting 50 units of regular insulin in 50 ml of 0.9% NS (1 unit = 1 ml).

Insulin infusion should be prepared and administered separately, without mixing it with fluid bags. The tubing should be flushed with insulin solution before administration. If no IV line is available, insulin can be given intramuscularly (IM), but central venous lines (CVL) should not be used for insulin infusion.<sup>34</sup>

The effectiveness of insulin should be monitored by checking venous pH and serum BOHB concentrations every 2 hours. If the insulin effect is adequate, as indicated by a reduction in blood glucose and ketosis, the insulin dose should be lowered. If there is a rapid drop in blood glucose, the insulin dose may also be reduced, provided metabolic acidosis continues to resolve. On the other hand, if pH does not improve despite a rapid fall in blood glucose, the insulin infusion should not be reduced or stopped; instead, the dextrose concentration in the fluid bag should be increased. The optimal blood glucose fall is 2-5 mmol/hour (36-90 mg/hour). To prevent an excessively rapid drop in blood glucose and hypoglycaemia after insulin infusion, a second bag of 10% dextrose fluids should be added (the two-bag method) within the total calculated fluids when blood glucose falls to 14-17 mmol/L (250-300 mg/dl), or when the blood glucose decreases by more than 5 mmol/L/hour (Table 3).

**Table-3:** Differential infusion rates of two-bag modulate glucose delivery between any concentrations ranging 0-10%.<sup>34</sup>

|                            |                                                                                                                                             |                               |
|----------------------------|---------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------|
| <b>Glucose &gt; 250:</b>   | Run "NS (only)" as calculated                                                                                                               |                               |
| <b>Glucose &lt; 225:</b>   | Run "NS" at 75 % rate of maintenance and "D10 with NS" 25% of maintenance, and need to titrate accordingly as per the fall of blood glucose |                               |
| <b>If Glucose &lt; 200</b> | Run "D10 with NS" as 100% calculated fluids.                                                                                                |                               |
| <b>e.g., glucose level</b> | <b>Bag A (NS Only)</b>                                                                                                                      | <b>Bag B (NS with 10 %DW)</b> |
| > 225 mg/dl                | 200 ml                                                                                                                                      | 00 ml                         |
| < 225 mg/dl                | 100 ml                                                                                                                                      | 100 ml                        |
| < 200 mg/dl                | 00 ml                                                                                                                                       | 200 ml                        |

With any fall or rise of blood glucose >50mg/dl, increase or decrease the fluids by 10-20% simultaneously to maintain blood glucose around 200. NS: Normal saline, DW: Dextrose water.

The two-bag method is useful for titrating blood glucose by adjusting the infusion rates of both types of fluid: one bag containing NS without dextrose and the other containing NS with dextrose. Both bags should also contain 20 mmol/L of KCl and 20 mmol/L of KPhos to maintain blood glucose levels between 8.3 and 13.9 mmol/L (150-250 mg/dl) (Figure-2).<sup>34,35</sup> If blood glucose continues to fall despite ongoing insulin to correct acidosis, 10% or 12.5% dextrose water (DW) may be used to prevent hypoglycaemia. If blood glucose drops to <4 mmol/L (70 mg/dl), a bolus of 2-4 mL/kg of 10% DW should be administered, and the dextrose concentration of the infusion should be increased by 10-15%, while blood glucose levels must be monitored every 30 minutes until they return to normal.

### Continuous Insulin Management

**Glucose Level <200:** If the blood glucose level is <200 mg/dl along with pH >7.3 and 10% dextrose-containing fluid is being administered, the insulin infusion rate may be reduced to 0.05 units/kg/hour. However, insulin should not be stopped at this point. If blood glucose becomes uncontrolled or the pH does not improve after 4-6 hours, senior medical staff should be consulted to re-evaluate possible sepsis, insulin administration errors, or other underlying conditions.<sup>33</sup>

**Glucose Level 81-100:** The rate of insulin infusion should be decreased to 0.05 unit/kg/hr and IV fluid (10% dextrose [D10] with NS) rate to 2 times maintenance. Blood glucose level should be rechecked in 30 minutes.

**Glucose Level <81:** Insulin drip must be decreased to 0.05 unit/kg/hr and a bolus of 10% DW be given at 2 ml/kg IV. IV fluid rate should be increased to 2 times maintenance. Blood glucose level should be rechecked in 15 minutes. If the glucose level is still <81 mg/dl, another bolus of 10% DW must be given at 2 ml/kg IV, and blood glucose should be rechecked in 15 minutes.

**Known Diabetic Child vs Newly Diagnosed Cases:** The patient's original insulin schedule may need to be resumed, and for newly diagnosed diabetics, insulin can be calculated as follows:

Total Daily Dose (TDD) = 0.75 U/kg/day in pre-pubertal children and 1 U/kg/day in pubertal patients.

Morning (pre-breakfast) 2/3 of TDD—1/3 regular and 2/3 (neutral protamine hagedorn (NPH)

Night (pre-dinner) 1/3 of TDD—1/3 regular and 2/3 NPH

Long-acting Lantus Insulin 0.4 units/kg SC as soon as glucose level is <225 mg/dl, and the patient is conscious and not acidotic, followed by oral initiation.

**SC Insulin:** When ketoacidosis is resolved, and the patient is fully conscious with a protected airway, the insulin infusion may be transitioned to SC administration. A bolus of long-acting basal insulin may be given, along with rapid or short-acting insulin at least 30 minutes before stopping the infusion and prior to the next meal.<sup>5</sup>

**When to Start Oral:** Patients should be kept NPO if they are in shock, drowsy, have impaired consciousness, or are experiencing severe acidosis. Oral fluids may be administered only when there is no vomiting, and the patients show significant clinical improvement with normal electrolyte levels. Oral intake may be allowed when the patients are clinically stable, and the acidosis (ketoacidosis) has resolved. If good clinical improvement occurs within 48 hours and rehydration is complete, oral intake may proceed, and the need for IV infusions can be reduced.

#### Consulting the Endocrinology Team

A consultation with the endocrinology team must be arranged in case the initial shock does not resolve with the first NS bolus, or when the child is aged <5 years, or there is severe hypokalaemia at the initial DKA stage, or there is severe and rapid fall in blood glucose despite the administration of full D10 fluids and minimal insulin dosage, or there is evidence of hypernatraemia, or there is non-resolving ketosis despite maximum insulin administration and declining blood glucose levels, or when there is severe acidosis or myocardial irritability, indicating that the use of  $\text{HCO}_3^-$  is necessary.

#### Documentation and Monitoring Protocol

**Fluid Balance and Insulin Infusion:** Fluid balance and the amount of insulin infusion must be documented accurately. All urine output and test for ketones must be measured, and all fluid intake, including oral fluids, must be recorded.

**Management of Diuresis:** If massive diuresis continues, physicians should consider increasing fluid intake. Large volumes of gastric aspirate must be replaced with 0.45% saline plus 10mmol/L KCl.

**Laboratory Monitoring:** Biochemistry, blood pH, BUN, calcium, magnesium, phosphorus, BOHB and laboratory blood glucose levels must be checked 2 hours after the start of resuscitation, and then at least every 4 hours. Fluid composition and infusion rates should be reviewed based on electrolyte results.

**Assessing Acidosis:** If acidosis is not corrected, this may indicate inadequate resuscitation, sepsis or insufficient insulin activity. Infusion lines and insulin doses need to be checked, and administering additional insulin, antibiotics and/or NS should be considered. The anion gap must be

calculated along with sodium and osmolality. Hourly capillary blood glucose measurements must be performed, and physicians should not rely on sudden changes. They should be verified with venous laboratory glucose measurements.

**Vital Signs and Neurological Monitoring:** Regular monitoring of vital signs is essential. Hourly or more frequent neurological observations should be conducted initially. The patient must be weighed twice daily to assess fluid balance.

**Symptoms and ECG Monitoring:** Symptoms, such as headache or any changes in consciousness or behaviour, must be monitored. Any changes in the ECG trace, especially T-wave changes that may suggest hyperkalaemia, must be watched.

#### Admission to Paediatric Intensive Care Unit (PICU) Criteria

Children with severe DKA (pH <7.1 or  $\text{HCO}_3^-$  <5mmol/L) should be promptly treated in a paediatric intensive care unit (PICU).

This includes children with prolonged symptoms, impaired circulation, reduced consciousness, and increased risk for cerebral injury (e.g., pH <7.1, partial pressure of  $\text{CO}_2$  [ $p\text{CO}_2$ ]  $\leq 20$ mmHg, BUN >20mg/dl).<sup>5,34</sup>

Children with significant electrolyte disturbances, such as hypo- or hyperkalemia, also require admission to PICU.<sup>25</sup>

Critical care consultation is recommended for less severe cases in children aged <5 years, particularly if they present outside of a tertiary care facility (35).

**Criteria for Discharge:** The following criteria need to be achieved prior to discharge:

**Laboratory Values:**  $\text{HCO}_3^-$  >15mmol/L, and anion gap <12 mmol/L.

**Clinical Status:** Tolerating oral intake, and ambulating independently.

**Education and Training (documented by a diabetes educator):** Use of blood glucose meter to test blood glucose; ability to draw up and administer insulin; ability to keep a Diabetes Diary; knowledge of carbohydrate counting; and knowledge of signs and symptoms of hypoglycaemia and treatment, including when and how to administer glucagon.

Laboratory Tests (for newly diagnosed patients only): ICAB (Islet Cell Antibodies), TSH, anti-TPO (Thyroid Peroxidase), Anti-MAB (Anti monoclonal Antibodies), and anti-TTG (Anti Transglutaminase antibodies).

**Home Situation:** Home environment should be safe and stable.

**Complications:** Cerebral oedema, hypoglycaemia, hypokalaemia, and aspiration pneumonia, which may be avoided by using an NG tube in children with impaired consciousness. Besides, abdominal pain may be noted due to liver swelling, gastritis, bladder retention and ileus. However, physicians should be aware of appendicitis, and arrange a consult for a surgical opinion once DKA is stable. A raised amylase level is common in DKA.

Few rare but potentially serious complication of DKA and its management may lead to:

Hypocalcaemia, Hypomagnesaemia, Severe Hypophosphatemia, Hyperchloraemic Acidosis, Hyperchloraemic Alkalosis. CNS includes Cerebral Venous Sinus Thrombosis, Basilar Artery Thrombosis, Intracranial Haemorrhage, Cerebral Infarction, Deep Venous Thrombosis and Cognitive Impairment.

Lungs related problems may be Pulmonary Embolism, Pulmonary Edema, Adult Respiratory Distress Syndrome, Pneumothorax, Pneumomediastinum, and Subcutaneous Emphysema. Other related consequences are Prolonged QTc, Rhabdomyolysis, Ischaemic Bowel Necrosis, Renal Failure, Acute Pancreatitis, Renal Damage, Thrombosis, Electrolyte Imbalance.

**Prevention and Education:** An early diagnosis of diabetes is helpful in designing and delivering diabetes awareness programmes and in identifying high-risk individuals. Paediatric diabetes can progress to DKA for several reasons, such as insulin omission, inadequate treatment, mismanagement, sepsis and adolescence. Importantly, DKA recurrences can be minimised by increasing parental involvement in care, reinforcing self-management education, and intensifying the diabetes care team's engagement with the family through frequent phone calls or clinic visits. Psychological counselling may also be beneficial. After children have recovered from DKA, it is important to discuss with them and their family members or caregivers the factors that may have contributed to the episode.

## Conclusion

Diabetic ketoacidosis is one of the commonest complications of either a partial or untreated diabetes in paediatric population especially among the adolescent. The management of DKA depends on early identification of the symptoms both in new onset and previous diabetes. Treatment of DKA generally based on consensus as develop by ISPAD, however there are few diversities as per the institutional policy and guidelines.

Fluid management with adequate amount and type is the corner stone followed by insulin therapy and electrolyte management as recommended. Special emphasize should be on minimizing the risk of cerebral oedema, electrolyte imbalance and fluid therapy.

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**NUAF:** Concept, drafting, final approval and agreement to be accountable for all aspects of the work.

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**BAK:** Concept, data interpretation, drafting and revision.

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