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Chapter

Diabetic Ketoacidosis

Hemchand K Prasad, Anand Kumar

ABSTRACT

Diabetic ketoacidosis (DKA) is a common but potentially dangerous presentation of type 1 diabetes mellitus in children. It indicates a profoundly insulin-deficient state with increased counter-regulatory hormones such as glucagon, growth hormone, and catecholamines, leading to hyperglycemia, lipolysis, and ketoacidosis and presenting with dehydration, electrolyte imbalance, and hyperosmolarity. Cerebral edema occurring in 0.5–0.9% of children with DKA is the most dreaded complication of DKA in children. Early recognition, protocol-based management, and meticulous monitoring form the crux of DKA management.

Keywords: Dehydration, Diabetic ketoacidosis, Electrolyte imbalance, Insulin, Cerebral edema, Hyperglycemia, Meticulous monitoring.

INTRODUCTION

Diabetic ketoacidosis (DKA) is a common presentation in children with type 1 diabetes mellitus (DM) and occasionally in older children and adolescents with type 2 DM. It is characterized by severe insulin deficiency causing a surge in counterregulatory hormones stimulating fat and protein catabolism, ketogenesis, hyperglycemia, and polyuria ultimately leading to dehydration, hyperosmolarity, and ketoacidosis.[1] Delay in diagnosis is the major cause of DKA in a previously unrecognized disease in younger children, whereas omission of insulin is the leading cause of DKA in previously diagnosed diabetic children, especially adolescents.

The *clinical signs of DKA* include:[1-3]

- Dehydration (which may be difficult to detect)
- Tachycardia
- Tachypnea (which may be mistaken for pneumonia or asthma)
- Deep, sighing (Kussmaul) respiration; breath has the smell of acetone (sometimes described as the odor of nail polish remover or rotten fruit)
- Nausea and vomiting (which may be mistaken for gastroenteritis)
- Abdominal pain that may mimic an acute abdominal condition

TABLE 1: Classification of diabetic ketoacidosis.

Severity	*Mild*	*Moderate*	*Severe*
Venous pH	<7.3	<7.2	<7.1
HCO_3 (mmol/L)	<15	<10	<5

- Confusion, drowsiness, progressive reduction in level of consciousness, and, eventually, loss of consciousness [mimics acute central nervous system (CNS) infection].

DEFINITION[1]

The *biochemical criteria* for the diagnosis of *DKA* are:

- Hyperglycemia [blood glucose (BG) >200 mg/dL (≈11 mmol/L)]
- Venous pH <7.3 or bicarbonate <15 mmol/L
- Ketonemia (≥3 mmol/L) and ketonuria (typically ≥2+).

Diabetic ketoacidosis is biochemically classified into mild, moderate, and severe depending on venous pH and bicarbonate level **(Table 1)**. A hypothetical case to explain the common pitfalls in the management of DKA is given in the following text.

A CLASSICAL CASE SCENARIO

A 4-year-old boy presented with difficulty in breathing. There was no history of cough, cold, or fever. He was treated with bronchodilator nebulization thrice and normal saline (NS) bolus 20 mL/kg. Respiratory distress persisted. Hence, he was referred to the tertiary care center. In the emergency room (ER), the observations were: tachypnea, pulses felt, capillary blood glucose (CBG)—high, point-of-care blood ketones—7.6 mmol/L, and pH—7.1. A diagnosis of severe DKA was made. As he was sick, a weight of 15 kg measured at the previous office visit was used. Let us assume the admission weight to be 14 kg. NS bolus 20 mL/kg intravenous (IV) bolus was given, IV fluids (maintenance with 8.5% deficit correction) NS started, and insulin infusion at 0.1 IU/kg/h started 1 hour later, and potassium 40 mEq/L added in fluid. He was shifted to the pediatric intensive care unit (PICU) and monitored as per DKA protocol **(Table 2)**; the child was found to be biochemically improving. 5-hour capillary blood glucose (CBG) was 245 mg/dL and acidosis started improving. Fluid changed to ½ DNS (dextrose normal saline), and there was a sudden deterioration in Glasgow Coma Scale (GCS) at 7 hours—9/15, bradycardia, high blood pressure (BP), airway not maintainable, intubated, and Inj. mannitol IV bolus given. Inj. sodium bicarbonate correction was given in view of persisting acidosis. There was a progressive deterioration in general condition with unequal pupils. Despite all efforts, he expired.

TABLE 2: Diabetic ketoacidosis monitoring chart.

	CBG (mg/dL)	*Ketone*	*Na*	*K*	*pH*	*HCO_3*	*GCS*	*HR (/min)*	*BP (mm Hg)*	*Fluid*	*Fluid rate (mL/h)*	*Insulin (IU/kg/h)*
4 pm	High	High			7.1	6	13	120	110/70	Normal saline	300	–
5 pm	620	High					14	130	110/60	Normal saline	73	0.1
6 pm	507	High	127	3.2	7.11	7	14	110	110/70	Normal saline	73	0.1
7 pm	300	High					13	110	110/70	Normal saline	73	0.1
8 pm	292	High	129	3.7	7.13	7	13	130	110/60	Normal saline	73	0.1
9 pm	245						14	120	110/70	½ DNS	73	0.1
10 pm	240		127	3.8	7.15	8	13	100	120/80	½ DNS	73	0.1
11 pm	230	Moderate					9	78	130/90	Inj. mannitol + ½ DNS	73	0.1
12 pm	230		125	3.9	7.17		7	68	80/50	Normal saline	73	
12.20 pm	Brainstem coning and death											

(BP: blood pressure; CBG: capillary blood glucose; DNS: dextrose normal saline; GCS: Glasgow Coma Scale; HR: heart rate)

Comments: This is an example of overaggressive and inappropriate management of DKA resulting in unfortunate death of the child. What went wrong? What should have been done? These are discussed as case audits to highlight the common pitfalls in the management of DKA.

MANAGEMENT

Emergency Assessment

- Initial assessment and stabilization of ABC (airway, breathing, circulation)—as per Pediatric Advanced Life Support (PALS) guidelines. Initial assessment should include evaluation of hydration status, the severity of dehydration, the level of consciousness through GCS, and body weight.
- *Weigh the child:* For all calculation purposes, the current weight of the child has to be used, and not the premorbid weight/weight measured in the previous outpatient visit.
- Estimation of dehydration—clinical or biochemical?[4-6] The assessment of dehydration in DKA is complicated by extra- and intravascular dehydration, metabolic acidosis affecting the clinical signs of dehydration, and the overall catabolic state of the patient. Despite their dehydration, patients generally continue to maintain normal or even have high BP, possibly due to elevated plasma catecholamine concentrations, increased release of antidiuretic hormone (ADH) in response to hyperosmolality, which increases BP via V2 receptors, or other factors. Hence, the assessment of severity of dehydration may be challenging and commonly leads to over- or underestimation.
  - *Clinical:* The useful individual signs for predicting dehydration in young children are as follows:
    - *5%:* Reduced skin turgor, dry mucous membranes, tachycardia
    - *10%:* Capillary refill time ≥3 seconds, sunken eyes
    - *>10%:* Weak or impalpable peripheral pulses, hypotension, shock, oliguria[2]
  - *Biochemical:* Dehydration/deficit depending on the biochemical severity of DKA: Mild: 3–5%, moderate: 5–7%, and severe: 7–10%[1]
- Take samples for BG, blood ketone [beta-hydroxybutyrate (BOHB)], electrolytes, urea, creatinine, blood gas, venous pH and pCO_2, serum osmolality, complete blood count (CBC), and blood and urine culture if indicated.
- If laboratory values are delayed, perform an electrocardiogram for baseline evaluation of potassium status.
- DKA should be managed in a unit with preformed management and monitoring guidelines with nursing staff trained in DKA management and a laboratory with quick turnaround times.

- Considering the fallacies in clinical assessment, it would be wise to take an individualized decision based on biochemical and clinical parameters to assess the severity and percentage of dehydration.

Case audit		
Intervention	***Right/wrong***	***Appropriate intervention***
History	×	Focused history with high index of suspicion. History of polyuria, polydipsia, and weight loss
Weight	×	Current weight should always be used
Vitals	×	CBG should be checked in all cases of unexplained tachypnea
Nebulization	×	No role in DKA. Might aggravate hyperglycemia by increasing glucagon secretion and hepatic glycogenolysis

The essential principles of DKA management include careful replacement of fluid deficits, correction of dehydration, correction of acidosis and hyperglycemia via insulin administration, maintenance of glucose levels within normal range, correction of electrolyte imbalance, and treatment of precipitating cause if any.

Fluid[1]

Fluid management includes immediate volume resuscitation with fluid boluses when necessary, deficit correction, and maintenance fluids.

- *Resuscitation fluids:*
  - Not required in mild-to-moderate DKA
  - In severe DKA without shock: 10–20 mL/kg over 1–2 hours
  - In severe DKA with shock (documented hypotension): 20 mL/kg over 20 minutes
  - In case of septic shock, 20 mL/kg multiple boluses
- *Deficit replacement:*
  - Usual fluid deficit in DKA depending on severity: *Mild:* 3–5%, *moderate:* 5–7%, *severe:* 7–10%
  - *NS versus ½ NS:* Replacement fluid should be isotonic [NS/Ringer's lactate (RL)/PlasmaLyte] for at least 4–6 hours to avoid rapid fall in osmolarity, which might precipitate cerebral edema. Then decide to change tonicity to ≥0.45% saline depending on hydration status, serum sodium, and serum osmolality as follows:
- If serum sodium is rising, hydration status improving, and serum osmolality gradually falling, change fluid to ½ NS (0.45%).
- If measured sodium is low or falling, hydration status is not improving, or serum osmolality rapidly falling, continue NS (0.9%).

- Deficit should be administered over 48 hours to avoid cerebral edema. Rate of fluid administration = (maintenance fluid for 48 hours + deficit) – (bolus + fluids prior to admission)/48. The total maintenance rate should not exceed 1.5–2 times the normal maintenance rate. Fluid administered in the first 4 hours should not exceed 50 mL/kg.
- Except for severely ill, oral intake usually begins within 24 hours. In that case, the remaining deficit has to be corrected using oral fluids.
- Measured serum sodium should increase with an appropriate increase in intravascular volume and fall in blood sugar. Failure of the rise in sodium indicates increased free water administration, hence the risk of cerebral edema. This mandates an increase in the tonicity of the fluid.
- Check BG hourly. Initially, there is a rapid fall in BG followed by a steady decline at 40–100 mg/dL/h. At any time, fall in glucose should not be >100 mg/dL/h.
- Add 5% dextrose to IV fluid once BG falls <250–300 mg/dL. If BG falls <150 mg/dL, 10% dextrose (or even 12.5% dextrose) has to be added. If the rate of fall in glucose is >100 mg/dL, add dextrose to in vitro fertilization (IVF) irrespective of CBG (even if >200 mg/dL).
- Caution: Chloride-rich fluids are associated with hyperchloremic metabolic acidosis which notoriously cause low bicarbonate and mask resolution of ketoacidosis. This problem may be avoided by using potassium acetate instead of potassium chloride or using RL or PlasmaLyte instead of NS.

Case audit—fluid

Intervention	***Right/wrong***	***Appropriate intervention***
Rapid fluid bolus	×	Only in severe DKA without shock, fluid bolus should be given over 1–2 hours
Use of normal saline	✓	Isotonic fluids are the fluid of choice initially
Fluid calculation	×	Preadmission fluids and fluid boluses should be subtracted
Initial choice of fluid	✓	Isotonic fluids—NS/RL/PlasmaLyte
Rate of fluid Initial 4 hours = 880 mL (300 + 300 + 280)	×	Not >50 mL/kg (14 × 50 = 700 mL in our case) over initial 4 hours including the bolus
Maintenance fluid rate	×	Premorbid weight was used
Fall in CBG and rise in sodium	×	Fall in CBG is >100 mg/dL/h and fall in sodium indicating increase in free water
Addition of dextrose to fluid	×	Though dextrose was added when CBG <300 mg/dL, it should have been added earlier as fall in CBG was precipitous
Change of fluid to 0.45% saline	×	Fluid should have been kept isotonic (0.9%) as measured sodium was not rising

Insulin

Insulin is required to restore normal glucose metabolism, reduce BG levels, suppress lipolysis and ketogenesis, and thereby resolve ketoacidosis. Insulin may be started at a low dose (0.05 IU/kg/h) or a standard dose (0.1 IU/kg/h); advantages and disadvantages of each are discussed in **Table 3**. It may be preferred to start insulin at 0.05 IU/kg/h and increase it gradually to 0.1 IU/kg/h in case of persistent acidosis or slow resolution of acidosis. Special conditions are as follow:

- In infants and children with marked insulin sensitivity, *0.025 IU/kg/h* may be used.
- In cases with severe insulin resistance, suspected type 2 DM, and sepsis, the dose may be escalated up to *0.3 IU/kg/h*.
  - Preparation/dilution—add 50 IU of regular insulin in 50 mL NS and flush 25 mL of this solution through the tubings (priming).
  - Start insulin 1–2 hours after the initial fluid therapy. Do not give insulin bolus.
  - Check potassium before starting insulin therapy; correct hypokalemia if present.
  - Monitor pH and blood ketones. pH should increase by 0.03/h. If ketoacidosis fails to improve, check insulin infusion preparation, priming of tubing, and rate of infusion before increasing the dose of insulin.
  - Do not decrease the insulin infusion if the BG concentration decreases too quickly (>100 mg/dL/h) or falls too low before DKA has resolved; rather, increase the amount of glucose administered.
  - *When to stop?* Ketoacidosis takes longer time to resolve than hyperglycemia. Hence, the *dose of insulin should remain at 0.05–0.1 IU/kg/h till complete resolution of DKA* (pH >7.30, bicarbonate >15 mmol/L, BOHB <1 mmol/L, or closure of anion gap).

TABLE 3: Comparison of low-dose and standard-dose insulin infusion in diabetic ketoacidosis.

Low-dose insulin therapy (0.05 IU/kg/h)	***Standard-dose insulin therapy (0.1 IU/kg/h)***
Equally efficacious in resolving hyperglycemia and ketoacidosis	Highly efficacious and well studied
Graded fall in blood glucose	Rapid fall in blood glucose
Chances of hypoglycemia and hypokalemia less	More risk of hypoglycemia and hypokalemia
Doubtful: Will it be efficacious in case of severe DKA?	*Doubtful:* Is risk of cerebral edema more?

(DKA: diabetic ketoacidosis)

- *Subcutaneous insulin* regimen:[7] Studies have shown that subcutaneous (SC) rapid insulin regimen can be effective in DKA (mild) without precipitating hypoglycemia or hypokalemia. This regimen can be used in circumstances such as primary healthcare settings where IV insulin infusion is not possible or in uncomplicated DKA. It is not to be used in patients with poor perfusion. The initial dose is 0.3 U/kg (rapid insulin lispro/aspart) followed 1 hour by SC insulin at 0.1 U/kg every hour or 0.15–0.20 IU/kg every 2 hours till resolution of DKA.

Case audit—insulin

Intervention	***Right/wrong***	***Appropriate intervention***
No insulin bolus	✓	Measure to prevent cerebral edema
Insulin therapy started 1 hour after fluid	✓	Measure to prevent cerebral edema
Rate of insulin infusion	×	Start at a low dose of 0.05 U/kg/h. Our goal is to avoid a rapid fall in blood glucose and precipitation of cerebral edema
Rate of pH change	×	Rise in pH <0.03/h. Insulin dilution, priming, and infusion should be checked; if proper, then the dose of insulin should be increased. A rapid rise pointer to onset of cerebral edema

Potassium

Diabetic ketoacidosis is associated with average total body potassium deficit of 3–6 mmol/kg due to the following mechanisms:

- Transcellular shifts of this ion caused by hypertonicity, glycogenolysis, and proteolysis secondary to insulin deficiency cause potassium efflux from cells.
- Vomiting
- Volume depletion causes secondary hyperaldosteronism, which promotes urinary potassium excretion.

Hence, IV potassium replacement is mandatory irrespective of initial serum potassium status, except in renal dysfunction. Renal dysfunction, by enhancing hyperglycemia and reducing potassium excretion, may contribute to hyperkalemia.

Document initial serum potassium. If it is unavailable or delayed, electrocardiogram (ECG) should be done. Prolongation of the PR interval, T-wave flattening and inversion, ST depression, prominent U waves, and apparent long QT indicate hypokalemia.

Tall, peaked, and symmetrical T waves and shortening of the QT interval indicate hyperkalemia.

- If hypokalemic, start potassium replacement simultaneously with initial fluid expansion.
- If hyperkalemic, start potassium replacement after documenting urine output.
- If normokalemic, start potassium after initial fluid resuscitation and concurrent with starting insulin therapy.
- Potassium concentration of infusate should be 40 mEq/L, in the form of potassium chloride 20 mEq/L with potassium acetate/phosphate 20 mEq/L. Potassium replacement entirely in the form of potassium chloride carries the risk of hyperchloremic metabolic acidosis as discussed earlier.
- The rate of potassium should not exceed 0.5 mEq/kg/h.

Case audit—potassium

Intervention	***Right/wrong***	***Appropriate intervention***
Timing of potassium supplementation	✓	After initial fluid bolus
Concentration of potassium supplementation	✓	Individualized, based on serum potassium (if available) or ECG findings

Role of Sodium Bicarbonate

There is no evidence suggesting faster metabolic recovery with bicarbonate therapy. Instead, it may cause paradoxical CNS acidosis, rapid correction of acidosis can cause hypokalemia, the additional sodium can add to hyperosmolality, and alkali therapy can increase hepatic ketone production, potentially slowing recovery.[8]

Indications: It may rarely be used in the cases with severe acidosis pH <6.9 with cardiocirculatory compromise and in life-threatening hyperkalemia.

Case audit—$NaHCO_3$

Intervention	***Right/wrong***	***Appropriate intervention***
Administration of sodium bicarbonate for cerebral edema	×	Strictly not to be used in DKA except in life-threatening hyperkalemia/ acidosis (pH <7) with cardiocirculatory compromise

Monitoring

Frequent meticulous monitoring of clinical and biochemical parameters is a very important part of management. The parameters to be monitored and frequency at which they need to be monitored are summarized in **Table 4**.

- Serum Na concentration is not reliable for determining extracellular fluid (ECF) deficit because of the osmotic effect of hyperglycemia-induced dilutional hyponatremia and the low Na content of the elevated

TABLE 4: Clinical and biochemical monitoring in diabetic ketoacidosis.

Hourly	*2nd–4th hourly*	*Once*
Vitals	Blood BOHB (superior than point-of-care BOHB)	Complete hemogram, blood culture in case of suspected infection
Sensorium/GCS	Electrolytes	Calcium, phosphorus, magnesium
CBG	BUN	Urine routine, ketone
Input/rate of fluid infusion	Blood gases	HbA1c
Output (urinary output need not be replaced except in rare cases)	Derived/calculated parameters: Corrected sodium, anion gap, effective osmolality	

(BOHB: beta-hydroxybutyrate; CBG: capillary blood glucose; BUN: blood urea nitrogen; GCS: Glasgow Coma Scale; HbA1c: hemoglobin A1c)

lipid fraction of the serum in DKA. Corrected Na, i.e., for normal glucose levels, can be estimated by adding 1.6 mEq to the measured value for each 100 mg/dL BG above normal.[8]

$$\text{Corrected sodium} = \text{Measured sodium} + 1.6 \times [(\text{plasma glucose} - 100)/100]$$

- As BG and ketones fall, pH and corrected sodium should rise indicating an appropriate improvement in hydration.

Case audit—monitoring and interpretation

Intervention	*Right/wrong*	*Appropriate intervention*
• Monitoring to recognize early risk factors for cerebral edema	×	Rapid fall of sugars and lack of rise of serum sodium should have been recognized early and a high index of suspicion for cerebral edema was needed in the child
• Rate of fall of pH >0.03/h	×	
• Rate of fall of sugar 40–100 mg/dL/h	×	
• Corrected sodium should increase with resolution of acidosis	×	

CEREBRAL EDEMA

Cerebral edema is the most dreaded complication in childhood DKA with an incidence of 0.5–0.9% and a mortality rate of 21–24%. Subclinical cerebral edema is much more common.

Risk factors

Risk factors include an initial pH of <7.1, abnormal baseline mental status, newly diagnosed patients who are <5 years old, patients suffering from dehydration and severe acidosis with lower partial pressure of carbon

dioxide, rapid rehydration (>50 mL/kg in the first 4 hours), insulin given before or within 1 hour of fluid initiation, persistent hypernatremia, and high blood urea at presentation.[9]

Criteria[10] (Table 5)

One diagnostic criterion and two major criteria/one major and two minor criteria have a sensitivity of 92% and a false-positive rate of only 4%.[1] Signs that occur prior to treatment should not be considered. The time of onset is distributed in a bimodal fashion; majority of patients develop signs and symptoms in the first 6–7 hours and the rest from 10–24 hours after the start of treatment, but can occur at any point of time, even before initiating therapy.[8]

Signs and Symptoms

Major symptoms are headache, slowing of heart rate, change in neurological status (restlessness, irritability, increased drowsiness, and incontinence), specific neurological signs (e.g., cranial nerve palsies, papilledema), recurrence of vomiting, rising BP, and decreased O_2 saturation.

Management

- Initiate treatment as soon as the condition is suspected.
- Reduce the rate of fluid administration by one third.
- Elevate the head of the bed to 30°.
- Give mannitol, 0.5–1 g/kg IV over 10–15 minutes, and repeat if there is no initial response in 30 minutes to 2 hours. Hypertonic (3%) saline

TABLE 5: Criteria for cerebral edema in diabetic ketoacidosis.

Diagnostic	***Major***	***Minor***
Abnormal motor/verbal response to pain	Altered mentation/fluctuating level of consciousness	Vomiting
Decorticate/decerebrate posture	Sustained heart rate deceleration (decrease >20 bpm) not attributable to improved intravascular volume or sleep state	Headache
Cranial nerve palsy (III, IV, VI)	Age-inappropriate incontinence	Lethargy or not easily arousable
Abnormal neurogenic respiratory pattern (grunting, tachypnea, Cheyne–Stokes/ apneustic respiration)		• Diastolic BP >90 mm Hg • Age <5 years

(BP: blood pressure)

TABLE 6: Sliding scale for insulin administration after resolution of diabetic ketoacidosis.

Sugar value (mg/dL)	*Trend (IU/kg)*
90–180	0.1
180–270	0.2
270–360	0.3
0	0.4

2.5–5 mL/kg over 10–15 minutes may be used as an alternative to mannitol, especially if there is no initial response to mannitol. Hence, hyperosmolar agents should be readily available at the bedside during DKA management.

TRANSITION TO ORAL FLUIDS/SUBCUTANEOUS INSULIN

- Except in severely ill, oral intake usually begins within 24 hours of therapy. IV fluid has to be reduced accordingly with increasing oral intake so that the total of oral and IV fluid should not exceed the calculated rate of maintenance fluid. This calculated fluid restriction should be continued for 48 hours from admission.
- If ketoacidosis has resolved and oral intake is adequate, transition to SC insulin should be planned preferably before a meal. To prevent rebound hyperglycemia, first SC insulin should be given 15–30 minutes (with rapid-acting insulin) or 1–2 hours (with regular insulin) prior to stopping insulin.
- Frequent BG monitoring is a must for the next 24 hours.
- *Sliding scale:* Administration of short-acting insulin every 6 hours based on sugar values **(Table 6)** may be useful in the initial phases (for quick transition) to assess daily insulin requirement and planning of an appropriate insulin regimen for a given child.

CONCLUSION

Diabetic ketoacidosis is the most common pediatric endocrine emergency due to insulin deficiency in milieu of excess counter-regulatory stress hormones leading to cascade of metabolic derangements progressing to dehydration and ketoacidosis. Though life threatening, it can be successfully managed if promptly diagnosed with fluid and insulin therapy and meticulous monitoring of hydration status, sensorium, and electrolytes. Cerebral edema is an uncommon but fatal complication that can be prevented with gentle/optimal use of fluid and insulin but aggressive monitoring of the patient.

KEY LEARNING POINTS

- Diabetic ketoacidosis may mimic pneumonia, acute gastroenteritis, and acute CNS infection. It is prudent to have a high index of suspicion and obtain CBG in such children and ask for history of polyuria, polydipsia, and weight loss.
- It is better to suspect and not find DKA, rather than not suspect it and then find it.
- No fluid bolus unless indicated, no insulin bolus
- Be gentle in fluid and insulin therapy. Always supplement potassium.
- Use infusion pumps. Use admission weight. Recheck all calculated infusion rates.
- Strict and programmed monitoring of vitals, GCS, and biochemical parameters is of utmost importance to avoid cerebral edema. Hyperosmotic agents should always be available bedside for its immediate management.
- Inappropriate management of DKA is as fatal as DKA itself.

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Recent Advances in PEDIATRICS

(Special Volume 29: *Emergencies and Intensive Care*)

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Contents

Suraj Gupte MD (PGIMER) FIAP FSAMS (Sweden) FRSTMH (London) hails from the alumni of the Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh, India. At present, he is Emeritus Professor and Head, Postgraduate Department of Pediatrics, Mamata Medical College and Mamata General and Superspecialty Hospitals, Khammam, Telangana, India. He occupies pride of place for outstanding contribution as a Pediatric Educationist, Author, Researcher, and Innovator globally. He is the Chief Editor of several books, including the international series, *Recent Advances in Pediatrics* (56 volumes so far), *The Short Textbook of Pediatrics* (now in 13th edition), *Pediatric Gastroenterology, Hepatology and Nutrition* (an Indo-American project), *Pediatric Infectious Diseases, Case-based Reviews in Pediatric Emergencies, Clinical Problem-solving in Neonatal Emergencies and Intensive Care, Long and Short Cases in Pediatrics*, etc. Among the prestigious books exclusively authored by him figure *Pediatric Drug Directory, Differential Diagnosis in Pediatrics, Instructive Case Studies in Pediatrics, Influenza, Perspectives in Influenza and Speaking of Child Care*. He has published/presented over 280 papers at national and international periodicals/conferences/workshops/symposia and delivered several orations.

He, besides holding important positions in the Indian Academy of Pediatrics (IAP) and its subspecialty chapters, has represented India and IAP at several international conferences, workshops, symposia, seminars, etc. worldwide as Plenary Speaker, Guest Speaker, Chairperson, Panelist, etc. He was an Advisor, 5th International Congress of Tropical Pediatrics (1999) and Executive, 8th Asian Congress of Pediatrics (1994), Co-Chairperson, WHO/UNICEF Workshop on Child Nutrition (2012), Organizing Chairman, Afro-Asian Workshop on Pediatric Gastroenterology, and Nutrition (2018). He is a recipient of several national and international awards, including International Achievement Award from International Society of Pediatric Gastroenterology, Hepatology, Hepatobiliary Transplant and Nutrition (2016) and Lifetime Achievement Awards from National Academy of Pediatric Neurology (2022) as also several Visiting Fellowships/Professorships. Furthermore, he has been an Examiner for several universities, including IGNOU, AIIMS, PGIMER, SKIMS, and a Faculty Selection Expert for AIIMS, UPSC, and State PSCs. He figures in most of the iconic national and international Who's Who volumes, including the *Morqui's Who's Who*—a pre-eminent database of outstanding global personalities.

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