# Guidelines
# For The Management Of

# Diabetic Ketoacidosis


By

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Clinical Standards
& Guidelines

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Diabetic ketoacidosis is a state of absolute or relative insulin deficiency aggravated by ensuing hyperglycemia, dehydration, and acidosis.

Diabetic ketoacidosis is typically characterized by hyperglycemia over 300 mg/dL, low bicarbonate level (<15 mEq/L), and acidosis (pH <7.30) with ketonemia and ketonuria.

**Moderate DKA**: pH <7.2 and serum bicarbonate <10 mEq/L

**Severe DKA**: pH <7.1 and serum bicarbonate <5 mEq/L.

**Presentation** ketoacidosis could be the first presentation of undiagnosed case of type 1 D.M with abdominal pain, vomiting, hyperventilation, polyurea, ultered mental status, hypotension and shock

**Precipitating factors** infection, MI, sever stress & disruption of insulin treatment

## Investigations

- Blood sugar, urea, creatinine, electrolytes, (Sugar & Potassium level needs to be checked every 1-2 hours)
- Arterial blood gases, anion gap (**higher than normal** )

*Anion gap* = $([Na+] + [K+]) - ([Cl-] + [HCO3-])$ normal value : 8 to 12 mEq/L

- Osmolality: $2(Na^+)$*mEq/L* $+2(K^+)$*mEq/L + glucose (mg/dL)/18 + BUN (mg/dL)/2.8.*

*If BUN not available, use B.Urea in (mgldL) & divide by 6, i.e., Urea/6 to convert to mEq/L*

Normal value 285 - 295 mOsm/kg, In Diabetic ketoacidosis, the osmolality is usually more than 330 mOsm/kg, if it is less than that, then think of other diagnosis.

- CBC (detect infection)
- Urinalysis for glucose, ketones and to detect underlying urinary infection
- Electrocardiography (ECG): Diabetic ketoacidosis may be precipitated by a cardiac event, in addition, it is a rapid way to asses significant hypokalemia or hyperkalemia
- Culture of blood, urine and CSF culture in selected cases.
- Chest radiography: to rule out pulmonary infection.
- CT scanning in selected cases

### Treatment

***Aim:***

***Correcting dehydration***

***Reversing the acidosis and ketosis***

***Reducing plasma glucose concentration to normal***

***Replenishing electrolyte and volume losses***

***Identifying the underlying cause***

- **Admit to ICU** or monitored unit depending on clinical status.
  Airway management and potential intubation should be a primary concern in any patient with a significantly depressed mental status or with respiratory distress.
  Bladder catheterization if no urine in the 1st 3 hours.
  N/G tube is needed if there is repeated vomiting or depressed mental state

- **Fluid** start fluid therapy before insulin therapy as follows
  - 1 liter of 0.9% saline stat. Then, typically, 1L over the next hour, 1L over 2h, 1L over 4h, then 1L over 6h
  - Use dextrose saline or 5% dextrose when blood glucose is < 250 mg /dL
  - Those >65yrs or with congestive heart failure CCF, need less saline more cautiously
  - Pediatric 10-20 mL/kg of normal saline (0.9%) solution over the first 1-2 hours without initial bolus, and then, start insulin, this is to avoid cerebral oedema

  Total volume over the first 4 hours should not exceed 40-50 mL/kg

- **Insulin** should be started about an hour after intravenous fluid replacement is started to allow for checking potassium levels and because insulin may be more dangerous and less effective before some fluid replacement has been obtained.

  Correct potassium first if the serum $K^+$ level < 3.3 mmol/L before giving any insulin
  - *Loading dose*: 0.1 - 0.15 U/kg IV bolus ( average 10 U )
  - *Maintenance doses*: 0.1 U/kg/h IV infusion, typically 4 - 6 U/hr according to the blood sugar .

  Insulin infusion dosage can be arranged by diluting 50 units of soluble insulin in 500 c.c normal saline i.e., I unit /10c.c. To infuse :

- 1 unit /hr = 3 drops/min
- 2 units/hr = 6 drops/min
- 4 units/hr = 12 drops/min
- 6 units/hr = 18 drops/min

*Goal* is to reduce glucose by **50-70 mg/dL/hr,**maximum **100 mg/dL/hr**
*Pediatric*: administer as in adults without loading dose (may increase risk of cerebral edema)
Change the IV fluid to 5% glucose when **blood sugar is 200 – 250 mg/dL** and keep it *to one hour after starting the SC insulin* according to the sliding scale done three times daily before meals when the patient starts oral feeding and he has no vomiting

| ***Premeal glucose result (mg/dL)*** | ***Soluble insulin SC*** |
|---|---|
| 100 – 140 | 2 U |
| 140 – 180 | 4 U |
| 180 – 220 | 6 U |
| 220 – 260 | 8 U |
| 260 – 300 | 10 U |

- **Potassium** don't add $K^+$ to the first bag unless the level is < 3.3 mmol/L less will be required in renal failure

  Check urea, creatinine and electrolytes hourly initially.

  If potassium level is not available, refer to T wave in ECG lead II.

| ***Serum $K^+$ (mmol/L)*** | ***Amount of KCl to add per liter of IV fluid*** |
|---|---|
| <3.0 | 40mmol |
| 3 – 4 | 30mmol |
| 4 - 5 | 20mmol |
| 5 – 6 | 10 mmol |
| >6 | Non |

- **Bicarbonate** typically is not replaced since acidosis will improve with the above treatments alone.
  Administration of bicarbonate has been correlated with cerebral edema in children.
  If acidosis is severe (pH <7), give IV bicarbonate 1mL/kg of 8.4% over 1h, and after the first liter of fluid and recheck arterial pH aiming for pH not more than 7.1

  **Magnesium** administration should be considered if
  - Patient is alcoholic, on diuretic
  - Cardiac arrhythmia, parasthesia, tremor, carpopedal spasm, seizure
  - Refractory hypokalemia (low potassium after full correction according to the above guidelines).
  - Magnesium level less than 1.8 mg/dl (0.74 mmol/L)
  - 1 - 2 gm magnesium sulfate in 50 c.c. solution IV over 15 to 30 minutes
  - In patient with even mild renal failure, magnisium replacement should be done with extreme caution due to the risk of hypermagnesemia

- **Find and treat infection** (lung, skin, perineum, urine after cultures).
- **Heparin** 5000U/8h (or low molecular weight version) SC in selected critical cases
- At any stage of treatment, consult senior doctor if the patient is not improving.

## Complications

***Cerebral edema,***

The leading cause of mortality in children
- Occurs 4-12 hours into treatment,
- Presented as abnormal response to pain, decorticate and decerebrate posturing, cranial nerve palsies, abnormal CNS respiratory patterns, fluctuating level of consciousness, sustained bradycardia, incontinence, vomiting, headache, lethargy, and elevated diastolic blood pressure.
- The risk is related to the severity and duration of diabetic ketoacidosis, associated with ongoing hyponatremia and with the administration of bicarbonate.
- 50% of children have subtle brain MRI findings, in particular narrowing of the lateral ventricles
- Hypertonic saline (3% saline, 3-5 ml /kg) or intravenous 20% Mannitol 0.5 g/kg (2.5 ml/kg) may be given over 20 minutes and repeated if no response is seen in 30-120 minutes,
if no response occurs, consider Dexamethasone with senior doctor consultation.

***Hypokalemia***

Manifested by abnormal ECG, arrhythmia, respiratory paralysis or sudden death

***Hypoglycemia***

May result from inadequate monitoring of glucose levels during insulin therapy.

***Acute pulmonary edema***

Related to aggressive or excessive fluid therapy especially in renal failure or congestive heart failure

***Others***

CVA, MI, DVT, Erosive gastritis, late hypoglycemia, Respiratory distress, Infection, Hypophosphatemia, Mucormycosis

**Medicolegal Pitfalls**

- Plasma glucose is usually high, but not always, levels may be as low as 250 mg/dL
-Failure to consider other coexisting illnesses, such as pelvic or rectal abscess, pneumonia, and silent MI
-High white cell counts may be seen in the absence of infection.
-Infection: there may be no fever.
-Failure to evaluate for other causes of coma if osmolality is relatively normal
-Creatinine: some assays for creatinine cross-react with ketone bodies, so plasma creatinine may not reflect true renal function, high levels of ketone bodies may lead to factitious elevation of creatinine level.
- High triglyceride levels may lead to factitious low glucose level;
-High serum glucose levels may lead to dilutional hyponatremia due to the osmotic effect of hyperglycemia, for each 100 mg/dL of glucose over 100 mg/dL, the serum sodium level is lowered by approximately 1.6 mEq/L & when glucose levels fall, the serum sodium level rises by a corresponding amount

*corrected plasma* $[Na^+] = Na^+ + 2.4 \left[ (glucose - 5.5) / 5.5 \right]$

-Ketonuria does not equate with ketoacidosis. Normal individuals may have up to ++ketonuria after an overnight fast. Not all ketones are due to diabetes, consider alcohol if glucose is normal.
-Recurrent ketoacidosis: blood glucose may return to normal long before ketones are removed from the blood, and a rapid reduction in the amount of insulin administered may lead to lack of clearance and return to DKA. This may be avoided by maintaining a constant rate of insulin, 4-5U/h IVI, and co-infusing dextrose 10-20% to keep plasma glucose at 108 –180mg/dL (6-10mmol/L )
-Acidosis but without gross elevation of glucose may occur, but consider overdose of aspirin and lactic acidosis (in elderly diabetics).
-Serum amylase is often raised (up to 10 x) and non-specific abdominal pain is common even in the absence of pancreatitis.

## References


www.emedicine.medscape.com/article/766275-overview/ Diabetic Ketoacidosis/ Donald W Rucker/ Jun 4, 2010

www.medscape.com/viewarticle/713278/ Paediatric Diabetic Ketoacidosis/ Simon Steel, Shane M Tibby, / 12/15/2009

www.emedicine.medscape.com/article/118361-overview/ Diabetic Ketoacidosis/ Osama Hamdy/ Sep 1, 2009

Oxford Handbook of Clinical Medicine/ Eighth Edition/Murray Longmore,Ian B Wilkinson, Tom Turmezei,Chee Kay Cheung/Diabetic Ketoacisosis

Oxford handbook of Endocrinology with Diabetes mellitus/2nd edition/ Emergency in Diabetes Mellitus/Ketoacidosis .Helen E. Turner/John A.H. Wass,

www.en.wikipedia.org/wiki/Plasma_osmolality

Harrison's Principles of Internal Medicine, 17th Edition: Anthony S. Fauci, Eugene Braunwald, Dennis L. Kasper and Stephen L. Hauser