Diabetic Ketoacidosis Management

Shahab Noorian, MD

Pediatric Endocrinologist

Alborz Medical University

Goals of Discussion

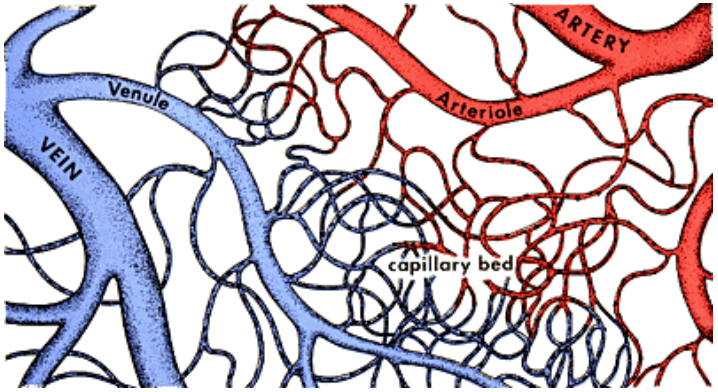
- Initial hospital management
  - Replace fluid and electrolytes
  - IV Insulin therapy
  - Glucose administration
  - Watch for complications
  - Disconnect insulin pump
- Once resolved
  - Convert to home insulin regimen
  - Prevent recurrence

Epidemiology

- Annual incidence in U.S.
  - 5-8 per 1000 diabetic subjects
- 2.8% of all diabetic admissions are due to DKA
- Overall mortality rate ranges from 2-10%

So, What are Ketones?

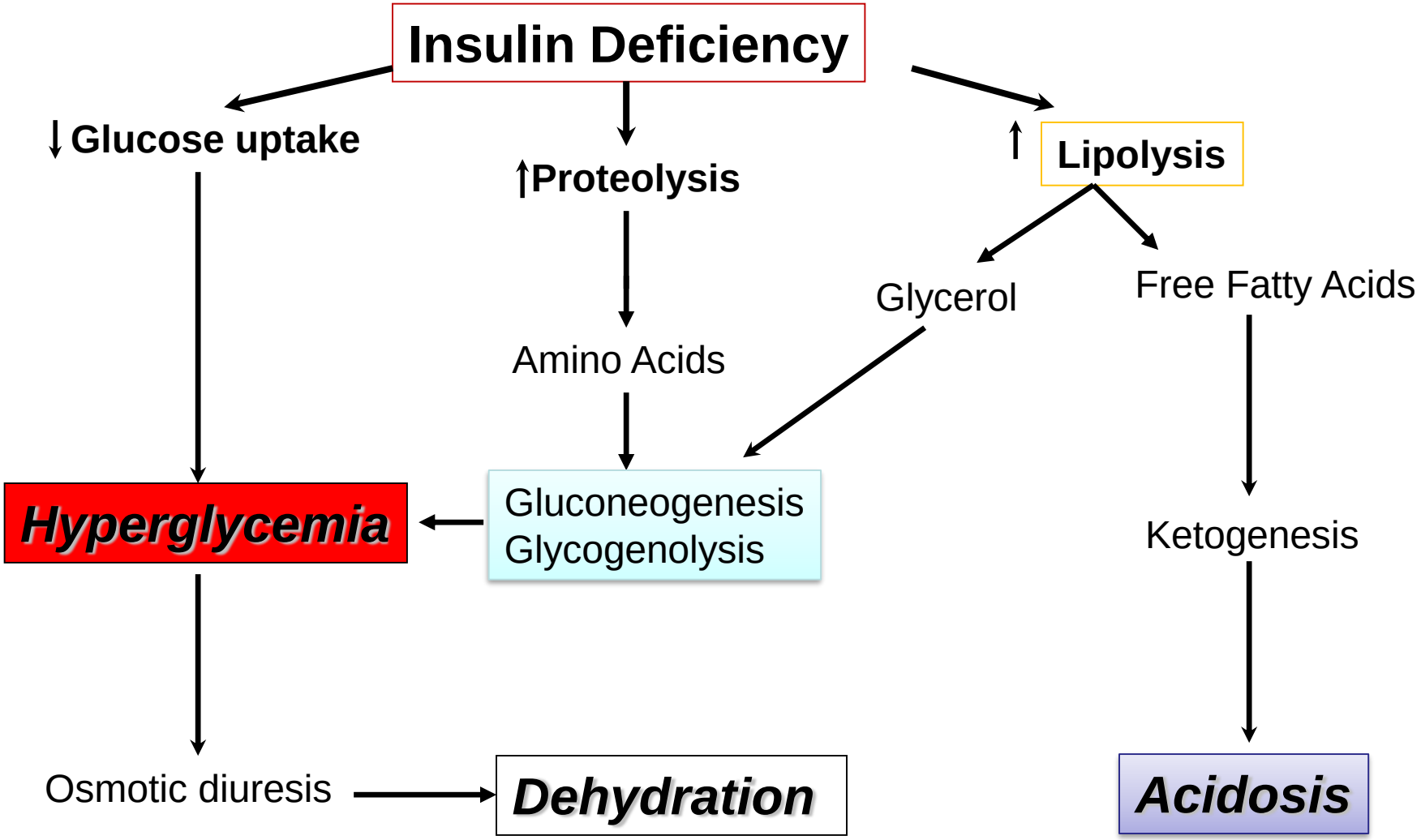
- Ketones are acids that build up in the blood.
- They appear in the urine when the body doesn't have enough insulin.
- They can poison the body.
- They are present in high amounts in a condition known as: <u>Ketoacidosis.</u>



Diabetic Ketoacidosis

Due to:

1. Severe insulin deficiency
2. Excess counterregulatory hormones

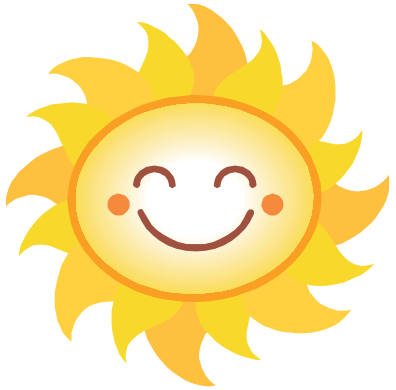

Insulin Deficiency
↓ Glucose uptake
↑Proteolysis
↑
Lipolysis
Glycerol
Free Fatty Acids
Amino Acids
Hyperglycemia
Gluconeogenesis
Glycogenolysis
Ketogenesis
Osmotic diuresis
Dehydration
Acidosis

Signs and Symptoms of DKA

- Polyuria, polydipsia ,Enuresis
- Dehydration
  - Tachycardia
  - Orthostasis
- Abdominal pain
  - Nausea
  - Vomiting
- weight loss

- Fruity breath
  - Acetone
- Kussmaul breathing
- Mental status changes
  - Combative
  - Drunk
  - Coma

- So,Classic presentation of diabetes in children is a history of:
- ***polyuria,***
- ***polydipsia,***
- ***polyphagia***,
- and, usually for up to one month



Lab Findings

- Hyperglycemia>200mg/dl
- Anion gap acidosis
  - (Na + K) – (Cl + Bicarb) >12
  - Venous pH <7.3,Arterial PH<7.35
  - Bicarbonate <15 mEq/L
- Urine ketones and serum ketones

- Hyperosmolality(300-350)
- Serum amylase may be elevated.
- Leukocytosis is common

hyperosmolality

- Hyperosmolality as a result of progressive hyperglycemia contributes to cerebral obtundation in DKA

- Serum osmolality:
  - $\{\text{Serum } Na^+ + K^+\} \times 2 + \frac{\text{glucose}}{18} + \frac{\text{BUN}}{3}$

two,initial Questions

1. Severity of DKA
2. Time of treatment

Table 1. Classification of diabetic ketoacidosis

	Blood glucose (mmol/L)	Venous pH	Bicarbonate (mmol/L)
Mild	>11	<7.3	<15
Moderate	>11	<7.2	<10
Severe	>11	<7.1	<5

Siafarikas A, O'Connell S. Australian Family Physician 2010;39(5):2

.

Address Severity of:DKA /Acidemia:

	CO2	PH (V)	Clinical
Normal	20-28	7.35 – 7.45	Normal Base line
Mild	16-20	7.25 – 7.35	Oriented, alert but Fatigued
Moderate	10-15	7.15 -7.25	Kussmaul Resp. Oriented, Sleepy but arousable.
Severe	<10	< 7.15	Kussmaul Or Depressed Resp./Sleep/ alter Mental>Coma.

Differential Diagnosis
Anion Gap Acidosis

- Alcoholic ketoacidosis
- Lactic acidosis
- Renal failure
- Ethylene glycol or methyl alcohol poisoning
- Metabolic acidosis

Treatment of DKA
Fluids

- Initial resuscitation
  - dehydration is most commonly in the order of 8.5%
  - initial hydrating fluid should be isotonic saline,or ringer
    - this alone will often slightly lower the blood glucose
    - rarely is more than 20 cc/kg fluid required to restore hemodynamics

Fluid Replacement Calculations
(CONTINUES)

3. Calculate patient's maintenance fluids (requirements); Wt. base OR per SA(m2)

- Wt base: 100 ml/kg for the first 10 kg
  50 ml/kg for the next 10 kg
  20 ml/kg for the rest…. kg.

Example: Pt. is 22 kg. **Maintenance** is **1540 mL**

Fluid Replacement Calculations
(*CONTINUES*)

4. Calculate deficit (pre-illness wt)

   Example:

   - Pt. current (dehydrated) wt is 22 kg
   - Pt. is assess to be 8.5% dehydrated.

     (22 kg is >>> =85 x 22)
   - **Deficit will** be = **1870 cc**

Treatment of DKA
Electrolytes

- Phosphate deficit
  - May want to use Kphos
  - Phosphate is depleted as well. Phosphate may be added as KPO_4 especially if serum chloride becomes elevated

BICARBONATE IS ALMOST NEVER ADMINISTERED

bicarbonate administration leads to increased cerebral acidosis

HCO_3^- combines with H^+ and dissociated to CO_2 and H_2O. Whereas bicarbonate passes the blood-brain barrier slowly, CO_2 diffuses freely, thereby exacerbating cerebral acidosis and cerebral depression

Bicarbonate not given unless pH <6.9 or PH<7 whit hemodynamic unstability, cardiorespiratory compromise

- "**Pseudohyponatremia**" is often present
  - Expect that the Na^+ level will rise during treatment
  - Corrected Na^+ = Measured Na^+ + 0.016(measured glucose - 100)
  - If Na^+ does *not* rise**, true hyponatremia** may be present (possibly increasing cerebral edema risk) and should be treated
  - You can Use NS for the first 1-2 h and 1/2 NS thearafter
  - If **hypernatremia>145 meq/l** develops, 1/4 NS can be used

Hyperkalemia ;

initially present,resolves quickly with insulin drip

- So serum K^+ is often elevated, though total body K^+ is depleted
- K^+ is started early as resolution of acidosis and the administration of insulin will cause a decrease in serum K

-Once urine output is present and K<5.0, add 20-40 meq KCL per liter

Treatment of DKA
Insulin Therapy

- infusion of 0.1 units/kg/h regular insulin
- Do not give an initial insulin bolus

- Glucose levels
  - Decrease 75-100 mg/dl hour
  - Minimize rapid fluid shifts
  - If acidosis resolved (ph>7.35) , you can decrease rate to 0.05 u/kg/h

Glucose Administration

Prevents rapid decline in plasma osmolality

Rapid decrease in glucose could lead to cerebral edema

- Glucose decreases before ketone levels decrease
- Start glucose when plasma glucose <250 mg/dl

Complications of DKA

- Infection
  - Precipitates DKA
  - Fever
  - Leukocytosis can be secondary to acidosis
- Shock
  - If not improving with fluids r/o MI
- Vascular thrombosis
  - Severe dehydration
  - Cerebral vessels
  - Occurs hours to days after DKA
- Pulmonary Edema
  - Result of aggressive fluid resuscitation

- Cerebral Edema
  - First 24 hours
  - Mental status changes
  - Tx: Mannitol
  - May require intubation with hyperventilation

FOLLOW - UP

1. Cardio-respiratory monitoring and Neuro checks
   - Neuro checks: observe for changes of metal status as signs of dehydration and or complications of DKA: Cerebral edema, strokes
   - Respiratory: Observe for changes/ type of respiration as sign of acidosis (Kussmaul respirations) and /or respiratory depression 2nd to CNS depression as an imminent CNS complication.

Observations

pulse and BP hourly

•notify if pulse <10/min or Diastolic BP>90 mmHg

neuro obs. hourly if drowsy or reduced conscious level

•notify if any deterioration in conscious level or increase in pupil size

capillary blood glucose hourly

fluid balance chart with hourly urine

•Ask the patient to pass urine every hour initially

• if has not passed urine after 2 hours catheterize.

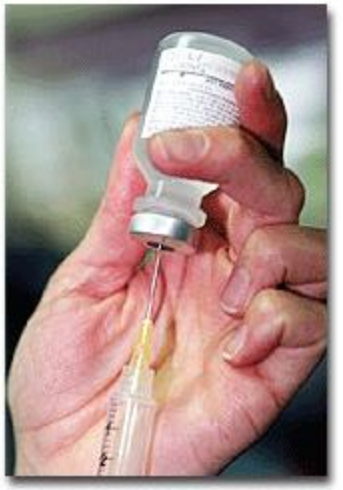
Withdraw catheter if good urine output after one hour

Once DKA Resolved
Treatment

- Patients 0-5 y require 0.6-0.7 units/kg/day
- Patients 5-12y require 0.8-1.0 units/kg/day
- Patients 12-18y require 1-1.2 units/kg/day

  – 2/3 daily requirement at morning
    (2/3NPH, 1/3 regular)

  – 1/3- given at night
  – (1/2 NPH,1/2 Regular)



- Give insulin SC ,at least 30 minutes prior to weaning insulin infusion.

Insulin

Type	Starts	Peaks	Duration
Humalog NovoRapid	5-10 min	0.5-1hrs	3.5 hrs
Regular	30 min	2-4 hrs	6-8 hrs
NPH Lente	1-2 hrs	6-10 hrs	16-24 hrs
Ultralente	4-6 hrs	8-24 hrs	24-36 hrs
Glargine	1.5h	None	Up to 24 hrs

Hyperosmolar Nonketotic Syndrome
Presentation

- Glucose >800 mg/dl
- Sodium
  - Normal, elevated or low
- Potassium
  - Normal or elevated
- Bicarbonate >15 mEq/L
- Osmolality >350 mOsm/L

Hyperosmolar Nonketotic Syndrome

- Extreme hyperglycemia and hyperosmolarity
- High mortality (12-46%)
- At risk
  - Older patients with intercurrent illness
  - Impaired ability to ingest fluids
- Urine volume falls
  - Decreased glucose excretion
- Elevated glucose causes CNS dysfunction and fluid intake impaired
- No ketones
  - Some insulin may be present
  - Extreme hyperglycemia inhibits lipolysis

Hyperosmolar Nonketotic Syndrome
Presentation

- Extreme dehydration
- Supine or orthostatic hypotension
- Confusion ⟶ coma
- Neurological findings
  - Seizures
  - Transient hemiparesis
  - Hyperreflexia
  - Generalized areflexia

Hyperosmolar Nonketotic Syndrome
Treatment

- Fluid replete
  - Replete ½ in first 12 hours
- Insulin
  - Make sure perfusion is adequ
  - Insulin drip 0.05U/kg/hr
- Treat underlying precipitating illness

Clinical Errors

- Fluid shift and shock
  - Giving insulin without sufficient fluids
  - Using hypertonic glucose solutions
- Hyperkalemia
  - Premature potassium administration before insulin has begun to act
- Hypokalemia
  - Failure to administer potassium once levels falling
- Recurrent ketoacidosis
  - Premature discontinuation of insulin and fluids when ketones still present
- Hypoglycemia
  - Insufficient glucose administration

Conclusion

- Successful management requires
  - correct use of fluids
    - Establish good perfusion
  - Insulin drip
    - Steady decline
    - Complete resolution of ketosis
  - Electrolyte replacement
  - Frequent neurological evaluations
  - High suspicion for complications
- Determine etiology to avoid recurrent episodes