Type 1 Diabetes Mellitus with Diabetic Ketoacidosis

By: Laura Valentine

Meet Susan Cheng

- 16 yrs old
- Type 1 DM
- Height: 5’3”
- Weight: 110 lbs
- BMI: 19.5
- Family history of diabetes
- Chief complaint: confusion, nausea, vomiting, fatigue, difficulty breathing & intense thirst

Nutrition HX

- 2,800 kcal diet
- Appetite is good, but has lost 5 lbs in 2 weeks
- %UBW = 110/115 x 100 = 95.65

Insulin Medications & Regimen

Split mixed insulin doses: Conventional Therapy

Morning (AM): 10 u NPH & 5 regular
Bedtime (HS): 4 u NPH & 4 u regular in PM

*No insulin during the middle of the day

Insulin Type	Brand Name	Onset of Action	Peak of Action	Duration of Action	Comments
Regular	Humulin or Novolin R	30 – 60 min.	2 – 4 2.5 – 5	5 – 8	Can be mixed w/longer-acting insulin
NPH	Humulin N or Novolin N	1 – 3 hours	8	20	Usually given in 2 daily doses

Intensive vs. Conventional Insulin Therapy

Conventional therapies are short- or rapid-acting insulin mixed with intermediate-acting insulins given before breakfast and before evening meal.

Intensive insulin therapy (MDI's – Multiple Daily Injections): intermediate insulins given once or twice daily and rapid- or short-acting insulin is given prior to meals

It allows more flexibility in the type of meals individuals are able to eat and the timing of meals. The amount of rapid- or short-acting insulin can be adjusted based on meal composition and/or its carbohydrate content.

Emergency Room

Physical Exam

- Urine tests
  - Glycosuria
  - Ketonuria
  - Blood glucose 400 mg/dL
- HEENT
  - Heart: tachycardia
  - Eyes: sunken
  - Ears: membranes dry
  - Nose: dry mucous membranes
  - Throat: N/A
- Blood pressure
  - 70/100 mm Hg = Stage 2 HTN

Neurologic

- Neurologic
  - Irritable, lethargic
- Skin
  - Dry, flushed skin, poor turgor
- Chest/Lungs
  - Deep, rapid Kussmaul's respirations
- Abdomen
  - Tender with guarding, decreased bowel sounds

***breath smelled like acetone

Diabetic Ketoacidosis (DKA)

DKA is a severe form of hyperglycemia. DKA results from dehydration during a state of relative insulin deficiency, associated with high blood levels of sugar level and organic acids called ketones.

Common causes include:

- Inadequate insulin due to missed insulin treatment, inadequate insulin dosing, and/or poor compliance
- Increased insulin needs with growth spurts
- Inadequately stored insulin
- Illness and/or infection (This can cause your body to produce certain hormones, such as adrenaline, which work against insulin to trigger an episode of diabetic ketoacidosis. Ex: pneumonia, urinary tract infections, etc…)
- Emotional stress
- High fever
- Surgery
- Stroke
- Alcohol or drug abuse
- Dehydration

Approximately 5% to 10% of cases have no identifiable cause.

Signs/Symptoms of DKA

- Nausea/vomiting
- Confusion/mental status change
- Kussmaul breathing
- Stomach pain
- Fruity or acetone smelling breath

Acute cerebral edema, a complication in about 1% of DKA patients, occurs primarily in children and less often in adolescents and young adults. Headache and fluctuating level of consciousness indicate this complication in some patients, but respiratory arrest is the initial manifestation in others. The cause is not well understood but may be related to too-rapid reductions in serum osmolality or to brain ischemia (lack of blood to the brain).

- Since type 1 diabetes typically starts before age 25 years, diabetic ketoacidosis is most common in this age group, but it may occur at any age.
- Males and females are equally affected.

Mechanism

lack of insulin→ liver produces excess glucose → excess glucose spilling into urine → dehydration → breakdown of fats for fuel → fatty acids are converted to ketones → excess ketones are excreted in urine → body consumes its own muscle, fat, and liver cells for fuel → weight loss

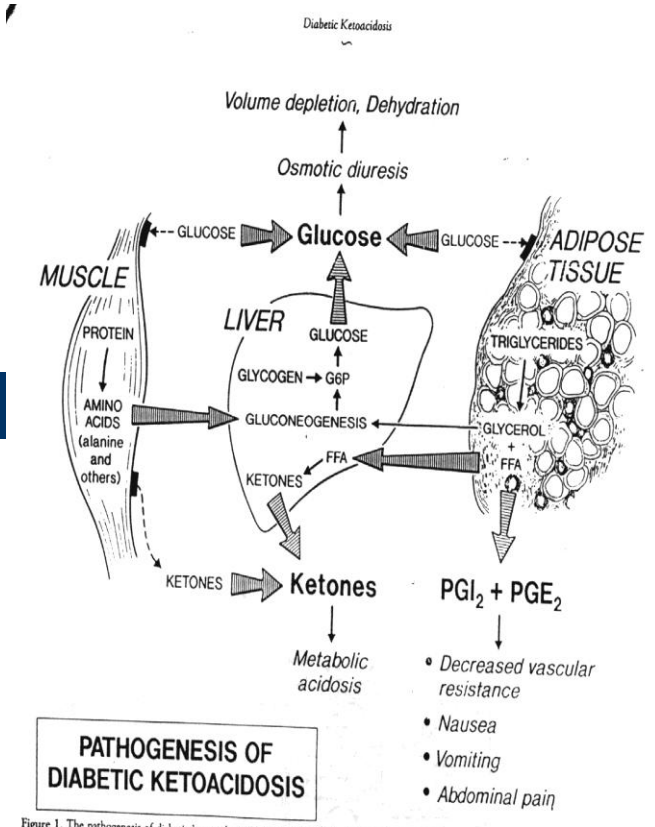


Figure 1. The pathogenesis of diabetic ketoacidosis. Severe insulin deficiency causes hyperglycemia, ketosis, and increased production of PGI_2 and PGE_2. Hyperglycemia is due to increased gluconeogenesis from amino acids, glycerol, and lactate and to decreased peripheral utilization of glucose. Ketosis is due to increased triglyceride lipolysis and increased FFA release from adipose tissue, to preferential utilization of FFAs for ketogenesis in the liver, and to deceased peripheral utilization of ketones. Increased PGI_2 and PGE_2 production by adipose tissue is due to accelerated triglyceride lipolysis and enhanced production of PGI_2 and PGE_2 in adipose tissue. Hyperglycemia causes an osmotic diuresis, volume depletion, hypotension, and dehydration. Ketosis causes an anion gap metabolic acidosis due to the dissociation of the ketoacids in the circulation and/or a hyperchloremic metabolic acidosis due to the loss of potential bicarbonate in the urine in the form of ketone bodies and the retention of chloride. Increased production of PGI_2 and PGE_2 causes decreased peripheral vascular resistance, hypotension, tachycardia, nausea, vomiting, and abdominal pain. Black rectangles denote impaired peripheral utilization of glucose and ketones, as indicated.

Abnormal Lab Values

Chemistry

	Normal	Admit	Day 2	Indication
Potassium	3.5 – 5.5	5. 8 (high)	5.1	Lack of insulin causes lack of distribution of potassium to cells and accumulation in blood
Chloride	98 – 108	110 (high)	102	
PO4	2.5 – 4.5	4.9 (high)	4.0	
Osmolality	275 – 295	336 (high)	298 (high)*	Dehydration → electrolyte imbalance
Total CO2	24 – 30	22 (low)	24	Respiratory compensation for metabolic acidosis. A forced increased respiration (blowing off the carbon dioxide).
Glucose	70 – 120	475 (high)	200 (high)*	Lack of insulin not able to rid blood of glucose
BUN	8 – 26	29 (high)	21	Decreased kidney function causing high levels of waste in blood.
Creatinine	0.6 – 1.3	1.8 (high)	1.2	Decreased kidney function
HbA1C	4.8 – 7.8	12.0 (high)		Large amount of glucose is attaching to the hemoglobin → degree of hyperglycemia
Cholesterol	140 – 199	201 (high)	200 (high)*	

Abnormal Lab Value

Arterial Blood Gases (ABG's)

pH	7.35 - 7.45	7.31 (low)	Excess ketones in the blood cause it to become acidic. Acidic blood causes acetone smelling breath.
CO2 content	23 – 30	22 (low)	The body initially buffers metabolic acidosis with the bicarbonate buffering system, but this is quickly overwhelmed and other mechanisms are used to compensate for the acidosis, such as hyperventilation to lower the blood carbon dioxide levels. This type of hyperventilation is observed through her Kussmaul Respirations.
HCO3-	24 – 28	21 (low)	A low bicarbonate level is not able to buffer the metabolic acidosis that is occurring because of the excess ketones she is excreting in the urine. This low buffering system is quickly overwhelmed and other mechanisms compensate for the acidosis, such as hyperventilation to lower the blood carbon dioxide levels.

Abnormal Lab Values

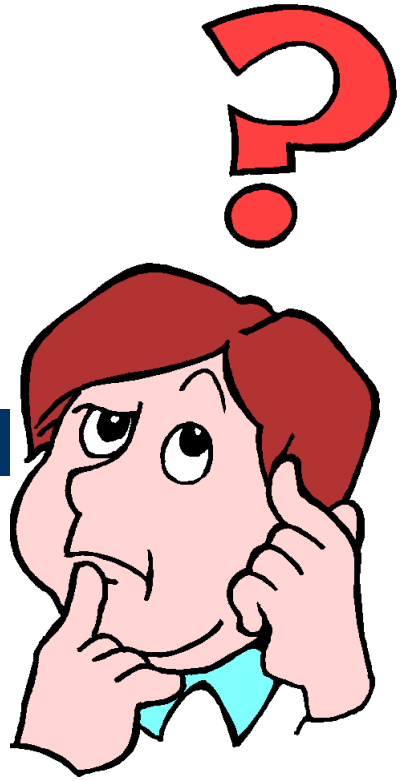
Urine Analysis

Lab	Admit	Indication
Glucose	pos	Inadequate insulin causes excess glucose
Ketones	pos	Break down of fat (lipolysis) causing excretion of ketones into urine

Blood Glucose Monitoring Record

Day/Date	Breakfast BG	Lunch BG	Dinner BG	Bedtime BG	Comments
9/20	115			130	Period started
9/21	140	135	120	130	Period
9/22	135	150	170	185	Period
9/23	200	170	150	200	Period
9/24	200	220	230	300	Volleyball tourney
9/25	250	250	275	280	My birthday!

What's causing Susan's DKA?



- Adherence to insulin regimen
- No other precipitating factors

- "Potentially, there may be a significant change in glucose metabolism that may occasionally be observed during the late luteal and decidual phases of the menstrual cycle."
- "Through unclear mechanisms, some women with diabetes mellitus demonstrate significant changes in glucose control around the time of their menses, including DKA. Accordingly, we propose that the terms catamenial DKA and catamenial hyperglycemia be used to refer to these disorders and that catamenial DKA be included in the differential diagnosis list of causes or precipitating events that can lead to DKA."

Treatment for DKA

- IV fluids and electrolytes to maintain hydration
  - Replace with NS initially (watch for CHF and fluid overloading)
  - As blood glucose drops, supplement with D5W or D10W
- Insulin regulation
- Regular blood glucose monitoring (test for ketones if glucose > 250 mg/dL)

Tx Plan: Insulin pump therapy combined with CHO counting

- 1 L 0.9 % NS w/10 u regular insulin IV over first 30 minutes
- Reduce rate to 1 L/hr 0.9% NS with 10 u regular insulin (per L)
- Measure glucose, acetone, electrolytes, and ABG's every hour
- At blood glucose 250 mg/dL; decrease insulin to 5 u in 5% dextrose and 0.4% NS; continue infusion until plasma cleared of ketones
- Maintain blood glucose at 250 mg/dL and continue dextrose/NS infusion until acidosis corrected

  Sliding scale for glucometer:

  BG > 400 Regular insulin SQ – 20 units
  BG > 300 Regular insulin SQ – 15 units
  BG > 200 Regular insulin SQ – 10 units
- When patient stable, begin sliding scale regular insulin SQ q 4 hr
- 300 mg Tagamet IV piggyback q 8 hr
- Liquids when PO fluids tolerated
- When stable, evaluate for insulin pump
- Diabetes education consult

Continuous Subcutaneous Insulin Infusion (CSII)

- A form of intensive therapy.
- Basal rapid- or short-acting insulin is pumped continuously in micro-amounts through a subcutaneous catheter and is received 24 hours a day
- Boluses of rapid- or short-acting insulin is given before meals

CHO Counting

Three Levels of Carbohydrate Counting

Level 1: Basic Carbohydrate Counting Skills

- Knowing carbohydrate sources, how to count grams of carbohydrate in foods, understanding the relationship between portion size and carbohydrate content, recording your usual carbohydrate intake and sharing it with an RD, and determining target amounts of carbohydrates for meals and snacks determined

Level 2: Intermediate Carbohydrate Counting Skills

- Pattern management: Identify blood glucose patterns impacted by food, insulin, and PA
- Identify and interpret patterns to make adjustments in diabetes regimens
- Insulin doses adjusted when deviations from usual carbohydrate content are made
- For every 15-20 g CHO added or subtracted from a meal, 1-2 units rapid- or short-acting insulin suggested.

Level 3: Advanced Carbohydrate Counting Skills

- Insulin adjusted on basis of ratio of grams of carbohydrate intake to doses of rapid or short-acting insulin
- Calculation of carbohydrate-to-insulin ratios
- Grams of CHO eaten at a meal divided by number of units of rapid- or short-acting insulin necessary to meet blood glucose goals.
- Large amounts of meat and/or fat at a meal may require adjustment of insulin administration after the meal instead of before the meal
- Grams of fiber may be subtracted from total carbohydrate content of a food if it contains >5 g fiber per serving, since fiber is not considered an available source

Individuals with intensive insulin therapy: Level 3 of CHO counting

Food Group	Carb Grams	Food Group	Carb Grams
Bread/Starch	15	Vegetable	5
Fruit	15	Meat	0
Milk	12	Fat	0

Grams of CHO eaten at a meal divided by number of units of rapid- or short-acting insulin necessary to meet blood glucose goals

Ratio of 1 U insulin to 9 g CHO or 2 U insulin for every I carbohydrate choice

- EX: 45 g CHO (3 CHO choices) at a meal and requires 5 units insulin

Grams of fiber may be subtracted from total carbohydrate content of a food if it contains >5 g fiber per serving (since fiber is not considered an available source of glucose)

- Ex: 22 g CHO w/5 g fiber = ~ 17 g CHO = ~1 exchange

ADIME

Diagnosis

- PES: Weight loss related to diabetic ketoacidosis as evidenced by BMI and UBW.

Monitoring/Evaluation

- Monitor blood glucose
  - Self-Monitoring of Blood Glucose (SMBG): collect detailed info about blood glucose at many time points to enable maintenance of a more constant glucose level by more precise regimens
- Diabetes education consult
- Evaluate adherence to 3,000 kcal diet order
  - Patient will be able to correctly identify carbohydrate content in a variety of regularly consumed foods
- Insulin pump education
  - Patient will be able to state carbohydrate goals with appropriate insulin coverage

What if Susan's symptoms were left untreated?

- Micro and macrovascular complications
  - Nephropathy (any disease of the kidney)
  - Retinopathy (disease of the retina)
  - Neuropathy (nervous system disorder)
- Hyperglycemic hyperosmolar
  - associated with polyuria (frequent urination)
  - polydipsia (excessive thirst)
  - polyphagia (excessing eating)
  - weight loss
- Death

Questions?