DIABETIC KETOACIDOSIS (DKA)

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DIABETIC KETOACIDOSIS (DKA)

- A state of absolute or relative insulin deficiency , which causes hyperglycemia, dehydration, ketone body production that leads to metabolic acidosis.
- Can occur in **both Type I Diabetes and Type II Diabetes**
  - In type II diabetics with insulin deficiency/dependence
- The presenting symptom for ~ 25% of Type I Diabetics.

FUNCTIONS OF INSULIN

1. Transports and metabolizes glucose for energy

2.Stimulates storage of glucose in the liver and muscle as glycogen.

3. Signals the liver to stop the release of glucose

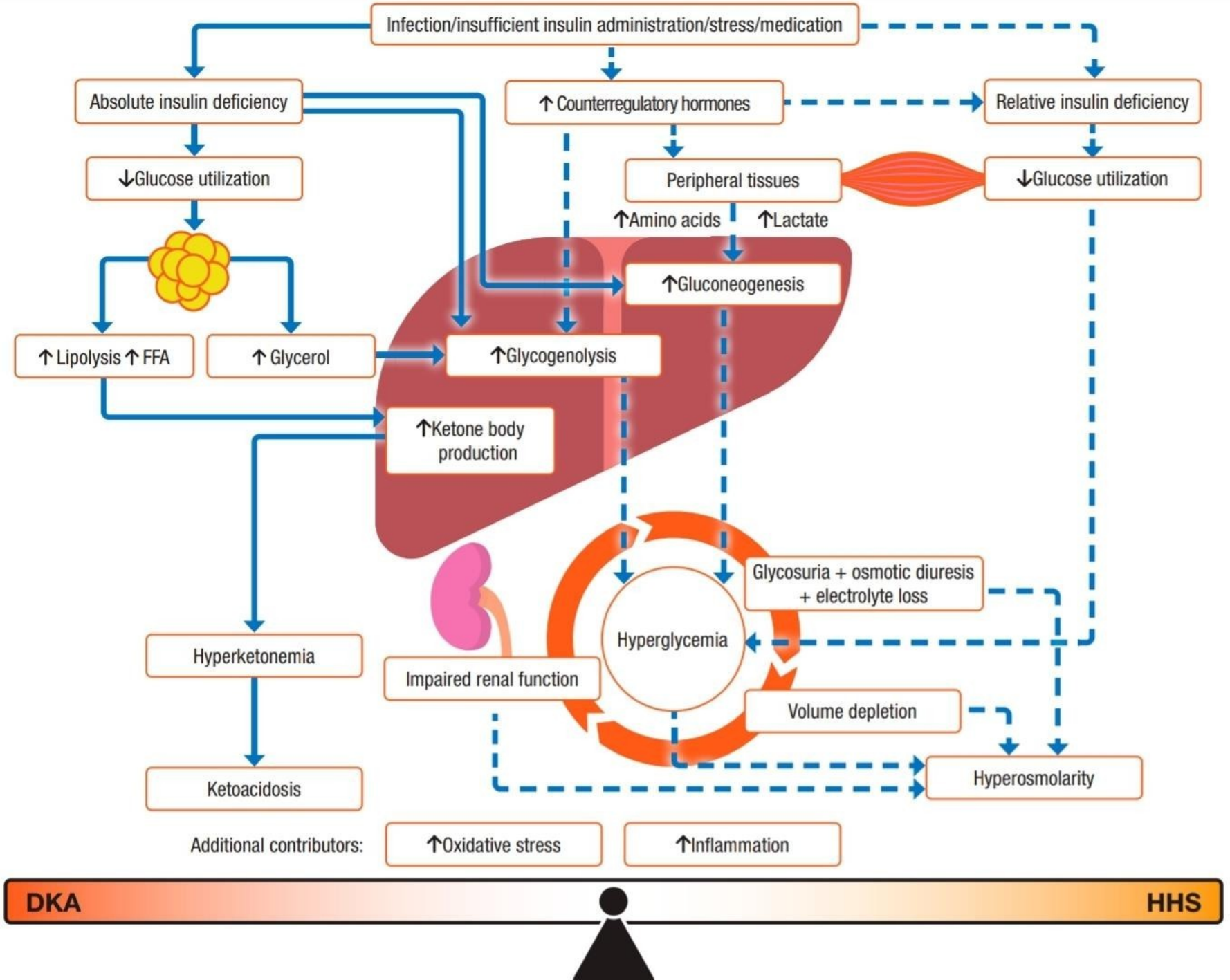
4. Enhances storage of fat in adipose tissue

5. Accelerates transport of amino acids into cells

6.Inhibits the breakdown of stored glucose, protein, and fat

PATHOPHYSIOLOGY

- DKA results from relative or absolute insulin deficiency combined with counterregulatory hormone excess (glucagon, catecholamines, cortisol, and growth hormone)
- Both insulin deficiency and glucagon excess, in particular, are necessary for DKA to develop.
- The decreased ratio of insulin to glucagon promotes gluconeogenesis, glycogenolysis, and ketone body formation in the liver, as well as increases in substrate delivery from fat and muscle (free fatty acids, amino acids) to the liver.


Infection/insufficient insulin administration/stress/medication
Absolute insulin deficiency
↑ Counterregulatory hormones
Relative insulin deficiency
↓Glucose utilization
Peripheral tissues
↓Glucose utilization
↑Amino acids
↑Lactate
↑Gluconeogenesis
↑ Lipolysis ↑ FFA
↑ Glycerol
↑Glycogenolysis
↑Ketone body production
Glycosuria + osmotic diuresis + electrolyte loss
Hyperglycemia
Hyperketonemia
Impaired renal function
Volume depletion
Hyperosmolarity
Ketoacidosis
Additional contributors:
↑Oxidative stress
↑Inflammation
DKA
HHS

RISK FACTORS

- Any Stressful precipitating event results in increased catecholamines, cortisol, glucagon.
  - Infection (pneumonia, UTI)
  - Non-compliance with insulin
  - Alcohol, drugs (COCAINE)
  - Stroke
  - Myocardial Infarction
  - Pancreatitis
  - Trauma
  - Medications (steroids, thiazide diuretics)
  - Pregnancy

CLINICAL

MANIFESTATION

Symptoms of DKA

- Polyuria
- Polydypsia
- Blurred vision
- Nausea/Vomiting
- Abdominal Pain
- Fatigue
- Confusion
- Obtundation

Signs in DKA.

- Hypotension, tachycardia
- Kussmaul breathing (deep, labored breaths)
- Fruity odor to breath (due to acetone)
- Dry mucus membranes
- Confusion
- Abdominal tenderness

DIAGNOSTIC INVESTIGATION

Classic Triad of DKA

The biochemical criteria for the diagnosis of DKA[3,4]

- **Hyperglycemia - blood glucose greater than 11.1 mmol/L**
- **Ketosis - ketones present in blood and/or urine**
- **Acidosis - pH less than 7.3 and/or bicarbonate less than 15 mmol/L**

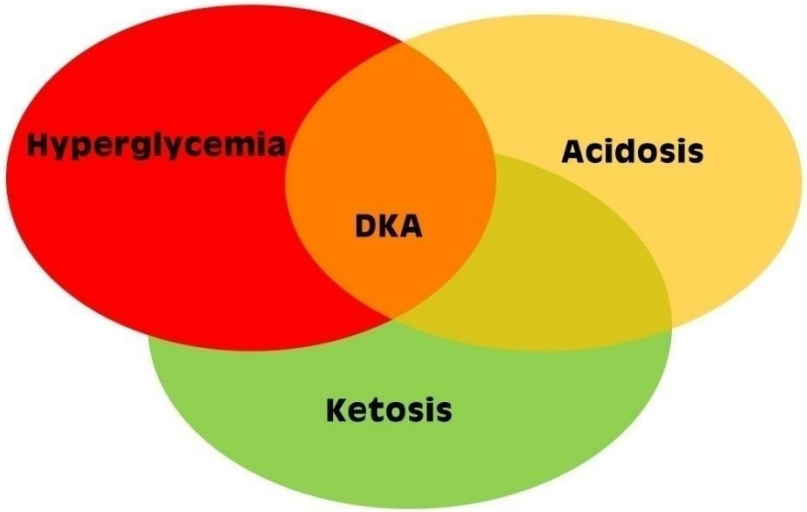


Table 2–DKA classification and suggested level of care by severity: mild, moderate, or severe

	Mild DKA	Moderate DKA	Severe DKA
"D": history of diabetes or elevated glucose level	Glucose ≥200 mg/dL (11.1 mmol/L)	Glucose ≥200 mg/dL (11.1 mmol/L)	Glucose ≥200 mg/dL (11.1 mmol/L)
"K": ketonemia	β-Hydroxybutyrate 3.0–6.0 mmol/L	β-Hydroxybutyrate 3.0–6.0 mmol/L	β-Hydroxybutyrate >6.0 mmol/L
"A": acidosis	• pH >7.25 to <7.30 or bicarbonate 15–18 mmol/L	• pH 7.0–7.25 • Bicarbonate 10 to <15 mmol/L	• pH <7.0 • Bicarbonate <10 mmol/L
Mental status	Alert	Alert/drowsy	Stupor/coma
Suggested level of care	Regular or observation nursing unit	Step-down unit or intermediate care unit	Intensive care unit

Not all variables need to be fulfilled to be defined as either mild, moderate, or severe, and the admission site and level of care are ultimately a clinical decision.

CASE STUDY ON DKA

Patient Profile

Name: Mr. Samuel R., 28 years old
Sex: Male
Medical History: Type 1 Diabetes Mellitus diagnosed at age 15

Home Medications:
Insulin glargine (Lantus) 20 units nightly, Insulin lispro with meals

Social History:
Works long shifts, recently had a flu-like illness, poor appetite for 2 days

Trigger Event:
Stopped taking mealtime insulin due to nausea and decreased oral intake

Presentation to the Emergency Department

Chief Complaints

- ✓ Increasing fatigue
- ✓ Nausea and vomiting for 24 hours
- ✓ Excessive thirst and frequent urination
- ✓ Rapid, deep respirations
- ✓ Confusion and headache

Nursing Assessment Findings

General Appearance: Pale, dehydrated, drowsy

Airway: Patent, breathing rapidly

Breathing: Kussmaul respirations (deep, rapid)

Circulation:

- ❖ HR: 122 bpm
- ❖ BP: 96/58 mmHg
- ❖ RR: 30/min
- ❖ SpO_2: 98% on room air
- ❖ Temp: 37.8°C

Skin: Dry, poor turgor

Breath: Fruity odor

Neurological: Oriented to person only, slow responses

Laboratory Results

Test	Result	Normal Range	Interpretation
Blood glucose	520 mg/dL	70–110	Severe hyperglycemia
Arterial pH	7.12	7.35–7.45	Metabolic acidosis
HCO_3	10 mEq/L	22–26	Low bicarbonate
Serum ketones	Large	Negative	Ketosis
Anion gap	28	8–16	High anion gap acidosis
Serum Na	130 mEq/L	135–145	Low (dilutional)
Serum K	5.8 mEq/L	3.5–5.0	Elevated due to acidosis
Urinalysis	Glucose +++ and ketones +++		Confirms DKA

Nursing Priorities in DKA

Priority #1: Restore Fluid Volume

- Start **0.9% normal saline** bolus to correct dehydration and improve perfusion.
- Monitor **urine output** (goal ≥ 0.5 mL/kg/hr).

Priority #2: Correct Hyperglycemia & Stop Ketone Production

- Start **regular insulin IV infusion** (not subcutaneous).
- Monitor glucose hourly.
- Transition to **D5½NS** when glucose reaches 200–250 mg/dL to prevent cerebral edema.

Priority #3: Correct Electrolyte Imbalances

Potassium replacement is critical.

- Although initial K^+ is high, insulin will drive it back into cells → risk for **hypokalemia**. Begin potassium replacement when $K^+ <$ 5.0 and patient has adequate urine output.

Priority #4: Manage Acidosis

- Insulin infusion will close the anion gap and correct acidosis.
- Bicarbonate therapy only if **pH < 6.9** (rare).

Priority #5: Identify and Treat the Trigger

- In this case, likely illness and missed insulin doses.
- Obtain blood cultures, assess for infection, and administer antibiotics if needed.

Nursing Interventions (Detailed)

A. Fluid Management

Initial:

- 1–1.5 L NS over 1 hour
- Continuous VS monitoring: BP, HR, respiratory pattern
- Daily weight and strict I&O

B. Insulin Therapy

- Start insulin drip per protocol (e.g., 0.1 units/kg/hr).
- Do NOT start insulin if potassium <3.3 mEq/L.
- Check blood glucose **hourly**.

C. Potassium & Electrolyte Replacement

- Monitor serum potassium every 2–4 hours.
- Add KCl to IV fluids when potassium normalizes or drops.

D. Monitoring for Complications

Cerebral edema (headache, decreased LOC, bradycardia)

Hypoglycemia

Cardiac arrhythmias due to potassium shifts

E. Patient & Family Education

- Reinforce importance of insulin during illness ("sick-day rules").
- Encourage frequent glucose and ketone checks.
- Teach early signs of DKA

OUTCOME / RESOLUTION

- After 12 hours of treatment:
- Glucose: 185 mg/dL
- pH: 7.32
- HCO_3: 18 mEq/L
- Anion gap closed
- Patient awake, alert, no respiratory distress
- Transitioned to subcutaneous insulin once stable


THANK YOU