DIABETIC KETOACIDOSIS (DKA)

- Diabetic ketoacidosis (DKA) is a **life-threatening** complication of **diabetes mellitus**, characterized by **hyperglycemia, metabolic acidosis, and ketonemia**.
- It primarily occurs in **type 1 diabetes mellitus (T1DM)** but can also be seen in type 2 diabetes (especially in cases of severe insulin deficiency).

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1. PATHOPHYSIOLOGY

The Core Problem: Absolute or Relative Insulin Deficiency

Insulin is essential for glucose uptake into cells. Without insulin, the body cannot use glucose for energy, leading to:

A. Hyperglycemia (High Blood Sugar)

- Without insulin, glucose remains in the blood → Hyperglycemia (>250 mg/dL)
- Kidneys try to remove excess glucose → Glycosuria (glucose in urine)
- Water follows glucose → Polyuria (excess urination) → Dehydration
- Leads to polydipsia (excess thirst) and hypovolemia (low blood volume)

B. Ketogenesis (Fat Breakdown → Ketone Production)

- Without insulin, the body breaks down fat for energy → Lipolysis
- Fatty acids are converted to ketones (β-hydroxybutyrate & acetoacetate) in the liver
- Ketones are acidic → Metabolic Acidosis (pH <7.3, low bicarbonate)

C. Electrolyte Imbalances

- Hyperkalemia (high K^+ in blood initially) due to acidemia (H^+ moves into cells, K^+ moves out)
- Total body potassium is low due to urinary losses
- Low sodium due to osmotic diuresis
- Phosphate depletion from cellular starvation

📌 **Summary:** DKA occurs due to insulin deficiency, leading to **hyperglycemia, dehydration, acidosis, and electrolyte imbalances**.

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2. RISK FACTORS FOR DKA

- Missed insulin doses (non-adherence, pump failure)
- New-onset diabetes (especially type 1 DM)
- Infections (pneumonia, UTI, sepsis → increased insulin demand)
- Stress conditions (surgery, trauma, myocardial infarction)
- Pancreatitis
- Medications (steroids, SGLT2 inhibitors in type 2 DM, diuretics)
- Alcohol, drug use (cocaine, methamphetamine → counteract insulin action)

3. CLINICAL FEATURES OF DKA

Symptoms (What the patient feels)	Signs (What you observe)
Polyuria, polydipsia (frequent urination, thirst)	Dehydration (dry skin, low BP, tachycardia)
Nausea, vomiting, abdominal pain (due to acidosis)	Fruity (acetone) breath
Weakness, fatigue	Kussmaul respiration (deep, labored breathing to compensate for acidosis)
Altered mental status (confusion, drowsiness, coma in severe cases)	Hypotension, tachycardia (due to fluid loss and shock)

📌 **Severe DKA = Altered mental status, severe dehydration, shock, or coma**

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4. COMPLICATIONS OF DKA

- Hypokalemia (during treatment if not replaced properly) → Can cause arrhythmias
- Cerebral edema (most feared complication, especially in children)
- Acute kidney injury (due to dehydration and shock)
- Severe metabolic acidosis → Respiratory failure
- Thrombosis (hypercoagulable state in DKA)

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5. INVESTIGATIONS & FINDINGS IN DKA

Test	Findings in DKA	Explanation
Blood Glucose	**>250 mg/dL (often 300-600 mg/dL)**	High due to insulin deficiency
Arterial pH	**<7.3 (Acidosis)**	Ketone accumulation
Serum Bicarbonate (HCO_3^-)	**<18 mEq/L**	Metabolic acidosis
Serum Ketones (β-hydroxybutyrate)	**Positive**	Marker of ketone production
Anion Gap	**>12 (high anion gap metabolic acidosis)**	Excess unmeasured acids
Serum Potassium (K^+)	**Normal or high initially, but total body K^+ is low**	K^+ shifts out of cells in acidosis
Urine Ketones	**Positive**	Confirms ketosis
Serum Sodium (Na^+)	**Low (due to osmotic dilution)**	Pseudohyponatremia
Serum Osmolality	**High (>290 mOsm/kg)**	Dehydration

Blood Urea Nitrogen (BUN)/Creatinine	**Elevated**	Due to dehydration and renal impairment

📌 **Key Finding: Hyperglycemia + Ketones + Metabolic Acidosis (pH <7.3, HCO_3^- <18, high anion gap)**

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6. MANAGEMENT OF DKA (STEP-BY-STEP APPROACH)

Step 1: Fluid Resuscitation (Correct Dehydration)

- 1st choice: IV Normal Saline (0.9% NaCl)
    - 1L in the first hour (unless contraindicated by heart failure)
    - Then 500 mL/hr for next 4 hours
- Switch to 0.45% NaCl if Na^+ is high
- Once glucose reaches 200-250 mg/dL, switch to Dextrose 5% + 0.45% NaCl to prevent hypoglycemia

Step 2: Insulin Therapy (Stop Ketogenesis)

- Start IV Regular Insulin:
    - 0.1 U/kg IV bolus, then
    - 0.1 U/kg/hr infusion
- Goal: Lower glucose by 50-100 mg/dL per hour
- When glucose reaches ~200 mg/dL, reduce insulin to 0.05 U/kg/hr and add Dextrose 5%

Step 3: Potassium (K^+) Replacement (Prevent Arrhythmias)

- Before giving insulin, check K^+:
    - If K^+ <3.3 mEq/L → Hold insulin, give IV KCl first
    - If K^+ 3.3-5.0 mEq/L → Give 20-40 mEq KCl per liter of fluids
    - If K^+ >5.0 mEq/L → Do not give K^+, monitor ECG
- Monitor K^+ every 2-4 hours

Step 4: Correct Acidosis (Ketone Clearance)

- Acidosis resolves once ketones clear → Continue insulin until β-hydroxybutyrate normalizes
- Bicarbonate is rarely needed, only if pH <6.9

Step 5: Identify & Treat Underlying Cause

- Infection? Start antibiotics
- Missed insulin dose? Educate patient
- Myocardial infarction? Check troponins

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7. TRANSITION TO SUBCUTANEOUS INSULIN

- Once DKA resolves (glucose <200 mg/dL, anion gap normal, ketones clear):
    - Start basal insulin (e.g., glargine) 2-4 hours before stopping IV insulin
    - Give meal-time insulin (e.g., lispro) with food