# DIABETIC KETOACIDOSIS

DKA is a syndrome in which insulin deficiency & glucagon excess combine to produce a hyperglycaemic, dehydrated, acidotic patient with profound electrolyte imbalance.



# Important Pathophysiology Points.

- Renal threshold for glucose is surpassed (>10mmol/L)
- Glucose in renal tubules draws *water, sodium, potassium, magnesium, calcium & phosphate* into urine.
  - Marked deficits in these electrolytes may be hidden by dehydration and free water deficits.
- Long-chain FFAs are partially oxidised in the liver to acetoacetate & betahydroxybutyrate.

# **Diagnostic Strategies.**

History.

- Recent onset polydipsia, polyuria, polyphagia.
- Associated weakness, blurred vision, weight-loss, N&V and abdominal pain.
  - Abdominal pain is usually idiopathic.

Physical Examination.

- Tachypnoea (Kussmaul's breathing).
- Tachycardia, dehydration.
- Hypotension is a late sign.
- Evidence of precipitating illness (?AMI, sepsis etc).

Laboratory Tests.

• Initial testing will confirm the diagnosis:

Biochemical Criteria for DKA.

- Hyperglycaemia;
  - Blood glucose > 11mmol/L (200mg/dL)

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- Venous pH < 7.3 or  $HCO_3^-$  < 15mmol/L
- Ketonaemia / ketonuria.
  - · Serum glucose, FBC, EUC, CMP.
  - Blood gas = raised anion gap, metabolic acidosis.
  - ECG (particularly if no immediate K+ is available)
  - Urine.
  - Ketone levels (point of care, urine, serum).
  - Cultures.

A word on Sodium... Misleading in hyperglycaemia. Strongly affected by glucose, triglycerides & insensible losses... **Corrected Sodium:** = measured sodium + [glucose/3] *Correcting Potassium for Acidosis* can be corrected by subtracting 0.6 mmol/L from the measured K+ value for every 0.1 decrease in pH.... eg. serum K = 5mmol/L w/ pH 6.90 (ie. 0.5 less than 7.4) corrected K+ = 5mmol/L - [ 0.6 x 5 ] = 5 - 3 = 2mmol/L.

## **Differential Considerations.**

- Alcoholic vs starvation ketoacidosis.
  - Alcohol > 20% all ketoacidoses.
- · Can develop in 3rd trimester of pregnancy (or nursing mothers who do not eat).
- Recall your other causes of raised anion-gap metabolic acidosis (particularly the toxic alcohols & salicylates).
- Hyperglycaemia without ketones = consider HHS (HONK).

# Management.

General Measures.

- The approach to the patient w/ severe DKA is the same as any patient in extremis.
- · ABCD approach.
- Obtunded & vomiting, requires airway protection.
- If you intubate; you must *match the patients minute ventilation* to avoid relative respiratory acidosis.
- Hypovolaemic shock requires aggressive fluid resuscitation.
- Identify & treat the precipitating illness.

### <u>Insulin.</u>

- DKA cannot be reversed without insulin.
- Low-dose therapy is preferable (no boluses !!)
  - 0.1 units/kg/hr via IV infusion.
- Dextrose is added to IV fluids once glucose levels reach 15mmol/L.
- Insulin infusion requires *priming of the tubing and burettes* to avoid insulin adhering to their walls.

### Dehydration.

- Patients may have fluid deficit of > 3-5L.
- Fluid boluses are acceptable if patient is in shocked state.
- Target:
  - 1st litre over 1st hour.
  - 2nd litre over 4 hours.
- Fluid therapy alone will reduce serum glucose levels (by ~ 18%).

## Potassium.

- Replacement is needed for all DKA patients.
- Levels will plummet with correction of acidosis and insulin administration.
- 20-40mmol/L in replacement fluid is req'd.

## Magnesium.

- Deficiency is a common problem in DKA.
- Deficiency promotes hypokalaemia.
- May need up to 0.3mmol/kg replacement.
  - 1-3grams in a 70kg patient.

#### Acidosis.

- · Acidosis will correct with fluids and insulin.
- Bicarbonate is rarely indicated & worsens intracellular acidosis.
- If bicarb is deemed necessary, pH should not be corrected beyond 7.1

#### BOX 124-7 SUMMARY OF TREATMENT FOR DIABETIC KETOACIDOSIS

Identify DKA: serum glucose, electrolytes, ketones, and ABG; also draw CBC with differential; urinalysis; chest radiograph film and ECG, if indicated.

- 1. Supplement insulin.  $\pm$  Bolus: 0.1 U/kg regular insulin IV Maintenance: 0.1 U/kg regular insulin IV Change IV solution to D<sub>5</sub>W 0.45% NS when glucose  $\leq$  300 mg/dL.
- 2. Rehydrate. 1–2 L NS IV over 1–3 hours Children: 20 mL/kg NS over first hour Follow with 0.45% NS
- 3. Correct electrolyte abnormalities. Sodium
  - Correct with administration of NS and 0.45% NS. Potassium
  - Ensure adequate renal function. Add 20–40 mEq KCl to each liter of fluid.
  - Phosphorus
  - Usually unnecessary to replenish
  - Magnesium

Correct with 1–2 g MgSO<sub>4</sub> (in first 2 L if magnesium is low).

- Correct acidosis.
  Add 44–88 mEq/L to first liter of IV fluids only if pH ≤ 7.0.
  - Correct to pH 7.1.
- 5. Search for and correct underlying precipitant.
- Monitor progress and keep meticulous flow sheets. Vital signs Fluid intake and urine output
  - Serum glucose,  $K^+$ ,  $Cl^-$ ,  $HCO_3^-$ ,  $CO_2$ , pH Amount of insulin administered
- 7. Admit to hospital or intensive care unit.
- Consider outpatient therapy in children with reliable caretaker and
  - Initial pH > 7.35
  - Initial  $HCO_3^- \ge 20 \text{ mEq/L}$
  - Can tolerate PO fluids
  - Resolution of symptoms after treatment in emergency department
  - No underlying precipitant requiring hospitalization

# **Complications of DKA.**

Morbidity from DKA is largely iatrogenic.

- Hypokalaemia
- Hypoglycaemia
- Alkalosis (aggressive bicarbonate therapy)
- CCF
- Cerebral oedema

Poor prognostic signs in DKA:

- Hypotension
- Azotaemia
- Coma
- Underlying illness.

Primary Causes of Death in DKA:

- Infection (especially pneumonia)
- Arterial thromboses
- Shock

# **Disposition.**

Most patients w/ DKA require hospital admission (and occasionally ICU).

Table 220-1 Important Causes of Diabetic Ketoacidosis
Omission or reduced daily insulin injections
Dislodgement/occlusion of insulin pump catheter
Infection
Pregnancy
Hyperthyroidism
Substance abuse (cocaine)
Medications: steroids, thiazides, antipsychotics, sympathomimetics
Heat-related illness
Cerebrovascular accident
GI hemorrhage
Myocardial infarction
Pulmonary embolism
Pancreatitis
Major trauma
Surgery