Huffin' and Puffin' Over Missed Insulin

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Dave's presentation

- Dave, 24, presents to the ED seeking insulin.
- He states that he has had diabetes mellitus Type 1 since childhood. He has not eaten since yesterday and took his last shot of insulin at 1 p.m.
- His friends, concerned about his worsening condition, called an ambulance at 8 p.m., which brought him to the ED.
- Dave is quite concerned about missing his insulin shot and repeatedly asks for it. He also complains of:
  - heavy breathing,
  - upper abdominal pain,
  - headache,
  - nausea,
  - vomiting and
  - increased urination.
- At this point, he is able to tell you that he has been hospitalized in the past for these kinds of episodes, but he is vague about the details.

Physical exam

- Dave is in obvious distress. He is breathing deeply and quickly, there is nasal flaring and accessory muscle use. A faint smell of acetone is present on his breath.
- He is unable to sit up and after giving the initial history, he is unable to focus on or answer any questions without repetitive questioning.
- Vitals are as follows:
  - Heart rate: 120 beats per minute
  - Respiration rate: 28 breaths per minute
  - Blood pressure: 126/73 mm Hg
  - Temperature: 36.5 C
  - Oxygen saturation: 98%
- His abdominal exam revealed slight tenderness to palpation, but is otherwise unremarkable.

For more on Dave see page 4.

Questions & Answers

1. What are the warning signs for DKA?

Several key factors will confirm a diagnosis of diabetic ketoacidosis (DKA):

- Does the patient have a history of diabetes Type I or II? If so, is the patient compliant with treatment? Have there been previous episodes of DKA?
- Check for precipitants of DKA. Is there evidence of infection, silent MI, stroke, mesenteric ischemia, pancreatitis or recently prescribed medications, including herbal remedies? (Note that two per cent to 10% of cases have no identified precipitating factor)
- Is the patient vomiting, nauseous, have polyuria or polydipsia?
- Is there hyperventilation, ketone breath, abdominal pain or dehydration?

2. What investigations should be done?

Once a diagnosis of DKA is made, investigations should include:

- A complete blood count, as well as electrolyte, creatinine, urea and blood glucose levels
- Calculate the anion gap (AG): Na – (Cl- + CO2). A value > 12 is abnormal
- Arterial blood gas workup
- Urine dip for ketones
- Serum ketones
- Chest X-ray, urine dip for infection or blood cultures, if there is evidence of infection. Note that DKA is associated with hypothermia, so normothermia or hyperthermia may indicate the presence of infection
- ECG monitoring effects of hypo- or hyperkalemia

3. Why do DKA patients get abdominal pain?

Abdominal pain, with nausea and vomiting, is common in DKA and is often mistaken as an acute surgical abdomen. It is likely due to the acidosis or decreased mesenteric perfusion, secondary to dehydration.

More on Dave

- Labs reveals a leukocyte count of 25.1 x 10^9 cells/L with metamyelocytes (toxic changes) present.
- Other lab results show that:
  - Glucose is 27.2 mmol/L
  - Potassium is 6.3 mEq/L
  - Anion gap is 33
  - Creatinine is 154 mg/dL
  - Urea is 12.3 mg/dL
- Arterial blood gases show pH 7.01, pCO2 9, HCO3 2. Ketonuria was present.

Dave's Treatment

- Three- to four-litre bolus of normal saline was given over two to three hours.
- When urine ketones and labs confirmed ketoacidosis, insulin was given intravenously at 0.15 units/kg bolus, followed by 0.1 units/ kg/hour. When glucose levels reached 12 mmol/L to 14 mmol/L, dextrose was administered in addition to normal saline and insulin infusion.
- Labs were rechecked every two hours. Urine input and output were monitored and Dave was put on a cardiac monitor due to hyperkalemia.
- When his labs demonstrated normokalemia, 20 mEq KCl was added to each litre of D5W/ 0.5 normal saline solution.

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4. What kinds of electrolyte abnormalities are present?

a) Hyponatremia: because of hyperosmolarity, water shifts extracellularly, leading to the dilution of sodium concentration. Osmotic diuresis causes the kidneys to excrete water, sodium, chloride and glucose.

b) Hyperkalemia: due to insulinopenia and acidosis, potassium shifts extracellularly. This can be misleading. The total body potassium is usually low because of diuresis, but appears high due to extracellular shifts.

c) High leukocyte count: is present in many patients, though it is not always indicative of an infection. It may be attributed to stress and dehydration.

d) Elevated amylase: can be present without pancreas pathology.

5. What is the treatment for DKA?

- Fluids, fluids and more fluids! This is the most important step! Patients with DKA are often depleted by five to seven litres. Infuse 15 mL/kg to 20 mL/kg normal saline for the first hour, then continue with hypotonic or normal saline after that. When the plasma glucose level reaches 12 mmol/L to 14 mmol/L, infuse five per cent dextrose solution.
- Once hypokalemia has been excluded, intravenous insulin is given in small doses through an infusion pump. The dosage is generally 0.1 units/kg/hr, increasing after the first hour until glucose level falls at a steady rate of 3 to 4 mmol/L.
- When the glucose level is between 12 mmol/L to 14 mmol/L, decrease the insulin infusion rate by 50% and start five per cent dextrose. Continue until ketoacidosis and ketonuria has resolved.
- Note that DKA patients have low total body potassium. Once insulin is given and acidosis resolves, potassium starts to move intracellularly and the hyperkalemia turns into hypokalemia. Potassium should be started when levels fall below 5 mmol/L. Add 20 mEq to 30 mEq potassium to each litre of infusion fluid. Measure potassium levels every one hour to two hours in the first five hours of treatment because of the rapid changes.
- Bicarbonate is generally *not* used, as there are few controlled studies demonstrating its benefit. There are risks in using bicarbonate, including hypokalemia, paradoxical central nervous system acidosis and increasing intracellular acidosis.
- In pediatrics cases, watch out for cerebral edema, a complication of treatment in this population. Sodium and water deficits should be corrected gradually and plasma glucose levels should not be decreased rapidly.
- Lastly, don't forget to treat the underlying cause, if present. Dx

References
1. Chiasson JL, Aris-Jilwan N, Bélanger R, et al: Diagnosis and treatment of diabetic ketoacidosis and the hyperglycemic hyperosmolar state. CMAJ 2003; 168(4):859-866.