

Diabetic Ketoacidosis

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Case

- A 19-year-old man is brought to the emergency department (ED) with diffuse **abdominal pain, vomiting, and altered level of consciousness**. The patient's symptoms began several days ago, when he complained of "**the flu**." His symptoms at that time included ~~profound fatigue, nausea, mild abdominal discomfort and some urinary frequency~~. Today he was found in bed moaning but otherwise **unresponsive**. **His past medical history** is unremarkable, and he is currently taking no medications. On physical examination, the patient appears pale and ill. His temperature is **36.0°C (96.8°F)**, pulse rate is **140 beats per minute**, blood pressure is **82/40 mm Hg**, and the respiratory rate is **40 breaths per minute**. **His head and neck examination shows dry mucous membranes and sunken eyes**; there is an unusual **odor to his breath**. The lungs are clear bilaterally with increased rate and depth of respiration. **The cardiac examination reveals tachycardia**, no murmurs, rubs, or gallops. The abdomen is diffusely tender to palpation, with hypoactive bowel sounds and involuntary guarding. The rectal examination is normal. **Skin is cool and dry with decreased turgor**. On neurologic examination, the patient moans and localizes pain but does not speak coherently. **Laboratory studies:** the leukocyte count is **16,000 cells/uL**, and the hemoglobin and hematocrit levels are normal. Electrolytes reveal a sodium of **124 mEq/L**, potassium **3.4 mEq/L**, chloride **98 mEq/L**, and bicarbonate **6 mEq/L**. **BUN and creatinine are mildly elevated**. The serum glucose is **740 mg/dL (41.1 mmol/L)**. The serum amylase, bilirubin, AST, ALT, and alkaline phosphatase are within normal limits. A 12-lead ECG shows **sinus tachycardia**. His CXR is normal.

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- What is the most likely diagnosis?
 - What is the next step?

Summary:

- This is a 19-year-old man with **acute diabetic ketoacidosis**.
- The patient has **new-onset diabetes mellitus**. The constellation of **severe hyperglycemia, anion gap acidosis, and ketosis** (manifested by the breath odor) is diagnostic. The other findings of **dehydration, hyponatremia, hypotension, altered level of consciousness, and diffuse abdominal pain**, are typical of a particularly severe episode of diabetic ketoacidosis.

Considerations

- Morbidity may result from either **underlying precipitating conditions, or from delayed or inadequate treatment.**
- In up to one-quarter of patients, **DKA is the initial presentation of type I diabetes**, so lack of a diabetic history cannot exclude the diagnosis.

CLINICAL APPROACH

- Diabetic ketoacidosis is a metabolic emergency. A delay in treatment leads to **increased morbidity and mortality**.
- Most cases occur in patients with **type I diabetes**, though some patients with type II diabetes may develop DKA during **severe physiologic stress**.

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- **Most likely diagnosis: Diabetic ketoacidosis**

Based on:

- hyperglycemia,
- ketosis,
- and metabolic acidosis
- **Next step: Management of the DKA including ;**
- effective resuscitation, and diligent attention to **fluid, electrolyte, and insulin** replacement are essential.
- **and a careful search for any precipitating or concomitant illness.**

Treatment

- Patients in DKA can have massive fluid deficits, sometimes as much as **5 to 10 L**. **Shock is fairly common**, and must be promptly treated with **crystalloid infusion** to prevent further organ damage.
- Adults with **clinical shock** should receive an initial **2-L bolus of normal saline** with frequent reassessment.
- **In children, shock** is treated with boluses of 20 mL/kg of normal saline. **Although overaggressive hydration** can present substantial **complications** later in the course of treatment, this concern takes a back seat to the reversal of shock. **Untreated shock** promotes **multiple-organ dysfunction** and contributes further to the **severe acidosis** seen in DKA.

Treatment

- The combination of **rehydration and insulin** will commonly ~~lower the serum glucose much faster than ketones are cleared.~~
- Regardless, **insulin infusion** should continue until the anion gap has returned to normal.
- Dextrose should be added to the IV infusion when the serum glucose falls to 250 mg/dL (14 mmol/L) to prevent hypoglycemia, a common complication of treatment.
- .Simply reverse shock with **normal saline**, and then continue an infusion of **half normal saline at two to three times maintenance**. Remember that **glucose should be added** before the serum glucose falls to the normal range, usually when the glucose level reaches 250 mg/dL.

Treatment

- An insulin dose of **0.1 U/kg/h (5-10 U/h in the adult)** is adequate for almost all clinical situations. This is sufficient to achieve maximum physiologic effect. Higher doses are no more effective, but do cause a higher rate of hypoglycemia.
- An **initial bolus of insulin**, equal to 1-hour infusion, is commonly given but has **not been shown to hasten recovery or provide any other benefit**. Insulin boluses are not recommended in **pediatric patients**.

Treatment

- **Potassium deficits** are usually quite large, yet the serum potassium at presentation may be low, normal, or even high. If the potassium is elevated initially, look for and treat any hyperkalemic changes on the ECG. **Give fluids without potassium until the serum K reaches the normal range, and then add potassium to the IV infusion.**
- **If the initial serum K is normal or low, potassium replacement** can be started immediately. **Magnesium supplementation** may be necessary to help the patient retain potassium. **Phosphate replacement** has not been shown to improve clinical outcomes, but extremely low phosphate levels (<1.0 mg/dL) are known to cause **muscle weakness and possibly rhabdomyolysis**. For such patients, some of the potassium can be given in the form of **potassium phosphate**.

Treatment

- Because **metabolic acidosis** is such a prominent feature in DKA, some clinicians have administered substantial doses of **sodium bicarbonate**.
- Many studies have failed to demonstrate any improvement from this treatment even at surprisingly low serum pH values, but even skeptical physicians sometimes encounter a patient who is so acidotic that they feel compelled to give bicarbonate. There are multiple theoretical and observed **complications** from **bicarbonate, including hypernatremia, hypokalemia, paradoxical CSF acidosis, and residual systemic alkalosis**. **Of course, in the case of hyperkalemia, bicarbonate might be lifesaving and should not be withheld**

Cerebral edema

- Cerebral edema is a rare but devastating complication of DKA, seen most often and most severely in children.
- It almost always **occurs during treatment**, and is a **leading cause of morbidity and mortality** in pediatric DKA.
- It has been variously associated with **overhydration** and **vigorous insulin therapy**, leading many pediatric centers to use extremely conservative, slow and low-dose treatment protocols for DKA.

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- **Cerebral oedema:** Children and adolescents are at the highest risk. Consider if:
 - headaches, or reduced conscious level. Monitoring for signs of cerebral oedema should start from the time of admission and should continue until up to at least 12 hours after admission. If there is a suspicion of cerebral oedema or the patient is not improving as expected, within 4 hours of admission, call the consultant. Check arterial blood gases and administer **Mannitol IV (100 ml of 20% over 20 minutes) or dexamethasone IV 8 mg (discuss with Consultant)** and undertake CT scan to confirm findings.



THANK
YOU