

CASE STUDY: DIABETIC KETOACIDOSIS (DKA)

One in a series of case studies developed to stimulate enhancement of problem-solving techniques for physicians and nurses and paramedical personnel when challenged by patients who present with unusual or complex clinical syndromes.

This Case Study is provider approved by the California Board of Registered Nursing, provider number 9697, for 1.0 contact hours. This course has been approved for one (1) hour of category One EMT-P Continuing Education by California EMT-P CE Provider #49-008.

LEARNING OBJECTIVES

After studying this case presentation, the student should be able to:

- Describe the major causative factors of diabetic ketoacidosis (DKA).
- List the clinical signs and symptoms of diabetes and DKA.
- Describe the major metabolic changes in DKA.
- Understand the fluid and electrolyte changes in DKA.
- Discuss the major objectives of medical management of DKA.

HISTORY

A 14-year-old male is brought to the Emergency Department via ambulance with a report of the patient being found unresponsive. The report given by the paramedics is that the patient's mother, who had not seen the child for over 24 hours, came home to find her son lying on the sofa unresponsive. Copious quantities of black colored vomit were evident. The paramedics report the child is a diabetic and gives himself his own "medication." His mother told the medics she was unsure when her son last took his medication. The patient himself offers no history whatsoever. The paramedics express deep concern regarding the social situation in which they found this child. The mother has not yet arrived in the Department to provide further information.

PHYSICAL EXAMINATION

Blood pressure: 101/72; heart rate: 123; respirations: 32; oral temperature: 34.8 °C; pulse oximetry: 100% on room air.

General: An approximately 65 Kg, thin male who responds to simple questions with moans, but is, in general, responsive only to very loud or painful stimuli. Head and neck are normal, except for his oropharynx, which demonstrates very dry mucous membranes and a moderate amount of dried, black material which is strongly Gastrocult positive.

His lungs are clear. His respiratory pattern is that of rapid and deep breathing ("Kussmaul" breathing).

Abdomen exam is negative. There are no other pathological findings on PE.

How would you proceed from here? Does this patient need an IV? If so, what types of fluid do you want to initiate and at what rate? What basic lab tests would you order? What other not-so-basic tests would you order? Formulate your answer before proceeding.

COURSE IN THE EMERGENCY DEPARTMENT

An intravenous infusion of normal saline was established and hydration was initiated at a rate of 200 cc/hr. A serum glucose determination (Accucheck) was "too high to read." A sample of the blood was sent to the lab for a definitive determination of the serum glucose level.

How does this affect your differential diagnosis? What additional care would you now render this patient?

The patient was given a bolus of 10 units of regular insulin IV while waiting for the lab results. The results came back shortly thereafter, and showed an arterial blood gas pH of 6.92, CO₂ of 9 and a bicarb of 2. The WBC count was 62.6 thousand (62,600), hemoglobin of 14.4 mg/dL, and hematocrit of 43.5%. His chemistry panel demonstrated a serum sodium of 127, potassium 5.2, chloride of 87, CO₂ of less than 5, BUN of 32, creatinine 1.5, and a blood sugar of 1,582. The serum ketones were positive at a dilution of 1:32.

What is your interpretation of these results? What additional treatment would you add? Would you administer antibiotics?

These results show severe metabolic acidosis secondary to DKA. The patient also has a very high WBC, which could be an indication of an infection, stress reaction, profound dehydration or perhaps all three. A search for a site of infection is warranted as part of the work up. The administration of broad spectrum antibiotics should be considered until a source of infection is found or ruled out.

The patient was started on an infusion of regular insulin at 10 units per hour. He was also given Zantac® (an H2 blocker) to decrease stomach acid production in light of his upper GI bleeding, which is most likely a result of stress gastritis. Blood and urine cultures were sent to the lab. Antibiotics were administered.

FURTHER HISTORY

The patient's mother arrived after care had begun. She had little to add to the case, but did confirm the child was responsible for administering his own insulin. This was the "medication" to which the paramedics referred in their first report in the emergency department.

FURTHER COURSE IN THE EMERGENCY DEPARTMENT

The patient's airway remained stable. His respiratory rate decreased. His vital signs stabilized. With hydration and insulin therapy as outlined above the patient showed an increased level of consciousness, was able to converse with his mother, and stated he was feeling "a little better." He was now able to tell us that he had not been taking his insulin "for a few days" and had been experiencing a mild cough.

TRANSFER ARRANGEMENTS

Arrangements were made to transfer this patient to hospital with a pediatric intensive care unit via helicopter. After consultation with the pediatric intensivist, fluids were adjusted to 160 cc/hr and the insulin infusion was decreased to 5 units/hr. The REACH critical care team after assessment determined the patient to be stable and initiated transport, maintaining the crystalloid and insulin infusions.

DISCUSSION

DKA is a disorder of glucose metabolism which is characterized by hyperglycemia, increased serum ketones (and other products by of fatty acid

metabolism), metabolic acidosis and dehydration. The basic metabolic problem is a lack of insulin or in some cases, lack of cellular capacity to utilize insulin. The result is the inability to transfer glucose through the cell membrane. This results in an increasing amount of glucose in the serum, i.e., it cannot "get into" the cell. The excess glucose is excreted by the kidneys, which must lose water to keep the glucose in solution. This leads to the dehydration and increased osmolarity of the serum (an increase in dissolved products), which is a major factor in the altered mental status associated with DKA.

Also, as water is drawn out through the kidneys, it carries with it sodium, potassium and other ions, contributing to the electrolyte abnormalities of DKA. A confounding factor is with profound dehydration and acidosis significant potassium loss can be "masked" in some instances. The serum level could be high but total body potassium, which is not measurable, is low. Observe the patient closely as you rehydrate because the serum potassium can drop quickly as the patient becomes normovolemic, and insulin enhances transport of potassium into the cell.

What occurs at the cellular level in DKA? Glucose can't get into the cell because of the lack of insulin to facilitate its entry. In the absence of glucose, the cell cannot maintain normal, aerobic metabolism. In an "effort" to continue function, the cell reverts to its "backup plan." It begins to use amino acids and fatty acids as its energy sources. Proteins and lipids are released from the body tissues. In the liver the proteins are broken down into amino acids and released back into the blood to be used by the cells as an energy source. The free fatty acids are metabolized in the liver, with ketoacids (acetoacetate, and beta-hydroxy butyrate) as byproducts. They are also used by the cells as an energy source. At a cellular level the use of amino acids and ketoacids energy sources is very inefficient, worsening the crisis at hand, contributing to the metabolic acidosis of DKA.

Of interest is the fact when serum ketone levels are measured in most labs, only acetoacetate and not beta-hydroxy butyrate is identified. For this reason, serum ketones, although present, are not always reported as positive in DKA.

The dehydration which evolves with DKA also leads to impaired renal function, further compounding the acidosis. These metabolic changes in the absence of insulin help explain the symptoms and signs typical of diabetes, polyuria (urinating a lot), polydypsia (drinking a lot, i.e., excessive thirst), and weight loss. In spite of good food intake by the patient, weight loss occurs

secondary to cellular starvation. All of the factors discussed above combine and affect one another in a manner which exacerbates the problem.

As in this case, the most common problem leading to DKA is the lack of insulin administration in an insulin-dependent diabetic. DKA can also be the presenting finding in new-onset diabetes. Physiologic or pathologic stress of any type can also lead to DKA by causing an increased utilization of insulin. These stresses include, but are not limited to, infection (urinary and lungs are most common), GI bleeding, myocardial infarction, congestive heart failure, and pregnancy.

Proper treatment of DKA consists of providing the necessary insulin for as long as is needed until the metabolic processes can readjust themselves, and correcting both the physiologic and pathologic conditions which lead to the insulin insufficiency. It is also essential to reverse dehydration.

After initial lab tests confirm the diagnosis of DKA, insulin in small doses is administered, usually as a bolus, followed by an infusion. Fluid replacement is indicated as DKA patients can have a large fluid deficit, sometimes as much as ten liters. Although it was not necessary in this case, in general crystalloid therapy is initiated "wide open" until approximately two liters are infused (don't do this in the child). Normal saline is an excellent fluid for this purpose. Serum potassium should be determined, and if low to low normal, replacement should be started early with crystalloid therapy. If potassium is high normal or high, it should be checked frequently and replacement begun as it begins to drop through the normal range. Serum glucose should be checked frequently and plain normal saline can be changed to fluid with dextrose when the levels fall to between 200-300 mg/dL. The glucose level should then be maintained at higher than normal levels until acidosis is corrected.

A common misunderstanding regarding the treatment of DKA is as the serum glucose level returns to normal, the problem has been corrected. It has not! It is important to continue insulin therapy until the acidosis is also corrected, and to replace glucose when its level gets into the 200-300 mg/dL range.

A note on treatment with bicarbonate: In the past, it was routine to treat the metabolic acidosis with bicarbonate. It is now widely understood this is of little, if any, benefit and indeed may be more harmful than helpful in the treatment of DKA. The reasons for alteration of this practice pattern are many (for example, exogenous administration of bicarbonate may lead to a paradoxical increase in intracellular acidosis), but outstanding among them is replacing the insulin and

correcting the dehydration will stimulate the body's own buffering systems to function more efficiently and correct the acidosis with fewer long term physiological side effects than will treatment with sodium bicarbonate. Another factor is the respiratory buffering system is working by means of an increased respiratory rate to compensate the metabolic by "blowing off" CO₂".

Although not a factor in this case another potential cause of altered level of consciousness leading to non-administration of insulin and consequently DKA is drug intoxication. It would have been a good idea to order a toxicology screen on this young man soon after his presentation to the emergency department.

Summary, DKA is a severe metabolic condition brought on by a lack of insulin, absolute or functional, which leads to profound dehydration, acidosis, and electrolyte disturbances. Although quite frightening at the outset, its treatment is relatively straightforward. Replace fluids with crystalloid and administer insulin early on. Later administer potassium and glucose. Treat infection with antibiotics. This treatment will in most instances result in DKA patients equilibrating to a normal physiological state.

KEY POINTS

- Lack of insulin or insulin utilization "sets up" a chain of events leading to DKA.
- A metabolic cascade results in profound dehydration, cellular starvation and acidosis.
- A search for infection is always indicated and presumptive treatment with antibiotics is indicated even if no source is found early on.
- Total body potassium depletion is almost always present despite high or normal serum levels. Observe closely and replace as indicated.
- Fluid deficits can be large, 5-10 liters, and should be replaced aggressively, initially with crystalloid.
- Continue treatment until not only the glucose imbalance but also the acidosis are corrected.

We would welcome any questions or comments about this case study. We would also welcome any suggestions relevant to developing a case study from an interesting case involving your unit and REACH.

Let us hear from you. Should you desire to read previously published case studies and the opportunity to receive additional CEUs, visit our website at www.reachair.com. You can do so online.

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CASE STUDY

POST TEST

DIABETIC KETOACIDOSIS (DKA) QUESTIONS: choose **all** correct answers

1. Which of the following **is not** a sign of diabetes?
 - A. Drinking a lot of water.
 - B. Frequent urination.
 - C. Increased intracranial pressure.
 - D. Weight loss despite good food intake.

2. Diabetes and DKA are related to which hormonal problem?
 - A. An increased insulin level.
 - B. A decreased insulin level.
 - C. An increased utilization of insulin by cells.
 - D. A decreased utilization of insulin by cells.
 - E. A and C
 - F. B and D.

3. Almost all patients in DKA are:
 - A. Only slightly dehydrated and can drink water to replace fluids.
 - B. At risk of becoming dehydrated and need an IV at a TKO rate.
 - C. Moderately dehydrated (1-2 liters) and need ½ normal saline.
 - D. Profoundly dehydrated (5-10 liters) and need aggressive hydration with normal saline.

4. Which electrolyte is depleted in DKA despite initial determinations which indicate normal or even high levels?
 - A. Sodium
 - B. Potassium
 - C. Chloride
 - D. Carbon Dioxide
 - E. Glucose

5. Early in the treatment of DKA, it is vital to administer:
 - A. Glucagon
 - B. Glucose
 - C. Insulin
 - D. Sodium
 - E. Keto acids

6. A patient who presents in DKA with fever will typically require the following IV therapy during the first 24 hours of treatment. All except:
 - A. Insulin
 - B. Crystalloid
 - C. Potassium
 - D. Antibiotics
 - E. Midazolam

7. Factors leading to DKA include all of the following **except**:
 - A. Infection.
 - B. Lack of insulin administration.
 - C. Stress.
 - D. Eating too much sugar quickly.
 - E. Undiagnosed type I diabetes mellitus.

8. Ketone tests in the lab are not always positive in DKA because:
 - A. Not all of the keto acids are measured.
 - B. Patients can be in DKA without ketones in the serum.
 - C. Ketones are not always present at first and may show up later.
 - D. As DKA gets very severe, the level of ketones drops until they disappear.

9. Altered levels of consciousness in DKA are related to:
 - A. Dehydration.
 - B. Acidosis.
 - C. Altered glucose metabolism.
 - D. All of the above.

10. When the glucose level begins to come down to the range of 200-300 mg/dL when treating DKA, it is time to:
- A. Stop treatment with insulin so the patient does not "bottom out."
 - B. Replace magnesium aggressively.
 - C. Begin glucose replacement.
 - D. Stop the rehydration.
 - E. Change the fluids to 1/4 normal saline and continue hydration.

