DIABETIC KETOACIDOSIS

Diabetic ketoacidosis (DKA) is a state of uncontrolled diabetes and it is characterized by hyperglycemia, a high anion gap acidosis, and the presence of ketonemia and ketonuria (ketone bodies in the blood and urine). Although it can occur in patients with type 2 diabetes (during periods of severe stress), DKA primarily occurs in patients with type 1. Type 1 diabetes is caused by the (usually autoimmune) destruction of the pancreatic beta cells, which leads to an absolute insulin deficiency. Thus, patients with type 1 have an absolute requirement for insulin and will develop DKA if they do not receive it.

The lack of insulin, the increase in glucagon (normally suppressed by insulin), and the high levels of the stress hormones epinephrine and cortisol all contribute to hyperglycemia in DKA. But despite such a large amount of sugar in the blood, the body’s organs are essentially starved for fuel because there is not enough insulin to stimulate their uptake of glucose. In an attempt to create an alternate source of energy, these organs begin diverting their metabolic resources to produce ketone bodies. Although ketone bodies allow cells to maintain a very minimal level of function, they are acids, and so can cause a dangerous anion gap acidosis.

The most common underlying causes of DKA are infection (which produces an increased need for insulin) and noncompliance with one’s insulin regimen. Because patients with type 1 diabetes have an absolute insulin deficiency, either of these scenarios can turn an already tenuously balanced metabolic state into a severely ketoacidotic one.

More information about diabetic ketoacidosis is discussed in the following articles:

History:
Normally, all glucose filtered by the kidney is reabsorbed. With hyperglycemia, this mechanism is overwhelmed and glucose “spills” into the filtrate, and pulls water with it. For these reasons, polyuria is a common feature of DKA. Because the hyperglycemia causes a rise in serum osmolarity, extreme thirst with polydipsia is also common. As the acidosis causes a very strong drive to breathe, patients may feel short of breath. Nausea and vomiting are also common in DKA (cause unknown), as well as a feeling of general malaise. These latter symptoms are often the reason for seeking medical attention.

Another fairly common patient report is blurred vision. While the vision problems in diabetic retinopathy result from years of hyperglycemia, blurry vision in DKA results from the rapid movement of water in and out of the orbit, a consequence of the osmotic shifts that occur with fluctuating serum glucose levels.

Physical Exam:
Inspection is quite valuable in DKA. Immediately upon talking with the patient, one may detect a pungent fruity smell on the patient’s breath, a result of the acetone (a ketone) that the body’s organs are producing. In addition, people with DKA have Kussmaul breathing (deep and rapid breaths), reflecting the body’s attempt to blow off carbon dioxide to compensate for the metabolic (keto)acidosis. Finally, because polyuria may cause severe dehydration (especially if the patient does not drink enough replacement fluids), the patient may be tachycardic and even hypotensive, and may show reduced skin turgor. Patients with severe hyperglycemia may exhibit signs of delirium as the associated shifts in fluid affect the brain.

Tests:
A finger prick to measure one’s plasma glucose level will reveal the presence of extreme hyperglycemia. (This should be confirmed with a serum glucose.) An electrolyte panel will reveal an
elevated anion gap metabolic acidosis, and the actual blood pH can be determined by an arterial blood gas.

Infection is a common precipitant of DKA; its presence is usually investigated with a complete blood count (to look for elevated white blood cells), urinalysis, and chest radiograph. In older patients, an ECG should be obtained since the medical stress from a myocardial infarction can precipitate DKA.

Treatment:
Because of the severe volume depletion that occurs as a result of hyperglycemia, isotonic fluids are given intravenously. As DKA reflects a state of insulin deficiency, IV insulin therapy is also administered. Finally, careful attention must be paid to electrolyte levels, with cautious addition of those ions whose levels have been depleted. For example, potassium re-enters cells as the acidosis resolves and will also be driven into cells by insulin. This exit of potassium from the blood will unveil a previously masked potassium depletion (from urine losses). Life-threatening hypokalemia can occur, so there should be close monitoring and replacement.

If infection is the precipitating cause of DKA, broad spectrum antibiotics should be administered.

More information about the treatment of diabetic ketoacidosis is discussed in the following article:

Self Assessments:
Which of the following would NOT be associated with diabetic ketoacidosis?

A. A fruity smell on the patient’s breath
B. Kussmaul breathing
C. A large anion gap
D. Hypertension

Explanations:
A. Incorrect. The fruity smell on the patient’s breath is from the production of acetone, an alternate source of fuel made by organs that are starved of glucose.
B. Incorrect. Kussmaul breathing is the body’s attempt to blow off as much carbon dioxide as possible in order to compensate for the metabolic acidosis from DKA.
C. Incorrect. A large anion gap indicates the presence of a metabolic acidosis, a key feature of DKA.
D. Correct! Due to the considerable amount of fluid lost in the urine (polyuria), the patient in DKA will be dehydrated. She may be normotensive, but tachycardic (reflecting her volume depletion), or she may even be hypotensive.

True of false: Blurry vision in DKA results from years of poor glycemic control.

A. True
B. False

Explanations:
A. Incorrect. Years of poor glycemic control leads to diabetic retinopathy, not the acutely fluctuating vision changes that occur because of the rapidly rising (or falling - if treatment has started) glucose levels in DKA.
B. Correct! The blurry vision in DKA results from rapidly fluctuating glucose levels, which cause fluid quantities in the eye to constantly change.