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**LEVEL: 300**

**1. DEFINE THE FOLLOWING:**

A. **KETOGENESIS**: Ketogenesis is a metabolic pathway that produces ketone bodies, which provide an alternative form of energy for the body.

B. **KETONAEMIA**: Ketonaemia the presence of an abnormally high concentration of ketone bodies in the blood.

C. **KETONURIA**: **Ketonuria** is a medical condition in which ketone bodies are present in the urine.

D. **KETOGENESIS**: **Ketogenesis** is the biochemical process through which organisms produce ketone bodies through breakdown of fatty acids and ketogenic amino acids.

**2. WHAT ARE THE CONSEQUENCES OF KETOSIS**

1. Headache

2. Fatigue

3. Brain fog

4. Increased hunger

5. Poor sleep

6. Nausea

7. Decreased physical performance

**3. WRITE CONSICELY ON THE MANAGEMENT OF KETOACIDOSIS**

An overproduction of ketone bodies through increased ketogenesis can pose a problem due to their acidic nature.

Diabetic ketoacidosis (DKA) is an example involving the overproduction of ketone bodies. It occurs when there is a lack of, or resistance to, insulin. This usually occurs in people with type I diabetes, although it can happen to people with advanced type II diabetes as well. In most cases of type II diabetes, enough insulin production continues to prevent excessive ketogenesis.

Due to the lack of glucose brought in by insulin, cells start to produce glucose via gluconeogenesis. This process, along with existing glucose that cannot be brought in with insulin, greatly elevates serum glucose levels. The threshold for DKA is a glucose level of 250. However, it is typically greater than this amount.

Once carbohydrate stores are depleted and gluconeogenesis cannot occur anymore, ketogenesis is substantially increased, and there are larger amounts of ketone bodies produced. Due to the acidic nature of beta-hydroxybutyrate and acetoacetate, this causes an anion gap metabolic acidosis.

On presentation, patients are usually very dehydrated from being hyperglycemic. The high glucose levels lead to osmotic diuresis, involving greater osmole concentrations (in this case the osmole being glucose) that cause an increased osmotic pressure, which leads to reduced water reabsorption in the kidneys. Along with being dehydrated, patients typically present with confusion, nausea, vomiting, and abdominal pain. Because of the acidosis, patients often breathe very deeply and rapidly to eliminate carbon dioxide and cause a respiratory alkalosis. This process is known as Kussmaul breathing, and, over time, a patient can experience respiratory distress due to the prolonged exertion of respiratory muscles. Cerebral edema can occur in severe cases of DKA. Because of the acetone produced by ketogenesis, patients can have breath that smells fruity or like nail polish remover.

The main goal of treating DKA is to resolve the metabolic acidosis, which involves giving glucose and insulin to not only lower blood glucose levels but also to downregulate the ketogenic pathway and decrease the number of ketone bodies produced.

Ketoacidosis also can occur with severe alcoholism and prolonged starvation.

The treatment for DKA usually involves a combination of approaches to normalize blood sugar levels and insulin levels. If you’re diagnosed with DKA but haven’t yet been diagnosed with diabetes, your doctor will create a diabetes treatment plan to keep ketoacidosis from recurring.

Infection can increase the risk of DKA. If your DKA is a result of an infection or illness, your doctor will treat that as well, usually with antibotics.

The therapeutic goals of DKA management include optimization of

1. Volume status;

2. Hyperglycemia and ketoacidosis;

3. Electrolyte abnormalities; and

4. Potential precipitating factors.

The majority of patients with DKA present to the emergency room. Therefore, emergency physicians should initiate the management of hyperglycemic crisis while a physical examination is performed, basic metabolic parameters are obtained, and final diagnosis is made. Several important steps should be followed in the early stages of DKA management:

1. Collect blood for metabolic profile before initiation of intravenous fluids;

2. Infuse 1 L of 0.9% sodium chloride over 1 hour after drawing initial blood samples;

3. Ensure potassium level of >3.3 mEq/L before initiation of insulin therapy (supplement potassium intravenously if needed);

4. Initiate insulin therapy only when steps 1–3 are executed.

**FLUID REPLACEMENT**: At the hospital, your physician will likely give you fluids. If possible, they can give them orally, but you may have to receive fluids through an iv. Fluid replacement helps treat dehydration which can cause even higher blood sugar levels. Fluid replacement. You'll receive fluids — either by mouth or through a vein (intravenously) — until you're rehydrated.

**INSULIN THERAPY:** Insulin will likely be administered to you intravenously until your blood sugar level falls below 240 mg/dL. When your blood sugar level is within an acceptable range, your doctor will work with you to help you avoid DKA in the future.

**ELECTROLYTE REPLACEMENT:** When your insulin levels are too low, your body electroltyes can also become abnormally low. Electrolytes are electrically charged minerals that help your body, including the heart and nerves, function properly. Electrolyte replacement is also commonly done through an IV. Electrolyte replacement. Electrolytes are minerals in your blood that carry an electric charge, such as sodium, potassium and chloride.