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ETIOLOGY AND PATHOGENIC ASPECTS OF KETOACIDOSIS

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This report discusses about the pathogenesis, diagnosis and treatment of diabetic ketoacidosis. Diabetic ketoacidosis is a life-threatening hyperglycemic condition which happens due to severe insulin deficiency and increase of counter regulatory hormones such as glycogen, cortisol, growth hormone and epinephrine.

Diabetic ketoacidosis happens in both type 1 diabetic and type 2 diabetic patients. It is known to be common in diabetic type 1 patients where there is lack of insulin secretion in the body, but in type 2 diabetic patients who are on irregular treatment also have a high chance of having diabetes ketoacidosis.

Due to a very low level of insulin, the insulin dependence glucose utilization is impaired and glucose level in the circulatory system and extra cellular fluid increases, where there is poor utilization of glucose in peripheral tissue. Because of this the peripheral tissue stimulates the counter regulatory hormones and increase the glucose level by glycogenolysis and gluconeogenesis.

Gluconeogenesis is the formation of glucose from non carbohydrate substances like free fatty acids and amino acids from the peripheral tissues which are utilized in the liver to produce glucose. The main source of protein is the muscle tissue where proteins are broken down into amino acids and sent to the liver and the glycogen in the muscle is broken into glucose-6-phosphate and through glycolysis transforms into lactate and sent to liver for gluconeogenesis.

When the insulin level is very low, and the adipose tissue doesn't get enough glucose the adipose tissue will secrete lipoprotein lipase enzyme. This will begin severe lipolysis, and fats are rapidly broken into free fatty acids. These free fatty acids are transported to the liver through the blood. Due to severe increase of free fatty acids in the liver, the liver could not utilize all the fatty acids. So, the remaining will go to the mitochondria of the liver and transform into ketone bodies through multiple steps.

The ketone bodies increase the pH of the blood by increasing the proton level. The increased proton will bind with HCO_3^- ion and form H_2CO_3. which will break down into water and Carbon dioxide. Carbon dioxide will increase the acidity and stimulate the respiratory center in the medulla and increase the respiratory rate by stimulating the inspiratory center. The increased protons will enter the cell in exchange of potassium and cause hyperkalemia (increase of potassium in blood), which will cause many cardiac problems like arrhythmia, muscle weakness, depress cardiac contractility.

Kussmaul's breathing is one of the key findings in diagnosis. During this the tidal volume will be increased, and this will be shown by rapid deep breathing of the patient. The patient's breath will also be sweet fruity smell which can be easily identified by the doctors.

In the urinary system the patients will have glycosuria where glucose won't be fully reabsorbed in the proximal convoluted tubule because of the saturation of glucose in the blood, polyuria where the reabsorption of water will be decreased, ketonuria where ketone bodies will be seen in the urine.

The main treatment for diabetic ketoacidosis is management of fluid balance, insulin replacement and electrolytes balance. Insulin should be given to the patient constantly in small amounts. If insulin is given to the patient in high dose hypokalemia will occur and cause serious cardiac arrhythmia and cause the patient to die.

Diabetic ketoacidosis is a fatal acute metabolic condition of diabetic Mellitus patients, where poor management of blood glucose levels can become fatal. Proper patient education regarding this and effective communication with the healthcare provider can improve the quality-of-life and life expectancy of the patient.