Diabetic ketoacidosis patients can have a wide range of serum potassium concentrations

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Introduction

Diabetic ketoacidosis is common in children with known type 1 diabetes. It affects about 1-10% of children with type 1 diabetes each year. Usually these children are either not taking insulin or have insulin delivery problems (such as insulin pump problems). Diabetic ketoacidosis can also occur when children do not get enough insulin when they are sick (when they are sick they need more insulin). Without insulin, cells cannot use glucose in the blood. Cells switch to backup mechanisms to gain energy and break down fat. Its by-products are compounds called ketones.

Ketones acidify the blood (ketoacidosis), causing nausea, vomiting, fatigue, and abdominal pain. Ketones make a child's breathe smell like nail polish remover.[1] Breathing becomes deeper and faster as the body tries to compensate for the acidity of the blood. Some children develop headaches, which can make them confused and less alert. These symptoms can be caused by fluid accumulation in the brain (cerebral edema).

Description

Diabetic ketoacidosis usually occurs because the body does not have enough insulin. Cells cannot use sugar in the blood for energy, so they use fat for fuel instead. Fat burning produces acids called ketones. If this process continues for some time, it can accumulate in the blood. This excess can alter the chemical balance of the blood and unbalance the entire system. A child is said to have diabetes if they have elevated blood sugar levels in addition to the typical diabetes symptoms. The diagnosis is confirmed by measuring blood sugar levels.[2] Blood sugar levels can be measured in the morning before the child eats (called fasting blood sugar) or outside meals (called control blood sugar). People with type 1 diabetes are at risk of ketoacidosis because their bodies don't produce insulin. Untreated ketoacidosis can lead to unconsciousness, coma, and even death. I have to go to the hospital to treat DKA. When you try to keep your blood sugar normal, you run the risk of getting your blood sugar too low.

Hyperglycemia by DKA results from accelerated gluconeogenesis, glycogenolysis, and decreased glucose utilization. All of these are due to absolute insulin deficiency. It may occur in diabetic patients who develop DKA during treatment with SGLT-2 inhibitors without hyperglycemia, i.e. H. euglycemic DKA.[3] Increased lipolysis and decreased lipogenesis convert abundant free fatty acids to ketone bodies: beta-hydroxybutyrate (beta-OHB), acetoacetate, and acetone. Hyperglycemia-induced osmotic diuresis, if not accompanied by adequate oral water intake, leads to dehydration, hyperosmolarity, electrolyte depletion, and subsequent decreased glomerular filtration. As renal function declines, glucosuria decreases and hyperglycemia/hyperosmolarity worsens. Impaired insulin action and hyperosmolarity significantly reduce potassium utilization by skeletal muscle, leading to intracellular potassium depletion.[4] Potassium is also lost through osmotic diuresis, leading to severe systemic potassium deficiency. Therefore, DKA patients can have a wide range of serum potassium concentrations.

Conclusion

Acidosis usually causes a respiratory response. Lowering the bicarbonate and pH will cause hyperventilation, reducing carbon dioxide and preventing further pH drop. When excess glucose enters the renal tubules, a lot of water is taken up and eventually a lot of urine is produced. This is called osmotic diuresis, and the patient loses fluid volume and becomes dehydrated. If left untreated, diabetic ketoacidosis can lead to coma and death. Symptoms of diabetic ketoacidosis include excessive thirst, frequent urination, nausea and vomiting, stomach pain, weakness or fatigue, shortness of breath, fruity breath, and confusion. Nonetheless, 'normal' plasma potassium concentrations indicate that the body's potassium stores are severely depleted, and that initiation of insulin therapy and correction of hyperglycemia will lead to future hypokalemia.

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Conflict of Interest

The author has nothing to disclose and also state no conflict of interest in the submission of this manuscript.

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