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Abstract: *Metabolic acidosis is a primary decrease in the level of bicarbonate (HCO_3^-), usually accompanied by a compensatory decrease in the partial pressure of carbon dioxide (Pco_2); pH can be significantly reduced or close to normal. Depending on the presence or absence of unmeasured anions in plasma, metabolic acidosis is distinguished with a large and normal anion gap.*

Keywords: *absence of unmeasured, intravenous sodium bicarbonate, metabolic acidosis.*

Ketoacidosis is a common complication of type 1 diabetes (see diabetic ketoacidosis), but it also develops in chronic alcoholism (see alcoholic ketoacidosis), malnutrition, and to a lesser extent, fasting from certain foods. Under these conditions, the body switches from glucose metabolism to free fatty acids (FFA). In the liver, FFAs are converted into keto acids - acetoacetic and beta-hydroxybutyric (immeasurable anions). Ketoacidosis is also a rare manifestation of congenital isovaleric acidemia or congenital methylmalonic acidemia.

Large anion gap acidosis. The most common causes of large anion gap acidosis are:

- ketoacidosis
- lactic acidosis
- kidney failure
- toxin poisoning

Lactic acidosis is the most common cause of metabolic acidosis in hospitalized patients. Lactate accumulates as a result of its increased formation in combination with reduced metabolism. Excess lactate production occurs during anaerobic metabolism. The most severe forms of lactic acidosis are observed in various types of shock. Reduced lactate metabolism is characteristic of liver dysfunction due to a local decrease in its perfusion or due to generalized shock. Diseases and drugs that interfere with mitochondrial function can cause lactic acidosis.

Renal failure contributes to the development of an acidosis with a large anion gap due to a decrease in acid excretion and reabsorption of HCO_3^- . The large anion gap is

due to the accumulation of sulfates, phosphates, urate and Hippurate. Toxins can be metabolized to form acidic products or induce lactic acidosis. A rare cause of metabolic acidosis is rhabdomyolysis; it is believed that the muscles at the same time directly release protons and anions.

Acidosis with normal anion gap. The most common causes of normal anion gap acidosis are: Loss of HCO_3^- through the gastrointestinal tract or kidneys. Impaired renal excretion of acids. Normal anion gap metabolic acidosis is also called hyperchloremic acidosis because chloride (Cl^-) is reabsorbed in the kidneys instead of HCO_3^- . Causes of metabolic acidosis include accumulation of ketone bodies and lactic acid, kidney failure or consumption of drugs or toxins (large anion gap), and loss of HCO_3^- through the gastrointestinal tract or kidneys (normal anion gap). In severe cases, nausea and vomiting, drowsiness and hyperpnea are observed. The diagnosis is established on the basis of clinical data and the results of the determination of arterial blood gases (ABG) and serum electrolytes. Treatment is to eliminate the cause; at very low pH, intravenous sodium bicarbonate may be indicated.

Loss of this ion through diarrhea, gastric drainage, or fistulas can cause acidosis. Many secretions of the gastrointestinal tract (eg, bile, pancreatic juice, and intestinal fluid) contain large amounts of HCO_3^- . During ureterosigmoidostomy (implantation of ureters into the sigmoid colon in case of obstruction or removal of the bladder), the intestine secretes and loses HCO_3^- in exchange for chloride ions (Cl^-) present in the urine and absorbs ammonium from the urine, which dissociates into ammonia (NH_3^+) and ions hydrogen (H^+). In rare cases, the loss of HCO_3^- is caused by the intake of ion exchange resins that bind HCO_3^- .

In different types of renal tubular acidosis, either H^+ secretion (types 1 and 4) or HCO_3^- absorption (type 2) is impaired. Impaired acid excretion and a normal anion gap have also been reported in early renal failure, tubulointerstitial nephritis, and carbonic anhydrase inhibitors (eg, acetazolamide).

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