Acid-base balance disorders

Acid-base balance disorders are conditions in which:

- the pH of the body's internal environment deviates from the norm (**acidemia, alkalemia**)

or

- there is an excess or deficiency of acids or bases in the organism, ie there is a change in the composition of the buffers (which may or may not be accompanied by a change in the resulting pH; **acidosis, alkalosis**).

Bicarbonate buffer is of the utmost importance for rapid pH maintenance. One of its components, (HCO_3^-), has a charge and is a relatively significant part of the ionogram. Acid-base balance is therefore closely linked to the metabolism of major ions. In practice, any major acid-base imbalance will also be accompanied by a disorder in the mineralogram. And conversely, major changes in the ionogram are usually accompanied by an acid-base imbalance. You can find more about the relationship between the acid-base balance and the ionogram here.

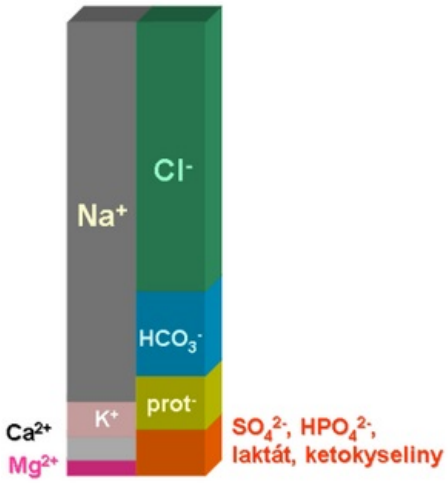
Respiratory disorders of the acid-base balance

If the ventilation changes, the partial pressure of carbon dioxide in the blood changes, and therefore also the concentration of the carbonic acid in the bicarbonate buffer changes. Specifically:

- **hyperventilation** accompanied by **hypocapnia** leads to **respiratory alkalosis**

and conversely

- **hypercapnia** caused by a ventilation disorder will result in **respiratory acidosis**.



Ionogram

Metabolic disorders of the acid-base balance

Metabolic acid-base disorders are conditions in which the concentration of bicarbonates changes significantly (more precisely: standard bicarbonates - see Laboratory evaluation of acid-base balance). At the same time, the concentration of one or more major ions always changes, because the bicarbonate anion must be in balance with other ions in the body fluids (more in the chapter Relationship between acid-base balance and ionogram).

Metabolic acidosis

Metabolic acidosis is a condition in which the **concentration of standard bicarbonates falls below reference values**. This can happen:

- due to the accumulation of an anion that "pushes" the bicarbonates out of the mineralogram;
- due to the loss of bicarbonates (accompanied by a cation, most likely as sodium bicarbonate);
- more rarely: due to losses of some cations, most likely sodium, which are compensated by a decrease in bicarbonate concentrations.

MAC:

Acidosis due to ESRD or ARF:

MAC 2:

Metabolic acidosis from anion accumulation

Lactic acidosis
lactic acid in a medium close to 7.4 dissociates almost completely into the lactate anion. Lactate concentration increases significantly, especially in tissue hypoxia.

Ketoacidosis
(in terms of ABR accumulation of β-hydroxybutyrate and acetate). It develops when glucose is not a sufficient source of energy and fats are broken down to an increased extent: during starvation, type 1 diabetes, extreme exercise, etc.

Renal acidosis
in renal failure, sulphates, phosphates and other anions accumulate that would normally be excreted in the urine.

Acidosis in some poisonings

- **ethanol intoxication - ethanol is metabolized to acetate.**

  In addition to acetate overproduction, NADH production plays an important role in ethanol degradation. The high concentration of reducing equivalents inhibits the breakdown of lactate that accumulates. Similarly, NADH inhibits glycolysis, which ultimately leads to stimulation of ketogenesis and accumulation of β-hydroxybutyrate and acetate.

- **methanol intoxication - methanol is metabolized to formate;**
- **ethylene glycol intoxication - metabolized to oxalate.**

Metabolic acidosis from bicarbonate losses

It is most often due to the loss of bicarbonates from the **gastrointestinal tract**. Duodenal and pancreatic juices are rich in bicarbonates, which are supposed to neutralize the digestion coming from the stomach. Normally, bicarbonates are resorbed back in the small intestine. In some GIT diseases (diarrhea, short bowel syndrome), resorption may be so low that blood bicarbonate levels drop significantly.

Renal loss of bicarbonate may be another cause (renal tubular acidosis, side effect of diuretic therapy, etc.). We can also include the so-called **dilution acidosis** in the group of metabolic acidoses from bicarbonate losses. It occurs during rapid infusions. Bicarbonates dilute in the blood faster than can be supplemented by metabolism. The processes that maintain the carbon dioxide partial pressure are much faster, so pCO2 does not change.

Renal failure

Metabolic acidosis typically develops in renal failure. There are several disorders that affect the acid-base balance in the same direction:

- accumulation of **sulphates,**
- accumulation of **phosphates**,
- **hyperuricaemia** - uric acid behaves like an anion at a pH close to physiological,
- **bicarbonate reabsorption fails** while maintaining diuresis and tubular damage.

Metabolic alkalosis

MAL:

Metabolic alkalosis is characterized by an increase in the concentration of standard bicarbonates. In principle, this may be due to:

- losses of an anion, usually chlorides or proteins, which are compensated in the ionogram by the addition of bicarbonates;
- an increase in the concentration of a cation, most often sodium.

Alkalosis from anion losses

Hypochloraemic alkalosis
: It is accompanied, for example, by prolonged vomiting, in which a large amount of chloride anion is lost through vomiting gastric juice. Diuretics may be another cause of hypochloraemic alkalosis.

Hypoproteinemia
: Proteins behave like polyanions, so the decrease in their concentration is also compensated by the addition of bicarbonates. Typical examples may be liver proteosynthesis failure, protein loss in nephrotic syndrome, or malnutrition.

Hypernatremic alkalosis

It is most often the result of hyperaldosteronism. Some adrenal tumors or other tumors producing this hormone lead to *primary hyperaldosteronism*. *Secondary hyperaldosteronism* is more common as a consequence of liver failure, as aldosterone is broken down in the liver. Another cause of secondary hyperaldosteronism may be overactivation of the renin-angiotensin-aldosterone system.

Elevated aldosterone levels cause the kidneys to retain more sodium, which is compensated in the ionogram by the addition of bicarbonate anion. In addition, sodium is being saved at the expense of increased urinary potassium and proton losses, leading to further deepening of alkalosis.

Sodium retention is also caused by corticosteroids, so metabolic alkalosis is accompanied by Cushing's syndrome.

Alkalosis from an excess of other cations

Rarely, metabolic alkalosis can be caused by an excess of another cation, such as ionized calcium. It occurs, for example, in bone tumors (multiple myeloma, metastases of breast cancer, prostate cancer, etc.). During the breakdown of bone tissue, a large amount of Ca2 + as well as HCO3- is released.

Liver failure

Liver failure is typically accompanied by metabolic alkalosis. Its causes are:

- hypoproteinemia in proteosynthesis failure;
- secondary hyperaldosteronism with sodium retention - aldosterone is normally broken down by the liver;
- slowing down the ureasynthetic cycle - a metabolic process that produces a proton for each molecule of urea formed.

Mixed disorders of the acid-base balance

In practice, **a combination of several acid-base disorders** can be encountered. **The combination of metabolic acidosis with metabolic alkalosis** is especially important: in the examination for acid-base disorder according to Astrup, the individual parameters may be normal or only slightly deviated. Therefore, the combined acid-base balance disorder may not be recognized or may be underestimated. At the same time, a treatment that affects one of the disorders can cause the other one to prevail quickly. This can lead to a sudden change in the pH of the internal environment and a severe metabolic breakdown.

Conditions leading to combined acid-base balance disorders are not uncommon. Typical examples are:

vomiting and diarrhea
: vomiting leads to hypochloremic alkalosis, diarrhea to acidosis due to bicarbonate losses

prolonged vomiting
: hypochloraemic alkalosis caused by vomiting is combined with fasting ketoacidosis and lactic acidosis due to insufficient tissue perfusion in hypovolemia

hepatorenal failure
: combines hepatic metabolic alkalosis with renal acidosis

liver failure with respiratory insufficiency
: severe hypoproteinemia in liver failure leads to pulmonary edema, hypoxia causes lactic acidosis

renal failure with nephrotic syndrome and severe hypoproteinemia
: renal acidosis from sulfate and phosphate accumulation is accompanied by alkalosis in hypoproteinemia

Correction and compensation of acid-base balance disorders

When the acid-base balance fails for any reason, the body begins to strive to maintain the pH of the internal environment. Ii fights it with another disorder that deflects the pH in the opposite direction. We distinguish two groups of such mechanisms:

- **Compensation** means that in the case of a metabolic disorder, the pH of the internal environment is maintained by changing respiration. For example, metabolic acidosis is compensated by respiratory alkalosis; the patient will breathe deeply ("Kussmaul's breathing").

- We talk about a **correction** only in the case of metabolic disorders: one metabolic deviation is corrected by another. For e.g. a patient with liver failure (and therefore metabolic alkalosis) will excrete more bicarbonate in the kidneys and the urine will less acidic.

The development of correction and compensation mechanisms takes some time. Respiration changes almost immediately after the disorder occurs. Respiratory compensation mechanisms then deepen, reaching a maximum in about 12 - 24 hours. Compensation and correction at the kidney level are much slower - some transport mechanisms have to be re-regulated, which often requires protein synthesis. These mechanisms reach their maximum in five days.

> *When arriving at high altitudes, it is necessary to count about five days of acclimatization. The cause of the alpine disease is hyperventilation, which the body uses to deal with hypoxia. However, strenuous breathing does not improve the oxygen saturation of hemoglobin too much - the O_2 partial pressure in the surrounding atmosphere is too low for that, but it does lead to respiratory alkalosis. Alkalosis and ionic imbalance are the causes of alpine disease, which include cerebral edema, pulmonary edema and tachycardia. Acclimatization consists of over-regulation of the kidneys - basically the development of metabolic acidosis, which lasts the mentioned 5 days. It can be accelerated by an intake of large amounts of fluids, as it increases urinary bicarbonate losses. As a part of the treatment of alpine disease, acetazolamide, a carbonic anhydrase inhibitor that reduces urinary acidification, is sometimes recommended (however, recent work considers acetazolamide to be ineffective).*

Principles of treatment of acid-base balance disorders

Treatment of metabolic acidosis

The treatment of more severe metabolic acidosis is usually the administration of **sodium bicarbonate**, either parenterally as a part of complex infusion therapy or orally. The advantage of enteral administration is that the body can regulate the absorption of bicarbonates, so there is no need to worry about excessive alkalization. On the other hand, this pathway is slower and less effective, and resorption may be impaired in more severe cases.

Milder and chronic metabolic acidosis is often treated by the administration of organic acids and their salts. Bicarbonates are then formed by their metabolism in the citrate cycle. This is under the condition of good liver function. The most commonly used are lactate (eg Ringer's infusion solution with lactate) and citrate (eg in oral rehydration solutions used to treat diarrhea).

If acidosis and acidemia have lasted for a long time, the pH of the internal environment must be adjusted slowly.

> *It should be remembered that the respiratory center responds to pCO_2 as an acid-base sensor: CO_2 diffuses from the blood into an environment rich in HCO_3^-, so a buffer is formed. Its pH depends on the current pCO_2. Nerve endings respond to the acidity of the environment. In case of acidosis that lasts several days, the respiratory center is over-regulated. A sudden alkalization of the internal environment could lead to the respiratory center behaving as it does in hypocapnia - hyperventilation: it would lead to respiratory depression, and eventually to respiratory insufficiency.*

Treatment of metabolic alkalosis

The treatment of metabolic alkalosis is most often based on the administration of physiological saline. While in the blood the concentration of sodium cations is higher than the concentration of chloride anions, in saline both ions are in a ratio of 1: 1. By administering it, we supply the body with an excess of chlorides. This displaces the bicarbonates in the ionogram and corrects the alkalosis.

References

Related articles

- Relationship between acid-base balance and ionogram
- Laboratory evaluation of acid-base balance
- Acid-Base balance