

Serum osmolality

Osmolality is the total amount of osmotically active particles dissolved in a kilogram of solvent (usually water). It depends on the number of particles in the solution regardless of their size. It can therefore also be defined as the total molal concentration of all dissolved components of the solution. **Blood osmolality** is mainly determined by the amount of ions, glucose, and urea in a kilogram of plasma.

A related quantity is osmolarity, indicating the total substance amount of dissolved particles in one liter of solution (it is therefore the total molar concentration of dissolved components).

The effective osmolality of body fluids considers only glucose and major ions (sodium, potassium, chloride). It does not include substances that quickly diffuse through semipermeable membranes (urea), and substances that do not occur physiologically in the body (medicines, xenobiotics).

If the concentration of osmotically active particles is different on both sides of a semipermeable membrane (e.g. the plasma membrane of cells), there is a different osmotic pressure on each side of the membrane. It is often compensated by moving water. The result is swelling or, conversely, cell dehydration.

A solution whose real osmolality is higher than the real osmolality of another (compared) solution is called **hyperosmolal**. In the body, the osmolality of solutions is most often compared with the real osmolality of plasma, i.e. solutions that have a higher real osmolality than plasma are referred to as **hyperosmolal**. Solutions with a lower osmolality than the comparison solution (usually blood plasma) are referred to as **hypoosmolal**, solutions with approximately the same osmolality are **iso-osmolal**.

Reference limits

- serum osmolality : 275–295 mmol/kg H₂O
- urine osmolality : 50–1200 mmol/kg H₂O

Note: urea can diffuse relatively freely across the plasma membrane, so it practically has no effect on the formation of osmotic gradients on the plasma membranes of cells. However, it cannot pass through membranes at unlimited speed. A rapid decrease in the concentration of urea in the plasma during, for example, hemodialysis can therefore lead to the so-called dysequilibrium syndrome, when due to the relatively slow diffusion of urea across the blood-brain barrier, a difference in osmotic pressures arises, leading to brain edema.

A change in glucose concentration in a certain compartment always has a fundamental effect on the effective osmotic pressure. The rise in glycemia thus leads to a rapid movement of water from the intracellular space, and thus, on the one hand, to cellular dehydration, and on the other hand, to the dilution of extracellular fluids. It is reported that for a rise in blood glucose of 5.5 mmol/l, natremia will decrease by 1.5 mmol/l.

Determination of urine osmolality will allow assessment of the concentrating function of the kidneys. In oliguria, this parameter will help decide whether the cause is renal or prerenal.

Osmolality regulation

Regulation of osmolality is carried out using antidiuretic hormone (ADH).

Changes in osmolality are sensed by osmoreceptors in the hypothalamus. The hypothalamus controls the secretion of ADH. the effect of ADH increases the reabsorption of water in the distal kidney tubule. A 1% change in osmolality causes a 100% rise in plasma ADH concentration.

Osmolality calculation, osmolal window

- Osmolality is measured in laboratories with osmometers, usually on the cryoscopic principle (an increase in the osmolality of a solution leads to a decrease in the freezing point). **Estimates of osmolality** are also used:

osmolality = 2 Na + + S-urea + S-glucose

effective osmolality = 2 Na + + S-glucose (mmol/kg) We refer to the difference between the actual serum osmolality and its calculated estimate as **the osmolal window (osmolal gap)**:

osmolal window = S-osm measured – S-osm calculated. The osmolal window mainly arises if foreign substances are present in the serum, which the osmolality calculation does not take into account:

ethanol

23 mmol/kg = 1 g/l = 1 ‰; osmolal gap (mmol/l) · 0.043 = S-ethanol (g/l = ‰); 1 ‰ of ethanol increases the osmolality by 23 mmol/l;

ethylene glycol

18 mmol/kg = 1 g/l;

mannitol

in anti-edematous treatment, the goal is an osmolal window of 25–35 mmol/kg.

Plasma hyperosmolality [edit | edit source]

An increase in plasma osmolality above reference limits is referred to as **plasma** hyperosmolality (hyperosmolality in the narrower sense of the word).

Causes

- water loss, acute catabolism, diabetic coma, burns , sepsis , intoxication, diabetes insipidus , drowning in salt water;
- movement of water into cells.

Clinical picture

- Metabolic encephalopathy – non-specific neuronal functional disorders (from mild disorders to delirium and coma), arises mainly as a result of the shriveling of ganglion cells. The development of the condition is accompanied by confusion and hallucinations. Especially in elderly patients, it can be confused with dementia.
- After 24-48 hours, metabolic changes occur in CNS cells that lead to normalization of their volume.

Plasma hypoosmolality

Causes

- **metabolic response to trauma, excess water (in case of inability to excrete it by the kidneys), replacement of fluid losses with water, chronic catabolism (hypoproteinemia), inappropriate secretion of ADH (SIADH in head trauma , barbiturates , etc.), poorly administered parenteral/enteral nutrition .**

Clinical and laboratory picture

- **brain edema ;**
- **even in this case, after 24-48 hours, metabolic changes occur in the cells of the CNS, which lead to the normalization of their volume;**
- **headache , epileptic paroxysms , muscle spasms, nausea, apathy ;**
- **low total serum protein;**
- **hyponatremia.**

Correction of osmolality disturbances

Correction of more severe osmolality disturbances is a major clinical problem. Incorrectly conducted therapy leads to severe neurological symptoms and can end fatally. In general, it can be said that the correction of osmolality disturbances must be slow in order to avoid rapid movements of water between compartments and ionic imbalances. Initially, effective treatment should change serum osmolality by 0.5–1 mmol/kg per hour. In 24 hours, serum osmolality should not change by more than 8–12 mmol/kg . Only in acute conditions (lasting less than 48 hours) is the procedure more aggressive, especially when it comes to hyponatremic hypoosmolality. In that case, the osmolality increases by 5 mmol/kg during the first one to two hours and then by 1–2 mmol/kg per hour .

CNS demyelination is described as a complication of hypoosmolal conditions (pseudobulbar paralysis, weak quadriplegia, ...); it is uncertain whether this is a consequence of the hypoosmolality itself or its rapid adjustment.

Blood sampling for osmolality testing

- - Heparinized plasma or serum.
 - Do not freeze, processing within 4 hours of collection.
 - Due to the strict requirements for analytical accuracy (coefficient of variation up to 0.4%), it is recommended to measure in triplicate .

Urine collection for osmolality testing

- - 1. morning urine.
 - Deliver to the laboratory within 1 hour at the latest.
 - If osmolality is determined in urine collected for 24 hours, the urine must be stored in a refrigerator at a temperature of 2–8 °C, not frozen.

Links

Related Articles

- - Urine osmolality

References

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