Sodium chloride

CAS: 7647-14-15
MF: NaCl
FW: 58.44
Solubility: dissolves easily in water (36 g/100 ml, at 20°C).

Major use
Sodium chloride (NaCl) is an essential food additive; daily consumption for adults varies between 2 and 10 g.

Human toxicity
High NaCl intake without corresponding intake of water can lead to hypernatremia: the ratio of sodium to water exceeds normal, and both the plasma sodium concentration and the total body sodium increase. Consequently, the balance of extracellular and intracellular osmolality is disturbed, which can result in several clinical symptoms, like excessive thirst, weakness, headache, irritability, pulmonary edema, hypertention, tachycardia, and also coma at high NaCl concentrations [1].

Chloride, the other component of NaCl, is also essential for water balance and for plasma osmolality, therefore abnormalities in sodium metabolism are accompanied by abnormalities in chloride metabolism [1].

Acute poisoning with NaCl occurs often in small children due to errors in formulating infant diet (use of salt instead of sugar), or due to administration of excess salt as a means of child abuse.

Salt intoxication and death also have been described when NaCl was used as an emetic [1].

Therapeutic doses of NaCl are in the range of 1 to 2 g three times daily, orally with food, or as a solution [2].

Ingestion of 0.5 to 1 g/kg will be toxic in most patients [1]. The maximum tolerated sodium intake in adult is about 15 g [2]. The estimated fatal amount of salt is about 1 to 3 g/kg [1].

When serum sodium ranges between 150 and 160 mEq/l (1 mEq, milliequivalent, is equal one mM of sodium, 23 mg; one gram of NaCl contains 17.2 mEq of sodium), which corresponds to 9-10 g/l of NaCl, the central nervous system (CNS) symptoms are common and seizures occur in approximately 10% of patients [2].

In one fatal case in an adult, with an acute oral overdose of NaCl (unknown amount, in combination with a large amount of jam), the concentration of sodium in serum was 11000 mg/l [3]. The minimum clinically measured acute lethal serum concentration of sodium, based on the data from several handbooks, was 11000 mg/l [4].

Kinetic data
Absorption: NaCl is rapidly and completely absorbed from the gastrointestinal tract. Sodium is absorbed mainly from the jejunum and ileum [1].

The volume of distribution (Vd): 0.64 l/kg [5].

Time to peak blood concentration: 5 h (at the acute poisoning) [5].

Elimination half-life: not reported [5].

Blood-protein binding: none [5].

The passage of blood-brain barrier is restricted [5].

Metabolism and excretion

Sodium maintains osmotic stability and is responsible for greater than 90% of the osmolality of extracellular fluid [2].

Extracellular concentration of Na^+ is about 150 mM, whereas its intracellular concentration is much low, less than 10 mM. It is opposite with K^+: extracellular concentration is between 120 and 160 mM, and intracellular concentration is less than 4 mM [4].

Sodium is a part of active transport system, the Na^+ and K^+ "pump", which utilizes metabolic energy to transport Na^+ out of the cell and K^+ into it, promoted by the enzyme Na^+K^+-ATPase.

Excretion of sodium occurs in the urine, sweat, and feces [1].

Toxicological mechanism

NaCl poisoning impairs the kidney ability to excrete excess of salt. Vacuolization of renal tubular cells and acute tubular necrosis may occur.

The most serious consequences of acute poisoning with NaCl are seen in the CNS due to hypernatremia. The damage of brain cells occurs when they are dehydrated following the acute osmotic shift of intracellular fluids to the extracellular space [1, 2]. The loss of brain water can lead to a rapid shrinkage of brain volume, and in severe cases – to intracerebral hemorrhage [6].

Target organs: CNS (histopathological lesions), lungs, kidney, vascular system [5].

References

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