Hypertonic Saline (3% Sodium Chloride)

Hypertonic saline is an osmotic agent uses to reduce the effects of secondary brain injury in patients with traumatic brain injury (TBI).

It is known as hypertonic saline because its concentration of sodium is higher than the physiologic concentration of plasma (0.9%).

This solution is given during the acute phase of the management of head injury because it is safe, inexpensive and has manageable side effects.

It can be given during resuscitation, via continuous infusion, or as a bolus dose.

The common clinically used preparations are 2%, 3%, 5%, 7%, and 23% sodium chloride.

3% saline contain 513 mmol/l of Na+ and Cl each, osmol of 1026 mOsm/l; pH 5.0

What is Secondary Brain Injury

Secondary brain injury is a pathological cascade that may occur withing a period of the hours and days after a primary traumatic head injury. This may be attributable to factors associated with cerebral ischemia that is as a result of cerebral hypoxia, systemic hypotension, or relative hypoperfusion due to intracranial hypertension.

Secondary injury can lead to development of cerebral edema and intractable increased intracranial pressure (ICP).

The resultant cerebral edema is treated with surgery and medications (osmotic agents such as mannitol and hypertonic saline).

Indications of hypertonic saline

Hypertonic saline is indicated in;

Management of c**erebral edema** and raised intractranial pressure (e.g. head injury),

Treatment of h**yponatraemic seizures (**severe symptomatic hyponatremia)

Resuscitation of patients with hypovolemic shock.

Mechanism of action of hypertonic saline.

Hypertonic saline works principally by exerting an osmotic effect. Due to its higher concentration of sodium and lower concentration of water when compared to plasma, it draws fluid out of the swollen (edematous) cerebral tissue.

Once hypertonic saline is administered parenterally, it increases the plasma osmolarity. This increase makes plasma to be hypertonic when compared to cerebral tissue therefore creating an osmotic gradient for drawing of water passively from the brain tissue via the process of osmosis.

This reduction in water content from the injured brain tissue reduces the mass effect controlling increased intracranial pressure eventually combating the cerebral edema and secondary brain injury.

Hypertonic saline is an effective plasma expander therefore it is used in hemodynamic stabilization of patients with hypovolemia. Volume expansion improves blood pressure and cerebral perfusion pressure leading to better brain oxygenation and reduced risk for secondary damage.

Decreasing edema in the vascular endothelium of injured tissues lowers vascular resistance, allowing more blood to flow through the vessels. Thus, it modulates the hypoperfusion often seen in secondary brain injury.

Hypertonic saline also has neurochemical properties whereby after brain trauma, neuronal membranes may become destabilized, and the neurochemical

environment can be disrupted. As a result, detrimental excitatory amino acids accumulate, leading to eventual cell death. Hypertonic saline is known to normalize neuronal cell membrane modulating this process. It does so by restoring normal electrolyte and neurotransmitter levels in brain cells, and by restoring normal cell volumes. Thus, HTS can limit secondary injury from neurochemical changes

It also counteracts hyponatremia in the brain. Brain hyponatremia results after traumatic brain injury can lead to cerebral edema, ischemia and extracellular volume depletion. The edema can all result in dangerous increases in intracranial pressure.

Hypertonic saline (HTS) helps to counteract the effects of hyponatremia by increasing serum sodium levels.

Hypertonic saline vs mannitol

When compared with mannitol;

1. Hypertonic saline is as effective for the treatment of raised ICP intraumatic brain injury as mannitol.
2. It has less “rebound” intracranial pressure (ICP)
3. With hypertonic saline there is o obligatory osmotic diuresis (plasma volume preserved/expanded)
4. Mannitol may be nephrotoxic
5. Hypertonic saline (3% NaCl) is reno-protective.
6. In monitoring osmolality; For 3% NaCl one can use plasma sodium levels whereas for mannitol need to infer osmolar gap monitoring.

Preparation and Dosage

Dose of 3% NaCl (or pre-made 2.7% NaCl when available)

In the management of cerebral oedema due to traumatic brain injury or DKA, the standard dose is **3-5 mls/kg infused** over 10–20 minutes.

For hyponatremic seizures control, aliquots of **1ml/kg hypertonic saline are used** to raise the **Na to greater than 125 mmol/L**

The same dose is used even for the pre-made 2.7% NaCl solutions

The dose is then repeated as clinically indicated

When 3mls/kg of 3% saline are used, there will be an **increase plasma sodium by approximately 2-3 mmol/L but the** increase may be greater if a large diuresis occurs and whenever in doubt you will need to check plasma sodium.

Administration

HTS can be administered via continuous infusion for acute head trauma care.

The HTS infusion rate can vary from 30 ml per hr to upwards of 150 ml per hr.

The rate is adjusted according to serum sodium levels.

The HTS solution can either supplement or replace the maintenance intravenous solution, depending on the patient's electrolyte levels and fluid requirements.

If the NaCl concentration is greater than 2%, HTS must be administered through a central line. It cannot be given peripherally, because HTS in concentrations of 3% or higher can cause local vascular irritation.

HTS would also be harmful to local tissues if the intravenous site became infiltrated.

The presence of HTS in the tissues of the arm and hand can cause cells of these tissues to become extremely edematous as fluid is pulled in. This dangerous edema can cause tissue damage and even lead to necrosis.

Patients receiving continuous HTS infusions should have their serum sodium levels checked at least every 6 hr.

The main objective of continuous HTS therapy is to provide an optimal osmolar gradient while avoiding the dangerous effects of hypernatremia. Maintaining serum sodium levels of 145 to 155 mmol/L is likely to achieve this goal.

Serum sodium levels should be maintained no higher than 155 mmol/L. Higher levels are dangerous. Patients with serum sodium levels higher than 160 mmol/L are at increased risk for treatment-related renal failure, pulmonary edema, and heart failure.

If serum sodium levels remain above 160 mmol/L for more than 48 hr, the risk of these problems increases even more.

Precautions

This solution must be administered slowly and preferably with central venous line because it carries risk of causing phlebitis, necrosis, hemolysis.

Furthermore, if serum sodium levels climb beyond 160 mmol/L, patients are at risk for seizures Serum osmolarity levels should also be monitored.

HTS is an osmolar agent, and it directly affects serum osmolarity levels.

These levels, which do not change as rapidly as serum sodium levels, should be checked every 12 hr while patients are receiving continuous HTS infusions. The target serum osmolarity is less than 320 mOsmol/L.

At higher levels, patients are at increased risk for treatment-related renal failure.

HTS needs to be administered at a carefully controlled rate.

Like other critical infusions, HTS should be run on a pump. HTS is not compatible with all medications.

Transfusions of blood or blood products cannot be given through the HTS line. These products must be given with normal saline, because the highly concentrated HTS solution could cause lysis of red blood cells.

On the other hand, giving HTS may cause some patients to become hypervolemic.

Nurses must carefully observe indicators of fluid status, such as intake and output, patient weight, and available hemodynamic values. Chest X rays should be obtained daily to assess for signs of pulmonary edema (Qureshi & Suarez, 2000).

Nurses need to exercise special caution when administering HTS to older adults via continuous infusion.

Normal age-related changes as well as concomitant renal, cardiac, and pulmonary illnesses can put older adults at increased risk for complications of HTS therapy. Older adults may develop treatment-related complications more rapidly than younger adults.

Older adults' serum sodium levels and fluid status should be carefully monitored to prevent or minimize these problems

Side effects

The most serious potential complication is central pontine myelinolysis (CPM). This occurs when serum sodium levels rise quickly in minutes. This syndrome, which is characterized by a rapid and irreversible demyelination of the pons, is manifested by a decreased level of consciousness and severe quadriparesis.

These symptoms are difficult to immediately identify in patients with severe TBI, although the

consequences of CPM would be devastating.

Careful monitoring is necessary to avoid rapid rises in serum sodium.

Gradual changes in serum sodium, no greater than 1020 mEq/L per day, are recommended.

As another precaution, HTS should not be started if serum sodium levels are lower than normal. Patients with hyponatremia should be treated with normal saline until their serum sodium levels normalize; then HTS can be started