Hyperosmolar therapy for intracranial hypertension

see Hyperosmolar therapy for children.

Hyperosmolar therapy has been the keystone of medical interventions used to control intracranial hypertension.

As early as 1783, Monro, Kellie, and other investigators advanced the notion that the volume of the brain is constant.

The landmark work of Weed and McKibben disproved this long-held dogma when they demonstrated dramatic changes in the volume of the brain resulting from administration of hypertonic or hypotonic intravenous solutions. Since that time, intravenous administration of hyperosmolar agents has become routine in the management of intracranial hypertension and herniation syndromes. However, the optimal agent, their optimal means of administration (i.e., dose and bolus vs. continuous infusion), and their precise mechanisms of action continue to be investigated.

In 1919 Weed and McKibben reported that hypertonic fluids could lower intracranial pressure and shrink nervous tissue ¹⁾.

Shortly thereafter Fay reported on "the treatment of cerebral trauma, by methods of dehydration" using intravenous hypertonic sodium and magnesium solutions ²⁾.

It was not until the early 1960s, following the introduction of ICP monitoring in head injury, that mannitol came into more widespread use $^{3)}$ and turn into the agent of choice.

Recently, hypertonic saline has challenged mannitol's role as the preferred osmotic agent ⁵⁾.

A recent pro-con debate on osmotic therapy provides a complete summary of the argument for and against its use ^{6) 7)}.

Single doses of 3 ml/kg of 20% mannitol and 3% hypertonic saline (HS) are safe and effective for intraoperative brain debulking during elective supratentorial craniotomy, but less effective in patients with pre-existing mass effect and midline shift ⁸.

Hyperosmolar therapy, using either hypertonic saline (HTS) or mannitol (MT), is considered the treatment of choice for intracranial hypertension.

Results do not lend a specific recommendation to select hypertonic saline or mannitol as a first-line for the patients with elevated ICP caused by TBI. However, for the refractory intracranial hypertension, hypertonic saline seems to be preferred ⁹⁾.

Complications

Hyperosmolar agents have been postulated to impair coagulation and platelet function.

Use of 3% HTS and 20% MT for the control of ICP did not significantly affect patients' coagulation function. Therefore, hyperosmotic solution is safe and does not increase the risk of intracranial rebleeding $^{10(11)}$.

Mannitol and hypertonic saline in equiosmolar concentrations produced comparable effects on ICP reduction, brain relaxation, and systemic hemodynamics ¹²⁾.

Case series

see Hyperosmolar therapy for intracranial hypertension case series

1)

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