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Mannitol Versus 3% NaCL for Management of Severe Pediatric Traumatic Brain Injury

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ABSTRACT

The study purpose was to investigate the relationship between the type of hyperosmolar therapy used in treating elevated intracranial pressure and the outcome of children with severe traumatic brain injury. Two outcomes were investigated: length of stay in the intensive care unit and disposition status at discharge from hospital. Children who received mannitol had the shortest length of stay and the highest mortality rate of 80%, whereas the group who received sodium chloride 3% had the longest length of stay in the intensive care unit. The group who received combined therapy of mannitol and sodium chloride 3% had the lowest mortality rate, which may suggest better modalities to manage increased intracranial pressures.

Keywords: 3% sodium chloride, children, hyperosmolar therapy, intracranial pressure, mannitol, traumatic brain injury

Traumatic brain injury (TBI) accounts for more than 600/100,000 causalities yearly[1] with high morbidity and mortality.[2] Epidemiologic studies estimate that 1.5 million head injuries occur yearly in the United States, and sport-related injuries accounted for an additional 1.6 to 3.8 million injuries yearly.[3] Over 5 million Americans live with disabilities after suffering a TBI, which accounts for almost 2% of the US population.[3]

TBI can be classified based on the injury location and severity, ranging from a mild concussion to a severe injury that may result in disability and death. The Glasgow Coma Scale (GCS) is a tool that provides a reliable, objective assessment of neurologic status using the individual and summed best eye, verbal, and motor responses.[4] The lowest possible summed score is 3, indicating a deep coma or death; the highest possible summed score is 15, reflecting a fully awake person. The GCS characterizes the TBI severity as mild (12-15), moderate (9-12), or severe (3-8).[5]

Critically ill children who sustain a severe TBI often require multiple treatment modalities to reduce their high intracranial pressure (ICP) and minimize the insult of secondary brain injury. ICP management is a crucial part of care rendered by nurses, acute care nurse practitioners, intensivists, and neurosurgeons. The most common treatment modality used to lower elevated ICPs is hyperosmolar medications,[6] such as mannitol, 3% sodium chloride (NaCl), and 7.5% NaCl, have been used widely in clinical settings.[7,8]

SCOPE OF THE PROBLEM

Mechanisms of brain injury may be the result of a blunt, penetrating, or blast impact.[9] The injury may be in 1 specific area of the brain or a focal brain injury, such as a contusion or laceration, or it may affect multiple regions of the brain, as in concussions and diffuse axonal injuries.[4] As a result of the initial injury, secondary injuries may occur.

Both primary and secondary injuries may lead to increased ICP. With high ICP, the direct brain tissue damage impairs the cerebral blood flow and the metabolic regulation.[10] Several cellular processes increase membrane permeability, resulting in an accumulation of lactic acid and edema formation. As a result, the anaerobic metabolism fails to meet the cellular energy demands of the cell and causes

depletion of the adenosine triphosphate (energy) stores.[10] In turn, a new cascade begins wherein cells' terminal membranes depolarize and start to secrete excitatory neurotransmitters, such as glutamate and aspartate. Concurrently, an ongoing cellular calcium and sodium influx leads to a catabolic, intracellular, autodigestion process. The calcium influx activates the lipid peroxidases, proteases, and phospholipase, resulting in an increased concentration of free fatty acids and free radicals that cause more brain tissue swelling after the initial injury.[10]

Complications resulting from brain injury overwhelm all the compensatory mechanisms and further increase ICP, brain swelling, and brain ischemia.[11] Different medical modalities have been used to decrease the effect of the cerebral edema and intracranial hypertension after a severe brain injury. The use of hyperosmolar medications to reduce high ICP in animals was documented in 1919 by Weed and McKibben.[12] Several hyperosmolar medications were used including urea, glycerol, sorbitol, mannitol, and hypertonic saline (HTS). In humans, Haden[13] showed that the use of hyperosmolar therapy had similar results in ICP reduction by lowering the intracranial volume. Specifically, serum osmolality increases with the administration of hyperosmolar medications, drawing fluid out of the brain's interstitial space into the vascular space and resulting in reduced brain tissue volume, and, subsequently, lowers ICP.[14,15]

Currently, mannitol is a diuretic widely used to treat high ICP and is recommended in TBI management guidelines for adults.[7] Although the latest pediatric TBI management guidelines reported no evidence to support or refute mannitol's use in managing high ICPs,[15] HTS is included as an effective intervention for the management of high ICP in children[15]; however, there is no published guideline or protocol that delineates the timing, concentration, and/or mode of administration (bolus vs continuous intravenous) of HTS in the management of severe TBI in children.[11,14] The decision about whether to use HTS, mannitol, or a combination of HTS and mannitol is left to provider preference,[14] and pediatric patient outcomes (intensive care unit [ICU] length of stay [LOS] and discharge disposition) associated with the treatment are unclear. Therefore, the study purpose was to compare the ICU LOS and discharge disposition in children admitted with severe TBI documented by an abnormal head computed tomographic (CT) scan and ICP > 20 mm Hg who receive standard care and either no hyperosmolar medication or hyperosmolar medications (mannitol only, 3% NaCl only, or mannitol plus NaCl). The children to whom no hyperosmolar medication was administered received and responded to the first line of TBI management (sedation and cerebrospinal fluid draining via an external ventricular drain). The children who did not respond to the first-line TBI management received hyperosmolar medications.

METHODS

Design and Data Collection

After institutional review board approval from the university and the medical center, a descriptive, retrospective chart review design was used. A list of patients (N = 306) admitted to the ICU from January 2003 through January 2009 was generated from the trauma database, which includes a record of all patients with traumatic injuries presenting to the level 1 pediatric trauma center. A review of all records by 2 reviewers yielded 96 patients who met the study inclusion criteria and constituted the sample. For additional eligibility verification, the potential subjects generated from the trauma database were cross-referenced and reconciled against the hospital admissions records by the primary investigator.

The sample selection criteria included patients from age 8 to 18 who were admitted with an isolated, severe TBI (GCS score of 3-8), an abnormal head CT scan on admission, and a documented high ICP (> 20 mm Hg) for more than 5 minutes. Exclusion criteria included a normal head CT or additional trauma such as abdominal, orthopedic, multiple system, and/or nonaccidental trauma.

STUDY SETTING

The study was conducted in 2 ICUs: a surgical ICU for children older than 15 and a pediatric ICU in a level 1 trauma center in Southern California.

SAMPLE AND MEASURES

The following data were extracted from the record of each patient included in the sample: age, sex, injury mechanism, GCS score on admission to the hospital