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REVIEW ARTICLE

Management of intracranial hypertension in pediatric traumatic brain injury

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Keywords:
Intracranial Hypertension, Pediatrics, Neurosurgery, Brain Injuries, Traumatic.

Abstract

Objective: To review current management of intracranial hypertension in pediatric population, its evidence levels and possible complications of its application. **Methods:** Bibliographic review, based on PubMed data. It was reviewed 47 articles, from April 17th to 25th of 2021, matching to "pediatric TBI, pediatric HICP, HICP management in pediatrics", we included those, in Portuguese and English, upper to 1999, and excluded 22 that were about adult TBI and HICP, scales application in TBI, management of any other complications instead HICP and genetic related outcomes post-TBI. **Results:** Authors noticed that some measures to manage the intracranial hypertension are consensual among some authors, even with its low evidence level, however, some remain controversial. **Conclusion:** Even though many measures still have a low evidence level, they have shown positive results preventing a second insult that may lead to a secondary injury. However, it is needed an individual assessment to decide which measures are more suitable and offer a lower complications risk to the patient.

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INTRODUCTION

Head trauma

Early identification and management of severe head trauma is essential to reduce the risks of secondary trauma and sequelae, with emphasis on avoiding hypotension and hypoxia[1].

Although brain plasticity in children plays a protective role against traumatic injuries, head trauma is the leading cause of mortality and morbidity in the pediatric age group. The causes of head trauma vary according to age[2].

Compared to adults, children have a higher incidence of brain damage, cerebral edema, and intracranial hypertension in head trauma events. A number of elements favor this outcome. An important role is played by the influx of calcium into neurons, which is instigated by the release of excitotoxic neurotransmitters (glutamate) during the early stages of injury. The influx of calcium triggers various cellular cascades that can result in apoptosis or necrosis. Children are more susceptible to these phenomena, since apoptosis plays a physiological role during development. In addition, the pediatric brain seems to undergo greater diffusion of excitotoxic agents with more pronounced inflammatory reaction. Another factor is that the cranial structure of children is less rigid than that of adults, which favors brain distortion during trauma[2].

Intracranial hypertension

The maintenance of intracranial pressure (ICP) within a normal range relies on the maintenance of the volume within the skull, which is occupied 80% by the compartments of the encephalon, 10% by blood, and 10% by cerebrospinal fluid. An increase in the volume of any of these components forces the others to decrease in volume order to avoid changes in intracranial pressure (ICP), working as a compensation mechanism. In most cases such compensation occurs through decreases in CSF or blood volume, with 30% of the intracranial volume reduction capacity being due to CSF displacement or reabsorption. If these mechanisms are exhausted, intracranial pressure increases, perfusion decreases, and ischemia sets in[3].

Normal ICP values for newborns and infants are less than 5 mmHg; in children, normal values range between 6 and 15 mmHg; in adolescents, normal ICP values are greater than 15 mmHg. Intracranial hypertension (ICH) is characterized when the ICP is above 20 mmHg[3,4].

The difference between mean arterial pressure (MAP) and intracranial pressure is called cerebral perfusion pressure (CPP), whose values required for maintaining adequate cerebral flow are specific for each age group. Normal values for newborns and infants range between 30 and 40 mmHg; for children, they range between 50 and 60 mmHg; and for adolescents, the values vary between 60 and 70 mmHg[3].

ICH is a severe complication for children in intensive care due to head trauma associated with poor outcomes. About 40-80% of patients with head trauma develop intracranial hypertension, with a third of the cases being refractory to treatment[5,6].

Intracranial hypertension secondary to head trauma results from increased intracranial volume, either from cerebral edema, hemorrhage, or hydrocephalus. A persistent increase in intracranial pressure is a key factor in the development of secondary brain injury[7,8].

Cerebral blood flow (CBF) is greater in children due to increased cerebral metabolism, whereas systemic blood flow is lesser. Thus, hypotension may quickly cause cerebral ischemia. A drop in pressure may increase ICP in children and decrease their ability to autoregulate cerebral blood flow, a common finding in critically ill pediatric patients[2].

ICH is one of the main causes of secondary brain injury in pediatrics. It has been strongly associated with morbidity and mortality in cases of head trauma. Thus, developing a better comprehension of the physiopathology and providing adequate treatment to children with intracranial hypertension is essential to improve the outcomes of pediatric head trauma[3].

Epidemiology

Trauma is the leading cause of death in children (40% of the deaths of children between the ages of five and nine years and 18% of the deaths of children aged between one and four years). Head trauma accounts for the majority of these events (75-97% of deaths by trauma in children). Approximately three patients are left with severe sequelae for every patient that dies[9,10].

Approximately 75% of the children with severe head trauma develop intracranial hypertension. In the general population, intracranial hypertension is the cause of about 50% of all deaths related to head trauma[5,11].

A scale used to evaluate the severity of traumatic brain injury categorizes head trauma as mild, moderate, severe, or very severe. Patients with mild head trauma have a Glasgow Coma Score (GCS) between 13 and 15 points and present signs of post-traumatic amnesia for less than one hour, and no coma. Subjects in this category progress well and do not require hospitalization in most cases. Patients with a GCS between nine and 12 points have moderate head trauma, with post-traumatic amnesia lasting from one to 24 hours. Individuals with moderate head trauma are at high risk of cognitive/behavioral impairment. When head trauma is severe, the GCS resides between 3 and 8. Patients in this category have post-traumatic amnesia lasting from one to seven days, and spend more than six hours in a coma. These individuals are at great risk of developing neurologic sequelae. In very severe cases, the GCS is 3 or 4 and patients have post-traumatic amnesia lasting for more than seven days and generally develop motor, behavioral, and/or physical disability[12].

After puberty, most cases of head trauma involve females, possibly due to hormonal changes. Head trauma has no preference for specific ethnicities and a characteristic genetic locus has not been identified[13].

Manifestations and diagnosis of ICH

In children, intracranial hypertension (ICH) constitutes a larger spectrum of clinical presentations compared to adults. Among clinical manifestations, headache stands out as the most common symptom in children and adults, which, when induced by elevated ICP, may be worse in the morning and awake patients from sleep or increase when the Valsalva maneuver is performed. Headaches may be associated with other symptoms such as nausea and vomiting. Ophthalmic symptoms such as blurred vision, optic disc edema, and diplopia are also frequent in cases of intracranial hypertension[13]. Transient visual obscurations may occur unilaterally or bilaterally, for only a few seconds. These events are probably caused by transient optic nerve ischemia and are more commonly seen in older children. Early vision loss can be caused by macular edema, subretinal or peri-macular hemorrhages, or anterior ischemic optic neuropathy[14].

In younger children and infants, the most notable clinical findings include irritability, apathy, and drowsiness, in addition to other nonspecific manifestations such as ataxia, dizziness, neck stiffness, facial or limb paresthesia, seizures, pulsatile tinnitus, and facial nerve palsy. Children may also be asymptomatic or present only with papilledema during routine ophthalmologic examination[14].

Papilledema is characteristic of hypertension. If this symptom is not apparent, the diagnosis of ICH must be made with caution, since papilledema might be present and unrecognized or might not have developed yet. It may be asymmetric and in rare occasions occurs unilaterally. Loss of visual acuity is usually a late finding of ICH. Neurological examination is usually uneventful, although a few patients have facial and hypoglossal nerve palsy, neck stiffness, hyperreflexia with Babinski signs, choreiform movements, and nystagmus[13].

In post-head trauma scenarios, indirect signs of ICP increase may be present and, therefore, the risk of ICH must be considered. If signs such as the ones listed above are present, a diagnosis of ICH must be considered so that the best management for pediatric patients is introduced. Two diagnostic references can be used to this end: the modified Dandy criteria or PTCS (pseudotumor cerebri syndrome). The modified Dandy criteria base establish the diagnosis of ICH based on signs and symptoms of increased intracranial pressure, elevated lumbar puncture opening pressure, normal CSF analysis, and normal neuroimaging in the absence of localizing signs. In turn, PTCS stratifies the diagnosis of ICH into definite, probable or suggested categories. PTCS does not use clinical but imaging evidence, which, when absent, should not impede a diagnosis of ICH, since it is present in the minority of patients. There is no consensus about which of the two should be used in pediatrics[13,15].

Modified Dandy criteria:

- Signs and symptoms of increased ICP (headache, nausea, vomiting, transient visual obscuration, papilledema).
- Absence of localizing neurologic signs (unilateral or bilateral, rule out abducens nerve palsy).
- CSF opening pressure >0.25 cm H_20 with normal composition.
- Symmetric normal to small ventricles as seen in computed tomography (CT) scans.

PTCS criteria:

The diagnosis is considered "definite" if the patient meets criteria A-E; "Probable" if patient meets criteria A-D and cerebrospinal fluid (CSF) pressure is less than specified for a "definite" diagnosis.

A. Papilledema.
B. Normal neurological examination, except for cranial nerve abnormalities.
C. Neuroimaging: normal brain parenchyma with no evidence of hydrocephalus, mass, or structural lesion; no abnormal meningeal enhancement on magnetic resonance imaging (MRI), and magnetic resonance angiography for others. If MRI is not available or is contraindicated, contrast-enhanced CT can be performed.
D. Normal CSF (cerebrospinal fluid) composition.
E. High opening pressure of the lumbar puncture (≥250 mm H2O CSF in adults and ≥280 mmH2O CSF in children [250 mmH2O CSF if the child is not sedated and not obese]) in a well-performed lumbar puncture.

Diagnosis of ICH without papilledema:

In the absence of papilledema, the diagnosis following the PTCS Criterion can be made if the above B-E criteria are met and, in addition, the patient has unilateral or bilateral abducens nerve palsy[15].

In the absence of papilledema or cranial nerve six palsy, a diagnosis based on PTCS can be suggested, but not established, if the above B-E criteria is met and, in addition, at least three of the following neuroimaging criteria are met[15]:

- Empty sella.
- Posterior globe flattening.
- Distension of the perioptic subarachnoid space with or without a tortuous optic nerve.
- Stenosis of the transverse venous sinus.

Although the PTCS criteria use objective data to establish a diagnosis of ICH, subjective symptoms may be useful indicators of disease. Distinguishing between a "probable" from a "definite" diagnosis may not be clinically relevant, since it does not change management after diagnostic definition. Further research is needed to clarify the disease process in patients presenting with signs and symptoms of elevated intracranial pressure and without ocular involvement[15].

Treatment

Since ICH is a life-threatening condition potentially caused by multiple factors, treatment must cover not only the primary cause of increased intracranial pressure, but also measures to prevent or minimize secondary injury, especially

hypoxia and hypotension, which are the main causes of sequelae and death. To this end, measures to stabilize the patient's condition and control intracranial pressure must be taken[3,16].

Early assessment must be performed concomitantly with resuscitation efforts, usually within the first 5 to 10 minutes. In this first phase, vital signs are evaluated and reassessed every five minutes, avoiding, as much as possible, maneuvers that might increase ICP while following the principles of ATLS (Advanced Trauma Life Support) and the ABCDE approach until the patient is in a stable condition[17].

Once the patient is stable, a second, more detailed assessment of the situation is performed, in which a detailed description of the patient's history is obtained, thorough physical and neurological examination is performed, and initial laboratory tests and imaging exams are ordered. This evaluation helps to identify the extent of the injuries, decide whether immediate surgical intervention is needed, and choose the best course of therapy[17]. Specific factors that can aggravate ICH and require prompt treatment include venous return obstruction, respiratory problems (hypoxia, hypercapnia, airway obstruction), fever, severe hypertension, hyponatremia, anemia, and seizures[16].

To maintain control of vital parameters, several protocols stipulate measures to keep adequate CPP and ICP levels, with general and specific measures to address ICH, namely[17]:

General measures

1. Position of the head: The head must be kept in a neutral position at an angle of 30º relative to the bed. The purpose of this measure is to improve venous return and promote cerebrospinal fluid (CSF) displacement from the intracranial to the spinal compartment. The prone position should be avoided, as it leads to increased intra-abdominal and intrathoracic pressure, resulting in increased ICP as a consequence[3].

2. Hemodynamic monitoring: Continuous measurement of mean arterial pressure (MAP) and central venous pressure (CVP) is recommended via arterial and central catheters, respectively. **Hypotension** must be promptly and aggressively treated, since it is one of the main factors that worsen patient outcomes when left unchecked[3]. Fluids must be administered initially (saline solution 0.9% at a dose of 20ml/kg for 10 to 20 minutes, or saline solution with 5% albumin); if there is no response, noradrenaline (0.05-1 µg/kg/min) or adrenaline (0.1-0.5 µg/kg/min) is indicated[17]. On the other hand, **hypertension** can be tolerated when at a mild level, since in cases of refractory ICH, blood pressure must be increased even when the ICP is elevated in order to maintain an adequate CPP level. It is common to find higher blood pressure levels in patients with ICH. Treatment must be individualized. Drugs such as beta-blockers or alpha-agonists with central action are preferred, since they lower blood pressure without affecting the ICP, although they should only be prescribed if they do not alter the CPP[16].

3. Temperature control: Fever increases the metabolic rate by 10-13% per degree Celsius and is a potent vasodilator, which can increase cerebral blood flow and consequently the ICP[16]. Prolonged hypothermia, on the other hand, decreases leukocyte counts, increases the risk of infection, and causes disturbances in ventricular conduction and the coagulation cascade[3]. Moderate prophylactic hypothermia to improve overall outcomes is discouraged with a level of evidence II. Patients must be kept on a normal temperature[7].

4. Respiratory management: It is important to remember that hypoxia and hypercapnia may dramatically increase the ICP. Therefore, orotracheal intubation is recommended in patients presenting signs of ICH, a GCS equal to or less than eight, respiratory effort, hypoxia, or refractory hypercapnia. This procedure aims to maintain saturation above 92% and $PaCO_2$ close to 35 mmHg (avoid decreases below 30 mmHg and increases above 38 mmHg). Prophylactic hyperventilation must be avoided, since it may cause vasoconstriction and worsen cerebral perfusion[3]. During mechanical ventilation, low positive end-expiratory pressures (PEEP) between 3 and 5 cm H_2O are recommended so as not to impair venous return and, consequently, cause ICH. When present, anemia must be treated promptly with packed red blood cells to maintain hemoglobin levels around 10 g/dL and hematocrit at 30%; greater values may increase blood viscosity and cause reduced cerebral blood flow[17].

5. Sedation/Analgesia: Patients must be kept free of pain and agitation, since both increase the cerebral metabolic rate and the ICP. Benzodiazepines are generally used for sedation, since they have no effects on the ICP; drugs in this group include midazolam (0.05-0.3 mg/kg/hour) in association with a more effective analgesic, such as fentanyl (0.5-2µg/kg/hour)[16]. Before intubation and/or aspiration, the use of lidocaine 1mg/kg intravenous (IV) is recommended in order to avoid ICP increases[3,16]. Neuromuscular blockade has been suggested to decrease the ICP due to its many modes of action, which decrease intrathoracic pressure, facilitate cerebral venous return, and reduce muscle jerks that increase the ICP, among others. Use of benzodiazepines must be accompanied by electroencephalographic monitoring, since it may mask the manifestations of seizures[17]. Propofol infusion should be limited to 12 hours because of the risk of metabolic acidosis and hypotension[3].

6. Seizure control: Seizures increase the metabolic rate and may lead to significant ICP increases. Therefore, children with repeated seizures, previous episodes of epilepsy, evidence of severe cortical contusion or surgical wounds, prophylaxis with anticonvulsants is indicated[3]. Diphenylhydantoin or carbamazepine should be used, especially in cases of head trauma, until the patient is in a stable condition. Patients having seizures must be provided the customary treatment with a fast-acting benzodiazepine, such as diazepam (0.2-0.3 mg/kg/dose), followed by maintenance with at least diphenylhydantoin. The goal is to avoid early seizures (first

week after head trauma). It is important follow patients with electroencephalography, since subclinical seizures can only be detected by an EEG[17].

7. Fluid replacement: electrolytes and osmolarity must be regularly monitored, as well as diuresis, to keep urinary output > 1 mL/kg/hour. Fluid restriction is indicated for patients with dilutional hyponatremia; sodium levels must be maintained in higher ranges (> 140 mEq/L)[3,17].

8. Glycemic control: There still is controversy regarding blood glucose in critical neurological care. However, 34% of the children are hyperglycemic within 48 hours of a head trauma event, even without glucose infusion. Since this state increases oxidative stress, inflammation, and cytotoxicity, and alters microcirculation, glucose levels must be kept between 8-10 mmol/L. Hypoglycemia also favors cytotoxicity, apoptosis, and free radical accumulation, and must therefore be avoided[2].

9. Nutritional support: Feeding should be started early and preferably using an enteral route[3]. In a secondary analysis, Meinert et al. (2018) concluded that patients started on nutritional support within 72 hours of a head trauma event had lower mortality and more favorable outcomes[19]. Patients must be administered the equivalent to 30-60% of their basal metabolic rate. If thiopental is administered in large doses, parenteral nutrition is preferred due to gastroparesis or paralytic ileus[3].

ICP monitoring

ICP monitoring is an invasive procedure that allows the correct evaluation of the ICP and cerebral perfusion pressure. It should be performed only under specific indications, since it is not a risk-free procedure. Its purpose is to reduce the risk of cerebral ischemia and secondary damage to brain tissues[3,16].

It is indicated only to patients with a GCS of 8 or less and head trauma victims with anomalous findings in admission CT scans, such as hematoma, contusion, edema, or compressed basal cisterns; other less common indications include patients with multiple system lesions with altered levels of consciousness individuals submitted to excisions of intracranial masses. ICP monitoring may be considered in non-traumatic patients with mass effect injuries that can cause increased ICP and clinical deterioration[3,16].

The procedure consists in the insertion of an intraventricular, intraparenchymal or subdural catheter, which allows the monitoring of the ICP[3]. The intraventricular access is still the most used, since its implantation, besides allowing ICP monitoring, enables the management of ICH through CSF drainage. Its advantages include a relative low cost, the possibility of draining CSF, and the ability to recalibrate to minimize measurement errors. Disadvantages include difficulty of insertion in case of compressed or displaced ventricles, risk of infection, decreased measurement accuracy in case of obstruction of the fluid column, and the need to maintain the transducer at a fixed point on the patient's head[16].

Noninvasive methods can be offered to patients who do not meet the criteria for invasive monitoring or have contraindications such as coagulopathy or skin infection, or when the invasive procedure cannot be performed. The ideal noninvasive monitor must be reliable, safe, cost-effective, affordable, and non-operator dependent[20].

Among the noninvasive options tested in pediatrics, transcranial Doppler and sonographic optic nerve sheath diameter evaluation are the most significant methods. The first involves positioning the probe in different acoustic windows corresponding to specific locations on the skull, where bone is thinner, to study the vascular distribution of the brain and its impact on ICP. The second method, on the other hand, has as a principle the evaluation of the diameter of the optic nerve sheath, which presents an anatomical continuity with the dura mater and the subarachnoid space. When there is an increase in ICP, the sheath expands and, consequently, its diameter increases. This change is visible within seconds in an acute episode of ICP elevation, unlike papilledema, which occurs later. A measurement above 4.5 mm in children over one year of age is considered anomalous[20].

Specific measures

Measures to treat ICH must be initiated when the ICP reaches 15 mmHg in infants, 18 mmHg in children under eight years of age, and > 20 mmHg in older children and adolescents. These measures aim to keep the CPP between 40-45 mmHg in infants and younger children and 50-55 mmHg in older children and adolescents[3].

1. CSF drainage: This is the first and simplest measure to reduce ICP and improve CBF with an intraventricular catheter. It is usually performed intermittently, although it can be done continuously, using gravity, after evaluation by the neurosurgeon[16]. It is recommended to drain 3-5 mL of CSF, with reassessment of ICP[3].

2. Hyperosmolar therapy: Hypertonic saline solution (HSS) at a concentration of 3% creates an osmotic force in the presence of an intact blood-brain barrier capable of draining fluid from the brain parenchyma into the intravascular space, decreasing intracranial volume and ICP. A recommendation with level of evidence II stipulates that a bolus infusion of 3% HSS be administered at a dose ranging from 2-5 mL/kg, for 10 to 20 minutes. Continuous infusion is suggested with level of evidence III, at a dose ranging from 0.1-1 mL/Kg, for one hour. In case of risk of herniation due to high ICP, HSS can be given at a dose of 7.5-10 mL/kg. The therapeutic target for sodium, according to previously published studies, resides between 155-160 mEq/L. Values above 165 mEq/L increase the need for blood transfusion due to anemia, while serum levels above 170 mEq/L increase the rates of renal failure, thrombocytopenia, neutropenia, and ARDS (acute respiratory distress syndrome)[7]. In order to avoid the risk of pontine myelinolysis, cerebral hemorrhage, and acute kidney injury during withdrawal, decreases of a maximum of 10 mEq/L/day should be enforced[3]. Mannitol infusion, which results in increased serum osmolarity, ultimately reduces cerebral

edema. Hematocrit and blood viscosity also decrease, thus potentially increasing CBF and improving oxygen delivery to the brain. This effect, however, is transient and lasts for less than 75 minutes. When autoregulation is intact, this drug can induce vasoconstriction, which keeps CBF constant, causing a significant drop in ICP[7,17]. When there is a hypertensive peak in ICP and an urgent reduction is required, the initial dose is 1mg/kg administered within 20 minutes[3]. Although both have similar osmotic effects, HSS seems to have a more favorable effect on hemodynamic stability and immunomodulation[2]. Feng et al. (2018)[5] randomized 30 head trauma patients given 3% HSS or mannitol, and showed that HSS was more effective than mannitol at decreasing the length of ICH, with a mean of 2.11 days versus 2.95 for mannitol. However, two-week mortality was unchanged[5].

3. Barbiturates: Barbiturates effectively decrease ICP and mortality in cases of ICH refractory to conventional medications and surgery. Administration requires close monitoring due to the risks of hemodynamic instability cerebral hypoxia with oligohemia. Beneficial effects include vascular tone alterations, inhibition of lipid peroxidation and oxygen free radical formation, and suppression of cerebral metabolism[16]. However, recommendations have a level of evidence III, with information supporting the use of barbiturates, although without indication of which specific drug and regimen of administration should be adopted for the treatment of refractory ICH (7). Prophylactic administration is not recommended[16].

4. Hyperventilation: Aggressive hyperventilation ($PaCO_2$ < 30 mmHg) is indicated for the treatment of refractory ICH, provided there is no sign of low CBF. It must be accompanied by monitoring of cerebral oxygenation and duration should not exceed two minutes. Its recommendation has a level of evidence III. Studies have shown a frequency of regional ischemia of 59.4% when $PaCO_2$ is between 25-35 mmHg, and 73.1% when $PaCO_2$ < 25 mmHg. When normocapnia was maintained, frequency of ischemia dropped to 28.9%[7].

5. Decompressive craniectomy: In this surgical procedure, a part of the skull cap is removed to create a window that allows pressure to decrease[17]. Although the procedure is not recommended as standard therapy, studies indicate better long-term functional outcomes for about 78-92% of patients undergoing decompressive craniectomy when it is performed within less than 48 hours for selected cases (GCS > 3)[2,3]. Tang Z et al. (2018)[21] analyzed 36 patients (23 boys and 13 girls) with a mean age of seven years, mostly victims of falls, assigned a mean GCS of 6[4-8]. The authors found that on day 14 after early (< 48h) decompressive craniectomy, mortality was 38.9%[21]. Although still a controversial procedure, it has been considered more widely. Therefore, it should be discussed as an option in the early management of uncontrolled ICH[18].

6. Mild hypothermia: Routine induction of hypothermia is not currently indicated. Studies have shown that, even when a drop in ICP was evidenced during hypothermia, no beneficial effects on patient neurologic outcomes were observed[17]. Tasker et al. (2017)[22] included seven pediatric centers in their study and compared hypothermia versus normothermia based conventional and Bayesian meta-analyses. They found no difference in mortality between hypothermia and normothermia in conventional meta-analysis. Bayesian meta-analysis showed a reduction in the relative risk of death of more than 20% in hypothermia compared to normothermia, but it provided a framework for decision making wrapped in a scenario of uncertainty. Therefore, further studies are needed to understand the clinical skepticism and optimism about the effects of hypothermia[22]. Safety recommendation, with level III evidence, stipulate that if moderate hypothermia (32-33oC) is performed, rewarming should be done at a rate of 0.5-1°C within 12-24h to avoid complications[7].

7. Corticosteroids: To date, studies have not shown that corticosteroids decrease ICP or improve prognosis[16]. The CRASH trial demonstrated a significant increase in the risk of death from 22.3% to 25.7% in patients given methylprednisolone for 48 hours. Therefore, use of corticosteroids is not recommended[17].

All these efforts are aimed at reducing morbidity and mortality associated with pediatric head trauma, which still remain higher in children compared to adults, especially in infants.

Treatment complications

The treatment of intracranial hypertension involves a number of precise approaches to achieve adequate management of the condition, however these therapies are not without risk.

ICP monitoring

The rates of complications caused by invasive ICP monitoring are very high. They include infection and intracranial hemorrhage, with incidence ranging from 7% to 11% and 15% to 20%, respectively[20]. Use of antibiotic-coated catheters has decreased the risk of infection in some cases. Other complications include catheter malfunction, obstruction, and malposition[16]. New noninvasive monitoring approaches have been sought to avoid complications, but none is equivalent to current invasive methods[20].

Hyperosmolar therapy

Hyperosmolar therapy is considered the standard treatment for ICH. Methods include mannitol and 3% hypertonic saline solution[23].

HSS proved to be more effective in reducing cerebral edema compared to mannitol. However, HSS has been associated with increased prevalence of complications such as liver and kidney failure, coagulopathy, immune dysfunction, ARDS, and increased need for transfusions when serum sodium levels were sustained above 165 mEq/L[24].

Mannitol may draw fluid into the CNS and aggravate vasogenic edema. Therefore, withdrawal, when indicated, must be performed gradually to prevent a rebound effect[16].

Barbiturates

In some cases, barbiturates are used to induce coma in children with refractory ICH. However, some complications such as hypotension, hypokalemia, respiratory depression, negative inotropism, infection, and liver and kidney dysfunction have been observed. In cases of hypotension caused by barbiturates, the first choice of treatment is volume replacement[16].

Decompressive craniectomy

The main complications described in decompressive craniectomy were hydrocephalus, ipsilateral hemorrhagic edema, subdural hygroma, contralateral subdural effusion, and hydrocephalus[2,16].

Lumboperitoneal *Shunt*

The placement of a lumboperitoneal shunt may cause complications such as infection, excessive drainage, iatrogenic Chiari I malformation, dislocation, and malfunction[25].

In view of the potential complications, the indication of each therapeutic approach must be precise and the effects triggered by them must be anticipated so that they are minimized if they appear.

CONCLUSION

The objective of the measures presented in this paper is to prevent imbalances that might cause secondary injury, which in turn may worsen the outcomes of pediatric patients. Although most of the measures have a low level of evidence and require further studies, their application has produced positive results in the treatment of ICH after head trauma in the pediatric population.

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