Vol 2 | Issue 1 | Pages 108-112

Journal of Head and Neck Surgery

ISSN: 2689-8713

Review Article

DOI: 10.36959/605/551

Approach for Emergency Management Patients with Increased Intracranial Pressure

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Abstract

Background: Traumatic brain injury (TBI) is a commonly medical problem also associated with increased intracranial pressure (ICP) induced by intracranial hematoma and/or cerebral edema. Therefore, we aim to look into the common pitfalls that both medical students and new physicians face in the recognition, diagnosis, and increased ICP management.

Targeted population: Increased ICP patients who are requiring urgent management in the ED, with Emergency Physicians for teaching approach protocol.

Aim of the study: Appropriate assessment and priorities for treatment the increased ICP patients by training protocol to Emergency Physicians. Based on patients' causes of increased ICP.

Methods: Collection of all possible available data about the TBI with increased ICP in the Emergency department. By many research questions to achieve these aims so a Midline literature search, PubMed, Scopus and etc, from a reputed database. Were performed with the keywords "critical care", "emergency medicine", "principals of resuscitation in ICP", " intracranial hematoma". Literature search included an overview of recent definition, causes and recent therapeutic strategies.

Results: All studies introduced that the initial diagnosis of TBI with increased ICP and their therapy is a serious condition that face patients of the emergency and critical care departments.

Conclusion: The application of the step-wise, three-tiered approach to the management of increased ICP, as it utilizes various treatment strategies to target different pathophysiological mechanisms.

Keywords

Emergency physicians, Intracranial pressure, Skill approach

Introduction

Traumatic brain injury (TBI) is a commonly encountered medical problem [1]. A major concern associated with head injuries is the management of intracranial pressure (ICP), a resulting factor of a TBI which facilitates into intracranial hematoma and/or cerebral edema. These conditions have adverse effects on the brain, and the immediate management and relief of intracranial hypertension (ICH) are crucial in avoiding permanent neurological damage or even death [2].

The Monro-Kellie hypothesis holds that the total intracranial volume is made up of brain tissue, cerebral spinal fluid (CSF), venous blood, and arterial blood. Cerebral blood flow (CBF) remains constant under normal conditions via cerebral auto regulatory mechanisms over a range of blood pressures. When one compartment is increased, by a hematoma for example, there must be a compensatory decrease in another compartment in order to prevent ICH. Cerebral perfusion pressure (CPP) is a surrogate for CBF. CPP is defined as mean arterial pressure (MAP) minus intracranial pressure (ICP). A decrease in CPP implies a decrease in CBF, although this association is not perfect. Decreased CBF ultimately leads to ischemia and hypoxia and worsening of the initial brain insult [3].

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Accepted: December 28, 2020

Published online: December 30, 2020

Citation: Elbaih AH, Ahmed OT (2020) Approach for Emergency Management Patients with Increased Intracranial Pressure. J Head Neck Surg 2(1):108-112

Scope of the problem

Trauma is the leading cause of death in individuals aged 1 to 45 years, with TBI being responsible for most these deaths (more than 50,000 deaths per year in the United States). TBI can be clinically stratified into mild, moderate, and severe based on the Glasgow Coma Scale (GCS) score, with associated permanent disability rates of 10%, 60%, and 100%, respectively, and overall mortalities of 20% to 30%. The economic impact is more than $80 billion in the United States alone according to the most recent US Centers for Disease Control and Prevention data [4].

What is the problem?

As TBI is commonly encountered in the emergency department, and the early treatment of the increased ICP is pivotal in such setting [5], it would be of great use to assemble a checklist that guides the practice of the skill of management of elevated ICP, as well as the assessment this skill in the training setting. This would greatly help in mastering such important skill, which would ensure timely delivery of appropriate management to patients, considerably improving the outcomes.

Why is this Study Necessary?

Aggressive treatment of ICH is effective in reducing mortality and improving outcome. Because of potential side effects of therapy and intensive ICP monitoring, identifying patients at risk for developing ICH is crucial in order to correctly select patients that need invasive ICP monitoring and aggressive treatment [6].

On the other hand, an elevated intracranial pressure goes untreated, two major complications arise. Firstly, and more importantly, is the possibility of the occurrence of brain herniation; brain herniation is a deadly condition that arises when the ICP is extremely high, and this condition presses the brain tissue against the hard cranium causing compression damage to the brain; this extreme pressure may also cause the brain to shift across vital structures that connect the brain to the spinal cord, such as the falxcerebri. Secondly, permanent loss of vision may occur (depending on the severity), which could irreparably disrupt the patient's quality of life [7].

Therefore, it is of utmost importance to know which ICH patients are to be treated using conservative measures, and which have to be monitored invasively and treated using aggressive measures.

Methodology

This section includes Collection of all possible available data about the TBI with increased ICP in the Emergency department. By many research questions to achieve these aims so a Midline literature search, PubMed, Scopus and etc. from a reputed database. were performed with the keywords "critical care", "emergency medicine", "principals of resuscitation in ICP", "intracranial hematoma". Literature search included an overview of recent definition, causes and recent therapeutic strategies.

So the main aims and outcome of the study: Initial assessment and evaluate the TBI with increased ICP to recognize potentially life-threatening conditions and to convey life-saving treatment so the key note here is that initial diagnosis in suspected cases with treatment.

Table 1: Normal ICP.

Age group	Normal range (mm Hg)
Adults and older children[a]	< 10-15
Young children	3-7
Term infants[b]	1.5-6

[a]The age of transition from "young" to "older" child is not precisely defined.

[b]May be subatmospheric in newborn.

Research Question

What are the steps of appropriate assessment and priorities for treatment the increased ICP patients? and training protocol to Emergency Physicians. Based on patients' causes of increased ICP?

Definitions

ICP is normally ≤ 15 mmHg in adults, and pathologic ICH is present at pressures ≥ 20 mm Hg. Homeostatic mechanisms stabilize ICP, with occasional transient elevations associated with physiologic events, including sneezing, coughing, or Valsalva maneuvers [5,8] (Table 1).

Pathologic structures, including mass lesions, abscesses, and hematomas, also may be present within the intracranial compartment. Since the overall volume of the cranial vault cannot change, an increase in the volume of one component, or the presence of pathologic components, necessitates the displacement of other structures, an increase in ICP, or both. Thus, ICP is a function of the volume and compliance of each component of the intracranial compartment, an interrelationship known as the Monro-Kellie doctrine. Elevated ICP may complicate trauma, central nervous system (CNS) tumors, hydrocephalus, hepatic encephalopathy, and impaired CNS venous outflow [4]. Furthermore, secondary increase in ICP is sometimes observed 3-10 days following the trauma, and may be associated with a worse prognosis [9].

The classic clinical presentation of ICH is Cushing's triad, which is defined as hypertension, bradycardia, and respiratory irregularity. In addition, patients suffering from significant ICP elevation, due to trauma or otherwise, are usually obtunded [10].

There are two types of injuries that occur after a TBI. There is the primary brain injury (e.g. cortical contusions, lacerations, bone fragmentation, diffuse axonal injury, and brainstem contusion), which occurs at the same time as the main traumatic event; this type of injury cannot be prevented by the treating physician. The second type is the secondary injury, which develops subsequent to the primary injury, and is happens as a result of systemic consequences of the primary injury (includes injuries from intracranial hematomas, edema, hypoxemia, ischemia (primarily due to elevated ICP and/or shock), vasospasm) [11]. This type injury can be prevented

by supportive measures and tight control of intracranial pressure; therefore, the main goal of treatment of TBI focuses on preventing such injuries.

Hypotension, previously defined as systolic blood pressure (SBP) less than 90 mm Hg, and hypoxia, defined as a PaO_2 less than or equal to 60 mm Hg, have been associated with doubling of mortality in patients with head injuries. Studies have shown an association between systemic insults (mainly hypotension, hypoxia, and hypercarbia) and increased mortality. Management strategies must therefore focus on the prevention of secondary injury (i.e., hypoxia, hypotension) through maintenance of adequate CBF and prevention of hypoxia [12].

Steps of the Initial Management of Increased ICP [3]

Prehospital management

As all other phases of elevated ICP management, prehospital strategies should focus on preventing secondary brain injury. Studies have shown that more advanced airway management modalities (e.g. rapid sequence intubation) in patients with head injuries who have a GCS less than 9 was associated with an increase in mortality. This result may be associated with the transient hypoxia during the prehospital procedures, excessive over ventilation causing hypocarbia, vasoconstriction, impaired CBF, and longer scene times. This implies the need for rapid transfer to definitive care and a focus on more basic airway strategies to maintain oxygenation in patients with head injuries.

In addition, researchers have found that utilization of special types of IV fluids (e.g. hypertonic saline) for volume resuscitation of patients with increased ICP cannot be recommended due to lack sufficient evidence.

Emergency department management [13]

The initial management of patients with increased ICP (especially if the etiology is TBI) is identical to that of all patients with trauma, focusing on the Advanced Trauma Life Support (ATLS) principles of management of airway, breathing, and circulation, followed by a rapid neurologic examination and exposure of the patient with prevention of hypothermia.

The airway should be secured according to local protocols. Induction agents such as propofol should be carefully used, given the risk of systemic hypotension with impaired CBF. Ketamine is an attractive agent in patients with trauma, given its favorable hemodynamic profile. Despite theoretic risks, a systematic review of ketamine use in TBI suggests that ketamine does not increase ICP.

Breathing should be optimized to maintain oxygenation and prevent ventilatory dysfunction, because extremes in CO_2 levels can lead to cerebral vasoconstriction and vasodilatation, and have been shown to be predictors of morbidity and mortality. Hyperventilation is used by some providers to acutely decrease ICP through hypocarbicvasoconstriction, despite evidence showing an association between even brief periods of hyperventilation and increased levels of mediators of secondary brain injury in areas adjacent to injured brain tissue as well as local reductions in cerebral perfusion. Therefore, this strategy should be used with caution, and perhaps only to acutely combat signs of active herniation while initiating more definitive treatment.

Circulation should be maintained to prevent hypotension and maintain CBF. There is a known coagulopathy related to head injury likely related to tissue factor release coupled with hypoperfusion, which may be exacerbated by a pure crystalloid resuscitation. A balanced blood product resuscitation has been shown to be beneficial in patients with trauma, and this likely extends to patients with elevated ICP related to TBI. Non-cross-matched packed red blood cells are an initial resuscitative fluid choice that is often used in hypotensive patients with trauma, with a goal to maintain SBP at greater than or equal to 90mmHg in patients suspected of having a TBI. The concept of permissive hypotension does not apply to patients with known or suspected TBI, and normal physiologic blood pressure parameters should be targeted in this population.

Disability should be looked for in the primary survey, starting by a rapid neurologic evaluation. The evaluation focuses on the pupillary examination, assesses for lateralizing signs suggesting a mass lesion with increased ICP, and calculates a GCS score to stratify the TBI severity. Agents such as hypertonic saline and/or mannitol can be given during this initial resuscitation if physical examination findings suggest a neurologic decline, significant head injury, or lateralizing neurologic examination.

Following the initial resuscitation, patients suspected of having a TBI usually undergo a non-contrasted head computed tomography (CT) scan, depending on the presence of other injuries that require more urgent attention. If rapid CT scanning is not available, providers can consider using one of the various criteria for determining need for additional imaging, such as the Canadian CT Head Rule and the New Orleans Criteria.

The three-tiered management of intracranial pressure

Tier 1:

- Head of bed elevated at 30 degrees (reverse Trendelenburg) to improve cerebral venous outflow
- Sedation and analgesia using recommended short-acting agents (for example, propofol, fentanyl, midazolam, ketamine) in intubated patients
- Ventricular drainage performed intermittently. Continuous drainage is not recommended unless an additional ICP monitor is placed, as when the drain is open, it does not accurately reflect the true ICP
- Repeat CT imaging and neurological examination should be considered to rule out the development of a surgical mass lesion and guide treatment

If ICP remains ≥ 20 - 25 mm Hg proceed to Tier 2

Tier 2:

- In patients with a parenchymal ICP monitor an external ventricular drain (EVD) should be considered to allow for intermittent CSF drainage
- Hyperosmolar therapy should be given intermittently as needed for ICP elevation and not on a routine schedule
    - Mannitol should be administered in intermittent boluses (0.25 - 1 gm/kg body weight). Caution should be taken in the hypovolemic patient when osmotic diuresis is instituted with mannitol. The serum sodium and osmolality must be assessed frequently (every 6 hours) and additional doses should be held if serum osmolality exceeds 320 mOsm/L. Mannitol may also be held if there is evidence of hypovolemia
    - Hypertonic saline may be administered in intermittent boluses of 3% sodium chloride solution (250 ml over 30 minutes) or other concentrations (e.g., 30cc of 23.4%). Serum sodium and osmolality must be assessed frequently (every 6 hours) and additional doses should be held if serum sodium exceeds 160 mEq/L
- Cerebral autoregulation should be assessed. If the patient is not autoregulating, the CPP goal should be lowered to reduce ICP (to no less than 50 mmHg).
- PaCO2 goal of 30 - 35 mmHg should be maintained, as long as brain hypoxia is not encountered.
- Repeat CT imaging and neurological examination should be considered to rule out development of a surgical mass lesion and guide treatment
- Neuromuscular paralysis achieved with a bolus "test dose" of a neuromuscular blocking agent should be considered if the above measures fail to adequately lower ICP and restore CPP. If there is a positive response, continuous infusion of a neuromuscular blocking agent should be employed (Tier 3)

If ICP remains ≥ 20 - 25 mmHg proceed to Tier 3

TIER 3 (not part of the initial management in the emergency department)

- Decompressive hemi-craniectomy or bilateral craniectomy should only be performed if treatments in Tiers 1 and 2 are not sufficient or are limited by development of side effects of medical treatment
- Neuromuscular paralysis via continuous infusion of a neuromuscular blocking agent can be employed if there is a positive response to a bolus dose. The infusion should be titrated to maintain at least two twitches (out of a train of four) using a peripheral nerve stimulator. Adequate sedation must be utilized
- Barbiturate or propofol (anesthesia dosage) coma may be induced for those patients who have failed to respond to aggressive measures to control malignant intracranial hypertension, however it should only be instituted if a test dose of barbituate or propofol results

Table 2: Summary of measures to control IC-HTN a Goals: Keep ICP < 22 mm Hg, and CPP ≥ 50 mm Hg

Skill	Done Sufficiently (2)	Done Insufficiently (1)	Not Done (0)
General measures (should be utilized routinely)			
Elevate head of the bed by 30 ºC			
Keep neck straight, avoid neck constrictions (tight trach tape, tight cervical collar…)			
Control hypertension if present			
Avoid arterial hypotension (SBP < 90 mm Hg)			
Avoid hypoxia (PaO_2< 60 mm Hg or O_2 sat < 90%)			
Ventilate to normocarbia ($PaCO_2$ = 35-40 mm Hg)			
Light sedation: e.g. codeine 30-60 mg IM q 4 hrs PRN			
Perform unenhanced head CT scan to identify etiology			
Specific measures for ICH (proceed to successive steps if documented ICH persists - each step is ADDED to the previous measure)			
Heavy sedation (e.g. fentanyl 1-2 ml or morphine 2-4 mg IV q 1 hr) and/or paralysis (e.g. vecuronium 8-10 mg IV)			
Drain 3-5 ml CSF if intraventricular catheter is present			
Hyperventilate to $PaCO_2$ = 30-35 mm Hg ("blows off" CO_2)			
Mannitol 0.25-1 gm/kg, then 0.25 gm/kg q 6 hrs, increase dose if ICH persists & serum osmol ≤ 320 (NB: Skip this step if hypovolemia or hypotension)			
If there is "osmotic room" (i.e., serum osmol < 320) bolus with 10-20 ml of 23.4% hypertonic saline			
Augmented hyperventilation to ↓ $PaCO_2$ to 25-30 mm Hg			
If ICH persists, consider unenhanced head CT & EEG. Proceed to "second tier" therapy			

in a decrease in ICP, thereby identifying the patient as a "responder." Hypotension is a frequent side effect of high dose therapy with these agents. Meticulous volume resuscitation should be ensured and infusion of vasopressor/inotropes may be required. Prolonged use or high dose of propofol can lead to propofol infusion syndrome. Continuous EEG may be used to ensure targeting of the infusion to burst suppression

- Hypothermia (< 36 °C) is not currently recommended as an initial TBI treatment. Hypothermia should be reserved for "rescue" or salvage therapy after reasonable attempts at ICP control via the previous Tier 3 treatments have failed (Table 2)

Summary and Recommendations

TBI is a leading cause of death and disability in patients with trauma. The rapid transfer of patients with TBI to trauma centers and the avoidance of secondary insults such as hypotension and hypoxia are paramount. Increased ICP should be managed in an algorithmic fashion using simple beside maneuvers, hyperosmolar agents, ventricular drainage, barbiturates, and operative intervention when appropriate.

We recommend the application of the step-wise, three-tiered approach to the management of increased ICP, as it utilizes various treatment strategies to target different pathophysiological mechanisms. In addition, it has the advantage of being step-wise and algorithmic, making it easier to remember and apply in high-demand situation, making the management of increased ICP in the ED more effective, evidence-based, and time-efficient. This would greatly improve patient outcomes while saving valuable resources for those in need.

We also recommend the use of the suggested OSCE checklist to assess the ability of ER physicians to manage patients with elevated ICP according to the three-tier approach, ensuring safe delivery of evidence-based medicine to patients with increased ICP, especially in the setting of TBI.

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DOI: 10.36959/605/551