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SOFT PACK IN TRAUMATIC BRAIN INJURY: A LITERATURE REVIEW

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Abstract: INTRODUCTION: Traumatic Brain Injury (TBI) is a complex neurological disorder resulting from external mechanical forces applied to the head, encompassing a spectrum of injury severity from mild concussions to severe cases with prolonged loss of consciousness and extensive neurological deficits. The pathophysiology involves both primary injuries, occurring at the moment of impact, and secondary injuries, involving biochemical and cellular processes evolving over time. Intracranial hypertension (ICH) is characterized by increased pressure within the cranial cavity, resulting from pathological mechanisms like CSF accumulation, cerebral edema, or mass lesions

OBJETIVE: To Analyze and describe the main aspects of TBI and its treatment in the last years.

METHODS: This is a narrative review. The base dates used were PubMed, sciELO and Medline, using as descriptors: "Traumatic Brain Injury" AND "Intracranial hypertention" AND "Trauma" AND "axonal injury" AND "treatment". Only studies writing in inglish and were included.

RESULTS AND DISCUSSION: The text delves into various aspects of TBI management, focusing on critical considerations such as intracranial pressure (ICP), physiological parameters maintenance, and therapeutic interventions. ICP, representing the balance within the skull, is pivotal for cerebral perfusion, and elevated ICP during TBI can compromise blood flow, leading to ischemia and exacerbating the initial injury. Monitoring and managing ICP, including maintaining it below 20 mmHg, are crucial in TBI care, with interventions like head elevation, osmotic therapy, and surgical procedures. The comprehensive management involves ensuring optimal physiological parameters, including oxygen saturation, blood pressure, and normocapnia, as deviations can impact cerebral blood flow and exacerbate intracranial hypertension. The text emphasizes the significance of maintaining the head in an elevated position to mitigate ICP, the importance of fever control, and the correction of coagulation disorders to prevent hemorrhagic complications. Furthermore, therapeutic interventions like hypertonic saline 3%, mannitol, sedation, and neuromuscular blockade are discussed in the context of TBI management. These interventions aim to reduce ICP, alleviate cerebral edema, ensure patient comfort, and control agitation. The use of hypothermia, barbiturates, and corticosteroids in TBI has been explored, but their efficacy remains inconclusive, prompting a reconsideration of their roles. Lastly, the text touches upon the prophylactic use of phenytoin within the first 7 days following TBI to prevent early post-traumatic seizures, highlighting the limited benefits observed in clinical trials and the current consensus against routine administration in the absence of specific indications.

CONCLUSION: In summary, the text delivers a comprehensive overview of TBI management, centering on the pivotal role of ICP in influencing cerebral perfusion. It covers the calculation and alterations of ICP during traumatic brain injuries, emphasizing recommended interventions to maintain optimal ICP levels. The significance of preserving physiological parameters, such as oxygen saturation, blood pressure, and normocapnia, is underscored for optimizing cerebral hemodynamics and minimizing secondary injuries in TBI patients. The text explores a multifaceted approach to TBI management, delving into therapeutic interventions like hypertonic saline, mannitol, sedation, and neuromuscular blockade. It critically evaluates the efficacy of hypothermia, barbiturates, corticosteroids,

and prophylactic phenytoin, highlighting the complexities and limitations associated with their use. Overall, the information provided serves as a valuable resource for clinicians and researchers, offering insights into current practices, challenges, and potential avenues for further investigation in the field of TBI management.

Keywords: Traumatic Brain Injury; intracranial pressure; Intracranial hypertention; Trauma; axonal injury;

INTRODUCTION

Traumatic Brain Injury (TBI) is a multifaceted neurological disorder resulting from external mechanical forces applied to the head, leading to complex and dynamic disruptions in brain structure and function[1]. These forces can be caused by various events, such as motor vehicle accidents, falls, or direct blows to the head[1]. TBI encompasses a spectrum of injury severity, ranging from mild, where consciousness may be briefly altered (concussion), to moderate and severe, where there may be prolonged loss of consciousness and more extensive neurological deficits[2]. The pathophysiology of TBI involves both primary injuries, occurring at the moment of impact, and secondary injury, which involves a cascade of biochemical and cellular processes evolving over time, leading to additional damage[1,2].

The primary injury in TBI includes focal injuries, such as contusions and lacerations, and diffuse injuries, such as axonal shearing and diffuse axonal injury[3]. The secondary injury mechanisms involve a complex interplay of neuroinflammatory responses, excitotoxicity, oxidative stress, and alterations in cerebral blood flow, which can contribute to ongoing neuronal dysfunction and cell death[4]. Neuroimaging techniques, such as computed tomography and magnetic resonance imaging, play a crucial role in diagnosing and characterizing the extent of primary injuries, while understanding the underlying molecular and cellular processes aids in developing targeted therapeutic interventions[2,4].

TBI can result from a variety of external mechanical forces applied to the head, with the leading causes encompassing motor vehicle accidents, falls, and assaults[5]. Motor vehicle accidents, including collisions involving automobiles, motorcycles, or pedestrians, are a significant contributor to TBI incidence[6]. The sheer kinetic energy involved in these events can lead to head impact or sudden deceleration, causing varying degrees of brain injury[5]. Falls, particularly among the elderly, are another prevalent cause of TBI, where the impact with the ground or other surfaces can result in head trauma. Additionally, intentional acts of violence, such as assaults, contribute to TBI cases, often involving direct blows to the head[6].

On the other hand, Intracranial hypertention (ICH) is a medical condition characterized by an abnormal increase in pressure within the cranial cavity, which houses the brain[7]. This elevated pressure arises from various pathological mechanisms, including the accumulation of CSF, cerebral edema, or the presence of mass lesions within the skull, such as tumors or hematomas[8]. The delicate balance between the volume of the brain tissue, blood, and CSF within the rigid confines of the skull is disrupted in intracranial hypertension, leading to increased pressure that can impede cerebral blood flow and cause potential damage to neural structures[8]. Clinical manifestations of ICH may include headache, altered mental status, and, in severe cases, neurological deficits. Timely diagnosis and management are critical to prevent irreversible brain damage associated with prolonged elevated intracranial pressure[7,8].

OBJETIVE

MAIN OBJETIVE

To Analyze and describe the main aspects of TBI and its treatment in the last years.

SECUNDARY OBJETIVES

1. To systematically review and synthesize existing literature on the use of soft pack interventions in the management of TBI: This objective aims to comprehensively analyze published studies, including clinical trials, case reports, and experimental research, to evaluate the effectiveness and safety of soft pack interventions in the context of TBI. The review will consider various aspects such as patient outcomes, feasibility, and potential benefits or limitations associated with the use of soft packs in the treatment of TBI.
2. To assess the role of non-pharmacological interventions in the rehabilitation of TBI patients: This objective focuses on exploring non-pharmacological treatment modalities, such as cognitive rehabilitation, physical therapy, and neuropsychological interventions. The article will critically evaluate the effectiveness of these interventions in promoting functional recovery, improving quality of life, and addressing cognitive and motor impairments associated with TBI.
3. To identify gaps in the current understanding of TBI treatment and propose directions for future research: This objective seeks to analyze the limitations and gaps in the existing literature on TBI treatment. By identifying areas where further research is needed, the article aims to contribute to the ongoing development of innovative and targeted therapeutic approaches for Traumatic Brain Injury.
4. To discuss the implications of recent advancements in treatment strategies for TBI on clinical practice: This objective focuses on translating research findings into practical insights for clinicians involved in the care of TBI patients. The article will explore how recent developments in treatment approaches can be integrated into clinical practice, emphasizing evidence-based recommendations and potential challenges in implementing novel strategies.

These specific objectives collectively aim to provide a comprehensive and up-to-date synthesis of the literature on the treatment of Traumatic Brain Injury, offering insights into both pharmacological and non-pharmacological interventions, identifying research gaps, and discussing the implications for clinical care.

METHODS

This is a narrative review, whose main objective is to recognize the main aspects of tbi and its treatment in the last 20 years. The beginning of the study was carried out with theoretical training using the following databases: PubMed, sciELO and Medline, using as descriptors: “Traumatic Brain Injury” AND “Intracranial hypertention” AND “Trauma” AND “axonal injury” AND “treatment” in the last 20 years. As it is a narrative review, this study does not have any risks.

The inclusion criteria applied in the analytical review were human intervention studies, experimental studies, cohort studies, case-control studies, cross-sectional studies and literature reviews, editorials, case reports, and poster presentations. Also, only studies writing in inglish were included.

RESULTS AND DISCUSSION

Intracranial Pressure ICP refers to the pressure exerted by the cerebrospinal fluid (CSF) within the rigid confines of the skull, representing the balance between the volume of brain tissue, blood, and CSF[8,9]. It is a crucial physiological parameter as it directly influences cerebral perfusion and can be a critical factor in the management of TBI[9]. Under normal conditions, ICP is maintained within a relatively narrow range to ensure optimal cerebral blood flow and prevent compression of vital brain structures[10].

ICP is calculated as the ratio of the total volume within the skull (brain tissue, blood, and CSF) to the compliance of the intracranial space. Mathematically, ICP = (Volume)/(Compliance)[10]. However, direct measurement of ICP is typically performed through invasive procedures, such as placing a catheter into the brain's ventricles (intraventricular catheter) or a pressure sensor within the brain tissue (intraparenchymal monitor)[9,10].

During a traumatic brain injury, the brain may swell, or there may be an accumulation of blood or other fluids, leading to an increase in ICP[10]. Elevated ICP can compromise cerebral blood flow, resulting in ischemia and potentially exacerbating the initial injury[9]. Therefore, monitoring and managing ICP are crucial aspects of TBI care. Clinical guidelines, such as those provided by the Brain Trauma Foundation, recommend maintaining ICP below 20 mmHg to reduce the risk of secondary brain injury and improve outcomes in TBI patients[10]. Interventions to control ICP may include elevation of the head, osmotic therapy, and, in severe cases, surgical procedures to remove intracranial masses or drain excess fluid[11].

Ensuring the maintenance of physiological parameters such as oxygen saturation, blood pressure, and normocapnia is critically important in the comprehensive management of TBI[11]. Adequate oxygen saturation is fundamental for meeting the cerebral metabolic demands, and episodes of hypoxia, characterized by low oxygen levels, can exacerbate secondary brain injuries, compromising cellular metabolism and contributing to neuronal damage following the initial trauma[12]. Vigilant monitoring and intervention to prevent and correct hypoxemia are emphasized as essential strategies in TBI management, recognizing the direct association between hypoxia and unfavorable outcomes[11].

In addition to oxygen saturation, maintaining blood pressure within appropriate limits is crucial for ensuring adequate cerebral perfusion[12]. Hypotension can compromise cerebral blood flow, exacerbate ischemia, and contribute to increased intracranial pressure, while hypertension may intensify intracranial pressure, elevating the risk of further damage[13]. Stabilizing blood pressure is, therefore, a priority in TBI management, with clinical guidelines underscoring the need to maintain a delicate balance to ensure optimal cerebral perfusion and thereby improve neurological outcomes[13,14].

Furthermore, normocapnia, or maintaining normal levels of arterial carbon dioxide, plays a critical role in ICP regulation and oxygen delivery to brain tissue[12]. Deviations from normocapnia can impact cerebral blood flow, exacerbating ICH in cases of hypercapnia or inducing detrimental cerebral vasoconstriction in cases of hypocapnia[13]. Thus, maintaining appropriate carbon dioxide levels is an essential consideration in TBI management to optimize cerebral hemodynamics and minimize the risk of secondary injuries[14].

Maintaining the head in an elevated position is of paramount importance in the management of TBI as it can help mitigate ICP and optimize cerebral perfusion[15].

Elevation of the head minimizes the risk of venous congestion in the brain, aiding in the reduction of intracranial blood volume and, consequently, lowering ICP[15]. This approach is a well-established practice in TBI management protocols, aiming to create an environment that facilitates optimal cerebral blood flow and reduces the likelihood of secondary injury[9]. Clinical guidelines, including those outlined by the Brain Trauma Foundation, emphasize the significance of maintaining the head in a slightly elevated position to support favorable outcomes in TBI patients[9].

Fighting fever is another critical aspect in TBI management as hyperthermia can exacerbate secondary brain injury and negatively impact outcomes[17]. Elevated body temperature can contribute to increased metabolic demands, excitotoxicity, and enhanced inflammatory responses, all of which may intensify neuronal damage in TBI patients[17]. Actively addressing and preventing hyperthermia through measures such as antipyretic medications and temperature control strategies are integral components of comprehensive TBI care. Studies highlight the association between elevated body temperature and worsened outcomes in TBI, reinforcing the importance of fever management in improving patient prognosis[17,18].

Correcting coagulation disorders is essential in TBI to prevent or manage hemorrhagic complications and reduce the risk of secondary injury[19]. Disruptions in coagulation can lead to increased bleeding, hematoma expansion, and worsened outcomes[20]. Prompt identification and targeted intervention to address coagulopathies, such as administering clotting factors or blood products, are critical components of TBI management protocols [19,20]. The recognition and correction of coagulation disorders are emphasized in clinical guidelines to optimize patient outcomes and minimize the risk of complications associated with impaired hemostasis in TBI patients[20].

Hypertonic saline 3% is often utilized in the management of TBI due to its osmotic properties, which help reduce ICP[21]. The hypertonicity of the saline draws water out of brain cells, leading to a decrease in cerebral edema[21]. This is particularly important in the acute phase of TBI, where elevated ICP can compromise cerebral perfusion and contribute to secondary brain injury[22]. Clinical studies and guidelines highlight the efficacy of hypertonic saline in lowering ICP and improving cerebral hemodynamics, emphasizing its role as a therapeutic option in TBI management[21,22].

Mannitol is an osmotic diuretic commonly used in the treatment of elevated ICP in traumatic brain injury[22]. Similar to hypertonic saline, mannitol works by drawing water out of brain tissue and into the blood vessels, reducing cerebral edema and alleviating intracranial hypertension[21,23]. Mannitol is known for its rapid onset of action and is often used in emergency settings to address acute increases in ICP[23]. However, its use requires careful monitoring, as excessive administration can lead to dehydration and potential adverse effects. Clinical guidelines recommend mannitol as a first-line treatment for elevated ICP in TBI, particularly in situations where other interventions may not be immediately available[22].

Sedation is a crucial component of TBI management to ensure patient comfort, facilitate mechanical ventilation, and control agitation[24]. By inducing a state of decreased consciousness, sedation helps manage pain and anxiety, preventing increases in blood pressure and ICP that may exacerbate the existing brain injury[23,24]. Commonly used sedatives in TBI include propofol, midazolam, and dexmedetomidine. The choice of sedative depends on factors such as the patient's

hemodynamic stability and desired depth of sedation[25]. Appropriate sedation in TBI contributes to the overall care plan by supporting neuroprotective strategies and preventing complications associated with increased intracranial pressure[24,25].

Neuromuscular blockade is occasionally employed in TBI cases to achieve controlled paralysis, facilitating mechanical ventilation and reducing patient movement. This intervention is typically reserved for situations where other measures to control ICP, such as sedation and osmotic agents, have been insufficient[25,26]. The use of neuromuscular blockade requires careful consideration, as it can mask neurological assessments and contribute to complications such as muscle atrophy and weakness[14]. It is typically employed for a short duration and in conjunction with sedation to optimize patient care and ventilation[28].

Hypothermia, barbiturates, and corticosteroids have been extensively investigated as potential therapeutic interventions for TBI, aiming to mitigate secondary injury processes and improve clinical outcomes[29,30,31]. However, the results from clinical trials and research studies have not consistently supported their efficacy, leading to a reconsideration of their roles in TBI management[28, 29,30,31].

Hypothermia, or the intentional cooling of the body, was initially explored with the hypothesis that reducing body temperature could attenuate inflammatory responses and cellular damage in the brain after TBI[22,28]. While some experimental studies showed promising results, large clinical trials like the National Acute Brain Injury Study: Hypothermia II (NABIS: H II) failed to demonstrate significant improvements in functional outcomes or mortality with hypothermia compared to normothermia in TBI patients[29]. The complexities of implementing hypothermia, potential complications, and the lack of consistent benefit across studies have contributed to the challenges in adopting it as a routine intervention[29].

Barbiturates, particularly sodium thiopental, were investigated for their neuroprotective potential in severe TBI cases[30]. The belief was that barbiturates could reduce cerebral metabolism, cerebral blood flow, and intracranial pressure. However, the Barbiturates for Head Injury trial and subsequent research did not provide clear evidence of improved neurological outcomes, and the use of barbiturates was associated with significant complications, including hypotension and infections[30]. Consequently, the routine use of barbiturates in TBI has been reevaluated due to the lack of consistent efficacy and the potential for adverse effects[27,28,30].

Corticosteroids, such as methylprednisolone, were investigated to address inflammation and edema in TBI[31]. The Corticosteroid Randomization After Significant Head Injury (CRASH) trial, a large-scale randomized controlled trial, revealed that the administration of methylprednisolone did not confer benefits and, in fact, was associated with an increased risk of death and disability in TBI patients[31]. The detrimental effects of corticosteroids, including complications like hyperglycemia and immunosuppression, have led to a shift away from routine corticosteroid use in the management of TBI[22, 26, 31].

Prophylactic administration of phenytoin, an antiepileptic medication, within the first 7 days following TBI has been a subject of clinical interest to prevent early post-traumatic seizures[9, 32]. While seizures can occur as a complication of TBI, the routine use of prophylactic phenytoin has shown limited benefits in terms of preventing early post-traumatic seizures and improving

overall outcomes. The Prophylactic Antiepileptic Drug Treatment in Early Post-TBI (PROTECT III) trial, a multicenter randomized controlled trial, did not find a significant reduction in the incidence of early post-traumatic seizures or improvement in neurological outcomes with prophylactic phenytoin compared to placebo[31]. The current consensus, as reflected in clinical guidelines, is that prophylactic phenytoin should not be routinely administered to all TBI patients in the absence of specific indications such as a known history of seizures or contusions involving the cortex[9]. The decision to initiate phenytoin prophylaxis is often made on a case-by-case basis, considering individual patient factors and the overall risk of seizures[9,31].

CONCLUSION

In conclusion, the text provides a comprehensive overview of the critical aspects involved in the management of Traumatic Brain Injury (TBI). Intracranial pressure (ICP) emerges as a central focus, highlighting its crucial role in influencing cerebral perfusion and its significance in TBI care. The discussion encompasses the calculation of ICP, its alterations during traumatic brain injuries, and the recommended interventions to maintain ICP within optimal limits. Additionally, the text emphasizes the importance of maintaining physiological parameters, such as oxygen saturation, blood pressure, and normocapnia, underscoring their role in optimizing cerebral hemodynamics and reducing the risk of secondary injuries in TBI patients.

The multifaceted approach to TBI management is evident in the exploration of various therapeutic interventions, including hypertonic saline, mannitol, sedation, and neuromuscular blockade. Each intervention is discussed in the context of its specific role in mitigating ICP, alleviating cerebral edema, ensuring patient comfort, and facilitating mechanical ventilation. Moreover, the text critically evaluates the efficacy of hypothermia, barbiturates, corticosteroids, and prophylactic phenytoin, pointing out the complexities and limitations associated with their use in TBI. The information provided serves as a valuable resource for clinicians and researchers involved in TBI management, offering insights into current practices, challenges, and areas for further investigation.

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