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Multidisciplinary Management of Traumatic Brain Injury: Emergency Care, Early Physical Therapy, Laboratory Monitoring, and Radiologic Evaluation

Muhannad Adel Ali Bajjah [(1)] , Aqeel Mohammad Ali Hadi [(2)] , Hasan Ahmed Yahya Abuhashim [(1)] , Ali Yahia Ibrahim Shabaani [(1)] , Hamdan Ali Hamdan Alghamdi [(3)] , Solaiman Rashed Alfaheed [(4)] , Naif Obaid Huda Alotaibi [(5)] , Muhannad Adel Ali Bajjah [(1)] , Ahmed Mohammed Hussain Abu Talib [(6)] , Mohammed Ahmed AlObaid [(7)] , Mohammed Salem Almotairi [(8)] , Ali Ahmad Hadi Dagriri [(9)] , Ali Yousef Ali Alhakami , Norah Gassim Ghulfan khormi [(10)]

(1) Damad Hospital – Damad,Ministry of Health, Saudi Arabia,
(2) Damad General Hospital – Damad,Ministry of Health, Saudi Arabia,
(3) Al-Aqiq General Hospital – Al-Baha,Ministry of Health, Saudi Arabia,
(4) Al-Asyah General Hospital,Ministry of Health, Saudi Arabia,
(5) Al-Bujadiyah Hospital,Ministry of Health, Saudi Arabia,
(6) King Fahad Central Hospital,Ministry of Health, Saudi Arabia,
(7) Al-Omran General Hospital,Ministry of Health, Saudi Arabia,
(8) Huraymila General Hospital Riyadh Third Health ,Ministry of Health, Saudi Arabia,
(9) Emergency Department-Jazan Health Cluster, Ministry of Health, Saudi Arabia,
(10) General Jazan hospital,Ministry Of Health, Saudi Arabia

Abstract

Background: Traumatic brain injury (TBI) is a major global health concern affecting millions annually and causing substantial morbidity, mortality, and long-term disability. It results from mechanical forces to the head, with severity ranging from mild concussion to severe intracranial injury. Closed head injuries—including contusions, diffuse axonal injury (DAI), and intracranial hematomas—represent the majority of cases.

Aim: This study aims to highlight multidisciplinary approaches in the emergency care, early rehabilitation, laboratory assessment, and radiologic evaluation of TBI to improve outcomes and reduce secondary brain injury.

Methods: A comprehensive review of TBI mechanisms, epidemiology, clinical presentation, diagnostic strategies, and acute management was conducted. Emphasis was placed on validated clinical tools such as the Glasgow Coma Scale (GCS), Canadian CT Head Rule, New Orleans Criteria, and PECARN guidelines for imaging decisions. Pathophysiological processes including primary and secondary injury cascades, intracranial pressure dynamics, and herniation syndromes were analyzed.

Results: Effective TBI management requires early airway stabilization, prevention of hypoxia and hypotension, and timely imaging. Evidence-based algorithms guide CT utilization and reduce unnecessary radiation exposure. Biomarkers such as GFAP and S100B show promise in predicting intracranial pathology in mild TBI. Severe TBI benefits from invasive ICP monitoring, targeted physiological goals, and multidisciplinary collaboration to reduce mortality. Rehabilitation involving physical, occupational, speech, and cognitive therapy significantly enhances functional outcomes.

Conclusion: Integrated emergency management, prevention of secondary injury, structured imaging strategies, and coordinated rehabilitation improve TBI outcomes. A multidisciplinary team approach is essential to optimize survival, functional recovery, and long-term quality of life.

Keywords: Traumatic brain injury, intracranial pressure, emergency management, CT imaging, diffuse axonal injury, rehabilitation.

Introduction

Traumatic Brain Injury (TBI) results from external mechanical forces applied to the head, including direct impact, rapid acceleration-deceleration, or penetrating injuries, and represents a significant global health concern. This discussion focuses on closed head injuries, which constitute the most common form of TBI, as opposed to penetrating brain injuries. Closed head injuries encompass a range of conditions, including concussions, parenchymal contusions, and intracranial hematomas of varying types. In the United States alone, approximately 1.7 million individuals experience a TBI annually, with the highest incidence observed among older adolescents aged 15 to 19 and adults over 65. These injuries frequently involve the frontal and temporal regions of the brain, contributing to cognitive, behavioral, and motor impairments, while placing a substantial economic and social burden on healthcare systems, especially in low-resource settings [1][2][3][4]. TBI severity is classified into mild, moderate, or severe categories based on clinical

*Corresponding author e-mail: mohanad70077@gmail.com (Muhannad Adel Ali Bajjah).
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features and standardized scales. Mild TBI, commonly referred to as a concussion, is defined by a brief loss of consciousness of less than 30 minutes, post-traumatic amnesia lasting under 24 hours, or any transient alteration of consciousness. Moderate and severe TBIs involve longer durations of unconsciousness, post-traumatic amnesia extending beyond 24 hours, or profound neurological deficits. The Glasgow Coma Scale (GCS), scored from 3 to 15, is widely used to assess neurological function in TBI patients. A GCS of 14–15 indicates mild TBI, 9–12 represents moderate injury, and 3–8 corresponds to severe TBI. Mild TBI accounts for over 90% of all cases, whereas moderate and severe injuries are less frequent but associated with higher morbidity and mortality [5][6]. Clinical presentation varies considerably, ranging from transient confusion or brief loss of consciousness in mild cases to prolonged coma in severe injuries. Long-term consequences may include persistent postconcussive symptoms such as headaches, dizziness, cognitive deficits, mood disturbances, and depressive symptoms. Repeated TBIs increase the risk of cumulative neurological damage, including chronic traumatic encephalopathy (CTE), highlighting the importance of early recognition, evidence-based management, and ongoing research to mitigate long-term sequelae. The widespread impact of TBI underscores the need for integrated emergency care, rehabilitation strategies, and public health initiatives to reduce morbidity and improve functional outcomes [7][8][9][10][9].

Etiology

Traumatic brain injuries arise from a spectrum of mechanical forces affecting the head, including minor or significant impacts, rapid acceleration-deceleration events, and penetrating injuries. Even in the absence of direct impact, sudden acceleration and deceleration of the brain can cause diffuse axonal injury (DAI), which is often associated with severe and long-lasting neurological deficits [11][12]. The degree of injury depends on the magnitude and nature of the force, the point of impact, and individual patient factors such as age and comorbidities. Epidemiological data indicate that falls represent the most common cause of TBI across age groups. In children aged 0 to 17 years, falls account for 49% of TBI-related emergency department visits, while in adults aged 65 years and older, falls are responsible for 81% of such visits [13][14]. Motor vehicle collisions constitute the second leading cause of TBI, followed by sports-related injuries, occupational hazards, and interpersonal assaults. Among patients requiring hospitalization for TBI, falls account for 52% of admissions, and motor vehicle accidents account for approximately 20% [15]. Penetrating head injuries, including those caused by firearms or blast trauma, represent the most lethal forms of TBI. These injuries carry a high risk of mortality due to extensive brain tissue damage, hemorrhage, and secondary complications such as infection or raised intracranial pressure [16]. Other contributing factors, including high-risk behaviors, unsafe working conditions, and inadequate protective measures during sports or transportation, further increase the likelihood of TBI. Understanding the diverse mechanisms and primary causes of TBI is critical for developing targeted prevention strategies, improving emergency response protocols, and optimizing patient outcomes through early intervention and rehabilitation.

Epidemiology

Traumatic brain injury (TBI) is a significant global health concern, affecting an estimated 70 million individuals annually. Epidemiological studies demonstrate that males are more frequently affected than females, with the highest incidence observed in children, young adults up to 24 years of age, and older adults over 75 [17][18][19][20]. Mild TBI, often referred to as concussion, represents the predominant form, accounting for over 90% of acute hospital presentations [21]. Although TBIs in the elderly comprise only 10% of cases, this population accounts for nearly half of all head trauma-related deaths, highlighting the increased vulnerability of older adults to poor outcomes. The distribution and causes of TBI vary between high-income and low- to middle-income countries. In low- and middle-income nations, the majority of head trauma results from road traffic accidents, with the highest incidence reported in Africa and Southeast Asia [17]. Conversely, in high-income countries, TBI is predominantly caused by falls, often related to age-associated frailty, and is frequently exacerbated by alcohol misuse [21]. This demographic pattern contributes substantially to the global burden of injury, emphasizing the need for preventive strategies tailored to regional risk factors. The Global Burden of Disease study provides further insight into the public health impact of TBI. In 2019, the worldwide age-standardized incidence of head trauma was 346 per 100,000 people. In 2016, TBI was estimated to account for 8.1 million years lived with disability and a pooled age-standardized rate of approximately 160 years of life lost per 100,000 individuals [21]. These figures underscore the extensive functional impairment, long-term disability, and mortality associated with TBI, illustrating the pressing need for improved prevention, early detection, and management strategies to reduce both individual and societal burdens [21].

Pathophysiology

Traumatic brain injury (TBI) is characterized by complex pathological mechanisms that arise from both primary and secondary insults to the brain. Understanding these mechanisms is essential for comprehending the acute and long-term consequences of head trauma and guiding appropriate management strategies. The primary injury in TBI occurs at the moment of trauma and results from the mechanical

forces directly applied to the cranial structures. These forces include blunt impact, rapid acceleration-deceleration, rotational forces, and penetrating injuries. Primary injury can manifest as skull fractures, focal cerebral contusions, and intracranial hematomas, including epidural, subdural, intraparenchymal, intraventricular, and subarachnoid hemorrhages. Additionally, diffuse axonal injury (DAI) is a hallmark of severe TBI caused by shearing forces that disrupt axonal integrity across widespread brain regions. The immediate mechanical disruption compromises neuronal, glial, and vascular structures, setting the stage for subsequent pathophysiological cascades. The severity and distribution of primary injury are influenced by the magnitude, direction, and location of the force applied, as well as individual factors such as age and pre-existing comorbidities.

Secondary Injury

Secondary injury refers to the delayed, progressive neurochemical and cellular damage that occurs hours to days after the initial insult. This phase is often triggered by hypoxia, hypotension, and raised intracranial pressure, exacerbating neuronal injury initiated by the primary event. Several neuropathological processes are implicated in secondary injury, including Wallerian degeneration, mitochondrial dysfunction, glutamate-mediated excitotoxicity, lactate accumulation, oxidative stress, lipid peroxidation, apoptotic cell death, neuroinflammation, impaired autophagy, and disruption of G-lymphatic pathways [23][24][25]. Glutamate excitotoxicity is central to secondary neuronal injury. Damaged neurons release excessive glutamate into the extracellular space, overstimulating NMDA and AMPA receptors. This leads to excessive sodium and calcium influx, activating enzymatic pathways that degrade cytoskeletal proteins and cell membranes, generate reactive oxygen species, and induce edema and cell death [23].

Intracranial Pressure and Herniation Syndromes

The Monro-Kellie doctrine explains the compensatory relationship between brain parenchyma, cerebrospinal fluid (CSF), and cerebral blood volume (CBV) within the rigid skull [26]. An increase in any of these components can elevate intracranial pressure (ICP), impair cerebral perfusion pressure (CPP), and compromise autoregulation (CPP = MAP – ICP). When ICP rises beyond compensatory limits, cerebral compliance diminishes, and sequential herniation syndromes may occur. Subfalcine herniation compresses pericallosal and callosomarginal vessels, resulting in contralateral lower limb weakness. Uncal herniation produces ipsilateral anisocoria and contralateral hemiparesis, with torsion of the diencephalon disrupting the reticular activating system and altering consciousness. Transtentorial herniation manifests as decorticate or decerebrate posturing, loss of brainstem reflexes, Cheyne-Stokes respiration, hyperventilation, ataxia, apnea, and ultimately respiratory arrest in cases of tonsillar herniation [27]. TBI pathophysiology represents a continuum from immediate mechanical injury to complex secondary biochemical cascades. The interplay of primary insult, secondary neurotoxic pathways, and compensatory mechanisms, including ICP and CPP regulation, defines the clinical severity and prognosis. Early recognition and intervention targeting both primary stabilization and mitigation of secondary injury are crucial in improving outcomes for patients with TBI. Understanding these processes also provides a foundation for the development of advanced therapeutic strategies, including neuroprotective agents, surgical interventions, and critical care monitoring protocols.

History and Physical

The clinical evaluation of traumatic brain injury (TBI) begins with a meticulous history, emphasizing the mechanism of injury and the presence and duration of any loss of consciousness. Determining the precise circumstances of the injury provides crucial insight into the potential severity and type of intracranial damage. A comprehensive assessment also requires reviewing the patient's medical history, including any use of anticoagulant or antiplatelet therapies, which may exacerbate intracranial bleeding risks. Patients with TBI frequently present with nonspecific symptoms, including nausea, vomiting, headache, tinnitus, visual disturbances, dizziness, cognitive slowing, and confusion. Long-term sequelae are common and may encompass persistent post-concussive symptoms such as impaired balance, memory deficits, cognitive dysfunction, emotional instability, anxiety, depression, sleep disturbances, and sensory changes, including visual alterations and headaches. These chronic manifestations underscore the necessity of ongoing monitoring and tailored rehabilitation strategies [28]. Initial physical examination should begin with vital sign assessment, particularly in patients at risk for elevated intracranial pressure (ICP). When ICP exceeds 20 mmHg (normal <15 mmHg), the Cushing triad—characterized by hypertension, bradycardia, and irregular respirations—may be present, signaling the need for urgent intervention. Once airway, breathing, and circulation are stabilized, the patient should undergo neurological assessment using the Glasgow Coma Scale (GCS), evaluating eye-opening, verbal, and motor responses. The GCS provides a standardized metric to categorize injury severity, with scores ranging from 3 to 15 [28].

Physical findings indicative of basilar skull fractures, such as Battle's sign, raccoon eyes, hemotympanum, and cerebrospinal fluid rhinorrhea or otorrhea, are strongly associated with intracranial hemorrhage [29]. Pupillary assessment is critical; a fixed, dilated pupil on one side may indicate ipsilateral intracranial hemorrhage and impending herniation. Neurological examination should extend to cranial nerve testing, assessment of motor strength, sensory function, reflexes, and clonus. Gait analysis may be

appropriate for patients without suspected cervical spine injury, although cervical immobilization remains standard for individuals with moderate to severe TBI until spinal injury is definitively excluded radiologically and clinically. In patients with persistent post-traumatic symptoms, neuropsychiatric evaluation is recommended to identify cognitive, emotional, or behavioral deficits that may require targeted intervention. Overall, a detailed history combined with a thorough physical and neurological examination allows clinicians to identify high-risk features, guide diagnostic imaging, and establish a structured plan for acute management and long-term rehabilitation following TBI [29].

Evaluation

The evaluation of traumatic brain injury (TBI) requires a nuanced and patient-specific approach that integrates clinical judgment, laboratory studies, and imaging modalities to guide diagnosis, prognostication, and treatment planning. Laboratory investigations are selectively employed based on the severity of injury, associated polytrauma, and underlying comorbidities. In cases of minor head injury, such as a ground-level fall resulting in a superficial contusion without additional trauma, laboratory testing may not be necessary, and clinical evaluation alongside imaging may suffice. Conversely, patients presenting after high-energy mechanisms, such as motor vehicle collisions, particularly those on anticoagulation therapy or with concomitant systemic injuries, typically warrant a comprehensive laboratory panel. Commonly indicated investigations include a complete blood count (CBC), comprehensive metabolic panel (CMP), coagulation studies including prothrombin time (PT) and partial thromboplastin time (PTT), and blood typing with crossmatch, as these tests provide critical information regarding hemodynamic stability, bleeding risk, and baseline metabolic function. Imaging plays a pivotal role in the initial evaluation of TBI, with non-contrast computed tomography (CT) of the brain considered the modality of choice for acute assessment due to its widespread availability and rapid acquisition. Skull radiographs have limited utility and are generally reserved for evaluating penetrating injuries or foreign bodies. Magnetic resonance imaging (MRI) provides superior soft tissue resolution and is particularly beneficial for assessing diffuse axonal injury (DAI), although its limited accessibility and longer acquisition time render it unsuitable for emergent evaluation. Advanced MRI techniques, including gradient-recalled echo (GRE) and diffusion tensor imaging (DTI), offer enhanced detection of subtle parenchymal injuries characteristic of DAI [30].

For adults with mild TBI, validated clinical decision rules guide the judicious use of CT imaging to minimize unnecessary radiation exposure while ensuring identification of clinically significant intracranial injury. The New Orleans Criteria (NOC) and Canadian CT Head Rule (CCHR) are two widely employed algorithms. The NOC recommends imaging in patients presenting with headache, vomiting, age over 60 years, intoxication, seizure, visible trauma above the clavicles, or short-term memory deficits. The CCHR incorporates criteria such as dangerous mechanisms of injury, repeated vomiting, age over 65, GCS less than 15 at two hours post-injury, seizure, evidence of basal skull fracture, open or depressed skull fractures, and retrograde amnesia exceeding 30 minutes. Comparative analyses indicate that the CCHR demonstrates greater specificity, reducing unnecessary CT scans without compromising sensitivity for neurosurgical intervention [31][32][33][34][35]. Pediatric head trauma assessment relies on the Pediatric Emergency Care Applied Research Network (PECARN) algorithms, which stratify risk based on age and injury characteristics. In children under two years, indications for CT include non-frontal scalp hematomas, loss of consciousness for at least five seconds, high-energy mechanisms such as falls greater than three feet or vehicular impact, abnormal behavior per caregiver report, and palpable skull fractures. For children aged two years and older, PECARN criteria include loss of consciousness, repeated vomiting, severe headache, significant mechanisms of injury, signs of basilar skull fracture, and altered mental status. PECARN demonstrates high sensitivity and negative predictive value in identifying clinically important TBI, thereby guiding imaging decisions while minimizing radiation exposure [36][37].

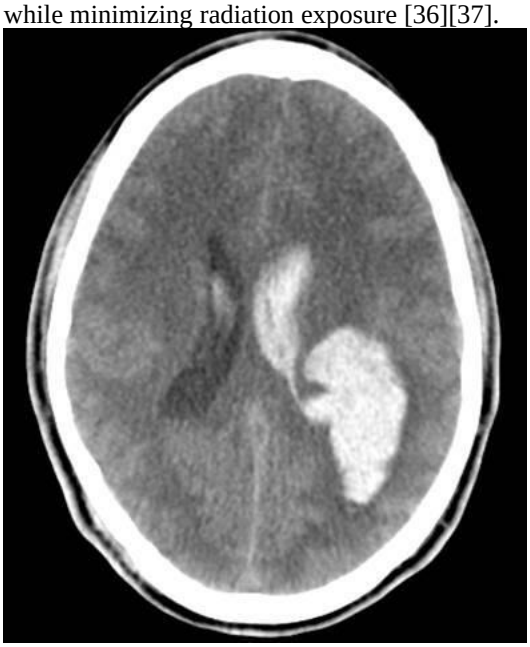

Fig. 1: Brain Trauma.

Additional prognostic and risk stratification tools support clinical decision-making beyond imaging indications. The CRASH (Corticosteroid Randomization After Significant Head Injury) model estimates 14-day mortality and six-month combined death or severe disability in adults with GCS 14 or less

but does not determine imaging necessity. The IMPACT (International Mission for Prognosis and Analysis of Clinical Trials) calculator predicts six-month functional outcomes in moderate to severe TBI, assisting in family counseling and resource allocation, although it is not used to guide acute imaging decisions [39][40]. These tools, in conjunction with validated clinical rules such as NOC, CCHR, and PECARN, facilitate a structured, evidence-based approach to evaluating patients with TBI across age groups and injury severities. Ultimately, the evaluation of TBI requires the integration of patient history, laboratory testing, clinical examination, and imaging guided by validated clinical rules and prognostic models. Such a comprehensive approach ensures early identification of life-threatening intracranial pathology, accurate risk stratification, and optimized patient outcomes while minimizing unnecessary interventions and radiation exposure.

Treatment and Management

The primary objective in the management of traumatic brain injury (TBI) is the prevention of secondary brain insults, which can exacerbate primary injury and worsen neurological outcomes. Effective management relies on a structured algorithm encompassing airway stabilization, maintenance of adequate cerebral perfusion, prevention of systemic derangements such as hypoxia, hypotension, hypercapnia, or hypocapnia, monitoring and management of elevated intracranial pressure (ICP), timely neurosurgical consultation for traumatic intracranial lesions, and the identification and treatment of concomitant life-threatening injuries [41][42][43]. Airway management is paramount in the acute care of TBI patients. Clinicians must evaluate for any conditions that may compromise ventilation, including pneumothorax, hemothorax, or pulmonary contusions. When sedation is required, short-acting agents with minimal hemodynamic effects, such as etomidate or propofol for induction and vecuronium or rocuronium for neuromuscular blockade, are preferred. Endotracheal intubation should be considered in patients with inadequate ventilation, severe head injury, inability to protect the airway, apnea, or severe agitation. Cervical spine precautions must be maintained during intubation, and nasotracheal intubation should be avoided in cases of facial trauma or suspected basilar skull fracture. Target parameters for oxygenation and ventilation include maintaining oxygen saturation above 90%, PaO_2 above 60 mm Hg, and $PaCO_2$ between 35–45 mm Hg. Hypotension must be avoided, as normal blood pressure may not suffice to maintain adequate cerebral perfusion when ICP is elevated. Systolic blood pressure should be maintained above 90 mm Hg and mean arterial pressure above 80 mm Hg. Hypotension in isolated head trauma is uncommon and should prompt evaluation for other causes, such as hypovolemia or hemorrhage.

Mild head trauma constitutes the majority of TBI cases. Patients with minor injuries may be discharged following a routine neurological assessment if no risk factors are present. The use of validated clinical decision rules, including the New Orleans Criteria and the Canadian CT Head Rule, can assist in determining which patients require imaging before discharge. In the absence of immediate imaging, observation for four to six hours may be warranted. Hospitalization should be considered for patients with bleeding disorders, those on anticoagulation or antiplatelet therapy, or individuals with prior neurosurgical interventions. Emerging evidence supports the use of biomarkers such as glial fibrillary acidic protein (GFAP) and S100B to predict intracranial injury and guide the need for CT imaging in patients with mild TBI. The CENTER-TBI study demonstrated that GFAP outperforms clinical characteristics in identifying patients with CT-positive findings, exhibiting a 99% probability of accurate detection [21][44]. Scandinavian guidelines recommend CT imaging in patients with loss of consciousness and repeated vomiting with elevated S100B, seizures, neurological deficits, skull base fractures, anticoagulation use, or age over 65 years with antiplatelet therapy [45]. Moderate to severe head trauma requires intensive prehospital and emergency care to stabilize physiological parameters and prevent secondary injury. Priority interventions include optimizing oxygenation, maintaining systolic blood pressure above 110 mm Hg, ensuring normothermia, correcting coagulopathy, and monitoring ICP using invasive or noninvasive techniques. Prehospital intubation is indicated for patients with a Glasgow Coma Scale (GCS) of eight or lower, whereas in-hospital intubation may be considered for GCS of ten or lower [21]. Oxygen supplementation is recommended, with end-tidal CO_2 maintained between 35–45 mm Hg. Serial assessments of pupillary response and GCS should be conducted at 30-minute intervals. Prophylactic hyperosmolar therapy or prehospital administration of tranexamic acid without evidence of herniation is generally discouraged [47].

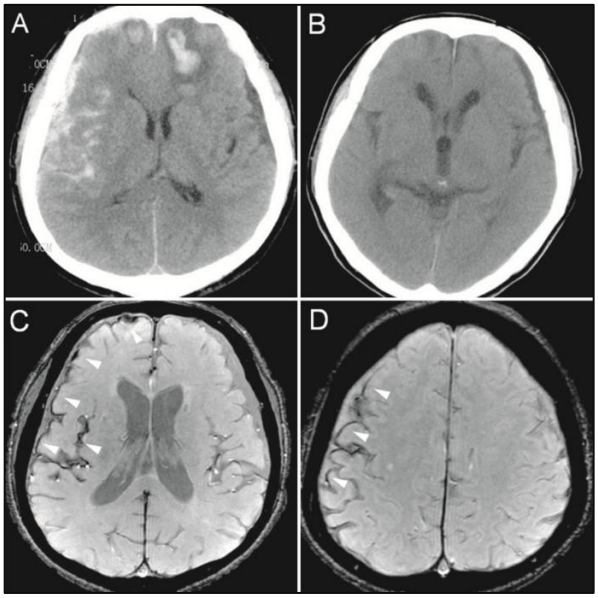

Fig. 2: Brain Trauma.

ICP management is central to preventing secondary injury. Ventricular catheters remain the gold standard for global ICP measurement, while parenchymal monitors offer easier placement but only provide localized measurements and cannot be recalibrated in vivo. Noninvasive techniques such as transcranial Doppler-derived pulsatility indices, tympanic membrane displacement, near-infrared spectroscopy, and optic nerve sheath diameter assessments have significant limitations and inter-rater variability, precluding their routine replacement of invasive monitoring [26]. ICP monitoring is recommended for patients with severe TBI (GCS 3–8) with abnormal CT findings or two or more of the following: age over 40, SBP below 90 mm Hg, or abnormal posturing [27]. Additional indications include initial normal CT scans with subsequent neurological deterioration, evidence of brain swelling, extensive bifrontal contusions, unreliable neurological exams, or inability to interrupt sedation for assessment [26]. The World Society of Emergency Surgery emphasizes a "Hub and Spoke" model for severe head trauma management, highlighting the role of telemedicine, rapid hemodynamic stabilization, multidisciplinary collaboration, and transfer by certified teams. During transfer, patients should remain sedated, intubated, and mechanically ventilated, with the head of the bed elevated to 30–45°. Continuous cardiorespiratory and invasive blood pressure monitoring is required, with targets including SBP above 110 mm Hg, MAP above 90 mm Hg, hemoglobin above 7 g/dL, serum sodium 140–145 mEq/L, SpO_2 above 94%, and $PaCO_2$ between 35–38 mm Hg. Increased sedation, osmotherapy, and short-term hyperventilation may be justified in patients with signs of brain herniation awaiting emergent surgery [48].

Refractory intracranial hypertension is managed using a tiered approach, beginning with evacuation of mass lesions or hematomas causing herniation, followed by physiological neuroprotection, sedation, analgesia, mechanical ventilation, cerebrospinal fluid drainage, osmotherapy with mannitol or hypertonic saline, hyperventilation, targeted hypothermia, barbiturate coma, and ultimately decompressive hemicraniectomy [27]. Early surgical intervention, including craniotomy or decompressive craniectomy, is essential in cases of mass effect, significant midline shift, or refractory intracranial hypertension to prevent irreversible neurological damage. The overarching principle in TBI management is the meticulous prevention of secondary brain injury while optimizing physiological and neurological parameters. This requires an integrated approach encompassing prehospital care, emergency stabilization, advanced monitoring, neuroprotective interventions, and timely surgical management, coordinated across multidisciplinary teams to improve functional outcomes and reduce morbidity and mortality associated with traumatic brain injury [27].

Differential Diagnosis

The evaluation of patients with traumatic brain injury (TBI) requires careful consideration of a broad differential diagnosis encompassing primary brain injuries, secondary complications, and associated conditions that may mimic or exacerbate neurological dysfunction. Primary brain injuries are those that occur at the time of the traumatic insult and include concussions, contusions, diffuse axonal injury (DAI), epidural and subdural hematomas, intraparenchymal hemorrhage, and traumatic subarachnoid hemorrhage. Concussions represent a mild form of TBI characterized by transient neurological dysfunction without structural brain damage detectable on conventional imaging. Contusions involve focal parenchymal bruising, often in the frontal and temporal lobes, and can evolve into hematomas if bleeding continues. Diffuse axonal injury results from shearing forces during rapid acceleration-deceleration events, leading to widespread neuronal injury that may not be immediately apparent on standard CT scans. Epidural hematomas typically arise from arterial bleeding, often associated with skull fractures, and can rapidly progress to life-threatening mass effect. Subdural hematomas, resulting from venous bleeding, may present acutely, subacutely, or chronically, particularly in elderly or anticoagulated patients. Intraparenchymal hemorrhages and traumatic subarachnoid hemorrhages similarly contribute to raised intracranial pressure and secondary injury if not promptly recognized. Secondary brain injuries develop as delayed consequences of the primary insult and include cerebral edema, ischemia or stroke, hypoxic-anoxic injury, and infections such as meningitis or ventriculitis. These complications exacerbate neuronal dysfunction and may be

influenced by systemic factors including hypotension, hypoxia, or impaired autoregulation [27]. Other associated injuries and conditions must also be considered in the differential diagnosis. Skull fractures and cervical spine injuries frequently co-occur with TBI and may require urgent stabilization. Post-concussion syndrome can manifest with persistent cognitive, emotional, or somatic symptoms beyond the expected recovery period. Repeated TBIs increase the risk of chronic traumatic encephalopathy (CTE), characterized by progressive neurodegeneration. Second impact syndrome, although rare, represents catastrophic cerebral swelling following a subsequent injury before recovery from an initial concussion. Psychiatric conditions such as post-traumatic stress disorder or adjustment disorders may complicate the clinical presentation and must be differentiated from persistent neurological deficits to ensure accurate diagnosis and appropriate management.

Prognosis

The prognosis following traumatic brain injury (TBI) is multifactorial and depends on a complex interplay of patient-specific, injury-specific, and management-related factors. Patient age is a significant determinant, with older individuals exhibiting higher mortality and morbidity due to reduced neuroplasticity, preexisting comorbidities, and impaired physiologic reserve. The initial Glasgow Coma Scale (GCS) score remains one of the most robust clinical predictors of outcome, with scores below eight post-resuscitation indicating severe injury and correlating with markedly increased mortality. Other critical prognostic indicators include the presence of anisocoria, reflecting potential uncal herniation; the rapidity of symptom onset following herniation; the existence of polytrauma; systemic insults such as hypoxia or hypotension; and the specific type of intracranial lesion, including epidural or subdural hematomas. Imaging-based scoring systems, such as the Marshall and Rotterdam scores, further stratify risk by evaluating structural damage and cerebral edema on CT imaging. The Rotterdam score, in particular, incorporates the presence of epidural lesions, intraventricular blood, subarachnoid hemorrhage, basal cistern compression, and midline shift. Patients with Rotterdam scores of 1 demonstrate mortality rates as low as 3.2%, whereas scores of 6 are associated with nearly 80% mortality, highlighting the predictive value of early imaging [49][50][51]. Large-scale clinical studies, including the International Mission for Prognosis And Clinical Trial (IMPACT), TRACK-TBI, and CENTER-TBI initiatives, have further refined prognostic models by integrating clinical, demographic, imaging, and laboratory data to improve outcome prediction and guide individualized management [6][52]. Genetic variability has also been identified as a determinant of recovery, with studies estimating that approximately 25% of outcome variance is attributable to genomic factors [21]. These findings underscore the potential for precision medicine approaches in the acute and rehabilitative management of TBI.

Advances in neurocritical care have enabled more sophisticated monitoring techniques, including continuous assessment of brain tissue oxygenation (PbtO2) and cerebral metabolism via microdialysis, alongside traditional intracranial pressure (ICP) and cerebral perfusion pressure (CPP) measurements [53]. Early initiation of structured rehabilitation programs is critical for improving long-term functional outcomes, emphasizing the necessity of a multidisciplinary approach incorporating neurology, critical care, physical therapy, occupational therapy, and cognitive rehabilitation. Emerging machine learning algorithms offer additional tools for outcome prediction, integrating real-time clinical and physiologic data to stratify risk and guide intervention [54][55][56]. In cases of mild TBI, or concussion, the prognosis is generally favorable. Most patients experience resolution of acute symptoms, such as headache, dizziness, or confusion, within three days to two weeks, although a subset may experience persistent postconcussive syndrome. Complications such as second impact syndrome, posttraumatic epilepsy, and chronic traumatic encephalopathy (CTE) remain rare but carry significant morbidity and mortality [57][58]. Sports-related concussions in adolescents demonstrate similar recovery trajectories between males and females, though symptom presentation may differ, with females more commonly reporting neurobehavioral and somatic symptoms and males experiencing cognitive deficits, including confusion and amnesia [57][59][60]. Return-to-play protocols following sport-related concussions are designed to mitigate the risk of re-injury and long-term sequelae. Consensus statements from professional organizations, such as the American Medical Society for Sports Medicine (AMSSM), recommend a structured six-step progression, with each step lasting approximately 24 hours. Medical clearance should be obtained at the completion of the protocol to ensure safe reintegration into physical activity and sports [61]. Overall, TBI prognosis is determined by a combination of acute clinical presentation, structural injury burden, systemic factors, genetic influences, and adherence to evidence-based management and rehabilitation strategies. While mild injuries generally have favorable outcomes, severe TBI continues to carry significant risk of mortality and long-term functional impairment. Early identification of high-risk patients through validated clinical and imaging-based scoring systems, coupled with targeted interventions and interdisciplinary rehabilitation, remains the cornerstone of improving survival and quality of life following traumatic brain injury.

Complications

Traumatic brain injury (TBI) can result in a wide spectrum of complications affecting multiple organ systems, ranging from immediate life-threatening conditions to long-term sequelae that

significantly impair quality of life. One of the most severe outcomes is brain death, which represents the irreversible cessation of all brain activity and requires immediate recognition for appropriate clinical and ethical management. Chronic traumatic encephalopathy (CTE) is increasingly recognized among military personnel and athletes exposed to repetitive head trauma, leading to progressive cognitive decline, behavioral changes, and neuropsychiatric disorders [62]. Central nervous system infections, including meningitis and ventriculitis, may occur secondary to open skull fractures or cerebrospinal fluid (CSF) leaks [63][64], with CSF leaks also increasing the risk of ascending infections. Cognitive sequelae, such as dementia, may manifest months to years following injury, particularly in patients with repeated head trauma or severe initial insult [65]. Vascular and thrombotic complications are common, with deep vein thrombosis (DVT) representing a significant risk due to prolonged immobility and impaired autonomic regulation. Neuropsychiatric consequences, including depression and post-traumatic stress disorder (PTSD), are frequently observed, reflecting the psychological impact of both injury and hospitalization [66]. Dysregulation of electrolytes may result in conditions such as cerebral salt wasting or the syndrome of inappropriate antidiuretic hormone secretion (SIADH), both of which can exacerbate cerebral edema and worsen neurological outcomes [67][68]. Endocrinopathies, particularly hypopituitarism, may develop due to pituitary damage and contribute to metabolic and hormonal imbalances [69]. Structural and hemodynamic complications include hydrocephalus, malignant cerebral edema, neurogenic pulmonary edema, and neurovascular injuries, all of which can threaten survival if not promptly identified and managed. Paroxysmal sympathetic hyperactivity, characterized by episodes of tachycardia, hypertension, and hyperthermia, may further complicate the course of TBI [71]. Additional complications include pneumocephalus [72], seizures, spasticity [73], stunned myocardium syndrome [74], vasospasm [75], and an increased risk of substance use disorders. Collectively, these complications underscore the necessity of multidisciplinary monitoring and long-term follow-up for patients with TBI, emphasizing proactive prevention, early detection, and timely intervention to mitigate morbidity and improve functional outcomes.

Patient Education

Prevention remains the cornerstone of reducing the incidence and severity of traumatic brain injuries (TBI). Given that the majority of TBIs result from accidental trauma, educating patients and the public on safety measures is paramount. Motor vehicle collisions are a leading cause of TBI, and although not entirely preventable, their risk can be significantly mitigated through adherence to safety protocols. Patients should be counseled to wear seatbelts at all times, avoid distracted driving, abstain from alcohol or drug-impaired driving, and ensure the appropriate use of child safety seats and booster seats based on age and size. For bicyclists and motorcyclists, consistent helmet use is critical to prevent severe head injuries. Sports-related TBIs are also increasingly recognized, and individuals recovering from a prior concussion or head trauma should be cautioned against premature return to athletic or high-risk activities. Recurrent injuries can lead to cumulative neurological damage, prolonged symptoms, and even permanent deficits, emphasizing the importance of patient adherence to return-to-play or activity guidelines. For patients recovering from TBI, education must extend beyond prevention to encompass posttrauma recovery, which involves physical, cognitive, and emotional rehabilitation. Patients and caregivers should be informed about common posttraumatic complications such as persistent headaches, dizziness, memory deficits, mood disturbances, and sleep disorders. Psychiatric and neurological sequelae, including depression, anxiety, and suicidal ideation, are prevalent, necessitating proactive discussion and monitoring. Patients should be encouraged to seek professional support when experiencing any neuropsychiatric symptoms and to engage in structured follow-up care. Education should also include guidance on managing daily activities safely, recognizing warning signs of complications, and understanding the long-term recovery trajectory. By emphasizing prevention, adherence to safety protocols, and early recognition of complications, patient education plays a critical role in mitigating the consequences of TBI and supporting holistic recovery [75].

Enhancing Healthcare Team Outcomes

Optimal management of traumatic brain injury requires a coordinated, interprofessional approach, as outcomes are closely linked to the collaboration of multiple healthcare disciplines. Case managers and social workers provide critical support in navigating post-injury care, including coordinating appointments, arranging rehabilitation services, facilitating insurance coverage, and connecting patients with community resources. Occupational therapists focus on improving functional independence, particularly in activities of daily living, and may recommend home modifications or assistive devices to optimize patient safety and autonomy. Physical therapists concentrate on restoring strength, endurance, coordination, and mobility, while also providing training in the use of adaptive equipment to maintain independence. Speech-language pathologists evaluate and treat deficits in communication and swallowing, which can significantly affect quality of life following TBI. Nurses are integral across all stages of care, from acute monitoring in intensive care units to ongoing education and support during

rehabilitation. They play a vital role in early detection of complications, medication administration, and patient and caregiver education. Physiatrists, or rehabilitation physicians, oversee the rehabilitation process, determine eligibility for intensive therapy programs, and coordinate care plans to optimize functional recovery. Primary care providers ensure long-term follow-up, monitor chronic complications, and coordinate specialist referrals. Neurologists manage the spectrum of posttraumatic neurological sequelae, while neurosurgeons perform emergent and elective surgical interventions as necessary. Neuropsychologists provide comprehensive cognitive assessments, guiding decisions regarding a patient's ability to manage personal, financial, and medical responsibilities. Pharmacists contribute by reviewing medication regimens, monitoring potential drug interactions, and counseling patients on adverse effects and adherence strategies. The synergy of this interprofessional team is essential to maximize functional recovery, minimize complications, and enhance overall patient outcomes. Effective communication among team members ensures timely interventions, individualized rehabilitation strategies, and continuity of care. Patients with TBI benefit from this coordinated approach, as it integrates medical management, cognitive and physical rehabilitation, psychosocial support, and patient education into a unified care pathway. By leveraging the expertise of each discipline, the healthcare team can address the multifaceted needs of TBI patients, promoting recovery, safety, and long-term quality of life. Interprofessional collaboration is therefore indispensable in the successful management and rehabilitation of individuals who have sustained traumatic brain injuries [76].

Conclusion:

Traumatic brain injury remains one of the most complex and impactful conditions managed in emergency and critical care settings. The article emphasizes that optimal outcomes depend on early recognition, rapid stabilization, and prevention of secondary insults such as hypoxia, hypotension, and elevated intracranial pressure. Evidence-based clinical rules—including CCHR, NOC, and PECARN—play a crucial role in guiding imaging decisions, preventing unnecessary CT scans while ensuring detection of clinically significant injuries. For moderate and severe TBI, invasive monitoring, targeted physiological parameters, and timely neurosurgical intervention remain central pillars of care. Long-term recovery hinges on a multidisciplinary rehabilitation framework integrating physical, cognitive, occupational, and speech-language therapies, along with psychosocial support for patients and families. The article highlights that complications such as cognitive decline, neuropsychiatric disorders, endocrine dysfunction, and chronic traumatic encephalopathy require ongoing monitoring and tailored interventions. Ultimately, improving outcomes requires a coordinated system of care—from prehospital stabilization to long-term rehabilitation—supported by emerging tools such as biomarkers, advanced imaging, and predictive models. By integrating clinical expertise, structured protocols, and interdisciplinary collaboration, healthcare teams can significantly reduce morbidity, enhance functional recovery, and improve quality of life for individuals affected by TBI.

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