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ORIGINAL RESEARCH

Critical care management in military warfare and blast-induced neurotrauma: Lessons from the Thai–Cambodian border conflict

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ABSTRACT:

Background: To describe the organization, delivery, and outcomes of critical care for combat casualties and blast-induced neurotrauma during active hostilities along the Thai–Cambodian border, with emphasis on operational strategies applicable to high-risk environments.

Method: This retrospective observational operational report reviews casualty management at Prasat Field Hospital between 24 July and 1 August 2025. Clinical data were obtained from hospital records, ICU logs, surgical reports, and Emergency Operations Center (EOC) documentation. All military and civilian casualties presenting alive during the study period were included.

Result: A total of 144 casualties were treated, including 32 patients requiring intensive care. Thirteen patients underwent emergent surgical intervention for severe injuries, including intracranial hemorrhage, major vascular trauma, airway disruption, and complex polytrauma. Integrated damage-control surgery, forward critical care capability, and coordinated MEDEVAC resulted in zero in-hospital mortality among patients who reached definitive care.

Conclusion: Effective critical care in conflict settings depends on forward deployment of ICU capability, structured triage, multidisciplinary coordination, and rapid evacuation pathways. Blast-induced neurotrauma requires early recognition, prevention of secondary injury, and sustained neurocritical care. Experience from the Thai–Cambodian border conflict highlights the evolving role of field hospitals as integrated trauma–critical care centers in modern warfare.

Trial registration: TCTR20240828001

Keywords: Military medicine; Critical care; Blast-induced neurotrauma; Combat casualty management; Trauma triage; Battlefield medicine; Thailand–Cambodia border conflict

INTRODUCTION

Military conflict produces a high burden of critically injured patients under conditions of time pressure, resource limitation, and operational threat. Modern warfare, characterized by high-energy explosives and complex blast environments, has increased both the frequency and severity of blast-related trauma, particularly blast-induced neurotrauma (BINT). During the Thai–Cambodian border conflict in July–August 2025, civilian and military healthcare teams jointly managed 144 combat casualties at Prasat Field Hospital in Surin Province, Thailand.

This manuscript reviews the organization and delivery of critical care in a wartime setting and examines clinical approaches to BINT, integrating operational experience from Prasat Field Hospital with contemporary evidence-based trauma and neurocritical care principles. The objective is to provide practical insights applicable to both military operations and civilian disaster preparedness.

CRITICAL CARE IN MILITARY WARFARE

Echelons of military medical care

Combat casualty care is structured across five escalating echelons of medical response, ranging from self-aid at the point of injury to definitive rehabilitation and strategic evacuation. During the 2025 conflict, Prasat Field Hospital functioned as an integrated Role II+/III facility, enabling advanced resuscitation, damage-control surgery, and intensive care within approximately 50 km of the frontline (Table 1). This proximity allowed critically injured patients to receive ICU-level support during the early post-injury phase, a period known to be decisive for survival and neurological outcome.

Command and coordination

An Emergency Operations Center (EOC) was established to coordinate real-time intelligence from military command, civil defense authorities, and hospital leadership. The EOC oversaw casualty flow, staff safety, infrastructure protection, and contingency planning. During a false alarm involving suspected hostile fire on 25 July 2025, rapid lockdown and communication protocols prevented panic and disruption of clinical care. This event informed subsequent evacuation and shelter-in-place procedures and highlighted the importance of structured command-and-control systems in sustaining critical care operations under threat (Table 1).

Critical care resource management

Sustaining ICU operations in a combat environment required rapid expansion of capacity and flexible use of resources. Portable ventilators, battery-powered monitors, and modular ICU layouts allowed scaling of critical care beds. Damage-control resuscitation principles were applied using whole blood and balanced component therapy, while rapid postoperative turnover ensured availability for incoming trauma and neurocritical patients. A modular supply chain supported from Suranaree Camp ensured continuous access to sterile equipment, medications, oxygen, and power.

KEY MESSAGES:

- Rapid triage and structured critical care protocols are essential in military conflict zones to optimize survival and outcomes for combat casualties.
- Blast-induced neurotrauma requires early recognition and aggressive neurocritical management, including prompt imaging, surgical intervention, and ICU support.
- Multidisciplinary teamwork and coordination between military and civilian healthcare teams significantly enhance patient outcomes in high-casualty scenarios.
- Preparedness, training, and robust emergency response systems in hospitals near conflict zones are critical for effective management of both military personnel and civilian casualties.

METHODS

Study design and setting

This study is a retrospective observational operational report with integrated narrative review elements. It describes the organization, clinical management, and outcomes of casualties treated at Prasat Field Hospital during active hostilities along the Thai–Cambodian border between 24 July and 1 August 2025.

Participants

All military and civilian casualties presenting alive to Prasat Field Hospital during the study period were included (n = 144). Patients declared dead on arrival or transferred without sufficient clinical documentation were excluded.

Data sources

Data were obtained from hospital medical records, emergency operations center (EOC) reports, surgical logs, ICU records, and direct operational observations by deployed medical personnel.

Ethical considerations

This study was conducted in accordance with Royal Thai Army medical regulations. All patient data were anonymized prior to analysis. Formal institutional review board approval was waived as this work constitutes a retrospective operational report using de-identified data collected during military medical operations.

BLAST-INDUCED NEUROTRAUMA (BINT)

Overview

Modern conflicts have demonstrated that blast exposure produces complex injury patterns that extend beyond focal head trauma. Advances in explosive weaponry and improved battlefield survival have increased the number

of patients living with acute and chronic consequences of BINT. Blast exposure may result in immediate death, rapidly evolving neurologic injury, or delayed manifestations that appear hours to years after the event. BINT represents a distinct clinical entity in which primary blast forces interact with systemic physiologic disturbances and concurrent multi-organ trauma, necessitating coordinated critical care management [1-5].

Physics and classification of blast injury

Detonation generates a high-energy blast wave consisting of an initial overpressure phase, a subsequent underpressure phase, and a high-velocity blast wind. Injury severity is influenced by peak overpressure, duration of exposure, distance from the blast, environmental confinement, and reflective surfaces, factors frequently encountered in jungle terrain, villages, and fortified structures along the Thai–Cambodian border.

Blast injuries are traditionally classified into primary, secondary, tertiary, quaternary, and quinary effects, often occurring simultaneously in "blast-plus" scenarios (Table 2). These overlapping mechanisms complicate early diagnosis and increase the risk of delayed neurologic deterioration.

Mechanisms of blast–body interaction

Blast waves interact with the human body through spallation at tissue interfaces, inertial effects from differential tissue acceleration, and implosion phenomena in gas-containing structures. Both high-frequency and low-frequency wave components contribute to microvascular disruption, diffuse axonal injury, and gray–white matter interface damage. These mechanisms explain why clinically significant neurotrauma may occur even in the absence of overt cranial injury.

PATHOPHYSIOLOGY OF BINT: IMPLICATIONS FOR CRITICAL CARE

Primary injury mechanisms

Primary blast forces transmit kinetic energy through the skull, producing rapid brain deformation and vascular shear. In severe cases, this results in subarachnoid hemorrhage, intracerebral hematoma, or malignant cerebral edema. In mild BINT, microscopic axonal and membrane disruptions may occur without loss of consciousness, yet predispose patients to delayed cognitive and vestibular deficits.

Blast exposure also triggers profound autonomic responses. Sudden thoracic overpressure can provoke vagovagal reflexes leading to apnea, bradycardia, and hypotension, while simultaneous sympathetic activation increases myocardial oxygen demand. These competing physiologic responses may mimic severe traumatic brain injury or shock in the field.

A rapid hydrodynamic pressure pulse transmitted through the venous system into the cerebral circulation contributes to endothelial activation, blood–brain barrier disruption, and early cerebral edema, even when the head is not directly impacted.

Secondary and chronic injury cascades

Within minutes to hours, secondary injury pathways emerge, including metabolic failure, oxidative stress, inflammatory activation, and progressive edema. If unmitigated, these processes exacerbate neuronal injury and worsen outcomes. Chronic sequelae may include persistent neuroinflammation, axonal degeneration, tau accumulation, cognitive decline, mood disorders, and neuroendocrine dysfunction.

Table 1. Combat casualty care operates across five levels of medical response.

Echelon of care	Capabilities	Example in Thai context (2025 conflict)
Role I	Self-aid, buddy aid, combat medic	Battlefield medical stations
Role II	Forward resuscitation, limited surgery	Mobile Army Surgical Unit
Role II+ / III	Damage control surgery, intensive care	Prasat Hospital (civil–military integrated)
Role IV	Definitive care, rehabilitation	Phramongkutklao Hospital
Role V	Strategic evacuation	National tertiary hospitals

The integration of Prasat Hospital as a Role II+ trauma node enabled advanced resuscitation and critical care within 50 km of the frontline.

Table 2. Blast-induced neurotrauma results from four synergistic mechanisms.

Injury type	Mechanism	Clinical manifestations
Primary	Shockwave propagation through brain tissue	Diffuse axonal injury, microhemorrhage
Secondary	Shrapnel or debris impact	Penetrating head wounds
Tertiary	Acceleration–deceleration	Contusion, skull fracture
Quaternary	Thermal or toxic exposure	Burns, inhalation injury

These findings reinforce that BINT is a whole-body injury, influenced by systemic hypoxia, hypotension, inflammation, and metabolic derangements, all of which are modifiable targets in critical care.

SYSTEMIC MODIFIERS OF BINT

Air embolism

Air emboli generated at air–fluid–tissue interfaces represent an underrecognized contributor to neurologic deterioration after blast exposure. Experimental and clinical observations demonstrate rapid embolic migration into the cerebral circulation, presenting as sudden hypoxia, neurologic decline, or cardiovascular collapse. Management priorities include high-flow oxygen, cautious ventilatory strategies, appropriate positioning, and rapid evacuation to advanced care.

Systemic inflammation

Blast trauma induces a systemic inflammatory response characterized by cytokine release, complement activation, and endothelial permeability. In the presence of blood–brain barrier disruption, circulating inflammatory mediators and leukocytes exacerbate neuroinflammation, increase intracranial pressure, and worsen both early and late neurologic outcomes. Early hemorrhage control, normoxia, normothermia, and judicious fluid resuscitation are therefore critical components of neuroprotection.

Diagnosis and management of BINT in austere environments

Diagnosing BINT in combat settings is challenging due to environmental chaos, competing injuries, and underreporting of symptoms. A high index of suspicion is required following close-range or confined-space blast exposure. Structured symptom assessment, focused neurologic and cardiopulmonary examination, targeted imaging, and laboratory evaluation support early identification. Because symptoms may resolve acutely yet recur later, long-term surveillance is essential.

Moderate and severe BINT typically occur as part of polytrauma. Early management prioritizes prevention of secondary injury through aggressive airway protection, maintenance of oxygenation, and avoidance of hypotension. Hypertonic saline was favored for suspected intracranial hypertension due to its combined effects on intravascular volume and cerebral edema, while large-volume crystalloid resuscitation was avoided.

Far-forward neurosurgical capability enabled timely decompressive procedures for refractory intracranial hypertension and space-occupying lesions. Combat experience consistently demonstrates improved outcomes with early surgical intervention combined with structured neurocritical care.

During the study period, 144 casualties were treated, with 32 requiring ICU admission. Integrated damage-control surgery, critical care, and coordinated MEDEVAC resulted in zero in-hospital mortality among patients who reached the ICU. Overall survival to transfer or discharge was 85% (Table 3). Average ICU length of stay was 2.6 days, reflecting rapid stabilization and evacuation to rear facilities.

The Incident Command System was adapted to include a dedicated Critical Care Operations Branch, enabling real-time tracking of ICU capacity, ventilator availability, blood supply, and evacuation status (Table 4). This structure ensured synchronization between surgical, critical care, and evacuation teams during mass casualty surges.

Operational and psychosocial dimensions

Sustaining morale and performance among healthcare personnel was essential. Leadership communication, structured briefings, mental health integration, and after-action reviews supported resilience. Despite ongoing security threats, the majority of staff voluntarily remained on duty, reflecting strong professional commitment.

Table 3. Case profile and clinical outcomes (Prasat Hospital, July–Aug 2025).

Injury type	No. of cases (n=144)	Major procedures performed	Survival (%)
Penetrating blast injury	52	Debridement, vascular repair	86
Blunt/Polytrauma	47	Orthopedic fixation, DCR	89
Neurotrauma (incl. BINT)	33	Craniotomy, ICP management	79
Burns / inhalation	12	Airway stabilization, fluid therapy	92

The overall survival rate was 85%, attributed to rapid evacuation and multidisciplinary critical care collaboration.

Table 4. The Incident Command System (ICS) was adapted to include a Critical Care Operations Branch.

Command element	Responsibility
Medical director	Oversight, triage prioritization
Critical care chief	Ventilator allocation, ICU management
Logistics officer	Power, oxygen, temperature control
EOC liaison	Data communication, MEDEVAC coordination

PHYSICS OF BLAST

Detonation and blast wave generation

Detonation is the rapid conversion of an explosive solid or liquid into gas, releasing energy that creates a region of intensely compressed air—the blast wave. This wave travels outward at supersonic speeds and is characterized by [6-10]:

1. Overpressure phase, a sudden rise in atmospheric pressure
2. Underpressure phase, a transient pressure drop following the initial peak
3. Blast wind, a high-velocity mass movement of air capable of causing traumatic amputation, disintegration, or displacement of the body

Determinants of blast injury severity

In practical battlefield settings such as the jungle, villages, and fortified encampments of the Thai–Cambodian border, the severity of blast injury depends on [11-14]:

1. Peak overpressure (690–1724 kPa / 100–250 psi may be lethal)
2. Duration of the overpressure pulse
3. Explosion medium (air, water, confined space)
4. Distance from the blast, with intensity decreasing by the cube of the distance
5. Environmental reflections, which may multiply blast pressures two- to ninefold due to walls, terrain, vehicles, or bunkers

These factors contribute to highly variable injury patterns. In the Thai–Cambodian conflict, terrain-induced reflection and confinement often exaggerated blast effects, producing injuries more severe than anticipated from explosive yield alone.

Classification of blast effects

Explosions produce five categories of injury-relevant effects [15-20]:

1. Primary blast effects
   - o Caused by the direct interaction of the blast wave with the body
   - o Includes pulmonary barotrauma, tympanic membrane rupture, and BINT
2. Secondary blast effects
   - o Result from debris, fragments, or projectiles accelerated by the explosion
3. Tertiary blast effects
   - o Due to bodily displacement or acceleration–deceleration forces
4. Quaternary blast effects
   - o Includes burns, inhalational injury, and other thermal or chemical exposures
5. Quinary blast effects
   - o Arising from postdetonation contaminants such as fuel residues, particulate metals, or toxic byproducts

Complex injury patterns, often termed "blast plus" scenarios, were frequently encountered in the Thai–Cambodian border region, where fragmentation, structural collapse, flash burns, and inhalation injuries occurred simultaneously. Such multimodal trauma complicates early diagnosis of BINT and increases the challenges of frontline critical care.

Mechanisms of blast–body interaction

When a blast wave strikes a living body, part of the wave is reflected at tissue boundaries while another portion propagates through tissue as a tissue-transmitted shock wave (Table 2). Three mechanical phenomena drive injury [21-25]:

1. Spallation

Occurs at interfaces between tissues of differing density. The reflected compression wave may cause fragmentation or formation of "spall" particles, particularly at bone–soft tissue interfaces.

2. Inertial Effects

Different tissues accelerate at different rates depending on density. These unequal forces can shear, stretch, or rupture tissues, especially organs suspended within cavities.

3. Implosion (Cavitation)

Gas-containing structures (lungs, intestines, brain vasculature) undergo compression followed by explosive re-expansion of gas bubbles, causing microvascular and parenchymal damage.

Frequency-dependent effects

Recent evidence indicates that:

- High-frequency, low-amplitude waves preferentially damage tissues with abrupt density transitions (e.g., lung air–blood interface, cerebral vasculature).
- Low-frequency, high-amplitude waves generate shear forces that disrupt tissue planes such as the gray–white matter junction.

Understanding these mechanisms is critical for anticipating delayed neurologic deterioration and designing appropriate triage and monitoring strategies in battlefield settings [26-28].

CRITICAL CARE MANAGEMENT IN MILITARY WARFARE: LESSONS FROM THE THAI–CAMBODIAN BORDER CONFLICT

The Thai–Cambodian border conflict provided essential insights into the early recognition, stabilization, and evacuation of blast-injured casualties, especially those with suspected neurotrauma.

Key principles of battlefield critical care

Key principles of battlefield critical care

1. Early Identification of BINT
   - o Symptoms may be subtle or overshadowed by external injuries.
   - o Continuous reassessment is essential due to risk of delayed deterioration.
2. Airway and Breathing Management
   - o Blast lung requires high suspicion and cautious ventilation strategies to prevent barotrauma.
   - o Oxygenation and avoidance of hypoxia are central to preventing secondary brain injury.
3. Circulatory Stabilization and Hemorrhage Control
   - o Rapid control of external bleeding, judicious fluid management, and prevention of hypotension are priorities.

   - o Permissive hypotension must be balanced carefully in patients with suspected BINT.
4. Prevention of Secondary Brain Injury
   - o Control of hypoxia, hypotension, hypothermia, and agitation.
   - o Early analgesia and sedation when appropriate.
5. Forward Deployment of Medical Personnel
   - o Having trained medics and rapid stabilization teams placed close to frontline units significantly improved outcomes.
6. Evacuation and Prolonged Field Care
   - o Delays imposed by terrain highlighted the importance of:
     - portable monitoring equipment
     - early neuroprotective measures
     - telemedicine consultation with neurosurgical specialists
7. Postexposure Surveillance
   - o The conflict underscored the need for long-term neurocognitive assessment and rehabilitation, as symptoms of BINT often emerged well after initial stabilization.

BINT remains a complex and potentially devastating form of injury in modern warfare. The interplay between blast physics, tissue biomechanics, and systemic responses requires clinicians to be proficient in both rapid battlefield assessment and sustained critical care management. Lessons from the Thai–Cambodian Border Conflict reinforce the importance of early recognition, aggressive prevention of secondary injury, and adaptable evacuation strategies in optimizing outcomes for blast-exposed personnel. These insights continue to inform contemporary military medical doctrine and guide improvements in trauma systems worldwide.

BLAST-INDUCED NEUROTRAUMA: PATHOPHYSIOLOGY FOR CRITICAL CARE MANAGEMENT IN MILITARY WARFARE

Blast-induced neurotrauma results from the complex interaction between blast overpressure, the body, and the brain. In modern military warfare, where improvised explosive devices, high-energy munitions, and enclosed-blast exposures are common, understanding the mechanisms of BINT is crucial for early recognition, triage, and targeted critical care management [29-32].

Phase I: Primary injury mechanisms

Ia. Mechanical injury to brain tissue

The initial blast wave transmits kinetic energy through the skull, causing brain deformation and rapid displacement. In moderate or severe BINT, this may shear bridging veins, causing subarachnoid hemorrhage (SAH), or rupture parenchymal vessels, producing intracerebral hematomas—injuries requiring rapid field recognition and evacuation.

In mild BINT—a frequent presentation in deployed environments—the mechanical strain is subtler but still clinically significant. High-rate shear forces may disrupt:

- Plasma membranes
- Organelle membranes
- Axonal cytoskeleton

These mechanical changes activate intracellular injury pathways such as calpain-2 and caspase-3, leading to degradation of cytoskeletal components and early axonal dysfunction. Evidence suggests that white matter structures (e.g., corpus callosum) may be more susceptible due to their higher stiffness under high-rate loading.

Operational relevance:

These microscopic injuries may present initially as "walking wounded," yet are associated with later cognitive, balance, and visual deficits. Providers must maintain a high index of suspicion in soldiers exposed to blasts, even without LOC or overt hemorrhage.

Ib. Autonomic Nervous System Activation

Blast overpressure transmitted to the thorax can cause sudden pulmonary hyperinflation, stimulating J-receptors and triggering a vagovagal reflex:

- Apnea
- Bradycardia
- Hypotension

These responses are frequently observed seconds after blast exposure. Severe bradycardia may be worsened by a Bezold-Jarisch reflex, mediated by hypoxia or air emboli.

Simultaneously, the sympathetic nervous system releases large amounts of epinephrine and norepinephrine, increasing cardiac oxygen demand.

Operational relevance:

In the field, early autonomic disturbances may mimic severe TBI or shock. Medics must prioritize:

- Airway support and prevention of hypoxia
- Monitoring for bradycardia and hypotension
- Rapid evacuation to higher-level care

Ic. Acute vascular response

Blood acts as a major conduit for blast energy. The venous system—especially the splanchnic veins—absorbs the majority of circulating blood volume and is highly compliant. Blast-induced thoracoabdominal overpressure increases:

- Intrathoracic pressure
- Central venous pressure (CVP)
- Pulmonary arterial resistance

Experimental data demonstrate a rapid, high-pressure hydrodynamic pulse transmitted from the chest into the venous system and upward into the cerebral vasculature. This pulse can produce [33-36]:

- Endothelial activation
- Platelet and neutrophil activation
- Early blood–brain barrier (BBB) dysfunction
- Cyclic cerebral edema

Operational relevance:

This mechanism explains why head injuries can occur even when the blast appears to be "thorax-only." For critical care teams, maintaining optimal cerebral perfusion pressure, minimizing secondary hypoxia, and monitoring for evolving edema are essential.

Phase II: Early secondary injury (minutes to 24 hours)

Once triggered, secondary injury cascades follow patterns similar to other forms of traumatic brain injury but may be more diffuse in blast exposures.

Key early metabolic and cellular disturbances [37-39]:

- Energy failure: decreased glucose, ATP, and magnesium
- Anaerobic shift: increased lactate and lactate/pyruvate ratio
- Membrane pump failure: impaired Na^+/K^+-ATPase
- Oxidative stress: increased reactive oxygen species
- BBB breakdown and early cerebral edema

Inflammatory activation occurs within hours, including:

- Complement activation
- TNF-α and IL-1β release
- Chemokine-driven leukocyte recruitment
- Early astrogliosis

Operational relevance:

Critical care management must address the modifiable secondary injurers:

- Prevent hypoxia and maintain normoxia
- Avoid hypotension; maintain MAP/cerebral perfusion pressure targets
- Control temperature (prevent hyperthermia)
- Manage ICP when indicated
- Early attention to electrolyte abnormalities (Mg^{2+}, Ca^{2+}, Na^+)

Phase III: Chronic consequences of blast neurotrauma

Chronic inflammation and neurodegeneration may continue long after the initial exposure. Persistent activation of microglia, sustained oxidative stress, and long-term inflammatory pathways can contribute to [40-42]:

- Progressive axonal degeneration
- Accumulation of phosphorylated tau
- Cognitive decline
- Mood and behavioral changes
- Endocrine dysfunction through hypothalamic–pituitary axis disruption

Even a single mild blast has been shown experimentally to elevate phosphorylated tau for at least 30 days, suggesting risk for chronic neurodegenerative processes (Figure 1).

Operational relevance:

Military personnel with blast exposure—especially those with repeated exposures—require:

- Long-term neurological and neuroendocrine follow-up
- Screening for cognitive and psychiatric sequelae
- Rehabilitation for balance, motor, and executive function deficits

These chronic effects underpin many post-deployment symptoms, including memory problems, irritability, sleep disruption, and depression.

Understanding BINT as a whole-body injury, not solely a cranial event, is essential. Blast overpressure affects [43-45]:

- Brain tissue mechanics
- Autonomic and cardiovascular physiology
- Cerebral and systemic vascular function
- Metabolic and biochemical homeostasis

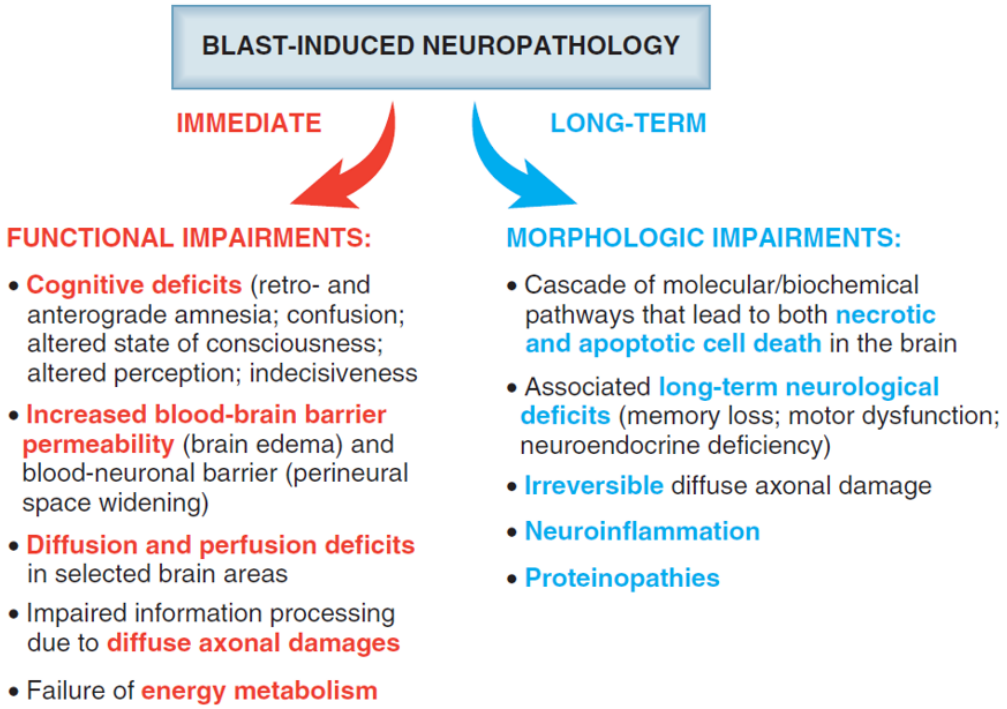

Figure 1. Summary of immediate and long-term impairments in blast-induced neuropathology. (Conceptual summary adapted from existing literatures)

For military critical care teams, optimal outcomes depend on:

1. Early airway & ventilation support to prevent hypoxia
2. Aggressive hemodynamic stabilization to prevent hypotension
3. Monitoring and mitigation of evolving cerebral edema
4. Recognition of autonomic instability post-blast
5. Long-term follow-up for chronic neurologic and endocrine sequelae

SYSTEMIC MODIFIERS OF BLAST-INDUCED NEUROTRAUMA: IMPLICATIONS FOR CRITICAL CARE IN MILITARY WARFARE

Blast injuries occur in an environment of extreme physiologic complexity. Multiple blast effects, primary blast overpressure, secondary fragmentation, tertiary acceleration, and quaternary burns or toxic exposures often interact simultaneously. Because these mechanisms unfold across the entire body, systemic alterations profoundly influence the severity, progression, and outcome of BINT. In the operational setting, early recognition and mitigation of these systemic modifiers is a core objective in military critical care.

Air emboli: A critical but underrecognized threat

Shock-wave transit through air–fluid–tissue interfaces can generate air emboli that propagate through the vasculature and impair cerebral perfusion. Animal models demonstrate emboli entering the carotid artery within seconds of blast exposure, with a cyclic release pattern noted at 0–10 seconds, 2 minutes, and 12 minutes after the blast, timing that correlates with hypoxia and decreased cerebral blood flow.

Human autopsy and clinical reports confirm:

- Massive arterial air embolism
- Venous air emboli within pulmonary vasculature
- Air within the renal interstitium compressing tubular structures

Operational significance:

Air emboli may present as sudden neurologic decline, cardiovascular collapse, or unexplained hypoxia after a blast. In field and critical care environments:

Management priorities

- High-flow oxygen to reduce nitrogen content in bubbles and enhance resorption
- Avoid high PEEP early on if possible, as it may worsen embolic migration
- Positioning: Left lateral decubitus or Trendelenburg may mitigate cerebral embolization for venous air cases
- Rapid evacuation to facilities capable of advanced cardiopulmonary and neurologic support

In severe cases, especially with concurrent lung injury, air emboli accelerate secondary brain injury by exacerbating ischemia and disrupting autoregulation.

Systemic inflammation: A driver of worsening neurotrauma

Blast exposure triggers a system-wide inflammatory surge, driven by tissue disruption and release of potent autacoids (e.g., prostaglandins, leukotrienes, and cytokines). These molecules [46-47]:

- Promote leukocyte recruitment
- Alter T and B lymphocyte activity
- Intensify complement activation
- Enhance endothelial permeability

Importantly, inflammatory cells mobilized systemically can cross a disrupted BBB, amplifying neuroinflammation and accelerating neurodegeneration.

Operational significance:

Systemic inflammation, even from non-CNS injuries, increases ICP, worsens edema, and contributes to early and late cognitive impairment. In deployed critical care settings:

Management priorities:

- Early control of hemorrhage and tissue destruction to reduce inflammatory load
- Tight oxygenation targets to avoid hypoxia-driven cytokine release
- Aggressive prevention of hypotension, a major amplifier of inflammation-induced brain injury
- Normothermia control, as fever accelerates inflammatory cascades
- Judicious fluid resuscitation: Over-resuscitation worsens capillary leak and cerebral edema

Systemic inflammation is one reason polytrauma BINT patients require highly coordinated, multidisciplinary care from point of injury through role for rehabilitation.

BINT VERSUS CIVILIAN TRAUMATIC BRAIN INJURY: IMPLICATIONS FOR MILITARY CARE

Debate continues regarding whether BINT represents a truly distinct clinical entity versus a subtype of TBI. Operationally, blast exposure creates diffuse, variable, and often delayed neurological effects that differ in presentation and progression compared with civilian blunt trauma [47].

Key findings in military studies include:

- More persistent post-concussive symptoms after blast mTBI
- Greater attentional and executive dysfunction
- Distinct metabolic and white matter abnormalities
- Disrupted interhemispheric coordination on EEG
- Diffuse white matter injury on DTI not captured by regional averaging

Operational significance:

Even in mild BINT, soldiers may appear neurologically intact yet have subtle deficits that impair decision-making, reaction time, and threat discrimination, critical functions in combat.

Diagnosis in military and austere environments:

Diagnosing mild BINT is challenging, especially immediately after an explosion when mission requirements, chaotic environments, and patient underreporting limit assessment.

Key principles for operational diagnosis:

1. High suspicion if the blast occurred within close range or in confined spaces.
2. Structured symptom inquiry, including auditory symptoms, respiratory distress, nausea, vertigo, amnesia, or altered consciousness.
3. Focused physical examination, emphasizing:
   - o ENT findings (eardrum rupture, hemotympanum)
   - o Lung examination (rales, pneumothorax suspicion)
   - o Neurologic screening for reaction time, balance, and reflexes
4. Targeted imaging (FAST, chest/brain CT when available)
5. Laboratory evaluation: ABG, lactate, and biomarkers such as UCHL1, GFAP, and SBDP-150
6. Vestibular and audiometric testing when feasible

Symptoms may resolve acutely yet recur months later, highlighting the need for long-term monitoring.

CRITICAL CARE MANAGEMENT OF MODERATE AND SEVERE BINT

Moderate and severe BINT typically occur as polytrauma, frequently with penetrating injuries, thoracic disruption, abdominal trauma, and limb destruction. Early management must target secondary injury prevention, particularly hypoxia, hypotension, coagulopathy, and rising ICP [47].

1. Immediate Prehospital Care
   - Secure airway (early RSI if GCS ≤8 or with signs of airway compromise)
   - Ensure adequate ventilation and oxygenation
   - Prevent hypotension; maintain MAP > 65–70 mmHg
   - Treat tension pneumothorax rapidly (high incidence post-blast)

This phase is decisive in limiting mortality and neurologic decline.

2. Resuscitation and Early Critical Care

Hypertonic saline is preferred for suspected or confirmed elevated ICP because it:

- Expands intravascular volume
- Reduces cerebral edema
- Avoids the detrimental effects of large-volume crystalloids

Key resuscitation guidelines:

- Bolus 3% NaCl for acute ICP crises
- Continuous infusion of 3% NaCl to maintain ICP control
- Avoid hypotension, permissive or otherwise, in TBI
- Avoid hypoxia and hypercapnia
- Early correction of coagulopathy with whole blood, plasma, platelets, and cryoprecipitate to maintain:
  - o INR < 1.5
  - o Platelets > 100,000
  - o Fibrinogen > 150 mg/dL

3. Surgical Management

Far-forward neurosurgical capability is critical in modern warfare.

Indications for emergent cranial decompression:

- Refractory intracranial hypertension
- Herniation syndromes
- Malignant cerebral edema post-blast
- Space-occupying hematomas

Combat experience from OIF/OEF demonstrated that early decompressive craniectomy in-theater, followed by medical evacuation, significantly improves outcomes.

Surgical considerations in blast casualties

- High risk of skull base, orbital, and sinus injuries
- Frequent CSF leak and contamination
- Delayed vascular injuries (AVFs, pseudoaneurysms, dissections)

Despite injury complexity, military outcomes for isolated blast/penetrating TBI surpassed civilian outcomes, attributed to more aggressive ICP monitoring and surgical intervention.

Critical care algorithm for blast-induced neurotrauma

Algorithm for Acute Management of Blast-Induced Neurotrauma in Combat Zones

1. Primary Survey (MARCH/ABCDE):
   - o Massive hemorrhage control → Airway protection → Breathing → Circulation → Disability → Exposure (Figure 2)
2. Airway & Ventilation:
   - o Early intubation; cricothyrotomy if upper airway injury is present
   - o Maintain SpO_2 > 94%, normocapnia ($PaCO_2$ 35–40 mmHg)
3. Hemodynamic Stabilization:
   - o Permissive hypotension (SBP 90 mmHg) until hemorrhage control
   - o Transfusion: 1:1:1 ratio (PRBC:FFP:Platelets) or whole blood
4. ICP and Brain Protection:
   - o Elevate head 30° and administer 3% hypertonic saline (2 mL/kg bolus)
   - o Avoid hypoxia, hypotension, and hyperthermia
5. Surgical Management:
   - o Early damage control craniectomy and debridement
   - o Repair of dural tears and vascular injuries
6. Postoperative ICU Care:
   - o ICP monitoring, sedation with propofol/midazolam
   - o Early nutrition and DVT prophylaxis
   - o Neurological assessment every 2 hours

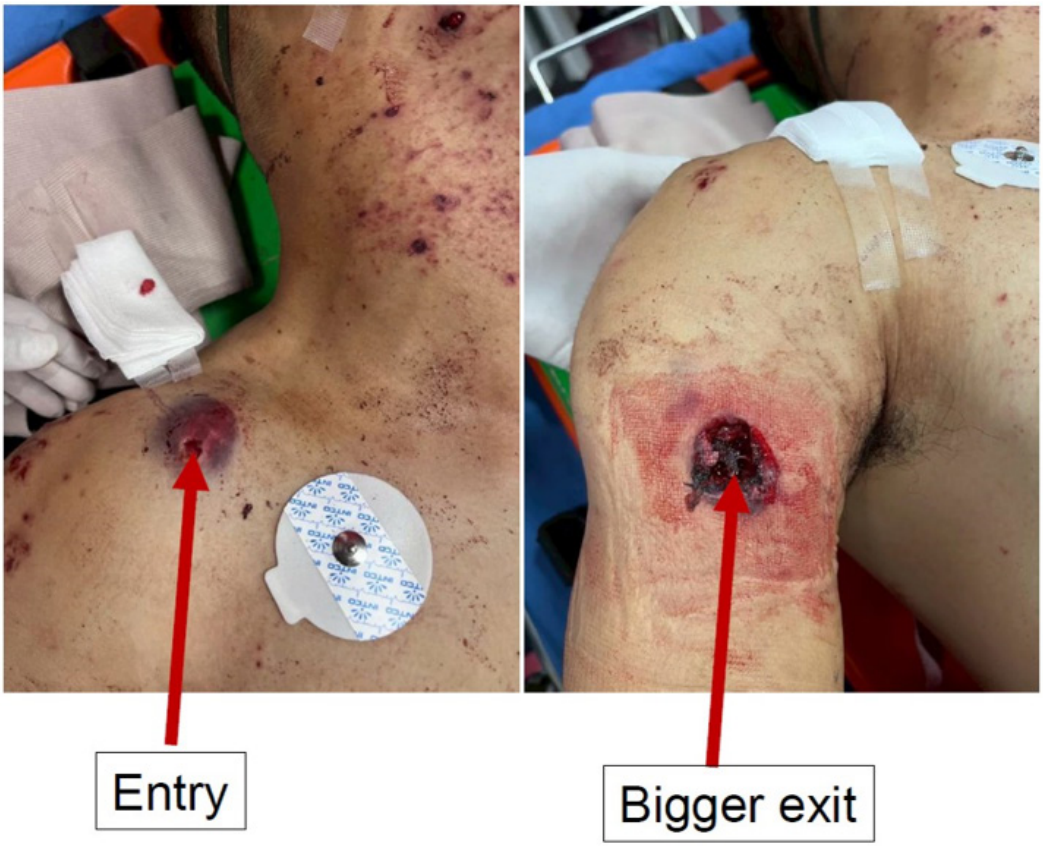

Figure 2. Shrapnel (ballistic) injury illustrating a small entry wound and a larger exit wound. The figure demonstrates the typical pattern of tissue damage caused by high-velocity projectiles, where kinetic energy is transferred to surrounding tissues, creating a wider exit tract and cavitation effect. Surrounding tissue may exhibit hemorrhage, edema, and necrosis depending on the projectile velocity and path. Clarification regarding anonymization and educational use.

Critical lessons learned

A 26-year-old soldier with epidural hematoma, carotid vein tear, and tracheal rupture was evacuated by helicopter to Prasat Hospital. After damage control craniotomy and vascular repair, he was transferred to Phramongkutklao Hospital and survived with intact neurological function.

This case underscores the importance of timely surgical intervention, coordinated airway management, and dedicated neurocritical monitoring in BINT.

Operational and psychosocial dimensions

Human Factors and Morale

Psychological resilience was sustained through:

- On-site leadership reassurance and safety communication,
- Team briefings and after-action reviews,
- Integration of mental health officers within the EOC.

Staff surveys revealed that 420 of 530 personnel (79%) voluntarily chose to remain on duty, reflecting profound commitment to both patients and national duty [47].

CRITICAL CARE MANAGEMENT IN MILITARY WARFARE AND BLAST-INDUCED NEUROTRAUMA: LESSONS FROM THE THAI–CAMBODIAN BORDER CONFLICT

In the 2025 Thai–Cambodian border conflict, the Royal Thai Army established a field hospital in Prasat District, Surin Province, to provide emergency and definitive care for wounded soldiers and civilians.

This report consolidates key events, operational management, critical care delivery under wartime conditions, and medical lessons learned. It draws directly from field logs, surgical records, and firsthand accounts of trauma surgeons, anesthesiologists, critical care teams, and field medical administrators who served in the mission (Table 3).

BACKGROUND AND MISSION OBJECTIVES

Between May and July 2025, recurrent clashes along the Thai–Cambodian border produced numerous combat casualties. The 2nd Army Area, in cooperation with Phramongkutklao Hospital, Suranaree Camp Hospital, and local public health offices, mobilized mobile surgical and critical care units to stabilize, operate on, and evacuate wounded personnel.

Mission objectives

1. Deliver rapid trauma and critical care to military and civilian casualties.
2. Establish an integrated field intensive care system capable of providing ventilation, hemodynamic monitoring, and postoperative stabilization.
3. Reduce combat mortality through effective triage, damage-control surgery, and time-sensitive evacuation.
4. Integrate field medicine with the national referral network through ground and air MEDEVAC.

PRE-DEPLOYMENT PREPARATIONS

Preparation involved a detailed mobilization plan for human resources, equipment, and critical care capability. Personnel from Phramongkutklao and Fort Suranaree hospitals formed multidisciplinary teams composed of:

- Trauma and general surgeons
- Orthopedic and vascular surgeons
- Anesthesiologists and critical care physicians
- ICU nurses and anesthesia nurses
- EMS teams with advanced pre-hospital trauma training
- Biomedical engineers and logistic support units

Critical care–focused assets

- 8 modular operating rooms capable of rapid reconfiguration for multiple cases
- 30 ICU beds, each equipped for mechanical ventilation and invasive monitoring
- Portable ventilators, multi-parameter monitors, defibrillators, and mobile ultrasound units for FAST and echocardiography
- Mobile imaging (digital X-ray, portable CT) for damage assessment
- Point-of-care laboratory for ABG, lactate, and cross-matching
- Blood bank with on-site storage and cold-chain support
- Backup generators and oxygen production units

OPERATIONAL OVERVIEW AND THE CRITICAL CARE CONTINUUM

The Prasat Field Hospital, situated roughly 35 km from the frontline, was close enough to receive casualties within the "golden hour." This location allowed for integration between frontline trauma stabilization and ICU-level post-resuscitative care, forming a continuous chain of survival.

Operational framework

1. Forward Aid Posts (CCP): Initial triage, hemorrhage control, airway protection.
2. Forward Stabilization Unit (Phanom Dong Rak): Pre-ICU care—intubation, fluid resuscitation, tourniquet verification.
3. Prasat Field Hospital:
   - o Advanced trauma life support (ATLS)
   - o Damage-control surgery (DCS)
   - o Postoperative critical care (ICU phase)
4. Rear Hospitals: Definitive reconstruction, rehabilitation, and long-term care.

The system emphasized seamless critical care transition — from prehospital stabilization to field ICU management, ensuring that every patient had continuous hemodynamic and airway support.

Command and control (ICS) for critical care operations

The Incident Command System (ICS) was adapted to incorporate Critical Care Operations Control, ensuring that ICU capacity, ventilator availability, and blood supply were tracked in real time (Table 4).

Key command roles

- Medical Director, strategic and clinical oversight, ICU triage prioritization.
- Critical Care Chief (Field Anesthesiologist-in-Charge), Coordination of ventilator allocation, ICU staffing, and sedation protocols.
- Logistics/Engineering, Maintenance of oxygen generation and climate control for ICU tents.
- Communications & EOC, Live dashboard of patient status and ICU occupancy via LINE/EOC digital network.

This command structure ensured that surgical and critical care activities were synchronized during mass casualty surges.

CRITICAL CARE AND MEDEVAC INTEGRATION

1. Air MEDEVAC (Rotary-Wing Evacuation):

Used for patients with TBI, massive thoraco-abdominal trauma, airway rupture, or uncontrolled bleeding requiring tertiary intervention. Each helicopter was equipped with an advanced life-support stretcher, ventilator, and portable monitor, staffed by an ICU nurse–paramedic pair.

2. Ground MEDEVAC (Convoy):

Used for stabilized patients with continuous monitoring capability. Vehicles were modified with portable suction, oxygen concentrators, and vibration-dampening mounts for stretchers.

Critical care lessons

- The availability of an LZ (Landing Zone) near the field ICU reduced "time-to-ventilation" and "time-to-surgery."
- Preflight stabilization checklists (ABCs, blood pressure >90 mmHg, SpO_2 >92%) minimized in-transit deterioration.

- Continuous telemetry communication between aircraft and EOC facilitated immediate handover upon landing.

BLOOD AND RESUSCITATION LOGISTICS

Massive transfusion capability was central to critical care management. Within one week, the hospital achieved a 250-unit blood reserve through regional mobilization and military–civilian cooperation.

Resuscitation protocols implemented

- Massive Transfusion Protocol (MTP): 1:1:1 ratio of RBC:FFP:Platelets
- DCR: Hypotensive resuscitation strategy to prevent rebleeding
- Use of Tranexamic Acid (TXA) within 3 hours of injury
- Calcium monitoring to prevent transfusion-induced hypocalcemia

Lessons

A responsive blood supply and adherence to DCR principles significantly reduced intraoperative and early ICU mortality.

ICU operations in combat environment

Field ICU setup

Each ICU module consisted of:

- 5–6 beds under negative-pressure-capable tents
- Central monitoring via wired and wireless networks
- Mobile suction and oxygen concentrators
- Temperature control and anti-dust filtration

Clinical focus

- Respiratory management: Mechanical ventilation (SIMV/AC), PEEP optimization for blast lung injuries.
- Hemodynamic monitoring: Arterial lines, central venous catheters, ultrasonography for fluid responsiveness.
- Sepsis prevention: Early antibiotic protocols, sterile zone management.
- Neurocritical care: Continuous ICP monitoring when available; sedation/analgesia titration for TBI.
- Postoperative recovery: Early enteral feeding, pain control, and DVT prophylaxis.

Performance outcome

All patients who reached the ICU alive were stabilized; mortality in the field ICU was 0% within the recorded period, indicating exceptional team coordination and rapid interventions.

TIMELINE HIGHLIGHTS

- 26–28 July 2025: Peak casualty period; 144 total patients, 3 admitted to ICU.
- Most common injuries: Extremity blast trauma (40%), penetrating chest/abdomen wounds (25%), head/neck trauma (20%), burns (5%).
- Average ICU stay: 2.6 days before evacuation or downgrade.
- Notable critical case: Multi-trauma with epidural hematoma, carotid vein tear, and tracheal rupture. Managed with rapid craniectomy, vascular repair, and tracheoplasty. Extubated on ICU day 3 and successfully transferred for rehabilitation — a testament to the synchronized MEDEVAC–OR–ICU system.

CRITICAL LESSONS LEARNED

1. Early Critical Care Saves Lives: Forward-deployed ICU capability transformed survival outcomes.
2. Damage-control surgery + damage-control resuscitation must operate hand-in-hand with continuous ICU support.
3. Ventilator allocation systems should be pre-planned; redundancy (manual bagging, portable ventilators) is vital.
4. Tele-ICU capability (e.g., LINE/EOC communication) enabled real-time consultation from tertiary intensivists.
5. Psychological care is integral to critical care — sedation, pain, and stress management for both patients and staff.
6. Maintenance of sterile zones and infection control in a warzone setting prevents secondary losses.
7. Power and oxygen redundancy (backup generators, compressors) are critical for sustaining ICU life support in unstable zones.

STRATEGIC RECOMMENDATIONS

1. Institutionalize Combat Critical Care Doctrine across the Royal Thai Army Medical Department.
2. Develop modular ICU units deployable within 12 hours, with portable ventilators and integrated monitoring systems.
3. Create a Critical Care Evacuation Team (CCET) trained for in-transit ICU-level care.
4. Implement joint training programs combining surgeons, anesthesiologists, and medics in combat critical care simulation.
5. Establish a digital Critical Care Registry for future data-driven improvement.
6. Provide ongoing mental health and resilience programs for medical personnel exposed to trauma care under fire.

EPILOGUE: THE SPIRIT OF MILITARY CRITICAL CARE

Operation Yutthabodin revealed the evolving face of military medicine, where field hospitals now function not merely as surgical outposts but as advanced critical care units capable of sustaining life under fire.

Every soldier and civilian saved represented the culmination of strategy, technology, and compassion under extreme conditions. The teamwork of Thai military doctors and nurses proved that critical care, when integrated from the frontlines to the tertiary center, is the decisive factor between life and death in modern warfare.

LIMITATIONS

This study has several limitations. First, as a retrospective operational report, it is subject to selection bias, particularly as outcomes reflect patients who survived long enough to reach definitive care. Second, the observation period was limited to a short conflict window, and long-term neurological and functional outcomes were not systematically captured. Third, resource availability and personnel concentration during this specific operation may limit generalizability to other conflict zones or civilian disaster settings. Nevertheless, the findings provide valuable real-world insights into integrated combat critical care and BINT management.

CONCLUSION

The Thai–Cambodian border conflict of 2025 demonstrates the evolving role of field hospitals as integrated trauma–critical care centers. Forward deployment of ICU capability, structured command and control, coordinated MEDEVAC, and adherence to neurocritical care principles resulted in exceptional survival despite austere and hostile conditions.

Blast-induced neurotrauma requires clinicians to look beyond overt head injury and address systemic physiologic disturbances that amplify neurologic damage. Early recognition, prevention of secondary injury, and long-term surveillance are central to optimizing outcomes. Lessons from this operation inform contemporary military doctrine and provide relevant insights for civilian disaster and mass casualty care.

ABBREVIATIONS

CABG: Arterial Blood Gas; ATLS: Advanced trauma life support; AVF: Arteriovenous fistula; BBB: Blood–brain barrier; BINT: Blast-induced neurotrauma; CCET: Critical Care Evacuation Team; CCP: Critical care point; CNS: Central nervous system; CPP: Cerebral perfusion pressure; CT: Computer Tomography; CVP: Central venous pressure; CXR: Chest x-ray; DCR: Damage control resuscitation; DTI: Diffusion Tensor Imaging; DVT: Deep vein thrombosis; EEG: Electroencephalography; EOC: Emergency Operations Center; FAST: The Focused Assessment with Sonography in Trauma; GCS: Glasgow Coma Scale; ICP: Intracranial pressure; ICS: The Incident Command System; ICU: Intensive care unit; IV: Intravenous administration; LZ: Landing Zone; MAP: Mean arterial pressure; MEDEVAC: Medical evacuation; mg: milligram; mmHg: Millimeters of mercury; mTBI: mild Traumatic brain injury; MTP: Massive Transfusion Protocol; NPO: Nothing by mouth; NSS: Normal saline; OIF/OEF: Organisation internationale de la francophonie; Operation Enduring Freedom; $PaCO_2$: Partial pressure of carbondioxide; PEEP: Positive end expiratory pressure; PTSD: Post-traumatic stress disorder; RSI: Rapid sequence intubation; SAH: Subarachnoid haemorrhage; SpO_2: Peripheral O_2 saturation; TBI: Traumatic brain injury; TXA: Tranexamic Acid;

CONFIDENTIALITY

Written informed consent was waived due to the retrospective nature of the study. All patient data used in this study were fully anonymized prior to analysis. Identifiable personal information was removed to ensure confidentiality and compliance with ethical standards governing military and civilian medical records. Data were securely stored within institutional systems accessible only to authorized personnel. The study was conducted in accordance with Royal Thai Army medical regulations and applicable data protection policies.

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