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GUIDELINE

Explosive Weapons Trauma Care Collective (EXTRACCT) Blast Injury Clinical Practice Guideline: Traumatic Brain Injury From Blast and Penetrating Mechanisms in Settings With Variable Resource Availability

Andres M. Rubiano[1,2] | Laura M. Loaiza-Cardona[2] | Ali Msheik[3] | Andrés Salazar Restrepo[2] | Santiago Cardona-Collazos[2] | Andrii Sirko[4] | Ruben Peralta[5,6] | on behalf of the EXTRACCT Clinical Practice Guidelines Collaboration

[1]Neuroscience Institute, El Bosque University, Bogotá, Colombia | [2]MEDITECH Foundation, Cali, Colombia | [3]Neurosurgery Resident, Hamad Medical Corporation, Doha, Qatar | [4]Head of the Center for Cerebral Neurosurgery, Communal Enterprise, Dnipropetrovsk Regional Clinical Hospital Named After I.I. Mechnikov, Dnipro, Ukraine | [5]Trauma Surgery, Hamad Medical Corporation, Doha, Qatar | [6]Department of Surgery, School of Medicine, Universidad Nacional Pedro Henríquez Urena, Santo Domingo, Dominican Republic

Correspondence: Andres M. Rubiano (rubianoam@outlook.com)

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ABSTRACT

Introduction: Blast- and penetrating-mechanism traumatic brain injury (TBI) is increasingly prevalent in settings affected by conflict and explosive violence, including among local casualties in environments where neurosurgical infrastructure, advanced imaging, and critical care resources may be limited or absent. No protocols or guidelines comprehensively address the full spectrum of TBI from blast and penetrating mechanisms across variable resource levels.

Methods: The Explosive Weapons Trauma Care Collective (EXTRACCT) conducted a targeted narrative literature review across key clinical domains, including initial assessment, hemodynamic and airway management, medical treatment of intracranial hypertension, noninvasive neuromonitoring, surgical decision-making, and interfacility transport. Searches were performed in PubMed and supplemented by military, penetrating and trauma system clinical practice guidelines (Joint Trauma System, Brain Trauma Foundation, TCCC). Evidence was synthesized by a multidisciplinary authorship team and consensus was achieved through iterative expert review, following the AGREE II framework.

Results: This document provides stratified recommendations across variable resource contexts. Key recommendations include: early physiological stabilization with avoidance of hypotension and hypoxia; structured neurological assessment using post-resuscitation GCS and pupillometry; noncontrast head CT as the primary imaging modality, with CT angiography for penetrating TBI and high-risk blunt mechanisms; hyperosmolar therapy as first-line treatment for intracranial hypertension; evidence-based sedation, analgesia, and neuromuscular blockade protocols; noninvasive multimodal neuromonitoring when invasive ICP monitoring is unavailable; surgical thresholds for decompressive craniectomy and craniotomy; and standardized transport protocols to minimize secondary brain injury during interfacility transfer.

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Conclusions: This document addresses a critical gap in context-appropriate blast-TBI management for settings with variable resource availability. Strict control of basic physiological parameters, structured clinical decision-making, and early transfer to neurosurgical care remain the primary determinants of outcome in resource-limited environments.

1 | Objectives

This clinical practice guideline aims to provide recommendations for the management of patients with blast-related head injuries in settings where such injuries may be new or uncommon, or settings where personnel, resources, and access to follow-up care may be limited. These guidelines are not intended to replace clinical judgment, but rather to support clinicians who may have limited experience in treating these injuries, and to serve as a complement to the judgment of more experienced providers. The primary goal is to reduce variability in treatment approaches and improve patient outcomes. These recommendations are specifically adapted to environments with limited access to neurosurgical care, advanced imaging, continuous monitoring, and critical care resources. In such settings, early stabilization, prevention of secondary brain injury, and pragmatic decision-making become the primary determinants of outcome.

2 | Background

Explosive injuries are becoming increasingly common across both military and civilian settings. In modern warfare, improvised explosive devices (IEDs) and other high-order munitions have become the leading causes of traumatic injuries. As a result, blast-related trauma has emerged as the signature injury of recent military conflicts, affecting not only combatants but also local casualties [1–4].

However, explosive-related injuries are not limited to combat zones. The use of explosives in terrorist attacks, criminal violence, and accidental detonations has also contributed to an increase in blast-related trauma among local casualties [5–7].

Traumatic brain injuries are frequent among individuals exposed to blasts. In military populations, 10%–20% of deployed personnel sustain a TBI during service, most commonly linked to blast exposure. Among local casualties of terrorist bombings, head trauma represents a significant share of both morbidity and mortality. For example, during the Madrid train bombings and the Oklahoma City bombing, head injuries accounted for 12%–31% of all casualties, and autopsy data revealed that up to 71% of fatalities were due to brain injuries [3–5].

Blast-induced brain injury results from complex and multifactorial pathophysiological mechanisms, commonly categorized into four components: (1) primary injury from rapid transmission of overpressure waves through the skull and cerebral vasculature; (2) secondary injury from shrapnel penetration; (3) tertiary injury from acceleration–deceleration forces; and (4) quaternary injury from thermal, chemical, or other related effects [8, 9].

These components are summarized in Table 1 and Figure 1. The combined impact of these mechanisms contributes significantly to the severity of the primary injuries observed in affected patients [10].

Positive patient outcomes are best achieved through a coordinated chain of interventions aimed at preventing secondary brain insults, particularly hypoxia and hypotension. With this goal in mind, the present document has been developed to compile and present evidence-based interventions providing a structured approach for basic medical and surgical management for nonneurosurgeons, advanced surgical management for neurosurgeons, and considerations during transport specific to injury patterns.

3 | Methods

The CPG development process for EXTRACCT guidelines has been previously described [11]. This guideline addresses a recognized gap in context-appropriate, evidence-based recommendations for an acute management of blast- and penetrating-mechanism TBI in settings where neurosurgical infrastructure,

TABLE 1 | Components of blast-related traumatic brain injury. Adapted from Rosenfeld et al. 2013 [10].

Components of Blast Injury		
Primary blast injury	Transmission of the blast pressure wave	Diffuse/concussive injury and cerebral edema from barotrauma; frequently no external wound; signature blast TBI
Secondary blast injury	Penetration of projectiles	Penetrating TBI, focal lacerations and retained metallic fragments; high risk of vascular injury (pseudoaneurysm, AVF) and intracranial infection
Tertiary blast injury	Acceleration and deceleration effects	Coup–contrecoup contusions, SDH and EDH, diffuse axonal injury, and skull fractures
Quaternary blast injury	Thermal, chemical, and other related injuries	Scalp/facial burns, inhalational and hypoxic–ischemic injury, and systemic/crush effects compounding the brain injury

advanced imaging, and critical care resources may be limited or absent—conditions that characterize many of the civilian health systems most affected by explosive weapons. A targeted narrative literature review was conducted across key clinical domains, including prehospital care, initial assessment, medical management of intracranial hypertension, surgical decision-making, and patient transport. Searches were performed in PubMed and supplemented by specialized literature, including military, penetrating and trauma system clinical practice guidelines (Joint Trauma System, Brain Trauma Foundation, and TCCC). Evidence was synthesized by the authorship team, composed of neurosurgeons, trauma surgeons, and critical care specialists with experience in both high- and low-resource settings. Consensus was achieved through iterative expert review and discussion. The process loosely followed the AGREE II framework within the constraints of a limited and heterogeneous evidence base [12]. Given the limited and heterogeneous body of high-quality evidence for blast- and penetrating-mechanism TBI in austere and conflict settings, the recommendations in this guideline rely substantially on the structured expertise of the authorship team, comprising clinicians with direct experience in resource-limited and conflict environments, rather than on high-certainty evidence. Recommendations should be adapted to the specific resources, personnel, and constraints of each setting, and updated as new evidence becomes available.

4 | Initial Assessment and Early Interventions

The initial management of the patient with brain trauma begins with addressing life-threatening injuries and resuscitation following established trauma protocols such as ATLS, ITLS, or TCCC [13–17]. See Box 1 and Table 2.

BOX 1 | Physiological targets to prevent secondary brain injury.

- SBP > 100–110 mmHg (avoid < 90; blood products first-line)
- SpO_2 > 93%; PaO_2 > 60 mmHg
- $EtCO_2/PaCO_2$ 35–45 mmHg; no routine hyperventilation
- CPP 60–70 mmHg; ICP < 22 mmHg (when measured)
- $PbtO_2$ > 20 (if available)
- Normothermia
- Glucose 70–180 mg/dL
- Head of bed 30°–45°

Securing the airway is crucial in patients with a GCS score ≤ 8, or in those with craniofacial trauma or airway burn signs that may compromise patency. In low-resource settings, where continuous monitoring and rapid reassessment may be limited, a lower threshold for airway protection (including intubation in patients with GCS ≤ 8) is justified to prevent hypoxia and secondary brain injury. The most advanced airway device available with which the provider is trained should be used, in accordance with airway management protocols [13, 14, 17, 18]. The need for motion restriction should be evaluated using validated clinical decision rules such as the NEXUS criteria or the Canadian C-Spine Rule; cervical collars should be applied only when indicated and without excessive tightness. The head of the bed should be elevated at 30°–45° to avoid aspiration, or placed in reverse Trendelenburg if spinal precautions are in place, and in cases of penetrating cervical injuries, a cervical collar should NOT be applied [14, 19].

The scalp should be examined carefully for open wounds. If a wound is identified, external bleeding control should be prioritized, followed by irrigation with normal saline, covering with sterile dressings, and documentation of the trajectory and the presence of any visible foreign bodies. Embedded fragments, foreign bodies, or exposed brain tissue should not be removed (Figure 2); definitive wound cleaning, debridement, and closure should be performed in a controlled surgical setting [20–22].

Only in austere environments where surgical access is delayed should a temporary superficial closure be considered, such as scalp approximation with sutures or staples, and only to control external bleeding; the wound must be reassessed and surgically

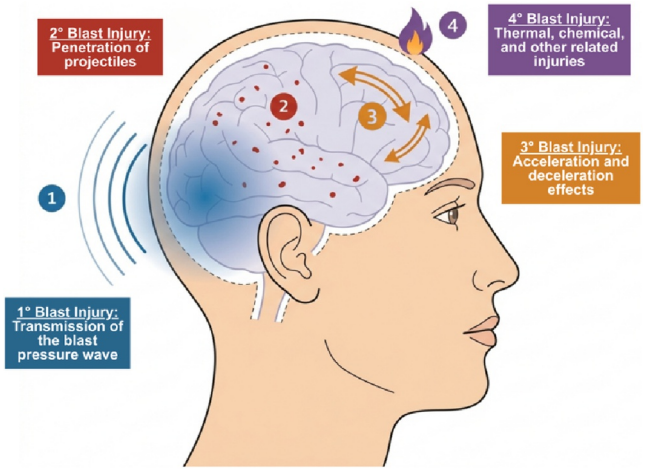


FIGURE 1 | Mechanisms of blast-related traumatic brain injury.

TABLE 2 | Suggested doses for vasopressor therapy. Adapted from Rubiano et al. 2026 [17].

Agent	Noradrenaline	Adrenaline
Presentation	4 mg/4 mL ampoule	1 mg/mL ampoule
Dose	0.05–0.5 μg/kg/min	0.1–2 μg/kg/min
Suggested preparation	4 ampules (16 mg) in 250 mL D5W or NS → 64 μg/mL	4 ampules (4 mg) in 250 mL D5W or NS → 16 μg/mL
Rate (mL/h)	Dose × weight (kg) × 60 ÷ 64	Dose × weight (kg) × 60 ÷ 16

explored once definitive care becomes available [20–22]. Capillary blood glucose should be checked, avoiding levels below 70 mg/dL or above 180 mg/dL [23].

A focused neurological assessment should be carried out to identify critical injuries requiring timely medical or surgical intervention. This assessment includes the Glasgow Coma Scale documented by individual components (Eye, Verbal, Motor), pupil size and reactivity, and the presence of focal neurological deficits such as asymmetric motor responses, limb drift, abnormal posturing, or impaired language comprehension [13, 16–18, 24].

The initial GCS may be misleading in the presence of hypoxia, hypotension, or sedative medications; therefore, the post-resuscitation GCS should be used to inform clinical decisions, prognosis estimation, triage, and neurosurgical consultation. Among its components, the motor response (GCS-M) is the most reliable for assessing neurological progression over time [25, 26].

In low-resource settings, strict control of basic physiological parameters, including oxygenation, blood pressure, glucose, and temperature, has a greater impact on outcomes than advanced monitoring technologies.

5 | Imaging Studies

5.1 | Computed Tomography (CT)

Blast injuries are considered a high-risk mechanism. All patients with suspected TBI from blast exposure should undergo a noncontrast head CT to evaluate for intracranial injury if they exhibit any signs or symptoms of neurologic dysfunction, even if transient, including in cases with GCS 14–15. If there are clinical or radiologic signs of skull fracture (periorbital ecchymosis, Battle's sign, otorrhagia, CSF rhinorrhea), a noncontrast head CT is also indicated. If the initial CT is performed within the first 4 hours after injury and shows any abnormality, a follow-up scan should be obtained at 6–8 hours (see Figure 3 for an example). Additional imaging is recommended at 24 and 72 hours postinjury, or earlier if neurological deterioration occurs [16, 24]. Online Resource 1 illustrates the suggested approach to the interpretation of CT scans, the ABCDzE mnemonic.

If CT is not available, noninvasive devices such as near-infrared spectroscopy (NIRS), pupillometry, and optic nerve sheath diameter (ONSD) ultrasound may be used to detect intracranial hemorrhage or loss of intracranial compliance, serving as triage tools to guide urgency and transfer decisions.

Although blood-based biomarkers are increasingly used in high-resource settings, they are not essential for decision-making in low-resource environments and should not delay imaging or transfer when clinically indicated [10].

5.2 | Vascular Imaging

For penetrating head injuries, in addition to noncontrast head CT, a CT angiogram of the brain should be obtained to assess vascular injuries such as arterial transections, pseudoaneurysms, arteriovenous fistulas, dissections, or thrombosis [14, 27]. In cases of blunt head trauma, clinical decision tools such as the Modified Denver Criteria (Table 3) should be used to identify patients at risk for traumatic cerebrovascular injury (TCVI) who may require CT angiography in addition to standard noncontrast head CT. In centers where vascular studies are available, early implementation of these diagnostic tools is recommended [28, 29].

5.3 | Magnetic Resonance Imaging (MRI)

Brain MRI is not a part of the routine acute evaluation of patients with TBI. It is primarily reserved for the subacute phase in cases with persistent clinical concerns, such as unexplained alterations in consciousness, persistent neurological symptoms, or when intracranial injuries are suspected despite a negative CT scan. MRI findings in this context may also have prognostic value, as they can help predict long-term disability [24, 30, 31].



FIGURE 2 | (a): Patient with explosive and shrapnel-related trauma presenting with exposed cerebral tissue. (b): Patient with thermal injury and shrapnel-related trauma secondary to an explosive event. Photo Credit: Dr. Andres M. Rubiano, Cali, Colombia.

MRI is contraindicated in patients with penetrating or blast-related TBI when an intracranial foreign body is ferromagnetic or of unknown composition. Because metals presumed to be nonferromagnetic may be contaminated with ferromagnetic material, MRI should be avoided in any patient with retained intracranial metal following penetrating injury, and should be considered only when the foreign body is known to be nonmetallic. This caveat is particularly relevant in blast and ballistic mechanisms, in which retained metallic shrapnel is common [32].

6 | Medical Management

6.1 | Use of Tranexamic Acid

In patients with isolated mild to moderate TBI and evidence of intracranial hemorrhage on CT, intravenous tranexamic acid (TXA) should be administered at 2 g (1 g bolus over 10 minutes, then 1 g over 8 hours) within the first 3 hours after injury [33]. Early TXA administration is particularly relevant in low-resource settings, where access to neurosurgical intervention and blood products may be limited. In patients with polytrauma with active internal or external bleeding and concurrent TBI of any severity (including severe TBI), the same TXA regimen should be administered within 3 hours of injury to support systemic hemorrhage control [34]. For patients with isolated severe TBI without significant systemic bleeding, the evidence of benefit is less clear, and clinical judgment is advised.

6.2 | Management and Prophylaxis of Posttraumatic Seizures

Early posttraumatic seizures (PTS) are associated with TBI severity and correlate with hemodynamic changes, increased cerebral metabolic demand, elevated intracranial pressure, and higher morbidity and mortality [24, 35–41]. Indications, agents, dosing, and duration for seizure prophylaxis are summarized in Box 2.

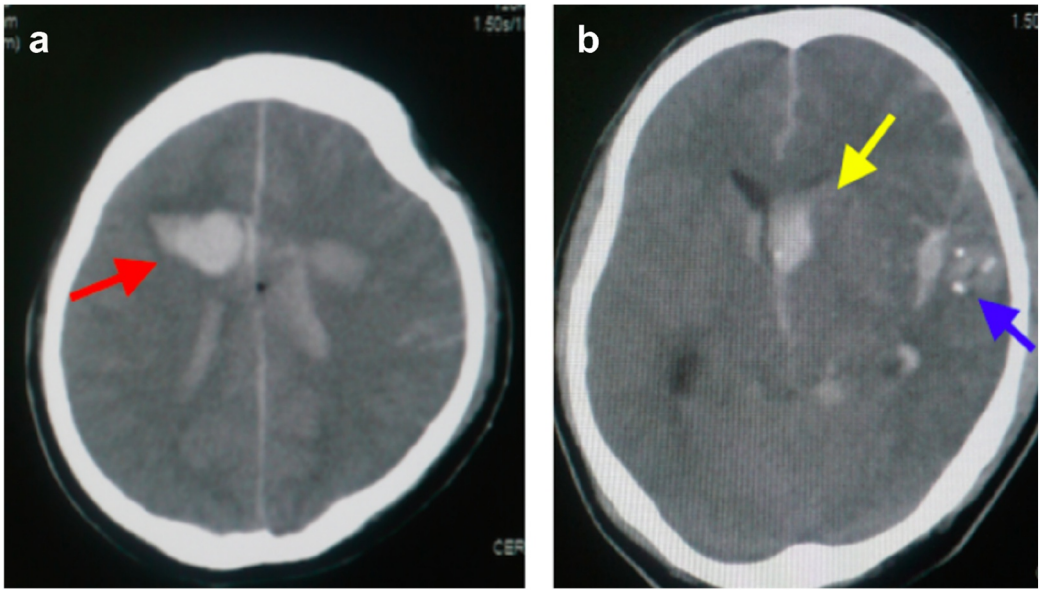


FIGURE 3 | CT scan of a patient with blast-related injuries. (a) Shows a more cranial slice demonstrating an intracranial hematoma/contusion (red arrow). (b) Depicts a more caudal slice from the same scan, revealing an intraventricular hemorrhage (yellow arrow) and intracranial shrapnel fragments (blue arrow). Subarachnoid hemorrhage is also evident in both slices. Photo Credit: Dr. Andres M. Rubiano, Cali, Colombia.

TABLE 3 | Denver Criteria for Blunt Cerebrovascular Injury.

Signs and Symptoms	Risk Factors
• Arterial hemorrhage	• Midface fractures (Le fort II or III)
• Cervical bruit	• Basilar skull fracture with carotid canal involvement
• Expanding neck hematoma	• DAI with GCS < 6
• Focal neurologic deficit	• Cervical spine fracture
• Neurological exam inconsistent with CT findings	• Hanging with anoxic brain injury
• Stroke on neuroimaging	• Seat belt mark with altered mental status
If any criterion is met, evaluate for blunt cerebrovascular injury with CT angiography.	

Abbreviation: DAI: Diffuse axonal injury.

BOX 2 | Posttraumatic seizure prophylaxis.

- Indications: penetrating TBI, severe TBI, or any TBI with depressed skull fracture, SDH, ICH, GCS < 10, or cortical contusions
- First-line: Levetiracetam 1500 mg IV load → 1000 mg IV q12h × 7 days
- Alternative: Phenytoin/fosphenytoin 15–20 mg/kg IV load → 4–7 mg/kg/day in 2–3 doses
- Duration: do not extend beyond 7 days absent seizure activity
- Acute seizure: lorazepam 4 mg IV over 2 minutes, or midazolam 5–10 mg IV/10 mg IM

6.3 | Antibiotic Use: Indications and Considerations

Prophylactic antibiotics are indicated for penetrating head injuries and open skull fractures; closed injuries do not require routine prophylaxis. Regimen, alternatives, and duration are summarized in Box 3 [42, 43].

BOX 3 | Antibiotic prophylaxis for penetrating/open cranial injuries.

- First-line: Cefazolin 2 g IV q6–8h
- Beta-lactam allergy: Clindamycin 600 mg IV q8h
- Gross contamination: add metronidazole 500 mg IV q8–12h
- Duration: 24 h standard; extend to 72 h if contamination or delayed surgical care
- Closed injuries: routine prophylaxis not indicated

6.4 | Stress Ulcer Prophylaxis

Gastric stress ulcer prevention with an IV proton pump inhibitor should be started within 12 hours of admission.

6.5 | Venous Thromboembolism Prophylaxis

All nonambulatory TBI patients require VTE prophylaxis, with timing of pharmacological agents determined by CT stability rather than injury severity alone [24, 44, 45]. Mechanical and chemical protocols are summarized in Box 4 [44, 45], including hold/restart criteria after hemorrhage progression [24, 45].

BOX 4 | Venous thromboembolism prophylaxis.

- Mechanical: sequential compression devices on admission, all nonambulatory patients
- Chemical agent: enoxaparin 30 mg SC BID, preferred over unfractionated heparin (UFH)
- Timing: start < 24 h if CT clear; > 72 h if hemorrhage present; postoperative (craniectomy/craniotomy/burr hole) 24–48 h if stable
- Hold/restart: hold if ICH progresses on repeat CT; restart after 24–48 h of confirmed stability
- Elderly patients: same approach; adjust LMWH dose for renal function, or use UFH

6.6 | Medical Management of Intracranial Hypertension

Management of intracranial hypertension is a cornerstone of TBI treatment, as without appropriate intervention, elevated intracranial pressure (ICP) can rapidly lead to cerebral ischemia and may be fatal. Due to their accuracy and reliability, invasive ICP monitors are currently preferred. Intracranial compliance assessment should not replace direct ICP measurement when available, but may serve as a surrogate indicator in low-resource environments [46]. In settings where invasive monitoring is unavailable, intracranial hypertension may be suspected based on clinical, imaging, and noninvasive neuromonitoring criteria summarized in Box 5.

BOX 5 | Suspect raised ICP without invasive monitoring.

- Suspect if 1 imaging criterion (MLS ≥ 5 mm; basal cistern effacement; nonevacuated (mass > 25 cc), OR ≥ 2 clinical/noninvasive criteria (mGCS ≤ 4; pupillary asymmetry/abnormal reactivity; ONSD ≥ 6 mm; NPi < 3; TCD PI > 1.3 + FVd < 20; NIRS positive; B4C P2/P1 > 1.0 or TTP > 0.25 s)
- If suspected → hyperosmolar therapy first-line (hypertonic saline preferred in polytrauma/hypotension (see Table 4)

Abbreviations: MLS: Midline Shift; mGCS: motor GCS; ONSD: Optic Nerve Sheath Diameter; NPi: Neurologic Pupil Index; TCD: Transcranial Doppler; FVd: End-Diastolic Velocity; B4C: Brain4Care; TTP: Time to Peak.

Hyperosmolar therapy can be administered through a peripheral IV line as a temporary measure while central venous access

TABLE 4 | Recommendations for the preparation and dosing of hyperosmolar therapy.

Agent	HTS 3%	HTS 7.5%	Mannitol 20%
Preparation	NS 400 mL + NaCl 100 mL (20 mEq/10 mL amp)	NS 100 mL + NaCl 150 mL (20 mEq/10 mL amp)	Ready to use
Dose	3–4 mL/kg	2 mL/kg	Bolus 5 mL/kg; then 1.25 mL/kg q6h

Abbreviations: HTS: Hypertonic Saline; NaCl: sodium chloride ampoules; NS: Normal Saline (0.9%).

is obtained. For central venous catheter placement, subclavian veins are preferred, followed by femoral and, lastly, internal jugular veins. When using osmotic agents, urine output and serum sodium should be monitored every 6 hours (if possible), with a target serum sodium goal of 150–160 mmol/L. Maintain physiological targets (See Box 1).

6.7 | Sedation

Sedation-analgesia in TBI controls pain, anxiety, and agitation, improves ventilator synchrony, and reduces $CMRO_2$—enhancing ischemic tolerance and lowering ICP [47, 48]. Depth should be titrated to specific targets (RASS −4 to −5, BIS 40–60, or isoelectric EEG) [48]; exceeding these doses offers no additional metabolic benefit and increases adverse effects. Routine neuromuscular blockade is not recommended; it should be reserved only for refractory ICP elevation or ventilator dyssynchrony despite adequate sedation (see Table 5).

7 | Noninvasive Intracranial Pressure Monitoring in TBI

When invasive ICP monitoring is unavailable, a multimodal noninvasive approach enables detection of intracranial pathology and guides therapeutic decisions, with several modalities having supporting evidence in resource-limited settings (Figure 4) [49]. These tools should be considered adjunctive and not equivalent to invasive ICP monitoring; however, in resource-limited environments they may provide critical guidance for triage and early management decisions [49–54]. A consolidated overview of the key noninvasive monitoring modalities and their respective diagnostic cut-off values is presented in Table 6.

8 | Basic Surgical Management for Nonneurosurgeons

Emergency cranial surgery by nonneurosurgeons is a temporizing measure that should be limited to specific scenarios, with strict adherence to preoperative stabilization principles and prior tele-consultation whenever feasible [15, 55]. Surgical competency requirements, indications and contraindications, radiological thresholds in austere settings (when transfer is impossible), and postoperative guidelines are detailed in Box 6; the specific surgical approach is illustrated in Figure 5, and the complete field protocol is provided in Online Resource 2. All decisions and procedures must be thoroughly documented, and transfer to a neurosurgical-capable facility must be prioritized at all times [15, 17, 20–22, 55, 56].

BOX 6 | Damage-control cranial surgery by nonneurosurgeons.

- Consider only when transfer is impossible and GCS $\leq$ 8 with unilateral fixed/dilated pupil
- Contraindicated: bilaterally fixed and dilated pupils
- Teleconsult neurosurgeon first whenever possible
- Surgeon competence: Documented training including $\geq$ 10 supervised cranial procedures, recent simulation-based training, access to an appropriate cranial surgical pack, and telemedicine capability
- Austere thresholds (only when definitive care unavailable): EDH > 30 mL or SDH > 10 mm
- Direct intervention to the side with the abnormality (NIRS/pupil) → then stabilize and transfer

9 | Advanced Surgical Management for Neurosurgeons

9.1 | Preoperative Evaluation and Advanced Imaging

Patients with penetrating or blast-related TBI should be considered at high risk for associated vascular injuries, including traumatic intracranial aneurysms (TICAs), traumatic arteriovenous fistulas (AVFs), direct carotid-cavernous fistulas, and intracranial vessel occlusions [17, 27, 55, 57]. When feasible, vascular imaging with computed tomography angiography (CTA) or digital subtraction angiography (DSA) should be performed at an early stage to detect these complications and guide management. Although CTA provides rapid and widely available screening, DSA remains the gold standard for vascular assessment in this context [58].

DSA offers two major advantages: first, it allows more accurate identification of vascular lesions (particularly pseudoaneurysms) in the presence of bone fragments or metallic debris; second, it serves not only as a diagnostic modality but also as a therapeutic platform, enabling immediate endovascular treatment of detected vascular complications [58].

Delayed vascular complications are common in penetrating and blast-related TBI, reinforcing the need for a low threshold for repeat vascular imaging. Repeating vascular imaging is therefore recommended in blast injuries and in delayed presentations, given the risk of evolving vascular pathology [1, 5, 7]. Preoperative planning must also anticipate blast-specific complications such as air embolism, delayed vasospasm, and complex coagulopathies. In centers where vascular studies are available, early implementation of these diagnostic tools is recommended.

9.2 | Indications and Surgical Thresholds

Multiple sources align in the definition of surgical indications for penetrating TBIs [15, 17, 27, 55, 57]. Surgical thresholds for advanced intervention should be grounded in clinical deterioration, radiographic mass effect, and evidence of refractory intracranial hypertension, while balancing potential survival against the risk of further morbidity. Advanced neuromonitoring parameters (e.g., intracranial compliance, $PbtO_2$) may support clinical decision-making when available but should not be used as sole indications for surgical intervention. In resource-limited settings, clinical examination and CT findings remain the primary determinants of surgical decision-making. The Zumkeller Index (MLS minus hematoma thickness) may serve as an adjunct measure of intracranial mass effect; however, it is not included in major guidelines and should not be used as an independent indication for surgery

TABLE 5 | Sedative, analgesic, and neuromuscular blocking agents for TBI management in mechanically ventilated patients.

Agent	Class	Mechanism	IV Dose (Infusion)	Onset	ICP/ $CMRO_2$	Key advantages in TBI	Main concerns
Sedatives							
Propofol	Alkylphenol	GABA-A potentiation	5–50 mcg/kg/min	30–60 s	↓↓/↓↓	Rapid titration; neurological reassessment windows; anticonvulsant	PRIS at > 4 mg/kg/h or > 48 h; hypotension; high lipid load
Midazolam	Benzodiazepine	GABA-A potentiation	0.02–0.1 mg/kg/h	2–5 min	↓/↓	Widely available; anticonvulsant; low cost	Accumulation; delayed neurological assessment; ICU delirium
Ketamine	Dissociative	NMDA antagonism	0.5–2 mg/kg/h	1–2 min	Neutral–↓[a]	Hemodynamic stability; analgesic; bronchodilator; useful in hypotensive TBI	Increased secretions; emergence phenomena; limited isolated-TBI evidence
Dexmedetomidine	Alpha-2 agonist	Central sympatholytic	0.2–0.7 mcg/kg/h	5–10 min	Minimal	Arousable sedation; preserves respiratory drive; opioid-sparing	Bradycardia; hypotension; limited ICP outcome data; higher cost
Analgesics							
Fentanyl	Opioid	μ-receptor agonist	25–200 mcg/h	1–2 min	Neutral[b]	Analgesia-first strategy; hemodynamic stability; short context-sensitive half-life	Tolerance; chest-wall rigidity (high bolus); cough on bolus
Morphine	Opioid	μ-receptor agonist	2–10 mg/h	5–10 min	Neutral[b]	Low cost; universal availability	Histamine release; active metabolite accumulation (renal impairment); ICP ↑ risk if hypotension
Neuromuscular blocking agents[c]							
Rocuronium	NDMR (amino steroid)	nAChR competitive blockade	0.3–0.6 mg/kg/h	1–2 min	Neutral	Fastest onset among NDMRs; sugammadex reversibility	Hepatic clearance; vagolytic at high doses; TOF monitoring is recommended when feasible
Vecuronium	NDMR (amino steroid)	nAChR competitive blockade	0.8–1.2 mcg/kg/min	3–5 min	Neutral	Low cardiovascular effects; widely available	Active metabolite accumulation in renal/hepatic failure; TOF monitoring is recommended when feasible
Cisatracurium	NDMR (benzylisoquinoline)	nAChR competitive blockade	0.5–10 mcg/kg/min	2–3 min	Neutral	Hofmann elimination; no renal/hepatic dependence; minimal histamine release; preferred in TBI + ARDS	Higher cost; requires adequate sedation before initiation; TOF monitoring is recommended when feasible

Abbreviations: BIS, bi-spectral index; $CMRO_2$, cerebral metabolic rate for oxygen; GABA, gamma-aminobutyric acid; ICP, intracranial pressure; nAChR, nicotinic acetylcholine receptor; NDMR, nondepolarizing muscle relaxant; NMDA, N-methyl-D-aspartate; PRIS, propofol infusion syndrome; RASS, Richmond Agitation-Sedation Scale; TOF, train-of-four; TBI, traumatic brain injury.

[a]Historical concern about ICP elevation with ketamine has not been confirmed under controlled ventilation; current evidence supports ICP-neutral or mildly reducing effects when combined with adequate sedation.

[b]ICP increase may occur indirectly through hypotension, cough, or CO_2 retention; bolus administration should be avoided.

[c]Neuromuscular blockade is not routine. Indication is restricted to: (1) adequate sedation with persistent ICP elevation unresponsive to other measures, or (2) refractory ventilator dis-synchrony. Continuous NMB without verified adequate depth of sedation is contraindicated. TOF monitoring (target: 1–2 twitches of 4) should guide dosing.

(Table 7). These decisions must be individualized but aligned with standardized institutional or consensus protocols.

9.3 | Staged Decompression and Debridement

In cases of uncontrolled intracranial hypertension not amenable to medical therapy, staged decompressive craniectomy followed by cautious debridement remains a mainstay of management [17, 27, 55–57]. For unilateral frontotemporoparietal decompressive craniectomy, adequate size and anatomical extent of the bone flap are critical to ensure effective decompression and to prevent secondary complications. The craniectomy should be large (typically $\geq$ 12–15 cm in anteroposterior diameter) and extend as close as safely possible to the skull base, including the



FIGURE 4 | Multimodal noninvasive neuromonitoring at the bedside in a mechanically ventilated patient with severe TBI. The image demonstrates simultaneous application of three complementary modalities: noninvasive intracranial compliance waveform analysis (Brain4Care), optic nerve sheath diameter measurement via point-of-care ultrasound (ONSD, linear transducer over closed eyelid), and automated quantitative pupillometry. Transcranial Doppler equipment is visible in the background, available for cerebral hemodynamic assessment. Photo Credit: Dr. Andres M. Rubiano, Cali, Colombia.

TABLE 6 | Noninvasive neuromonitoring technologies and diagnostic cut-off values.

Modality	Interpretation	Clinical Utility
NIRS	Significant interhemispheric optical density difference → intracranial hematoma	Detection of intracranial hematomas. NIRS is sensitive to superficial hematomas but limited for deep or small collections ($<$ 3.5 mL). Prone to false positives from extracranial hematomas, scalp injuries, or dark hair/skin.
Quantitative pupillometry	NPi $<$ 3 or anisocoria $>$ 2 mm → intracranial hypertension; ↓MCV → rising ICP	Detection and monitoring of intracranial hypertension. Reliability may be reduced in preexisting cranial nerve disorders, cataracts, or significant ambient light changes.
ONSD ultrasound	ONSD $>$ 6.0 mm → high probability elevated ICP	Noninvasive ICP assessment and monitoring. The technique is contraindicated for penetrating globe injuries and requires operator experience.
Transcranial Doppler	PI $>$ 1.3 + diastolic FV $<$ 20 cm/sec → possible ICP elevation (hemodynamically stable patients)	Monitoring cerebral hemodynamics and surrogate ICP. TCD is operator-dependent and significantly limited in unstable patients.
Brain4Care system	P2/P1 $>$ 1.0 or TTP $>$ 0.25 sec → reduced intracranial compliance	Real-time assessment of intracranial compliance, detect micrometric cranial bone pulsations. The device is sensitive to external movement during patient transfer.

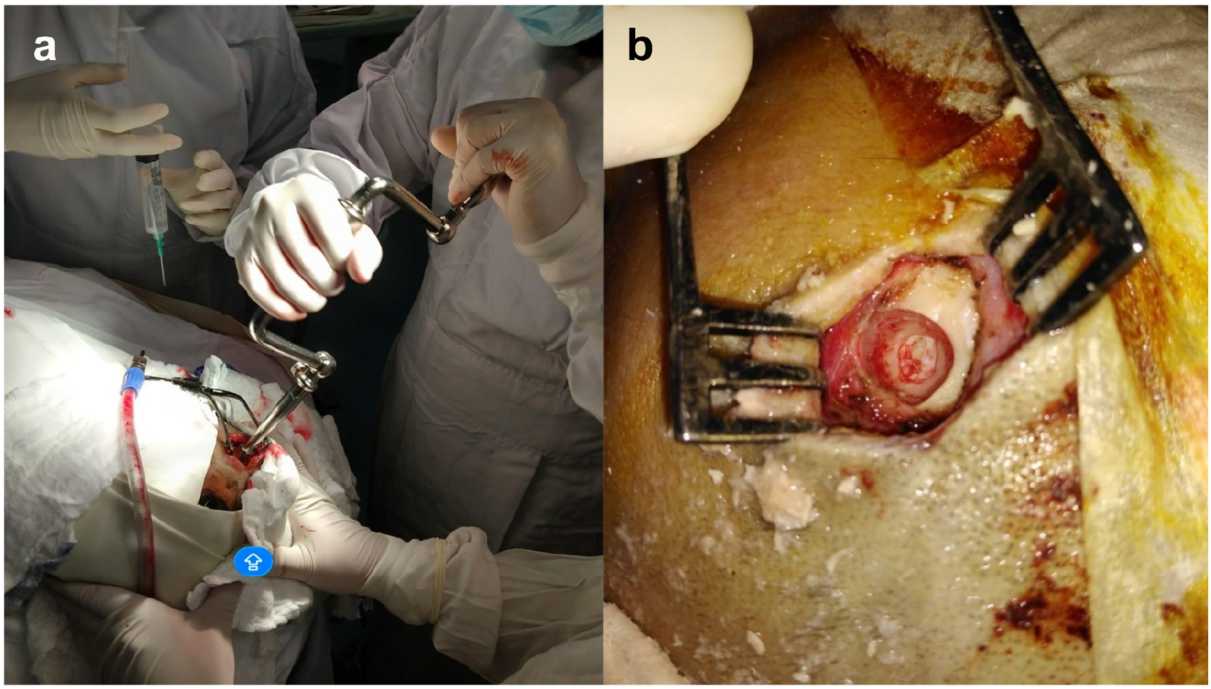


FIGURE 5 | Manual Burr Hole Technique. (a): Intraoperative creation of a burr hole using a manual hand-crank drill (Hudson brace) with syringe irrigation. (b): Direct visualization of the completed burr hole with soft tissue held by self-retaining retractors. Photo Credit: Dr. Andres M. Rubiano, Cali, Colombia.

TABLE 7 | Recommended surgical indications and procedures in advanced-level settings.

Decompressive Craniectomy	Craniotomy	EVD	Debridement
• Large ICH + zumkeller index > 3 • Significant cerebral edema on CT • Persistent intracranial compliance impairment despite medical therapy, hematoma drainage and/or EVD • Posterior fossa hematoma > 10 mL + hydrocephalus • Persistent PbtO2 < 20 mmHg not attributable to systemic causes	• Large ICH + zumkeller index < 3 • ICH any size + persistent impaired intracranial compliance despite optimal medical therapy	• Persistent intracranial compliance impairment with normal/near-normal CT despite optimal medical therapy and/or hematoma drainage	• Contaminated wound, exposed devitalized tissue, or bone fragments/foreign bodies within an accessible cortical trajectory

Definitions: Large ICH: EDH > 30 mL, ICH/contusion > 50 mL, SDH > 10 mm. Significant edema: MLS ≥ 5 mm or basal cistern effacement (grade III edema). Intracranial compliance impairment: ≥ 2 abnormal noninvasive monitoring systems or abnormal ICP waveform. Emergency surgery is IMMEDIATE when nonresponsive unilateral mydriasis is present.

floor of the middle cranial fossa, to allow optimal temporal lobe decompression.

Superiorly, the bone flap should be placed no closer than approximately 2 cm from the superior sagittal sinus to reduce the risk of venous injury and subsequent complications, including impaired venous outflow and hydrocephalus.

A pedicled pericranial flap is recommended for dural reconstruction to achieve adequate expansion and watertight closure. Care must be taken to avoid compression of cortical veins at the margins of the craniectomy, as this may exacerbate venous congestion and worsen intracranial hypertension. Undersized craniectomy significantly reduces the effectiveness of decompression and is associated with worse clinical outcomes. See Figure 6.

Deep intraparenchymal exploration should be avoided to minimize secondary injury and reduce the risk of uncontrollable hemorrhage. Debridement should focus on the removal of

devitalized tissue and foreign bodies only when safely accessible.

9.4 | Vascular and Skull Base Repairs

When vascular injuries are identified, including traumatic intracranial aneurysms (TICAs), traumatic arteriovenous fistulas (AVFs), direct carotid-cavernous fistulas, and intracranial vessel occlusions, management should be individualized and coordinated with available endovascular capabilities (Table 8). [56, 57]. Whenever feasible, early vascular imaging with CTA and/or DSA is recommended to guide treatment strategy. DSA remains the gold standard, particularly in complex penetrating injuries, as it allows precise characterization of vascular lesions and provides the opportunity for immediate endovascular intervention [57, 59, 60]. Parent vessel sacrifice may be required in selected cases, provided that adequate collateral circulation is confirmed prior to intervention [55].

In cases involving skull base disruption, paranasal sinus violation, or dural defects, meticulous reconstruction is essential. Techniques such as cranialization, sinus obliteration, and multilayer dural closure using grafts or vascularized flaps (preferably pedicled pericranial flaps) are critical to reduce the risk of CSF leakage and intracranial infection.

Surgeons should maintain a high index of suspicion for delayed vascular complications, including pseudoaneurysm formation and vasospasm, particularly in patients with subarachnoid hemorrhage or diffuse blast exposure. Serial vascular imaging or transcranial Doppler monitoring should be considered when clinically feasible [57].

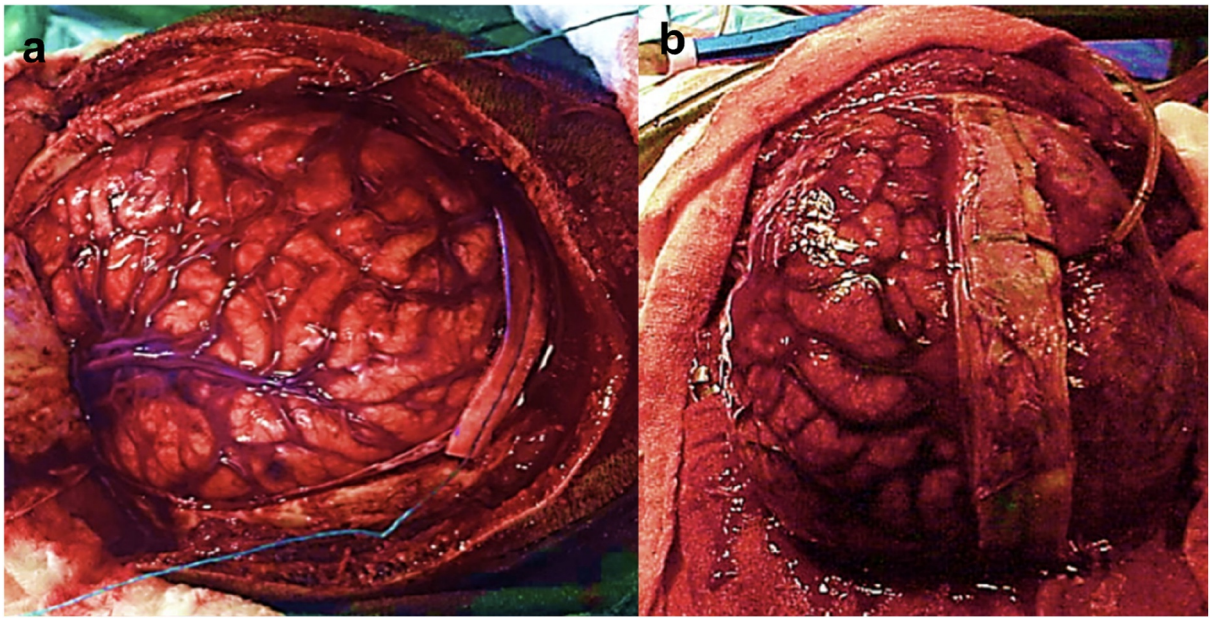


FIGURE 6 | Intraoperative views of decompressive craniectomy in severe TBI. (a) intraoperative view following completion of a wide frontotemporal-parietal craniectomy and dural opening. The extent of the bony decompression is reflected by the broad surgical exposure, allowing full visualization of the underlying hemisphere. The dura is reflected and secured with tack-up sutures, with cortical surface and vasculature visible across the entire craniectomy window. (b) Diffuse transosseous cerebral herniation ("mushrooming") through the craniectomy defect, reflecting the severity of underlying intracranial hypertension. An undersized craniectomy risks cortical strangulation at the bone edges, underscoring the importance of guideline-recommended dimensions. Photo Credit: Dr. Andres M. Rubiano, Cali, Colombia.

TABLE 8 | Endovascular management options for vascular injuries in penetrating and blast TBI.

Vascular Injury Type	Imaging Required	Endovascular Option	Key Notes
TICA/ Pseudoaneurysm	CTA ± DSA	Coiling, stent-assisted coiling, parent vessel sacrifice (if collaterals adequate)	Early detection critical; monitor for delayed rupture or regrowth
Direct carotid-cavernous fistula	DSA preferred	Coil embolization, covered stent, balloon-assisted	Look for orbital signs, bruit, chemosis
Traumatic arteriovenous fistula	CTA/DSA	Liquid embolics (onyx, glue), coils, combination	Coordinate with neuro-interventionalist; risk of high-flow shunts
Intracranial vessel occlusion	CTA/DSA	Mechanical thrombectomy, intra-arterial vasodilators (for vasospasm), stenting	May present with delayed ischemia; often associated with vasospasm

Abbreviations: AV: arteriovenous; CTA: computed tomography angiography; DSA: digital subtraction angiography; TICA: traumatic intracranial aneurysm.

In resource-limited settings, where advanced vascular imaging or endovascular therapy may not be immediately available, early recognition of high-risk injury patterns and timely transfer to specialized centers are essential to optimize outcomes. Failure to recognize and treat vascular injuries in penetrating and blast-related TBI is associated with a high risk of secondary neurological deterioration and mortality.

9.5 | Postoperative Intensive Care Monitoring and Management

Postoperative management requires vigilant neurocritical care (see Box 1). Surveillance for vasospasm is essential, particularly following vascular injury or subarachnoid hemorrhage [57, 61]. When feasible, monitoring strategies such as transcranial Doppler or repeat vascular imaging should be considered [17, 55]. Infection prevention protocols, seizure prophylaxis, venous tromboembolism prophylaxis, and measures to prevent CSF leakage must be implemented rigorously [27, 56].

Documentation of operative details, intraoperative decision-making, and postoperative status should be comprehensive. Timely communication with referral neurosurgical centers is critical when staged transfer is anticipated. In resource-limited settings, where advanced monitoring may not be available, strict control of basic physiological parameters and serial neurological assessment remain the primary determinants of outcome. Early involvement of rehabilitation services and structured discharge planning should begin within the ICU whenever feasible [15]. Failure to maintain physiological targets in the postoperative period is a major contributor to secondary brain injury and poor outcomes.

Rehabilitation planning should begin early, even in resource-limited settings. All survivors of blast-related TBI should receive structured discharge instructions and follow-up planning, including screening for cognitive, vestibular, visual, and psychological sequelae.

The Brain Trauma Foundation (BTF) has published comprehensive recommendations for the management of penetrating TBI [32]. A summary of BTF Penetrating TBI Guidelines stratified by resource level (basic, intermediate, and advanced) is provided in Online Resource 3.

10 | Considerations During Transport

10.1 | General Principles for Transport in Severe TBI

The interfacility transport of patients with severe penetrating or blast-related TBI must be executed with the primary aim of preventing secondary brain injury through rigorous adherence to standardized protocols that prioritize physiological stability. See Box 1. The timing of transfer should balance urgency and adequate pretransfer stabilization, prolonged on-scene times exceeding 30 minutes are associated with increased mortality risk [17].

10.2 | Monitoring and Stabilization Before Transfer

Before transport, patients must undergo reassessment for neurological deterioration [17]. If elevated ICP is suspected or documented, hyperosmolar therapy (preferably hypertonic saline) should be administered in accordance with established protocols [55]. Mannitol should be avoided in hypotensive patients. Hemodynamic support with vasopressors may be necessary in patients with polytrauma or those who remain unstable despite resuscitation [56]. All interventions, vital signs, neurologic exams, and clinical decisions must be meticulously documented prior to departure with clear timestamps [15]. See Online Resource 4 for pre transfer check list and standardized Inter-facility TBI Transfer Form.

10.3 | Injury-Pattern Specific Considerations

For blast-related TBI, clinicians must account for the elevated risk of air embolism and delayed cerebral vasospasm;

TABLE 9 | Specific transport considerations according to injury pattern and key transport risk.

Injury Pattern	Key Risk	Transport Considerations
Severe TBI without raised ICP [2]	Hypoxia/hypotension	SBP $\geq$ 100–110 mmHg, $SaO^2 > 93\%$, normoglycemia, normothermia, and secure airway.
Severe TBI with raised ICP [5]	Neurological deterioration	Hyperosmolar therapy if needed. Reassess pupils/GCS pretransfer
Blast-related TBI [5, 9]	Air embolism, vasospasm, polytrauma	Monitor vascular compromise. Limit cabin altitude (air evacuation). Rapid coordination
Skull base fracture + CSF leak [6, 7]	Infection	Occlusive wound dressing. Prophylactic antibiotics
Confirmed vascular injury (TICA, AVF) [2, 8]	Rupture in transit	CTA/DSA pretransfer if possible. BP management. Blood products available
Multiple injury patterns [1]	Long on-scene time increases risk	Keep on-scene time < 30 min. Use AET if available. Preplan staging between facilities.
All moderate–severe TBI [1, 2, 5]	Incomplete handover	Standardized checklist. Document vitals, interventions, timeline

serial neurologic assessment and vascular monitoring are recommended in such cases [27, 57, 61]. In cases involving skull base fractures with CSF leak, prophylactic antibiotics should be administered before transfer, and all open wounds must be sealed with sterile occlusive dressings [27, 55]. Although routine antibiotic prophylaxis for CSF leaks is not recommended in high-resource settings, in austere environments with delayed surgical access and higher contamination risk, selective use may be considered. For patients with suspected vascular injuries (pseudoaneurysms or AVFs), advanced imaging (CTA or DSA) should be completed pretransfer if logistically feasible [27, 57, 61]. Table 9 summarizes injury-specific transport considerations.

10.4 | Equipment and Evacuation Protocols

The evacuation team's level should correspond to the severity of injury. Whenever possible, moderate to severe TBI patients should be transferred by advanced evacuation teams (AETs) trained in neurocritical care [17]. Teams must verify the availability of essential equipment including airway tools, portable vital sign monitors, hyperosmolar agents, and backup supplies [15]. A standardized transfer checklist should be used to ensure continuity of care and prevent the omission of critical clinical data, imaging studies, or administered medications; coordination with the receiving facility should include real-time communication to prepare the team for arrival [46, 55].

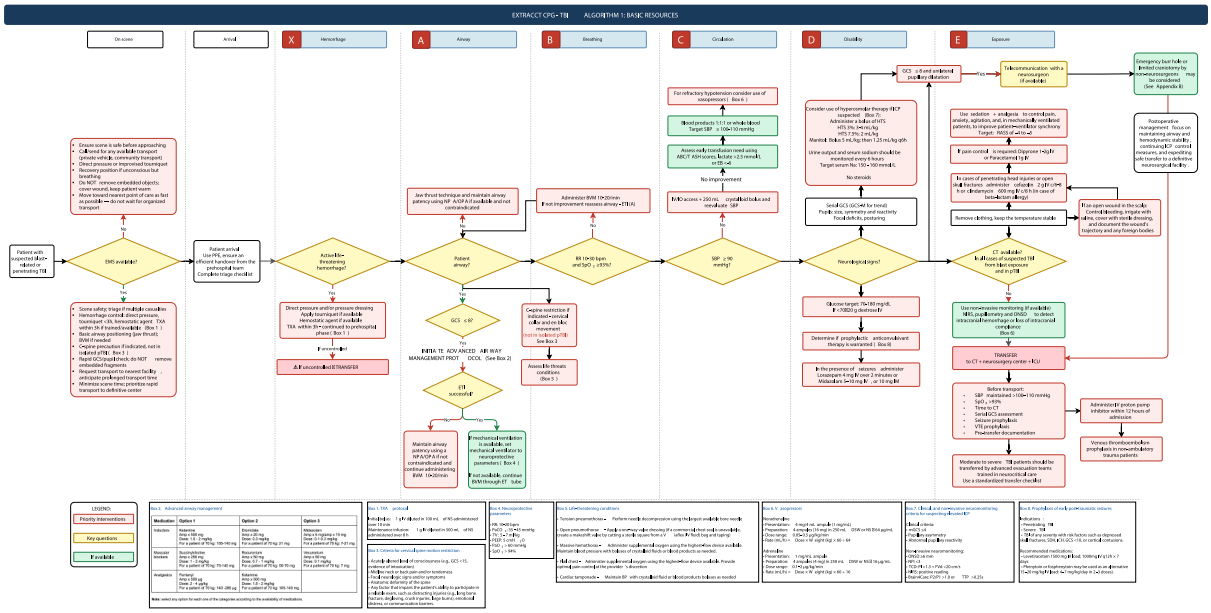


FIGURE 7 | EXTRACCT CPG—TBI Algorithm 1: Basic Resources. Sequential management pathway (Hemorrhage, Airway, Breathing, Circulation, Disability, Exposure) for settings with basic resources only, from point of injury through transfer.

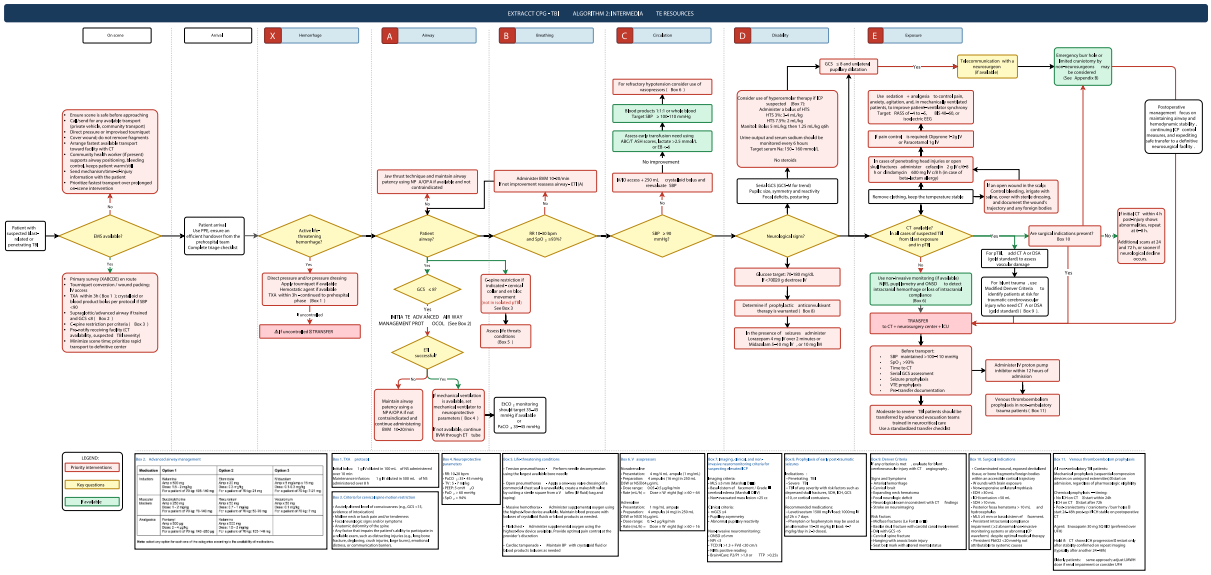


FIGURE 8 | EXTRACCT CPG—TBI Algorithm 2: Intermediate Resources. Management pathway for settings with CT availability but without neurosurgical capability.

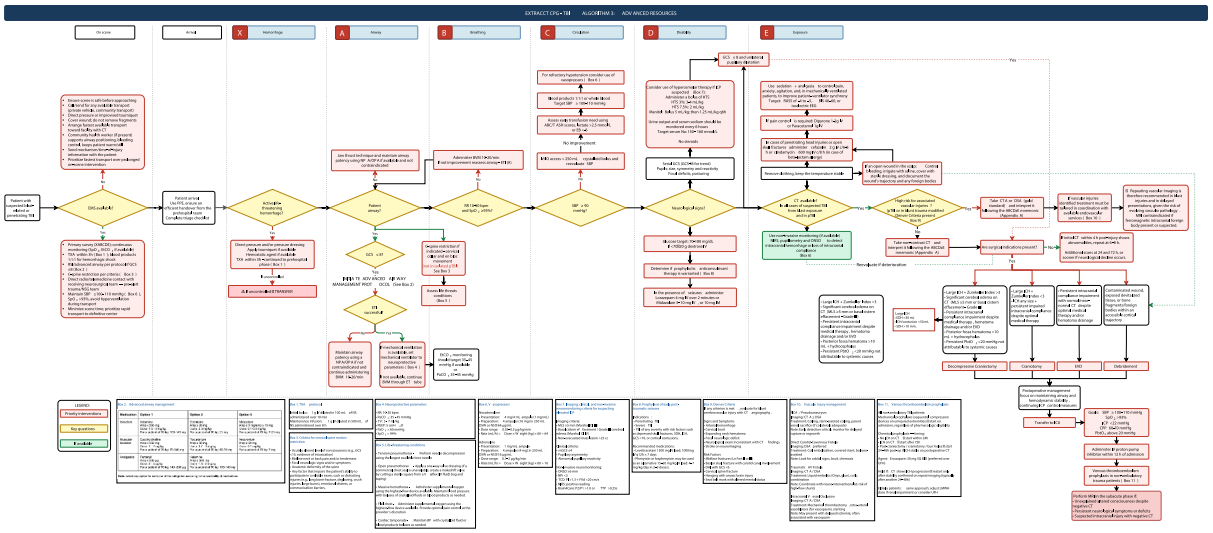


FIGURE 9 | EXTRACCT CPG—TBI Algorithm 3: Advanced Resources. Management pathway for settings with CT, neurosurgery, and ICU capability.

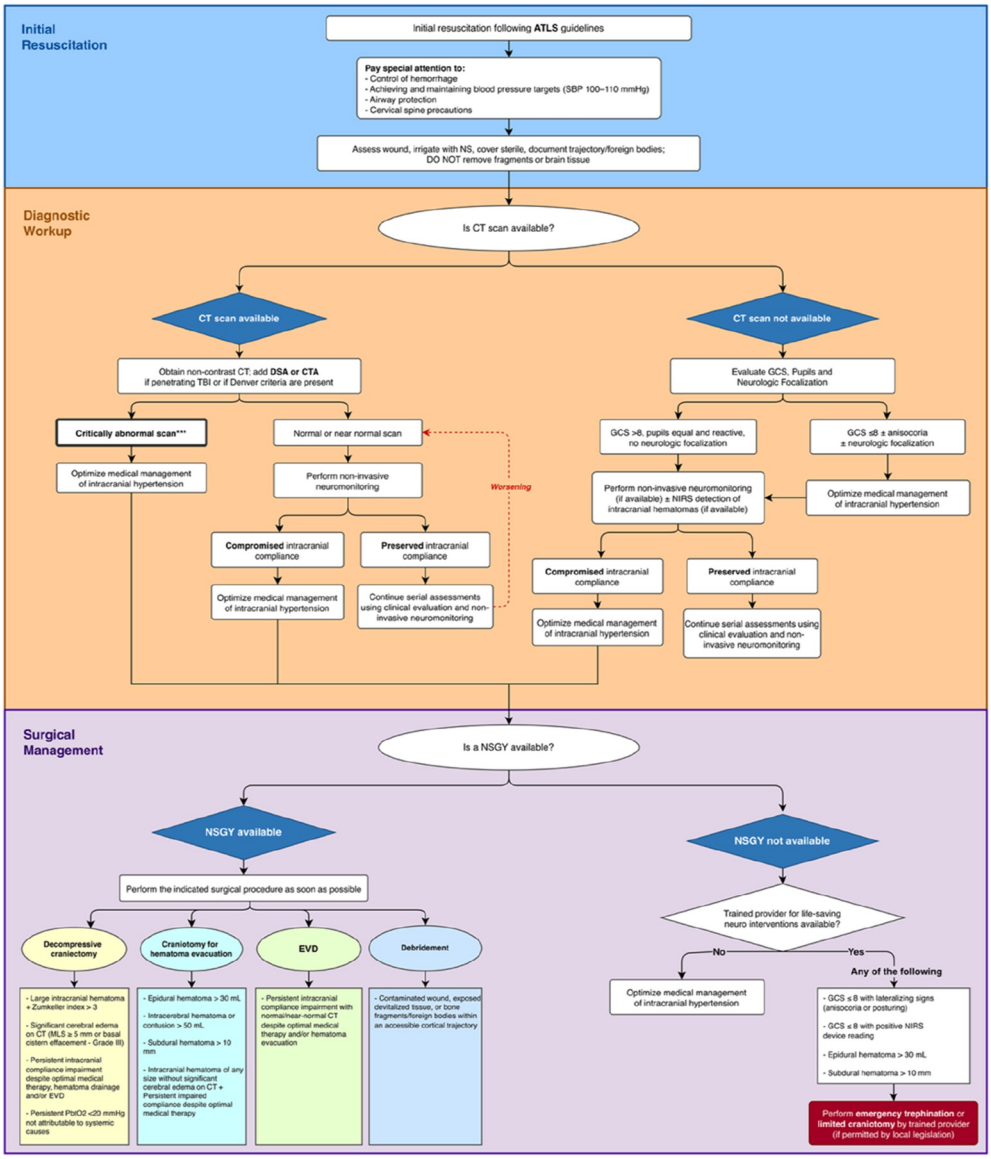


FIGURE 10 | General management algorithm for traumatic brain injury secondary to blast injuries.

10.5 | Data Sharing and Communication

Effective communication and documentation are critical components of safe TBI transport. Transport notes should detail the patient's baseline neurologic status, all pretransfer stabilization measures, and any events or deterioration during transit. The receiving facility must be provided with updated imaging, a log of all procedures performed, and a narrative summary of the injury pattern [15, 62]. When multiple care facilities are involved, staged transfer pathways should be precoordinated and clearly delineated [27]. The implementation of regionally standardized transport protocols and integrated digital handoff tools can significantly enhance care quality and reduce transition-related errors.

11 | Special Populations

Special consideration is required for pediatric and pregnant patients, including adaptations in imaging thresholds, drug dosing, and physiological targets. Comprehensive management of blast injury in pediatric patients and in pregnant patients are addressed in dedicated EXTRACCT Clinical Practice Guidelines; clinicians managing these populations should refer to those guidelines for resuscitation targets, imaging considerations, and medication dosing specific to each group. Within the scope of this TBI guideline, the following considerations are particularly relevant: in pediatric patients, the postresuscitation GCS may be less reliable for predicting outcomes and should be interpreted alongside age-appropriate neurological assessments; normal physiological targets for blood pressure, heart rate, and oxygenation differ from adults and must be adjusted accordingly. In pregnant patients beyond the first trimester, left lateral decubitus positioning should be employed to avoid aortocaval compression during resuscitation; fetal monitoring should be initiated as soon as resources allow.

12 | Proposed Management Algorithms

Based on the principles outlined above, this section proposes a set of stratified management algorithms adapted to different resource contexts, including a specific protocol for blast-related TBI (Figures 7–10). Figure 11 synthesizes these pathways into a single Chain of Survival that follows the patient from the point of injury (with or without EMS) through trauma stabilization, damage control, and definitive neurosurgical care.

13 | Conclusion

This CPG has provided a structured, evidence-informed approach to the assessment, diagnosis, and management of traumatic brain injury from blast and penetrating mechanisms in settings with variable resource availability. The recommendations are intended to support clinicians across a range of resource contexts from austere field environments to facilities with neurosurgical capability and are applicable to local

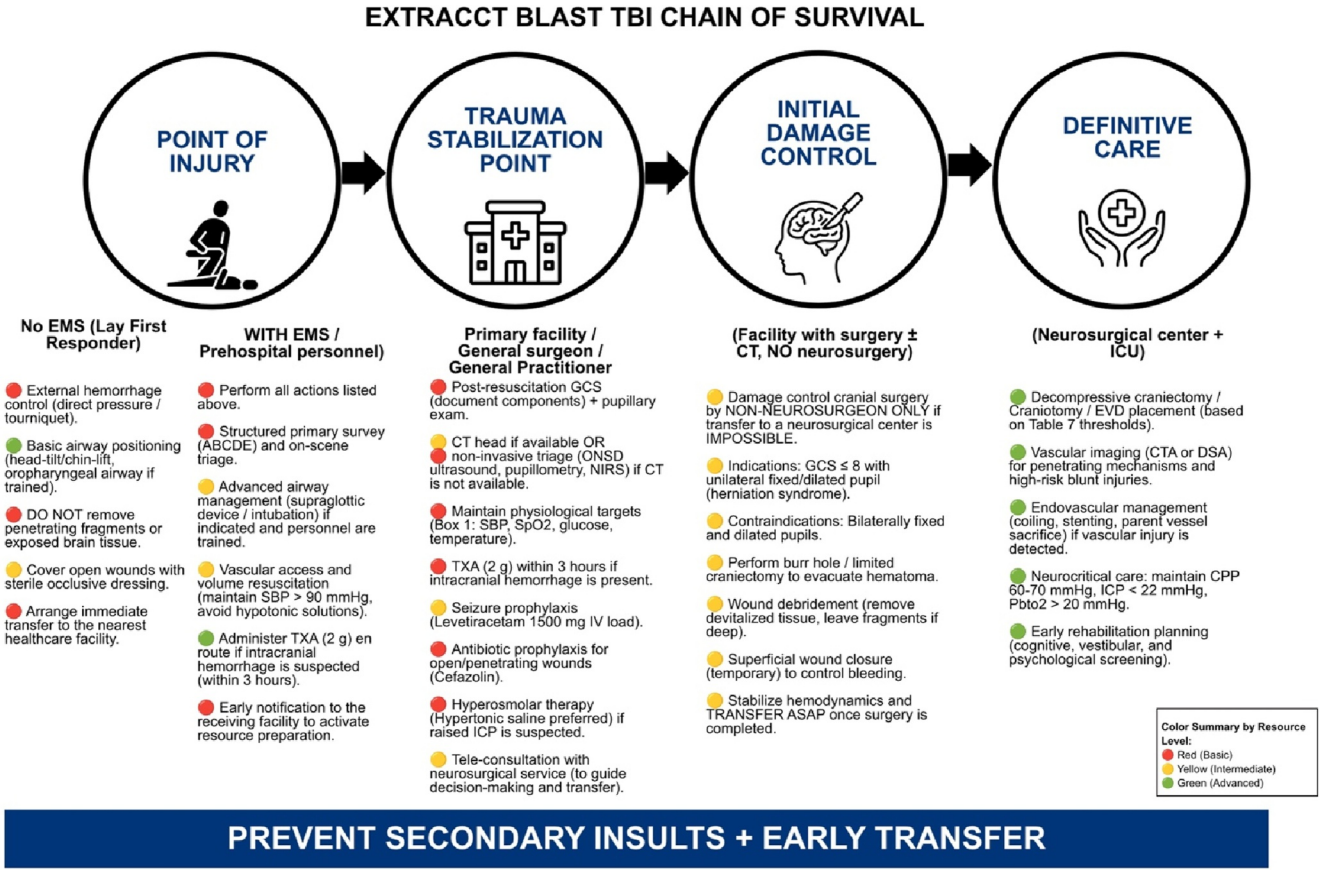


FIGURE 11 | EXTRACCT Blast TBI Chain of Survival. Resource-stratified care pathway from point of injury to definitive care. Color-coded by resource level: red (Basic), yellow (Intermediate), and green (Advanced). Early transfer and prevention of secondary insults are priorities across all levels.

casualties in both conflict and nonconflict situations involving explosive weapons.

Author Contributions

Andres M. Rubiano: conceptualization, methodology, supervision, writing – original draft, writing – review and editing, project administration. **Laura M. Loaiza-Cardona:** investigation, data curation, writing – original draft, writing – review and editing. **Ali Msheik:** formal analysis, writing – review and editing. **Andrés Salazar Restrepo:** investigation, writing – review and editing. **Santiago Cardona-Collazos:** investigation, writing – review and editing. **Andrii Sirko:** visualization, writing – review and editing. **Ruben Peralta:** writing – review and editing.

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Ethics Statement

The authors have nothing to report.

Consent

Informed consent was obtained from all individual patients whose clinical photographs are included in this manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.

Supporting Information S1: wjs70522-sup-0001-suppl-data.docx.
Supporting Information S2: wjs70522-sup-0002-suppl-data.docx.
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