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High risk and low prevalence diseases: Blast injuries

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ABSTRACT

Introduction: Blast injury is a unique condition that carries a high rate of morbidity and mortality, often with mixed penetrating and blunt injuries.
Objective: This review highlights the pearls and pitfalls of blast injuries, including presentation, diagnosis, and management in the emergency department (ED) based on current evidence.
Discussion: Explosions may impact multiple organ systems through several mechanisms. Patients with suspected blast injury and multisystem trauma require a systematic evaluation and resuscitation, as well as investigation for injuries specific to blast injuries. Blast injuries most commonly affect air-filled organs but can also result in severe cardiac and brain injury. Understanding blast injury patterns and presentations is essential to avoid misdiagnosis and balance treatment of competing interests of patients with polytrauma. Management of blast victims can also be further complicated by burns, crush injury, resource limitation, and wound infection. Given the significant morbidity and mortality associated with blast injury, identification of various injury patterns and appropriate management are essential.
Conclusions: An understanding of blast injuries can assist emergency clinicians in diagnosing and managing this potentially deadly disease.

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1. Introduction

This article series addresses high risk and low prevalence diseases that are encountered in the emergency department (ED). Much of the primary literature evaluating these conditions is not emergency medicine focused. By their very nature, many of these disease states and clinical presentations have little useful evidence available to guide the emergency physician in diagnosis and management. The format of each article defines the disease or clinical presentation to be reviewed, provides an overview of the extent of what we currently understand, and finally discusses pearls and pitfalls using a question and answer format. This article will discuss blast injuries. This condition's low prevalence in the civilian setting but high morbidity and mortality, as well as its variable atypical patient presentations and challenging diagnosis, makes it a high risk and low prevalence disease.

1.1. Definition

A blast injury is the result of exposure to an instantaneous transformation of a liquid or solid matter to gaseous matter, producing energy in the form of heat, light, pressure, and sound [1,2]. These are most commonly the result of an explosive device, which are divided into high-order and low-order [3-5]. High-order explosives (e.g., C-4, Semtex, dynamite, ammonium nitrate, trinitrotoluene, gelignite, etc.) create supersonic blasts, which can travel at 8000 m per second and result in pressures up to 30,000 times atmospheric pressure [6-9]. Low-order explosives (e.g., gunpowder, Molotov cocktails, pipe bombs, etc.) create subsonic blasts and less sheer velocity and are often used as propellants or pyrotechnics [3]. While associated with less forceful primary blast compared to high-order explosives, damage from low-order explosives can be more severe with secondary missiles (e.g., nails, metal fragments, etc.) or infectious agents creating multiple vectors for injury [4].

Blast injuries are categorized as primary, secondary, tertiary, quaternary, and quinary (Table 1) [4]. The blast wave itself causes primary blast injuries, which most commonly affect gas-containing organs such as the eardrum, lungs, and bowel [10]. Specific injuries include hemothorax, pneumothorax, arterial gas embolism, intestinal perforation, globe rupture, and tympanic membrane perforation [6]. Primary blast injuries typically result from three different physical mechanisms: spallation, implosion, and shearing injuries. Spallation occurs when the pressure blast wave passes from a dense medium to a less dense medium (e.g., bowel wall into the gas-filled bowel). Implosion results

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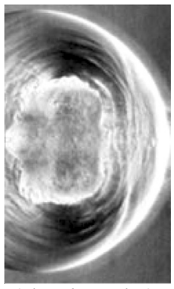
Table 1
Mechanisms of blast injury. CDC Web site. Available at: https://www.cdc.gov/masstrauma/preparedness/primer.pdf

	Primary	Secondary	Tertiary	Quaternary	Quinary
Mechanism					
Definition	High-order explosives. Impact of over pressurization wave on body surfaces.	High- and low-order explosives. Due to flying debris, bomb fragments, other projectiles.	High-order explosions. Due to individuals being thrown by blast winds or structural collapse. "Flying people injury"	Any explosion-related injury, illness, or disease not due to primary, secondary, or tertiary mechanisms. Includes exacerbations and complications of pre-existing illnesses.	Hyperinflammatory behavior, unrelated to their injury complex and severity of trauma
High Yield-Injuries	-Blast Lung -TM Rupture -Globe Rupture -Abdominal Hemorrhage	-Soft Tissue Injury -Globe Penetration -Wound Contamination	-Bony Fracture -Traumatic Amputation -Closed and Open Brain Injuries	-Burns -Crush Injury -Inhalation Injury	-Prolonged shock and hypotension

from compression of tissue that is otherwise not typically compressible (e.g., solid organs). Shearing results from acceleration/deceleration resulting in displacement of the tissue causing tearing injuries. A blast wind follows dissipation of the energy from the blast wave, which consists of fast-moving superheated air that can result in further injury [6,11,12]. Blast lung is the most common cause of death associated with primary blast injury (PBI) [6,12]. Blast lung is marked by alveolar capillary rupture, resulting in hemorrhage and pulmonary edema, reduced gas exchange, and hypoxia and hypercarbia [6,8,13-15].

Secondary injury is the result of direct impact of debris caused by blast wind and can comprise shrapnel from the bomb device or secondary fragments from the environment [6,16]. Secondary injuries can be thought of as the "flying objects" injuries. These penetrating injuries can be limited with the use of body armor, though body armor has little effect on the damage from the primary blast wave to the lungs. Tertiary injury is the result of the transposition of the whole body from the blast wind (flying people injury) or a structural collapse [6,16,17]. Quaternary injuries are other blast effects including burns, inhalational injury, radiation, crush injuries, psychological effects, bacterial contamination, and those immediate injuries not classified by primary to tertiary injuries [6,16,17]. Finally, quinary blast injury, a relatively newly described classification, is a delayed hyper-inflammatory response which may be due to chemicals associated with the blast and is manifested as hyperpyrexia, sweating, low central venous pressure, and positive fluid balance that does not correlate with the complexity of injuries sustained and lasts anywhere from a few hours to 5 days [18-20]. Some definitions also include radiation and bacterial exposure in this class rather than quaternary [21].

1.2. Epidemiology

The global prevalence of blast injuries has increased over the last decade. Terrorist attacks globally over the last decade have tripled from approximately 5000 in 2007 to over 16,000 in 2017 [22]. Many of these involve the shift to asymmetric warfare through the use of improvised explosive devices (IEDs) or suicide attacks by terrorist groups and have led to an increase in military casualties due to blast injury [12,22,23]. U.S. Department of Defense statistics from 2007 to 2016 described blast exposure as the main cause of combat casualties, comprising 55% of combat injuries of U.S. forces [9,24,25]. While the majority of these have been relegated to war zones, recent events such as the Boston Marathon bombing in 2013 and the United Kingdom train bombing in 2017 have illustrated an increase in terrorist attacks outside of war zones and areas of high terrorist activity [22,26]. Kapur et al., reported 5931 injuries and 699 deaths due to blasts in the U.S. from 1983 to 2002, making it a rare but significant mechanism of injury [27].

2. Discussion

2.1. ED presentation, evaluation, and management

Explosions by their very nature generate a multitude of vectors causing harm. This makes the presentation differ greatly depending on the type of explosive, the direction of the blast wave, presence of shrapnel, and sources of quaternary blast injury [4,6]. The direction of the blast may also result in damage to one organ system while sparing others. These difficulties make it beneficial to take note of the common ways that blasts can injure organ systems knowing that all or none of them can affect a patient with blast exposure.

Evaluation of blast injury patients should be systematic, given the extent to which large blasts result in polytrauma with critical injuries. This evaluation must include the primary and secondary survey, with emphasis on hemorrhage control due to the tendency for this mechanism to cause extremity and junctional hemorrhage. The incidence of polytrauma in this patient population makes serial evaluation necessary to reduce the risk of missing life-threatening injury. A review of over 3000 victims of terrorist bombings found an immediate fatality rate of 13% and a hospitalization rate for survivors of 30%, indicating the severity of the potential injuries sustained from blasts [28].

2.1.1. Auditory system

The auditory system is frequently affected by the primary blast wave. The tympanic membrane (TM) can be easily stressed from the incoming pressure differential and can perforate at pressures <0.5 atm, much less than needed to damage other organs [4,29-31]. Rupture of the TM occurs at a high incidence in blast victims, as seen in the 2004 Madrid train bombings where 41% of the 243 victims were found to have TM rupture, while a study of military blast victims found TM

perforation occurred in 16% of explosion-injured patients [29,32]. TM perforation may lead to dizziness, hearing loss, otalgia, tinnitus, and hemotympanum. TM rupture was traditionally used as a marker of additional PBI and middle ear injury [4]. However, a study evaluating 167 patients with blast injuries demonstrated that only half of those with additional PBI had TM rupture, suggesting that TM rupture is not a sensitive or specific marker for PBI affecting other systems [33]. This study indicates that the presence of TM rupture cannot reliably predict other injuries, and the absence of TM rupture cannot exclude other injuries. A review of blast victims from conflict in Iraq did show a strong association between TM perforation and loss of consciousness inferring possible concussive brain injury [34].

All patients exposed to a blast should be asked specifically about hearing loss and tinnitus during their initial trauma evaluation. Routine otoscopy is recommended on all blast injury patients, given the low threshold for TM rupture from a blast wave [35]. While TM rupture was previously thought to be a marker of severity of blast injury, more recent evidence suggests this is not the case [18]. Ossicular disruption, cochlear damage, and foreign bodies may occur secondary to blast injury, and these injuries may not appear with obvious damage to the TM on otoscopy [35]. If hearing loss, otalgia, or dizziness are noted without TM rupture, high suspicion for these other causes should be investigated [35].

While small, simple, and isolated non-blast TM ruptures without signs of additional middle ear injury (vertigo, dizziness) may be managed with observation and primary care follow up, blast TM ruptures are likely to benefit from urgent otolaryngology evaluation given the higher rate of large perforation, reduced spontaneous healing, and need for operative intervention [36,37]. These patients should be ideally evaluated by otolaryngology within 48 h to further stratify the extent of middle ear injury and hearing loss.

Management of TM rupture is mainly conservative and includes mitigation of additional barotrauma. Antibiotic otic drops are only indicated if the ear is contaminated with debris or signs of secondary infection are present. If debris are found in the external auditory canal or middle ear due to a TM perforation, ear irrigation should be avoided, as this will result in severe pain and vertigo, move debris medially in the canal and middle ear, and promote infection [35]. These patients should be treated with a fluoroquinolone and steroid-containing topical antibiotic. Topical drops containing aminoglycosides should be avoided given their ototoxicity [81]. Those with debris or TM rupture should be advised to keep the external auditory canal dry and away from water until the TM has healed or been repaired [35]. Removal of debris should only be completed by an otolaryngology specialist in order to reduce the risk of further injury to the TM and middle ear [35].

Preventing further auditory injury with noise reduction is important to long term healing. A study of 433 soldiers from Israel indicated those with auditory injury (tinnitus, hearing loss) who were moved to quieter non-combat units compared to those who remained in noisy units had over 3 times the rate of hearing improvement [38]. Patients with persistently diminished or lost hearing for >72 h should undergo audiogram or hearing test and if possible be kept from high noise environments to allow healing [35].

A review of 210 military blast related TM perforations found a lower rate of spontaneous closure (74%) compared to non-blast traumatic TM perforations (89%) [36,39,40]. A study of 2004 Madrid civilian bombing survivors with TM perforation found a rate of 38% spontaneous closure rate at 6 months [30]. The reason for this lower rate of spontaneous closure is unclear, though it has been suggested that the difference in spontaneous closure between traumatic and blast caused TM rupture is due to shrapnel or increased keratin debris, which may require tympanoplasty for definitive repair [41].

Additional PBI should be considered in those with TM rupture. However, no specific guidelines exist, and some literature suggests that during a resource limited event such as mass casualty incident (MCI), a patient can be safely discharged with TM rupture if they remain hemodynamically stable, have an unremarkable chest radiograph, have otolaryngology follow up, and have no additional symptoms or signs of blast injury after an observation period of 4–6 h. The majority of those with TM rupture have good prognosis and experience healing without any intervention, though up to 30% develop some degree of permanent hearing loss [3,11,33,42]. In blast victims with or without TM rupture, dizziness, vertigo, and sensorineural hearing loss may indicate a perilymphatic fistula and are indications for immediate referral to otolaryngology.

2.1.2. Brain

The impact of kinetic energy from the primary blast wave on the brain may induce a wide spectrum of injury traditionally classified in the literature as a traumatic brain injury (TBI) or head injury [21]. There is often a blunt force in addition to the blast wave which may complicate the injury [21]. Regardless of the underlying pathophysiology, TBI is a leading cause of mortality in blast victims, with one analysis of 3357 terrorist bombing victims indicating head injury accounted for 52–71% of fatalities [28,43].

The blast can further afflict the brain with secondary injury in the form of debris leading to penetrating injury. While the specific mortality rate of penetrating head injury during blasts is unknown, it is likely significant, with one study finding 62% of patients with a penetrating head injury from gunshots were dead within the first 48 h [44]. While TBIs due to blast injury occur by a unique mechanism, their clinical consequences appear to be similar to penetrating injuries sustained from non-blast projectiles [45]. In addition to penetrating brain injury, secondary and tertiary blast injuries can cause closed head injuries (or blunt head trauma) as well.

TBIs are typically classified into primary and secondary phases of injury. The primary phase is the direct damage sustained from the transfer of force on the intracranial contents [45-47]. In the case of blast injuries it is unclear if this direct damage is due to the direct shearing, stretching, and/or angular forces of the blast wave on brain tissue or from the blast wave causing gas emboli leading to infarction [48,49]. This can include the combination of cerebral contusion, cerebral edema, diffuse axonal injury, and extra-axial hematomas (e.g., epidural, subdural, and subarachnoid hemorrhages) [50]. The secondary phase of injury includes a cascade of molecular mechanisms initiated at the time of the first contact and sustained for hours or days [45,50]. This phase is multifactorial and involves inflammatory responses, vasospasm, secondary ischemia, and delayed physiologic events that occur in response to the initial injury [45,50]. The secondary phase is also modifiable in the prehospital and ED setting by limiting exposure to hypoxia, hypotension, and hypercarbia. The eventual tissue loss and cell death after TBI depends on the interplay of these two phases of injury.

Evidence of a TBI in a blast victim can be assessed via the Glasgow Coma Scale (GCS) which can help delineate between a mild TBI (GCS of 13–15) and moderate/severe TBI (GCS of <13 or post-trauma amnesia >24 h) [50]. GCS is a good predictive marker for short term mortality in TBI patients [51]. Additional signs and symptoms of TBI include headache, seizure, dizziness, amnesia, weakness, numbness, and difficulty concentrating [11,50]. Continued tracking of a blast victim's GCS and neurological progression is also necessary to determine the extent of the secondary phase of brain injury.

Contact with the primary blast wave can cause a spectrum of brain injury from concussion to intracranial hemorrhage and severe TBI (GCS <9). In the last 30 years, mortality from severe TBI for those patients who survive to the hospital has been reduced from 50% to 25% [52,53]. This reduction of both mortality and long term neurologic deficit may be due to better out-of-hospital and emergency care, given that many of the factors associated with outcome are determined in the first few hours after the TBI onset [54].

While mild TBI patients may lack acute hemorrhage and many of the large structural changes seen in moderate/severe TBI, animal models have shown that blasts cause histological and chemical changes to the

brain following exposure [21,55]. Mild TBI from blasts have been linked to post-traumatic stress disorder (PTSD) although the exact mechanism is poorly understood [21,55-57]. Persistent depression, anxiety, insomnia, lethargy, fatigue, and poor concentration well after exposure to a blast may indicate undiagnosed TBI, post-concussive syndrome, or PTSD.

After optimization of hemodynamics and ensuring cerebral perfusion, assessment of mental status and neurologic examination (i.e., cranial nerves, motor, sensory, cerebellar testing) is necessary to identify the degree of TBI. The clinician should assess for headache, loss of consciousness, amnesia, nausea, fatigue, and poor concentration. If a deficit or concerning symptoms are noted, additional imaging including computed tomography (CT) of the head is necessary to assess for hemorrhage and signs of herniation. Of note, while CT imaging may identify signs of TBI (e.g., cerebral hemorrhage, edema, ischemia, herniation, hematoma, vascular injury, skull fracture), up to half of those with TBI after blast injury have no significant findings on initial CT [58].

There is no significant differentiation between the treatment of blast TBI patients and non-blast TBI patients [45]. Observation is recommended for patients with mild TBI and hospitalization for those with GCS <15, abnormal CT findings, or seizures [45,59,60]. Resuscitation should focus on ensuring systemic perfusion and reduction of intracranial pressure. Hypoxia, hypercarbia, hypotension, hypothermia, and hypoglycemia should be avoided. In moderate and severe TBIs without surgical indications the primary goal is to limit the extent of secondary injury from posttraumatic hypotension and hypoxia. Targeting a systolic blood pressure > 90 mmHg and PaO2 > 60 mmHg, as well as keeping the patient euvolemic to prevent worsening of intracranial pressure from cerebral edema is necessary [45]. Hyperoxia with a PaO2 > 300 mmHg should be avoided in severe TBI given the association with higher in-hospital mortality [61]. These goals can conflict with the treatment of other injuries commonly seen in blast injuries, most notably blast lung injury (BLI) and burns. There is further controversy and little consensus on a general approach on management of penetrating brain trauma associated with blast injury, and prompt neurosurgical consultation is advised.

2.1.3. Lung

The lungs can be heavily damaged by the primary blast wave given the large contrast in density throughout the organ. One review found that 10% of military casualties and 60–90% civilian victims of terror incidents suffer pulmonary blast injuries when explosives were used [62]. The large difference was suspected to be due to the higher prevalence for civilian blast victims to be in enclosed settings [63]. Primary BLI occurs more frequently when high explosives are used in confined spaces increasing the duration of the blast wave or where the victim is in close proximity to the explosion [64]. The mortality of BLI is difficult to assess given it rarely occurs in isolation without additional lethal PBI. The incidence of BLI in those who die immediately at the blast scene can be relatively high, ranging from 13% to 47% [28,64]. While older studies evaluating BLI found mortality rates of 11% in those who survive to the hospital, more recent cohorts have found a mortality rate of 5% [28,62,65,66].

Lung damage from the primary blast wave includes rupture of the alveolar capillaries, intrapulmonary hemorrhage, and pulmonary contusions. The immediate consequence of this dissipation of energy may present as pneumothorax, hemothorax, respiratory distress, and hypoxia. There is also the potential of immediate air embolism formation from the rupture of alveolar capillaries, which can cause stroke syndromes or myocardial infarct due to coronary artery air embolism, which can rapidly lead to cardiogenic shock or cardiac arrest. If the patient survives these initial injuries, leukocyte accumulation will cause additional epithelial cell damage that usually manifests at 12–24 h and endothelial cell damage in 24–56 h, leading to lung edema and acute respiratory distress syndrome (ARDS). This rapid deterioration of lung tissue and development of ARDS is classified as blast lung, which peaks at 48 h, and the majority of patients demonstrate a fulminate clinical presentation within 6 h from the point of injury. The amount of lung injury correlates with the degree of energy absorbed from the primary blast wave. Patients may present with dyspnea, hypoxia, and/or cough that progressively becomes more productive and with hemoptysis. Decreased breath sounds, rhonchi, and tachycardia can also be found. Hypoxia often precedes the onset of the major symptoms or imaging findings, and thus hypoxia should prompt consideration for a longer period of observation, even in the setting of normal imaging.

Secondary blast lung injury from flying shrapnel or debris may be more obvious on examination given the presence of a penetrating wound or signs of blunt injury over the thorax. Penetrating injury may also present with pathologies such as pneumothorax, hemothorax, and lung contusion. A review of United Kingdom blast injuries from the Afghanistan conflict suggested that primary BLI is more frequently seen in non-survivors than in survivors due to the necessity for close proximity to the explosive source, and such proximity often results in death because of penetrating injury or traumatic amputations [4,21,64,67-69].

BLI may not be immediately obvious on initial evaluation, and signs and symptoms vary, including dyspnea, hypoxia, respiratory distress, hemoptysis, or restlessness. These symptoms may be delayed up 6 h from initial injury [70]. BLI is associated with multi-system injury including limb fractures, vascular injury, abdominal injury, and TM rupture [71]. Identification of these injuries should increase suspicion for BLI. Plain radiographs can help identify pneumothorax, hemothorax, and unilateral or bilateral opacities that signify barotrauma or poor lung expansion. Classically seen findings of BLI include patchy or fluffy nebulous infiltrates resembling a “batwing” or “butterfly” on chest radiograph. However, plain chest radiographs demonstrate poor sensitivity in diagnosing blast lung with abnormalities on chest radiograph present in 52–91.7% of patients [62]. CT may better demonstrate the distribution and extent of alveolar and parenchymal hemorrhage as well as subtle pneumatoceles and pneumothoraces missed on plain radiograph [63]. In one study of pediatric blast victims, plain radiograph demonstrated a sensitivity of 80% for BLI, compared to 100% for CT [72]. In a study of blunt force trauma, chest radiograph had a sensitivity for thoracic injury ranging from 34 to 61%, compared to 100% for CT [73]. CT of the chest is more sensitive than plain radiographs but may suffer from a delay of radiographic findings that trail symptoms or radiographic findings without any clinical correlation [69,74]. CT is recommended if the patient has evidence of respiratory signs, cardiovascular decompensation, or hypoxia [64].

Symptomatic patients should receive supplemental oxygen, high-flow oxygen, and other non-invasive forms of respiratory support based on the oxygen saturation and work of breathing. Patients with BLI require supportive care in a high dependency or intensive care environment, and approximately 80% will require mechanical ventilation [63,75]. The target oxygen saturation is ≥92–94%, but hyperoxia should be avoided unless there is a clear clinical indication (e.g. pneumothorax, pre-intubation, etc.) [76]. Positive pressure ventilation and positive end expiratory pressure (PEEP) should be avoided whenever possible because of the risk of pulmonary alveolar rupture and subsequent arterial air embolism [77]. If respiratory failure does occur, endotracheal intubation should be performed. PBI associated ARDS is a more localized injury than traditional ARDS resulting from systemic inflammatory insults such as sepsis and as such is a milder disease [64]. Conventional mechanical ventilation strategies for ARDS are associated with good outcomes in patients with BLI, including utilization of low tidal volumes, relatively high respiration rates, and PEEP and FiO2 relationship guided by the ARDSnet protocol [66,78]. Patients should be evaluated for the presence of a pneumothorax or hemothorax, and if these are present, chest thoracostomy is recommended prior to CT imaging. Previous literature has suggested that prophylactic bilateral chest tubes can be beneficial in blast lung patients given their tendency to develop

pneumothoraces. More recent studies have suggested that thoracostomy should not be performed prophylactically given the potential damage associated with this procedure in already injured pulmonary tissue [11,42]. Exceptions include need for air transport and those who respond inadequately to intubation and mechanical ventilation [79].

2.1.4. Abdomen

The gastrointestinal (GI) tract is susceptible to injury from the primary blast wave given its heterogeneous density and gas containing components. While an uncommon finding in survivors of blast injury, the bowel can be damaged from the primary blast wave leading to rupture, infarction, ischemia, and hemorrhage [4]. These injuries require a high level of energy transfer from the primary blast wave and are usually found in those in close proximity to the blast. Literature suggests that injuries to the GI tract (stomach, small intestine, large intestine) comprise 48% of the injuries found in abdominal blast victims [8,42,80]. These gas-containing sections of the GI tract are most vulnerable to primary blast effect, but solid organs may also be injured [80]. GI injury also occurs more frequently in underwater explosions given the medium's ability to conduct pressure and deliver higher forces [21,81]. If there is a significant blast, immediate peritonitis can occur from bowel wall rupture, but patients can have a delayed course with nausea, non-peritonitic abdominal pain, testicular pain, tenesmus, or bloody diarrhea as the symptoms of bowel injury [67]. Injuries to the intestinal wall can be masked by damage to the lungs and other organ systems and occur in a more delayed time frame, making serial examination essential [82]. Injury to solid organs such as the liver, spleen, and kidney are generally associated with high intensity primary blast wave and proximity of the patient to the origin of primary blast wave [32,80]. Failure to diagnose abdominal injuries can be catastrophic given the high mortality rate (19%) among immediate survivors [28].

Patients may develop abdominal symptoms up to 14 days after blast exposure [21]. CT is specific but poorly sensitive for abdominal injury, and there is no reliable non-invasive test [67]. Repeat evaluation and monitoring of symptoms are critical to diagnosis of GI injury [8].

The treatment of GI blast injury follows traditional management of non-blast injury with surgical intervention necessary in cases of bowel necrosis and perforation. Delayed perforation and presentation of injury is common in this subset of patients, and an initially unremarkable CT should not be used to definitively exclude injury [8,81]. Patients who are discharged must be counseled that peritonitis and GI injury can be delayed up to 14 days after blast exposure, and thus they should return for further evaluation if they experience further GI symptoms (e.g., pain, vomiting) [21].

2.1.5. Cardiac

Though not gas containing, the heart and its major vascular structures can sustain injuries from the pressure and kinetic forces of blasts. Blast cardiac injuries appear to have pathology and presentation comparable to blunt cardiac trauma, including cardiac contusion, free/septal wall rupture, tamponade, papillary muscle rupture, valve injury, aortic arch injury, and coronary artery dissection [4,12,83-85]. The incidence of these injuries appears to be less common and related to the proximity to the initial blast, but there is little literature on this subject. Krohn et al., first described a novel form of cardiovascular dysfunction secondary to blast injury that involved transiently decreased cardiovascular output and hypotension [86]. A later study found that a unique form of cardiogenic shock occurs when in close proximity to the primary blast wave that involves myocardial depression in the absence of compensatory peripheral vasoconstriction [87]. This dysfunction is suspected to be due to vagally mediated reflex and occurs in a bimodal period seconds after a blast injury and again up to 3 h after [88]. This reflex can result in severe shock without signs of hemorrhage in blast victims, and the cardiac dysfunction is frequently refractory to initial resuscitative efforts [84]. This dysfunction may also lead to bradycardia or normal heart rate even in the setting of hemorrhage from another injury. A rare cardiac complication of blast injury is thoracic compartment syndrome, which likely arises from edematous tissue and hematoma in the mediastinum, resulting in hemodynamic compromise [4,79]. The hallmark of this condition is decreasing blood pressure with positive pressure ventilation [4,79].

Blast patients presenting with chest pain, dyspnea, or hemodynamic instability concerning for cardiovascular injury should be evaluated similarly to those with blunt thoracic trauma given their similar pathology and presentation [12,85]. Current Eastern Association for the Surgery of Trauma (EAST) guidelines recommend screening electrocardiogram (ECG) as a level 1 recommendation. Troponin and echocardiogram are also recommended if cardiac injury is suspected, or if there is hemodynamic instability or persistent new arrhythmia [89].

When cardiac dysfunction secondary to blasts is suspected, reduction of positive pressure ventilation and inotropic support rather than aggressive fluid administration is recommended given the high likelihood of associated lung injury [4,90].

2.1.6. Eye

The eye has a heterogeneous density and is highly sensitive to trauma. As such the organ is susceptible to damage from both the primary blast wave and secondary blast injury. While these injuries are rarely fatal, they are relatively common in this patient population. Ocular injuries were sustained in 8% of the victims of the Oklahoma City bombings [91]. It is also theorized that the bony orbit magnifies the effect of the primary blast wave leading to complex reflections and thus increased damage [92,93]. The most common eye blast injuries include corneal abrasions, eyelid lacerations, globe rupture, and both superficial and intraocular foreign bodies [94]. Secondary blast injury from flying debris makes up the vast majority of the eye injuries in blast patients comprising corneal abrasions, conjunctivitis, superficial foreign bodies, globe perforation, and lid lacerations [95]. However, the primary blast wave may also result in injury, including globe rupture, iris rupture, subconjunctival hemorrhage, and retrobulbar hematoma. In patients with visual acuity changes, eye pain, and orbital swelling, intraorbital trauma should be considered, given blast-related eye injuries have a generally poor prognosis, with only up to one-third achieving resolution in visual acuity [96,97].

If a patient endorses eye pain, double vision, or decreased visual acuity, further investigation including a comprehensive visual examination is recommended, including slit lamp examination and fluorescein. All patients with eye symptoms should be assumed to have globe rupture until proven otherwise. Patients who are unable to communicate symptoms should undergo ocular examination rapidly following stabilization, given the significant propensity of ocular blast injuries to result in vision loss. Ocular ultrasound can be performed and has been found to have specificity of 95% and sensitivity of 95% for identifying signs of intraocular trauma, retrobulbar hematoma, and foreign bodies, but it must be used with caution given the risk of worsening injury if globe rupture is present [98-100]. CT is preferred when available to identify intraocular foreign bodies and globe injury, but this should not be relied on alone for diagnosis, given the sensitivity and specificity of only 76% and 85%, respectively, for globe rupture [101]. Magnetic resonance imaging (MRI) should be used with caution due to the risk of metallic foreign body [101].

Patients with findings of globe rupture, intraocular foreign bodies, and eyelid lacerations should receive ophthalmology consultation. Patients with retrobulbar hematoma and signs of increased intraocular pressure >40 mmHg should emergently receive lateral canthotomy and cantholysis within 60 min of injury to reduce risk of vision loss from orbital compartment syndrome [102,103].

2.1.7. Musculoskeletal

The musculoskeletal system is frequently affected in blast injuries. In one cohort of 101 blast victims, 57% had extremity injuries, and of those 75.2% had at least one fracture with over 90% being open fractures [104]. There is a tendency for more fractures the closer a victim is to the explosive epicenter, with more severe open fractures being associated with increased morbidity [104]. Traumatic amputation of the limbs affects 1–3% of victims and also indicates close proximity to the blast center [105]. The mechanism of traumatic amputations requires a combination of the primary blast wave damaging skin, soft tissue, and skeletal structures and the secondary fragmentation projectiles stressing the limb to the critical point of separation [106]. Significant morbidity has been associated in dismounted (on foot rather than in vehicle) blast victims who sustained pelvic fractures, with over 25% of deaths of U.S. military casualties in 2008 from blasts having pelvic fractures [107].

A thorough examination of the extremities is warranted, include appearance, palpation, range of motion, and neurovascular status. Tourniquet application is necessary for those with severe extremity hemorrhage. Plain radiographs of the affected area should be obtained after initial stabilization for those with evidence of traumatic injury. Fractures or dislocations should be emergently reduced if causing neurovascular compromise. Other extremity injuries may be splinted after initial stabilization. Soft tissue wounds will be discussed in detail later in this review.

2.2. Mass casualty incidents

An important aspect in the evaluation of blast injuries is resource management given that blasts may result in a mass casualty incident (MCI). Explosions can cause an influx of critically injured patients in a short amount of time. In the Madrid bombings of 2004, 43% of patients had severe or life-threatening injuries, and over 30 patients required CT imaging within a 3-h period [108]. The Boston Marathon Bombing in 2013 resulted in 3 deaths and over 250 injured [109]. Emergency physicians must attempt to balance the outsized demand for and relative lack of available diagnostic tools when evaluating these patients due to oversaturated hospitals, and when able, use modalities such as ultrasound. An “upside-down” triage model should be expected if scene control and triage was not maintained where the most severely injured arrive after the less injured who bypass emergency medical services (EMS) triage and go directly to the closest hospitals. In these events it is recommended to double the first hour's casualties for a rough prediction of the total “first wave” of casualties [42]. Additionally, when the decision to complete advanced imaging is made in a stable critically ill patient, some literature suggests that selective CT scans of one body part should be avoided. Instead, standard-protocol contrast enhanced whole body CT should be implemented from the head to pelvis given the high incidence of multiple sources of injury in these patients and to identify foreign bodies not discovered on physical examination.

3. Pearls and pitfalls

3.1. What are important considerations in establishing scene safety?

An explosion is a sudden and rapid increase in volume and releases energy in an extreme manner, which can injure patients and damage surrounding structures, making patient assessment and retrieval at the scene dangerous. A blast wave with a pressure of 275 kPa can significantly damage reinforced buildings. In contrast, it takes 480 kPa to commonly cause significant pulmonary injury [31,42,105,110,111]. Weakened structures may collapse and pin victims, preventing extraction and resulting in additional crush injury. Immediately after a blast the scene may be complicated by secondary explosive devices placed in waiting to harm any would-be rescue teams. Scattered explosive fuel may lead to a delayed detonation. Fire and smoke from incendiary bombs make rescue dangerous. Shrapnel may also litter the surroundings, hampering efficient entrance to the area. Explosive devices can inject various materials into the air that harm the respiratory systems and impair the vision of patients and responders. Multiagent bombs that include chemical, biologic, or radioactive release can further contaminate the scene, adding another layer of difficulty in safely reaching and evacuating patients. Patients can also harm responders and other injured patients through acting as a vector for the spread of bomb contaminates or directly harm others (i.e., terrorist, brain injured or agitated patient).

In order to minimize these risks, emergency personnel should survey the area carefully and utilize appropriate personal protective equipment (e.g., eye protection, breathing mask for toxic fumes, hard hat, heavy gloves, etc.) [42,112,113]. Once the area has been deemed safe to enter and precautions taken, it is also prudent to set up a staging point outside of the immediate, threatened area to allow patients to be screened, searched for weapons, decontaminated, and appropriately triaged to the appropriate medical facilities [114]. The mass influx of potential patients commonly leads to confusion with up to 50–80% of victims at a blast event going to hospitals on their own or taken to hospitals by people at the scene [42,113]. The lack of effective triage and staging can increase the potential for the spread of biologic/chemical contamination and unnecessary overutilization of any one hospital. Studies of civilian bombings report that approximately 18.7% of those involved have serious injuries, while a significant number of patients may present for care who do not need immediate medical attention and overwhelm medical resources [28]. Lack of effective triage at the scene of an MCI and patients transporting themselves can lead to poor patient distribution; this was seen in the 2017 Las Vegas shooting where 215 gunshot wound victims presented to one hospital while there was additional capacity in the local healthcare system [115]. Failure of effective triage may delay or prolong diagnosis and management and allow those patients with critical and salvageable life-threatening injuries to go untreated. Events that involve building collapse and crush injury may also result in delayed presentation of critically ill patients to healthcare facilities due to the time needed for extraction.

3.2. How can characteristics of the blast impact injury patterns?

3.2.1. Enclosed vs open space

An important factor in blast injury includes explosions that occur in enclosed versus open spaces. Enclosed spaces enhance the effect of the primary blast wave and result in greater injury. When the blast wave is confined, it is able to rebound and reflect back to the patient from additional directions. This allows for even small explosions in enclosed spaces to have a significantly greater effect, compared to open air explosions [116,117]. Bombings that occur in confined spaces have a higher amount of primary blast wave injury (e.g., pneumothorax, BLI, TM rupture) [117,118]. In open air explosive events, injuries are primarily penetrating soft tissue injuries caused by shrapnel, and blast wave injury typically affects only those patients close to the blast epicenter [119].

3.2.2. Burn injury

Patients exposed to blasts frequently have concomitant burn injury, with 52% of all burns from Operation Iraqi and Enduring Freedom sustained from explosions secondary to hostile action [120]. Similar rates were noted by U.S. service members in the Vietnam War suggesting a high rate in modern combat scenarios, while the rate in civilian blasts can vary greatly depending on the source and cause of the blast [120]. In the prehospital setting the traditional strategy of fluid resuscitation applies with emphasis on targeting fluid boluses only to treat hypotension and preventing any significant volume overload, which increases the risk of BLI and subsequent ARDS [42]. Blast victims with burns are also likely to have additional traumatic injury and will require transport to a hospital with burn and trauma specialists. Additional fluids can be administered after admission targeting urine output and systemic perfusion to prevent respiratory injury [42]. These patients also have a

high incidence of smoke inhalation injury, occurring in 55% of blast victims [121]. Smoke inhalation injury can include burns to the respiratory mucosa and/or carbon monoxide poisoning which further complicate patient management [122,123]. Pulmonary manifestations do not usually appear in thermal injuries without BLI and are unique to this mechanism [121]. Severe burns may also cause a delay in diagnosing additional blast damage to other organs beyond the lungs by making examination difficult, and burns can also distract from other injuries as providers may fixate on a severe burn injury.

3.2.3. Crush injury

Explosions commonly cause structural damage and may lead to entrapment of victims under debris. Crush injury is the compression of a body part that causes muscle edema and neurovascular compromise, and crush syndrome is a series of systemic metabolic changes that occur secondary to the crush injury and release of cellular contents into the circulation [124]. Reperfusion of the crush injury leads to release of toxic muscle cell breakdown metabolites and electrolytes throughout the body which may lead to metabolic derangements, hypotension, and cardiac arrhythmias [125,126]. The edema of crushed body parts can result in significant third spacing and subsequent hypovolemia. Patients may sequester >12 L of fluid in the crushed area over a 48-h period requiring substantial fluid resuscitation [42]. The swelling can also lead to compartment syndrome further endangering vascular supply. Keys in assessment and initial treatment of compartment syndrome include immobilization of the affected part, measurement of compartment pressure, and fasciotomy of the affected compartment while resuscitating the patient and ensuring perfusion without over hydrating and causing more edema. A low threshold for fasciotomy is recommended, as patients are often critically ill, and an accurate neurovascular assessment is difficult to obtain [127]. In crush syndrome the release of myoglobin and other muscle cell components into the systemic circulation may cause rhabdomyolysis and lead to renal injury/failure. If not effectively hydrated to maintain renal perfusion and diuresis of 200–300 mL/h, severe renal injury may occur, which may require hemodialysis [125,126,128]. Additional metabolic abnormalities of crush syndrome include hyperkalemia, hypocalcemia, and acidosis that worsens renal injury and may lead to fatal arrhythmias if not treated appropriately. Early mortality in untreated crush is due to hyperkalemia and hypovolemia making close cardiac monitoring and hydration integral. Urine alkalinization is controversial but can be utilized in those with crush syndrome in the setting of increasing creatine kinase and urinary pH < 6.5. However, mannitol may worsen oliguria and dehydration. If the patient survives the initial injury, they remain at risk of renal failure, coagulopathy, hemorrhage, and sepsis during hospitalization [125,126,128].

3.2.4. Combined injury pattern

A combined injury pattern is common in patients with blast injury. In the 1995 Oklahoma City bombing, 98% suffered soft tissue injuries, 57% were treated for fractures or dislocations, 53% were treated for head injuries, 24% had severe lacerations, 37% had eye injuries, and 11% were treated for burns [129]. Similar injury diversity was illustrated in the Madrid train bombings of 2004 where the majority of the 512 casualties had minor injury, but 72 patients with severe illness had a combination of injury patterns, most commonly maxillofacial and long bone fractures combined with head trauma and/or BLI [81].

The overlap of injuries in this patient population is unique, with a variety of possible injury patterns. Clinicians must ensure a systematic approach in the evaluation and management of these patients, with frequent reassessments. Patient hemodynamics and volume status must be balanced. Injuries can also be missed on initial evaluation, and repeat assessment is necessary. These combined injuries also benefit from a multidisciplinary team in the operating room given the likelihood of multiple lethal wounds and concurrent orthopedic trauma.

3.2.5. Wound management

Soft tissue wounds are the most prevalent injury pattern in blast victims, occurring in 70–98% of patients [129,130]. Explosions can result in penetrating injury with shrapnel, dirt, debris, and human remains in the case of a suicide bombing. Wounds can be grossly contaminated and require extensive irrigation and debridement [127]. If significant soft tissue wounds are present, broad-spectrum antibiotics and tetanus prophylaxis should be administered, as blast wounds tend to be colonized by multiple pathogens with the most common cultured pathogen in one cohort being Acinetobacter species [127]. There appears to be little to no consensus in what constitutes broad antimicrobial coverage, with current U.S. military guidelines recommending single-dose cefazolin IV for war wounds, the U.S. Joint Trauma System recommending moxifloxacin oral or ertapenem IV, and other government public health agencies recommending co-amoxiclav IV or cefuroxime/metronidazole IV [131-134]. In wounds with underlying fractures, administration of antibiotics within 3 h of injury reduces infection, and a more recent study suggests dosing >66 min increases infection risk [135]. Complex soft tissue wounds that occur in blasts have a high incidence of invasive fungal infections due to the transmission of environmental matter into the wound. In a cohort of combat casualties from Afghanistan, all diagnosed invasive fungal infections occurred in blast patients [136]. In less complex wounds, delayed primary closure should be considered. Tetanus vaccination status should be assessed, and patients require close follow-up.

3.3. What imaging is recommended in these patients? Should these patients undergo the "trauma pan-scan"?

Blast victims can range from those with little to no injury to a complex polytrauma patient. This wide spectrum of acuity makes decisions of resource utilization and imaging dependent on a multitude of factors. The need for imaging should be based on the mechanism of the blast (e.g., type of explosive; distance; evidence of secondary, tertiary, and quaternary injury), the patient's symptoms, vital signs, and examination. Any abnormality in either of these greatly increases the risk of not only injury, but also polytrauma, given the ability of the energy for the blast wave to transmit across the body. The extent to which each imaging modality is utilized should be tailored to the severity of illness and mechanism of the blast. There is some consensus that critically ill patients should receive at minimum a chest x-ray and pelvis x-ray or their equivalents [4]. However, given the increased sensitivity of CT, the critical state of these patients, and the extent of injuries, obtaining CT imaging of the head, cervical spine, chest, abdomen, and pelvis is a viable option. The high likelihood of polytrauma in this patient population and risk of missed injury makes this a reasonable approach in patients who are critically ill but must be balanced with other factors. Plain films and ultrasound of the chest and abdomen offer increased speed and require less resources compared to CT and can be considered prior to CT imaging given their ability to uncover rapidly worsening injury that requires emergent intervention.

In these scenarios, appropriate triage is integral to limit bottlenecks that can cause delay and thus harm.

3.3.1. Ultrasonography

The *E*-FAST examination is specific (98%) in identification of free fluid in the abdomen, pneumothorax, hemothorax, and cardiac tamponade, all of which can be seen in blast injury, but the absence of these pathologies on ultrasound does not definitively exclude them (sensitivity of 85–96%) [31,137]. However, given the speed, lack of radiation, and ease of use, ultrasound can assist in appropriately triaging patients until further imaging can be obtained.

3.3.2. Plain radiographs

In the acute setting, chest and abdominal radiographs can diagnose rib fractures, hemothorax, pneumothorax, foreign bodies, abdominal

perforation, and the characteristic findings of BLI [31]. Plain radiographs are also useful in diagnosing musculoskeletal fractures, dislocations, and extremity foreign bodies. Radiographs can rule in but not exclude severe intrathoracic or abdominal pathologies.

3.3.3. Computed tomography

CT offers a rapid means of identifying abdominal trauma with high sensitivity (97–98%) and specificity (97–99%) [138-140]. One disadvantage of CT imaging in abdominal injury is its poor sensitivity detecting mesenteric and bowel injuries without oral contrast [141-143]. However, CT has high sensitivity (94%) and specificity (95%) in detecting organ injury in those with penetrating abdominal trauma [144]. CT for evaluation of cardiac injury from blunt chest trauma has poor sensitivity, but it has high sensitivity for other thoracic injuries due to blunt trauma (95.4%) [145-147]. CT may be used acutely to investigate for intracranial bleeds, cerebral edema, loss of grey-white matter differentiation from TBI, traumatic fractures, and foreign bodies. CT angiography with IV contrast can also assist in the diagnosis of aortic injury, active extravasation/hemorrhage, and limb perfusion [148]. While CT of the head is useful in evaluating for severe intracerebral injury such as intracerebral hemorrhage, one study found that over half of patients affected with blast injury to the brain had no evidence of injury on CT [149]. It is also important to not delay life-saving surgery in order to obtain CT imaging.

3.3.4. Magnetic resonance imaging (MRI)

Given the time needed to complete MRI and the likelihood of metal shrapnel in blast injury, MRI has little relevance in the acute setting with critical patients [31]. While MRI is useful in stratifying spinal cord injury and TBI, it is not recommended until CT has been completed to evaluate for foreign bodies [42].

3.4. What blast injuries do clinicians miss?

The primary blast wave is the most unique aspect of explosion injury and also contributes to the injuries that can be missed on first assessment. The pressure wave is able to diffuse through the body without necessarily causing any outside indication of entry, and patients may not have evidence of secondary, tertiary, or quaternary injury. Patients in close proximity to the blast may have significant internal injury without any external evidence, unlike traditional trauma where external examination typically indicates injury. While visual inspection may be misleading, additional components of the physical examination such as assessing for tenderness, respiratory distress, hypoxia, vital sign abnormalities, neurological deficits, and GCS may offer insight into injury. Imaging based on history and evaluation is also an essential component of ED assessment of blast injury victims.

Unlike traditional traumatic injuries seen commonly in the ED, blast related abdominal injuries and BLI tend to have a delayed course until symptoms and signs arise. Symptoms from BLI typically occur within 6 h of injury but could be missed in the setting of other injuries [42]. Severely delayed blast lung is uncommon, and most with pulmonary blast injury have their maximal clinical course evident within several hours of the blast exposure [68].

Symptoms from PBI may not manifest until 14 days after the event, and PBI may not be initially evident on CT. This delay makes misdiagnosis more likely and increases morbidity and mortality. Return precautions and education regarding the delayed nature of abdominal blast injury is needed for all blast injury patients.

Another factor that contributes to misdiagnosis is the overwhelming scope of injuries seen in blast victims and the prioritization of emergency resuscitation and damage control surgery. In a sample of military veterans who survived polytrauma due to blasts, the most commonly overlooked conditions included concussion, soft-tissue damage, PTSD/acute stress reaction, nerve damage, hearing loss, and chronic infection [150]. While these pathologies are not acutely life-threatening, early identification and treatment would likely prevent harm and morbidity in those survivors. Thus, it is important to remain systematic in the approach to blast injury patients, consider these common conditions, and frequently reassess for their development in the post-acute phase of treatment. It is critical to assess for auditory, optical, neurologic, and abdominal injury in blast patients. Assessment should focus on the mechanism of injury, "the blast", and the various injuries to prevent failure in diagnosis [150].

While not prevalent initially in the ED, the survivors of blasts frequently suffer from PTSD and/or acute stress reactions that may go unrecognized for years [15,151-153]. This predisposition for PTSD makes regular screening and education of patients on the neurologic and psychiatric symptoms imperative to prevent residual harm from the initial blast and to allow patients and their providers the ability to identify the condition when it arises.

Table 2 lists pearls concerning the evaluation and management of blast injuries.

Table 2
Blast injuries pearls.

- Damage from the primary blast wave is enhanced in enclosed spaces.
- Blast victims are prone to suffering polytrauma, and thus a comprehensive examination is recommended. All blast patients should be evaluated for injury to the organ systems prone to insult from the primary blast wave.
- While the tympanic membrane is sensitive to blast forces, the presence or absence of tympanic membrane rupture is not a reliable marker for further blast injury.
- Injury caused by the primary blast wave (blast lung injury, abdominal injury, traumatic brain injury, ocular injury) may present with little evidence of external trauma.
- Brain injury is common, including cerebral contusion, cerebral edema, diffuse axonal injury, and extra-axial hematomas. In moderate and severe TBIs without surgical indications, secondary injury from posttraumatic hypotension, hypoxia, hypercarbia, and hypoglycemia must be prevented.
- Blast lung is the most common cause of death from the primary blast wave and is not significantly prevented by body armor.
- Blast lung classically presents on chest radiographs as bilateral patchy lung contusions with a "batwing" distribution.
- IV fluids should be limited in blast injury patients given the increased risk of blast lung injury.
- Non-invasive positive pressure ventilation and mechanical ventilation may improve hypoxia and work of breathing but may increase the risk of barotrauma in blast victims.
- Intubated patients should be ventilated with a lung protective strategy following the ARDSnet protocol, and peak airway pressures should be minimized.
- Gastrointestinal and abdominal injury from the primary blast wave can present in a delayed fashion and is not always detected on initial CT imaging.
- Cardiac injury may result in hemodynamic compromise.
- Effective triage is important when evaluating victims of blasts. Flow in the ED should be prioritized with the utilization of advanced imaging and surgical resources activated based on triage.
- Blast wounds are prone to contamination, and a low threshold for antibiotic administration is recommended.
- Crush injuries and burns are common in blast victims.

4. Conclusion

Blast injury is associated with significant morbidity and mortality. The blast mechanism can lead to damage across multiple organ systems. The primary blast wave causes unique effects, with blast injuries affecting the pulmonary, neurologic, auditory, cardiac, ocular, musculoskeletal, and GI systems. Identification of these effects is important for emergency clinicians managing the care of these potentially complex patients. An understanding of the mechanism of blast injury and the corresponding common injuries can assist emergency clinicians in diagnosing and managing this potentially deadly spectrum of injuries.

CRediT authorship contribution statement

Josh Bukowski: Writing – review & editing, Writing – original draft, Visualization, Validation, Resources, Conceptualization. **Craig D.**

Nowadly: Writing – review & editing, Writing – original draft, Validation, Supervision, Resources, Conceptualization. **Steven G. Schauer:** Writing – review & editing, Validation, Supervision, Resources, Conceptualization. **Alex Koyfman:** Writing – original draft, Visualization, Validation, Supervision, Resources. **Brit Long:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Conceptualization.

Declaration of Competing Interest

None.

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