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PATHOPHYSIOLOGICAL MECHANISMS OF COMBAT INJURIES OF THE LOWER EXTREMITIES AND THEIR IMPACT ON THE CHOICE OF SURGICAL TACTICS

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ABSTRACT

Introduction. Combat injuries of the lower extremities remain one of the leading causes of severe disability among military personnel. The high-energy nature of blast, gunshot, fragment, thermal, and barotraumatic injuries gives rise to complex local and systemic pathophysiological responses that largely determine the course of traumatic disease and the prospects for limb salvage.

Aim. To systematise current data on the pathophysiological mechanisms of tissue injury in the lower extremities across different types of combat trauma and to clarify their significance for the selection of surgical tactics.

Materials and methods. A narrative review of the literature indexed in PubMed/MEDLINE, Scopus, and Google Scholar for the period 2016–2026 was performed using the keywords "blast injury", "gunshot wounds", "fragment wounds", "compartment syndrome", "reperfusion injury", "endothelial dysfunction", and "combat trauma". English-language full-text publications addressing the pathogenesis of combat trauma were included. After screening and removal of duplicates, 45 sources were assessed, of which 15 were included in the final analysis.

Results. Regardless of the physical mechanism of injury, combat trauma may be characterized by a three-zone pattern comprising a zone of primary necrosis, a zone of secondary injury (zone of molecular concussion), and an area of potentially reversible tissue changes. Blast injuries combine the effects of the blast wave, fragments, and thermal energy and may result in extensive muscle destruction and rhabdomyolysis; gunshot and fragment wounds are characterized by kinetic energy transfer, temporary cavitation, and substantial tissue contamination; thermal injury and barotrauma may produce zones of tissue stasis associated with increased capillary permeability and interstitial oedema. Progression of tissue injury within these zones is driven by a common cascade involving endothelial dysfunction and glycocalyx damage, the no-reflow phenomenon, intracellular calcium overload, mitochondrial dysfunction, reperfusion-associated oxidative stress, systemic inflammation, and compartment syndrome.

Conclusions. Despite differences in their physical mechanisms, various types of combat trauma share common pathogenetic mechanisms of secondary tissue injury and necrosis, providing a basis for unified approaches to monitoring tissue viability. Understanding these mechanisms supports the rational selection of surgical tactics, timely assessment of tissue viability, and improved limb salvage outcomes.

Keywords: blast injuries, compartment syndromes, necrosis, war-related injuries, wounds, gunshot.

INTRODUCTION

Combat trauma remains one of the principal causes of death and disability among military personnel in contemporary armed conflicts. Despite advances in personal protective equipment and continued improvements in medical evacuation systems, extremity injuries account for the largest proportion of combat wounds. Injuries of the lower extremities are of particular clinical importance because they are frequently associated with extensive soft-tissue destruction, long-bone fractures, vascular injury, and a high risk of limb loss [1, 2, 23].

Contemporary combat injuries are typically characterized by high-energy mechanisms. The most common include blast injuries, gunshot and fragment wounds, and combined thermal and barotraumatic injuries. Unlike civilian trauma, these injuries involve the transfer of substantial mechanical energy to tissues, the formation of extensive zones of indirect tissue injury, and the initiation of complex local and systemic pathophysiological responses [1, 3, 28].

For a long time, attention was focused primarily on the immediate zone of mechanical tissue destruction.

Current research, however, demonstrates that the severity and clinical course of injury depend to a large extent on secondary processes that develop over the hours and days following trauma. These include endothelial dysfunction, microcirculatory disturbances, activation of the coagulation cascade, oxidative stress, reperfusion injury, and the systemic inflammatory response [4-6, 29].

One of the key concepts in modern military traumatology is the so-called zone of molecular concussion, or parabiosis. In contrast to the zone of primary necrosis, in which tissues undergo irreversible injury at the time of trauma, structural integrity within the zone of parabiosis may be preserved for a certain period. Nevertheless, profound metabolic disturbances persist at the cellular and subcellular levels, potentially resulting either in restoration of tissue viability or progression to secondary necrosis [4, 7, 8, 32, 35].

Despite differences in their physical mechanisms, blast injuries, gunshot and fragment wounds, and thermal and barotraumatic injuries share several common pathogenetic mechanisms. These include microvascular disturbances, the no-reflow phenomenon, progressive tissue hypoxia, rhabdomyolysis, compartment syndrome, and the systemic inflammatory response. These mechanisms substantially influence the extent of surgical debridement, the need for fasciotomy, the feasibility of reconstructive interventions, and the prospects for limb salvage [5, 9-11].

Therefore, summarizing current knowledge of the pathophysiology of combat injuries of the lower extremities is essential for refining treatment strategies and improving outcomes in injured patients.

AIM

The aim of this work was to systematise current data on the pathophysiological mechanisms of tissue injury in the lower extremities across different types of combat trauma and to clarify their significance for the choice of treatment strategy.

MATERIALS AND METHODS

The literature search was conducted in the PubMed/MEDLINE, Scopus and Google Scholar databases for the period 2016-2026, using the keywords "blast injury", "gunshot wounds", "fragment wounds", "compartment syndrome", "reperfusion injury", "endothelial dysfunction" and "combat trauma", together with their combinations. English-language articles reflecting current understanding of the pathophysiological mechanisms of combat trauma to the lower extremities were included in the review; publications without full-text access and studies not directly related to the subject of the review were excluded. After screening and removal of duplicates, 45 sources were examined, of which 15 publications that most fully reflect the current state of the problem were included in the final analysis.

RESULTS

Blast injury

Blast injury is among the most severe forms of combat injury to the lower extremities and is characterised by the combined action of the blast wave, fragments, thermal energy and mechanical acceleration of the body. Unlike other types of trauma, injury from explosions arises not only from the direct destruction of tissue but also as a consequence of substantial secondary pathophysiological changes [1, 2, 17, 19].

Primary tissue necrosis develops within the zone of direct blast effect, and around it a zone of secondary injury, or zone of molecular concussion, is formed. In this area the structural integrity of the tissues may still be preserved, yet pronounced disturbances of microcirculation and cellular metabolism are already developing and prepare the ground for the progression of necrosis [1, 4].

Endothelial damage and activation of the coagulation cascade play a prominent role in the pathogenesis. The formation of microthrombi and the development of the no-reflow phenomenon lead to tissue hypoxia even when major vessel blood flow is not substantially compromised [4, 5]. Oxidative stress, reperfusion injury and the development of acute compartment syndrome act as additional factors that aggravate the damage [6, 9, 12, 30].

Extensive destruction of muscle tissue is frequently accompanied by rhabdomyolysis and a systemic inflammatory response, which increases the risk of multiple-organ complications [6, 11, 13]. The severity of blast injury is thus determined not only by the extent of primary tissue destruction but also by the development of secondary ischaemic damage.

Gunshot wounds

The pathophysiology of gunshot wounds is largely determined by the transfer of the projectile's kinetic energy to the tissues. In addition to the formation of the wound tract, bullet passage generates a temporary pulsating cavity that causes tissue stretching and injury at a considerable distance from the projectile trajectory [3, 14].

Muscle tissue and the microvascular bed are particularly vulnerable to cavitation-related injury. Damage to cell membranes, mitochondria, and endothelial cells triggers a cascade of secondary injury involving impaired microcirculation, tissue hypoxia, and the development of secondary necrosis [3, 4, 15].

A characteristic feature is the formation of a zone of molecular concussion, in which macroscopic signs of tissue destruction may not yet be apparent, although pronounced cellular and vascular disturbances are already present. This zone may substantially influence the final extent of viable tissue and the need for further surgical intervention [3, 8, 32].

Heavy wound contamination and the risk of delayed vascular injury are also clinically important, as vascular complications may result in the progression of ischemia several hours or even days after injury [3, 14, 23].

Fragment wounds

Fragment wounds are characterised by multiple foci of injury and considerable variability of clinical presentation. The irregular shape and unstable trajectory of fragments favour the formation of complex wound tracts and multiple zones of tissue destruction [1, 2].

Each fragment creates a local zone of primary injury and a surrounding area of secondary changes. In multiple wounding, these zones may overlap, forming extensive areas of ischaemia and secondary necrosis [8].

Important features of fragment wounds include considerable contamination of tissue with foreign material, pronounced microcirculatory disturbances and a high risk of infectious complications, particularly when combined with massive soft-tissue damage [2].

Thermal injury and barotrauma

Thermal and barotraumatic injuries frequently accompany combat trauma and may substantially worsen the course of the wound-healing process. Thermal injury is characterized by the development of coagulative necrosis and the formation of a zone of stasis, the viability of which influences the subsequent progression of tissue damage [10].

Microcirculatory disturbances, tissue hypoxia, and cellular energy deficiency develop within the zone of stasis and contribute to the progression of secondary necrosis. Similar processes are observed in electrical injuries, in which severe damage to deep tissues may be disproportionate to the external manifestations of trauma [10, 11]. Barotrauma is associated with endothelial injury, increased capillary permeability, and the development of interstitial oedema. Impaired microcirculation, microthrombosis, and tissue hypoxia create conditions conducive to secondary injury similar to those observed in other high-energy forms of combat trauma [4, 15].

Despite differences in their underlying mechanisms, various types of combat injury exhibit both specific features and shared pathophysiological patterns. The principal characteristics of individual injury types are presented in Table 1.

Analysis of these data indicates that, despite the differing nature of the primary injury, all types of combat trauma exert their effects through a number of shared pathophysiological mechanisms. It is these mechanisms that determine the progression of secondary necrosis, tissue viability and treatment outcomes in the injured.

From pathophysiological mechanism to surgical decision

Each pathophysiological mechanism has a specific therapeutic window that determines the timing and type of surgery.

Restoration of perfusion (0-8 h). The priority is revascularisation within 4–6 h after vascular injury. Temporary intravascular shunts may maintain perfusion during damage-control surgery. External fixation is preferred initially, while debridement should be limited to clearly non-viable tissue. Gra-

Table 1. Pathophysiological features of different types of combat injury of the lower extremities

Type of injury	Leading mechanism of primary injury	Principal mechanisms of secondary injury	Clinical significance
Blast injury	Blast wave, fragment impact, mechanical acceleration	Endothelial dysfunction, microthrombosis, reperfusion injury, compartment syndrome	High risk of limb loss and multiple-organ complications
Gunshot wounds	Transfer of kinetic energy and cavitation injury	Zone of molecular concussion, microcirculatory disturbance, secondary necrosis	Tissue damage beyond the wound tract
Fragment wounds	Multiple wound tracts and polyfocal tissue destruction	Ischaemia, microvascular disturbance, infectious complications	High level of contamination and occult necrosis
Thermal injury	Coagulative necrosis	Impaired microcirculation within the zone of stasis, tissue hypoxia	Progression of injury depth during the first days after trauma
Barotrauma	Action of the blast wave and pressure differentials	Endothelial dysfunction, oedema, microthrombosis	Occult tissue damage and impaired perfusion

Fig. 1. Algorithm linking pathophysiological changes, clinical and laboratory markers and surgical tactics in combat injuries of the lower extremities

Note: ABI — ankle–brachial index; BTSS — Blast Trauma Severity Scale; CK — creatine kinase; DAMPs — damage-associated molecular patterns; ICG-FA — indocyanine green fluorescence angiography; ICP — intracompartmental pressure; MDR — multidrug-resistant; MESS — Mangled Extremity Severity Score; NIRS — near-infrared spectroscopy; NPWT — negative pressure wound therapy; ΔP — perfusion pressure.

dual tourniquet release reduces reperfusion injury [14, 26, 27].

Fasciotomy (6-24 h). Indicated for confirmed compartment syndrome (pressure >30 mmHg or ΔP ≤30 mmHg) and selected high-risk injuries. Complete decompression of all compartments is essential. Routine prophylactic fasciotomy has not consistently reduced amputation rates and increases wound complications; therefore, indications should be guided by clinical and pathophysiological findings rather than applied routinely [24, 28-30].

Staged debridement (24-72 h). Initial surgery removes only non-viable tissue, followed by planned re-exploration every 24-48 h until no further necrosis remains. Primary closure is contraindicated. Negative-pressure wound therapy supports wound-bed preparation between procedures [15, 31].

Timing of initial surgery. The traditional 6-hour rule has been replaced by evidence supporting debridement within 24 h, with earlier intervention reserved for vascular compromise, gross contamination, or Gustilo IIIB injuries [13, 32-34].

Systemic consequences (24 h-7 d). Reperfusion and muscle destruction may cause rhabdomyolysis and acute kidney injury, requiring aggressive resuscitation and renal support when indicated. At this stage, limb salvage versus amputation should be reassessed using an integrated severity assessment [11, 14, 18, 25, 27, 35,].

Reconstruction (5-14 d). Definitive fixation and soft-tissue coverage should be performed only after complete wound control, with reconstruction delayed until the wound is clean and free of progressive necrosis. Antibiotic therapy should be culture-guided [12, 21, 22, 31].

DISCUSSION

Common mechanisms of secondary tissue injury in combat trauma

Despite substantial differences among blast, gunshot, fragment, thermal, and barotraumatic injuries, current research points to common pathogenetic mechanisms that influence the subsequent fate of damaged tissue. These processes substantially shape the clinical course of injury, the extent of secondary necrosis, and the prospects for limb salvage [4, 5, 7].

Irrespective of the type of injury, the primary mechanical or thermal insult initiates a cascade of cellular and vascular responses that continues to evolve over the following hours and days. This may result in a classical three-zone pattern of injury comprising a zone of primary necrosis, a zone of secondary injury, and an area of potentially reversible changes. Whereas tissues within the zone of primary necrosis are non-viable from the time of injury, the subsequent fate of tissues within the zone of secondary injury depends on the severity of local and systemic pathophysiological processes [1, 3, 10].

Endothelial dysfunction is one of the central mechanisms of secondary injury. Damage to the endothelial glycocalyx is associated with disruption of vascular barrier function, platelet activation, and initiation of the coagulation cascade. Impaired regulation of vascular tone may lead to vasospasm, increased capillary permeability, and progressive tissue oedema. At the same time, conditions conducive to microthrombus formation may develop, impairing tissue perfusion even when blood flow through major vessels is preserved [4].

Endothelial injury may contribute to the development of the no-reflow phenomenon, in which restoration of systemic haemodynamics is not accompanied by adequate tissue reperfusion at the level of the microcirculation. Microvascular thrombosis, blood cell stasis, endothelial swelling, and compression of capillaries by interstitial oedema may create conditions in which tissue hypoxia persists even after macroscopic circulatory disturbances have been corrected [4, 8].

Disruption of intracellular ionic homeostasis plays a prominent role in the progression of tissue injury. Mechanical, thermal, or ischaemic damage to cell membranes may result in excessive calcium influx into the cytosol. Increased intracellular calcium concentrations activate proteases, phospholipases, and endonucleases, which contribute to the degradation of cellular structural components. Mitochondrial function is impaired concurrently, leading to reduced adenosine triphosphate (ATP) synthesis and cellular energy failure [7, 10, 11].

Further progression of tissue injury is associated with oxidative stress. Reperfusion of ischaemic tissue is accompanied by increased generation of reactive oxygen species, which can damage cell membranes, proteins, and nucleic acids. Reperfusion injury may not only exacerbate local tissue destruction but also contribute to persistence of the systemic inflammatory response [6, 7].

Activation of systemic inflammation is a common consequence of severe combat trauma. Cell destruction is accompanied by the release of intracellular components that can act as danger-associated molecular patterns (DAMPs). In response, neutrophils, macrophages, and other immune cells become activated and produce pro-inflammatory cytokines and other inflammatory mediators. In the setting of extensive injury, the local inflammatory response may progress to systemic inflammatory

response syndrome and contribute to multiorgan dysfunction [6].

Compartment syndrome occupies a distinct place among the secondary mechanisms of tissue injury. Regardless of the initial cause, the combination of tissue oedema, haemorrhage, and impaired venous outflow can lead to increased intrafascial pressure. Compression of blood vessels and nerves causes further deterioration of microcirculation, establishing a vicious cycle of progressive ischaemia. Without timely decompression, compartment syndrome may become an independent cause of limb loss even after the primary injury has been adequately managed [9].

Thus, despite the diversity of the physical mechanisms of injury, the pathophysiology of combat trauma to the lower extremities involves a shared cascade of interrelated processes, including endothelial dysfunction, microcirculatory disturbances, the no-reflow phenomenon, oxidative stress, systemic inflammation, and compartment syndrome. These mechanisms substantially influence tissue viability, the extent of secondary necrosis, and treatment outcomes in injured patients.

CONCLUSIONS

1. Notwithstanding the diversity of the mechanisms by which they arise, combat injuries of the lower extremities share common pathophysiological patterns in the development of secondary tissue damage. The leading roles belong to endothelial dysfunction with glycocalyx shedding, microcirculatory disturbance and the no-reflow phenomenon, intracellular calcium overload with mitochondrial dysfunction, reperfusion-related oxidative stress, rhabdomyolysis, systemic inflammation and compartment syndrome.

2. Each element of this cascade has a measurable clinical or laboratory correlate – lactate and base deficit for shock and endotheliopathy; total ischaemia and tourniquet time, ankle–brachial index, tissue oxygen saturation and indocyanine green fluorescence for perfusion; perfusion pressure ΔP for intrafascial hypertension; creatine kinase, myoglobinuria and diuresis for muscle necrosis and its systemic consequences. This permits the extent of injury to be assessed objectively rather than by inspection alone, and it is of particular value in the sedated or intubated casualty, in whom the classical clinical signs are unavailable.

3. Each mechanism has a defined temporal window, and that window determines the operation: revascularisation with temporary intravascular shunting within 4-6 h; four-compartment fasciotomy over the full length of the segment when $\Delta P \leq 30$ mmHg, or when ischaemia exceeds 4-6 h, combined arterial and venous injury or arterial ligation is present, or evacuation precludes serial observation; staged wound excision with planned re-inspection every 24-48 h and preservation of tissue of doubtful viability at the first operation; negative pressure wound therapy between stages; and delayed closure, grafting or flap coverage only after a clean granulating bed has been obtained.

4. Fasciotomy corrects a single mechanism and should be indicated by the marker and the injury pattern rather than applied prophylactically to every high-energy limb wound; a liberal policy has been associated with limb infection, motor dysfunction and contracture without a demonstrable reduction in amputation. Conversely, delayed or incomplete decompression remains one of the principal avoidable causes of limb loss.

5. The algorithm presented in Figure 1 formalises the transition from pathophysiological mechanism through diagnostic marker to surgical decision. It requires prospective clinical validation, in particular with respect to perfusion-imaging thresholds for the extent of excision and to the integration of severity assessment instruments into the decision between limb salvage and amputation.

Article Declarations

Perspectives for Further Research. Further research should be directed towards the large-scale clinical testing and validation of objective methods for monitoring the viability of tissues within the zone of molecular concussion, including intraoperative fluorescence angiography, tissue oximetry and laboratory markers of endothelial injury and rhabdomyolysis. Of separate interest is the prospective validation of integrated severity assessment instruments for blast-related injuries, together with the development of unified algorithms determining the optimal timing and extent of surgical debridement, the indications for fasciotomy and the criteria for staged reconstructive intervention in casualties with combat injuries of the lower extremities.

Study Limitations. The authors expressly acknowledge that the limitations of this review arise both from the predefined boundaries of its subject matter, time span and study types, and from the availability of sources. The search strategy covered PubMed (https://pubmed.ncbi.nlm.nih.gov), Scopus (https://www.scopus.com) and Google Scholar (https://scholar.google.com) for the period 2016–2026 and was restricted to English-language publications available in full text, which does not exclude the omission of relevant work published in other languages or in sources with restricted access. As the review is narrative rather than systematic, the selection of the final 35 publications reflects the expert judgement of the authors and was not accompanied by a formal assessment of the risk of bias or by quantitative synthesis of the data.

Primary data and materials. The authors expressly certify that no primary medical records (case histories, outpatient charts, examination protocols, or laboratory and instrumental findings of individual patients) and no statistical databases were used in this work. The article is a review of the published scientific literature and contains no individually identifiable patient data or images.

Research Ethics. The researchers complied with all protocols and procedures established by the Committee on the Ethics of Biomedical Research, as well as with the requirements of the health care legislation of Ukraine and the provisions of the Declaration of Helsinki (2000). As the work is a review of published literature and involved neither human participants nor primary medical documentation, separate approval by an ethics committee was not required.

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ПАТОФІЗІОЛОГІЧНІ МЕХАНІЗМИ БОЙОВИХ УШКОДЖЕНЬ НИЖНІХ КІНЦІВОК ТА ЇХ ВПЛИВ НА ВИБІР ХІРУРГІЧНОЇ ТАКТИКИ

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РЕЗЮМЕ

Вступ. Бойові ушкодження нижніх кінцівок залишаються однією з основних причин тяжкої інвалідизації військовослужбовців. Високоенергетичний характер мінно-вибухових, вогнепальних, осколкових, термічних та баротравматичних ушкоджень зумовлює складні місцеві й системні патофізіологічні реакції, від яких залежать перебіг травматичної хвороби та можливість збереження кінцівки.

Мета. Систематизувати сучасні дані щодо патофізіологічних механізмів ушкодження тканин нижніх кінцівок за різних видів бойової травми та з'ясувати їхнє значення для вибору хірургічної тактики.

Матеріали та методи. Проведено наративний огляд літератури, індексованої в PubMed/MEDLINE, Scopus та Google Scholar за період 2016–2026 рр., за ключовими словами "blast injury", "gunshot wounds", "fragment wounds", "compartment syndrome", "reperfusion injury", "endothelial dysfunction" і "combat trauma". До аналізу включали повнотекстові англомовні публікації, присвячені патогенезу бойової травми. Після відбору та вилучення дублікатів опрацьовано 45 джерел, з яких 15 увійшли до остаточного аналізу.

Результати. Незалежно від фізичного механізму ушкодження бойова травма характеризується тризональною моделлю: зоною первинного некрозу, зоною вторинного ушкодження (зоною молекулярного струсу) та ділянкою потенційно зворотних змін. Мінно-вибухова травма поєднує дію ударної хвилі, осколків і термічної енергії з масштабним руйнуванням м'язів та рабдоміолізом; вогнепальні й осколкові поранення характеризуються передачею кінетичної енергії, тимчасовою кавітацією та високим рівнем контамінації; термічна травма та баротравма супроводжуються формуванням зони стазу з підвищеною проникністю капілярів та інтерстиціальним набряком. Прогресування ушкодження в цій зоні зумовлене спільним каскадом патофізіологічних процесів, що включає ендотеліальну дисфункцію з ушкодженням глікокаліксу, феномен no-reflow, перевантаження клітин кальцієм, мітохондріальну дисфункцію, реперфузійний оксидативний стрес, системне запалення та компартмент-синдром.

Висновки. Попри відмінності у фізичних механізмах, усі види бойової травми мають спільні патогенетичні механізми розвитку вторинного некрозу, що створює підґрунтя для уніфікації підходів до моніторингу життєздатності тканин. Розуміння цих механізмів є підґрунтям для раціонального вибору хірургічної тактики, своєчасної оцінки меж життєздатності тканин та покращення результатів збереження кінцівки.

Ключові слова: мінно-вибухова травма; компартмент-синдром; некроз; бойова травма; вогнепальні поранення.

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