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Features of Acute Kidney Injury in Combat Casualties During Modern Armed Conflict

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ABSTRACT

BACKGROUND: In modern armed conflicts, wounds and injuries of various etiologies have become an inherent component of combat operations. The main research directions concerning such injuries are aimed at identifying causal relationships between the nature of the trauma, the features of its acute phase, and the development of complications, including acute kidney injury. A review of the literature revealed sufficient information on the leading pathophysiological mechanisms underlying the development of acute kidney injury. However, early diagnostic methods for this complication have not yet been developed, which prompted the conduct of this study.

AIM: To identify factors influencing the risk of acute kidney injury in combat casualties and to determine the frequency of the leading pathogenetic mechanisms in the early phase of traumatic disease.

MATERIALS AND METHODS: A prospective analysis was conducted involving 104 patients with wounds who were admitted to the 1602 Military Clinical Hospital of the Ministry of Defense of the Russian Federation (Rostov-on-Don) between January and March 2024. Upon admission to the multidisciplinary hospital, the severity of patients' conditions was assessed using the admission condition scale developed by the Department of military field surgery. Based on the assessment results, patients were divided into three categories: 1—patients with decompensated traumatic disease (score >34; 17 patients, 16.4%); 2—patients with subcompensated traumatic disease (score 25–34; 49 patients, 47.1%); 3—patients with compensated traumatic disease (score ⩽24; 38 patients, 36.5% of the total). Assessment included medical history, evaluation of vital signs, 24-hour urine output measurement, complete blood count, urinalysis, serial blood chemical analysis (three measurements), electrocardiography, and chest X-ray. Statistical processing of the experimental data was performed using SPSS Statistics 27 (IBM).

RESULTS: As a result of the study, diagnostic factors influencing the risk of this complication in the early phase of traumatic disease were identified, including hemoglobin, hematocrit, creatinine, estimated glomerular filtration rate, urea, 24-hour urine output, and potassium levels; their statistical significance was confirmed. Based on the intergroup analysis of the obtained data, no cases of acute kidney injury were recorded in the group of patients with compensated traumatic disease. The incidence of acute kidney injury was 82.3% among patients with decompensated traumatic disease and 24.5% among those with subcompensated disease. Of the 26 patients diagnosed with acute kidney injury, 12 (46.1%) presented with a non-oliguric form. The leading pathogenetic mechanisms were prerenal acute kidney injury due to hypovolemia (69.2%), renal injury associated with rhabdomyolysis (23%), and metabolic acidosis (8.3%).

CONCLUSION: The results obtained in this study indicate a high incidence of acute kidney injury (up to 25%) among patients with wounds and injuries in modern armed conflict, with prerenal mechanisms being the predominant pathogenetic variant. Due to the limited diagnostic value of standard methods, particularly in patients presenting with the non-oliguric form, it is essential to develop a comprehensive approach to the diagnosis of acute kidney injury in wounded and injured individuals. This approach should incorporate modern laboratory biomarkers and be applied during the early phase of traumatic disease.

Keywords: armed conflict; acute kidney injury; pathogenetic mechanism; prerenal acute kidney injury; combat casualty; early phase; traumatic disease.

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Особенности острого повреждения почек у раненых в современном вооруженном конфликте

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АННОТАЦИЯ

Актуальность. В условиях современного вооруженного конфликта особенности ранений и повреждений различной этиологии стали неотъемлемой частью боевых действий. Основные направления исследований в отношении подобных повреждений сводятся к установлению причинно-следственных связей между характером травмы, особенностями ее острого периода и развития осложнений, в том числе острого повреждения почек. При анализе литературных источников получен достаточный объем информации по ведущим патофизиологическим механизмам развития острого повреждения почек. Однако на данный момент не разработано методик ранней диагностики данного осложнения, чем и обусловлено проведение этого исследования.

Цель — определение факторов, влияющиех на риск развития острого повреждения почек у раненых, а также установить частоту ведущих механизмов патогенеза развития в ранний период травматической болезни.

Материалы и методы. Представлен проспективный анализ 104 пациентов с ранениями, которые были госпитализированы в ФГКУ «1602 военный клинический госпиталь» МО РФ, Ростов-на-Дону, в период с января по март 2024 г. При поступлении в многопрофильный стационар проводилась оценка тяжести состояния пациентов с использованием шкалы кафедры военно-полевой хирургии состояния при поступлении, по результатам которой были выделены три категории раненых: 1 — пациенты с декомпенсированным течением травматической болезни с суммой баллов более 34 (17 человек, 16,4%); 2 — пациенты с субкомпенсированным течением травматической болезни с суммой баллов от 25 до 34 (49 человек, 47,1%); 3 — пациенты с компенсированным течением травматической болезни с суммой баллов до 24 (38 человек, что составляет 36,5% от общего числа). Осуществлялись сбор анамнеза, оценка витальных функций пациентов с подсчетом суточного диуреза, выполнялись общеклинический анализ крови и мочи, трехкратное биохимическое исследование крови, электрокардиография, рентгенография органов грудной клетки. Статистическую обработку экспериментальных данных проводили с использованием программы SPSS Statistics 27 (IBM).

Результаты. В результате исследования определены диагностические факторы, влияющие на риск развития осложнения в ранний период травматической болезни (гемоглобин, гематокрит, креатинин, расчетная скорость клубочковой фильтрации, мочевина, величина суточного диуреза, калий) и их статистическую значимость. На основании межгруппового анализа полученных данных отмечено, что в группе пациентов с компенсированным течением травматической болезни случаев развития острого повреждения почек не зарегистрировано. В группе пациентов с декомпенсированным течением травматической болезни частота развития острого повреждения почек составила 82,3%, с субкомпенсированным течением травматической болезни — 24,5%. У 12 из 26 пациентов (46,1%) с установленным диагнозом острое повреждение почек отмечалась неолигурическая форма. Ведущими патогенетическими механизмами развития осложнения явилось преренальное острое повреждение почек, обусловленное гиповолемией (69,2%); ренальное, связанное с рабдомиолизом (23%) и метаболическим ацидозом (8,3%).

Заключение. Полученные в работе результаты указывают на высокую распространенность острого повреждения почек среди пациентов с ранениями и повреждениями в условиях современного вооруженного конфликта (до 25%), ведущим патогенетическим вариантом которого является преренальный. С учетом низкой информативности стандартных методов диагностики осложнения, особенно у пациентов с неолигурическим вариантом, требуется разработка комплексного подхода к диагностике острого повреждения почек у раненых и пострадавших с использованием современных лабораторных биомаркеров в ранний период травматической болезни.

Ключевые слова: вооруженный конфликт; острое повреждение почек; патогенетический механизм; преренальный патогенетический вариант; раненый; ранний период; травматическая болезнь.

Как цитировать

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BACKGROUND

The mortality rate of patients with severe multiple and combined trauma (polytrauma) depends on various factors, including morphology and severity of single injuries, number of affected anatomical areas, speed and quality of pre-hospital care, delivery time of the injured person to a qualified and specialized healthcare facility, patient's age, comorbidity, condition severity, and general level of healthcare organization, and can reach ⩾30%. Considering the specific features of the traumatic disease (TD) course, detection of complications at Stages I and II of the pathological process is relevant in determining the patient's prognosis and quality of life.

In modern military conflict situations, the sanitary loss structure analysis at the stage of first aid measures indicates the predominant character of missile injuries, accounting for 79.3%, and blast injuries, occurring with a frequency of 15.1%. The prevalence of severe and extremely severe injuries is 26% and 8.6%, respectively. Statistics show an increase in the severe and extremely severe injuries rate compared with other military conflicts, particularly in North Caucasus, with the total number of such injuries not exceeding 20%. More than 90% of injuries are combined and multiple injuries. Several studies showed that 5.3% of injured individuals suffer from thermal and thermomechanical injuries, mostly sustained inside attacked vehicles or combat vehicles [1]. The changes observed in the sanitary casualty structure are explained by progress in weapon development and introduction of high-energy munitions into the armory [2–4].

Scientific data reveal that the complication rate in patients with multiple and combined trauma is >70%, mainly in the TD early period. Acute kidney injury (AKI) is a life-threatening complication frequently diagnosed in injured individuals. Studies have reported that AKI likelihood depends on trauma type, wound and injury location, and the time of delivery to qualified and specialized healthcare facilities. AKI is commonly observed in abdominal mine blast wounds and combined injuries [2–6].

The major factors contributing to AKI include traumatic shock, hypovolemia, rhabdomyolysis, sepsis, metabolic acidosis (MA), and urinary tract obstruction due to trauma [4–6].

There are three main AKI pathogenetic variants: prerenal (associated with renal hypoperfusion); renal (associated with direct damage to the main compartments of the organ, i.e., intrarenal vessels, glomeruli, tubules, and interstitium); and postrenal (associated with urinary tract obstruction). In patients who are brought to the hospital after a wounding or an injury directly from the battlefield, prerenal AKI is more common, whereas renal AKI is more likely to occur as a hospital complication. The continuity disruption between medical care stages can lead to undiagnosed complications. It should be considered that the risk of AKI is increased in injured patients with pre-existing renal disease, diabetes mellitus, and arterial hypertension and in those on or already taking potentially nephrotoxic drugs [6, 7].

Prerenal kidney damage causes include:

- Hypovolemia caused by increased fluid loss (due to bleeding, burns, massive vomiting and diarrhea);
- Decreased cardiac output in heart failure, cardiac tamponade, massive pulmonary embolism;
- Intrarenal vasomodulation due to hypercalcemia, hepatorenal syndrome, abdominal compartment syndrome; and
- Systemic vasodilation due to sepsis, systemic inflammatory response syndrome, and hepatorenal syndrome [4, 6].

Acute blood and plasma loss in burns, leading to gross hemodynamic disturbance, negatively affects organ perfusion. Ischemia leads to decreased glomerular filtration, toxic substance accumulation in the blood, and water–electrolyte balance disruption. To compensate for hypovolemia, the renin–angiotensin–aldosterone system is activated; however, this may lead to additional damage. Increased renin levels contribute to increased angiotensin II production, causing renal vasoconstriction and further renal perfusion deterioration [8–10]. Notably, AKI severity depends on blood loss volume and rate. The complication risk significantly increases at a >30% blood volume loss [10, 11].

The most common causes of renal AKI include acute interstitial nephritis, pigment nephropathies, rhabdomyolysis and massive hemolysis, and acute tubular necrosis with ischemia due to shock and sepsis [6, 12].

Traumatic rhabdomyolysis (RM) is characterized by striated muscle sarcolemma damage with myocyte contents leaking into the blood. The main indicator in the pathogenesis of injury-related pigment nephropathy is myoglobin. This complication is common in patients with crush syndrome (CS) [4, 6, 13].

CS is a serious injury involving soft tissue compression. It may occur in combat conditions involving falling of building debris, use of military vehicles, barricades, and roadblocks. Myoglobin release in sarcolemma damage leads to renal tubular necrosis. In addition to its ability to penetrate the glomerular basal membrane and bind to Tamm-Horsfall protein, it damages the convoluted tubule epithelium. The urine acidic pH in the distal tubule lumen creates conditions for the formation of poorly soluble cylinder shape precipitate, which causes tubular obstruction [4, 6, 14].

Unlike in crush syndrome, muscles are damaged in compartment syndrome by the body weight of the patient who is unconscious due to poisoning or fainting [4, 13, 15–17].

In addition to trauma, RM may be caused by improper application of tourniquet for limb wounds and injuries. Noncompliance with clinical guidelines for tourniquet use is characterized by ischemic muscle damage, leading to hypermyoglobinemia and systemic disorders after its withdrawal. The myoglobinemia level correlates with tourniquet exposure duration. RM-associated AKI occurs in 10%–40% of cases [6, 18].

Postrenal causes include bladder and ureter trauma, bladder strictures and its blockage with blood clots, papillary necrosis in the renal pelvis, and ureter and renal pelvis obstruction by concretions [5–7].

In addition to the above-described pathogenesis chains in burns, MA is a critical physiologic change triggering renal dysfunction [19].

MA is manifested by an increase in the hydrogen ion blood concentration and results in a pH decrease. Burns, especially severe burns, cause complex physiologic changes, including fluid and electrolyte loss, which contributes to acidosis and AKI as a consequence of metabolic imbalance [6, 20, 21]. AKI in burn injury occurs at a frequency of up to 30%.It is classified into early (up to 72 hours after the injury) and late (4–14 days after the injury).

AKI progression depends on the total burn area. According to published data, the risk of AKI increases by 10%–15% for every 10% increase in the burn area [4, 21].

Analysis of studies provided sufficient data on AKI development mechanisms in early TD. Considering the high frequency of various etiologies of renal dysfunction, resulting in worse prognosis for wounded and injured individuals, the search for early diagnostic criteria for complications in early TD should be a priority.

This study aimed to identify factors affecting the risk of AKI in injured patients and determine the frequency of the leading pathogenesis mechanisms of AKI in early TD.

MATERIALS AND METHODS

The study examined 104 patients with wounds and injuries admitted for qualified medical care in a multidisciplinary hospital (Federal State Institution "The 1602nd Military Clinical Hospital of the Ministry of Defense of the Russian Federation, Rostov-on-Don) between January and March 2024.

At admission, the patients' condition severity was assessed using the Department of Military Field Surgery scale (Military Field Surgery Scale for assessing the severity of the condition at admission) in the reception unit. This method was used to further categorize patients into risk groups for early TD complications. Chronic somatic pathology, nephrotoxic drug use, blood component transfusion, and tourniquet application were evaluated based on medical records from previous healthcare levels. The impact of these factors on potential renal dysfunction complications were investigated. In the multidisciplinary hospital, the patients' vital functions were determined by calculating daily diuresis as the simplest and most informative renal function assessment method; complete blood count and urinalysis were performed; a three-time blood chemistry study with determination of the calculated glomerular filtration rate (GFR) using the new Chronic Kidney Disease Epidemiology (CKD-EPI) creatinine formula was conducted; and electrocardiography and chest X-ray were registered.

The patients were grouped by condition severity. The AKI time indexes were obtained and analyzed, as well as the frequency of prevalent AKI pathogenetic mechanism

In the study, the inclusion criteria were a wound or injury history of <120 hours prior to hospital admission and a nursing care facility stay of at least 72 hours. The choice of wounding time of <120 hours before hospital admission is explained by the need to cover the period of the highest risk of TD complications, which starts from 48 hours after wounding and lasts up to 3–5 days.

Study setting

This study was performed in a multidisciplinary clinical hospital (Federal State Institution, 1602nd Military Clinical Hospital of the Ministry of Defense of the Russian Federation, Rostov-on-Don).

Study duration

Patient follow-up lasted 72 hours, which corresponds to the early TD period. The rationale for the choice of follow-up duration was associated with the activation of main AKI pathogenetic mechanisms in any variant (prerenal, renal, and postrenal) within 72 hours.

Intervention

History data from available medical records, Military Field Surgery Scale scores, and investigation results were evaluated.

Main study outcome

The criteria for early diagnosis of AKI at the stages of care were defined. Moreover, the complication incidence rate was calculated, and the leading complication pathogenetic mechanisms in the wounded in TD periods I–II were determined.

Additional study outcomes

As an additional outcome of the present research, the prognostic model for early diagnosis of AKI should be considered as an outcome of this study.

Subgroup analysis

The main groups of participants were formed: wounded and injured patients with compensated, subcompensated,

and decompensated TD. Injury time and the patient's age and healthcare characteristics during the evacuation stages were assessed.

Outcomes registration

The condition of the injured was examined using the Military Field Surgery Scale, assessment of vital functions (heart rate, blood pressure, blood oxygen saturation, and daily diuresis), blood chemistry (creatinine, urea, and potassium), complete blood count, urinalysis, electrocardiogram, and chest X-ray.

The Military Field Surgery Scale comprises 12 criteria: skin color, respiratory pattern, auscultatory changes in the lungs, speech contact, pain reaction, pupillary reflex, pupil size, pulse pattern, pulse rate, systolic blood pressure, estimated blood loss, and intestinal peristalsis [5].

To estimate the diuresis in patients with wounds and injuries, daily diuresis was counted rather than the rate (mL/kg/h) in accordance with the 2012 KDIGO Practical Clinical Guidelines owing to care organization peculiarities at this stage.

Statistical analysis

Statistical analysis of experimental data was performed using SPSS Statistics 27 (IBM). Quantitative trait distribution was examined for conformity to the normal distribution law using the Shapiro–Wilk test and descriptive statistics analysis. In normal or near-normal distribution, parametric analysis methods were employed to compare the distributions of quantitative indicators in the groups (analysis of variance to compare three or more groups and Student's *t*-test to compare two groups). Results were presented as mean of the trait and standard deviation ($M \pm SD$). In cases of non-normal distribution, nonparametric criteria were utilized, and results were presented as median and interquartile range (Me [Q_1; Q_3]). The Pearson chi-squared test of independence was used to compare qualitative measures. For assessing changes in the nitrogen metabolism parameters by groups, analysis of variance with repeated measurements (general linear model) was used. The time factor with three measurements (on admission and after 24 and 48 hours) corresponded to a change in the in-group variance of the controlled parameter, and the group factor (patients with decompensated, subcompensated, and decompensated TD) corresponded to the change in the in-group variance.

RESULTS

Participants

The characteristics of the patients' wounds and injuries were determined by assessing the objective status and accompanying medical documentation. Most of the cases were mine blast injury (MBI), which were detected in 67 patients (64.4%); missile injury (MI) in 20 patients (19.2%); bullet injury (BI) in 12 patients (11.5%); CS in 3 patients (2.8%); and burns with >40% skin damage in 2 patients (1.92%) (Table 1).

Notably, analysis of the wound and injury structure depending on the main (dominant) lesion location showed that chest and abdomen injuries were the most common (Table 2). This distribution is possibly due to explosive munitions (heavy artillery systems, cluster munitions, and reconnaissance and strike drones) used by the enemy.

The Military Field Surgery Scale was utilized to assess severity of the patient's condition on admission. Assessment results revealed that the injured were grouped as follows: group 1, patients with decompensated TD (total score: >34, n=17, 16.4%); group 2, patients with subcompensated TD (total score: 24–33, n=49, 47.1%); and group 3, patients with compensated TD (total score: ⩽24, n=38, 36.5%).

Primary results

Based on the 2012 KDIGO Clinical Practice Guidelines, AKI was diagnosed in 26 wounded and injured patients (25% of all patients). In the group of decompensated TD patients, AKI was detected in 14 patients, accounting for 82.3% of all patients in this group. In analyzing the diagnostic time indicators, complications were observed in 11 patients upon admission and within the first 24 hours after admission in 3 patients. In the group of subcompensated TD patients, AKI was diagnosed in 12 patients, which is 24.5% of all patients in this group. Notably, this complication was diagnosed in 3 patients upon admission, in 5 patients after 24 hours, and in 4 patients after 48 hours. In the group of compensated TD patients, no evidence of AKI was found (Table 3).

At the initial stage, the patients were assessed for age, admission time after injury (trauma), and history of chronic pathology. One-factor analysis of variance was used. No differences were noted between the groups regarding age and wounding time. According to medical documentation, 27 patients had a history of chronic pathology. When comparing the groups with decompensated and subcompensated TD ($p_{1,2} \leqslant 0.05$) and subcompensated and compensated TD ($p_{2,3} \leqslant 0.05$), the differences in the trait frequency were significant. When comparing the groups with decompensated and compensated TD course, the history of chronic pathology was not statistically significant ($p_{1,3} \geqslant 0.05$) (Table 4).

In evaluating interventions during previous healthcare stages, differences contributing to AKI frequency should be noted. Table 5 shows that in the groups with decompensated and subcompensated TD, tourniquet was applied in 9 and 12 patients, accounting for 52.9% and 24.5% of patients in the groups, respectively, which is statistically significant ($p_{1,2} \leqslant 0.05$). When comparing the

Table 1. Group distribution of patients by type of wounds and injuries
Таблица 1. Групповое распределение пациентов по типу ранений и повреждений

Frequency of occurrence	Type of wound/injury					Total
	MBI	MI	BI	CS	Burn disease	
Number of cases	67	20	12	3	2	104
Percentage of patients, %	64.4	19.2	11.6	2.9	1.9	100

Table 2. Frequency of wounds and injuries by major clinical groups (based on the localization of the primary dominant injury)
Таблица 2. Частота ранений и повреждений по основным клиническим группам (в зависимости от локализации основного доминирующего поражения)

Location of the dominant or single lesion/ the leading injury mechanism	Number of patients	Percentage of patients, %
Head	20	19.2
Chest	25	24.1
Abdomen	25	24.1
Limbs	18	17.3
Soft tissues	7	6.7
Spine with spinal cord injury	4	3.8
CS	3	2.9
Burn disease	2	1.9
Total	104	100

subcompensated and compensated TD groups, this indicator was also significant ($p_{2, 3} \leqslant 0.05$). No significant differences were observed in tourniquet application between the compensated and decompensated TD groups ($p_{1, 3} \geqslant 0.05$). Intergroup analysis revealed significant differences in blood substitute solutions administration at the previous evacuation stage in all groups ($p \leqslant 0.05$). No significant differences were found between the groups regarding administration of nephrotoxic drugs ($p = 0.640$) (Table 5).

The mean hemoglobin value on admission was 114.4±14.8 g/L in the compensated TD patients, 99.7±17.8 g/L in the subcompensated group, and 81.2±7.9 g/L in the decompensated group (Table 6). The average hematocrit value was 38.5±3.1% in the compensated group, 35.2±3.4% in the subcompensated group, and 32.1 ± 1.7% in the decompensated group, indicating severity of injuries. No significant differences were observed in the results of the urinalysis, namely, specific gravity, between the groups. Focus was directed to hyperkalemia that co-occur with AKI in injuries. Based on biochemical blood test results, the highest values of this index were noted in the decompensated patients group. In intergroup analysis, significant differences were observed in potassium levels when comparing the decompensated and subcompensated TD groups ($p_{1, 2} \leqslant 0.001$) and the decompensated and compensated TD groups ($p_{1, 3} \leqslant 0.001$). No significant differences were found between the subcompensated and compensated TD groups ($p_{2, 3} = 0.991$) (Table 6).

Analysis of variance with repeated measurements (general linear model) was utilized to examine the changes over time in creatinine, GFR, urea, and daily diuresis by groups. The time factor with three measurements (on admission and after 24 and 48 hours) corresponded to the change in the in-group variance of the controlled index, and the group factor (patients with compensated, subcompensated, and decompensated TD) corresponded to the change in intergroup variance. The statistical hypotheses were tested in analysis of variance with repeated measures:

1. The mean values of the dependent indicators for significance at all three time points are the same regardless of the group.

2. The mean values of the dependent indicators are the same between groups at each time point.

3. The mean values of dependent indicators do not differ by time, indicating no interaction between time and the group factor (Table 7).

Table 7 shows an increase in creatinine levels in all three groups ($p_1 < 0.001$); this change is statistically significant. Compared with the established reference values (76–110 μmol/L), significant overrange values were observed in the decompensated TD group. An insignificant outlier compared with reference values was noted in the subcompensated TD group 24 hours after admission. Moreover, other differences were found between the groups ($p_2 < 0.001$). The creatinine levels in the

Table 3. Comparative analysis of the incidence of acute kidney injury depending on the course of traumatic disease, *n*

Таблица 3. Сравнительная характеристика частоты развития острого повреждения почек в зависимости от течения ТБ, *n*

AKI, h	Groups of patients		
	with decompensated TD, *n*=17	With subcompensated TD, *n*=49	With compensated TD, *n*=38
On admission	11	3	–
24	3	5	–
48	–	4	–
Total	14	12	–

Таблица 4. Сравнительная характеристика входящего потока раненых и пострадавших по возрасту, времени от получения ранения (травмы), наличия хронической патологии в анамнезе, *M*±SD

Table 4. Comparative characteristics of wounded and injured individuals by age, time from injury (trauma), and history of chronic conditions, *M*±SD

Indicator, measurement units	Patients with decompensated TD, *n*=17	Patients with subcompensated TD, *n*=49	Patients with compensated TD, *n*=38	*p*-value
Time since wounding, h	28.2±8.4	33.3±14.1	31.6±14.1	p=0.402 $p_{1,2} \geqslant 0.05$ $p_{1,3} \geqslant 0.05$ $p_{2,3} \geqslant 0.05$
Age, years	37.5±4.4	37.4±5.2	36.7±7.8	p=0.827 $p_{1,2} \geqslant 0.05$ $p_{1,3} \geqslant 0.05$ $p_{2,3} \geqslant 0.05$
Chronic pathology in history absolute, %	1 (5.9)	20 (40.8)	6 (15.8)	p = 0.004 $p_{1,2} \leqslant 0.05$ $p_{1,3} \geqslant 0.05$ $p_{2,3} \leqslant 0.05$

Table 5. Characteristics of interventions at earlier stages of care and relevant history data, abs., %

Таблица 5. Характеристика мероприятий, проведенных на предыдущих этапах оказания помощи, и анамнестических данных, абс., %

Parameter	Patients with decompensated TD, *n*=17	Patients with subcompensated TD, *n*=49	Patients with compensated TD, *n*=38	*p*-value
Tourniquet	9 (52.9)	12 (24.5)	2(55.3)	p=0.008 $p_{1,2} \leqslant 0.05$ $p_{1,3} \geqslant 0.05$ $p_{2,3} \leqslant 0.05$
Injection of blood substitute solutions	15 (88.2)	19 (38.8)	4 (10.5)	$p \leqslant 0.05$ $p_{1,2} \leqslant 0.05$ $p_{1,3} \leqslant 0.05$ $p_{2,3} \leqslant 0.05$
Injection of nephrotoxic drugs	17 (100)	47 (95.9)	36 (94.7)	p=0.640 $p_{1,2} \geqslant 0.05$ $p_{1,3} \geqslant 0.05$ $p_{2,3} \geqslant 0.05$

decompensated TD group were higher than those in other groups upon admission and after 24 and 48 hours; the difference between the compensated and subcompensated groups was insignificant as far as confidence intervals overlapped. Notably, the level of creatinine increase in the decompensated TD group after 24 hours was more dynamic compared with that in other groups (p_3 <0.001).

GFR levels decreased in all three groups (p_1 <0.001). Considering a correlation between creatinine and GFR (calculated using the CKD-EPI formula), overlapping confidence intervals between the compensated and subcompensated TD groups were observed. This results in low statistical significance within both groups. Similar to creatinine levels, the low initial GFR level in

Table 6. Comparative analysis of clinical examination findings and the Military Field Surgery Admission Scale, *M*±SD
Таблица 6. Сравнительная характеристика показателей, полученных при обследовании пациентов, и оценочной шкалы ВПХ-СП, *M*±SD

Parameter	Patients with decompensated TD, *n*=17	Patients with subcompensated TD, *n*=49	Patients with compensated TD, *n*=38	*p*-value
Military Field Surgery Scale, scores	36.7±1.3	30.0±3.4	21.7±2.6	$p \leqslant 0.001$ $p_{1,2} \leqslant 0.001$ $p_{1,3} \leqslant 0.001$ $p_{2,3} \leqslant 0.001$
Hemoglobin, g/L	81.2±7.9	99.7±17.8	114.4±14.8	$p \leqslant 0.001$ $p_{1,2} \leqslant 0.001$ $p_{1,3} \leqslant 0.001$ $p_{2,3} \leqslant 0.001$
Hematocrit, %	32.1±1.7	35.2±3.4	38.5±3.1	$p \leqslant 0.001$ $p_{1,2}=0.002$ $p_{1,3} \leqslant 0.001$ $p_{2,3} \leqslant 0.001$
Urine specific gravity	1.02±0.01	1.02±0.01	1.02±0.00	$p=0.029$ $p_{1,2}=0.772$ $p_{1,3}=0.070$ $p_{2,3}=0.092$
Potassium, mmol/L	4.9±1.2	4.0±0.4	3.9±0.2	$p \leqslant 0.001$ $p_{1,2} \leqslant 0.001$ $p_{1,3} \leqslant 0.001$ $p_{2,3}=0.991$

the decompensated TD group progressively decreased throughout the examination (p_3=0.014).

In estimating the average urea level in the biochemical blood test and daily diuresis level in the decompensated TD group, high indices at the first measurement and their progressive increase (p_3 <0.001) are expected. No significant difference was observed between the compensated and subcompensated TD groups.

Notably, 12 of 26 patients (46.1%) with AKI had a non-oliguric form. This indicates the low informativeness of the daily diuresis parameter in isolation at the initial stage of TD and the need for clinical observation over time in injured patients at high risk for complications.

DISCUSSION

Summary of Primary Results

The main factors that can be used for early AKI diagnosis are routine blood serum creatinine and urine output; Military Field Surgery Scale score; hemoglobin content; and hematocrit, urea, and potassium levels in the blood serum.

Early verification of the leading AKI pathogenetic mechanism at the initial healthcare stages allows for determining the patient evacuation destination and choosing the optimal treatment strategy for complication management.

The main AKI pathogenetic mechanisms were hypovolemia (69.2% of total AKI cases), rhabdomyolysis (23% of total AKI cases), and MA (8.3% of total AKI cases). Hypovolemia is one of the most common mechanisms leading to prerenal AKI. This mechanism is prevalent in combined trauma, accompanied by external blood loss and internal bleeding in the cavity, without proper circulating blood volume replenishment at the healthcare stages. Renal AKI due to rhabdomyolysis and MA was significantly less common. This mechanism was often seen in CS and mine blast injuries of the extensive muscle layers. MA was prevalent in 8.3% of cases. This mechanism was most relevant for burn injuries of ⩾40% of the skin.

CONCLUSION

The main diagnostic factors identified in this study can be used in developing a prognostic model for AKI early diagnosis when patients are admitted to a qualified healthcare facility.

Various combined, multiple, and penetrating injuries and severe limb injuries require a comprehensive treatment approach. It implies the development and practical implementation of advanced AKI verification techniques based on laboratory biomarkers to avoid misinterpretation of standard biochemical and instrumental studies during the diagnostic window (72 hours).

The leading pathophysiological mechanism of AKI was prerenal AKI characterized by hypovolemia (69.2% of the total AKI cases). Renal AKI due to rhabdomyolysis was detected in 23% of patients with AKI, and MA was detected in 8.3% of patient with AKI.

Table 7. Renal function parameters over time and across patient groups, *Me* [Q_1; Q_3]

Таблица 7. Показатели функционального состояния почек в зависимости от времени и принадлежности к группам, *Me* [Q_1; Q_3]

Parameter		Patients with decompensated TD, *n*=17	Patients with subcompensated TD, *n*=49	Patients with compensated TD, *n*=38	*p*-value
Creatinine, μmol/L	0	226 [126; 416]	96 [90; 108]	90 [86; 98]	p_1 <0.001
	24 h	273 [174; 588]	110 [97; 129]	94 [94; 99]	p_2 <0.001
	48 h	229 [203; 522]	118 [104; 167]	105.5 [98; 110]	p_3 <0.001
GFR, mL/min/1.73 м^2	0	31.4 [15.5; 60.2]	86.7 [73.2; 92.3]	91.2 [86.7; 99.8]	p_1 <0.001
	24 h	24 [9.7; 41.9]	72 [59.0; 86.1]	86.8 [79.5; 92.3]	p_2 <0.001
	48 h	22.6 [11.8; 34.8]	67.1 [47.1; 82.1]	77.4 [73.2; 87.6]	p_3=0.014
Urea, mmol/L	0	8.4 [6.9; 10.8]	6.2 [5.7; 6.9]	5.4 [4.8; 6.2]	p_1 <0.001
	24 h	12 [8; 14.2]	6.4 [5.8; 7.1]	5.7 [5.2; 6.2]	p_2 <0.001
	48 h	11.3 [8; 18.7]	6.8 [5.6; 7.3]	5.6 [5.2; 6.0]	p_3 <0.001
Diuresis, L	0	1 [0.7; 1.4]	1.6 [1.4; 1.7]	1.8 [1.6; 2.3]	p_1=0.167
	24 h	0.7 [0.4; 1.3]	1.5 [1.3; 1.8]	1.6 [1.4; 2.3]	p_2=0.196
	48 h	0.7 [0.5; 1.2]	1.4 [1.1; 1.5]	1.6 [1.4; 1.8]	p_3 <0.001

Note. p_1, value for hypothesis 1; p_2, value for hypothesis 2; p_3, value for hypothesis 3

Примечание. p_1 — значение для гипотезы 1; p_2 — значение для гипотезы 2; p_3 — значение для гипотезы 3.

ADDITIONAL INFO

Author contribution. A.V. Yazenok, writing the text and editing the article; A.A. Ivanov, writing the text and editing the article, literature review, mathematical analysis of the obtained results; P.V. Agafonov, writing the text and editing the article; M.V. Zakharov, writing the text and editing the article; A.V. Marukhov, collection and analysis of literature sources; A.V. Popov, literature review, editing the article; L.A. Kudryavtseva, collection and analysis of literature sources. All authors confirm that their authorship meets the international ICMJE (all authors have made a significant contribution to the development of the concept, research and preparation of the article, read and approved the final version before publication).

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