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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform_requirements.html
В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or compu-ter-printed on a single side of standard typing paper, with the left margin of **3** centimeters width, and **1.5** spacing between the lines, typeface - **Times New Roman** (**Cyrillic**), print size - **12** (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf
In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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SEVERE UPPER EXTREMITY CRUSH SYNDROME IN A NON-DISASTER SETTING: A CASE REPORT OF SUCCESSFUL MULTIMODAL MANAGEMENT WITH COMPLETE RENAL RECOVERY

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Abstract.

Background: Crush syndrome is a severe systemic condition caused by prolonged compression of skeletal muscles, leading to ischemia–reperfusion injury and traumatic rhabdomyolysis. It is most commonly associated with natural disasters and predominantly affects the lower extremities, whereas isolated upper extremity involvement in non-disaster settings is rare.

Objective: To present a rare case of severe crush syndrome of the upper extremity in a non-disaster setting and to evaluate the effectiveness of early multimodal treatment.

Materials and Methods: A 28-year-old male developed crush syndrome after 6–8 hours of prolonged compression of the left upper extremity following loss of consciousness. Clinical, laboratory, and instrumental data were analyzed. The patient underwent early fasciotomy, intensive care management, therapeutic plasma exchange (4 sessions), and renal replacement therapy (5 sessions of hemodialysis).

Results: The patient developed severe rhabdomyolysis (creatine kinase up to 93,994 U/L), acute kidney injury (creatinine up to 740 μmol/L), hyperkalemia, and multi-organ dysfunction. Early fasciotomy revealed initially non-viable muscle tissue with subsequent partial recovery of viability. Combined treatment resulted in significant clinical improvement, restoration of diuresis (up to 4800 mL/day), and complete recovery of renal function.

Conclusion: Severe upper extremity crush syndrome in non-disaster conditions can have a favorable outcome when managed with early and aggressive multimodal therapy. Early surgical decompression combined with therapeutic plasma exchange and hemodialysis may significantly improve prognosis and reduce mortality and long-term disability.

Key words. Crush syndrome, rhabdomyolysis, upper extremity, fasciotomy, plasmapheresis, acute kidney injury.

Introduction.

Crush Injury (CI) is defined as ischemia, edema, and rhabdomyolysis (muscle breakdown) of muscle tissue resulting from prolonged and sustained compression of the extremities or other parts of the body [1-4]. Such injuries are frequently observed during natural disasters (earthquakes, landslides), industrial accidents (mining disasters, explosions), as well as man-made events — terrorist attacks and local armed conflicts [3,5-7].

Severe crush injury can rapidly progress into a life-threatening systemic condition known as Crush Syndrome (CS), which is characterized by traumatic rhabdomyolysis and endogenous intoxication [1-4,7]. According to recent meta-analyses and comprehensive reviews, the incidence of crush syndrome following earthquakes ranges from 2% to 20% among casualties (depending on the severity of the collapsed structures, duration of compression, and the speed of rescue operations) [3,5]. Mortality rates with timely treatment range from 4.6% to 32%; however, in untreated or delayed cases, mortality can reach up to 40% [3-4,]. Crush syndrome remains the second leading cause of death in earthquake victims after direct trauma [3,5].

The pathogenesis of crush syndrome is based on traumatic rhabdomyolysis caused by prolonged ischemia of muscle tissue. Disruption of the continuity of rhabdomyocyte membranes leads to cell lysis, massive release of intracellular components (myoglobin, potassium, phosphate, and other toxic metabolites) into the systemic circulation, and subsequent endogenous intoxication [1-2,4,8-10]. This process results in electrolyte disturbances (hyperkalemia, hyperphosphatemia, hypocalcemia), myoglobinuria, and ischemia–reperfusion injury [9-10,3- 4].

In the acute phase, patients typically develop hypovolemia, life-threatening arrhythmias, and acute kidney injury (AKI) [1,5]. Despite aggressive fluid resuscitation and renal replacement therapy, patients with crush syndrome frequently develop systemic inflammatory response syndrome (SIRS) and may progress to the often-fatal multiple organ dysfunction syndrome (MODS) [1,3-5].

Crush syndrome was first described in 1941 by British physicians Eric Bywaters and D. Beall in victims of the London Blitz during the Second World War [11]. However, research on this condition remains limited to this day due to ethical and safety constraints that prohibit large-scale human experimentation [3,8,12]. As a result, despite significant efforts by the medical community, knowledge regarding the pathophysiology and clinical management of crush syndrome is still incomplete [3-4].

Currently, there is a scientific consensus that the pathophysiological mechanisms of crush injuries primarily involve two key components: ischemia–reperfusion injury and ischemia–reperfusion-induced rhabdomyolysis [3-4,8-10].

Clinically, crush syndrome is characterized by a clear traumatic history and visible signs of compression on the affected limbs. Myoglobinuria typically develops rapidly after decompression. Laboratory tests (creatine kinase (CK), serum potassium level, and myoglobin (Mb)) play a crucial role in diagnosis and severity assessment [2,13]. Nevertheless, standardized clinical criteria for the objective assessment of the severity or prognosis of crush syndrome are still lacking.

Case Presentation.

A 28-year-old male contract soldier from Military Unit N (born 02.02.1996) was urgently hospitalized on May 30, 2024,

in the Resuscitation Department of the Central Clinical Military Hospital. He was transferred by ambulance from Ijevan Medical Center in a state of inhibited consciousness.

The patient had no significant past medical history, was not taking any regular medications, and had no known allergies.

On admission, the patient had difficulty establishing contact, answered questions with delay but according to the essence, and did not remember some things. According to the history, the previous evening he had experienced prolonged loss of consciousness and had remained lying on his left side for approximately 6–8 hours, compressing the left upper extremity.

Additional diagnostic evaluation was performed to determine the etiology of the patient's prolonged loss of consciousness, which lasted 6 to 8 hours. Toxicological screening revealed a high serum ethanol level with no evidence of other intoxicants or illicit substances. Electroencephalography (EEG) and comprehensive neurological assessment showed no epileptic activity or focal neurological deficits. Cardiovascular evaluation, including continuous electrocardiographic monitoring, did not demonstrate any clinically significant arrhythmias or acute cardiac abnormalities. The patient had no prior history of neurological, psychiatric, or cardiovascular disease.

Although the cause of loss of consciousness was initially classified as "unknown etiology," after thorough investigation, exogenous alcohol intoxication leading to prolonged immobilization was determined to be the most probable precipitating factor for the development of crush syndrome of the left upper extremity, severe rhabdomyolysis, compartment syndrome, and subsequent acute kidney injury.

Physical examination:

The external coverings were pale. Respiration was spontaneous, respiratory rate 22/min, SpO_2 95%. On the right lung vesicular breathing was heard, on the left – weakened vesicular breathing. Heart sounds were muffled, pulse 100 bpm, regular, soft, of moderate filling. Blood pressure 90/60 mmHg. The abdomen was soft and painless, liver and spleen not enlarged. Diuresis was adequate but meat-wash-like in appearance.

The left upper extremity was markedly swollen in the shoulder joint and arm region, with pastosity at the wrist level, skin darkening and cyanotic tint.

Preliminary diagnosis:

Loss of consciousness of unknown etiology, prolonged crush syndrome of the left upper extremity, polyorgan failure.

Investigations:

Doppler examination of the left upper extremity showed that the main and superficial veins were patent, blood flow was магистральный but slowed, no thrombi were present. There was pronounced edema of the subcutaneous tissue and muscular system.

Chest X-ray showed no focal infiltrative changes. Abdominal ultrasound revealed a small amount of free fluid around both kidneys. Right kidney 11.8×6.2×7.4 cm, parenchyma 2.3 cm, calyceal width 0.8–1.1 cm. Left kidney 12×6.2×7.8 cm, parenchyma 2.3–2.4 cm, calyceal width 0.8–0.9 cm. Corticomedullary differentiation was preserved. Parenchyma was diffusely changed on both sides. No urine outflow was recorded from either side at the time of examination. There was 50–60 ml of urine in the bladder around the urinary catheter.

Echocardiography showed no myocardial hypertrophy or chamber dilatation. Interventricular septum 10 mm. Diffuse decrease in left ventricular contractility, ejection fraction 43–35%. Right ventricular function normal (TAPSE 17 mm). No pericardial fluid. Trivial TR and MR.

ECG showed sinus rhythm 93 bpm with mild ST-segment elevation in V1–V3 leads.

Non-contrast CT of the head and chest revealed hypoventilation-hyperperfusion foci in the lungs and infarct-pneumonia in the left lingular segments.

Laboratory findings:

Tables 1-3 and Figure 1.

Clinical diagnosis:

Exogenous intoxication of unknown origin with loss of consciousness; prolonged crush syndrome of the left upper extremity with rhabdomyolysis; polyorgan failure (acute kidney injury in oliguric stage, subclinical hepatitis, myocarditis, encephalopathy); compartment syndrome of the left upper extremity, arm and shoulder girdle.

Treatment and clinical course:

On May 30, 2024, under general endotracheal anesthesia, fasciotomy of the shoulder girdle, arm and forearm was performed. Approximately 0.8 L of serous-hemorrhagic fluid was obtained. At the beginning of the operation the biceps and triceps muscles were non-viable with a "cooked meat" appearance. Reflex contractions were absent during electrocoagulation. At the end of the operation viability was assessed as satisfactory and reflexes were restored. Wound sanitation and drainage were performed.

Along with drug therapy (detoxification, albumin 20% 100 ml IV, furosemide, ceftriaxone, pantoprazole, heparin 20,000 IU, actrapid, glucose, venoruton, heptal, tramadol) four sessions of therapeutic plasma exchange were performed:

- May 30, 2024 – 760 ml plasma removed, replaced with 3 packs of fresh frozen plasma.
- May 31, 2024 – 900 ml removed, replaced with 4 packs.
- June 1, 2024 – 1,820 ml removed (2 sessions), replaced with 7 packs.
- June 2, 2024 – 910 ml removed, replaced with 4 packs.
- On June 3, 2024, due to progressive acute kidney injury, the patient was transferred to the specialized nephrology department. Under ultrasound guidance, a dialysis catheter was inserted into the right internal jugular vein using the Seldinger technique, and five sessions of hemodialysis were performed. Positive dynamics were observed with restoration of diuresis (1,500–2,300–4,000–4,800 ml).

Serial laboratory monitoring demonstrated a marked and progressive decline in rhabdomyolysis markers following therapeutic plasma exchange (TPE) and hemodialysis. On admission, creatine kinase (CK) was extremely elevated at 93,994 U/L and myoglobin level reached 1,575.3 ng/mL. After sequential TPE sessions, CK levels progressively decreased to 7,927 U/L, 4,707 U/L, 807 U/L, and finally 479 U/L. Myoglobin levels showed a parallel decline from 1,575.3 ng/mL to 936.5 ng/

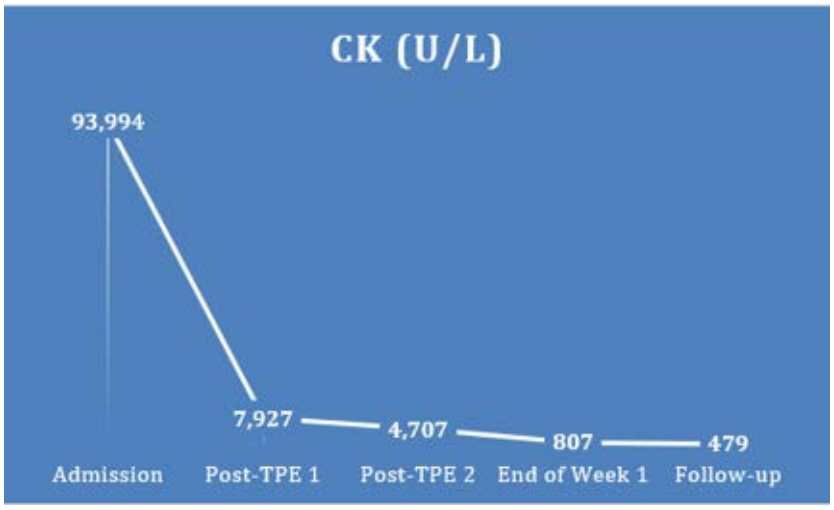

Figure 1. *Serial decline in creatine kinase (CK) levels following therapeutic plasma exchange and hemodialysis.*

Table 1. *Urinalysis Results During Hospitalization.*

Parameter	**Results**			**Normal Reference Range**	**Unit of Measurement**
	Admission	**Day 2**	**Week 1**	**End of Treatment**	
Specific Gravity	1020	1005	1010	1.015-1.025	1020
PH	5.0	7.0	7.0	5-7	6
Protein	5.9	0.75	0.75	<0.10	0.25g/l
Glucose	3.0	0	0	<0.84	0 mmol/L
RBC	250	250	250	-	0 /hpf

Table 2. *Complete Blood Count (CBC) Results During Hospitalization.*

Parameter	**Result**			**Reference Range**	**Unit of Measurement**
	Admission	**Week 1**	**End of Treatment**		
WBC	25.9	18.5	12.1	3.5-11.4 3.8-11.2	$x10^3$/ µL
LYM%	5.8	8.8	11.2	21.3-40.3 13.4-47.4	%
NEUT%	90.5	87.4	76.8	47.1-72.5 42.1-78.4	%
LYM#	1.5	1.6	1.4	1.2-3.3 0.9-3.6	$x10^3$/ µL
NEUT#	23.4	16.2	9.2	1.5-7.6 1.7-7.4	$x10^3$/ µL
RBC	5.4	4.38	3.08	3.85-5.07 4.24-5.72	$x10^3$/ µL
HGB	17.9	14.0	9,70	11.6-14.4 13.1-16.7	g/dL
HCT	51.1	50.3	28.3	32.7-45.0 39.8-52.0	%
MCV	94.3	93.4	91.9	84.7-100 86.6-97.9	fL
MCH	33.0	32.1	31.5	26.5-31.9 28.2-31.7	pg
MCHC	35.0	34.4	34.3	30.0-33.3 31.2-33.8	g/dL
PLT	316	260	130	154-410 138-350	$x10^3$/ µL
RDW-SD	45.8	42.8	42.6	41.0-49.8 38.9-49.2	fL
RDW-CV	13.5	12.6	13.1	11.4-14.7 11.5-13.8	%
ESR	140	130	100	5.0-15.0 2.0-10.0 30	17 mm/h

Table 3. *Selected Blood Biochemistry Parameters During Hospitalization.*

Parameter	**Result**								**Unit of Measurement**
	Admission	**Day 2**	**Week 1**	**End of Treatment**					
Glucose	10.9	8.67	6.3	4.5					3.88-6.38 mmol/L
Total Protein	70.5	51.8	46.7	45.0					64-85 g/L
Albumin	34.9	30.1	29.5	29.8					34-48g/L
Total Bilirubin	7.5	6.2	5.4	-					17 µmol/L
Urea	15.02	24.9	25.3	31.8	21	14	8	7	1.7-8.3 mmol/L
Creatinine (M/F)	242	412	490	740	539	276	117		62-106/44-80 µmol/L
AST (Aspartate Aminotransferase) (M/F)	535	559.1	228	172	116	19			up to 37/31 U/L
ALT (Alanine Aminotransferase) (M/F)	161	180	122	143	12.6				up to 42/32U/L
CRP (C-Reactive Protein)	112	75.5	37	23.1	18.2	7.5			<5 mg/L
Potassium (K^+	6.41	6.48	4.1						3.5-5.3 mmol/L
Sodium (Na^+)	128	128.6	138						135-148 mmol/L
Ionized Calcium	0.969	0.686	1.1						1.13-1.32 mmol/L
Prothrombin Index	90.1	87.2	90.1	82	109.4				73-111%
Prothrombin Time	11.0	11.2	10.4	15.2	9.9				9.9-11.8"
INR	1.06	1.12	1.06	1.00	1.14	0.95			1-2
APTT (Activated Partial Thromboplastin Time)	22.3	45.9	47.5	28.9	35.7	22.4			23.2-34.7"
Fibrinogen (Factor I)	3.7	4.5	2.5	4.5	2.55				1.8-3.5g/L
Creatine Kinase (CK)	93994	7927	4707	807	479				99-336U/L
Creatine Kinase MB (CK-MB)	1229.4	33							7-25U/L
Myoglobin	1575.3	936.5	478.7	252.3	79.4				23-72ng/mL
Procalcitonin									
Troponin	405	365	180	32.0	10.0				40 ng/mL
D-Dimer	0.71	2.30	1.80	0.30					0.50 ng/mL
eGFR (Estimated Glomerular Filtration Rate)	8	11	25	59					69 mL/min/1.73 m²

mL, 478.7 ng/mL, 252.3 ng/mL, and 79.4 ng/mL, respectively. This biochemical improvement coincided with restoration of diuresis, improvement of renal function, and overall clinical stabilization.

From August 16 to September 2, 2024, the patient received physiotherapy at Alfa-Beta Medical Center for mixed-type contractures of the left elbow and wrist joints and postoperative median nerve neuropathy. Pain syndrome decreased and range of motion in the left elbow joint improved.

Outcome. Positive clinical and laboratory dynamics were observed with restoration of renal function.

Discussion.

The presented clinical case describes severe prolonged crush syndrome (CS) in a 28-year-old male, which developed due to 6–8 hours of sustained compression of the left upper extremity following loss of consciousness while lying on his side. This case is notable in several aspects: it is unrelated to mass disasters, occurred solely in the upper limb (which accounts for only ~10% of CS cases, while lower limbs comprise 74%), and was accompanied by severe rhabdomyolysis (creatine kinase up to 93,994 U/L), acute kidney injury (creatinine up to 740 µmol/L), hyperkalemia, and multi-organ dysfunction, yet resulted in complete renal function recovery thanks to aggressive multimodal treatment [3-6].

Compared with the literature, the patient's clinical picture fully corresponds to the classic pathophysiology of CS, based on ischemia-reperfusion injury with rhabdomyocyte lysis and massive release of intracellular toxins into the circulation [14-16,22-25]. Laboratory findings (CK up to 93,994 U/L, myoglobin 1,575.3 ng/mL) confirm severe rhabdomyolysis, a clear marker of CS severity, and align with contemporary reviews [3,6].

For comparison, in the 2023 Türkiye earthquake, the mortality rate among 128 patients with CS was 4.6% (Onan et al., 2024), rising to 14.2% in those with acute kidney injury [15,]. Our patient's survival and full renal recovery (diuresis restored to 4,800 mL/day) demonstrate that timely intervention can significantly reduce mortality to the 4.6–15% range reported in UpToDate and Akrivos et al. (2025) [3,6].

The treatment approach fully complies with current guidelines and has important specific features. Early fasciotomy (performed on the first day of hospitalization) was carried out for compartment syndrome and resulted in muscle viability restoration without amputation. Data from the 2023 Türkiye earthquake confirm that fasciotomy performed <24 hours prevent amputation and reduces the volume of muscle excision, whereas delayed fasciotomy (>48 hours) is associated with a significantly higher amputation risk (Demir et al., 2025) [17].

Particular attention should be paid to the use of four sessions of therapeutic plasma exchange (TPE) as a detoxification measure. The literature shows that TPE is safe and effective for myoglobin clearance in severe rhabdomyolysis when standard therapy is insufficient (Boparai et al., 2023) [18].

The decision to initiate early therapeutic plasma exchange (TPE) was based on several high-risk clinical and laboratory findings. The patient presented with extremely elevated creatine kinase levels (CK 93,994 U/L), exceeding the threshold commonly associated with severe rhabdomyolysis, together with markedly increased myoglobin levels (1,575.3 ng/mL),

indicating a high risk of myoglobin-induced acute kidney injury. In addition, progressive oligoanuria, worsening renal dysfunction despite aggressive fluid resuscitation, severe crush syndrome-associated muscle injury, hyperkalemia, and the risk of systemic complications supported the need for aggressive multimodal treatment. TPE was therefore initiated early as an adjunctive detoxification strategy aimed at accelerating the clearance of myoglobin and other intracellular toxic metabolites in combination with hemodialysis and standard supportive therapy.

In our case, serial laboratory monitoring demonstrated a rapid and marked decline in both CK and myoglobin levels following sequential TPE sessions and hemodialysis. CK decreased from 93,994 U/L on admission to 479 U/L, while myoglobin declined from 1,575.3 ng/mL to 79.4 ng/mL. These findings provide objective evidence supporting the potential role of TPE in accelerating clearance of myoglobin and other intracellular muscle breakdown products in severe crush syndrome-associated rhabdomyolysis [4,19].

The favorable outcome (renal function recovery and improvement of neuropathy and contractures with physiotherapy) underscores the importance of timely diagnosis and a multidisciplinary approach. This case adds to the limited literature on non-traumatic upper-extremity CS and demonstrates that even with extreme laboratory changes (CK >90,000 U/L), aggressive treatment can prevent death and permanent disability [4,20].

Limitations.

This is a single case report without standardized protocols for therapeutic plasma exchange in crush syndrome. Although serial CK and myoglobin measurements demonstrated significant biochemical improvement following TPE, larger studies are required to objectively evaluate the efficacy and optimal indications for TPE in severe rhabdomyolysis-associated crush syndrome. Clinical photographs and Doppler ultrasonography images were not available for publication due to patient confidentiality considerations and institutional limitations.

In conclusion, the presented case confirms the pathophysiological mechanisms of CS and shows that timely, individualized treatment (early fasciotomy + TPE + hemodialysis) can dramatically improve prognosis even in severe cases. This experience is valuable for Armenian and regional medical institutions where similar injuries may occur not only in disasters but also in everyday life.

Conclusion.

The presented clinical case clearly demonstrates that even severe prolonged crush syndrome (CS), developed under non-traumatic conditions (6–8 hours of sustained compression of the upper extremity following loss of consciousness), can have a favorable outcome with timely and aggressive multimodal treatment, avoiding death or permanent severe disability.

In our patient, extreme rhabdomyolysis (creatine kinase up to 93,994 U/L), acute kidney injury in the oligoanuric phase (creatinine up to 740 μmol/L), and multi-organ dysfunction were successfully managed through early fasciotomy, four sessions of therapeutic plasma exchange (TPE), hemodialysis, and intensive pharmacotherapy. This combined approach led to complete restoration of renal function and significant clinical improvement.

In comparison with the literature, this case confirms that early fasciotomy (<24 hours) substantially reduces the risk of amputation and the volume of muscle excision, aligning with conclusions drawn from the 2023 Türkiye earthquake experience. Although the role of therapeutic plasma exchange in severe rhabdomyolysis remains controversial and lacks support from large randomized trials, in our case it was applied safely and contributed to rapid clinical dynamics, particularly in non-traumatic “found-down” type CS.

This case emphasizes that the prognosis of CS depends not only on the severity of injury and laboratory parameters, but primarily on the speed of diagnosis, a multidisciplinary approach, and individualized intensive therapy. Such non-disaster-related cases, although rarely reported in the literature, hold significant practical value for Armenian and regional healthcare systems, where similar conditions may occur in everyday life (loss of consciousness, prolonged immobility).

Future research should focus on developing standardized indications for TPE and early surgical intervention in non-traumatic CS, which will help improve outcomes for such patients in the future.

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АННОТАЦИЯ

Введение: Синдром длительного сдавления (краш-синдром) представляет собой тяжёлое системное состояние, возникающее в результате продолжительного сдавления скелетных мышц, что приводит к ишемически-реперфузионному повреждению и травматическому рабдомиолизу. Наиболее часто он ассоциируется с природными катастрофами и преимущественно поражает нижние конечности, в то время как изолированное поражение верхней конечности в условиях, не связанных со стихийными бедствиями, встречается крайне редко.

Цель: Представить редкий случай тяжёлого краш-синдрома верхней конечности в некатастрофических условиях и оценить эффективность раннего мультимодального лечения.

Материалы и методы : У пациента 28 лет развился краш-синдром после 6–8 часов продолжительного сдавления левой верхней конечности на фоне потери сознания. Проанализированы клинические, лабораторные и инструментальные данные. Больному была выполнена ранняя фасциотомия, интенсивная терапия, терапевтический плазмаферез (4 сеанса) и заместительная почечная терапия (5 сеансов гемодиализа).

Результаты: У пациента развился тяжёлый рабдомиолиз (креатинфосфокиназа до 93 994 ЕД/л), острое повреждение почек (креатинин до 740 мкмоль/л), гиперкалиемия и полиорганная дисфункция. При ранней фасциотомии выявлены первоначально нежизнеспособные мышечные ткани с последующим частичным восстановлением жизнеспособности. Комплексное лечение привело к значительному клиническому улучшению, восстановлению диуреза (до 4800 мл/сут) и полному восстановлению функции почек.

Заключение: Тяжёлый краш-синдром верхней конечности в условиях, не связанных со стихийными бедствиями, может иметь благоприятный исход при проведении раннего и агрессивного мультимодального лечения. Ранняя хирургическая декомпрессия в сочетании с терапевтическим плазмаферезом и гемодиализом может существенно улучшить прогноз и снизить летальность и инвалидизацию в отдалённом периоде.

Ключевые слова: Синдром длительного сдавления, рабдомиолиз, верхняя конечность, фасциотомия, плазмаферез, острое повреждение почек.