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Successful Multidisciplinary Management of Crush Syndrome with a Nine-Year Longitudinal Psychological–Behavioral Follow-Up: A Case Report

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Case Report

Keywords: crush syndrome, acute kidney injury, continuous renal replacement therapy, fasciotomy, post-traumatic growth, case report

Posted Date: November 25th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11215794/v1

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Additional Declarations: No competing interests reported.

Abstract

Background

Crush syndrome is a life-threatening complication of prolonged compression of skeletal muscle, characterized by rhabdomyolysis, hyperkalemia, and acute kidney injury (AKI). Although its acute management has been extensively described, long-term psychological and behavioral outcomes after successful treatment remain rarely reported.

Case presentation:

A 25-year-old man sustained approximately 7 hours of continuous compression of the chest, abdomen, and bilateral lower extremities in a tractor rollover accident. On admission, laboratory tests revealed marked leukocytosis (white blood cells 29.67 × 10^9/L), hyperkalemia (5.8 mmol/L), and prolonged prothrombin time (15.4 s). Ten hours after admission, oliguria and myoglobinuria developed, progressing to anuria on hospital day 3, with serum myoglobin peaking at 2,708 ng/mL and serum creatinine at 920.6 μmol/L. Compartment syndrome of the right lower extremity required fasciotomy on hospital day 2. Continuous renal replacement therapy (CRRT) was initiated approximately 14 hours after admission and continued throughout the 17-day oliguric/anuric phase, after which renal function recovered progressively. During 9 years of follow-up, the patient's psychological state evolved from initial unawareness and denial, through hopelessness and despair, to a positive, resilient, and health-conscious lifestyle with sustained commitment to family responsibilities, consistent with post-traumatic growth.

Conclusions

This case demonstrates that timely fasciotomy and early CRRT, combined with integrated psychological support, can achieve favorable long-term outcomes after severe crush syndrome. The 9-year psychological–behavioral trajectory supports embedding structured psychological screening and stepped-care interventions into crush syndrome management and argues for prospective cohort studies of long-term outcomes.

1. Introduction

Crush syndrome, first systematically described in victims of the London Blitz, results from prolonged compression of skeletal muscle and is characterized by rhabdomyolysis, myoglobinuria, hyperkalemia, hypovolemia, and AKI [1–3]. Approximately 2%–5% of earthquake victims develop crush syndrome; the incidence may reach 10.5% in severely compressed victims, and mortality without aggressive treatment ranges from 40% to 100% [1, 2]. Although rhabdomyolysis is the common final pathway, the rapid and severe systemic manifestations of crush syndrome distinguish it from uncomplicated rhabdomyolysis [4]. The syndrome is not confined to disasters: similar mechanisms occur in industrial accidents,

building collapses, and road-traffic accidents [5–7]. The treatment evidence base derives mainly from retrospective earthquake cohorts [8–11]; randomized controlled trials are lacking, and current management relies on early aggressive fluid resuscitation [3, 12, 13], correction of electrolyte disturbances, renal replacement therapy (RRT) [14], and selective fasciotomy for compartment syndrome [15, 16]. In contrast to the well-characterized acute phase, the psychological–behavioral consequences of surviving crush syndrome are poorly documented, and case reports with follow-up exceeding several years are rare. We report a 25-year-old male patient with severe crush syndrome who was treated successfully and followed for 9 years, focusing on the trajectory of psychological–behavioral recovery.

2. Case presentation

2.1. History and admission findings

A 25-year-old previously healthy man was admitted after a tractor rollover accident in which his chest, abdomen, and bilateral lower extremities had been compressed for approximately 7 hours. He reported persistent dull pain over the chest, abdomen, and lower extremities, numbness and immobility of both lower limbs, chest tightness, dyspnea, and nausea; the right lower limb was the most severely affected. Color Doppler ultrasonography on admission revealed marked soft-tissue edema of the medial right thigh. Initial laboratory results showed a white blood cell count of 29.67 × 10^9/L (neutrophils 26.38 × 10^9/L, 88.9%), a platelet count of 117 × 10^9/L, a prothrombin time of 15.4 seconds, and a serum potassium concentration of 5.8 mmol/L.

Ten hours after admission, urine output decreased markedly and myoglobinuria appeared. On hospital day 2, the 24-hour urine output fell to 355 mL (oliguria), serum myoglobin was 2,708 ng/mL, and creatine kinase-MB exceeded 300 ng/mL. The right lower limb showed increasing tension with circulatory impairment and skin blistering, and both lower limbs exhibited sensory and motor deficits. Progressive systemic inflammatory response, hyperkalemia, hypermyoglobinemia, and acute renal failure developed, and the patient became anuric on hospital day 3. A diagnosis of crush syndrome complicated by compartment syndrome of the right lower extremity and crush-related AKI was established.

2.2. Treatment course

Management comprised (1) local decompression of the right thigh; (2) CRRT with urine alkalinization, targeting a urine output above 100 mL/h to protect renal function; (3) prevention and management of complications; (4) neurotrophic therapy; and (5) fluid resuscitation combined with diuretics. The patient and his family were fully informed of the disease severity, risks, costs, and prognosis. Right-thigh fasciotomy was performed on hospital day 2, and CRRT was initiated on the first hospital day, approximately 14 hours after admission. CRRT was delivered as continuous veno-venous hemofiltration (blood flow 200–250 mL/min; replacement-fluid rate 3–4 L/h; anticoagulant administered as an initial dose of 15–30 mg followed by 2–8 mg/h).

The patient remained oliguric or anuric for 17 days and received CRRT throughout this period, with serial laboratory monitoring showing progressive improvement (Table 1). Because of coagulopathy and severe anemia, he received 1,000 mL of virus-inactivated fresh frozen plasma and 14 units of leukoreduced packed red blood cells, together with recombinant human erythropoietin and human albumin. After 17 days, urine output recovered (polyuric phase), serum creatinine declined, and CRRT was discontinued; anti-infective therapy, organ-supportive care, and correction of hypoalbuminemia and multiorgan dysfunction were continued until discharge.

Table 1
Laboratory parameters before and after treatment

Parameter	Before treatment	After treatment	Adult reference range
Hemoglobin (g/L)	47	104	120–160
Blood urea nitrogen (mmol/L)	36.2	9.7	2.9–8.2
Serum creatinine (μmol/L)	920.6	101.5	57–97
Prothrombin time (s)	17.1	12.5	11–14
Serum myoglobin (ng/mL)	> 3,000	67	< 70
Serum potassium (mmol/L)	6.2	3.9	3.5–5.3
Serum albumin (g/L)	22.3	38.7	40–55

Values are transcribed from the patient's medical records; reference ranges are representative adult values and may vary among laboratories. The unit of serum myoglobin in the original record (ng/L) was corrected to ng/mL, the standard clinical unit; please verify against the original laboratory report.

2.3. Follow-up and psychological–behavioral trajectory

The patient was discharged in stable condition with substantially recovered renal and limb function. During 9 years of continuous follow-up, his psychological–behavioral status evolved through three phases: (i) an initial phase of unawareness and denial, with limited appreciation of the severity of the injury; (ii) a phase of hopelessness and despair during the prolonged hospitalization and rehabilitation; and (iii) gradual adaptation culminating in a positive, optimistic, and resilient attitude characterized by regular physical exercise, a health-conscious lifestyle, self-discipline, and a strong sense of responsibility toward his family and children, with these attitudes translated into sustained daily behavior.

3. Discussion

3.1. Determinants of successful treatment in the present case

Timely decompression and early RRT were the cornerstones of management. Compartment syndrome is a limb-threatening complication of crush injury whose clinical signs are insensitive [15, 16]; prompt fasciotomy relieves compartment pressure and permits reperfusion of viable muscle. Although evidence that routine fasciotomy improves long-term limb function is conflicting and fasciotomy may increase the risk of wound infection [17, 12], the clear clinical evidence of compartment syndrome in the present case—tense limb, circulatory impairment, blistering, and neurological deficit—justified early decompression. Early aggressive fluid resuscitation [3, 12, 13] and CRRT initiated within the first 24 hours prevented fatal hyperkalemia and maintained metabolic control [14]. CRRT was continued through the 17-day oliguric/anuric phase until renal recovery; although myoglobin clearance is governed mainly by endogenous metabolism and is not substantially influenced by the blood purification modality [14], CRRT provides stable fluid and electrolyte management in hemodynamically unstable patients [10, 11].

3.2. Current limitations in the clinical management of crush syndrome

Despite these advances, several limitations persist. First, the evidence base is dominated by retrospective earthquake cohorts, and no randomized controlled trials have evaluated the core interventions; treatment is largely extrapolated from rhabdomyolysis research [4, 8–11]. Extracorporeal membrane oxygenation (ECMO) has also been reported to improve survival in trauma patients with refractory hypoxemic respiratory failure and may serve as a salvage strategy in crush-associated ARDS [18]. Second, early risk stratification remains difficult: admission vital signs do not reliably reflect the extent of muscle necrosis [8]; creatine kinase peaks late and is not a specific early predictor of AKI requiring RRT [19]; established predictors such as entrapment time, male sex, multiple crushed limbs, and high creatine kinase level identify higher-risk patients [20]; and although the McMahon score improves early risk prediction in rhabdomyolysis, its performance in crush cohorts requires prospective validation [21, 22]. Third, the optimal fluid strategy—volume targets and the roles of urinary alkalinization and mannitol—remains debated [4, 23]. Fourth, the optimal timing and modality of RRT are unsettled, and hyperkalemia may recur despite dialysis [11, 14]. Fifth, in mass-casualty settings, limited dialysis capacity and complex evacuation logistics, the so-called “secondary renal disaster,” delay timely treatment [24, 25]. Sixth, surgical decision-making regarding fasciotomy and amputation remains contentious [15–17]. Finally, the psychosocial dimension is largely neglected: approximately one in four earthquake survivors develops post-traumatic stress disorder (PTSD) [26–28], physical injury is an independent risk factor [26, 28], and psychiatric morbidity was recorded in only 1% of a large crush-AKI cohort [10], indicating substantial under-recognition.

3.3. Psychological–behavioral recovery after crush syndrome

The 9-year trajectory observed in our patient—from unawareness and denial, through despair, to resilience and positive lifestyle change—illustrates the dynamic nature of psychological adaptation after life-threatening injury. PTSD is common after earthquakes, affecting approximately one quarter of

survivors [26–28], and ICU diaries reduce new-onset PTSD after critical illness [29]. At the same time, a substantial proportion of survivors of severe physical trauma report post-traumatic growth (PTG), which is associated with approach-oriented coping and social support [30]. Several interventions can support this adaptation: music therapy has a medium-to-large effect on stress-related outcomes [31]; and stepped-care psychological interventions and trauma-focused cognitive-behavioral therapy are effective in preventing the onset of PTSD [32]. In the present case, continuous family involvement, repeated explanation of the disease and prognosis, and structured rehabilitation likely contributed to the patient's eventual psychological recovery, although these were not delivered within a formal, scale-based psychological care pathway.

3.4. Strengths and limitations of the single-case design

The present findings must be interpreted with caution, as with all case reports [33, 34]. A single observation cannot establish generalizability or causality; retrospective data collection, unmeasured co-interventions, and the absence of a control group limit causal inference [34]; and the psychological outcomes were assessed qualitatively rather than with validated instruments (e.g., PCL-5, HADS, or PTGI), precluding quantitative comparison with published norms. Publication bias toward successful or unusual outcomes further constrains interpretation [33, 34]. The present report contributes a rare 9-year psychological–behavioral trajectory after crush syndrome and has been prepared in accordance with the CARE reporting guideline [35]. Nevertheless, case reports retain genuine value in detecting novelties, generating hypotheses, and documenting feasibility [33, 36].

3.5. Outlook: toward a prospective clinical cohort

On the basis of the limitations identified above, we plan to expand this work into a prospective clinical cohort. Four priorities are planned: (1) a protocolized care pathway standardizing fluid targets, criteria for RRT initiation and modality selection, fasciotomy indications, and staged amputation decisions, using current AKI definitions [37]; (2) prospective validation of the McMahon score and candidate urinary biomarkers (e.g., neutrophil gelatinase-associated lipocalin, kidney injury molecule-1) in crush syndrome to improve early risk stratification [21, 22]; (3) integration of routine psychological screening and stepped-care interventions—including psychoeducation, ICU diaries [29], and trauma-focused cognitive-behavioral therapy [32]—with longitudinal assessments of PTSD, depression, PTG, quality of life, and return-to-work at 6, 12, and 24 months; and (4) establishment of a multicenter registry with standardized data elements and telemedicine-based referral networks to enable adequately powered analyses of treatment effects and long-term outcomes, including renal recovery and limb function [25, 17].

4. Conclusions

This case demonstrates that timely fasciotomy, early CRRT, and comprehensive supportive care can achieve full recovery from severe crush syndrome complicated by AKI and compartment syndrome. The 9-year psychological–behavioral follow-up reveals a trajectory of adaptation consistent with post-traumatic growth and underscores the importance of integrating psychological care into crush syndrome

management. Prospective cohort studies that combine standardized acute treatment with structured, longitudinal psychobehavioral assessment are needed to transform crush syndrome management from an experience-based into an evidence-based discipline.

Declarations

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and any accompanying data.

Availability of data and materials: The data supporting the findings of this case report are available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no competing interests.

Funding: This work received no funding.

Ethics statement: Not applicable. Written informed consent for publication was obtained from the patient.

Author Contribution

1.2. wrote the main manuscript text and 3.4. prepared figures . All authors reviewed the manuscript."

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