

High-Voltage Electrical Injury with rhabdomyolysis: a case report

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Case Report

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Abstract

Introduction

High-voltage electrical injuries are uncommon but potentially limb- and life-threatening because the extent of internal tissue damage may greatly exceed the apparent cutaneous injury. Early recognition of deep muscle injury, metabolic complications, and occult systemic involvement is essential, particularly in patients with upper-extremity involvement and functional impairment.

Case presentation

A 24-year-old man with no relevant past medical history presented after a workplace high-voltage electrical accident involving the left upper extremity. Physical examination revealed a circumferential second-degree burn of the left shoulder and a third-degree burn of the left hand with tissue loss, painful edema of the left arm, and forced flexion of the fingers and wrist with difficulty in extension. Initial laboratory evaluation showed leukocytosis, hypoxemia, mild acidemia, hypokalemia, and marked muscle injury with high levels of creatine kinase. Serial testing over the following day demonstrated worsening rhabdomyolysis and severe hyperlactatemia with metabolic acidosis. Despite this, renal function remained within the normal range in all reported samples. The electrocardiogram showed sinus rhythm, normal QRS duration and no acute ischemic or arrhythmic abnormalities. After computed tomography and plastic surgery evaluation, urgent surgery was performed. The patient received cefazolin, tetanus prophylaxis, intravenous fluids, postoperative intensive care monitoring, and transfer to a referral center for specialized plastic surgery care.

Conclusion

This case illustrates that high-voltage electrical trauma may present with relatively localized external burns while causing profound muscle injury, major biochemical evidence of rhabdomyolysis, and transient metabolic derangement. Serial laboratory assessment and multidisciplinary management are critical, even when early hemodynamic status, renal function, and ECG findings appear reassuring.

Introduction

Electrical injuries differ from conventional thermal burns because current passage may produce extensive deep tissue destruction, muscle necrosis, compartment syndrome, rhabdomyolysis, renal injury, and arrhythmia, even when visible skin findings seem limited. High-voltage exposure is associated with greater morbidity, and burns involving the hand or any confirmed high-voltage electrical injury warrant specialist burn-center consultation and consideration for transfer.

We report the case of a young worker with high-voltage electrical injury of the left upper extremity, notable not only for mixed-depth burns and acute functional impairment, but also for serial biochemical evidence of severe rhabdomyolysis without early acute kidney injury and an ECG without acute electrical

complications. This combination makes the case clinically relevant because it illustrates the disconnect between external burn extent, internal muscle injury burden, and early organ function.

Patient Information

A 24-year-old man presented to the emergency department after a workplace accident in which, while wearing a safety harness and rubber gloves, he touched a high-voltage electrical cable with his left hand. He immediately developed pain and functional loss of the left upper extremity. He denied head trauma, syncope, vomiting, chest pain nor dyspnea. He had no known drug allergies, denied toxic habits, had no relevant prior medical or surgical history, and was not taking regular medication.

Clinical Findings

Initial physical examination showed a circumferential second-degree burn of the left shoulder and a third-degree burn of the ventral left hand with tissue loss (Fig. 1). The left hand was held in forced flexion of the fingers and wrist, with difficulty extending them (Fig. 2). The left arm was painful and edematous, although peripheral pulses were present and symmetrical. These findings suggested significant deep tissue involvement despite the relatively limited external burn distribution. After surgery, on arrival to the intensive care unit, the patient was alert, conscious, and oriented. Vital signs were stable, with blood pressure 139/81 mmHg, heart rate 90 beats/min, respiratory rate 16 breaths/min, and oxygen saturation 95%. The surgical dressing was clean, and mobility and sensation were preserved in the fingers of the left hand.

Diagnostic Assessment

The diagnosis was based on the mechanism of injury, physical findings, and subsequent biochemical and electrocardiographic assessment. Clinically, the patient had a high-voltage electrical burn of the left upper extremity with mixed-depth burns, tissue loss, edema, and acute motor dysfunction. CT of the left upper extremity was performed, showing muscle swelling and edema.

The first urgent laboratory sample showed leukocytosis of $19.20 \times 10^3/\mu\text{L}$ with neutrophilia, potassium 3.3 mmol/L, CK 9,705 U/L, LDH 439 U/L, AST 130 U/L, pH 7.34, pO₂ 57 mmHg, and oxygen saturation 87.3%, consistent with acute stress, hypoxemia, and major muscle injury after electrical trauma. Coagulation parameters were initially normal, and creatinine was 0.88 mg/dL.

Two further samples documented dynamic worsening of muscle injury. One sample showed CK 13,247 U/L, AST 273 U/L, ALT 53.4 U/L, LDH 501 U/L, mild coagulation derangement with Quick index 68% and INR 1.29, and severe hyperlactatemia at 10.48 mmol/L with metabolic acidosis, reflected by pH 7.31, bicarbonate 15.1 mmol/L, base excess - 10, and compensatory low pCO₂. Another sample showed CK 25,959 U/L, AST 391 U/L, ALT 73 U/L, and LDH 946 U/L, confirming severe rhabdomyolysis and evolving tissue injury burden.

Despite the marked enzymatic evidence of muscle necrosis, renal function remained preserved in all reported tests, with creatinine values of 0.88 mg/dL, 0.52 mg/dL, and 0.72 mg/dL, and estimated glomerular filtration rate > 90 mL/min when available. This suggests early biochemical rhabdomyolysis without documented early acute kidney injury.

Inflammatory markers did not strongly support early infection: C-reactive protein remained low at 0.47 and 0.82 mg/dL, and procalcitonin was 0.08 and 0.06 ng/mL, supporting the interpretation that the biochemical abnormalities were primarily driven by acute trauma and tissue necrosis.

The ECG showed sinus rhythm at 95 bpm, PR interval 121 ms, QRS duration 95 ms, QT 362 ms, QTc 455 ms, and normal frontal axes; no clear acute ischemic changes, conduction disturbances, or sustained arrhythmias were identified (Fig. 3).

Therapeutic Intervention

After CT assessment and evaluation by the plastic surgery team, urgent surgery was indicated. Perioperative management included cefazolin 2 g, as antibiotic prophylaxis, tetanus vaccination, and 1000 mL of intravenous fluid therapy during surgery. Urgent fasciotomy was carried out without incident.

The patient was subsequently admitted to the intensive care unit for monitoring and treatment. Given the magnitude of CK elevation and the risk of pigment nephropathy after electrical rhabdomyolysis, serial laboratory monitoring and supportive fluid management were clinically important components of care.

Transfer to a referral center for specialized plastic surgery management was appropriate because the case combined high-voltage exposure, a deep hand burn with tissue loss, and acute functional compromise of the affected limb.

Follow-up and Outcomes

In the immediate postoperative period, the patient remained hemodynamically and respiratory stable. On ICU arrival, he was alert, adequately perfused, and had clean surgical dressings. Finger mobility and sensation were preserved, suggesting that despite substantial tissue injury, some distal neurological and functional integrity remained at that early stage.

The serial laboratory course showed marked ongoing muscle injury over the first hospital day, with CK rising from 9,705 U/L to 13,247 U/L and later to 25,959 U/L, accompanied by rising AST and LDH (Table 1). Nevertheless, creatinine remained normal throughout the measurements, and no documented arrhythmia or acute ECG abnormality was identified. This combination of severe rhabdomyolysis with preserved early renal function makes the evolution particularly noteworthy.

Discussion

This case demonstrates a classic but often underappreciated feature of high-voltage electrical trauma: the skin injury may only partially reflect the true systemic and deep-muscle burden. Externally, the patient had localized mixed-depth burns centered on the left shoulder and hand. Internally, however, he developed profound biochemical evidence of muscle necrosis, with CK increasing to 25,959 U/L and associated marked AST and LDH elevation.

A second notable feature is the evidence of transient metabolic stress. One venous sample revealed severe hyperlactatemia and metabolic acidosis, with lactate 10.48 mmol/L, pH 7.31, and bicarbonate 15.1 mmol/L. In the context of electrical injury, this most likely reflects acute tissue hypoperfusion and anaerobic metabolism related to muscle damage and systemic stress rather than infection, especially because procalcitonin and CRP remained low in the available samples.

A third clinically relevant point is the dissociation between the severity of rhabdomyolysis and the absence of early kidney injury. Despite CK values in the severe range, creatinine remained normal across serial determinations. This does not exclude later renal complications, but it shows that early renal function may remain preserved even in the presence of dramatic muscle breakdown, reinforcing the importance of close serial monitoring rather than relying on a single reassuring creatinine value.

The ECG findings also add value to the report. Cardiac complications are a well-known concern after electrical injury, but in this patient the tracing demonstrated sinus rhythm without clear acute ischemic, conduction, or sustained arrhythmic abnormalities. This helps frame the case as one in which the major early threat was deep local and muscular injury rather than overt electrical cardiac instability, while still supporting the appropriateness of cardiac evaluation in the acute setting.

From a management standpoint, the case supports current recommendations for rapid multidisciplinary care. The combination of high-voltage exposure, a deep hand burn, tissue loss, edema, and functional impairment justified urgent surgery, ICU monitoring, and transfer to a specialized reconstructive center.

Conclusion

This case report describes a work-related high-voltage electrical injury of the left upper extremity in a young man, resulting in mixed-depth burns, tissue loss, acute functional impairment, severe rhabdomyolysis, transient metabolic derangement, and preserved early renal function. The case illustrates why electrical burns should not be judged solely by external appearance and why serial laboratory follow-up can substantially increase the clinical value of a case report.

Table 1
Key serial laboratory findings

Date	Main findings	Clinical interpretation
1st day	WBC $19.20 \times 10^3/\mu\text{L}$, neutrophilia, K 3.3 mmol/L, CK 9,705 U/L, AST 130 U/L, LDH 439 U/L, pH 7.34, pO ₂ 57 mmHg, SaO ₂ 87.3%, creatinine 0.88 mg/dL	Early stress response, hypoxemia, mild acidemia, and marked muscle injury without renal failure
2nd day	CK 13,247 U/L, AST 273 U/L, LDH 501 U/L, lactate 10.48 mmol/L, pH 7.31, HCO ₃ 15.1 mmol/L, INR 1.29, creatinine 0.52 mg/dL	Severe rhabdomyolysis with lactic/metabolic acidosis and mild coagulation abnormality, renal function preserved
3rd day	CK 25,959 U/L, AST 391 U/L, ALT 73 U/L, LDH 946 U/L, WBC $12.90 \times 10^3/\mu\text{L}$, creatinine 0.72 mg/dL, CRP 0.47 mg/dL, procalcitonin 0.08 ng/mL	Biochemical peak of muscle injury with preserved renal function and no strong evidence of early infection

Declarations

Competing interests

The authors declare that they have no competing interests.

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The authors received no specific funding for this work.

Ethics statement

Ethical approval was not required for this case report because it describes the retrospective clinical course of a single patient managed according to standard care, without experimental intervention or protocol-driven research procedures.

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Figures



Figure 1

Circumferential second-degree burn of the left shoulder and a third-degree burn of the ventral left hand with tissue loss. The left hand was held in forced flexion of the fingers and wrist.



Figure 2

Urgent fasciotomy of the left arm.

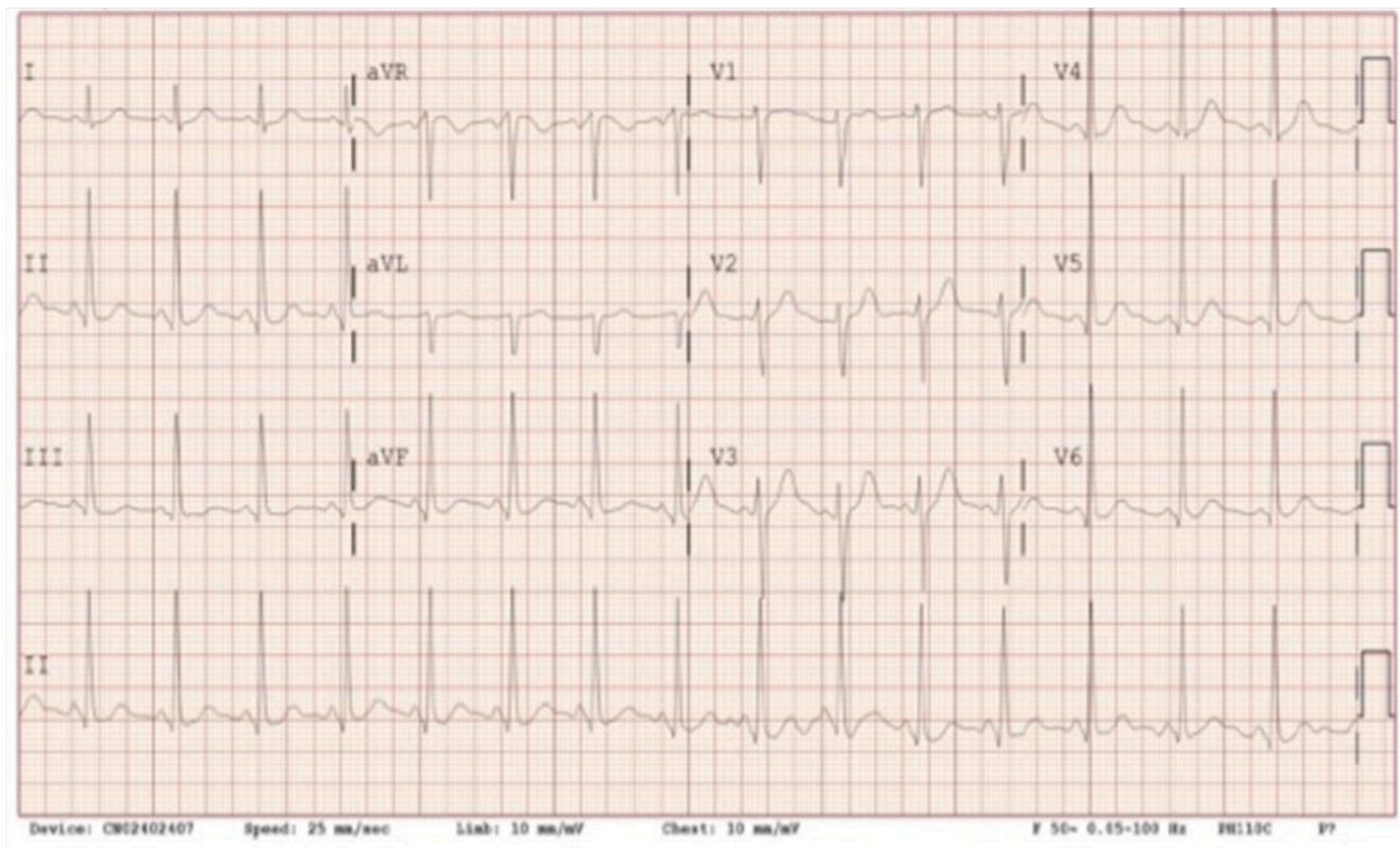


Figure 3

Electrocardiogram. Twelve-lead ECG showing sinus rhythm at 95 beats/min, PR 121 ms, QRS 95 ms, QT 362 ms, QTc 455 ms, normal frontal axes, and no clear acute ischemic or sustained arrhythmic abnormality on the available tracing.