



CASE REPORT: DELAYED ONSET MALIGNANT ARRHYTHMIA SECONDARY TO ELECTROCUTION

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Abstract

Background: High-voltage electrical injuries can precipitate acute cardiac instability. Malignant arrhythmias may arise days to weeks later. Reports of delayed-onset bradyarrhythmia after electrocution are scarce and optimal surveillance strategies remain ill-defined.

Case Presentation: A 28-year-old male electrician sustained a 10,000-volt electrocution to the left upper limb - with entry and exit burns at the hand and forearm. He was hemodynamically stable on arrival; electrocardiography (ECG) and serum electrolytes were normal. Because of the high-voltage exposure he was admitted to the intensive-care unit for continuous monitoring. Rapidly progressive compartment syndrome mandated emergent fasciotomy. This was followed by guillotine amputation of the right hand when tissue perfusion proved non-viable. Post-operatively he developed stump infection requiring debridement and split-skin grafting.

Four weeks after injury the patient acutely deteriorated with profound bradycardia (heart rate 30 beats min⁻¹) and hypotension. Repeat ECG indicated multiple bradycardic runs with absent P-waves - consistent with malignant bradyarrhythmia. Immediate intravenous atropine restored perfusion and he was transferred to a cardiac care unit. Echocardiography demonstrated only trace mitral regurgitation; a 24-h Holter confirmed bradyarrhythmia with a minimum heart rate of 39 beats min⁻¹ and 59 % of total beats in bradycardia. Supportive therapy, β -blockade and an ACE inhibitor were initiated. The patient remained stable under prolonged telemetry and was discharged with plans for outpatient cardiology follow-up and rehabilitation of the amputated limb.

Conclusion: This case illustrates that life-threatening bradyarrhythmia can manifest weeks after apparently uncomplicated high-voltage electrocution. Prolonged cardiac monitoring and a multidisciplinary approach are essential to detect and manage delayed electrical injury sequelae.

Keywords: high-voltage electrocution; delayed arrhythmia; bradycardia; guillotine amputation; cardiac monitoring; case report

Introduction

Electrical injuries represent a unique subset of trauma that can produce multisystem damage out of proportion to the visible cutaneous findings. Globally they account for up to 5 % of burn-centre admissions and are a leading occupational hazard among electricians, construction workers and agricultural labourers.¹⁻³ High-voltage exposures (>1,000 V) are lethal as they produce deep tissue necrosis, compartment syndrome and devastating neurologic and cardiac sequelae. Survival depends on initial resuscitation, surgical management of the burn wound and also on vigilance for occult, delayed complications that may arise long after the apparent stabilization of the patient.⁴⁻⁶

Cardiac manifestations after electrocution span an immediate to delayed continuum. Immediately, the trans-thoracic passage of current can precipitate ventricular fibrillation, asystole or nonspecific ST-T changes. Most surveillance protocols recommend at least 24 h of telemetry for high-risk patients. Accumulating evidence suggests that malignant dysrhythmias may appear days to weeks later despite an unremarkable early course [4]. Proposed mechanisms include (A) electrical disruption of autonomic fibres (B) progressive microvascular ischemia and (C) delayed fibrotic remodelling of myocardial tissue. These are processes that can evolve much beyond the time window of standard observation periods [5, 6].⁷⁻¹⁰

Bradyarrhythmias constitute a rare but life-threatening subset of these late complications. In the largest contemporary review - fewer than 2 % of high-voltage survivors developed clinically significant conduction defects after 48 h; among those affected - mortality exceeded 30 %. The low incidence has hampered large prospective studies and left clinicians to extrapolate monitoring strategies from isolated case reports and small series. There is no consensus on the optimal duration of cardiac telemetry or the indications for prophylactic pacing in apparently stable survivors.^{8,9}

The surgical management of electrical burns represents a race against progressive tissue necrosis. Electrical current travels along fascial planes and produces deep muscle and vascular injury while sparing superficial skin i.e., it masks the true extent of damage. Fasciotomy to relieve compartment pressures, aggressive debridement and early amputation (when necessary) are critical to prevent systemic sepsis and improve rehabilitation [8]. Multidisciplinary coordination between burn surgeons, intensivists and cardiologists is therefore indispensable.

The present report describes a 28-year-old electrician who - after a seemingly uncomplicated acute course - developed malignant bradyarrhythmia four weeks after a 10,000-volt injury that had already required guillotine amputation¹¹ of the right hand. This case emphasizes three key themes: (1) the insidious nature of delayed electrical cardiac injury (2) the need for extended surveillance protocols beyond current standards and (3) the importance of integrated surgical and cardiologic care paths in mitigating long-term morbidity.^{7,8,12,13}

Case Presentation

Patient Description

A 28-year-old male electrician with no known prior cardiac illness or significant comorbidities.

Case History

The patient was admitted to the emergency department after an accidental contact with a live 10,000-volt electrical wire while at work. He experienced a brief loss of consciousness but regained it spontaneously on-site. He did not report chest pain, muscle soreness or other complaints on arrival.

Physical Examination Results

On examination, the patient was alert and hemodynamically stable. Notable findings included (A) **Entry burn:** Right hand (B) **Exit burn:** Right forearm; No other significant abnormalities were noted on initial systemic examination.

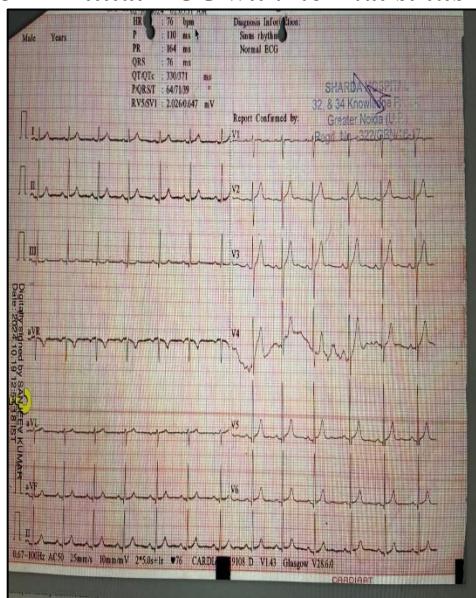
Figure 1 – Patient's left hand - extensive tissue necrosis, discoloration & demarcated burn injuries following high-voltage electrical contact injury.



Results of Pathological Tests and Other Investigations

After admission the patient underwent an initial electrocardiogram (ECG) which demonstrated normal sinus rhythm.

Figure 2 - Initial ECG with normal sinus rhythm



His serum electrolytes were within normal ranges. Despite stable initial findings - he was admitted to the intensive care unit (ICU) for close cardiac monitoring due to the high-voltage nature of his injury.

Table 1 – Case record details

Time Point	Investigation	Result/Findings	Remarks
Admission	ECG	Normal sinus rhythm	No acute changes
	Serum Electrolytes	Within normal limits	No electrolyte imbalance
Initial	Physical Exam	Entry burn (right hand), exit burn (right forearm)	Signs of compartment syndrome
Early hospital	Surgical Exploration	Fasciotomy, non-viable tissue; guillotine amputation	Prevent sepsis
Post-op	Wound Exam	Fever, purulent discharge at amputation stump	Wound infection
	Wound Debridement	Necrotic tissue removed, split skin graft applied	For wound closure

Treatment Plan

After admission - the patient was closely monitored in the intensive care unit (ICU) due to the high-voltage nature of his electrical injury, despite initially stable vital signs and a normal ECG. During early management the focus was on monitoring for potential cardiac complications and addressing the significant soft tissue injury to his right hand - which showed clinical signs of compartment syndrome.

After the development of compartment syndrome in the right hand - surgical intervention was performed. An emergency fasciotomy was performed to relieve compartment pressure and the exposed tissues were assessed for viability. Due to poor perfusion and the risk of developing sepsis - a guillotine-type amputation of the right hand was carried out. Postoperatively - the patient developed fever and there was evidence of local infection at the amputation stump. Surgical debridement was performed followed by split skin grafting to facilitate wound closure.

Figure 3 - Postoperative image after guillotine amputation and wound closure with sutures



Figure 4 - Guillotine amputation specimen - extensive tissue necrosis & burn injury secondary to high-voltage electrocution



Throughout his hospital course - continuous cardiac monitoring was maintained.

Approximately four weeks after the initial electrocution - the patient experienced sudden hemodynamic instability with a markedly slow heart rate (bradyarrhythmia, HR 30 bpm). Repeat ECG at this time showed multiple bradycardic episodes and absent P waves. This indicated malignant arrhythmia. An echocardiogram showed trace mitral regurgitation but no other significant structural heart disease. A 24-hour Holter study confirmed the presence of bradyarrhythmia. A minimum recorded heart rate of 39 beats per minute was observed and 59% of total beats in the bradycardia range.

4 weeks post-injury	ECG	Multiple bradycardic runs, absent P wave	Malignant arrhythmia diagnosed
	Echocardiogram	Trace mitral regurgitation; otherwise normal	No structural heart disease
	24-hour Holter	Bradyarrhythmia, min HR: 39 bpm, 59% beats in bradycardia	Confirmed diagnosis

Considering the delayed malignant arrhythmia - the treatment approach included supportive care, close cardiac observation and initiation of pharmacological therapy i.e., beta-blockers and ACE inhibitors as indicated for cardiac dysfunction secondary to electrical injury. The multidisciplinary team followed up on ongoing wound care, infection prevention and regular follow-up for both his cardiac and surgical needs. Long-term plans included outpatient cardiology follow-up, serial echocardiography and rehabilitation for his upper limb amputation.

Outcome of the Treatment Plan

The patient's bradyarrhythmic event was acutely managed and he was stabilized in the cardiac care unit. He remained under close cardiac monitoring and continued postoperative care for his amputation stump. Long-term follow-up was planned for continued cardiac and surgical rehabilitation.

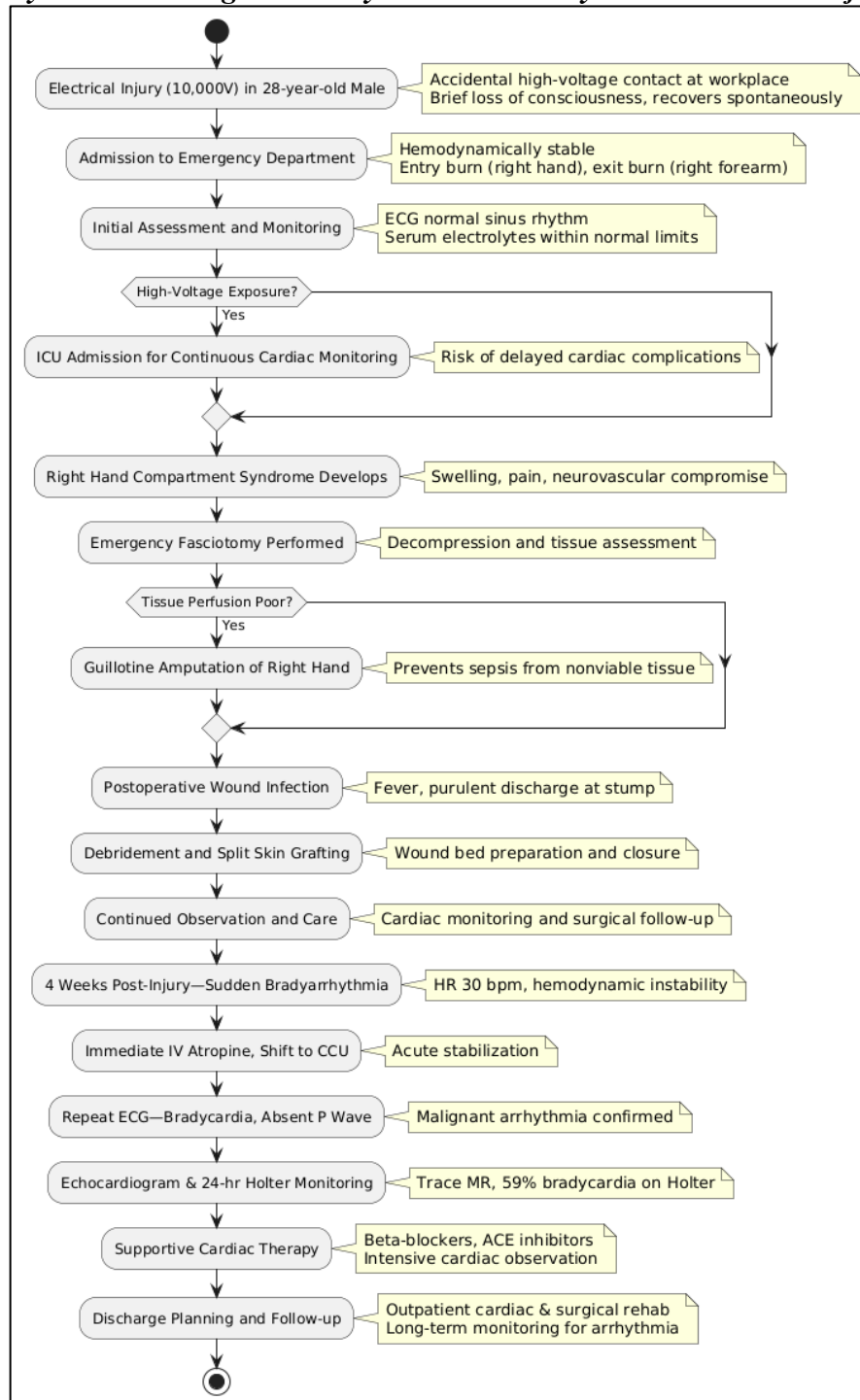
Case Significance

This case highlights a rare but severe complication: **delayed onset malignant arrhythmia** after high-voltage electrocution. Although the initial cardiac workup was unremarkable, the patient developed a life-threatening bradyarrhythmia weeks later. This emphasized the need for prolonged cardiac monitoring in all high-voltage electrical injury cases. Early recognition and intervention are vital.

Delayed arrhythmogenic effects may be fatal without timely management. The case also emphasized the importance of multidisciplinary care which involves both surgical and cardiac teams - in managing complex electrocution injuries.

Overall Case Flow chart

Figure 5 - Delayed Onset Malignant Arrhythmia Secondary to Electrocutation Injury: Case Flow



Discussion

Electrical injuries are complex events that may produce immediate and delayed consequences, particularly for the cardiovascular system. High-voltage electrical trauma - as illustrated in this case - carries substantial risk for local tissue necrosis and also for life-threatening cardiac arrhythmias.

Immediate vs. delayed arrhythmias: While most arrhythmic complications after electrical injury tend to occur immediately, there are rare cases of delayed-onset malignant arrhythmias which can occur days or even weeks after the initial event; these have been reported in the literature.

Incidence and risk assessment: Large cohort studies, such as that by Pilecky et al. (2019), suggest that most arrhythmias associated with electrical accidents are detected on admission ECG, and the risk of late-onset malignant arrhythmia is exceedingly low. In their analysis of 480 patients, no clinically relevant late arrhythmias were observed during monitoring or at 30-day follow-up, even among those with workplace high-voltage injuries. This is consistent with Fatovich's (2007) literature review, which concluded that delayed lethal arrhythmias after electrocution are “exceptionally rare,” and that in asymptomatic patients with a normal ECG, routine prolonged cardiac monitoring may not be required.^{7,10,13–15}

Individual case reports and smaller series still document rare occurrences of delayed malignant arrhythmia and some with fatal outcomes. Fatovich described instances of lethal arrhythmias occurring hours to days after low- and high-voltage shocks. Some of the instances they detailed were with initial ECG changes and others with none.¹⁴ Jensen et al. also reported delayed ventricular arrhythmias occurring up to two months post-injury - particularly in patients who had initial ECG abnormalities.¹⁶

Pathophysiology and risk factors: The mechanisms which form the basis for delayed arrhythmias are not fully understood. Proposed theories include progressive microvascular ischemia, myocyte necrosis and fibrotic changes in the cardiac conduction system. These factors could potentially evolve for weeks after the initial injury. This is supported by both clinical and histopathological studies. High-voltage injuries, loss of consciousness, transthoracic current path and severe soft tissue damage have been considered risk factors in some studies. Neither Pilecky et al. nor Fatovich found clear predictive clinical markers for delayed arrhythmia in their large cohorts.^{10,14}

Biomarkers and monitoring: The role of cardiac biomarkers e.g., troponin I and CK-MB in risk stratification after electrical injury is limited. Pilecky et al. found that troponin I elevation was rare, and when present, was not predictive of arrhythmia. Most arrhythmic complications, when they did occur, were detected on ECG at presentation. Routine serial measurement of cardiac necroenzymes in stable patients without ECG changes may not be warranted and could increase healthcare costs without improving outcomes.¹⁰

Clinical implications: Our case of delayed, life-threatening bradyarrhythmia developing weeks after a high-voltage injury. It demonstrates an important exception to the generally positive outcomes reported in the literature. It highlights the limitations of current guidelines, which often focus on short-term monitoring for at-risk patients but may not address rare, late complications. Extended or outpatient cardiac monitoring may be considered in select high-risk individuals – e.g., those with severe burns, extensive tissue injury or those who have undergone major surgical intervention post-electrocution. Patient education regarding warning symptoms and the potential for late cardiac effects is crucial at discharge.

Limitations: The available evidence is limited by the predominance of low-voltage injuries in most large cohorts, and by the retrospective nature of many studies. As noted by Pilecky et al., results may not be fully generalizable to high-voltage cases - which are less common but can be more severe.¹⁰ Not all arrhythmias (i.e., non-fatal or transient events) may be captured unless continuous monitoring is performed.

Conclusion

The risk of delayed malignant arrhythmias after electrical injury is very low overall. But clinicians should be aware that such complications can occur following high-voltage exposures. Our case supports the need for a cautious and individualized approach to monitoring and follow-up. We emphasize the importance of multidisciplinary care in managing the complex sequelae of electrical trauma.

References

1. Maghsoudi H, Adyani Y, Ahmadian N. Electrical and lightning injuries. *Journal of Burn Care and Research*. 2007;28(2).
2. Duff K, McCaffrey RJ. Electrical injury and lightning injury: A review of their mechanisms and neuropsychological, psychiatric, and neurological sequelae. Vol. 11, *Neuropsychology Review*. 2001.
3. Shaha KK, Joe AE. Electrocution-related mortality: A retrospective review of 118 deaths in Coimbatore, India, between January 2002 and December 2006. Vol. 50, *Medicine, Science and the Law*. 2010.
4. An SJ, Kayange L, Davis D, Peiffer S, Gallaher J, Charles A. Predictors of mortality following electrical and lightning injuries in Malawi: A decade of experience. *Burns*. 2024;50(3).
5. Cooper MA. Emergent care of lightning and electrical injuries. Vol. 15, *Seminars in Neurology*. 1995.
6. Arnoldo BD, Purdue GF, Kowalske K, Helm PA, Burris A, Hunt JL. Electrical injuries: A 20-year review. Vol. 25, *Journal of Burn Care and Rehabilitation*. 2004.
7. Searle J, Slagman A, Maaß W, Möckel M. Cardiac Monitoring in Patients With Electrical Injuries. *Dtsch Arztebl Int*. 2013;
8. Claudet I, Maréchal C, Debuissou C, Salanne S. Risk of arrhythmia and domestic low-voltage electrical injury. *Arch Pediatr*. 2010;17.
9. Searle J, Slagman A, Maaß W, Möckel M. Cardiac monitoring in patients with electrical injuries. An analysis of 268 patients at the Charité Hospital. *Dtsch Arztebl Int*. 2013;110(50).
10. Pilecky D, Vamos M, Bogyi P, Muk B, Stauder D, Racz H, et al. Risk of cardiac arrhythmias after electrical accident: a single-center study of 480 patients. *Clinical Research in Cardiology*. 2019;108(8).
11. Schulze SM, Weeks D, Choo J, Cooney D, Moore AL, Sebens M, et al. Amputation Following Hand Escharotomy in Patients with Burn Injury. *Eplasty*. 2016;16.
12. Waldmann V, Narayanan K, Combes N, Jost D, Jouven X, Marijon E. Electrical cardiac injuries: Current concepts and management. Vol. 39, *European Heart Journal*. 2018.
13. Yew K. Electrocution induced symptomatic bradycardia necessitating pacemaker implantation. *Heart Views*. 2014;15(2).
14. Fatovich DM. Delayed lethal arrhythmia after an electrical injury [1]. Vol. 24, *Emergency Medicine Journal*. 2007.
15. Ghazal Asswad A, Holm S, Engström O, Huss F, Lipcsey M, Rudolph A. Delayed, Unprovoked, Hemodynamic Collapse with Following Asystole in a Pediatric Patient Following a High-Voltage Injury: A Case Report and Literature Review. *Pediatr Cardiol*. 2022;43(5).
16. Jensen PJ, Thomsen PE, Bagger JP, Nørgaard A, Baandrup U. Electrical injury causing ventricular arrhythmias. *Br Heart J*. 1987;57(3).