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CASE REPORT

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V-A ECMO for refractory malignant arrhythmia and cardiogenic shock following acute hydrofluoric acid burns: a case report and pathophysiology review

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Abstract

Background Hydrofluoric acid (HF) exposure causes severe systemic toxicity through fluoride ion-mediated electrolyte disturbances, leading to life-threatening cardiovascular complications. While extracorporeal membrane oxygenation (ECMO) has been reported for respiratory failure in HF poisoning, its use for refractory malignant arrhythmias and cardiogenic shock remains extremely rare.

Case presentation A 52-year-old male worker sustained extensive HF burns (30% of total body surface area) following an industrial accident. Despite aggressive electrolyte correction and blood purification, he developed refractory ventricular arrhythmias requiring more than 37 defibrillations within 24 h and progressive cardiogenic shock requiring escalating vasopressor support (norepinephrine equivalent up to 1.3 μg/kg/min). On the advice of a multidisciplinary consultation, veno-arterial (V-A) ECMO was successfully implemented, resulting in immediate cessation of malignant arrhythmias and rapid hemodynamic stabilization. The patient was weaned from ECMO after 4 days and discharged home after complete recovery.

Conclusion This case demonstrates the life-saving potential of V-A ECMO in severe HF poisoning complicated by refractory malignant arrhythmias and cardiogenic shock. Early recognition of the need for mechanical circulatory support and timely ECMO implementation may be crucial for survival in such cases.

Keywords Hydrofluoric acid, Chemical burns, Malignant arrhythmia, Cardiogenic shock, Veno-arterial ECMO, Blood purification

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Background

Hydrofluoric acid (HF), despite being a weak acid, exhibits extreme biological toxicity [1]. Unlike conventional acids causing superficial coagulative necrosis, HF demonstrates dual toxicity: hydrogen ions cause surface corrosion while fluoride ions rapidly penetrate body tissues and enter systemic circulation, causing severe systemic toxicity [2]. Cardiovascular complications are the primary cause of death in HF poisoning [3, 4]. Fluoride ions chelate calcium and magnesium while inhibiting Na^+/K^+-ATPase, creating the characteristic electrolyte triad: severe hypocalcemia, hypomagnesemia, and hyperkalemia [2, 5, 6]. These disturbances prolong cardiac action potentials, increase QT intervals, and trigger malignant arrhythmias including torsades de pointes [3, 7, 8]. Fluoride ions also inhibit essential enzymes and induce oxidative stress, while hypocalcemia impairs myocardial excitation-contraction coupling, leading to myocardial depression and potentially refractory cardiogenic shock [3, 8, 9].

Conventional treatments often show limited efficacy in severe cases with circulatory collapse [2, 10]. Veno-arterial extracorporeal membrane oxygenation (V-A ECMO) provides mechanical circulatory support for refractory arrhythmias and cardiogenic shock [11], creating therapeutic windows for recovery. However, ECMO use in HF burns is extremely rare, previously reported only for severe hypoxemia [12]. We report a case of extensive HF burns with refractory malignant arrhythmias and cardiogenic shock, successfully managed with V-A ECMO.

Case presentation

Initial exposure and emergency response

A 52-year-old male silicon industry worker sustained extensive HF burns on Day 1 at approximately 10:00 AM when his forklift punctured an HF storage tank. Massive spillage involved the head, face, neck, chest, back, waist, and buttocks (approximately 30% total body surface area). He experienced severe pain and acute vision loss but remained conscious without respiratory distress. Following immediate clothing removal and 10 min of water irrigation, he was transferred to a local hospital, then to our burn center at 11:45 AM after peripheral IV access and crystalloid administration.

Initial presentation and early management

On admission, he was agitated and tachypneic, with SpO_2 89%, throat secretions, bilateral wet rales, heart rate (HR) 108/min, and blood pressure (BP) 105/84 mmHg. Examination revealed severe eyelid edema with corneal opacities, conjunctival hyperemia, facial swelling, and oral vesicles. Burns appeared erythematous with desquamation and red-white mottling. Due to respiratory compromise and burn extent, he underwent immediate intubation, wound debridement, and treatment with topical calcium gluconate, IV methylprednisolone, and furosemide before intensive care unit (ICU) transfer.

At ICU admission, he had unmeasurable non-invasive BP, mottled extremities, HR 132/min, respiratory rete (RR) 33/min, and peripheral oxygen saturation (SpO_2) 85% (mechanical ventilation [MV], fraction of inspired oxygen [FiO_2] 100%). Arterial blood gas (ABG) showed mixed acidosis, severe hypoxemia, and severe hypocalcemia (Ca^{2+} 0.48 mmol/L). Lactate was 3.6 mmol/L. Electrocardiogram (ECG) showed complete right bundle branch block and QTc prolongation (564ms). Immediate interventions included ventilation optimization, sedation with midazolam/remifentanil, central venous access, crystalloid resuscitation, bicarbonate, and calcium gluconate. Left radial arterial monitoring revealed BP 55/35 mmHg, prompting norepinephrine 0.22 μg/kg/min to maintain target mean arterial pressure (MAP) > 65 mmHg. Bedside echocardiography revealed preserved ejection fraction (EF) of 61%.

Refractory arrhythmias and cardiogenic shock

At 13:57, ventricular fibrillation (VF) occurred and was successfully defibrillated. Cardiac biomarkers were elevated: troponin I 0.68 ng/ml, creatine kinase-myocardial band (CK-MB) 8.36 ng/ml, myoglobin 1132.0 ng/ml. Laboratory findings revealed inflammation (white blood cell [WBC] 18.78×10^9/L, neutrophils 88.1%, procalcitonin [PCT] 2.06 ng/ml), renal impairment (urea 8.42 mmol/L, creatinine 150 μmol/L), and hypomagnesemia (0.492 mmol/L). Piperacillin-tazobactam 4.5 g q8h was started with continuous calcium gluconate and magnesium sulfate. Bronchoscopy at 16:00 showed bilateral mucosal inflammation with secretions, managed with saline lavage. BP became increasingly difficult to maintain despite escalating norepinephrine (Fig. 1).

Despite correction attempts, recurrent malignant arrhythmias occurred. Over the next two hours, he experienced 17 VF or polymorphic ventricular tachycardia (VT) episodes (Fig. 2), each requiring defibrillation. Right femoral venous cannulation for hemoadsorption (HA130, Jafron, Zhuhai City, China) and continuous renal replacement therapy (CRRT) was initiated to enhance fluoride elimination, achieving partial biochemical improvement. However, more than 20 additional malignant arrhythmias occurred overnight, requiring repeated shocks. Progressive cardiac dysfunction developed (EF 47%), with rising lactate (5 mmol/L) and escalating vasopressor support (norepinephrine equivalent 1.3 μg/kg/min) (Fig. 1).

ECMO support and recovery

On Day 2, refractory arrhythmias and cardiogenic shock persisted despite electrolyte correction. Multidisciplinary consultation determined that ongoing hypoxemia and

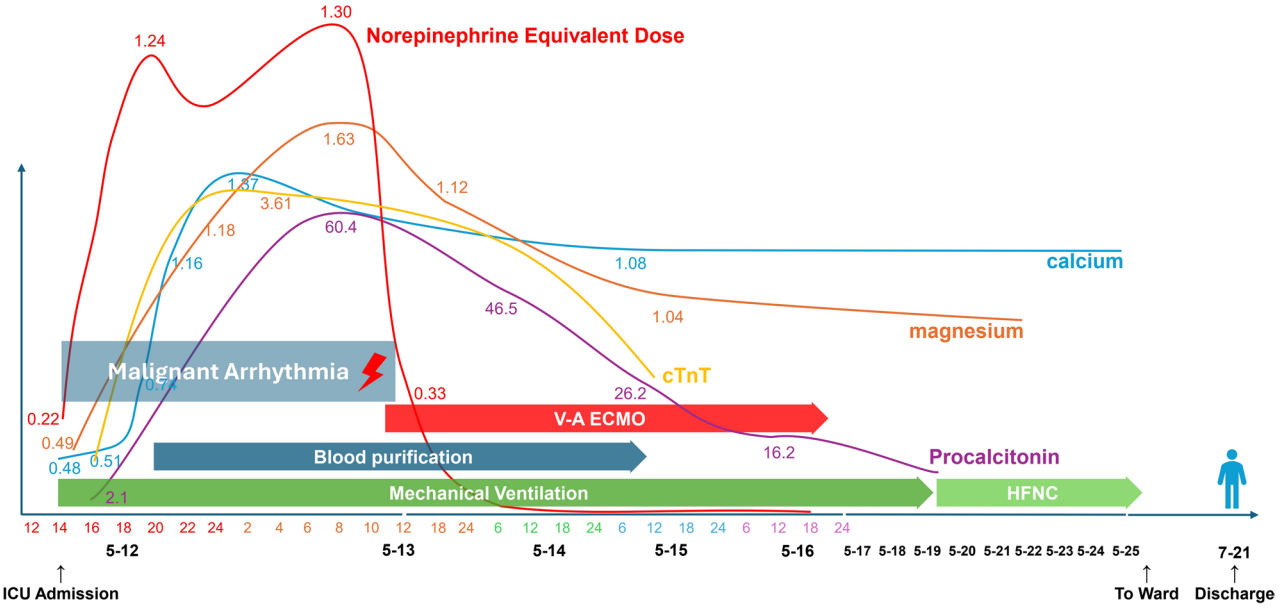

Fig. 1 Clinical course and treatment timeline. Abbreviations: cTnT Cardiac troponin T; ECMO Extracorporeal membrane oxygenation; HFNC High-flow nasal cannula; ICU Intensive care unit; V-A Veno-arterial

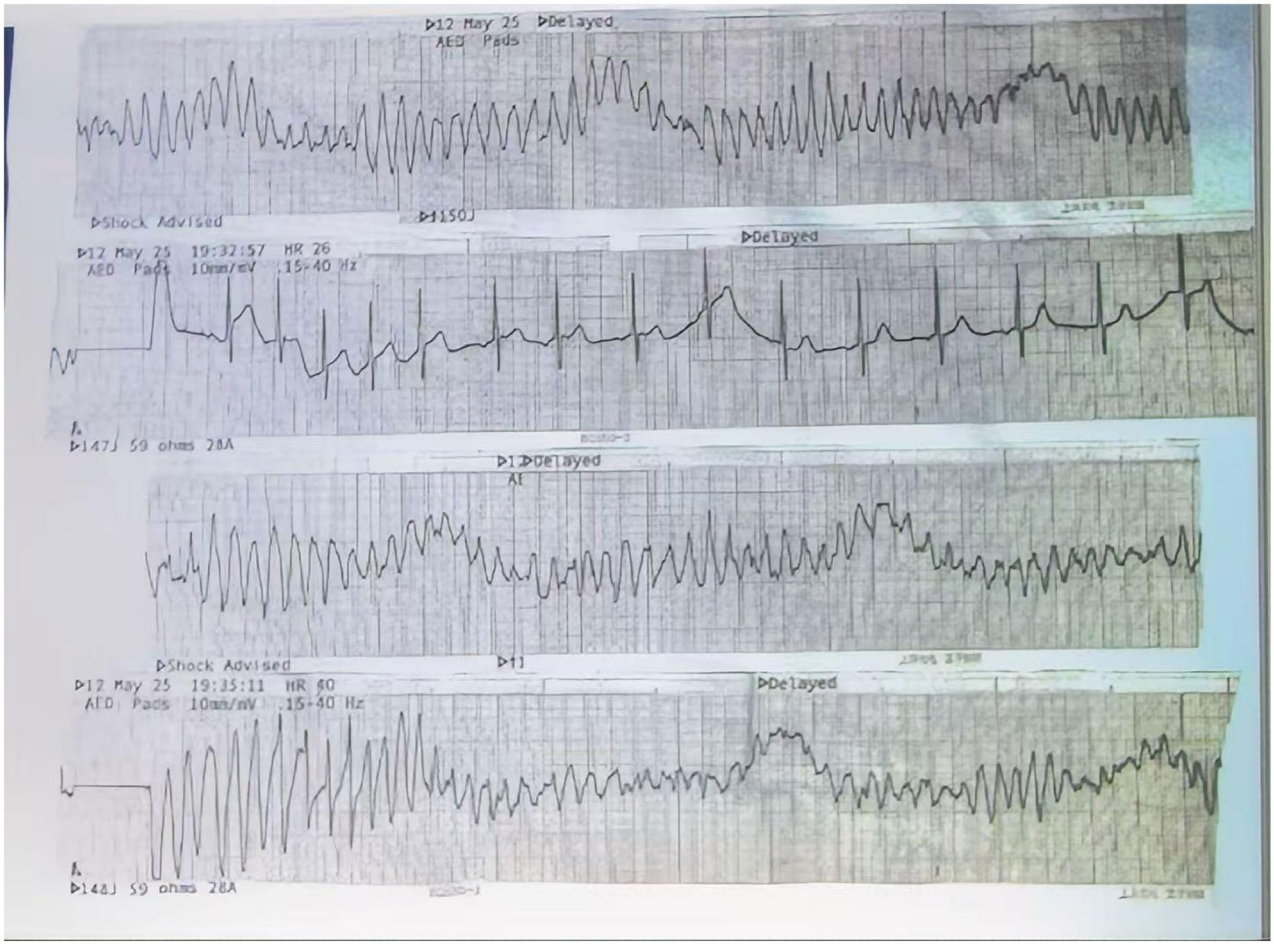

Fig. 2 Representative electrocardiograms demonstrating malignant arrhythmia

CO_2 retention (PCO_2 59 mmHg) despite lung-protective ventilation, combined with refractory malignant arrhythmias and cardiogenic shock, met V-A ECMO criteria. At 11:00 (21 h after first VF), left femoral arteriovenous cannulation with distal perfusion catheter successfully established V-A ECMO (flow 3 L/min, with anticoagulation targets of activated clotting time [ACT] 180–200 s and activated partial thromboplastin time [APTT] 40–50 s under heparin administration). Post-ECMO, circulation dramatically improved with cessation of arrhythmias and rapid vasopressor reduction (Fig. 1).

Subsequently, cardiac function improved (EF 52–54%), vasopressors were tapered, and electrolytes normalized (Fig. 1). Levosimendan and recombinant human B-type natriuretic peptide (BNP) were initiated for cardiac function improvement and decongestion. Bronchoscopy showed resolving mucosal inflammation. Chest computed tomography (CT) on Day 4 revealed bilateral interstitial changes, patchy opacities, and lower lobe consolidation consistent with chemical pneumonitis and pulmonary edema.

ECMO weaning

Infectious complications emerged during ICU stay. PCT levels steadily increased post-admission, reaching 60.4 ng/ml on Day 2 before gradually declining while remaining elevated (Fig. 1). Clinical deterioration occurred on Day 5 with fever (38.5 °C), leukocytosis, and elevated PCT (16.2 ng/ml). Sequencing of bronchial alveolar lavage identified multiple pathogens (Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, and Pneumocystis jirovecii), leading to antimicrobial therapy modification with piperacillin-tazobactam, vancomycin, and trimethoprim-sulfamethoxazole.

Given elevated infection risk, comprehensive ECMO weaning assessment was initiated based on favorable parameters: improved cardiac function (EF 54% with ECMO flow 2.5 L/min), reduced vasopressor requirement (norepinephrine decreased to 0.10 μg/kg/min), normalized metabolic parameters, and downtrending cardiac enzymes (Fig. 1). Following Fried et al.'s protocol [13], systematic evaluation confirmed weaning readiness. Successful ECMO decannulation was performed by cardiothoracic surgeons with direct arterial and venous repair.

Clinical outcome

This patient was successfully extubated on Day 8. Visual function began to improve on Day 9 and returned to normal by Day 13. He was transferred from the ICU to the burn unit on Day 14 for ongoing wound care and rehabilitation. Following complete recovery, he was discharged on Day 71.

Discussion

Pathophysiology of hydrofluoric acid burns

As a weak acid, hydrofluoric acid exhibits pH-dependent dissociation characteristics. In its concentrated industrial form, HF remains predominantly undissociated due to the acidic environment, which enhances its tissue penetration capability. The undissociated HF molecules, despite their polar nature, exhibit unique penetration ability due to their small molecular size and ability to interact directly with tissue structures—dissolving the stratum corneum and destroying cell membrane integrity to achieve rapid deep tissue penetration [11, 13]. Once these undissociated molecules penetrate into tissues and encounter the physiological pH environment of body fluids, they dissociate predominantly into fluoride ions (F^-), which become the primary toxic factor causing local tissue necrosis and systemic toxicity. Fluoride ions exhibit extremely high affinity for calcium and magnesium, forming insoluble precipitates that cause severe hypocalcemia and hypomagnesemia—the primary mechanism underlying fatal arrhythmias [2, 6]. Unlike coagulative necrosis from other strong acids, HF causes progressive liquefactive necrosis extending to bone with severe pain. In the cardiovascular system, electrolyte disturbances prolong QT interval and impair myocardial contractility, while fluoride ions directly affect cardiac ion channels and mitochondria, inducing refractory malignant arrhythmias and cardiogenic shock [2, 14]. Additionally, fluoride acts as a potent enzyme inhibitor, causing multi-organ toxicity through Na^+/K^+-ATPase inhibition, oxidative stress induction, and apoptotic pathway activation [2, 14]. This dual toxicity mechanism—local destruction combined with systemic multi-organ failure—makes even small surface exposures potentially fatal.

Insidious systemic toxicity and arrhythmia risk

HF poisoning exhibits insidious, progressive pathophysiology with ongoing fluoride penetration leading to progressive electrolyte disturbances. Each 1% increase in body surface area involvement is associated with 2.28-fold higher systemic toxicity risk [7]. However, industrial sites typically lack specialized neutralizing agents (Hexafluorine, calcium gluconate solutions/gels), relying only on water irrigation [15]. Industrial facilities with high HF usage are often located remotely with only community hospitals nearby, and even larger emergency departments lack HF burn experience due to rarity, resulting in missed optimal treatment windows. High-risk patients should receive prophylactic IV calcium even without measured levels, with simultaneous magnesium once hypocalcemia is confirmed [13, 14]. Early ECG assessment is crucial—QTc prolongation predicts fatal arrhythmias and supports preventive measures like ECMO standby, while

normal QTc followed by VF emphasizes vigilance even with initially normal findings.

Multi-factorial cardiac injury and mechanical circulatory support

Despite relatively prompt treatment, severe cardiac impairment still occurred in our case. Previous literature described a 91% burn patient developing hypotension 4 h post-injury with initially normal cardiac function [16], similar to our presentation. However, our patient had lower calcium/magnesium levels with more severe arrhythmias and shock than typically reported [16, 17]. Cases have described delayed fatal electrolyte disturbances and cardiac arrest 16 h post-exposure [18], emphasizing continuous monitoring needs. HF's direct effects on cardiac pacemaker and conduction systems remain unclear, with multiple cases of persistent VF despite calcium correction, suggesting direct myocardial toxicity beyond electrolyte disturbances [19]. Additionally, repeated electrical cardioversion may contribute to myocardial injury through high-energy stimulation and prolonged ischemia-reperfusion cycles, potentially compounding cardiac dysfunction in HF poisoning patients requiring multiple defibrillations. Unlike previous reports using veno-venous (V-V) ECMO for respiratory failure [12], our primary V-A ECMO indication was refractory malignant arrhythmias and cardiogenic shock—both representing established ECMO indications, yet their application in HF poisoning contexts remains unreported.

Reflection on V-A ECMO initiation timing

Despite the decision to implement V-A ECMO following multidisciplinary consultation, our implementation of V-A ECMO was insufficiently proactive. Initially, while domestic recommendations [20–22] suggested aggressive electrolyte correction, our literature review revealed no V-A ECMO reports for HF poisoning, and experienced colleagues advised electrolyte correction first. We were concerned about ECMO's lack of definitive mortality benefit in cardiogenic shock and initially perceived HF poisoning as reversible. However, HF creates an "electrolyte black hole" where fluoride continuously consumes supplemented calcium, trapping us in prolonged futile correction attempts. Although malignant arrhythmias and cardiogenic shock met established cardiovascular specialist criteria for mechanical support [11], we delayed by attempting to optimize conservative treatment. This delay represented a critical learning point—emergency physicians and intensivists managing HF poisoning may become overly focused on electrolyte correction while overlooking optimal timing for mechanical circulatory support. Similarly, Prasad reported a patient considered for V-A ECMO who suffered multiple cardiac arrests during preparation and died [23], emphasizing early implementation once criteria are met. Repeated defibrillation attempts, while life-saving, may contribute to myocardial injury and stunning through high-energy electrical stimulation, potentially delaying critical timing for V-A ECMO initiation. This represents a valuable lesson for medical colleagues—early V-A ECMO consideration should not await prolonged electrolyte correction attempts in patients with refractory arrhythmias and hemodynamic instability.

ECMO management and blood purification strategy

During recovery, we used levosimendan, whose calcium sensitization mechanism provides theoretical advantages in HF-induced hypocalcemic cardiomyopathy by enhancing contractility without increasing intracellular calcium burden [24]. Meta-analyses show levosimendan significantly improves V-A ECMO weaning success [25]. Recombinant human BNP provides additional benefits through congestion relief and sodium-water excretion during recovery [26]. Our blood purification strategy evolved during treatment—initially employing hemoadsorption (HA130) based on empirical assumptions of HF lipophilicity, then transitioning to CRRT upon recognizing that fluoride ions (F^-) exist primarily as water-soluble small molecules (19 Da) more amenable to diffusion and convection clearance. Although fluoride removal efficiency is limited due to tissue reservoir effects and continuous release from bone deposits, CRRT provided multifold benefits including electrolyte balance maintenance, precise volume control, and adjunctive fluoride clearance [27]. Blood purification has become routine for severe HF poisoning regardless of renal function [13, 14, 16, 28]. Additionally, evidence from cardiac surgery populations supports earlier CRRT initiation for improved survival outcomes [29].

Infection risk management

V-A ECMO weaning was partly necessitated by infectious complications. Extensive HF burn skin barrier destruction creates extreme infection susceptibility from wounds, invasive procedures, and immune compromise. Airway mucosal burns plus intubation predispose to ventilator-associated pneumonia. Our patient developed multi-pathogen pulmonary infection requiring antimicrobial modification. Chitosan-based hydrogels demonstrate potential for promoting HF burn healing with antibacterial effects [30]. Timely pathogen identification and precision antimicrobials are crucial for maintaining ECMO support, requiring multidisciplinary collaboration to balance infection risk with circulatory support needs. Post-ECMO, circulation dramatically improved with arrhythmia cessation and rapid vasopressor reduction, confirming the effectiveness of mechanical support in bridging recovery time for HF toxicity resolution.

Conclusion

This case highlights the life-saving role of V-A ECMO in the management of severe HF burns complicated by refractory malignant arrhythmias and cardiogenic shock. Key factors contributing to success included CRRT support, ECMO implementation and cardiac function optimization, and close multidisciplinary collaboration.

Abbreviations

ABG	Arterial blood gas
ACT	Activated clotting time
AKI	Acute kidney injury
APTT	Activated partial thromboplastin time
BAL	Bronchoalveolar lavage
BE	Base excess
BNP	Brain natriuretic peptide
BP	Blood pressure
CK-MB	Creatine kinase-myocardial band
CO_2	Carbon dioxide
CRRT	Continuous renal replacement therapy
CT	Computed tomography
ECG	Electrocardiogram
ECMO	Extracorporeal membrane oxygenation
EF	Ejection fraction
FiO_2	Fraction of inspired oxygen
HF	Hydrofluoric acid
HR	Heart rate
ICU	Intensive care unit
IV	Intravenous
MAP	Mean arterial pressure
mNGS	Metagenomic next-generation sequencing
MV	Mechanical ventilation
PCT	Procalcitonin
PRBC	Packed red blood cells
QTc	Corrected QT interval
RR	Respiratory rate
SpO_2	Peripheral oxygen saturation
TBSA	Total body surface area
V-A	Veno-arterial
VAP	Ventilator-associated pneumonia
VF	Ventricular fibrillation
V-V	Veno-venous
VT	Ventricular tachycardia
WBC	White blood cell

Supplementary Information

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Supplementary Material 1

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Author contributions

PZ, JCL and MHL drafted the manuscript. ZZS, RS, YPL and ZAZ contributed substantially to its revision. All authors take responsibility for the paper as a whole and approved the final manuscript.

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Data availability

The data used to support the findings of this case study are included within the article. The detailed data regarding the case presented are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The publication of the case report was approved by the patient and his family and submitted for records to the local institutional ethics committee.

Consent for publication

The patient gave written consent for publication of the case and photo documentation.

Competing interests

The authors declare no competing interests.

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