

Extracorporeal membrane oxygenation combined with continuous renal replacement therapy in cutaneous burn and inhalation injury caused by hydrofluoric acid and nitric acid

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Abstract

Rationale: Hydrofluoric acid (HF) is a highly corrosive agent and can cause corrosive burns. HF can penetrate deeply into tissues through intact skin and the lipid barrier, leading to painful liquefactive necrosis, and inducing hypocalcemia and hypomagnesemia. In this study, we hypothesize that continuous renal replacement therapy (CRRT) may be beneficial in addressing hemodynamic instability in cases of HF poisoning.

Patient concerns: A 25-year-old man fell into an electroplating pool containing 10% HF and 50% nitric acid.

Diagnoses: He had severe cutaneous injuries involving approximately 60% of his total body surface area including the head, face, neck, right upper arm, right hand, trunk, perineum, and both lower limbs and feet. Examination at admission showed the following electrolyte concentrations: ionic calcium 0.192 mmol/L, total calcium 0.72 mmol/L, magnesium 0.4 mmol/L, potassium 5.49 mmol/L, and sodium 136.8 mmol/L.

Interventions: An initial 20 mL intravenous bolus of 10% calcium gluconate was followed by a continuous infusion at 6 g/h plus continuous intravenous drip 25% magnesium sulfate at 1.5 g/h. Continuous cardiac monitoring was performed in the intensive care unit. Extracorporeal membrane oxygenation (ECMO) was used to improve oxygenation function at 38 hours post exposure. Antibiotic therapy using imipenem/cilastin plus vancomycin was required.

Outcomes: After treatment for 12 hours, electrolyte concentrations returned to normal. On day 11, the hemodynamic parameters were stable and oxygenation function had improved. On day 26, the patient was weaned off CRRT. One month later, the patient twice received skin grafting, then was discharged from the hospital without pulmonary, cardiac, or neurological complications 3 months later.

Lessons: The present case study demonstrates that CRRT may be an effective and potentially lifesaving therapy after severe exposure to HF. Prolonged hemodialysis is recommended to remove delayed release fluoride ions to avoid delayed systemic injury. When conventional therapy can not improve oxygenation and/or carbon dioxide retention, ECMO should be performed as soon as possible.

Abbreviations: ARDS = acute respiratory distress syndrome, BSA = burn surface area, CI = cardiac index, CRRT = continuous renal replacement therapy, CVVHD = continuous venovenous hemodialysis, ECMO = extracorporeal membrane oxygenation, ELWI = extravascular lung water index, HF = hydrofluoric acid, PEEP = positive end expiratory pressure, PICCO = pulse index continuous cardiac output, TBSA = total body surface area.

Keywords: extracorporeal membrane oxygenation, hydrofluoric acid, inhalation injury, nitric acid

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1. Introduction

Hydrofluoric acid (HF) and nitric acid are widely used in various chemical industries, metal refining and electroplating, electronics manufacturing, semiconductor preparation, glass etching, and other industrial and household applications.^[1–3] HF is a highly corrosive agent and a contact poison. In addition, when exposed to skin, HF can penetrate into through intact skin and lipid barriers into deep tissues, leading to painful liquefactive necrosis. After tissues are penetrated by HF, free fluoride ions are strongly reactive with calcium and magnesium ions, forming neutralizing salts and inducing hypocalcemia and hypomagnesemia.^[4,5] In addition, fluoride has a direct toxic effect on a number of cellular enzymes and organs including the heart, liver, kidney, central nervous system, and the pulmonary endothelium.^[5]

The majority of HF injuries are local burns, which mainly manifest as erythema and intense pain. The degree of systemic toxicity of extensive burns depends on the duration of exposure, HF concentration, burn surface area, burn depth, and the time elapsed between exposure and hospital care. The main cause of

patient death is due to hypocalcemia-related cardiac arrhythmia soon after exposure. A previous study reported a patient death with a burn area of only 2.5% of total body surface area (TBSA) after exposure to 70% HF.^[6] A number of case reports shows that mortality rates approach 100% when over 20% of the body surface area is exposed to high concentrations of HF.^[6–8] In addition, exposure to HF can also result in acute lung injury or acute respiratory distress syndrome (ARDS). In particular, routine mechanical ventilation or higher level of applied positive end expiratory pressure (PEEP) cannot improve oxygenation; extracorporeal membrane oxygenation (ECMO) may provide a new method for relief from hypoxemia and/or carbon dioxide retention.

We report here a case of occupational HF and nitric acid exposure with cutaneous burn and inhalation injury, which was complicated by recurrent ventricular fibrillation, serious ARDS, and HF poisoning. Because continuous renal replacement therapy (CRRT) may be beneficial in treating HF poisoning by addressing hemodynamic instability, it was combined with ECMO in the present case.

2. Case report

The ethics committee of Suzhou Municipal Hospital Affiliated with Nanjing Medical University approved the present study. A healthy 25-year-old man accidentally fell into an electroplating pool containing 10% HF and 50% nitric acid while he was working in a chemical plant. His whole body was soaked in the mixture of acids, immediately after which his co-workers removed his clothes and irrigated his entire body with plenty of running water for approximately 20 minutes. Calcium gluconate gel was not applied at the plant. Two hours later, he was sent to the hospital emergency room by ambulance. Upon admission, he was fully conscious and complained of mild dyspnea, hoarseness, and severe pain in the burn area with erythema or edematous. The vital signs were as follows: heart rate of 120 beats/min, respiratory rate of 28 breaths/min, axillary temperature at 37.5°, SaO₂ at 92%, and blood pressure of 124/78 mm Hg. Cutaneous injuries were present on approximately 60% of the TBSA, including the patient's head, face, neck, right upper arm, hand, trunk, perineum, both lower limbs, and feet. After the initial physical examination, third degree burns were present on approximately 13% of the burn area, whereas the remaining burn area showed deep partial thickness burns. The conjunctiva was congested and edematous, and the cornea was without ulcer. The eyes were treated by frequent rinsing with normal saline for 30 minutes, followed by levofloxacin eye drops and 1% calcium gluconate every 4 hours. A portion of the nasal and oral mucosa was pale, and profuse secretions had partially filled in the oral cavity, which was treated by rinsing with 5% calcium gluconate. Because of hypoxemia and inhalational injury, tracheotomy and bedside mechanic ventilation were immediately conducted. All burn areas were rinsed using 5% sodium bicarbonate and covered with wet sterile gauze containing 10% calcium gluconate. Electrocardiography was normal upon admission. Evaluation of serum electrolyte concentrations revealed that the concentration of ionic calcium was 0.192 mmol/L (normal range 1.12–1.32 mmol/L), total calcium was 0.72 mmol/L (normal range 1.8–2.6 mmol/L), magnesium was 0.4 mmol (normal range 0.66–1.2 mmol/L), potassium was 5.49 mmol/L (normal range 3.5–5.5 mmol/L), and sodium was 136.8 mmol/L (normal range 135–145 mmol/L) (Fig. 1). In addition, liver and renal functions were normal.

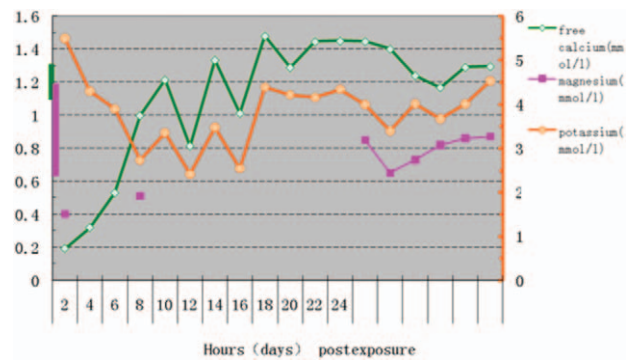


Figure 1. Serum ion concentration. The green line indicates free calcium concentration, the orange line indicates magnesium concentration, and the red line indicates potassium concentration.

Continuous cardiac monitoring was performed in the intensive care unit. The patient had immediately undergone fluid resuscitation, and also intravenous administration of 10% calcium gluconate and 25% magnesium sulfate. The initial 20 mL intravenous bolus of 10% calcium gluconate was followed by a continuous infusion at 6g/h based on the measured concentration of serum ionic calcium and continuous intravenous drip 25% magnesium sulfate at 1.5g/h according to the concentration of magnesium. Invasive hemodynamic monitoring was performed by pulse index continuous cardiac output (PICCO) to guide fluid resuscitation. The initial parameters of PICCO were as follows: cardiac index (CI) 3.02 L/min/m², extravascular lung water index (ELWI) 16 mL/kg, central venous pressure 5 mm Hg, systemic vascular resistance index 2213 dyn.s/cm⁵.m², and global end-diastolic index 450 mL/m². Because of fluoride intoxication, CRRT was applied to avoid further harm to the patient. However, despite the great efforts made, ventricular fibrillation occurred at 4 hours postexposure, and another 10s episodes of ventricular fibrillation took place within the next 3 hours. The patient was successfully defibrillated 10 times, and 10% calcium gluconate was administered immediately after defibrillation. Invasive arterial blood pressure (ABP 86/48 mm Hg) and CI (2.5 L/min/m²) had decreased significantly after defibrillation, and dobutamine was administered to improve left ventricular contraction. Additionally, glucocorticoid (methylprednisolone 40 mg q8h) and antibiotic (piperacillin tazobactam 4.5 g q8h) were given.

On the second day of hospitalization, the patient's oxygenation index had deteriorated rapidly (PaO₂/FiO₂ was 55.2/1.0) and hypoxemia had worsened. Meanwhile, the ELWI increased to 25 mL/kg, an abundance of frothy sputum was suctioned via tracheotomy, and pulmonary edema was confirmed by chest x-ray (Fig. 2). We attempted to increase ultrafiltration and PEEP; however, hypoxemia could not be redressed. Therefore, ECMO was used to improve oxygenation at 38 hours postexposure. The patient's cardiac function insufficiency was also taken into consideration and he was treated with venoarterial ECMO. The initial flow rate was 3.0 L/min and the activated clotting time of whole blood was maintained at 160–200 seconds (heparin 10–14 u/kg/h). The arterial oxygen saturation of the opposite upper limb was about 98%. During ECMO, hypoxemia was effectively corrected. On day 3, fiberoptic bronchoscopies were performed, and results showed the presence of pale mucus on the main bronchus, the left principal bronchus, the right principal bronchus, and the right inferior lobar bronchus. At the same

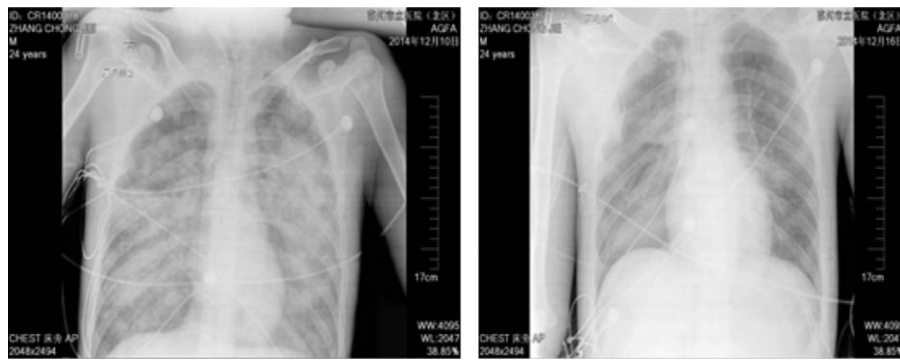


Figure 2. Chest radiograph. Left (December 10, 2014): on the second day after admission, chest x-ray shows diffuse, bilateral infiltrates consistent with pulmonary edema. Right (December 16, 2014): 1 week later, chest radiography shows infiltrates when compared with previous x-ray images.

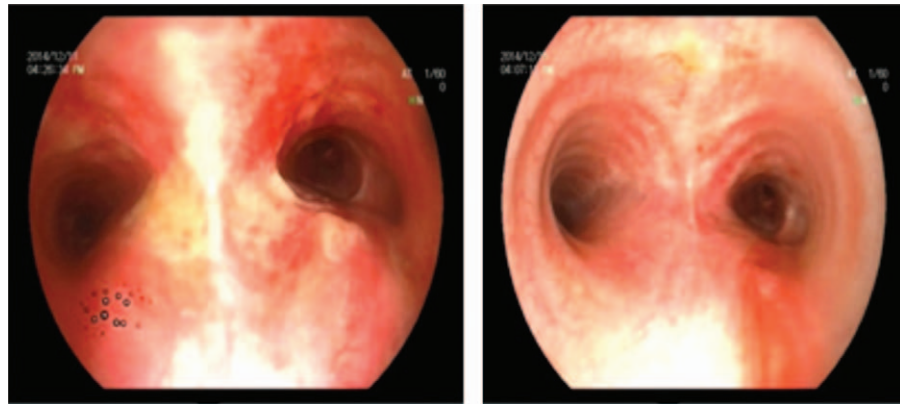


Figure 3. Image of fiber bronchoscopy. Left (December 11, 2014): the trachea, the main branch, and the bronchial mucosa show congestion and edema, the tracheal mucosa show signs of erosion; right (December 15, 2014): a section of bronchial mucosa shows congestion, significantly improved edema.

time, aspiration and lavage of the affected bronchi were performed and postirrigation fluid was yellow. On day 5, the patient developed burn wound infections. On day 7, fiberoptic bronchoscopies were performed again and showed congested, edematous mucosa (Fig. 3). On day 11, the patient’s hemodynamic profile was

stable and his oxygenation functions were improved. As a result, the patient was weaned off of ECMO, his catheter was removed, and vessels were repaired (Fig. 4). On day 16, a significant deterioration occurred due to pulmonary infection and burn wound infections. Microbiological identification showed that methicillin-resistant *Staphylococcus aureus* and *Pseudomonas aeruginosa* were positive in all wound smears and sputum, and antibiotic therapy using a combination of imipenem/cilastin plus vancomycin was commenced. On day 26, the patient was weaned off of CRRT. One month later, the patient twice received skin grafting. However, the patient was fearful of stopping CRRT and did not successfully cease CRRT until 2 months after the accident. The patient was discharged from the hospital without pulmonary, cardiac, or neurological complications 3 months later.

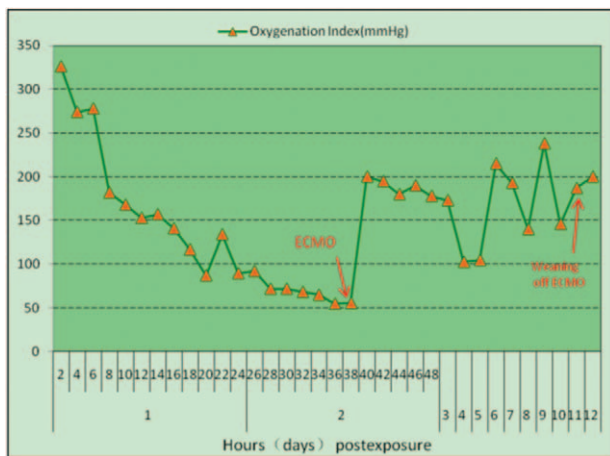


Figure 4. Time course of oxygenation index. Progressive decrease in oxygenation after admission, ECMO treatment 38 hours after injury, improved oxygenation after ECMO treatment, improved oxygenation after 1 week, ECMO 11 days after injury. ECMO=extracorporeal membrane oxygenation.

3. Discussion

Hydrofluoric acid is a relatively weak inorganic acid when compared with nitric acid, sulfuric acid, and hydrochloric acid. The clinical manifestation of cutaneous HF burns mainly depends on the concentration of the acid and the duration of contact. Therefore, HF burn injuries are divided into 3 categories based on the concentration of HF: <20%, 21%–50%, and >50%. When exposed to >50% HF, cutaneous symptoms express immediately, and are usually accompanied by intense pain and tissue destruction, whereas a moderate concentration of HF induces clinical symptoms only after 1 to 8 hours. Skin symptoms and

pain may be delayed for up to 24 hours after exposure to <20% HF. Various therapies are recommended for HF burn injuries. To our best knowledge, this is the first report on the use of ECMO combined with CRRT in an HF burn patient.

In patients suffering from HF burns, cardiac arrhythmia is the leading cause of death during the early stage, mainly due to polymorphic ventricular tachycardia and ventricular dysrhythmias. However, the mechanisms of cardiac arrhythmias owing to HF are still debated. Large numbers of case reports have demonstrated electrolyte imbalance in HF burn patients, akin to the severe hypocalcemia observed in our case. HF penetrates into the tissue, and then dissociates into hydrogen and free fluoride ions, which bind to calcium and magnesium, resulting in cellular dysfunction. Hence, it is vital to correct hypocalcemia in treatment to avoid the lethal systemic effects of cardiac complications.^[9] So far, there is a lack of reliable data on the duration and dose of calcium and magnesium therapy in cases of fluoride toxicity. In our case, we administered 55 g of 10% calcium gluconate during the first 24 hours. Calcium gluconate gel is an appropriate and typical treatment for HF burn injuries, but it may not be available in a timely manner in some situations. In the present case, the burned area was rinsed using 5% sodium bicarbonate and covered with wet sterile gauze containing 10% calcium gluconate. Greco et al^[10] reported that hypocalcemia may develop when: >1% body surface area is exposed to >50% HF; >5% body surface area is exposed to any concentration of HF; inhalation or ingestion of HF at any concentration. Wu et al^[11] reported a case of hypocalcemia after dermal exposure to 20% HF with a burn surface area (BSA) of 3% of TBSA. Despite these considerations, ventricular dysrhythmias may occur without electrolyte imbalance, as observed with the last 2 episodes of ventricular dysrhythmias in this case. A previous case report documented that ventricular dysrhythmias occurred 3.5 hours after exposure to 71% HF, even though the patient presented with normal serum electrolytes and blood gases.^[12]

Additional electrolyte imbalance has been reported in HF burn injuries including hyperkalemia, hypomagnesemia, and hyponatremia. Hyperkalemia has been proposed as another etiology of fluoride-associated ventricular dysrhythmias. Fluoride ions induce hyperkalemia mainly in 2 ways: (1) fluoride-mediated inactivation of the Na⁺/K⁺ATPase, leading to accumulation of intracellular sodium and extracellular potassium; (2) activation of Na⁺-Ca⁺⁺ ion exchanger, resulting in the intracellular accumulation of calcium.^[3] Hypokalemia has also been reported in several reports, including the study by Vohra et al, who reviewed the literature and found that hyperkalemia occurred only in cases of poisoning with sodium fluoride or sodium silicofluoride, but was rarely observed in HF poisoning.^[9] Hypokalemia is usually associated with prolonged potassium deficiency, loss of potassium, and transcellular shift, for example, through the administration of large volumes of fluids and calcium gluconate or calcium chloride. Severe hypomagnesemia can also result in hypokalemia.

Fluoride is a low-molecular-weight anion that is easily absorbed through the gastrointestinal tract, respiratory tract, skin, and mucosa. Fluoride accumulates in bone and is eliminated primarily through renal excretion. The half-life of hydrogen fluoride is only 12 to 24 hours. Thus, improvement of fluoride excretion may play a vital role in cases of fluoride poisoning because of its rapid toxicity and high rates of associated mortality. Previous studies have demonstrated that diuretic therapy and alkalization of the urine with sodium bicarbonate may increase renal elimination of fluoride ions. However,

hemodialysis or high-volume hemofiltration should be initiated in patients with severe systemic HF toxicity. There are a few reports on the use of hemodialysis in fluoride intoxication after ingestion of sodium fluoride,^[2,13] but only 1 case has shown successful performance of hemodialysis after dermal exposure to HF.^[12] Bjornhagen et al^[12] reported a patient who demonstrated repeated ventricular fibrillation without electrolyte abnormalities, in whom hemodialysis was initiated despite normal renal function after exposure to 71% HF. The hemodialysis was performed 8 hours after exposure and continued for 4 hours. Continuous veno-venous hemodialysis (CVVHD) was used at 30 hours postexposure for 48 hours. However, the concentration of serum fluoride was still 6 times above the upper reference limit. Antar-Shultz et al^[14] documented a case in which the patient received hemodialysis for 4 hours approximately 16 hours after ingestion of a mixture containing HF. In that case, the concentration of serum fluoride decreased from 688 to 36 μmol/L after hemodialysis. However, initial urine fluoride levels increased from 138 to 1376 μmol/L postdialysis. These reports show that serum fluoride concentrations can be reduced by 30% to 70% after 1 standard hemodialysis.^[12–14] Prolonged or repeated hemodialysis is recommended to avoid fluoride-related cardiac toxicity. Obviously, it is difficult to estimate how much fluoride ions are truly removed by hemodialysis, as the fluoride ions can transform rapidly into a nonionic form. Unfortunately, we could not detect serum and urine concentrations of fluoride. The extremely low level of ionic calcium (0.192 mmol/L) and repeated ventricular fibrillation in our case reflects the severity of HF burn injury in this case. We applied continuous venovenous hemodialysis (CVVHD) to the patient to further reduce fluoride concentration because of hemodynamic instability at 4 hours postexposure lasting for 72 hours. He developed sepsis approximately 72 hours after exposure due to pulmonary infection and wound infection including on the perineum, both lower extremities, and below the trunk. Therefore, we changed the treatment to continuous venovenous hemofiltration after 72 hours and continued treatment for 10 days.

Serious ARDS due to HF and nitric acid is rare. Inhalation of nitric acid causes pulmonary edema because of the generation of various nitrogen oxides and the increase of vascular permeability, which further damages pulmonary epithelial cells. HF is very volatile in its anhydrous state, and fumes may directly cause severe respiratory damage, including acute hemorrhagic pulmonary edema with acute respiratory distress syndrome. Any inhalation of HF causes a high risk of systemic toxicity regardless of the concentration of HF. The respiratory symptoms may occur several hours later, but can take up to 2 days after exposure to HF to manifest. The first line of therapy is to remove the patient from the harmful environment to a 100% oxygen environment, followed by treatment with 2.5% to 3.0% calcium gluconate, which is used to neutralize fluoride ions. Dieffenbacher and Thompson^[15] reported a patient who died 2 hours after inhalation of 10% HF for merely 30 to 35 seconds, despite the use of calcium gluconate therapy. In our case, the patient's hypoxemia worsened and his condition deteriorated rapidly 14 hours after the accident, as we had failed to increase positive pressure to sustain sufficient oxygenation. Apart from oxygenation, repeated defibrillation and/or direct cardiotoxicity may have resulted in the patient's hemodynamic instability. Considering his decreasing contractile function, we performed venoarterial (VA) ECMO not only to improve oxygenation, but also to support the patient's hemodynamic profile. As hemodynamic parameters may be affected by CRRT and/or

ECMO, hemodynamic monitoring was stopped. Bur et al^[16] tried ECMO 7 hours after admission in a case of fatal pulmonary edema after nitric acid inhalation. However, the patient's lung function improved temporarily, even though the patient ultimately died due to respiratory failure. Shin et al^[17] described a case of potential fatal pulmonary edema after exposure to HF and nitric acid fumes, where they successfully used VV-ECMO to correct hypoxemia and carbon dioxide retention at the early stages of pulmonary edema. In contrast to the other cases involving patient fatality, Shin et al suggested that ECMO should be initiated as early as possible. We applied VA-ECMO 38 hours after HF exposure, and the patient's oxygenation function and hemodynamics began to stabilize after ECMO. There is no well-characterized time at which it is ideal to initiate ECMO, but it is an effective therapy when conventional therapies fail to ameliorate oxygenation in ARDS.^[18,19] Although glucocorticoid therapy remains controversial in ARDS, glucocorticoid can up-regulate cyclic adenosine monophosphate (cAMP) to reduce the permeability of the epithelial barrier to resolve alveolar edema.^[20] The risks and complications of ECMO should be noted. These include bleeding, inflammation, and thrombosis.^[21] Here, we propose that when traditional treatments fail to ameliorate the patient's condition, ECMO should be taken into consideration as a potential treatment.

4. Conclusions

After severe exposure to HF, it is vital to correct electrolyte imbalance in a timely manner. If hemodynamic instability occurs, CRRT may be an effective and potentially life-saving therapy. Prolonged hemodialysis is recommended to prevent the delayed release of fluoride ions to avoid delayed systemic injury. When conventional therapies cannot improve oxygenation and/or carbon dioxide retention, ECMO should be performed as soon as possible.

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