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CASE REPORT

Successful extracorporeal life support after potentially fatal pulmonary oedema caused by inhalation of nitric and hydrofluoric acid fumes☆

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Calcium gluconate

Summary Two patients presented with potentially fatal pulmonary oedema after accidental exposure to nitric and hydrofluoric acid fumes during electroplating. Despite aggressive respiratory support, one succumbed to respiratory failure 3.5 h after inhalation. The other patient also rapidly progressed to respiratory failure. Extracorporeal life support (ECLS) was started 5 h after exposure at the ED. During ECLS, hypoxia improved, but pulmonary oedema shown by chest radiography became aggravated. *N*-Acetyl cysteine and calcium gluconate were given i.v. on the first day of admission and nebulised for 48 h after exposure. Pulmonary secretions were significantly reduced 24 h after the nebulising therapy began. Ultimately, the patient was discharged without serious pulmonary or neurological complications after 28 days of hospitalisation. In this case, early ECLS, nebulised antioxidant and antidote were available to treat potentially fatal pulmonary oedema after exposure to nitric and hydrofluoric acid fumes.

Introduction

Nitric and hydrofluoric acid are potent oxidants and corrosive agents. They are used in various industries, e.g., metal refining and electroplating.[1—3] The clinical symptoms of nitric acid and hydrofluoric acid inhalation range from mild irritation of the upper respiratory tract to third degree burns, pulmonary oedema, and systemic hypocalcaemia. Clinical presentation depends on the duration and intensity of exposure. Severe exposure to

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Table 1 Initial clinical and laboratory data of the patients

	37-Year-old man		43-Year-old man	
ED arrival time	11:03 a.m.		11:36 a.m.	
Blood pressure (mmHg)	86/56		140/74	
Pulse rate (pulse/min)	116		134	
Respiratory rate (breath/min)	30		28	
Arterial blood gas	11:04 a.m.	11:57 a.m.	11:37 a.m.	1:15 p.m.
pH	7.26	6.96	7.34	6.93
$PaCO_2$ (mmHg)	37	85	36	105
PaO_2 (mmHg)/Fio_2	28/0.21	54/1.0	43/0.21	51/1.0
HCO_3^- (mmol/L)	16.0	18.5	18.9	21.8
SaO_2 (%)	39	60	74.6	55.2
Hemoglobin—hematocrit	19.8 mg/dL — 60.5%		18.3 mg/dL — 52.5%	
White blood cell count	26,850 mm^{-3}		12,990 mm^{-3}	
$Na^+/K^+/Cl^-$ (mmol/L)	132/3.9/101		137/3.8/107	
Total Ca—Ca^{2+} (mg/dL)	9.9—4.5		5.8—3.5	
CO-Hb	1.2%		1.9%	
Serum lactate (mg/dL)	8.3		6.3	
Central venous pressure	0 cm H_2O		0 cm H_2O	
Amount of secretion	Approximately 2 L		Approximately 2 L	
Infusion of catecholamine	Yes		Yes	
Extracorporeal life support	No		Yes (at 1:51 p.m.)	
Outcome	Arrest at 12:25 p.m.		Survival to discharge	

Accidental exposure time was November 20, about 9:00 a.m.

nitric or hydrofluoric acid results in acute lung injury, described as non-cardiogenic pulmonary oedema and haemorrhage.[4,5] In several reports, patients with severe exposure succumbed despite aggressive support with mechanical ventilation and extracorporeal membrane oxygenation.[1,6]

We describe two cases of accidental nitric and hydrofluoric acid inhalation injury that caused serious pulmonary oedema. One patient died, while the other received extracorporeal life support (ECLS) during the early phase of acute lung injury and concomitant nebulised therapy comprised of *N*-acetylcysteine and calcium gluconate. This patient ultimately survived without neurological or serious respiratory complications.

Case report

Two men were accidentally exposed to a mixture of nitric acid and hydrofluoric acid fumes for approximately 5 min in an industrial electroplating plant. The mixture was composed of 4 volumes of 65% nitric acid to 1 volume of 55% hydrofluoric acid. Initially, they experienced a mild throat irritation. Dyspnoea occurred approximately 1—2 h later. At arrival in the emergency department both were conscious, but showed dyspnoea at rest, cyanosis, and general pallor. One of the patients, a 37-year-old man, rapidly deteriorated with massive pulmonary secretions of about 2 L during resuscitation. Despite respiratory support including mechanical ventilation with PEEP, he died 3.5 h after exposure. The other patient, a 43-year-old man, also developed rapid deterioration in respiratory function. He received both mechanical ventilation and ECLS in the emergency department. The characteristics and laboratory data of the two patients are summarised in Table 1. The sequence of events for the patient supported by ECLS, are outlined in the following paragraphs.

November 20, about 09:00 h: exposure to nitric and hydrofluoric acid fumes (Table 2).

11:35 h: Arrival at the ED. Frothy fluid issuing from nose and mouth. Expiratory stridor and intercostal muscle retraction were recorded. Pulmonary oedema was confirmed by chest X-ray. Mechanical ventilation with PEEP was started immediately with a rapidly progressive increment in FiO_2 to 100%. Calcium gluconate (20 mL, 10%) and *N*-aceylcystein (300 mg) were infused intravenously. However, the hypoxia became worse and the patient deteriorated rapidly into respiratory failure. A decision was made to place the patient on a twin pulse extracorporeal life support system (T-PLS: NewHeartbio, Seoul). A 17-F arterial and a 21-F venous catheter were inserted into the femoral artery and vein percutaneously using a modified Seldinger method.

13:50 h: ECLS was started at an initial flow rate of 3.0 L/min. Arterial oxygen saturation was 96%, cyanosis improved. During initial ECLS vital signs were; blood pressure 80 mmHg/50 mmHg and heart