Case report: Acute respiratory distress syndrome and shock caused by severe chlorine gas poisoning was successfully cured by venous-arterial extracorporeal membrane oxygenation

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Abstract

Objectives: To report a severe case of severe chlorine poisoning inducing acute respiratory distress syndrome (ARDS) and shock required venous-arterial extracorporeal membrane oxygenation (V-A ECMO). Design: Case report. Setting: Pediatric intensive care unit (PICU). Patients: A 11-year-old boy who admitted to our PICU after inhaled chlorine poisoning. Interventions: V-A ECMO. Measurements and Main Results: After inhaling chlorine, the children quickly developed hypoxia, cyanosis and unconsciousness. After high-condition mechanical ventilation, hypoxia was only slightly improved for a short time, and then deteriorated rapidly and shock occurred. The highest oxygen saturation index was 27.3, and the chest X-ray showed extensive diffuse interstitial parenchyma changes. The oxygenation and circulation was recovered quickly under ECMO. The children received methylprednisolone intravenous injection for 3 days, the pulmonary lesions basically recovered 5 days after onset. He was successfully removed from the ventilator 1 day after the successful removal of ECMO. Follow-up 3 months after discharge showed the pulmonary lesions were completely absorbed and there were no other sequelae. Conclusion: Chemical pneumonia caused by chlorine inhalation can lead to severe ARDS or even shock, but the prognosis is often good. ECMO support should be considered when conventional treatment is ineffective.

Introduction

Chlorine gas can damage respiratory tract mucosa directly. It can also produce hypochlorous and hydrochloric acid with high oxidizing potential which all cause unspecific cell damage in skin and mucosa [1]. Acute chlorine poisoning refers to a systemic disease characterized by acute respiratory damage caused by inhaling a large amount of chlorine in a short period. Mild patients usually do not need to be hospitalized, severe patients can be life-threatening due to larynx and epiglottis edema, acute respiratory distress syndrome (ARDS), often need mechanical ventilation, but rare of them need extracorporeal membrane oxygenation (ECMO) support. The overall survival rate is high [2]. Shock, a rare complication of chlorine poisoning, is reported to be caused by heart failure secondary to toxic cardiomyopathy in some reports [3, 4]. Here, we reported a severe obese child with acute chlorine poisoning whose oxygenation was unable to be improved by high-conditioned invasive mechanical ventilation (MV) and soon developed non-cardiogenic decompensated shock, and finally recovered via venous-arterial ECMO (VA-ECMO) life support without obvious sequelae.

Methods

This retrospective case report was exempt from institutional review board evaluation. Written authorization for case report publication was obtained from the patient's guardian.

Case report

A 12-year-old boy, weighing 114 kg and 170 cm in height, admitted to our pediatric intensive care unit (PICU) due to severe chlorine gas poisoning. He inhaled chlorine for about 3 minutes due to the burst of

disinfection pipe in a swimming pool 4.5 hours before admission, and showed extra breathing effort, shortness of breath and white foamy sputum when evacuated the toxic environment. Four hours before admission, he arrived at a local hospital and received budesonide atomization and oxygen inhalation, but gradually deteriorated to cyanosis, irritability and confusion. Thirty minutes before admission, he arrived at the emergency department of our hospital. The monitoring showed heart rate (HR) 130 bpm, respiration rate (RR) 30 pm, transcutaneous oxygen saturation (SPO2) 80%, blood pressure (BP) 130/80 mmHg. Physical examination showed unconsciousness, shortness of breath but regular rhythm, extensive wet rales in both lungs and warm extremities. Immediately endotracheal intubation and bag-mask positive pressure ventilation was done with pure oxygen inhalation, his $SP0_2$ gradually increased to 85%. Large amount of pink foam was sucked from tracheal cannula. He was transferred to PICU immediately after tracheal intubation.

Main auxiliary investigation after PICU admission included:

1. Chest X-ray: diffuse parenchyma and interstitial lesions in both lungs (Fig1-1).

2. Blood routine: 21.19×10^9/L of leukocytes, 88.6% of neutrophils.

3. C reactive protein 8 mg/L, procalcitonin is normal.

4. Venous blood gas: PH7.169, PCO_2 60.3 mmHg, PO_2 37.2 mmHg, SO_2 57.8%, lactic acid 4.5 mmol/L.

5. The electrolytes, liver and kidney function and blood coagulation function were nearly normal. He was diagnosed as severe chlorine poisoning, severe ARDS and inhaled toxic gaseous pneumonia. Normal frequency MV was initiated with inspired oxygen fraction (FiO2) 100%, positive end-expiratory airway pressure (PEEP) 20 cmH2O, RR 15 pm, tidal volume (VT) 500ml, peak inspiratory pressure monitored was 48 cmH2O. Methylprednisolone 2 mg/kg/d was given intravenously for anti-inflammation, midazolam/fentanyl for analgesia and sedation, and rocuronium muscle relaxation. Under such support, themonitoring heart rate was around 110 bpm, SPO_2 92%, BP 100/80 mmHg. We calculated oxygen saturation index (OSI) 27.3 instead of oxygen index (OI) due to the difficulty of arterial blood collection.

Three hours after admission, the child's BP suddenly dropped to 72/50 mmHg, SPO_2 75%, with HR rose to130 bpm, adrenaline 0.8 ug/kg/min was given to increase blood pressure, but with poor effect. Bedside transthoracic echocardiography was done immediately by some senior PICU doctors, but we failed to get a clear image, which may be related to the higher condition of MV, extreme obesity and operators' experience. Since the child's oxygenation couldn't be maintained under routine treatment and combined with unstable circulation, we prepared to undergo V-AECMO life support.

Seven hours after admission, the catheterization through the right internal jugular vein and jugular artery was completed by cardiac surgeon due to difficulty of catheterization in the femoral vein, and the VA-ECMO was initiated with rotational speed 3000 rpm, blood flow 3L pm, airflow 5L pm, FiO_2 100%. The $SPO_2$of the child rose to 98%, then we successfully down-regulated the MV parameters to FiO_2 60%, PEEP 12 cmH2O, RR 20 pm, VT 300 ml. X-ray showed pulmonary transmittance was greatly elevated (figure 1-2). The circulation condition was improved significantly to BP 116/80 mmHg and stable pulse pressure gap. Bed side cardiac ultrasound performed by our certified ultrasound doctor showed normal left ventricular systolic function and the adrenaline was successfully withdrawn within 2 hours. 15 hours after admission, the patient presented with continuous oliguria accompanied by progressive increase of urea nitrogen and creatinine, and was dealt with continuous renal replacement therapy (CRRT).

On the second day of admission, MV's FiO_2 was down-regulated to 40%. On the 3rd day of admission, the urine output recovered and urea nitrogen and creatinine decreased gradually, we stopped CRRT successfully. On the 5th day of admission, the chest X-ray showed that the pulmonary lesions were mostly absorbed (figure 1-3), and the vital signs were stable. The ECMO was removed when the ventilator was set to FiO_2 60%, PEEP 12 cmH2O, VT 450 ml, RR 20/min. After the removal of ECMO, the child presented a transient aggravation of pulmonary exudation (figure 1-4) with SPO_2 decreased to 90%, his oxygenation gradually stabilized after the PEEP was increased to 16cmH2O and furosemide intravenous injected at a dose of 1mg/kg.

On the 7th day after admission, we down-regulated the PEEP to 6 cmH_2O, FiO_2 to 40%. The child's OI was 3.68 with normal spontaneous breath and clearconscious, and the pulmonary exudation was nearly absorbed (Fig. 1-5). After MV was successfully removed, he was supported by bi-level non-invasive positive pressure ventilation for 12 hours, and then we gave him nasal catheter. Fiberoptic bronchoscopy was performed before withdrawing the ventilator, which showed inflammation of tracheobronchial intima and poor ventilation in some subbranches of the left lower basal segment. Enteral feeding was started from 5% glucose on the same day.

From the 8th day of admission, the child gradually weaned off oxygen inhalation and could get out of bed and do some physical exercise. The lung CT showed exudation lesion was absorbed (Fig 1-6 and 1-7), while the pulmonary function showed mild mixed ventilatory dysfunction may be due to his extreme obesity. The transcranial doppler, brain magnetic resonance image, cervical vascular ultrasound and cardiac ultrasound were all normal. During the recovery period of enteral feeding, a transient pancreatic injury occurred with amylase and lipase increased to 18U/L and 195.5 U/L respectively, they all returned to normal after several days' somatostatin infusion. During the 2nd-4th day after admission, the child was given nitroprusside and nicardipine pump successively due to the increase of BP (up to 170/100 mmHg) for unknown reason. His BP returned to normal on the 5th day of admission with no reliance on long-term oral antihypertensive drugs.

The child hospitalized in our hospital relatively longer due to the incision fat liquefaction and discharged one month after admission. The lung CT in the 2 month's follow-up showed no abnormality (Fig.1-8). There were no sequelae in the follow-up 3 months after admission, his physical ability returned to the basic level, and could do physical exercise as before.

Discussion

Most chlorine poisoning onsets in where chlorine-containing disinfectants are used, such as chemical plants and swimming pools. The treatment mainly includes conjunctival cleaning, high PEEP mechanical ventilation, bronchiectasis drugs inhalation, glucocorticoid administration and so on. Rare patients need ECMO support. The prognosis is usually good with few sequelae [1, 2]. The case we described has the following characteristics: Complicated with shock and acute renal injury. VA-ECMO was established instead of the common chosen veno-venous ECMO (VV-ECMO).

Shock develops in 23-50% of severe ARDS patients due to acute corpulmonale (ACP) [5, 6]. In a large-scale prospective study by Mekontso et al.[7], the four independent risk factors for ACP in ARDS were ARDS caused by pneumonia, driving pressure>18 cmH_2O, PaO_2/FiO_2<150 mmHg and $PaCO_2$>48 mmHg. The main pathogenic mechanisms is a sharp rising in pulmonary arterial pressure and pulmonary circulatory resistance caused by hypoxic/hypercapnia pulmonary vasoconstriction, interstitial edema compression on pulmonary vessels, high PEEP ventilation and so on. Clinically, acute right heart failure can occur due to high right ventricular after load, followed by a decrease in pulmonary blood flow that aggravates hypoxia and hypercapnia, and leads to a significant decrease in blood pressure. Bedside echocardiography is very important in the early diagnosis of ACP and the continuous monitoring of cardiac function in children [8, 9]. Transthoracic echocardiography is non-invasive, but sometimes can hardly get clear images in obese and mechanically ventilated patients. Transesophageal echocardiography is invasive, but the image is clear. The child we discussed had high risk factors for ACP, while no pathophysiological basis of distributed shock or hypovolemic shock; after VA-ECMO, his pulse pressure difference was normal, and the cardiac ultrasound proved normal left ventricular function, his troponin was normal too, so cardiogenic shock could be excluded; chest X-ray and coagulation test could also exclude tension pneumothorax and pulmonary embolism. Therefore, we considered as obstructive shock caused by ACP. Unfortunately, transthoracic ultrasound before VA-ECMO failed to get clear images, and our ICU was not equipped with transesophageal probe, so objective evidence for the diagnosis of ACP caused by ARDS hadn't been obtained.

ECMO should be considered in patients with ACP caused by ARDS who can't be maintained by routine treatment. VV-ECMO can improve oxygenation and acidosis, reduce carbon dioxide, and then reduce pulmonary circulatory resistance, several reports have reported successful application of it on patients with

ACP caused by ARDS duetoacute poisoned gasinhalation [1, 10]. VA-ECMO can significantly reduce blood flow in pulmonary circulation, which is more beneficial to reduce pulmonary exudation during ARDS, and can immediately reduce the pre- and after load of right ventricular during ACP so as to improve hemodynamics. But VA-ECMO will affect left cardiac function, reduce coronary oxygen supply, and cause different limb and cerebral perfusion according to the location of arterial catheterization. Morbidities are relatively more, and may cause myocardial injury when used for a long time. The ECMO support time required for ARDS is generally longer, so VV-ECMO is more commonly used. For this patient, VA-ECMO was chosen because the possibility of cardiogenic shock couldn't be excluded before ECMO establishment and the existence of extremely unstable hemodynamics. The severe hypotension and hypoxia were corrected quickly after VA-ECMO so as the circulatory condition. Acute kidney injury occurred a few hours after hypotension due to insufficient perfusion, but returned to normal in 2 days, which may also be related to the rapid correction of hemodynamic abnormalities by VA-ECMO. For patients with ARDS complicated with ACP, it suggests that the duration of lung injury caused by the primary disease is expected to be short and complicated with severe hemodynamic disturbance, VA-ECMO might be considered first. Temporary aggravation of pulmonary exudation after withdrawal of VA-ECMO suggests that in children with ARDS treated by VA-ECMO, the PEEP and volume load should be properly adjusted before a dafter weaning.

Chlorine inhalation can cause chemical aspiration pneumonia. Severe ARDS, even shock can occur in the acute stage, but the prognosis is mostly good. Therefore, if conventional treatment is ineffective and patients do not have severe underlying diseases, ECMO should be considered promptly. Transesophageal probe had better be equipped in advanced ICUs to facilitate the completion of bedside cardiacultrasound in special patients.

Proofread Report

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Address of above authors: Pediatric Intensive Care Unit, Beijing Children's Hospital, Capital Medical University, National Center for Children's Health, No.56 Nan-Li-Shi Road, Beijing, 100045, China.**Figure Legend**Figure 1. Patient's x-ray and CT scan imaging studies. 1:extensive and diffuse interparenchymal changes in both lungs before ECMO; 2:the transmittance of both lungs increased after ECMO initiation; 3:the lesions of both lungs were basically absorbed before ECMO withdraw; 4:the exudation of the lungs was aggravated temporarily after ECMO withdraw; 5:before the removal of invasive ventilator, the pathological changes in the lungs were basically absorbed; 6:lung CT in the recovery stage after weaning from the invasive ventilator; 7:pulmonary lesions were basically absorbed 2 weeks after the ventilator withdraw; 8:follow-up CT in 1 month after discharge showed nearly normal lungs;