

Extinguishing the Fire Within: Dry Powder Fire Extinguisher Ingestion in a Critical COVID-19 Patient

Poster
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INTRODUCTION

Exposure of dry powder in fire extinguisher can occur accidentally or intentionally because it is equipped everywhere, for public use. Respiratory tract is a common route of exposure, causing pulmonary toxicity. In addition, systemic toxicity occurs via absorption. Even though fire extinguisher inhalation accidents and the systemic complications are rarely reported, it can be fatal. As mild respiratory symptoms such as cough and sore throat occur initially, primary physicians could disregard it as common cold, delaying proper treatment. Furthermore, life threatening conditions such as seizures, cardiac arrhythmias, acidosis, and cardiac arrest can develop. We report a case of dry powder fire extinguisher inhalation, which contained monoammonium phosphate (MAP) as its active ingredient. This is further complicated by the presence of Coronavirus-disease-2019 (COVID-19).

CASE REPORT

A 43-year-old man with underlying schizophrenia and concomitant methamphetamine use disorder was brought into the Emergency Department 2 hours after ingesting dry powder fire extinguisher, as shown in Figure 1. He presented with episodes of persistent vomiting consisting of yellowish substance and food particles. There was no haematemesis. He also had difficulty breathing. Patient was compliant to his medications.

On arrival, the patient was alert but tachypnoeic with a respiratory rate of 45 breaths per minute, and oxygen saturation of 87% under room air. His blood pressure was 160/70 mmHg, and heart rate was 112 beats per minute. His arterial blood gas on high flow oxygen 15 litres per minute showed type 1 respiratory failure with severe high anion gap metabolic acidosis (HAGMA) as shown in Figure 2. He had mild hyponatraemia (Na) and hypokalaemia (K). His calcium (Ca) and magnesium (Mg) were of normal range. However, there was marked hyperphosphatemia (PO₄). Otherwise, he recorded no acute kidney injury (AKI) at presentation. Chest radiograph (CXR) showed right lower zone consolidation [Figure 3].



Figure 1: Dry Powder Fire Extinguisher

Parameters	Results
pH	6.9
pCO ₂	23 mmHg
pO ₂	96 mmHg
HCO ₃	7.6 mmol/L
BE	-27 mmol/L
Na	130 mmol/L
K	3.2 mmol/L
Ca	2.28 mmol/L
Mg	0.9 mmol/L
PO ₄	4.9 mmol/L
Urea	2.8 mmol/L
Creatinine	73 µmol/L

Figure 2: Laboratory Investigation Results

Patient had persistent yellowish powdery vomitus [Figure 4]. Oral suction failed to remove the clumping powdery substance in his oral cavity. Otolaryngology team was consulted and an immediate flexible naso-pharyngo-laryngoscopy showed no obstructing airway with non-oedematous vocal cord, epiglottis, and arytenoids. However, yellowish powder was seen in the pharynx. Patient then was decided for intubation. He was positioned in bed-up, head elevated to 30 degrees. Apnoeic pre-oxygenation was used. There was moderate yellowish secretion, and suction-assisted laryngoscopy and airway decontamination method was used. The percentage of glottic opening was 100%, and intubation was done via video laryngoscope with first pass success.

As COVID-19 rapid antigen test came back positive, diagnosis was revised to dry powder fire extinguisher ingestion complicated with critical COVID-19 (category 5), severe type 1 respiratory failure, and severe HAGMA. Post-intubation, patient was connected to the ventilator with a combination of lung-protective and metabolic acidosis ventilator strategy. Patient was admitted to the intensive care unit (ICU).

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CASE REPORT (continued)

He subsequently developed AKI with worsening metabolic acidosis, and was put on continuous veno-venous haemodialysis (HD). He also developed ventilator-associated pneumonia (VAP) while in ICU. 3 weeks later, he was successfully extubated, and discharged the following week.

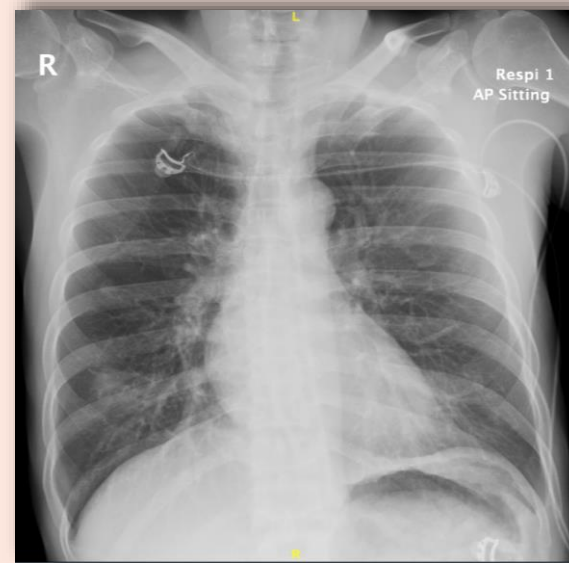


Figure 3: Pre-intubation Chest Radiograph



Figure 4: Yellowish Powdery Vomitus

DISCUSSION

MAP, a white crystalline powder, is the major ingredient of dry powder in fire extinguishers. MAP can be absorbed via respiratory tract and gastrointestinal tract. When MAP is metabolised, PO₄ is released as intermediate product. The process causes elevation of serum PO₄ level that results in AKI from PO₄ nephropathy and metabolic acidosis.

Although exposure to MAP is not common, anyone who uses fire extinguisher or people who are at a scene of fire can be intoxicated by MAP via gastrointestinal tract or respiratory tract. In this case, exposure via respiratory tract led to acute toxicity when fire extinguisher was sprayed. Innumerable fine particles which floated in the air could readily have entered the respiratory tract. The absorption of MAP might occur through the respiratory tract in a significant manner due to the high concentration of MAP in the spray. Linear relationship between serum PO₄ level and mucosal PO₄ concentration has been reported [1].

Biochemical findings in this patient was consistent with a case report in Hong Kong, consisting of hyponatraemia and severe metabolic acidosis [1]. This is due to MAP that is metabolised into PO₄ causing severe AKI with metabolic acidosis. In a case report from Iran, quite the opposite occurs as the authors reported metabolic alkalosis and hypernatremia. This is possibly because the reported type of dry powder fire extinguisher consisted of sodium bicarbonate, potassium bicarbonate, urea bicarbonate, and potassium chloride suitable for extinguishing Class B and C fire [2]. It is interesting to note that both our patient and the Hong Kong patient survived this toxicity while the Iranian patient succumbed. Perhaps a study of prognosis between different types of dry powder fire extinguisher inhalation can be proposed for future research.

Apart from nephropathy and metabolic acidosis, MAP inhalation also causes acute respiratory distress syndrome (ARDS). A Norwegian case reported that fire extinguisher powder was found adhered to the airways post-inhalation, where it was postulated to be the cause of ARDS [3]. This is comparable in our case where yellowish powder was found in patient's pharynx. Another similar case in Japan proposed that the pathophysiology of lung injury in fire extinguisher exposure is akin to that of pneumoconiosis [4].

As no antidote for MAP exists, treatment is mainly supportive. Hypoxia in this patient is multifactorial; both from MAP and COVID-19, causing ARDS. Thus, early intubation is necessary, and ventilation should take into consideration both metabolic acidosis and ARDS strategies. HD should also be initiated early for extracorporeal toxin clearance.

CONCLUSION

MAP toxicity is debilitating, further exacerbated by concomitant COVID-19 infection. Clinicians should have a high index of suspicion for systemic toxicity when dealing with MAP.

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DECLARATION OF CONFLICT

The authors do not have any conflicts of interest to declare.