CASE STUDY: Cinnamon Aspiration in a Toddler Causing Severe ARDS Requiring Surfactant and Extracorporeal Membrane Oxygenation

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Abstract

As many as 6% of reported cinnamon poisonings cause significant clinical effects, however descriptions of pulmonary toxicity have not yet been reported. Here, we present a pediatric patient's hospital course following powdered cinnamon aspiration. The early presentation with hypercapnia and lower airways obstructions evolved to hypoxemic respiratory failure and severe pediatric acute respiratory distress syndrome requiring a 7-day course of veno-venous extracorporeal membrane oxygenation, 16 ventilator-days, and 3 diagnostic and therapeutic bronchoscopies with 2 applications of surfactant therapy. The sum of these modalities contributed to this patient's survival and subsequent return to respiratory baseline 6 months post-hospitalization.

Introduction:

Reports of the pulmonary sequelae of cinnamon aspiration are absent despite cinnamon's ubiquity and the popularity of "the cinnamon challenge" around 2012[1]. Nevertheless, our review of the National Poison Data System (2009-2018) found that cinnamon poisonings lead to "moderate" (e.g. wheezing, moderate shortness of breath) or "major" (e.g. respiratory/cardiac arrest) effects in 5.9% and 0.11% of cases, respectively. Here, we detail a case of cinnamon-induced respiratory failure requiring extracorporeal membrane oxygenation (ECMO) and therapeutic bronchoscopies for survival.

Case Presentation:

A previously healthy 2 year-old male was transported from home to our hospital following an unwitnessed powdered cinnamon aspiration. He was described as mottled and wheezing with visibly increased work of breathing. He was hypoxemic (SpO_2 55%) in ambient air, necessitating escalation to non-invasive bi-level positive pressure ventilation and continuous albuterol nebulization. His first blood gas analysis, from a venous sample, found significant respiratory acidosis (pH 6.98, pCO_2 112 mm Hg). After increasing respiratory support, an arterial sample revealed persistent respiratory acidosis (pH 7.19, pCO_2 65.2 mm Hg) without significant metabolic contribution (bicarbonate 26 mmol/L, anion gap 14 mmol/L, lactate 1.26 mmol/L) and impaired oxygenation (pO_2 66.2 mm Hg on 100% FiO_2). Upon transfer to the PICU, he underwent endotracheal intubation and initiation of synchronized intermittent mandatory ventilation. His chest radiograph revealed lung hyper-expansion and bilateral patchy opacities sparing the apices (Figure 1A), and his ventilator scalars were consistent with increased airway resistance and lower airways obstruction. Intravenous methylprednisolone (loading dose of 2 mg/kg followed by 4 mg/kg/d) was initiated. By hospital day (HD) 4, his ventilator scalars demonstrated resolution of lower airways obstruction, and his hypercarbia

improved, but his oxygenation index (OI) rose to the mid-20's (Figure 1D). We transitioned him to airway pressure release ventilation (APRV) and started inhaled nitric oxide.

The first diagnostic flexible bronchoscopy with lavage (30 mL of normal saline instilled in total) was performed on HD 6. The airways of the left lung appeared more heavily deposited than the right, with moderate amounts of brown debris in all of the left lobes. The mucosa appeared normal, but with mild friability after the lavage. Analysis of the lavage fluid found increased white blood cells with a neutrophilic predominance and the moderate presence of lipid-laden macrophages; no analysis could be performed on the debris.

A chest radiograph attained just prior the bronchoscopy was later interpreted as showing pneumomediastinum and subcutaneous emphysema. Given worsening oxygenation (OI 32), the presence of air leak, ongoing intermittent obstructions of the endotracheal tube with mucoid cinnamon, and the need for additional bronchoalveolar lavages, the patient was deployed at bedside onto veno-venous extracorporeal membrane oxygenation (VV-ECMO, Figure 1B and 1D). On HD 7, he developed a new pneumothorax requiring chest tube placement.

He underwent repeated bronchoscopies on HD 9 and HD 10 with the goal of debris removal through high volume, total left lung lavage (120-150 mL normal saline instilled in total per procedure). At that time, the left lung demonstrated significantly worse mucosal inflammation than on HD 6, with a large quantity of visible debris, most heavily deposited in the left lower lobe (Figure 1E). After each lavage, we instilled surfactant (1.25ml/kg and 0.625ml/kg on HD 9 and HD 10, respectively). Each bronchoscopy was accompanied by an immediate worsening in oxygenation followed by slow, steady improvement; this was most dramatic on HD 10, with increasing lung compliance and tolerance of extracorporeal oxygen blender weaning. The patient was decannulated on HD 14, extubated on HD 18, and transferred on HD 28 to a sub-acute inpatient rehabilitation program, requiring no supplemental respiratory support. He completed the program and is now at home. At a pulmonary medicine follow-up visit 6 months after the aspiration, his chest radiograph demonstrated persistence of patchy opacities in the right lung base, with no abnormalities on the left (Figure 1C), which had directly received the bronchoalveolar lavages and surfactant. He was doing well, with no ongoing respiratory symptoms or need for respiratory support or medications.

Discussion:

The initial response to cinnamon aspiration in our patient appeared to be consistent with lower airways obstruction, possibly bronchospasm. As the air-trapping resolved, his oxygenation defect worsened, consistent with V/Q-mismatch and increased oxygen diffusion barrier. Moreso complicating his already precarious condition was the frequent plugging of his endotracheal tube with tenacious secretions and the onset of pulmonary air leak. Thus, in our case, deployment of ECMO was necessary for continued gas exchange.

The cellulose fibers of cinnamon can be retained for years[2] and appear to elicit an acute inflammatory cell infiltration as well as a chronic, fibrosing granulomatous alveobronchiolitis[3-4]. Thus, in addition to other external and intrapulmonary percussive therapies, we relied on bronchoscopies for enhanced airway clearance while on ECMO. Given the apparent worsening in mucosal inflammation between the first and second bronchoscopies, and the return of cinnamon with each lavage, we may consider earlier and more frequent lavages for similar aspirations in the future, but with cautious anticipation that each session may cause temporary worsening in oxygenation.

We also opted to provide this patient with surfactant therapy following each high-volume lavage. It is presumed to help replace iatrogenic surfactant deficiency caused by total lung lavage, while another report employed this strategy to aid in the removal of a poorly water-soluble, powdered material[5]. That said, the contribution of surfactant therapy alone to the clinical and/or radiographic improvement in our patient is indeterminate, and more research is required before the practice can be widely adopted.

Conclusion

Pediatric cinnamon aspiration leading to severe respiratory failure requiring ECMO has not been previously reported. In our case, we supported the patient with VV-ECMO to allow the lungs to recover while pro-

viding repeated directed bronchoalveolar lavages with surfactant administration. The short-term functional outcome at 6 months appears to be good, but our patient needs ongoing evaluations for long-term sequelae. Aspects of our approach warrant consideration when treating severe respiratory failure from the aspiration of cinnamon and similar agents.

Ethical Considerations:

Two-physician witnessed consent was provided by the patient's legal guardian specifically for publication of this report.

References:

[1] Grant-Alfieri A, Schaechter J, Lipshultz SE. Ingesting and aspirating dry cinnamon by children and adolescents: the "cinnamon challenge". Pediatrics. 2013;131(5):833-835.

[2] Muhle H, Ernst H, Bellmann B. Investigation of the durability of cellulose fibres in rat lungs. Ann Occup Hyg. 1997 ;41(supp1):184–188

[3] Tátrai E, Adamis Z, Ungváry G. The pulmonary toxicity of cinnamon dust in rats. Indian J Med Res. 1995;102:287–92.

[4] Adamis Z, Tátrai E, Honma K, Ungváry G. In vitro and in vivo assessment of the pulmonary toxicity of cellulose. J Appl Toxicol. 1997 Mar-Apr;17(2):137-41.

[5] Matina F, Collura M, Maggio MC, et al. Inhaled surfactant in the treatment of accidental talc powder inhalation: a new case report. Ital J Pediatr. 2011;37:47.

FIGURE LEGEND:

Figure 1. Chest radiographs, clinical metrics, and bronchoscopic images after cinnamon aspiration. Radiographs were taken (A) on hospital day 1 at approximately 12 hours after admission, (B) on hospital day 6 after deployment of ECMO, and (C) on outpatient follow-up approximately 6 months after admission. (A) The radiologist's interpretation included findings of hyperexpanded lungs with patchy right lower and left retrocardiac opacities, with a possible small left pleural effusion. (B) At the time of ECMO initiation, the radiograph revealed evolution of the bilateral lower lobe opacities, pneumomediastinum, and pneumopericardium; no pneumothorax was present at this time. (C) Upon outpatient follow-up, the radiologist reported findings of persistent patchy opacities in the right lower lobe, with resolution of any obvious radio-opaque findings on the left. Of note, an incidental finding of a left anterior six rib contour abnormality was also noted on all films. In (D), the patient's oxygenation index (OI), pCO_2, and minute ventilation are depicted over the period during which the patient was invasively ventilated. The duration of venovenous extracorporeal membrane oxygenation (VV-ECMO) is shaded in pink. The vertical dotted lines indicate days of three bronchoscopies. Attained on HD 9 (E), bronchoscopic images demonstrate occlusion of the airways with brown debris and significant mucosal erythema.

Figure 1