Development of an esophageal stricture following paradichlorobenzene mothball ingestion

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Abstract
A 2-year-old patient presented with a paradichlorobenzene mothball ingestion. The foreign body was removed with a straight Miller blade and a curved Allis clamp. Two weeks following the removal of the mothball, the patient developed solid food dysphagia due to an esophageal stricture. This complication after a short exposure to mothballs is unreported in the literature to date, making this a unique and interesting case. In addition, a comparison of the clinical presentation and treatment of naphthalene and paradichlorobenzene mothballs was reviewed in this article.

Keywords
Paradichlorobenzene, naphthalene, mothball, dysphagia, esophageal stricture

Introduction
Mothballs are composed of either naphthalene or paradichlorobenzene. Paradichlorobenzene is used in both household and industrial products.1 Paradichlorobenzene is more benign but less well known of the two chemicals.2 In addition to mothballs, it can be found in toilet bowl fresheners, household deodorants, and pesticides.1 Paradichlorobenzene ingestion has been associated mostly with neurological symptoms such as central nervous system (CNS) depression,1 depression,3 and skin changes.4–6 Naphthalene similarly is found in mothballs, pesticides, soil fumigant, lavatory deodorants, and in industrial products and waste materials.7 Naphthalene use in products is under strict control by the US Environmental Protection Agency, and very few products with this chemical are approved.8 In certain states like California, the use of naphthalene in pesticides has been banned since 1992.8 Naphthalene has been associated mostly with hematological disorders.2 Individuals with glucose-6-phosphate dehydrogenase (G6PD) deficiency are at most risk for methemoglobinemia and hemolysis when exposed to naphthalene.8 Typical exposure is via inhalation, dermal contact, or ingestion.7 Other toxic effects are to the eyes, lungs, kidney, brain, and liver.7 It can also cause cataracts, and it is carcinogenic.7,8 To date, there has not been a report of esophageal strictures post either paradichlorobenzene or naphthalene ingestion.

Case report
A 26-month-old male was airlifted to our hospital after ingesting a mothball. Manual removal of the mothball at home was unsuccessful. He soon exhibited respiratory distress, coughing, foamy sputum, drooling, and vomiting. He was taken to the local emergency center and subsequently airlifted to the local children’s hospital. During transport, he had hypoxia requiring 10 L on nonrebreather mask. If the oxygen was removed, his pulse oximetry immediately dropped to 83% on room air. He was pale, lethargic, and responsive only to painful stimuli. A chest radiograph showed a foreign body in the esophageal inlet with compressive effects on the trachea. Initial arterial blood gas showed pH of 7.34, CO2 of 43, O2 of 105, and bicarbonate of 22.8.

The patient was intubated and underwent emergent endoscopy, during which the mothball was identified in the upper esophagus. A straight Miller blade and a curved Allis clamp were used for removal. Two weeks following the removal of the mothball, the patient developed solid food dysphagia due to an esophageal stricture. This complication after a short exposure to mothballs is unreported in the literature to date, making this a unique and interesting case. In addition, a comparison of the clinical presentation and treatment of naphthalene and paradichlorobenzene mothballs was reviewed in this article.

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esophagus and was exerting a compressive effect on the trachea (Figure 1(a) and (b)). Several unsuccessful attempts were made (using a Roth Net® retrieval device endoscopically (STERIS Healthcare, Mentor, OH); using a Parsons laryngoscope and a 4 × 30 rigid esophagoscope and peanut forceps/alligator forceps; using a Boston Scientific CRE™ Pro Wire Guided Esophageal Balloon dilation catheter inflated to 10-French and introduced over the rigid esophagoscope distal to the foreign body and pulled back; and using a Rosen needle and tonsil clamps). Removal was difficult because the foreign body was too slippery, and the esophageal tissue was tightly adhered to the mothball. Ultimately, successful removal was accomplished using a straight Miller blade to locate the mothball and removal with a curved Allis clamp. Post removal, endoscopic esophageal exploration with an Olympus™ GIF-160 endoscope demonstrated minimal mucosal erosions and no perforations (Figure 1(c)). A nasogastric tube was placed at the completion of the procedure.

The patient was admitted to the pediatric intensive care unit (PICU) and remained intubated for 24 h due to concern for airway cricopharyngeal edema. He was given a 3-day course of dexamethasone, kept nil-per-os (NPO) for 24 h after the procedure, and put on famotidine intravenously. He was extubated to room air. Esophagram was normal without any signs of perforation or strictures. Clear liquid diet was started the next day and advanced to a regular diet. The rest of the hospital course was unremarkable, and he was sent home. The mothball was sent to pathology for evaluation and was found be an ovoid firm, tan-white object measuring 1.7 cm in diameter and composed of paradichlorobenzene.

Two weeks following hospital discharge, he returned to clinic due to solid food dysphagia. Repeat endoscopy identified a stricture in the proximal esophagus at the location where the mothball had come into contact with the mucosa (Figure 2(a)). The stricture was serially balloon dilated (Figure 2(b)), and triamcinolone was injected into the four quadrants of the stricture area. At a follow-up appointment, he had improved and able to tolerate solid foods. A third endoscopy showed resolution of the stricture (Figure 2(c)).

**Discussion**

Paradichlorobenzene and naphthalene are aromatic compound hydrocarbons which are toxic and lipid soluble.9 It is clinically useful to know which substance was ingested, as

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**Figure 1.** (a) Mothball at esophageal inlet. (b) Close-up of mothball. (c) Upper esophageal mucosa showing minimal damage post removal of mothball.

**Figure 2.** (a) Esophageal stricture. (b) Esophagus post balloon dilation. (c) Repeat endoscopy in 1 month demonstrating resolution of stricture following balloon dilation.
paradichlorobenzene is less toxic than naphthalene and has a different mechanism of action. It may also be inhaled and creates depression CNS effects. It has abusive potential, and there are some cases of addiction reported in the literature. In the setting of chronic ingestion, some case studies have reported withdrawal symptoms. Neurological symptoms with chronic exposure may include ataxia, limb weakness, bradyphrenia, gait ataxia, tremor, and dysarthria. Another case study described a 21-year-old female who developed leukoen cephalopathy after ingestion of one to two mothballs per day for 7 months. Furthermore, a report linked chronic paradichlorobenzene mothball ingestion with the development of clinical depression.

In addition to neurological symptoms, generalized hyperpigmented pruritic skin rash due to months of chronic exposure has been reported. Another case study described a 50-year-old woman with chronic mothball ingestion who presented with hyperpigmented acanthotic and ichthyosisiform plaques. Symptoms resolved 3 months after discontinuation of the mothball ingestion. Skin findings have also been reported in children, and mainly present as ichthyosis-like dermatosis with associated neurological symptoms.

In the acute setting, high doses of paradichlorobenzene may cause hepatic, pulmonary, renal, and dermatological effects. Aplastic anemia, hepatic insufficiency, acute renal failure, and pulmonary granulomatosis have been reported. Ingestion of paradichlorobenzene may also lead to hematologic effects as seen in naphthalene ingestion described below.

A thorough history is critical to identifying patients with acute or chronic paradichlorobenzene exposure. Paradichlorobenzene toxicity can be detected in both urine and blood. The metabolite detected in the urine is 2,5-dichlorophenol, while the toxin detected in the serum is p-dichlorobenzene. A fat biopsy may also be performed as paradichlorobenzene is highly lipophilic.

Naphthalene can be absorbed via dermal or oral routes or inhaled. It is metabolized hepatically, and its effects are mostly hematologic. Heinz bodies may be seen on blood smear as well as red cell fragmentation, anisocytosis, and poikilocytosis. Labs may also reveal leukocytosis, reticulocytosis, and anemia. One case study reported hemolytic anemia as a presenting sign of naphthalene toxicity. Toxicity is due to production of free oxygen radicals (metabolites like alpha-naphthol), which lead to oxidative stress and ultimately hemolysis and hemoglobinuria. Patients may also acutely develop fever, abdominal pain, nausea, diarrhea, lethargy, icterus, pallor, convulsions, dark urine, and vomiting. As previously mentioned, there are certain populations such as those with G6PD deficiency who are at greater risk of severe hemolysis and methemoglobinemia after exposure.

Unfortunately, few treatments exist for either paradichlorobenzene or naphthalene ingestion. In both cases, management is supportive unless there is an underlying problem such as methemoglobinemia. Treatment of anemia and methemoglobinemia may include blood transfusion, N-acetylcysteine, and methylene blue. Cessation of the exposure is important. Patients require close follow-up for chronic complications such as hepatic, pulmonary, neurological, hematologic, and renal effects.

Although systemic side effects of mothball ingestion have been well described, there are no reports of esophageal strictures or the mechanism of esophageal injury after paradichlorobenzene mothball ingestion. However, there are reports of esophageal mucosal injury by other caustic ingestions, such as acid and alkaline solutions. Acid ingestions can cause coagulative necrosis, and eschar formation that prevents deeper damage of the tissue. In comparison, alkaline ingestion causes liquefactive necrosis, saponification of fat, creating a gelatinous substance that allows further penetration, and full-thickness lesions. Similarly, button batteries create a high pH exposure and mucosal injury due to hydroxide radical formation. Button batteries have the ability to cause esophageal strictures and perforation even after a 2-h exposure, whereas a 24-h exposure of a blunt object rarely cause strictures. Development of an esophageal stricture after the mothball ingestion was not expected because the object is a blunt object and the duration of the exposure was short. From the time of ingestion, to the transfer via helicopter, and to the operating room was approximately 3 h. Noninvasive techniques were used first, but were unsuccessful because the mothball had adhered to the esophageal mucosa. Significant force was not used with the other techniques due to concern for perforation. The straight Miller blade to locate the mothball and removal with a curved Allis clamp was the best technique for this object. The endoscopic picture post removal did not show significant damage, and therefore should not have caused an esophageal stricture. What is alarming is that our patient developed solid food dysphagia and an esophageal stricture 2 weeks after such a short exposure to the paradichlorobenzene mothball. Potentially, there may be a caustic mechanism resulting in liquefactive necrosis mimicking an alkaline full-thickness tissue damage. Further studies are needed to identify the true mechanism of injury of a paradichlorobenzene mothball ingestion in the esophagus.

Conclusion

We report a young child who ingested a paradichlorobenzene mothball and subsequently developed an esophageal
stricture after a very short exposure. A straight Miller blade to locate the mothball and removal with a curved Allis clamp was the best technique for this object. To date, this is an unreported complication in the literature, and the pathomechanism of injury is unknown. Close clinical follow-up is critical in patients who have ingested mothballs to monitor for complications.

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Ethics approval
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Informed consent
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