Turpentine-induced Chemical Pneumonitis with Broncho-pleural Fistula

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Abstract

Turpentine is a volatile hydrocarbon used in polishes, solvents, paints and textile industry. When hydrocarbons are aspirated into the lung, they cause chemical pneumonitis, acute respiratory distress syndrome (ARDS), and rarely pneumatoceles and pneumothorax. We report a 20-year old boy with turpentine-induced chemical pneumonitis that evolved into a bronchopleural fistula. He was treated with oxygen, steroids and intercostal tube drainage. This is the first reported case of turpentine-associated bronchopleural fistula.

INTRODUCTION

Hydrocarbons are organic compounds that cause pulmonary toxicity either by direct aspiration into the lungs or via inhalation. Turpentine is a pinewood distillate and belongs to a separate class of hydrocarbons called terpene derivatives.1 Hydrocarbons commonly cause a chemical pneumonitis in the lung. Rarely this evolves into a pneumatocele which ruptures causing a pneumothorax.2 We report a case of left bronchopleural fistula and pneumothorax following accidental turpentine ingestion.

CASE REPORT

A 20 year old boy presented to the accident and emergency department with breathlessness and vomiting following accidental ingestion of unspecified quantity of turpentine at his lubricant manufacturing factory. At the time of admission, he had a respiratory rate of 30/min, blood pressure of 120/80 mm of Hg and a pulse rate of 88/min. He was afebrile and not cyanosed. His systemic examination was unremarkable except for a few rhonchi on auscultation of the chest. The chest X-ray showed bilateral patchy consolidation in the mid and lower zones (Fig. 1). The arterial blood gases on FiO2 of 0.35 showed pH - 7.308, paO2 - 65.8 mm of Hg, paCO2 - 41.7 mm of Hg, bicarbonate of 20.4 meq/L and an O2 saturation of 90%.

The sputum Gram stain showed occasional Gram-positive cocci and plenty of inflammatory cells (> 25 PMN/high power field). The culture was negative, suggesting a chemical pneumonitis. He was treated with nebulized salbutamol and antibiotics. On day 5, he complained of increased breathlessness and the chest X-ray showed bilateral effusions and an increase in opacities. Ultrasound of the chest revealed 108 ml of fluid on the right side and 198 ml on the left. His oxygen saturation dropped to 78%. He was started on glucocorticoids - initially intravenous dexamethasone followed by oral predinsolone in the dose of 30 mg daily for a
total duration of seven days. The patient’s breathlessness improved over the next 48 hours and oxygen saturation returned to normal. On the 12th day of admission, the patient developed acute breathlessness and had findings of a left sided pneumothorax (Fig. 2). On auscultation he had amphoric breath sounds on the left side. An intercostal drainage (ICD) was introduced which relieved his breathlessness. He had a persistent airleak for 10 days. The air leak diminished from the 10th to the 14th day. His chest x-ray at this stage showed a small pocket of loculated pneumothorax (Fig. 3) and the ICD was removed. Two weeks later the x-ray showed only evidence of pleural thickening. One month after discharge he was fine with no recurrence of the pneumothorax.

**DISCUSSION**

Turpentine is a pinewood distillate belonging to the terpene group of hydrocarbons. Aspiration or inhalation of hydrocarbons commonly causes a chemical pneumonitis (40-50%). Pleural effusions (3%), ARDS (2%) and secondary bacterial pneumonias are some of the less frequently reported pulmonary complications. Pneumatoceles, pneumomediastinum and pneumothorax are very rare. Our patient had a chemical pneumonitis on admission. He later developed, both a pleural effusion and a pneumothorax with a bronchopleural fistula. Kamijo Y, et al. from Japan have reported a case of bronchopleural fistula following naphtha ingestion another hydrocarbon, occurring nearly 17 days after ingestion.

Turpentine has low viscosity, which increases the risk of aspiration. Hydrocarbons cause a chemical pneumonitis by direct injury to the pulmonary parenchyma, disruption of lipid surfactant layer, destruction of alveolar and capillary membranes. This results in increased vascular permeability, edema and collapse of distal airways and alveoli. The resultant ventilation and perfusion mismatch cause hypoxemia.

Treatment is mainly supportive. There is anecdotal evidence that glucocorticoids, which were used in these patients, are useful but they do not prevent the development of bronchopleural fistula. Kamijo Y, et al. have also reported remarkable resolution in a case of naphtha poisoning with pulse steroid therapy. However, there is no role for prophylactic use of antibiotics and steroids.

**REFERENCES**