Open Journal of
Clinical & Medical
Case Reports

Volume 6 (2020)
Issue 18

ISSN: 2379-1039

A Rare case of necrotizing pneumonia after incidental lighter's fluid ingestion

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Abstract

Hydrocarbon pneumonitis is a type of chemical pneumonitis that often occurs from accidental oral ingestion of hydrocarbons, which can be found in lighter fluid, kerosene, or gasoline. It accounts for over 28,000 cases reported annually to the United States regional poison control centers [1], with about 85% of hydrocarbon exposures are unintentional. These patients often demonstrated significant mental health and behavioral comorbidities as they are more likely to have an episode of major depression, suicidality, conduct disorder, and are at an increased risk for future drug abuse problems [2]. We present a rare case of a forty-three-year-old female who ingested two ounces of lighter fluid and consequently developed hydrocarbon pneumonitis that worsened to necrotizing pneumonia.

Keywords

Lighter's fluid; necrotizing pneumonia; chemical pneumonitis; hydrocarbon pneumonitis.

Introduction

Liquid hydrocarbons are ubiquitously spotted in household items, including petroleum solvents, kerosene, gasoline, lighter fluids, liquid polishes, and waxes [1]. One can have exposure to hydrocarbons in the form of inhalation, ingestion with or without aspiration, or dermal exposure. A handful of cases present in developing third world countries due to people stealing gasoline by siphoning from cars. The consequences range from encephalopathy, metabolic acidosis, central nervous depression, dermatitis to more fatal pneumonitis or malignant arrhythmia. Here we present a rare case of a forty-three-year-old female who ingested two ounces of lighter fluid and consequently developed hydrocarbon pneumonitis that worsened to necrotizing pneumonia.

Case presentation

The patient is a 43-year-old caucasian female with a past medical history of major depressive disorder, bipolar disorder, and alcohol dependence. She presents with increased shortness of breath, bilateral pleuritic chest pain, productive cough and dysphagia after accidentally swallowing lighter fluid from a water bottle. On admission, vitals were stable except for a temperature of 100.4 F. Physical exam showed diminished lung sounds bilaterally, no crepitus on palpation, epigastric and left-sided abdominal tenderness; other systems were unremarkable. Lab revealed leukocytosis of 17K without other abnormalities. Initial chest x-ray showed small extraluminal gas adjacent to the right posterior trachea with a concern for perforation; however, esophagram and esophagogastroduodenoscopy were both unremarkable (Figure 1). The patient was initially thought to have aspiration pneumonitis and treated with Unasyn and then transitioned to PO Augmentin upon discharge to complete a seven-day course. However, she returned three days later with worsening shortness of breath, and pleuritic chest pain. Vitals were within normal limits except O2 92% on room air. Labs results (Table 1). The lungs had significant crackles auscultated in the lower lung fields on physical exam, and some tenderness noted in the epigastric region. CT chest (Figure 2) showed moderate complex bilateral pleural effusion (Left >Right) with multiple fluid loculations concerned for possible necrotizing pneumonia; she was also found to have pulmonary emboli over at right medial lobe medial segment. The patient was started on vancomycin, cefepime, and metronidazole for presumed hospital acquired pneumonia versus necrotizing pneumonia, and Lovonox for pulmonary emboli. After a pulmonology consult, a chest tube was placed on the left side and drained serosanguinous fluid for four days. Light's criteria revealed exudative pleural effusion (serum: LDH 210, protein 5.1, glucose 113; pleural fluid: LDH 496, protein 3.8, glucose 108; no organisms seen on pleural fluid culture). On repeated ultrasound, no loculation found. The patient completed a seven-day course of Vanc, Cefepime and Metronidazole prior to discharge, and symptoms resolved.

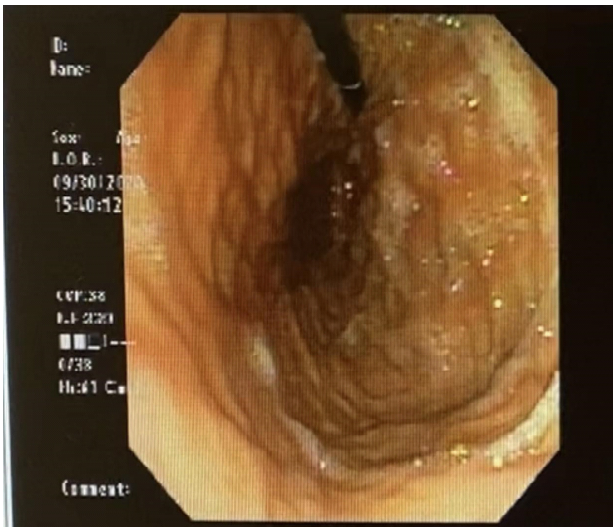

Figure 1: EGD revealed normal esophagus.

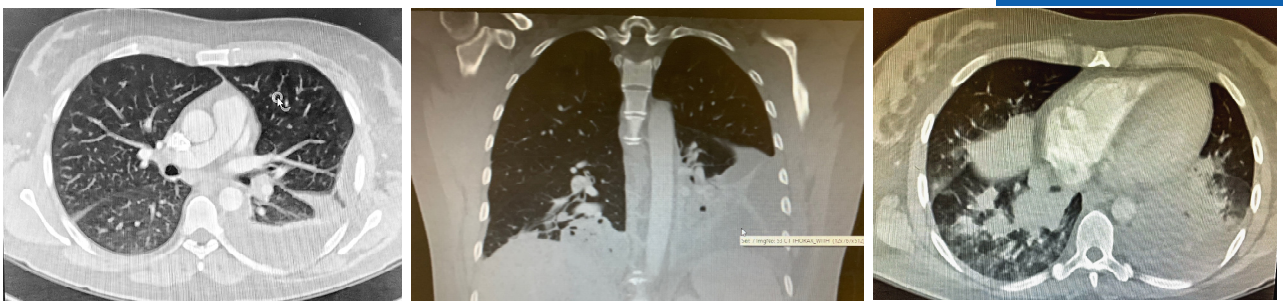

Figure 2: Axial view of CT Chest on first admission (left), CT chest coronal plane (middle), and second admission (right): Multiple abnormal pulmonary emboli in right lung with complex left pleural effusion with multiple loculations

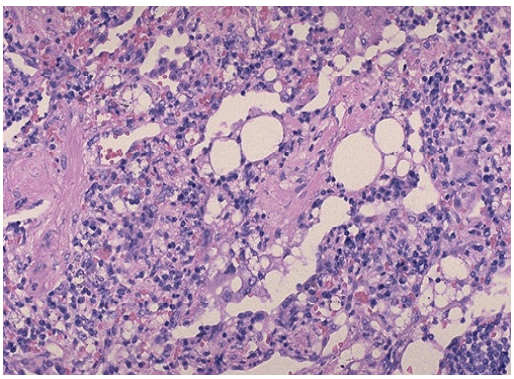

Figure 3: Microscopic appearance of an exogenous lipoid pneumonia in which lipid vacuoles appear, mainly along airways, accompanied by an inflammatory response that can contain foreign body giant cells [6].

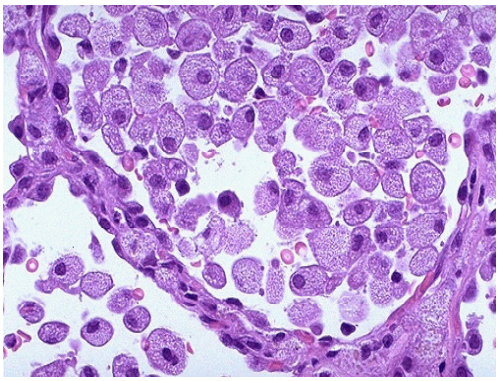

Figure 4: Microscopic appearance of an endogenous lipoid pneumonia in which numerous foamy lipid laden macrophages are present in alveolar spaces [1].

Table 1: Labs on admission

		Our Value	Reference Range
CBC	WBC	15.07 Bill/L	4-10
	Hct	38.7%	36-46
	Hgb	12.65 g/dL	12.5-15.5
	Plt	389 Bill/L	150-450
CMP	Na	135 mMol/L	136-144
	K	3.4 mMol/L	3.6-5.1
	Cl	98 mMol/L	101-111
	CO_2	25 mMol/L	22-32
	BUN	9 mg/dL	8-20
	Cr	0.7 mg/dL	0.44-1
	Glucose:	132 mg/dL	74-118
Inflammatory markers	Lactate	1.1 mMol/l	0.5-2.2
	CRP	294.2 mg/L	0-7.48
	ESR	117 mm/hr	1-25
	Procalcitonin	0.31 ng/mL	<= 0.05

Discussion

Hydrocarbon aspiration primarily presents with pulmonary symptoms such as cough (70%), chest pain (62.5%), dyspnea (55%), and fever (52.5%) [4]. It can range in the form of an acute fatal course such as tension pneumatocele, which is an expanding intraparenchymal cyst that compresses the adjacent lung parenchyma and can lead to a bronchopleural fistula, pneumothorax, and even to cardiopulmonary collapse [3] to the form of a chronic indolent sequelae such as a series of sustained repeated episodes of aspiration and presenting with an indolent disease progression similar to trapped lung disease [4]. Children five-years-of-age or younger still account for the most of the nearly 14,000 annual pediatric exposures [1]; among adolescents and adults, hydrocarbon exposure often arises from recreational inhalant abuse [2].

In terms of pathophysiology, it was hypothesized that hydrocarbon could disrupt alveolar surfactant, which strained the flow of her lungs' productivity. As a result, it increased breathing effort while simultaneously diminishing pulmonary compliance. The mixed monolayer localized in the lungs impaired the mucociliary clearance and inhibited the cough reflex which increased the risk for aspiration. The histopathology distinctively resembles lipoid pneumonia similar to a vaping lung injury (Figure 3,4). Furthermore, the process increases vulnerability to recurrent acute respiratory infections and superimposed bacterial effusions, in its severe form, could lead to necrotizing pneumonia. This figure has been quoted as less than 1% in adults, and Streptococcus pneumoniae and Staph aureus superinfection most commonly cause it [3].

In terms of treatment, contrary to common belief, because hydrocarbon ingestion rarely causes esophageal or gastric injury, active charcoal is contraindicated as they do not bind hydrocarbons and increase the risk of hydrocarbon aspiration [5]. Options of steroids have remained controversial [4] as there is no evidence in the literature supporting its correlations. Furthermore, an empiric antibiotic is standard practice; however, the role of antibiotics can be limited; certain patients still develop multiple organ failure despite appropriate antibiotic therapy and intensive care. It was hypothesized that hydrocarbons lead to the compromise of bronchial and pulmonary vascular supply which decreased the perfusion and bioavailability [2]. Therefore, aggressive treatment options such as chest tube placement, surgical pneumonectomy is warranted and should be initiated early for those with deteriorating stability and failing medical therapy as it is associated with an extremely high rate of mortality [6].

Source of financial support

Loma Linda Veterans Affairs Hospital, This publication's authors certify that they have no affiliations with or involvement with any organization with any financial interest in the subject matter discussed.

Conflict of Interest

The authors of this publication certify that they have no affiliations with or involvement with any organization with any financial interest in the subject matter discussed.

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Manuscript Information: Received: November 14, 2020; Accepted: December 21, 2020; Published: December 30, 2020

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Citation: Gao Y, Dulanto J, Chujutalli R, Huang J. A Rare case of necrotizing pneumonia after incidental lighter's fluid ingestion. Open J Clin Med Case Rep. 2020; 1719.

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