Lipoid Pneumonia Caused by Diesel Aspiration: A Case Report and Literature Review

Fei Tian1,2†, Fei Jiang3‡, Xingguang Qu1, Jinglan Liu2,4, Shengmin Gui4, Liu Min1, Chaxiang Li2,4, Zhaohui Zhang1*, Zuyang Xi2,4*

1Department of Critical Care Medicine, The First Clinical Medical College of China Three Gorges University & Yichang Central People’s Hospital, Yichang 433000, Hubei Province, China
2Center of Clinical Nursing Research, China Three Gorges University, Yichang 443003, Hubei Province, China
3Department of Hematology, The First Clinical Medical College of Three Gorges University, Yichang 443003, Hubei Province, China
4Department of Nursing, The First Clinical Medical College of Three Gorges University, Yichang 443003, Hubei Province, China
†These authors contributed equally to this work.
*Corresponding author: Zuyang Xi, 980347043@qq.com; Zhaohui Zhang, 1410956890@qq.com

Abstract: Diesel poisoning is a rare clinical condition. On September 27, 2021, a 55-year-old male who mistakenly inhaled 20 mL of diesel through a siphon was admitted to our hospital. The main symptoms were cough and asthma. Chest computed tomography (CT) showed both lungs scattered with patchy consolidation, ground-glass shadow, exudation, and pleural effusion. After 61 days of lung rehabilitation training and other supportive treatment, including oxygen therapy, postural drainage, ventilator support, bronchoalveolar lavage, hemoperfusion, continuous renal replacement therapy (CRRT), hormones, and antibiotics, the patient’s condition improved, and the patient was discharged. Through literature review, we found that lung consolidation, ground-glass shadow, nodular lesions, and pleural effusion can be observed on chest images of patients with lipoid pneumonia, with severe cases showing diffuse lesions involving both lungs, possibly secondary to respiratory failure. Children with acute critical illness deteriorates rapidly and have poor prognosis, whereas adults or patients with chronic poisoning have better prognosis after active treatment.

Keywords: Diesel poisoning; Aspiration pneumonia; Lipoid pneumonia

Online publication: July 27, 2023

1. Introduction
Poisoning is a major health issue worldwide. Lipoid pneumonia (LP) refers to the chronic inflammatory response of the lung to lipid substances, which can be categorized as endogenous or exogenous [1,2]. Exogenous LP (ELP) is primarily caused by the aspiration of diesel, gasoline, paraffin oil, and other fatty substances into the lungs, causing acute and chronic pulmonary inflammatory reactions, local pulmonary fibrosis, and even affecting gas exchange, which could lead to respiratory failure [3,4]. Diesel is a commonly used hydrocarbon, a complex mixture of chemicals obtained mainly from the distillation of crude oil, and mainly composed of carbon and hydrogen atoms arranged in aliphatic chains or aromatic (benzene) rings [5]. Diesel poisoning is caused by inhalation of low-viscosity and high-volatile hydrocarbons. Patients with
diesel poisoning may present with a range of symptoms such as coughing, vomiting, or choking within half an hour, ranging from mild respiratory discomfort to severe acute respiratory distress syndrome (ARDS) [6,7]. It is a rare condition seen in clinical practice. We report and analyze the diagnosis and treatment process of one case of ELP caused by mistaken diesel inhalation, review the relevant literature, and summarize the clinical characteristics and treatment experience in order to improve the diagnosis and treatment of this kind of condition. This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Department Yichang Central People’s Hospital (approval number: 2022-081-01).

2. Case report
2.1. Case history
The patient, a 55-year-old farmer, with a history of hypertension on oral nifedipine, mistakenly inhaled about 20 mL of diesel oil through a siphon while working on September 26, 2021. He self-reported diesel swallowing, coughing, choking, and burning sensation behind the sternum. Chest computed tomography (CT) from the local hospital showed pleural effusion on both lungs. The patient was transferred to our hospital for further diagnosis and treatment and admitted to our department on September 27, 2021, after completing the COVID-19 nucleic acid test in the emergency department. The patient had a slight cough and wheeze, apparent after physical activities at the time of admission. Upon physical examination, his body temperature was 38.5°C, pulse rate was 90 beats/min, respiratory rate was 28 breaths/min, and blood pressure was 110/65 mmHg; his breathing was regular, with thick breath sounds in both lungs and audible moist rales, without any pleural rub; he had normal limb muscle tone and strength. The patient was diagnosed with aspiration pneumonia, lipid pneumonia, severe pneumonia, organic solvent poisoning, acute respiratory distress syndrome (ARDS), respiratory failure, and acute gastric mucosal lesions.

The treatment plan was as follows: indwelling gastric tube, supplemental oxygen therapy via nasal cannula, antibiotic (piperacillin-tazobactam), anti-inflammatory (methylprednisolone 80 mg), gastric protection (omeprazole), blood perfusion, bronchoalveolar lavage (BAL), and other symptomatic and supportive treatments. At 6 p.m. on September 28, the patient’s dyspnea worsened, ARDS occurred, and his blood oxygen saturation dropped to 79% under 5 L/min of oxygen; after sedation and analgesia, tracheal intubation and ventilator-assisted ventilation were initiated. On October 1, bedside continuous renal replacement therapy (CRRT) was performed due to low urine output. On October 3, chest CT showed that the pulmonary infection was worse than before, and the consolidation was still serious; prone position ventilation was initiated. On October 6, we detected a *Klebsiella pneumoniae* carbapenemase (KPC)-producing strain, *Acinetobacter baumannii*, subject to contact isolation. On October 7, we detected methicillin-resistant *Staphylococcus aureus* (MRSA) strain, subject to contact isolation, and bronchoalveolar lavage fluid was collected under bedside bronchoscopy for next-generation sequencing technology (NGS) detection. If *Stenotrophomonas maltophilia* was present, cefoperazone sodium, sulbactam sodium, sulfamethoxazole, and minocycline were given. On October 18, bedside tracheotomy was performed, ventilator-assisted ventilation was initiated, and the patient was given 2U of red blood cells via intravenous infusion and a combination of polymyxin and tigecycline. On October 25, the patient’s ARDS and respiratory failure improved, and he was transferred to the emergency medical ward for continued treatment. After 61 days of treatment, the patient’s condition improved, and the patient was discharged from the hospital on November 7, 2021. After a year of follow-up visit, the patient was in good condition without complications. The imaging and laboratory investigation results during treatment are shown in Figure 1 and Table 1.
Table 1. Laboratory investigation results of patients before and after treatment

<table>
<thead>
<tr>
<th>Investigations</th>
<th>Reference</th>
<th>Detection time (2021)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body temperature (°C)</td>
<td></td>
<td>36.3–36.9</td>
</tr>
<tr>
<td>White blood cells (10^9/L)</td>
<td>3.5–9.9</td>
<td>8.55</td>
</tr>
<tr>
<td>Neutrophil percentage (%)</td>
<td>40.0–75.0</td>
<td>96.4</td>
</tr>
<tr>
<td>CRP (mg/L)</td>
<td>0–10</td>
<td>211.28</td>
</tr>
<tr>
<td>PCT (ng/mL)</td>
<td>0–0.046</td>
<td>3.46</td>
</tr>
<tr>
<td>Hemoglobin (g/L)</td>
<td>130–175</td>
<td>114</td>
</tr>
<tr>
<td>Oxygenation index (mmHg)</td>
<td>400–500</td>
<td>324.09</td>
</tr>
<tr>
<td>Partial pressure of oxygen (PO₂)</td>
<td>80–100</td>
<td>68.06</td>
</tr>
<tr>
<td>Oxygen saturation (%)</td>
<td>95–98</td>
<td>94.98</td>
</tr>
<tr>
<td>Partial pressure of carbon dioxide (PCO₂)</td>
<td>35–45</td>
<td>32.24</td>
</tr>
<tr>
<td>IL-6(pg/mL)</td>
<td>0–7</td>
<td>237</td>
</tr>
</tbody>
</table>

Abbreviations: CRP, C-reactive protein; IL-6, interleukin 6; Oct, October; PCT, procalcitonin; Sept, September.

3. Literature review
PubMed, CNKI database, and Wanfang database were searched for clinical studies on aspiration pneumonia caused by diesel aspiration. The search period was from January 2015 to October 2021, and the search languages were Chinese and English. The Chinese search strategy was as follows: (“diesel” OR “diesel poisoning” OR “diesel aspiration”) AND (“aspiration pneumonia” OR “aspiration”). The English search
terms were as follows: (“Diesel poisoning” OR “Diesel Fuel” OR “Diesel”) AND (“Aspiration pneumonia” OR “Lipoid pneumonia” OR “Exogenous lipoid pneumonia”). A search method combining subject words and free words was used. A total of 37 literatures were retrieved; reviews, conferences, and other literatures were excluded; and 9 case reports were obtained [8-16], as shown in Table 2. Based on the 9 case reports and the data of the patient with diesel aspiration pneumonia in our hospital, a clinical analysis was performed. The main clinical manifestations of diesel aspiration are cough, dyspnea, nausea, vomiting, fever, and chest pain; pulmonary consolidation, reticular nodules, ground-glass opacities, pleural effusion, and pneumothorax lesions are commonly observed on imaging, involving the lower lobes of both lungs, the right middle lobe, and the left lingual lobe; the commonly used treatments are anti-inflammatory, anti-infectives, oxygen therapy, respiratory support, blood, BAL, and pulmonary rehabilitation.

Table 2. Nine case reports of diesel aspiration

<table>
<thead>
<tr>
<th>Ref.</th>
<th>Age (years)/ gender</th>
<th>Onset</th>
<th>Symptom(s)</th>
<th>Imaging results</th>
<th>Laboratory investigations</th>
<th>Treatment plan</th>
<th>Treatment duration (days)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>[8]</td>
<td>25/ male</td>
<td>4 days</td>
<td>Fever, sore throat, cough, difficulty breathing, diaphoresis, rash, and headache</td>
<td>Consolidation in the middle and lower regions of both lungs and emphysema</td>
<td>Elevated white blood cells and neutrophils</td>
<td>Ceftriaxone, hemodialysis, methylprednisolone 1 g/day, piperacillin, and hemodialysis</td>
<td>23</td>
<td>Death</td>
</tr>
<tr>
<td>[9]</td>
<td>1/ male</td>
<td>Within hours</td>
<td>Vomiting and difficulty breathing</td>
<td>Consolidation of right middle and lower lobe and bilateral parietal lobe</td>
<td>pH 6.7, increased PaCO2, and decreased PaO2</td>
<td>Acid and antibiotics</td>
<td>3</td>
<td>Death</td>
</tr>
<tr>
<td>[10]</td>
<td>1.5/ female</td>
<td>Within 1 day</td>
<td>Vomiting, dyspnea, decreased SpO2, and generalized cyanosis</td>
<td>Left upper lung infiltrates, bilateral pneumothorax, and alveolar collapse</td>
<td>pH 6.98, increased PaCO2, decreased PaO2, and elevated white blood cells</td>
<td>Antibiotic</td>
<td>12</td>
<td>Death</td>
</tr>
<tr>
<td>[11]</td>
<td>24/ male</td>
<td>8 hours</td>
<td>Difficulty breathing</td>
<td>Infection in the middle and lower lungs, patchy parenchyma, reticular nodules, and ground-glass opacities</td>
<td>Decreased PaO2, lung biopsy showed lipid-laden macrophages in alveoli and interstitium</td>
<td>Ceftriaxone, azithromycin, and oxygen therapy</td>
<td>2</td>
<td>Recovery</td>
</tr>
<tr>
<td>[12]</td>
<td>55/ male</td>
<td>14 days</td>
<td>Expectoration, cough, chest pain, and chest tightness.</td>
<td>Right upper lobe consolidation with necrosis and gas shadows</td>
<td>Increased ratio of white blood cells to neutrophils, lung biopsy showed a large number of neutrophil inflammatory exudation, purulent necrosis, and fibrous tissue hyperplasia</td>
<td>Methylprednisolone, cefoperazone-sulbactam, and amebrolex</td>
<td>40</td>
<td>Recovery</td>
</tr>
<tr>
<td>[13]</td>
<td>Unknown /Male</td>
<td>4 days</td>
<td>Expectoration, cough, nausea, vomiting, and fever</td>
<td>Right middle lobe mass shadow and pleural effusion</td>
<td>Increased neutrophil ratio, PCT, and CRP; lung biopsy showed adipoid cells and a little necrotic tissue</td>
<td>Methylprednisolone, cefoperazone-sulbactam, and antidote</td>
<td>22</td>
<td>Recovery</td>
</tr>
<tr>
<td>[14]</td>
<td>28/ female</td>
<td>14 days</td>
<td>Nausea, vomiting, difficulty breathing, chest pain, and mouth sores</td>
<td>Multiple patchy exudates</td>
<td>Increased WBC, PCT, and neutrophil ratio</td>
<td>Pipericillin, amikacin, and methylprednisolone</td>
<td>20</td>
<td>Recovery</td>
</tr>
<tr>
<td>[15]</td>
<td>30/ male</td>
<td>2 days</td>
<td>Cough, nausea, and vomiting</td>
<td>Increased markings in both lungs, strip-like high-density shadows, ground-glass shadows, and nodular calcifications in the middle lobe of the right lung</td>
<td>Elevated white blood cell ratio, PCT, CRP, and neutrophils</td>
<td>Antibiotics, antivirals, and dexamethasone</td>
<td>14</td>
<td>Recovery</td>
</tr>
<tr>
<td>[16]</td>
<td>39/ male</td>
<td>2 days</td>
<td>Cough, chest pain, and fever</td>
<td>Ground-glass opacities, nodules, paving stones, and pleural effusion</td>
<td>Elevated white blood cell ratio, PCT, CRP, and neutrophils</td>
<td>Cefmetazole, methylprednisolone, omeprazole, and bronchoalveolar lavage</td>
<td>7</td>
<td>Recovery</td>
</tr>
</tbody>
</table>

Abbreviations: CRP, C-reactive protein; PaCO2, partial pressure of carbon dioxide; PaO2, partial pressure of oxygen; PCT, procalcitonin; SpO2, oxygen saturation; WBC, white blood cell.
4. Discussion
Diesel aspiration is extremely rare, and the literature published is dominated by case series and case reports. Diesel aspiration can cause multi-organ toxicity, mainly respiratory system damage and pulmonary toxicity [17]. There are no specific clinical manifestations. Diesel is insoluble in water, but soluble in fat, alcohol, and benzene. Upon inhalation, the respiratory mucosal damage induces bronchial smooth muscle spasm and causes airway hyperresponsiveness. The damage to pulmonary surfactant increases alveolar vascular permeability and causes alveolar exudation. The damage to the alveolar and vascular cells caused by chemical substances deposited in the lungs can stimulate local inflammatory responses and lead to pulmonary fibrosis, thus resulting in impaired lung function and lung volume. Based on literature reports, the onset of symptoms is different depending on the amount and nature of aspiration. Many acute cases, such as this patient, would be admitted to the hospital after developing symptoms within a few hours, secondary to ARDS, but cases of chronic poisoning are those who continue to inhale small amounts of diesel. Symptoms may only appear for days or even a week or two, and in most of these patients, their condition progresses rapidly. Literature reports have shown that diesel inhalation rarely affects the entire lung. Considering that the anatomical structure of the right main bronchus is thick, short, and straight, the lesions are more common in the middle and lower lobes of the right lung, accompanied by pleural effusion. In our case, due to the inhalation of a large amount of diesel oil, the patient immediately choked, presenting with symptoms like fever, shortness of breath, dyspnea, and a large amount of yellowish-white sputum, along with large lung lesions and multiple patchy shadows in both lungs. Chemical pneumonia and acute lung injury should be considered when treatment with anti-infectives is unsuccessful. At present, the diagnosis of LP is mainly based on a history of lipid aspiration, radiography, CT imaging, BALF, bronchoscopy lung biopsy, and lung puncture biopsy. The gold standard for diagnosis is the presence of lipid-rich macrophages on lung biopsy. This case provides a clear history of diesel inhalation with BAL cytology, which is consistent with the diagnosis of ELP.

At present, there are no guidelines or expert consensus for the diagnosis and treatment of LP. In addition to oxygen therapy, ventilator-assisted ventilation, extracorporeal membrane oxygenation, and supportive treatment, intravenous antibiotics, steroids, and BAL are commonly used for treatment. Although there is no recommendation on the duration and dose of steroid use in these patients due to the lack of evidence, the use of systemic glucocorticoid therapy is recommended in severe patients [19]. Sen et al. [20] reported in their retrospective study that patients with hydrocarbon pneumonia responded well to steroid therapy [20]. Our patient had severe chemical pneumonia and extrapulmonary involvement, and glucocorticoid therapy was used to reduce the inflammatory response; after the symptoms had subsided, the dose was reduced. On the other hand, BAL is currently used as a safe and effective treatment method since hydrocarbons cannot be metabolized by the human body in the alveolar space. There are patients who have shown significant improvement after BAL treatment for hydrocarbon pneumonia [21]. In our case, BAL was performed with 0.9% sodium chloride injection, since normal saline is incompatible with oil. A study has reported that emulsifier and 3% sodium bicarbonate + 0.02% nitrofurazone solution can be used for BAL [21]. However, these recommendations are made based on case reports. There is no further clinical validation on their efficacy and safety. The role of corticosteroids and antibiotics in the treatment of hydrocarbon aspiration pneumonia is controversial, as shown in an animal study [22]. Although antibiotics may be ineffective in the treatment of diesel aspiration pneumonia, the majority of patients with diesel aspiration pneumonia developed leukocytosis, increased neutrophil percentage, and lung infection, and received antibiotics when inflammatory markers such as C-reactive protein (CRP) and procalcitonin (PCT) were elevated. The bacterial cultures in our patient were positive for Acinetobacter baumannii, MRSA strain, and Xanthomonas maltophilia. Therefore, for the treatment of critically ill patients with a long course of disease, it is necessary to first identify the pathogen and then target the infection.
5. Conclusion
Currently, there are no standardized treatment and evidence-based guidelines for diesel aspiration pneumonia. Compared with the cases reported in literature, our patient presented with respiratory failure in the early stage, with diffuse exudation in both lungs. Our patient also had severe disease, rapid development, long course of disease, and various complications. The patient was under treatment with oxygen therapy, postural drainage, and ventilatory support. BAL, hemoperfusion, CRRT, steroid, antibiotics, pulmonary rehabilitation training, and other supportive treatments were also initiated. The patient’s condition improved, and he was transferred out of the intensive care unit and followed-up. Through literature analysis, severe patients with acute diesel aspiration pneumonia, especially children, may be in danger, and their condition may develop rapidly, leading to poor prognosis. However, most patients with chronic diesel aspiration pneumonia have good prognosis. Therefore, patients with acute diesel aspiration pneumonia should be treated with early, and active intervention should be given to ensure a good curative effect and prognosis. Due to the limitations of the present study, we hope that there will be more reports and higher-quality studies to further explore the diagnosis, treatment methods, and therapeutic effects of diesel aspiration pneumonia.

Acknowledgements
We would like to express our gratitude to all participants for their support and cooperation in this study.

Disclosure statement
The authors declare no conflict of interest.

References


Publisher’s note
Bio-Byword Scientific Publishing remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.