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

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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

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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, lipoid 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**.

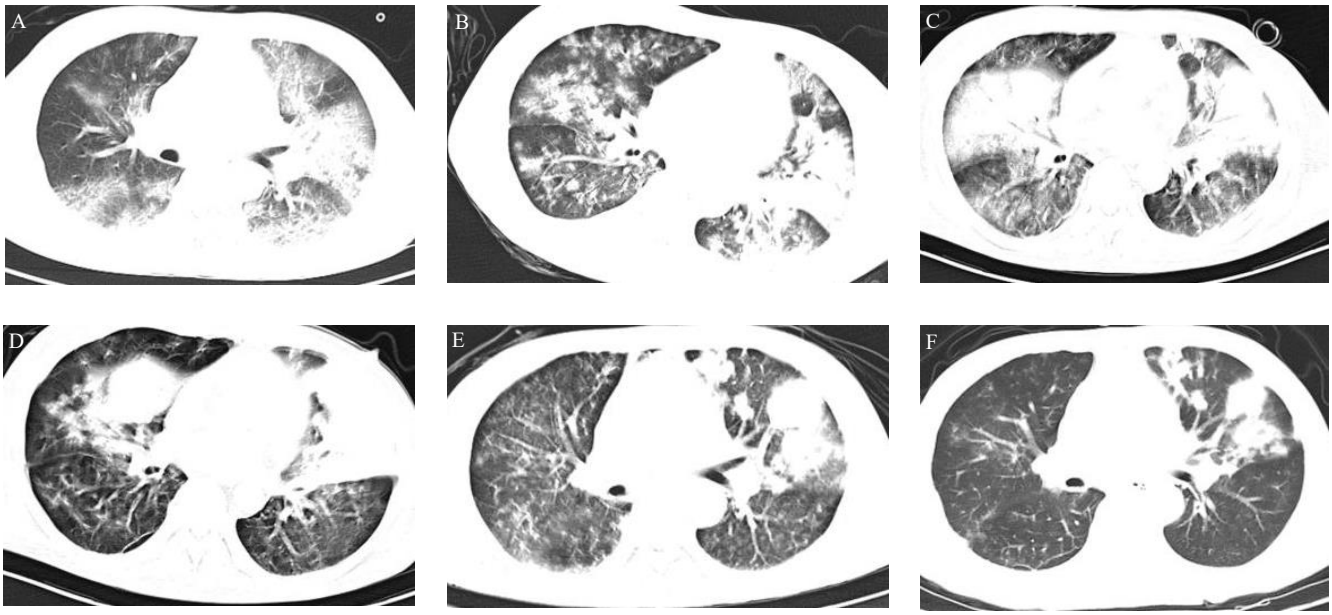

Figure 1. Computed tomography of the patient during treatment. (A) September 28, 2021: Exudation or infection in both lungs. (B) October 2, 2021: Increased exudation or infection and flaky blurred shadows in both lungs, with left pleural effusion. (C) October 9, 2021: Both lung markings increased, with presence of exudation or infection, but the flaky blurred shadows in both lungs decreased. (D) October 16, 2021: Smaller area of flaky blurred shadows. (E) October 21, 2021: Reducing area of flaky blurred shadows. (F) November 14, 2021: Patchy high-density shadows in both lungs, with partial consolidation, and slightly reduced absorption of bilateral pleural effusion.

Table 1. Laboratory investigation results of patients before and after treatment

Investigations	Reference	Detection time (2021)							
		Sept 27	Oct 1	Oct 5	Oct 9	Oct 13	Oct 17	Oct 21	Oct 25
Body temperature (℃)	36.3–36.9	36.6	37.5	37.1	38.2	38.2	39.2	38.5	38.1
White blood cells (10^9/L)	3.5–9.9	8.55	5.48	13.06	7.67	10.53	10.55	14.98	13.02
Neutrophil percentage (%)	40.0–75.0	96.4	93	89.7	94.9	96.2	93.6	94	93.8
CRP (mg/L)	0–10	211.28	77.65	214.22	225.32	139.76	60.46	142.78	46.96
PCT (ng/mL)	0–0.046	3.46	2.24	1.12	0.44	0.3	0.45	0.38	0.25
Hemoglobin (g/L)	130–175	114	90	85	79	83	75	95	88
Oxygenation index (mmHg)	400–500	324.09	151.69	283.01	549.81	829.9	535.02	374.1	746.54
Partial pressure of oxygen (PO_2)	80–100	68.06	75.85	59.43	115.46	174.28	112.35	107.16	156.77
Oxygen saturation (%)	95–98	94.98	96.71	91.77	99.71	100	100	99.12	100
Partial pressure of carbon dioxide (PCO_2)	35–45	32.24	47.15	54.56	46.55	42.87	40.17	38.41	40.14
IL-6(pg/mL)	0–7	237	412.9	322.0	244.9	77.22	157.9	144.96	

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

Ref.	Age (years)/ gender	Onset	Symptom(s)	Imaging results	Laboratory investigations	Treatment plan	Treatment duration (days)	Outcome
[8]	25/ male	4 days	Fever, sore throat, cough, difficulty breathing, diarrhea, rash, and headache	Consolidation in the middle and lower regions of both lungs and emphysema	Elevated white blood cells and neutrophils	Ceftriaxone, hemodialysis, methylprednisolone 1 g/day, piperacillin, and hemodialysis	23	Death
[9]	1/ male	Within hours	Vomiting and difficulty breathing	Consolidation of right middle and lower lobe and bilateral parietal lobe	pH 6.7, increased $PaCO_2$, and decreased PaO_2	Acid and antibiotics	3	Death
[10]	1.5/ female	Within 1 day	Vomiting, dyspnea, decreased SpO_2, and generalized cyanosis	Left upper lung infiltrates, bilateral pneumothorax, and alveolar collapse	pH 6.98, increased $PaCO_2$, decreased PaO_2, and elevated white blood cells	Antibiotic	12	Death
[11]	24/ male	8 hours	Difficulty breathing	Infection in the middle and lower lungs, patchy parenchyma, reticular nodules, and ground-glass opacities	Decreased PaO2, lung biopsy showed lipid-laden macrophages in alveoli and interstitium	Ceftriaxone, azithromycin, and oxygen therapy	2	Recovery
[12]	55/ male	14 days	Expectoration, cough, chest pain, and chest tightness,	Right upper lobe consolidation with necrosis and gas shadows	Increased ratio of white blood cells to neutrophils; lung biopsy showed a large number of neutrophilic inflammatory exudation, purulent necrosis, and fibrous tissue hyperplasia	Mezlocillin, doxofylline, and ambroxol	40	Recovery
[13]	Unknown /Male	4 days	Expectoration, cough, nausea, vomiting, and fever	Right middle lobe mass shadow and pleural effusion	Increased neutrophil ratio, PCT, and CRP; lung biopsy showed adipoid cells and a little necrotic tissue	Methylprednisolone, cefoperazone-sulbactam, and antidote	22	Recovery
[14]	28/ female	14 days	Nausea, vomiting, difficulty breathing, chest pain, and mouth sores	Multiple patchy exudates	Increased WBC, PCT, and neutrophil ratio	Piperacillin, amikacin, and methylprednisolone	20	Recovery
[15]	30/ male	2 days	Cough, nausea, and vomiting	Increased markings in both lungs, strip-like high-density shadows, ground-glass shadows, and nodular calcifications in the middle lobe of the right lung	Elevated white blood cell ratio, PCT, CRP, and neutrophils	Antibiotics, antivirals, and dexamethasone	14	Recovery
[16]	39/ male	2 days	Cough, chest pain, and fever	Ground-glass opacities, nodules, paving stones, and pleural effusion	Elevated white blood cell ratio, PCT, CRP, and neutrophils	Cefmetazole, methylprednisolone, omeprazole, and bronchoalveolar lavage	7	Recovery

Abbreviations: CRP, C-reactive protein; $PaCO_2$, partial pressure of carbon dioxide; PaO_2, partial pressure of oxygen; PCT, procalcitonin; SpO_2, 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.

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Disclosure statement

The authors declare no conflict of interest.

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