



Case Report

Hydrocarbon-induced chemical pneumonitis complicated by organizing pneumonia: A case report.

Brabaharan Subhani, Rasika Ranaweera, Upul Dissanayake

National Hospital, Sri Lanka.

Key words: hydrocarbon inhalation, chemical pneumonitis, organizing pneumonia

Corresponding Author: Brabaharan Subhani E-mail:< brabaharan197subhani@gmail.com >	
 https://orcid.org/0000-0003-4865-3791	
Received: 19 Mar 2023, accepted revised version: 27 Mar 2023 Published: 02 Nov 2023	
Competing Interests: Authors have declared that no competing interests exist	
© Authors. This is an open-access article distributed under a Creative Commons Attribution-Share Alike 4.0 International License (CC BY-SA 4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are attributed and materials are shared under the same license.	

Introduction

Hydrocarbon inhalation induced chemical pneumonitis complicated by organizing pneumonia is a rare entity [1, 2]. Chemical pneumonitis is defined as an inflammatory reaction in response to aspiration of a chemical substance [1]. The recent economic and fuel crisis in Sri Lanka spurred siphonage and massive black-market peddling of fuel. Many lacked the appropriate equipment to siphon fuel leading to inevitable accidental ingestion and inhalation of diesel and petrol. Here we present a case of diesel-induced chemical pneumonitis resulting in an organizing pneumonia.

Case report

A male in his early forties, employed as an assistant to a truck driver, was admitted with acute onset abdominal pain following accidental inhalation of diesel. He was a habitual consumer of heroin, nicotine and alcohol. He was initially admitted to a surgical unit at a tertiary hospital where he underwent emergency laparotomy due to a clinical suspicion of pneumoperitoneum which yielded a negative result. Subsequent contrast enhanced CT abdomen did not reveal any significant findings. He subsequently left the ward against medical advice as heroin withdrawal set in. A day later, he got admitted to the National Hospital of Sri Lanka with acute onset shortness of breath. On clinical examination, he was tachypnoeic and tachycardic with evidence of bilateral, fine and coarse lung crepitations.

SpO₂ was 80% on air. Arterial blood gas analysis (ABG) showed a type 1 respiratory failure. His WBC was 18,000/mm³ with a neutrophilic predominance and 15% eosinophilia. The CRP was 400 mg/dL. His coagulation profile and renal biochemistry were normal. His liver

biochemistry was deranged with evidence of raised AST (237) and ALT (230). HIV and VDRL tests were negative. Rh factor was negative. CXR showed evidence of bilateral consolidation. HRCT chest demonstrated patchy and coalescing areas of consolidation in the basal segments of bilateral lower lobes, lingular segment of left upper lobe, right middle lobe and anterior segment of right upper lobe. Crazy paving appearance was seen in the right middle lobe. Tree in bud opacities with peri-bronchial thickening was seen in both lungs, with fairly preserved apical segments. The findings were suggestive of hydrocarbon pneumonitis.

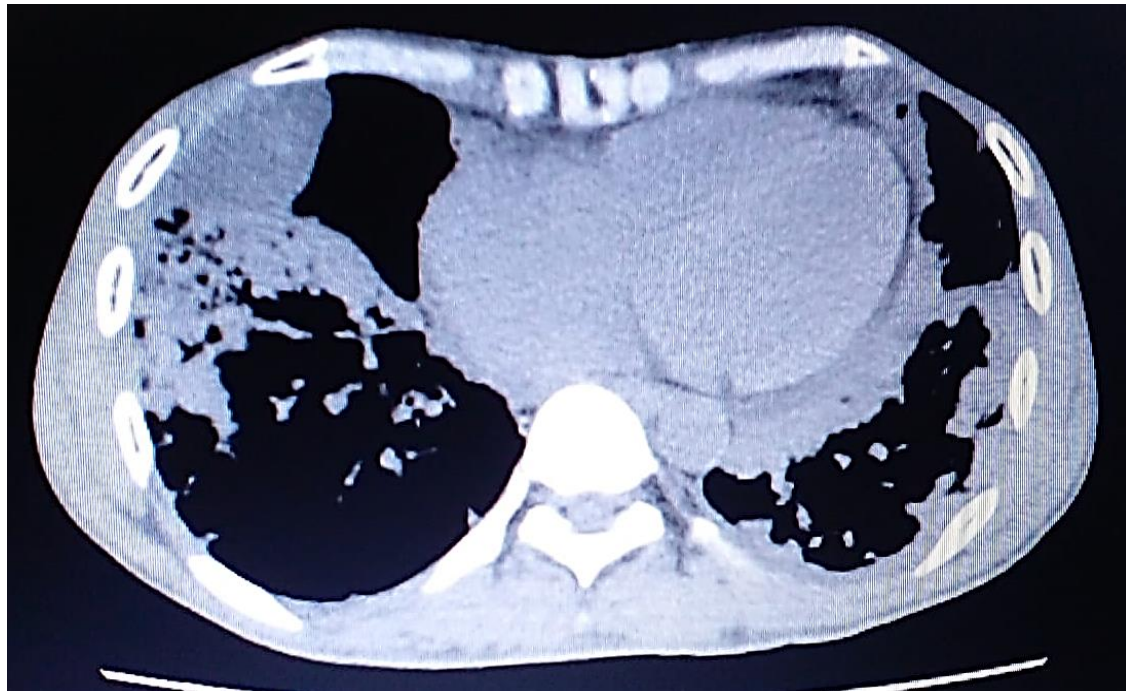


Figure 1: HRCT chest

Bronchoalveolar lavage (BAL) revealed neutrophil predominance with less than 5% hemosiderin laden macrophages. Pneumocystis pneumonia (PCP) in toluidine blue and acid fast bacilli (AFB) were negative but there was significant growth of *Pseudomonas* sp in the sputum culture.

Initially, the patient was placed in the ICU and a short duration of non-invasive ventilation (NIV) was given. Subsequently, the patient was managed with intravenous (IV) glucocorticoids (hydrocortisone and prednisolone), antibiotics and chest physiotherapy. He required a prolonged course of antibiotics for almost three weeks, including 3rd generation cephalosporins and piperacillin tazobactam [2], oxygen supplementation and frequent chest physiotherapy. The subjective sensation of shortness of breath persisted despite normal saturation on air with minimal desaturation on the six-minute walk test (6MWT). Despite being satisfied by the treatment he received, he had a subjective

sensation of breathlessness which persisted months after the initial event despite a normal 6MWT and absence of desaturation. A timeline of the events is shown below.

Discussion

Though hydrocarbon-induced chemical pneumonitis complicated by organizing pneumonia is a rare entity, it can lead to significant morbidity. However supportive treatment along with glucocorticoids can lead to significant improvement reducing complications.

The damage caused by aspiration depends on the viscosity, volatility and chemical side chains of the hydrocarbon. Lower viscosity and higher volatility are associated with a greater chance of aspiration and resultant pulmonary injury [3]. Aspiration may result in pulmonary oedema, bronchospasm and resultant hypoxia. These substances have the ability to rapidly diffuse throughout the bronchial tree and disrupt the surfactant barrier. Pathology may reveal a necrotizing pneumonia along with direct destruction of capillaries, alveolar septae and the pulmonary epithelium. They then provoke the activation of macrophages, leading to an increased release of cytokines and a prolonged inflammatory reaction [4]. The surfactant layer, which is composed of lipids, is made soluble by hydrocarbons causing further damage. Subsequently, there can be atelectasis, interstitial inflammation and hyaline membrane formation [3].

If the pathological findings, derived from bronchoscopic or percutaneous biopsy, demonstrate lipid-laden macrophages, hydrocarbon pneumonitis needs to be suspected. However, our patient had a predominant neutrophilic count in the BAL indicative of a secondary bacterial infection [5].

CT findings that may raise suspicion of aspiration pneumonitis include posterior pulmonary opacities and a pattern of focal, peripheral or peribronchiolar consolidation involving one or more segments [2,6]. Pulmonary infiltration and opacification were found in most cases following fuel siphonage. The right middle lobe (80%) was the predominant lung field involved, followed by the right lower lobe (46.7%), the left lower lobe (40%), and the left upper lobe (3.3%). More than one-third of patients (36.7%) showed the involvement of two lobes and nearly one-sixth of patients (16.7%) showed involvement of more than two lobes [5]. There are four common parenchymal abnormalities of hydrocarbon pneumonitis seen in the chest CT, including consolidations with an air bronchogram, ground-glass opacifications, air-space nodules and crazy-paving pattern. Nevertheless, none of these findings are unique to hydrocarbon pneumonitis [7]. Areas of fat attenuation within the consolidation were reported as a characteristic manifestation of hydrocarbon pneumonitis. However, fat attenuation may later be obscured by superimposed inflammation [8]. This is similar to our patient who demonstrated patchy and coalescing areas of consolidation in the basal segments of bilateral lower lobes, lingular segment of left upper lobe, right middle lobe and anterior segment of right upper lobe with a crazy-paving appearance. Acute complications of chemical pneumonitis include secondary pneumonia and acute respiratory distress

syndrome (ARDS). Long-term complications include bronchiectasis, bronchiolitis obliterans, and lung destruction [9].

The treatment of hydrocarbon pneumonia is aimed at reducing the symptoms. Management usually includes glucocorticoids (GC), broad spectrum antibiotics, broncho-alveolar lavage and chest physiotherapy [1,10]. Although antibiotics are ineffective in the treatment of hydrocarbon pneumonitis, most patients with hydrocarbon pneumonitis undergo treatment with antibiotics because radiologically differentiating between hydrocarbon pneumonitis and superimposed pulmonary infection may be difficult and furthermore many develop secondary bacterial infections.

The use of GC for treatment is still controversial. Experimental and clinical studies in animals and humans have found different results and are inconclusive. A retrospective study has demonstrated that patients with hydrocarbon pneumonitis responded well to GC therapy [11]. The combined use of inhaled and intravenous steroids had positive effects on the clinical and radiological recovery of patients [12]. Some studies suggest that GC usage reduces inflammation and subsequent fibrosis. [12]. However, it is important to emphasize that GCs can increase the risk of secondary infections.

In a study by Sen et al, evaluating cases of pneumonia that occurred secondary to hydrocarbon exposure in children, 18 patients (33.3%) with an oxygen saturation of < 92 who had findings of respiratory failure and who were admitted to the ICU were treated with GCs. The oxygen saturation of 17 patients returned to normal in the first 24 hours of GC treatment. Moreover, none of the patients treated with steroids had a superinfection [13].

Glucocorticosteroids are the standard treatment for organizing pneumonia. Regression is observed quickly after treatment initiation. However, the tapering of steroids can induce relapse in about 50% of cases. Other immunosuppressive therapies, such as cyclophosphamide, cyclosporine and macrolides, also seem to be effective [14]. A few case reports have reported dramatic improvements after BAL in patients with hydrocarbon pneumonitis [5,15].

The progress and severity of subsequent pulmonary symptoms determine the requirements for further care and length of hospital stay. Similar to patients with pneumonia, symptomatic improvement might precede the resolution of lesions on CXR. Thus, decisions on treatment should depend on the patients' pulmonary symptoms. According to Chen YJ et al and Chang HY et al nearly 20% of patients demonstrated an oxygenation saturation level of less than 90% on arrival at hospital and one case eventually required continuous positive airway pressure mask ventilation and another case underwent tracheal intubation with ventilator support. Of those patients whose hospital course was fully documented, 40.9% had to be admitted for more than 1 week. Moreover, three cases required intensive care and one case was fatal. They found three complications, all pulmonary sequelae, including one empyema, one pulmonary abscess,

and one pneumothorax. The complication and mortality rates in their studies were 7.5% and 2.5%, respectively. [5,16]

Conclusion

Hydrocarbon-induced chemical pneumonitis may lead to long term pulmonary morbidity with recurrent hospital admission, poor quality of life and loss of productivity. However early supportive treatment along with GC and BAL may lead to improvement in the overall symptoms.

Acknowledgements

We would like to thank the staff in our wards who tirelessly devote their time to taking care of our patients.

References

1. Walls R, Hockberger R, Gausche-Hill M, Bakes K. Rosen's Emergency Medicine: Concepts and Clinical Practice. 9th ed. Philadelphia PA: Elsevier; 2018.
2. Komiya K, Ishii H, Umeki K, Kawamura T, Okada F, Okabe E, et al. Computed tomography findings of aspiration pneumonia in 53 patients. *Geriatrics & gerontology international*. 2013;13(3):580-5.
<https://doi.org/10.1111/j.1447-0594.2012.00940.x>
3. Pham K, Sverchek J, McPheeters RA. Chemical pneumonitis from hydrocarbon aspiration. *The western journal of emergency medicine*. 2008;9(3):165.
4. Mylonaki E, Voutsas V, Antoniou D, Papakosta D, Kontakiotis T, Skordalaki A, et al. Hydrocarbon pneumonitis following liquid paraffin aspiration during a fire-eating performance: a case report. *Journal of Medical Case Reports*. 2008;2(1):214.
<https://doi.org/10.1186/1752-1947-2-214>
5. Chen YJ, Hsu CC, Chen KT. Hydrocarbon pneumonitis following fuel siphonage: A case report and literature review. *World journal of emergency medicine*. 2019;10(2):69-74. <https://doi.org/10.5847/wjem.j.1920-8642.2019.02.001>
6. Bonte FJ, Reynolds J. Hydrocarbon Pneumonitis. *Radiology*. 1958;71(3):391-7.
<https://doi.org/10.1148/71.3.391>
7. Betancourt SL, Martinez-Jimenez S, Rossi SE, Truong MT, Carrillo J, Erasmus JJ. Lipoid pneumonia: spectrum of clinical and radiologic manifestations. *AJR American journal of roentgenology*. 2010;194(1):103-9.
<https://doi.org/10.2214/AJR.09.3040>
8. Marchiori E, Zanetti G, Mano CM, Irion KL, Daltro PA, Hochegger B. Lipoid pneumonia in 53 patients after aspiration of mineral oil: comparison of high-resolution computed tomography findings in adults and children. *Journal of computer assisted tomography*. 2010;34(1):9-12.
<https://doi.org/10.1097/RCT.0b013e3181a9ec9f>
9. White CS, Templeton PA. CHEMICAL PNEUMONITIS. *Radiologic Clinics of North America*. 1992;30(6):1231-43.
[https://doi.org/10.1016/S0033-8389\(22\)00864-8](https://doi.org/10.1016/S0033-8389(22)00864-8)

10. Tormoehlen LM, Tekulve KJ, Nañagas KA. Hydrocarbon toxicity: A review. *Clinical toxicology (Philadelphia, Pa)*. 2014;52(5):479-89.
<https://doi.org/10.3109/15563650.2014.923904>
11. Sen V, Kelekci S, Selimoglu Sen H, Yolbas I, Günes A, Abakay O, et al. An evaluation of cases of pneumonia that occurred secondary to hydrocarbon exposure in children. *European review for medical and pharmacological sciences*. 2013;17 Suppl 1:9-12.
12. Konca C, Tekin M, Turgut M. The Combined Use of Inhaled and Intravenous Steroids for Children With Chemical Pneumonitis After Ingestion of Paint Thinner. *Iranian Red Crescent medical journal*. 2016;18(7):e24300.
<https://doi.org/10.5812/ircmj.24300>
13. Sen V, Kelekci S, Selimoglu Sen H, Yolbas I, Günes A, Abakay O, Fuat Gurkan M. An evaluation of cases of pneumonia that occurred secondary to hydrocarbon exposure in children. *Eur Rev Med Pharmacol Sci*. 2013 Feb 1;17(Suppl 1):9-12.
14. Basarakodu KR, Aronow WS, Nair CK, Lakkireddy D, Kondur A, Korlakunta H, et al. Differences in treatment and in outcomes between idiopathic and secondary forms of organizing pneumonia. *American journal of therapeutics*. 2007;14(5):422-6.
<https://doi.org/10.1097/01.pap.0000249905.63211.a1>
15. Russo R, Chiumello D, Cassani G, Maiocchi G, Gattinoni L. Case of Exogenous Lipoid Pneumonia: Steroid Therapy and Lung Lavage with an Emulsifier. *Anesthesiology*. 2006;104(1):197-8.
<https://doi.org/10.1097/00000542-200601000-00027>
16. Chang HY, Chen CW, Chen CY, Hsue TR, Chen CR, Lei WW, et al. Successful treatment of diffuse lipoid pneumonitis with whole lung lavage. *Thorax*. 1993;48(9):947-8.
<https://doi.org/10.1136/thx.48.9.947>