A Rare Case of Chemical Pneumonia Caused by Hydrocarbon Ingestion

Hidrokarbon Yutulmasının Neden Olduğu Nadir Bir Kimyasal Pnömoni Vakası Fevzi Yılmaz¹, Muhammed Furkan Albayrak¹, Engin Deniz Arslan¹, Önder Acar¹

ABSTRACT

Aim: Hydrocarbon compounds that are ingested orally show their effects by tissue hypoxia, pulmonary irritation, and systemic toxicity. Chemical pneumonia is one of the most serious complications of hydrocarbon poisoning. The clinical and radiological detection of chemical pneumonia is important in terms of treatment and prognosis. In this article, a 54-year-old patient diagnosed with chemical pneumonia due to kerosene drinking is presented.

Case Report: A 54-year-old man was admitted to the Emergency Department with complaints of cough, shortness of breath, and epigastric pain. On admission, his vital signs were as follows: BP: 150/95 mmHg, SPO2: % 97, the pulse rate: 97/min, body temperature: 36.5 C. His medical history was notable for drinking approximately 100 ccs of kerosene accidentally while cleaning his house about 5 days ago. The patient was found to have chemical pneumonia and hospitalized.

Conclusion: Chemical pneumonia should be remembered in patients admitted to the emergency department after drinking hydrocarbons. In addition, most of these poisoning episodes are preventable.

Keywords: Hydrocarbon toxicity, chemical pneumonia, lamp oil

ÖZ

Amaç: Ağızdan yutulan hidrokarbonlu bileşikler doku hipoksisi, pulmoner irritasyon vesistemik toksisite ile etkilerini gösterirler. Kimyasal pnömoni hidrokarbon zehirlenmesinin en ciddi görülen komplikasyonlardan biridir. Kimyasal pnömoninin klinik ve radyolojik olarak saptanması tedavi ve prognoz açısından önemlidir. Bu yazıda gazyağı içmeye bağlı şimik pnömoni teşhisi konulan 54 yaşında bir hasta sunuldu.

Olgu Sunumu: 54 yaşında erkek hasta Acil Servis'e öksürük, nefes darlığı ve epigastrik ağrı şikayetleri ile başvurdu. Kabulde vital bulguları; TA: 150/95 mmHg, SPO2: % 97, nabız: 97/dk, ateş: 36,5 C idi. Hastanın öyküsünde yaklaşık 5 gün önce evini temizlerken yanlışlıkla yaklaşık 100 cc kadar gaz yağı içtiği öğrenildi. Yapılan tetkiklerinde şimik pnömoni saptanan hasta hastaneye yatırıldı.

Sonuç: Acil servise hidrokarbon içilmesi sonucu getirilen hastalarda şimik pnömoni unutulmamalıdır. Ayrıca bu tür zehirlenmelerin büyük çoğunluğu önlenebilir

Anahtar Kelimeler: Hidrokarbon toksisitesi, kimyasal pnömoni, gaz yağı

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Introduction

In excess of 28,000 cases of hydrocarbon, exposure is reported to United States regional poison control centers each year, and these intoxications are among the leading poisoning types across the globe. Accidental intakes constitute some 85% of all hydrocarbon exposures. Most of the approximately 14,000 annual pediatric cases affect children aged 5 years or younger (1). Despite a very low death rate, moderately severe intoxications particularly with hydrocarbon ingestion are common and require supportive care. Hydrocarbon ingestion ranks first in the frequency list of exposures affecting this age group, being responsible for some 75 percent of cases (2). Young children (≤5 years of age) are usually tempted by their exploratory behavior and get intoxicated by ingesting hydrocarbons. Hydrocarbons are usually insecurely and improperly stored in drinking containers such as soda or water bottles. On the other hand, adolescents and adults are typically exposed to inhaled hydrocarbons through recreational abuse of these substances (3).

The route of intake, type of chemical compound, and amount of substance taken are important determinants of the toxicity of a given substance. Despite the fact that hydrocarbons injure nearly all systems in the human body, the lungs, brain, and heart are the most severely affected organs (4-6). Animal studies suggest that severe necrotizing pneumonia represents the principle pathologic change (7). Direct severe injury to the airway epithelium, alveolar septae, and pulmonary capillaries are among other findings, which also include the dissolution of the lipid surfactant layer. Atelectasis, interstitial inflammation, and hyaline membrane formation are secondary pathological findings. Ingestion of hydrocarbon-containing substances primarily causes aspiration and chemical pneumonia, leading to morbidity and mortality through lung injury and subsequent complications. Acute hydrocarbon exposure diagnosed by a clinical evaluation with patient history and physical examination findings. Symptoms vary according to chemical class and route of exposure. Most of the time, exposure and type of substance are identifiable in the patient's history (5). With this case report, we aimed to draw attention to hydrocarbon poisoning, which mostly occurs as a result of home accidents and causes preventable lung complications.

Case Report

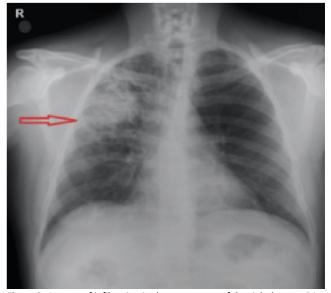
A 54-year-old man was admitted to the Emergency Department with complaints of cough, shortness of breath, and epigastric pain. On admission his vital signs were as follows: BP: 150/95 mmHg, SPO2: % 97, the pulse rate: 97/min, body temperature: 36.5 C. His medical history was remarkable for accidental drinking of approximately 100 ccs

of kerosene while cleaning his house about 5 days ago (figure 1). The patient had no smoking history or known



Figure 1. The kerosene bottle from which that the patient drank kerosene and which he brought to the emergency department

comorbidity. His physical examination revealed a normal oropharynx, reduced lung sounds, and ronchus localized to the right hemithorax on pulmonary auscultation. His other organ systems showed normal examination findings. In the radiological examinations, a posteroanterior chest X-Ray showed an infiltrative image consisting of a large number of small, irregular densities with ill-defined borders in the right upper lung (figure 2). In chest CT, there were cystic bronchectasic areas, opacities compatible with secretion in dilated bronchi (pneumatocele), parenchymal glass ground densities, and centrilobular opacities in the upper lobe of the right lung (figure 3).



 $\label{eq:Figure 2.} \textbf{An area of infiltration in the upper zone of the right lung on PA chest X-Ray.}$

The laboratory test results were as the following: Hb: 11.7 g/dL, HTC: 34.8%, Glucose 117 mg/dL, BUN:10 mg/dL,



Figure 3. Chest CT findings compatible with chemical pneumonia in the upper zone of the right lung.

Creatinine: 0.89 mg/dL, Sodium:133 mmol/L, Alanine aminotransferase (ALT): 132 U/L, Aspartate transaminase (AST): 56 U/L; in arterial blood gas analysis, pH: 7.42, PO2:83 mmHg, PCO2: 42 mmHg, and HCO3: 25 mmol/L. A 12-lead electrocardiogram (ECG) was recorded, and continuous cardiac monitoring was initiated to monitor ventricular arrhythmias.

Based on the above findings, the patient was consulted with the department of pulmonary diseases with a preliminary diagnosis of chemical pneumonia and hospitalized for further diagnostic workup and treatment. He was treated with O2, bronchodilators, nebulizers, and administered Piperacillin-Tazobactam against superinfections. Having recovered both clinically and radiologically the patient was discharged with full recovery 12 days later.

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Discussion

The aliphatic hydrocarbons are straight-chain compounds, which include butane, propane, kerosene, and mineral seal oil. They are used in furniture polishes, lamp oil, and lighter fluid. Gasoline and naphtha are composed of mixtures of aliphatic hydrocarbons which additionally contain other substances such as xylene, toluene, benzene, naphthalene, and tetraethyl lead that is added to enhance octane. Petroleum distillates are produced their contents processing crude oil; include gasoline, naphtha, mineral spirits, kerosene, paraffin wax, and tar. Terpene hydrocarbons, family of cyclic hydrocarbons, include turpentine (commonly utilized Anatolian J Emerg Med 2020;3(4); 129-132

as paint thinner) and pine oil (incorporated into a variety of cleaning products enriched with pine odor). Aromatic hydrocarbons are cyclic compounds that incorporate a benzene ring such as benzene, toluene, and xylene. Their main area of use is the production of solvents, glues, nail polishes, paints, and paint removers (4,5).

Clinical manifestations of hydrocarbon intoxications depend on the type of hydrocarbon and the route and amount of hydrocarbon intake. The primary mechanism of toxicity of aromatic hydrocarbons, halogenated hydrocarbons (e.g., trichloroethylene), and pine oil is systemic absorption from the gastrointestinal tract or the pulmonary tree. Pulmonary aspiration and chemical pneumonitis, however, may also develop by vomiting the compounds. Central nervous system (CNS), the liver, kidney, myocardium, and bone marrow may also be damaged (6). Aliphatic hydrocarbons damage the respiratory system. Lung findings depend on aspiration occurring during drinking or vomiting of these substances (7). Our patient has also been forced to vomit by their relatives after drinking a toxic agent. We think that these practices cause complications due to a high risk of aspiration.

Among these substances, intoxications due to drinking kerosene are the most common ones in our country (8). Severe fatal pneumonia can occur by aspirating a few drops of these substances into the lungs. The main symptoms of aspiration are cough, dyspnea, cyanosis, and rales. In severe cases, massive pulmonary edema, pulmonary bleeding, secondary infection, and sometimes pneumothorax and pleurisy may develop (9). Our patient drank approximately 100 ccs of kerosene, and the history and physical examination were taken at the admission to the emergency department showed findings suggesting respiratory system damage.

Hydrocarbons may also injure the central nervous system (CNS) and the cardiovascular system, with the latter mainly via arrhythmias and myocardial occurring dysfunction. Especially solvent hydrocarbons (e.g., halogenated hydrocarbons) cause fatal ventricular arrhythmias by making the myocardium susceptible to the effects of endogenous and exogenous catecholamines. A variety of hydrocarbons depress the central nervous system (CNS) through some compounds' direct neuronal effects and/or hypoxia caused by profound lung damage. Patients may suffer restlessness, confusion, sleepiness, and rarely, convulsions. They are lost from a coma or respiratory arrest. Cardiomegaly, heart failure, hepatosplenomegaly, and proteinuria are also signs of severe intoxication (5). In our patient, an ECG taken at admission and cardiac enzymes measured during follow-up were normal. In addition, except for the respiratory system, no abnormalities of the CNS or other systems were detected.

ingested, hydrocarbons irritate the pharynx, esophagus, stomach, and small intestine by direct contact and cause edema and mucosal ulceration of the affected sites. Orogastric and intestinal irritation may result in nausea, vomiting, and hematemesis, which are typical of mild severity and do not usually require care (4,5,10). Our patient also had epigastric complaints, but he did not have signs or symptoms such as nausea-vomiting, hematemesis, and melena, and his physical examination showed no irritative lesions in the oropharynx. However, superficial ulceration was detected at the esophagogastric junction in upper gastrointestinal endoscopy while he was hospitalized, and H2 blockers and proton pump inhibitors (PPIs) were initiated accordingly.

Plain films of the chest usually suffice for the determination of hydrocarbon-induced chemical pneumonitis. The radiograms initially manifest pulmonary aspiration/chemical pneumonitis by a large number of small densities with indiscernible borders that are scattered all over the lungs. Progressive cases tend to develop larger lesions as individual lesions congregate (4,11). In our patient, a chest X-Ray showed an infiltrative lesion consisting of multiple, small, irregular densities with unclear borders in the right upper lung. Subsequent thorax CT revealed radiological findings compatible with chemical pneumonia in the upper zone of the right lung (figures 2, 3).

As with all patients visiting the ED, airway, breathing, and circulation should be secured in these cases. Gastric lavage is risky in these patients and may lead to aspiration. Antidotes such as activated carbon should not be used either, as they will also cause vomiting. Oxygen, nebule therapy, bronchodilators as needed, as well as antibiotics if there are signs of superinfection, are administered to patients with pulmonary symptoms. Corticosteroids are not used. Treatment of CNS symptoms is symptomatic. Oxygen, hydration and, if necessary, sedation is applied.

Conclusion

Even if fluids containing hydrocarbons are taken orally, they can lead mainly to pulmonary complications, as well as cardiac and neurological complications. These poisonings are largely preventable by taking appropriate measures. Among the measures to be taken for this purpose, raising the awareness of family members about not keeping such substances in places where children can easily reach, particularly in water containers, is of utmost importance. We are of the opinion that regular training of emergency physicians is necessary for the diagnosis and treatment of patients who present after ingesting these substances.

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Authors' Contribution: FY conceived the case report. FY, MFA contributed reagents, materials, analysis tools or data. FY, MFA, EDA, OA drafted the manuscript and all authors contributed substantially to its revision.

Informed Consent Statement: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review in this journal.

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