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An evaluation of cases of pneumonia that occurred secondary to hydrocarbon exposure in children

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Abstract. – OBJECTIVE: Hydrocarbon pneumonia is distinct among the types of childhood pneumonia in that it has a different pathogenesis and treatment and is preventable. In this study, the cases of 54 children with hydrocarbon pneumonia admitted to the Dicle University Medical Faculty Pediatric Chest Diseases Unit between the years 2006 and 2010 were analyzed retrospectively.

PATIENTS AND METHODS: The medical records of 54 patients diagnosed with pneumonia after ingesting/inhaling hydrocarbons were analyzed retrospectively. Age, sex, presenting symptoms, clinical status, radiological and laboratory findings and response to treatment and prognosis were noted.

RESULTS: 35 (64.8%) of the patients were male, 19 (35.2%) were female and the ages of the patients ranged from 1 to 5 with an average of 2.49 ± 0.80. The etiologies of the pneumonia were thinner (33%), naphta (3.7%) and kerosene. In 49 of the patients (90.7%), the symptoms started to occur the day the patient was exposed to hydrocarbons. The average length of hospital stay was 4.0 ± 2.3 days. Six patients were treated in the intensive care unit (ICU), and one patient with hydrocarbon pneumonia due to kerosene ingestion died. Inhaled corticosteroids were administered to 18 patients who were progressively deteriorating and inhaled salbutamol was given to 16 patients with bronchospasm. Patients with radiological findings on their chest X-rays and auscultatory findings were found to have longer hospital stays ($p < 0.05$).

CONCLUSIONS: To prevent chemical pneumonia, precautions must be taken to stop children under 5 years of age from using/abusing chemical substances. Although some cases of chemical pneumonia lead to death, with diligent care and treatment, the outcomes are promising. The patients in this study group responded well to treatment with inhaled corticosteroids and salbutamol.

Key Words:
Childhood, Etiology, Treatment, Hydrocarbons, Pneumonia, Steroids

Introduction

Pneumonia is the inflammation of lung tissue and is caused by infectious and non-infectious agents. Chemical pneumonia is caused by aspiration of substances such as volatile hydrocarbons, animal (milk) or mineral oils that are toxic for the lungs. Chemical pneumonia is generally diagnosed based on information from family members of the patient or by clinical suspicion. A sudden onset of dyspnea and coughing, together with lower lobe infiltrates on chest X-ray are important findings that suggest the presence of chemical pneumonia. Prognosis and clinical improvement depends on the type, volume and pH of the substance aspirated and complications are due to aspiration and response of the patient to treatment. Another cause of chemical pneumonia is hydrocarbon aspiration. The prevalence of severe pneumonia after hydrocarbon aspiration is less than 2%. The importance of chemical pneumonia is underscored by the increasing number of new cases of death in children and adults due to hydrocarbon aspiration that are reported each year[1]. Chemical pneumonia is of particular importance because it is preventable and it differs from other forms of childhood pneumonia in treatment and pathogenesis. There are not many studies on chemical pneumonia due to hydrocarbon exposure with large series of patients in the literature. The goal of this study was to retrospectively analyze 54 cases of pediatric chemical pneumonia admitted to our Hospital between the years 2006 though 2010 in order to identify high-risk groups, preventable causes and optimal treatment and to reduce morbidity and mortality.

Patients and Methods

In our study, the medical records of 54 children with hydrocarbon pneumonia who were admitted

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to the Dicle University Medical Faculty Pediatric Chest Diseases Unit were analyzed retrospectively and the demographic information, clinical and laboratory findings and etiologies of chemical pneumonia due to hydrocarbon exposure were evaluated. Pneumonia was diagnosed based on the history, radiological and laboratory findings. Radiographic findings were used as the gold standard. In children under three years of age, tachypnea was considered the best clinical sign of pneumonia.

Statistical Analysis

All numerical values are represented as mean ± standard deviation, counted values represented as frequency and percentage. Statistical analysis was performed with the aid of SPSS 12.0 for Windows program (SPSS Inc., Chicago, IL, USA). Fisher's exact test and Chi-square test was used. p value < 0.05 was considered as statistically significant.

Results

In our study, the medical records of 54 children with hydrocarbon pneumonia who were admitted to the Dicle University Medical Faculty Pediatric Chest Diseases Unit were analyzed retrospectively. Out of the 54 cases included in the study, 35 were male (64.8%) and 19 were female (35.2%). All patients were between the ages of 1 and 5 and were an average of 2.49±0.80 years. Patients who were 2 and 3 years old accounted for 86.9% of the study group. The median age was 3 in male and 2 in female patients with a male/female ratio of 1.84.

The most common etiology of hydrocarbon pneumonia was thinner (63%), followed by naphta (33%) and kerosene (3.7%) (Table I). The most common presenting symptoms were vomiting, coughing and high fever. Most of the patients' symptoms started the day they were exposed to hydrocarbons. The most common physical exam findings at admission were high fever, tachycardia and tachypnea. In 6 of the cases (11.1%), the clinical situation deteriorated and respiratory failure ensued. Only one of the patients in the study died, secondary to hydrocarbon pneumonia caused by kerosene ingestion. The deceased case was a 1.5 year-old male. Half an hour after drinking kerosene, he started coughing and respiratory distress occurred. At admission the case was intubated and was cyanotic and lethargic. His blood pH was less than 7.35, rales were heard on auscultation and there were distinct areas of consolidation on his PA chest X-ray. The case was admitted to the intensive care unit and received mechanical ventilation and died after six days.

Table I. The etiologies of cases of hydrocarbon pneumonia.

Etiology	Numbers	%
Thinner	34	63.0
Naphta	18	33.3
Kerosene	2	3.7
Total	54	100.0

In this study 77.7% of the cases were admitted between April and August. On the PA chest X-rays of patients taken at admission, 26 were normal (48.1%), 17 had consolidations (31.5%) and 7 had air bronchograms (13%). Upon auscultation at admission, 23 patients had normal breath sounds (42.6%), 22 patient had rales (40.7%) and 9 patients had rhonchi (16.7%).

Inhaled corticosteroids were administered to patients with respiratory failure, patients who were admitted to the intensive care unit (ICU) and patients who continued to have a high fever 48 hours after admission. Inhaled salbutamol was used to treat 16 patients (29.6%) and inhaled corticosteroids were used to treat 18 patients (33.3%). Patients with hydrocarbon pneumonia who had abnormal radiological findings on their PA chest X-rays taken at admission were found to have longer hospital stays. The difference in admission times between patients who had abnormal radiological findings and who had a normal chest X-ray were statistically meaningful ($p < 0.05$) (Table II).

Patients who had abnormal breath sounds at admission were found to have longer hospital stays and the difference in admission times between the patients who had auscultatory findings and the patients who had normal breath sounds was statistically significant ($p < 0.05$) (Table II). The majority of patients who received inhaled corticosteroids had abnormal radiological findings on the PA chest X-rays taken at admission. There was a statistically significant difference in the number of patients on inhaled corticosteroids who had abnormal radiological findings at admission and the number of patients on inhaled corticosteroids who had normal radiological findings ($p < 0.05$).

Table II. Factors that effect admission time in patients with hydrocarbon pneumonia.

	Number (n)	Average ± standard deviation	*p* value
Auscultatory findings			
No	23	3.3 ± 1.2	0.016
Yes	31	4.6 ± 2.7	
Radiological findings			
No	26	3.1 ± 1.2	0.003
Yes	28	4.9 ± 2.6	
Elevated ALT levels			
No	52	3.9 ± 2.2	0.355
Yes	2	5.5 ± 0.7	
Sex			
Male	35	4.2 ± 2.4	0.477
Female	19	3.7 ± 1.7	
Inhaled steroids			
No	36	3.2 ± 1.2	0.006
Yes	18	5.5 ± 3.0	

ALT: Alanine amino transferase.

Discussion

In children, cough and fever that present along with tachypnea, intercostal retractions, rales, rhonchi, decreased breath sounds and, in severe cases, nasal flaring and cyanosis, suggest pneumonia[2]. In our study, 88.9% (51/54) of the patients had a cough and 81.5% (44/54) had a fever. On physical examination, 40.7% (22/54) of the patients had rales, 25.9% (14/54) had wheezing, 11.1% (6/54) had nasal flaring and 9.3% (5/54) had cyanosis. In our study, we examined 54 patients diagnosed with hydrocarbon pneumonia after drinking or inhaling products containing hydrocarbons. Thirty-five of the patients were male (64.8%), 19 were female (35.4%) and the male/female ratio was 1.84:1. The most common compounds containing hydrocarbons are petroleum products. Some household chemical products contain hydrocarbons and accidentally or deliberately drinking/breathing in these products results in poisoning. Hydrocarbon compounds can be addictive and may be inhaled by children with substance abuse.

In this study 77.7% of the cases were admitted between April and August, suggesting that in our region, people are more likely to have hydrocarbon-containing products for household use (for example, paint) in these months.

Aspiration of high viscosity and less volatile hydrocarbons can cause exogenous lipoid pneumonia which can be chronic and mostly asymptomatic[3]. Aspiration of low viscosity and volatile hydrocarbons can cause pseudo-infectious pneumonia[3,4].

Based on experiments performed in animals, it is known that ethyl alcohol and kerosene penetrate the alveoli and rapidly reduce the amount of oxygen in the alveolar air. It is reported that 1 hour after aspiration, progressive bronchiolitis, alveolar hemorrhage and edema start to form. After 24 hours, desquamation of type II pneumocytes, fibrinoid leukocytosis and microabscess occur and these lesions can be present for several weeks[5].

Grossi et al[6] found that one week after pyrofluid aspiration, necrotizing acute fibrinous pneumonia and necrotizing acute bronchiolitis were histologically observed in lung tissue. Burkhardt et al[7] found inflammation using an electron microscope in tissue from the cases in their study and suggested that the inflammation may be the result of pseudo-infections. The severity of pulmonary symptoms correlates with the degree of exposure to hydrocarbons. Clinical findings are not specific and may manifest as a fever, cough, dyspnea and gastrointestinal symptoms such as vomiting[6,8]. Symptoms such as cyanosis, hemoptysis, chest pain and/or diarrhea have also been reported. The diagnosis is easy if there is a history of hydrocarbon exposure. Hydrocarbon pneumonia can be fatal in 1-2% of cases[9]. It has been found that hydrocarbon pneumonia caused by kerosene tends to be more severe and have a higher mortality rate. Similarly, in our study, the 1 case (1.8%) that died had in-

gested kerosene. In a study of hydrogen pneumonia due to kerosene aspiration by Gondoin et al. (10), the radiological findings included alveolar consolidation (51%), ground glass appearance (39%) and alveolar nodules (23%). In our study, 17 of the cases had alveolar consolidation (31.5%). Radiological regression generally occurred between 2 weeks and 8 months. Pneumothorax and pneumomediastinum are rare complications of hydrocarbon pneumonia.

The treatment of hydrocarbon pneumonia is aimed at reducing the symptoms[8]. The use of steroids and antibiotics for treatment is still controversial. Experimental and clinical studies in animals and humans have found different results and are inconclusive. Some studies suggest that steroid usage reduces inflammation and the subsequent fibrosis[11]. Corticosteroids may reduce the development of fibrosis[12]. However, it is important to emphasize that steroids can increase the risk of secondary infections. In our study, 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 steroids. The oxygen saturation of 17 patients returned to normal in the first 24 hours of steroid treatment. Moreover, none of the patients treated with steroids had a superinfection.

The use of antibiotics is controversial. Generally, the prophylactic use of antibiotics for hydrocarbon pneumonia patients is not recommended because experimental and clinical studies have shown that during the acute phase of chemical pneumonia, the pH environment of the lung makes bacterial colonization unlikely.

Experimental studies suggest that the cause of infection is generally due to secondary bacterial infections that occur after the acute clinical symptoms of chemical pneumonia. However, it is difficult to determine whether the changes that occur in the early period of these cases are due to an infection or not. Thus, a risk-benefit analysis suggests that antibiotics should generally be administered as an early treatment[13]. Borer and Koelz[14] have reported that treatment is vital and that in cases with complications, the usage of antibiotics is beneficial. In the literature[15], antibiotics are used in almost all cases whereas steroids are only used in some of the cases. In our study, 18 patients (33.3%) were given steroids. Antibiotics were used in 48 patients (88.9%). The outcomes of using antibiotics and inhaled steroids in our cases were generally positive.

References

1) Colombo JL. Aspiration syndromes. In: Behrman RE, Kliegman RM, Jenson HB (eds). Nelson textbook of pediatrics (17th edition) Philadelphia: WB Saunders Co; 2004. pp. 1427-1428.
2) American Academy of Pediatrics. In: Pickering LK, ed. 2000 RedBook: Report the Comminittee on Infectious Disease. 25 th ed. Ellk Grove Village; IL: American Academy of Pediatrics; 2000, p. 408.
3) Haas C, Lebas FX, Le Jeunne C, Lowenstein W, Durand H, Hugues FC. Pneumopathies caused by inhalation of hydrocarbons: a propos of 3 cases. Ann Med Int 2000; 151: 438-447.
4) Lee JS, Im JG, Song KS, Seo JB, Lim TH. Exogenous lipoid pneumonia: high-resolution CT findings. Eur Radiol 1999; 9: 287-291.
5) Scharf SM, Heimer D, Goldenstein J. Pathologic and physiologic effects of aspiration of hydrocarbons in therat. Am Rev Respir Dis 1981; 124: 625-629.
6) Grossi E, Crisanti E, Poletti G, Poletti V. Fireeater's pneumonitis. Monaldi Arch Chest Disease 2006; 65: 59-61.
7) Burkhardt O, Merker HJ, Shakibaei M, Lode H. Electron microscopic findings in BAL of fire-eater after petroleum aspiration. Chest 2003; 124: 398-400.
8) Facon D, Coumbaras J, Bigot E, Bahlouli F, Boissonnas A, Bellin MF. Acute hydrocarbon pneumonia after white spirit aspiration: sequential HRCT findings. Eur Radiol 2005; 15: 31-33.
9) Franquet T, Gimenez A, Roson N, et al. Aspiration diseases: findings, pitfalls, and differential diagnosis. Radiographics 2000; 20: 673-685.
10) Gondouin A, Manzoni P, Ranfaing E, Brun J, Cadranel J, Sadoun D, Cordier JF, Depierre A, Dalphin JC. Exogenous lipid pneumonia: a retrospective multicentre study of 44 cases in France. Eur Respir J 1996; 9: 1463-1469.
11) Svimli ZU, Dogan N, Kizilkaya M, Gundoğdu C. Experimental acute lung injury. Effects of methylprednisolone and lidocaine on histopathology and neutrophils. Saudi Med J 2004; 25: 1831-1834.
12) Behrman RE, Kliegman RM, Jenson HB. In: John L Colombo. Pneumonia. Nelson Textbook of Pediatrics. 17th Ed, Philadelphia: WB Saunders, 2008: pp. 1427-1428.
13) Bartlett JG. Aspiration disease and anaerobic infection. In: Fishman AP et al. Fishman's pulmonary diseases and disorders (3rd edition) Vol 2. New York: The Mc Graw-Hill Co; 1998, pp. 2011-2019.
14) Borer H, Koelz AM. Fire-eater's lung (hydrocarbon pneumonitis). Schweiz Med Wochenschr 1994; 124: 362-367.
15) Cok G, Erdinc M, Savas R, Alper H. Fire eater pneumonia. Turkish Thor J 2003; 4: 279-282.