VIA MEDICA

CASE REPORT

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Fire-eater's lung

The authors declare no finacial disclosure

Abstract

Fire eater's lung (FEL) is an acute hydrocarbon pneumonitis caused by aspiration or inhalation into airways of liquid hydrocarbons. This disorder is classified into distinct form of chemical toxic pneumonitis.

An amateur fire-eater is presented in this work. He accidentally aspirated into airways about 1/3 of glass of grill lighter fluid composed of mixture of liquid hydrocarbons. A few hours after this incident he had severe symptoms like weakness, high temperature, midsternal pleuritic chest pain, myalgia of the back, shortness of breath, and dry cough. Radiologic examination revealed consolidations with well–defined cavitary lesions (pneumatoceles) in lower lobes mainly in the left lower lobe. After one week of this event clinical improvement was observed. The lesions resolved nearly completly during three months. The review of the literature connected with fire-eater's lung is also presented.

Key words: hydrocarbon pneumonitis, liquid hydrocarbons with low viscosity, pneumatoceles

Adv. Respir. Med. 2016; 84: 337–341

Introduction

Fire eater's lung (FEL) or fire-breather's lung is an acute hydrocarbon pneumonitis caused by accidental aspiration into airways of liquid hydrocarbons. This disorder is a distinct form of chemical pneumonitis [1–5]. Liquid hydrocarbons (LHs) are widely present in our environment, households and in industry [6]. The most frequently used products of petroleum are as follows: gasoline, motor oils, lubricating oils, diesel oil, kerosene, lighter fluid, and lamp oil [3, 4, 6].

An acute hydrocarbon pneumonitis usually occurs after accidental ingestion or inhalation of LHs by infants, then by elderly persons, by fire-eaters, by industry workers, mainly petrochemical workers, painters or sometimes by the waste workers. Some intentional uses are also reported [4, 5, 7]. The first description of FEL was done by Gerbeaux *et al.* [8] in 1971. They reported a history of an infant who ingested fluid with liquid hydrocarbons.

The street entertainers use different flammable petroleum derivatives for the flame blowing. The entaintainer blows out some pyrofluid into a burning stick, it looks like the flames are coming from the entertainer's mouth („ dragon's breath"). After blowing of the flame the showman is also at risk to aspirate some residual liquid hydrocarbons from the mouth [1, 4, 6].

In this paper we present the clinical history of an amateur fire-eater's performer who accidentally aspirated grill lighter fluid into airways.

Case report

A 26-year-old male, a teacher, was admitted to the IIIrd Clinic of TB and Lung Diseases in

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DOI: 10.5603/ARM.2016.0044
Received: 9.08.2016

ISSN 2451–4934

the Institute of TB and Lung Diseases in Warsaw because of exertional dyspnea and lung lesions on the Chest X-ray.

In July 2011 he spent holidays by the Baltic Sea.

At that time during one evening he and his college, also teacher, decided to do the fire-eater's performance by the sea shore. Unfortunately at the beginning of this show he aspirated about 1/3 of glass of a lighter fluid for grill containing mixture of Lhs. After this episode he started to be very weak, he had also some shortness of breath. A few hours (5 to 6 hours) later the clinical status of the patient had deteriorated, he had severe dyspnea, tachycardia, midsternal pleuritic chest pain, myalgia mainly of the back, high temperature up to 39 degrees of Celsius and mild dry cough. Physician from the district out-patient clinic visited the patient two times during that night and decided to direct him to the hospital. On admission to the hospital the patient had very severe dyspnea. On auscultation of lungs crackles in the lower parts were heard. Blood gases revealed partial respiratory insufficiency, PaO_2 58 mm Hg, $PaCO_2$ 37.7 mm Hg, pH 7.45, HCO_3 25.5 mmol/l. CRP was elevated to 246.7 mg/l, WBC was increased to 16.3 × 10^6/mm^3, neutrophil count was 81.7 %. Prokalcytonin level was slightly elevated to 0.56 ng/ml (the normal upper limit do 0.5).

Chest X-ray (Fig. 1) revealed bilateral consolidations in the lower lung zones, more extensive on the left side. On CT scans consolidations with pneumatoceles in the lower parts of lungs, mainly on the left side were seen (Figs 2, 3). In spirometry FVC was decreased to 2.82 l, 45.5% of predicted value, FEV_1 was decreased to 0.72 l — 14.6% of predicted value.

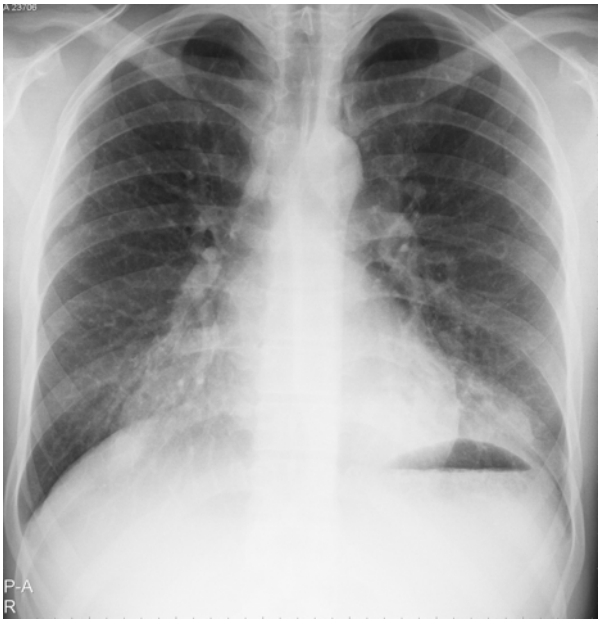

Figure 1. Posteroanterior chest X-ray. Bilateral consolidations in lower lung zones, more extensive on the left side

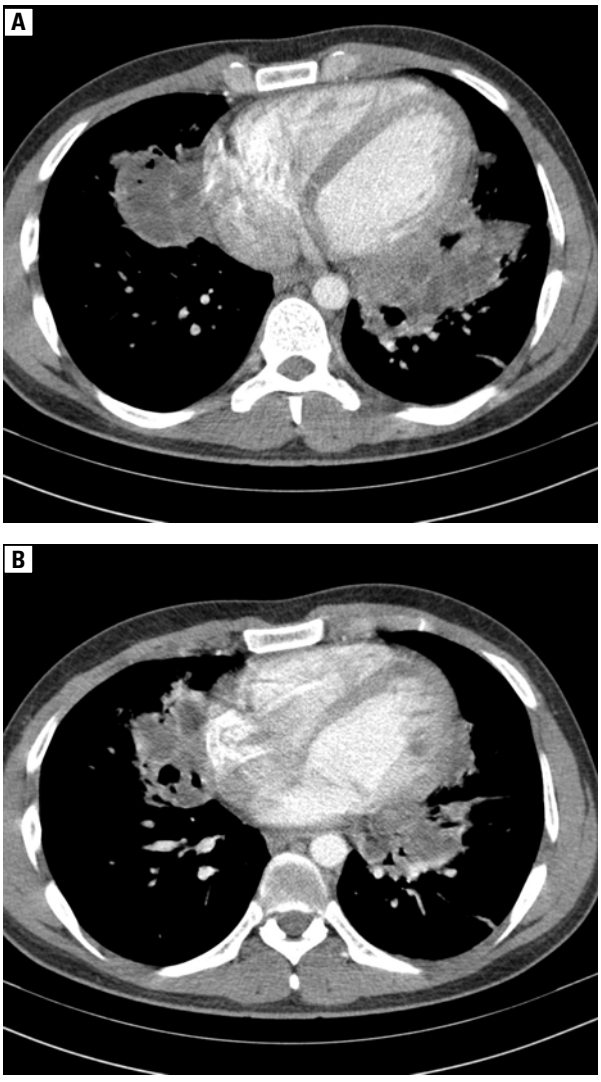

Figure 2. A, B. CT — mediastinal window. Pneumatoceles within consolidative lesions in the middle and left lower lobes

He was treated with oxygen and with the broad-spectrum antibiotics. After a few days he started to expectorate a lot of transparent sputum. The high temperature, 39−38.5 degrees of Celsius had still lasted for 7 days. The results of bacteriological examination of blood and sputum were negative.

After one week he was admitted to the National Institute of TB and Lung Diseases in Warsaw. On admission he had exertional dyspnea, dry cough, sometimes with expectorating of small amount of transparent sputum. He was feeling better than one week earlier.

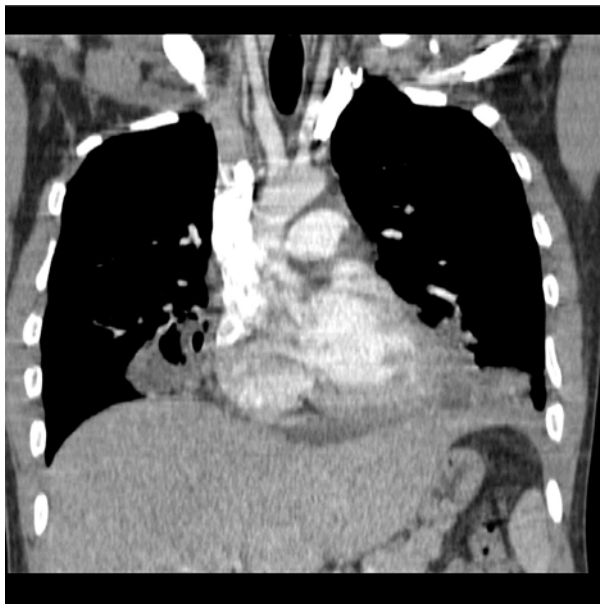

Figure 3. CT — mediastinal window, coronal view, confirming the presence of pneumatoceles within extensive consolidations with low-attenuation areas bilaterally in the lower parts of lungs

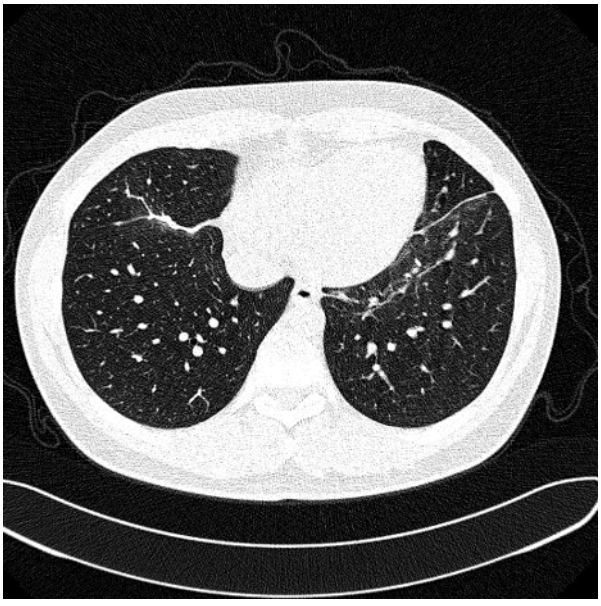

Figure 4. CT — after three months near — complete regression of lung consolidations

On auscultation crackles were heard in the lower parts of lungs, predominantly on the left side. In blood gases normalization of values was observed, PaO_2 was 78.4 mm Hg, $PaCO_2$ 42.1 mm Hg, pH 7.4, HCO_3 27.6 mmol/l. Spirometry volumes were also higher than before, FVC was 4.29 l, 72% of the predicted value, FEV_1 3.6 l, 73% of the predicted value. WBC was 7.96×10^6 /mm^3, neutrophil count 74%. CRP was only slightly elevated up to 27.2 mg/l (normal value up to 10 mg/l). During bronchoscopy a lot of transparent foamy secretion was found in the bronchial tree. Cytological examination revealed granulocytes and epithelial cells. Bacteriological examination of bronchial secretion was negative. The patient had intensive respiratory rehabilitation (bronchial drainage) two times per day.

Further improvement was soon observed. PaO_2 increased to 85.4 mm Hg, $PaCO_2$ was 44 mm Hg, Ph 7.4, HCO_3 25 mmol/l. In control spirometry FVC was 5.09 l — 86% of predicted value, FEV_1 4.91 l — 100% of predicted value. CRP < 5 mg/l. WBC and neutrophil count had normalized. The patient was discharged after two weeks, continuation of rehabilitation at home was indicated.

He had control examinations after three months in November 2011. He did not complained of any symptoms. On lungs auscultation normal breath sounds were heard. Spirometry and blood gases were within normal limits. Chest x-ray and HRCT showed nearly complete regression of lung consolidations (Fig. 4).

Discussion

The fire-eaters use diferent volatile petroleum derivatives such as kerdan which is composed of tolouene, xylene, ethylbenzene, propylbenzen, and methylbenzene, they use also kerosene or other individually prepared mixtures [1, 2, 5, 6].

Hydrocarbon pneumonitis is caused by aspiration of volatile hydrocarbons which are characterized by low viscosity and low surface tension. The LHs diffuse very quickly to the alveoli causing severe inflammatory response of lung with impairment of the surfactant effectivness [1, 2, 5, 6, 9–11].

Hydrocarbons characterized by high viscosity and high surface tension like mineral oil used for example in laxatives or nasal droplets tend to cause exogenous lipoid pneumonia. The fire-eater's pneumonia is a different disorder from exogenous lipoid pneumonia although some similarities in histologic picture can be observed [1, 2, 5, 6, 9–11].

An acute hydrocarbon pneumonitis occurs predominantly in infants or elderly persons after accidental or erroneous aspiration or ingestion of petroleum-based products like lamp oil or kerosene [8, 12, 13].

Severe hydrocarbon pneumonitis was also diagnosed in an active soldier after accidental aspiration of jet fuel, and in a person siphoning diesel oil from the container [5, 14].

Some patients intentionally aspirated or ingested some volume of liquid LHs in attempt to

commit suicide, one of those patients drank some amount of insecticide fluid containing petroleum as a solvent [7].

We present the clinical history of a young teacher who aspirated lighter fluid during an amateur fire-eater's performance. He had severe weakness, and some shortness of breath just after the show. A few hours later his status deteriorated, he had severe symptoms such as high temperature, midsternal pleuritic pain, myalgia of back, weakness, severe dyspnea, and mild dry cough. Symptoms usually occur during the first 12 hours after aspiration of liquid hydrocarbons and can vary from mild to the life-threatening [1, 2, 11]. According to the report of Gentina *et al.* [1] who analyzed clinical history of the 17 fire-eaters symptoms were as follows: pleuritic pain (100%), fever (97%), dyspnea (97%), cough (70.55), hemoptysis (26.5%). The severe symptoms usually have lasted for one week.

Radiologic lesions appear after 30 min up to 12 hours after aspiration [1, 6]. Chest x-ray shows local or diffuse perihilar or basal opacities predominantly in lower lobes and in the middle lobe like in a case of aspiration pneumonia [1, 6]. The most often seen lung lesions on CT scans of fire-eaters are pneumotoceles (well-defined cavitary lesions) within consolidative or ground-glass opacities [1, 2, 6, 15]. The other lung lesions like pleural effusion, pneumothorax, pneumomediatinum and broncho-pleural fistula are sparsly observed [1, 2, 3, 14].

On HRCT scans of our patient consolidations with decreased attenuation and pneumatoceles were observed. The radiologic lesions have to be differentiated mainly with inflammatory lesions and with neoplastic disease [1, 2, 5, 7].

The obstructive ventilatory pattern with significant decrease of vital capacity was observed in our patient. After about two months normalization of ventilatory values was found. However in children after hydrocarbon pneumonitis lung function tests had remained obstructive even for 8–14 years after ingestion or aspiration of liquid hydrocarbons [6, 16].

Franzen *et al.* [2] stated that in the cases of FEL there are rather no indications for bronchoscopy and bronchoalveolar lavage when quick regression of lesions is observed. Gentina *et. al.* [1] reported that BAL was performed in 3 out of 17 fire-eaters and lipoid-laden macrophages with vacuoles were not detected. However according to the report of Brander *et. al*. [6] vacuolated macrophages may be seen in a case of toxic lung injuries [6]. These findings were in agreement with results from electron microscopic examination of BAL performed in a patient with hydrocarbon pneumonitis published by Burkhardt *et al.* [17]. They found features of lipoid-containing inclusions in macrophages indicating an increased activity of macrophages that led to increased release of cytokines which could trigger prolonged inflammatory response. Histopathologic examinations of lung in the experimental studies after hydrocarbon aspiration revealed the acute phase within 24 hours,characterized by lung inflammatory cells infiltration with edema, and the chronic phase within 1 to 2 weeks after aspiration or inhalation of LHs manifested by proliferative bronchiolitis, and lung fibrosis [6, 16].

The diagnosis of the fire-eater's pneumonia is based on the information on the recent exposure to volatile hydrocarbons, clinical presentation , and on the results of radiologic examinations [10, 15].

The optimal treatment of FEL has not been established. Antibiotic therapy is controversial but the majority of published patients were treated with antibiotics when inflammatory markers were elevated [2, 5, 6, 15]. Our patient was also treated with the broad–spectrum antibiotics although the results of bacteriological examination of blood and bronchial washings were negative. During the first week severe symptoms had still lasted irrespective of antibiotic therapy. Then after 1 week the patient felt better, and the inflammatory markers had decreased. In the severe ill patients systemic corticosteroid therapy is often administered [5–7]. Inhalation or aspiration of large amount of LHs sometimes leads to acute lung injury requring treatment with mechanical ventilation [1]. The prognosis in these cases is concerned to be severe. The mortality rate is assesed for less than 1% [6]. However the overall prognosis is favorable. The lung lesions in the majority of patients resolve spontaneously between a few weeks up to 3 months [1, 2].

Conclusions

The fire-eaters are at occupational risk for hydrocarbon pneumonitis called the fire-eater's pneumonia. After aspiration of some volume of pyrofluid performers have severe symptoms. Radiologic lesions are localized mainly in the lower and middle lobes like in aspiration pneumonia. The well-defined cavitary lesions (pneumatoceles) within consolidative or ground–glass opacities are the most often seen lesions in hydrocarbon pneumonitis. The patients are predominantly treated with antibiotics because the fire-eater's lung

resembles bacterial pneumonia, however results of bacteriological examinations are usually negative. The corticosteroid therapy is advised in severe course of hydrocarbon pneumonitis with the clinical appearance of respiratory failure. The fire-eater's lung lesions in the majority of patients resolve spontaneusly between a few weeks up to 3 months.

The presented patient is a young man, a teacher, who decided to try to be a fire-eater. The performance was complicated by aspiration of liquid hydrocarbons. Young adults should be warned about the consequences of such trials.

Acknowledgements

We are grateful to dr Dawid Janasiak from Antoria Chemie for information on the physicochemical properties of grill lighter fluid.

Conflict of interest

The authors declare no conflict of interest.

References:

1. Gentina T, Tillie-Leblond I, Birolleau S et al. Fire-eater's lung: seventeen cases and a review of literature. Medicine 2001; 80: 291–297.
2. Franzen D, Kohler M, Degrandi C et al. Fire-eater's lung: retrospective analysis of 123 cases reported to a National Poison Center. Respiration 2014: 87: 98–104.
3. Kakadal F, Uysal M, Gulhan N et al. Fire-eater's pneumonia characterized by pneumatocele formation and spontaneous resolution. Diagn Interv Radiol 2010; 16: 201–203.
4. Karacan O, Yilmaz I, Eyuboglu O. Fire-eater's pneumonia after aspiration of liquid parafin. The Turkish Journal of Pediatrics 2006; 48: 85–88.
5. Aboudara M,Joon Y.A case of Fire-eatere's pneumonia in an active-duty soldier. Med Gen Med 2006; 8: 67–70.
6. Brander P, Taskinen E, Stenius-Aarniala B. Fire-eater's lung. Eur Respir J 1992; 5: 112–114.
7. Ishimatsu K, Kamitani T, Matsuo Y et al. Exogenous lipoid pneumonia induced after aspiration of insecticide. J Thorac Imaging 2012; 27: W 18–W20.
8. Gerbeaux J, Couvreur J, Tournier G, Lesage B. Une cause rare de penumatocele chez l'enfant: l'ingestion d'hydrocarbures (kerdane). Ann Med Interne (Paris) 1971; 122: 589–596.
9. Haas C, Lebas F, Le Jeunne C et al. Pneumopathies caused by inhalation of hydrocarbons: apropos of 3 cases. Ann Med Intern 2000; 151: 438–447.
10. Mylonaki E, Voutsas V, Antoniou D et al. Hydrocarbon pneumonitis following liquid paraffin aspiration during a fire-eating performance: a case report. Journal of Medical Case Reports 2008; 2: 214–216.
11. Marchiori E, Zanetti G, Mano C, Hochhegger B. Exogenous lipoid pneumonia. Clinical and radiological manifestations. Respiratory Medicine 2011; 105: 659–666.
12. Gotanda H, Kameyama Y, Yamaguchi Y et al. Acute exogenous lipoid pneumonia caused by accidental kerosene ingestion in an elderly patient with dementia: a case report. Geriatr Gerontol Int 2013; 13: 222–225.
13. Marandian M,Youssefian H,Saboury M et al. Accidental hydrocarbon ingestion in children. Clinical, radiologixcal, biological, and pathological findings. Ann Pediatr (Paris) 1981; 28: 601–609.
14. Hadda V, Khilnani G.Lipoid pneumonia: an overwiev. Expert Rev Respir Med 2010; 4: 799–807.
15. Franzen D, Kohler M. Severe pneumonitis after fire eating. BMJ Case Reports 2012; 10: 1–3.
16. Brown J, Burke B, Dajani A. Experimental kerosene pneumonia: evaluation of some therapeutic regimens. J Pediatr 1974; 84: 396–401.
17. Burkhardt O, Merker H-J, Shakibaei E et al. Electron microscopic findings in BAL of a Fire-eater after petroleum aspiration. Chest 2003; 124: 398–400.