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Spontaneous pneumothorax as a complication of chronic Jet propulsion fuel-8 exposure

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ABSTRACT

Jet Propulsion Fuel 8 (JP-8) is a kerosene based fuel commonly used in aviation. Occupational exposure to JP-8 may lead to negative health outcomes, which were described in a small number of studies. We report a case of 33-year-old Caucasian male veteran with a history of JP-8 exposure who presented with chronic dyspnea and recurrent spontaneous pneumothorax. To our knowledge, this is the first case of chronic inhalation injury from JP-8 exposure complicated with recurrent secondary spontaneous pneumothorax.

Introduction

Jet propulsion fuel 8 (JP-8) is a kerosene-based fuel that is widely used for aircraft by the United States (US) military. Due to its versatile use, it has become a common occupational exposure for military workforce. Several nonspecific respiratory symptoms were found to be related to chronic JP-8 exposure.[1] However, its association with pneumothorax and dyspnea has not be documented in the literature. We report a case involving a 33-year-old gentleman, a military veteran, who developed spontaneous pneumothorax and prolonged dyspnea after JP-8 exposure.

Case presentation

A 33-year-old, caucasian male veteran, who worked at a military air base, and had no past medical history, presented to the emergency department (ED) with sudden onset, right sided, sharp, pleuritic chest pain for 3 h duration. The pain developed while he was eating dinner with his family. He had no history of smoking or illicit drug use and had no family history of heart or lung disease. On review of systems he reported intermittent dry cough weeks prior to admission.

On examination, the patient's vital signs were stable, but he appeared in mild distress. His lung exam revealed decreased breath sounds over the right hemithorax. Chest x-ray showed moderate to large right pneumothorax (Fig. 1) with no mediastinal shift, pleural effusion or consolidation. Chest tube placement was recommended at that time but the patient left against medical advice.

The next day, he presented back to the ED with minimal symptomatic improvement. Chest x-ray was repeated, and it showed a complete right side pneumothorax. He underwent needle thoracostomy and subsequent chest tube placement (Fig. 2). Repeat chest x-ray showed re-expansion of the right lung, and the patient endorsed symptomatic improvement. The chest tube was removed three days later and he was then discharged home.

Twenty eight days after his discharge, the patient presented to the ED again for right sided chest pain which he had for 2 days. Once again he was found to have decreased breath sounds on the right hemithorax and his chest x-ray revealed a right sided pneumothorax (Fig. 3). Vital signs were stable. He then underwent a second chest tube insertion. Bronchoscopy was performed, which demonstrated a normal endobronchial examination. He then underwent right-sided mechanical pleurodesis using video assisted thoracoscopic surgery (VATS). No blebs or subpleural bullae were seen. The surgery was successful, uncomplicated, and the patient was subsequently discharged home.

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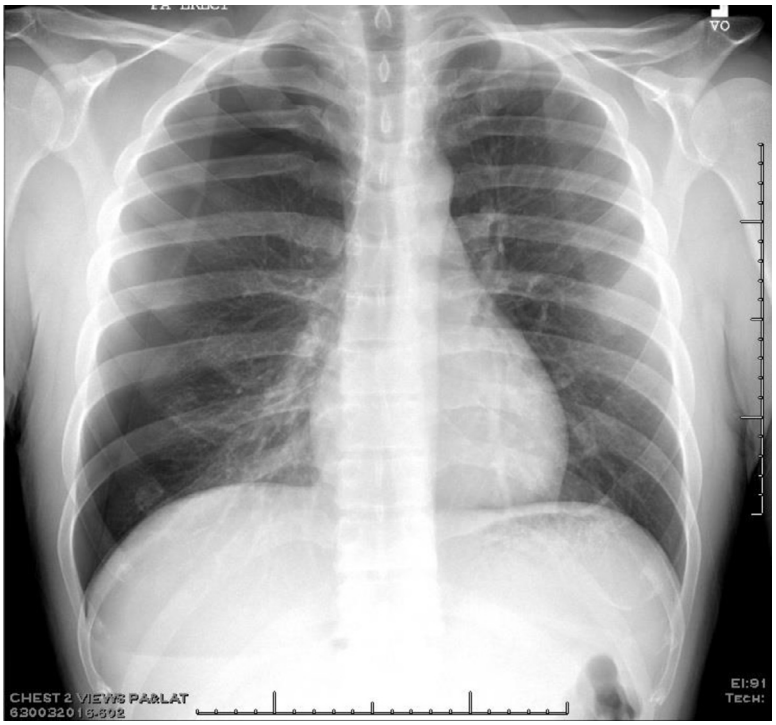

Fig. 1. Chest X-ray showing a large right-sided pneumothorax.

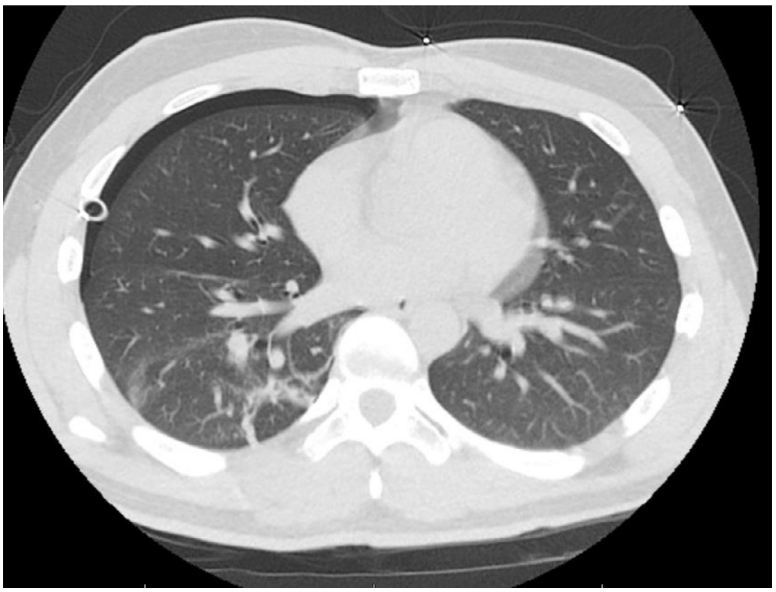

Fig. 2. Axial computed tomography image of the chest showing re-expansion of the right lung after insertion of a chest tube.

One month after surgery, he followed up with his primary care provider (PCP) and stated that he had been suffering from anosmia for over 10 years, without a known etiology; He also reported a history of mild, slowly progressive exertional dyspnea that started around the same time, for which he did not seek medical advice. At that point he was referred for magnetic resonance imaging (MRI) of the brain, which was normal. He was also referred to the Ear, Nose and Throat service, and his symptoms were thought to be related to chronic allergies.

Upon follow up with his PCP four months later, he endorsed worsening dyspnea on exertion, right sided chest pressure, and a decline in his exercise tolerance from baseline to being short of breath after walking one block. Computed tomography angiography (CTA) of his chest was done, and it showed no pulmonary embolism (PE).

At this point he was referred to the pulmonary service, where he continued to report persistent dyspnea on exertion and decreased exercise tolerance. A repeat computed tomography (CT) scan without contrast of his chest showed mild emphysematous changes, but no pneumothorax (Fig. 4). Pulmonary function test (PFT) showed no obstruction, restriction, or gas transfer defect (Table 1). His ventilation/perfusion (V/Q) scan was normal. Echocardiogram showed ejection fraction (EF) > 55% with no impairment. Alpha-1 Antitrypsin level was normal. Upon further investigation on his history, the patient endorsed an inhalational exposure to Jet-Propulsion fuel 8

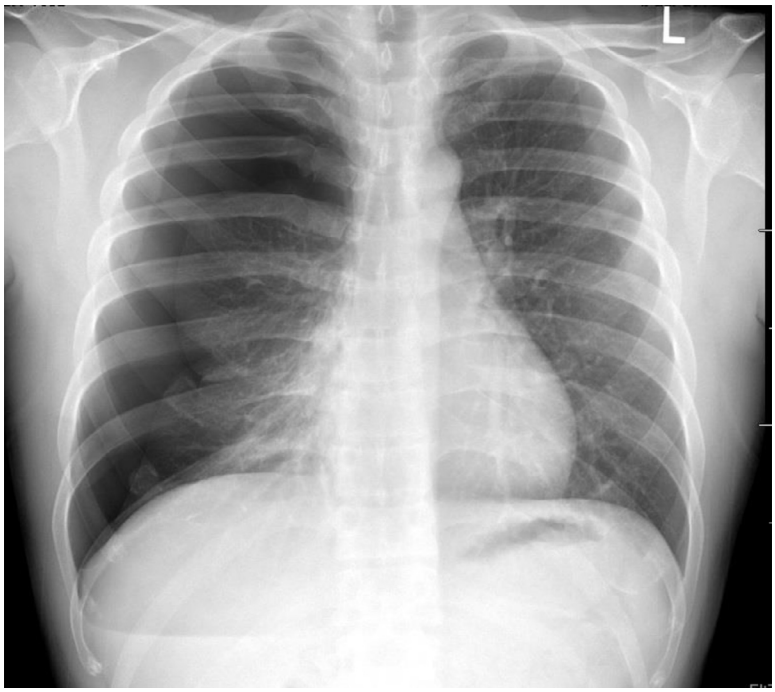

Fig. 3. Chest X-ray showing recurrence of the right-sided pneumothorax.

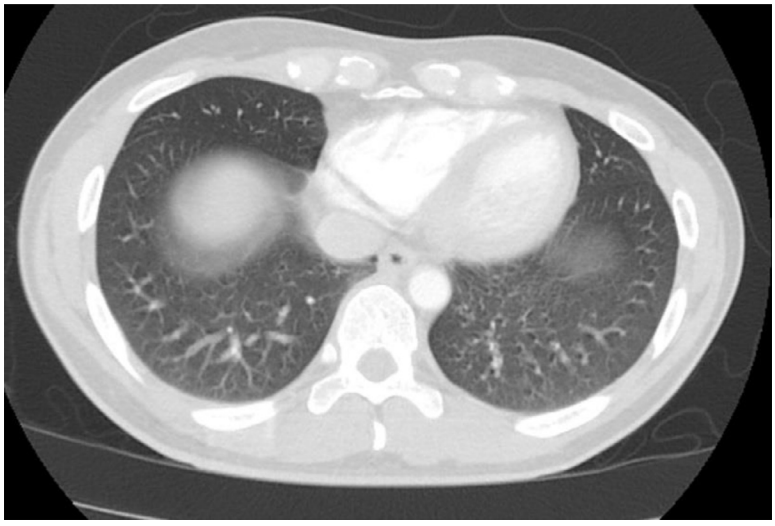

Fig. 4. Axial computed tomography image of the chest showing emphysematous changes at the lung bases bilaterally.

Table 1
Pulmonary function test

Parameters	Results (Value, % predicted)
FVC (L)	4.64, (91%)
FEV1 (L)	3.63, (86%)
FEV1/FVC ratio	0.78
VC (L)	4.70, (92%)
TLC (L)	5.74, (86%)
RV (L)	1.04, (63%)
DLCO (mm/Hg/min)	31.2, (92%)

(JP-8) from 2001 to 2005. He used to work as military police, guarding aircrafts at a military air base, where JP-8 was used as a fuel for aircrafts and heaters. He reported exposure to JP-8 on a daily basis, both in open air and confined spaces, and was not wearing any personal protective equipment to prevent that exposure. After evaluation of our patient's data, and thorough literature search, we concluded that our patient had lung injury most likely related to JP-8 exposure. We advised our patient to continue with routine pulmonary follow up.

Discussion

Spontaneous pneumothorax can be subclassified as primary (occurring in patients without clinically apparent lung disease) or secondary (as a complication of pre-existing lung disease). Primary spontaneous pneumothorax has an estimated incidence of between 7.4 and 18 cases per 100,000 population per year among men and between 1.2 and 6 cases per 100,000 population per year among women. It typically occurs in thin, tall men between the ages of 10 and 30 years old with no apparent triggers. The risk increases if the

patient has a family history of pneumothorax, exposure to cocaine, heroin, or cigarette smoking.

Secondary spontaneous pneumothorax has an incidence of approximately 6.3 cases per 100,000 population per year among men and 2.0 cases per 100,000 population per year among women. And usually affects older individuals (age, 60 to 65 years). Chronic obstructive pulmonary disease and *Pneumocystis carinii* pneumonia in human immunodeficiency virus (HIV) infected patients are the most common conditions associated with secondary spontaneous pneumothorax.[2]

Spontaneous pneumothorax from chronic exposure to JP-8 has not been reported in the literature. Our patient developed two episodes of spontaneous pneumothorax, which most clinicians will likely diagnose him with primary spontaneous pneumothorax with recurrence. Despite the fact that our patient's history does not indicate any toxic habits or family history of pneumothorax, he does have a history of prolonged exposure to JP-8. Additionally, the patient's workup has been unremarkable except for emphysematous change on CT scan of the chest, which potentially reflects a JP-8-induced pathology given that the patient is a non-smoker and his alpha-1 antitrypsin level is normal. It is most likely that our patient has suffered from a secondary spontaneous pneumothorax, which can be attributed to chronic inhalational lung injury from exposure to JP-8.

The pulmonary effects of JP-8 have been investigated through multiple animal studies, and most of them demonstrated cellular and structural damage in the lungs. In one study, experimental rats were exposed to high dose (813−1094 g/m^3) or low dose (500 mg/m^3) JP-8 aerosol for 1 h per day for 7, 28, or 56 days, and rats in all groups showed interstitial edema from endothelial damage, thickening of alveolar septa, increase in alveolar permeability and number of macrophages.[3] In a later study by Wong et. al., the authors showed that JP-8 inhalation resulted in structural damage to the extended alveolar area involving cytoplasmic vacuolization and apical membrane blebs. The results from both studies illustrate how JP-8 can potentially induce bleb formation from damaged alveolar wall, which is believed to be a major cause of pneumothorax.[4] This may provide an explanation to our patient's symptoms and recurrent pathology. It is important to emphasize that the available literature has studied toxicology profile of JP-8 in subjects with maximum exposure time of 90 days, while our patient was exposed for a total period of 6 years, from 2001 to 2005, 5 days a week.

Alveolar septal thickening signifies chronic inflammation and fibrosis, which can be a result of increased macrophages and interstitial edema that are seen in rats that are exposed to JP-8.[3] These pathological changes are typically seen in patients with chronic obstructive pulmonary disease (COPD), especially in those with emphysema.[5] Our patient, who has no exposure to cigarette smoke, no family history of pulmonary disease and normal alpha-1 antitrypsin level, showed emphysematous changes on CT scan, an uncommon finding at his age. This radiologic finding is likely attributed to a JP-8 induced chronic lung injury which resemble early emphysema despite normal pulmonary function test results.

The progressive dyspnea after pneumothorax is another pattern that is not commonly seen in this patient's age group who have history of pneumothorax. In a study by Robb et al., JP-8 fuel exposure was shown to be highly toxic to alveolar type 2 epithelial cells of rats.[6] The authors explained that the cytotoxic effects of JP-8 on type 2 alveolar cells of rats could be accounted for and potentially subdued by inhibiting the apoptosis cascade to reduce JP-8 toxicity. Type 2 alveolar cells are important for surfactant production and lung repair by differentiating into type 1 alveolar cells.[7] In our case, recurrent pneumothorax may have triggered an irreversible damage to patient's altered lung structure. His decreased type 2 alveolar cells may have led to an inability to compensate for loss of type 1 cells from chronic endothelial damage, and worsened with recent lung injury from recurrent pneumothorax. As a result, his dyspnea progressed.

Anosmia can indicate an early damage to a patient's nose through JP-8 exposure. According to a dosimetry and exposure assessment of JP-8, 60% of JP-8 aerosol particle will deposit in the nose through nasal breathing, and 80% will deposit in pulmonary region upon oral breathing.[8] Continuous inhalation of JP-8 may result in production of toxic metabolites of toluene and xylene, which can be produced by enzymes in the respiratory tracts.[8] The accumulation of those toxic metabolites may cause damage at the deposited site. In a study by Carpenter et al., six human volunteers were exposed to kerosene at 20 ppm for 15 min, and 3 reported slight olfactory fatigue.[9] It is likely that the patient suffered olfactory damage from chronic inhalation injury from JP-8 exposure.

The number of studies on the direct pulmonary effects of JP-8 exposure on humans is very limited. The existing studies are mainly reviews from medical records, observations or questionnaires from subjects who have been exposed to JP-8 fuel, and they all lack the quantitative exposure assessment and sufficient sample size.[10] There is no established connection between a clinically significant sequelae and chronic JP-8 exposure, especially JP-8-induced pneumothorax and chronic dyspnea. To our knowledge, this is the first reported case of secondary spontaneous pneumothorax likely due to chronic exposure to JP-8.

Conclusion

Although most of the studies on exposure to JP-8 fuel do not show any clinically significant sequelae, JP-8 fuel can still be potentially harmful to humans. Our patient likely developed pulmonary damage from prolonged exposure to JP-8, and ultimately developed secondary spontaneous pneumothorax and chronic dyspnea. It is important to remain cautious when handling JP-8 fuel, and appropriate measure should be carried out to protect workers who will be exposed to this fuel in any workplace.

Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.hrtlng.2018.08.001.

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