CASE REPORT OF ELECTRONIC CIGARETTES POSSIBLY ASSOCIATED WITH EOSINOPHILIC PNEUMONITIS IN A PREVIOUSLY HEALTHY ACTIVE-DUTY SAILOR

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Abstract—Background: Electronic cigarettes (e-cigarettes) are a technology that has been touted as a safe and effective alternative to traditional cigarettes. There is, however, a paucity of literature showing the adverse outcomes of e-cigarettes and a correlation with acute eosinophilic pneumonia (AEP). Objective: To present a possible association between e-cigarettes and AEP. Case Report: A 20-year-old previously healthy man was found to develop AEP after smoking an e-cigarette. He was treated with antibiotics and steroids and his symptoms improved. Conclusion: Though an alternative to traditional cigarettes, e-cigarettes can have unpredictable and potentially serious adverse effects. More research needs to be conducted to determine their safety. If seeing a patient in the ED with pulmonary symptoms after use of e-cigarettes, AEP should be considered in the differential. Published by Elsevier Inc.

Keywords—electronic; cigarette; smoking; eosinophilic; pneumonitis

INTRODUCTION

Electronic cigarettes (e-cigarettes) can be marketed for therapeutic purposes (tobacco cessation) and are Food and Drug Administration (FDA) regulated (1). In the past few years there has been a viral marketing campaign by manufactures to promote e-cigarettes as “healthy alternatives” to smoking (2). Different models have different mechanisms, but essentially there are five components to the e-cigarette: a metal sheath, atomizer, cartridge, smart chip, and lithium battery (2). The metal sheath houses the entire apparatus, and the cartridge contains nicotine and other materials to be aerosolized. The atomizer is responsible for changing a liquid to a gaseous form for inhalation, and the lithium battery powers the apparatus. The smart chip acts as the mechanical interface and relay point between the user and the atomizer. Propylene glycol can be used to create a smoke effect. One of the breakdown products after atomization of propylene glycol is diethylene glycol, which has associations with carcinogenesis, as seen in one FDA study (2). Diethylene glycol has been implicated as a contributing factor to various forms of pulmonary disease, including inflammation, infection, and neoplasm. Some of the other impurities that can be found in the e-cigarette are cotinine, anabasine, myosmine, and β-nicotyrine; there have been no data to support a direct causal relationship to these molecules and adverse clinical outcomes (2).

CASE REPORT

A 20-year-old active-duty healthy male sailor presented to the Emergency Department (ED) for the chief complaint of 3 days of persistent cough, shortness of breath, and facial flushing. The patient noted the onset of the...
symptom cluster beginning 1 h after smoking an e-cigarette 3 days prior. The patient denied supplement use and had no history of exposure to any pulmonary irritants while aboard the ship he was stationed on (USS Bunker Hill [CG-52]). The patient had deployed to Southeast Asia 1 year prior, but never to the Middle East, and denied any illness during his deployment. The patient presented to his primary care manager, who started him on albuterol, and there was no improvement in his symptoms. While en route to the ED, the patient had another e-cigarette, with exacerbation of his symptoms.

The patient’s initial vitals demonstrated mild hypertension, with blood pressure of 140/78 mm Hg, tachycardia at 128 beats/min, tachypnea at 32 breaths/min, with an O2 saturation of 100% on room air, yet was afebrile at 36.9°C (98.4°F). The patient’s heart rate was elevated without murmurs, rubs, or gallops. The patient’s lungs were clear to auscultation bilaterally and he was speaking in full sentences without signs of respiratory distress. An electrocardiogram showed sinus tachycardia without dynamic changes, delta waves, or an S1Q3T3 pattern. The patient had a mild leukocytosis of 13.0 (1000 cells/µL), 82.1% neutrophils, 6.8% lymphocytes, 8.2% monocytes, 2.0% eosinophils, and 0.9% basophils. Because the patient had shortness of breath, tachycardia, and a family history of pulmonary embolism, a chest x-ray study was obtained. The results demonstrated “subtle diffuse patchy reticulonodular opacities” (Figure 1). A computed tomography (CT) scan of the chest was ordered to further characterize the opacity seen on the chest x-ray study and to evaluate for pulmonary embolism. The final read of the chest CT showed “no evidence of pulmonary embolism with predominantly diffuse ground-glass opacities involving the upper and middle lobes of the lungs more than lower lobes … Considerations include opportunistic infection such as pneumocystis jirovecii versus atypical or viral pneumonias. Acute alveolar disease such as diffuse alveolar hemorrhage, drug toxicity, or hypersensitivity pneumonitis is on the differential … Possible tuberculosis cannot be excluded” (Figure 2). The patient was admitted to the hospital and started on 1 gm of ceftriaxone and 100 mg of doxycycline for initial treatment of community-acquired pneumonia. Concern for tuberculosis (TB) prompted the pulmonary service to perform a bronchoscopy. The bronchial alveolar lavage demonstrated “abundant macrophages, eosinophils, and scattered benign respiratory epithelial cells. There are no chunky eosinophilic inclusions identified in the macrophages … The right upper lobe cell count yielded 3268 WBCs [white blood cells] with 3% neutrophils, 2% basophil, 17% macrophages and 74% eosinophils.” The pulmonary report suggested no evidence of bacteria, viruses, fungus, parasites, or neoplasm via bronchoscopy, culture, and serum laboratory tests. No other infectious etiologies were found, including TB, Aspergillus, Nocardia, herpes simplex virus, influenza, parainfluenza, Legionella, and cytomegalovirus; and the diagnosis of acute eosinophilic pneumonia (AEP) was made. The patient was started on 60 mg of prednisone and was discharged from the hospital on hospital day 5 with improvement in his symptoms. The patient had a follow-up 2 days later with Pulmonology, at which time he stated he was feeling better and that his symptoms had almost completely resolved. A repeat chest x-ray study performed 1 week later showed significant interval improvement of the left lung base opacity (Figure 3).
AEP is an idiopathic inflammation of the alveoli and intima first discovered in 1989. An article by Allen and Davis suggests a correlation between inhaled antigenic triggers and the onset of AEP (3). AEP is defined by fever lasting < 5 days, diffuse infiltrates on chest x-ray study, bronchoalveolar lavage results > 25% eosinophils, no concomitant infection (bacterial, fungal, or viral), improvement with steroids, and no recurrence of symptoms after steroid use. Symptoms often progress rapidly, and most patients develop respiratory failure requiring intubation and ventilatory support. Plain chest radiographs will usually demonstrate diffuse infiltrate, and CT can reveal diffuse alveolar infiltrates, pleural effusions, and no lymphadenopathy. Most patients will not have an elevation of serum eosinophils. Pulmonary function tests demonstrate a restrictive pattern, and lung biopsies demonstrate eosinophilia and edema in the alveoli, bronchi, and intima. Patients will usually respond to intravenous steroids, methylprednisolone in particular, within 1–2 days (3).

There is a paucity of literature linking e-cigarettes with AEP. Several case reports exist for the association of smoking traditional cigarettes, but not e-cigarettes. One such case involves a young healthy male who developed AEP from smoking one pack of traditional cigarettes. He was intubated, admitted to the hospital, and started on azithromycin and ceftriaxone without improvement. A chest CT scan demonstrated bilateral diffuse infiltrates, and bronchoscopy revealed many white blood cells with eosinophilia in the lavage. Antibiotics were discontinued, and the patient was started on methylprednisolone, with symptoms resolving in 2 days (4). Another study suggesting a link between AEP and traditional smoking reviewed 18 cases presenting in 2003–2004 of AEP in previously healthy active duty soldiers deployed to Iraq. All patients were smokers, and more than 75% had just recently starting using tobacco products. All were treated with steroids and showed complete resolution of symptoms without recurrence of disease (5). However, our patient, in particular, was not deployed to the Middle East.

A study reported earlier this year demonstrated acute adverse pulmonary effects in e-cigarette users similar to traditional smokers. Within the first 5 min of smoking an e-cigarette, pulmonary function tests were obtained showing an increase in pulmonary impedance, resistance, and a decrease in forced expiratory nitric oxide (6). Although this study does not demonstrate an association with AEP, it does lend credence to the notion that e-cigarettes adversely affect pulmonary physiology, as suspected in our patient.

**CONCLUSION**

E-cigarettes are an alternative to traditional cigarettes; there is a paucity of data suggesting pulmonary toxicity. Although no clear link has been made between e-cigarettes and AEP, existing data suggest there may be a possible correlation. In our patient, it is unclear what the definite cause of AEP was, but it is suggested to be his e-cigarette use. Further vigilance, monitoring, and evaluation will be needed to prove a causal relationship between e-cigarettes and AEP. When evaluating patients in the ED with respiratory distress and e-cigarette use, one may consider AEP in the differential. Further studies are needed to establish a correlation.

**REFERENCES**