UCLA Proceedings of UCLA Health

Title

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Permalink https://escholarship.org/uc/item/6m87q7v6

Journal Proceedings of UCLA Health, 24(1)

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Publication Date 2020-04-14

A Case of E-Cigarette, or Vaping, Product Use-Associated Lung Injury

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Introduction

E-cigarette, or Vaping, Product-Use Associated Lung Injury (EVALI) is a spectrum of acute pulmonary injury, including hypersensitivity pneumonitis and acute respiratory distress syndrome, resulting from e-cigarette use.¹ Recently, there has been a rise in cases of EVALI primarily associated with vaping THC oil, though nicotine and other products have been implicated. We present a case of a patient who recently began vaping THC oil and subsequently developed acute respiratory issues concerning for EVALI.

Case Presentation

A 49-year-old male with no past medical history was referred to the emergency room by his primary care physician for cough and shortness of breath. He reported developing generalized abdominal pain and diarrhea six days before. Over the ensuing days he felt increasingly weak with decreased appetite and oral intake. He also developed a nonproductive cough and chest tightness. He denied any sputum production or hemoptysis. He had no sick contacts or recent travel. He did admit to previously smoking marijuana and recently switching to vaping THC oil.

On presentation to the emergency room he was febrile with a temperature of 39.3°C and tachycardic with a heart rate of 117. He had tachypnea with a respiratory rate of 31/min. He was also hypoxic with pulse oximetry of 81% on room air. He appeared fatigued but comfortable. Physical exam was notable for rhonchi in the midlung fields. He had normal heart sounds but was tachycardic. Jugular venous pulse (JVP) was normal and there was no lower extremity edema. His abdomen was soft and non-tender. Laboratory evaluation was notable for an elevated white blood cell count of 13.3k/uL and platelet count 432k/uL. He was not anemic. Metabolic panel was significant for a low sodium of 131 mEq/L, chloride of 88 mEq/L and potassium of 3.3 mEq/L. Bicarbonate was normal at 25 mEq/L. Kidney function was normal with a blood urea nitrogen of 22 mg/dL and creatinine of 0.94 mg/dL. D-dimer was elevated at 2.76 ug/mL as was his procalcitonin of 2.02 ng/mL. Chest xray showed patchy bilateral diffuse air space opacities (Figure 1). CT pulmonary angiogram showed no evidence of pulmonary embolus but did show extensive ground glass opacities with septal thickening throughout the lungs consistent with diffuse pneumonitis or pulmonary edema (Figure 2). He was admitted to the inpatient medicine service for further management.



Figure 1.



Figure 2.

Additional testing after admission included Influenza A and B antigen, blood cultures, sputum culture, respiratory pathogen panel, Legionella antibody, HIV and coccidioides testing. These were all negative. Hypersensitivity pneumonitis panel was also negative. An echocardiogram showed a normal ejection fraction and normal diastolic function without valvular abnormalities. Given his recent use of a THC vaping product it was likely he had vaping associated lung injury.

Upon admission to the hospital was treated for sepsis with fluid resuscitation and broad-spectrum antibiotics with vancomycin and piperacillin and tazobactam. Patient received supplemental oxygen and after 24 hours was increasingly hypoxic requiring high flow 100% oxygen therapy. He required bilevel positive airway pressure (BiPAP) ventilator support while asleep. After 3 days, with a negative infectious workup, patient was started on methylprednisolone 40 mg intravenously three times a day. After initiation of the steroids the patient's symptoms began to improve and his oxygen requirements began to decrease. After four days of intravenous steroid therapy he was no longer requiring supplemental oxygen and his respiratory status had returned to normal. His chest x-ray also improved (Figure 3). He was discharged home in stable condition with a two-week prednisone taper.



Figure 3.

Discussion

Our patient presented in acute hypoxic respiratory failure, with past medical history significant for recent initiation of ecigarette use, primarily with THC oil. Beginning in June 2019, there was a sudden rise in cases of patients with a history of vaping presenting with various degrees of acute respiratory failure, ranging from mild hypoxemic respiratory failure to acute respiratory distress syndrome requiring mechanical ventilation support.¹ The Centers for Disease Control and Prevention began an investigation into what was ultimately named E-cigarette, or Vaping, Product-Use Associated Lung Injury (EVALI). As of January 14, 2020, there have been 2,668 hospitalized cases of EVALI reported to the CDC.¹ Eighty-two percent of cases reported the use of THC-containing products, primarily from informal sources such as family, friends, inperson and online dealers.² Our patient reported purchasing the THC oil on-line, but did not provide further details.

A recent study reported a possible link between vitamin E acetate and EVALI.³ Amongst 51 bronchoalveolar lavage fluid samples from EVALI patients, 49 contained vitamin E acetate, a synthetic form of vitamin E. Two possible mechanisms of lung injury were proposed: 1) disruption of pulmonary surfactant due to the conversion of phosphatidylcholines from gel to a liquid crystalline phase in the presence of tocopherols, and 2) ketene formation when vitamin E acetate is exposed to heat (in this case, when the oil is exposed to the heating coil inside the vaping device). Ketene, a toxic gas, is a known lung irritant.

The CDC has developed guidelines for diagnostic evaluation and management of patients with suspected EVALI.⁴ Once acute hypoxia is stabilized with supplemental oxygen support, patients should have a full respiratory infectious evaluation. Our patient was appropriately evaluated for respiratory infections including influenza, respiratory syncytial virus, and bacterial pneumonia. It is then recommended that empiric antibiotics for acute community-acquired pneumonia be initiated. Because of the severity of respiratory failure, our patient was initiated on broad-spectrum antibiotics with vancomycin and piperacillin-tazobactam. If, after 48 hours of antibiotic therapy, there is no significant improvement in respiratory symptoms, the CDC recommends considering initiation of systemic steroid therapy.⁴ Our patient was started on IV methylprednisolone with prompt improvement in symptoms. Once patients have showed clinical improvement over 24-48 hours, they may be considered for discharge to complete a prolonged oral steroid taper. Patients should have close outpatient follow-up with the primary care physician to ensure ongoing clinical improvement.⁴

Conclusion

Individuals who use e-cigarettes are at risk of developing acute lung injury, regardless of the substance that is inhaled. Any patient with a history of e-cigarette use who presents with acute respiratory failure should be evaluated for EVALI. Prompt diagnostic work-up and consideration of systemic steroid treatment may prevent any further deterioration in respiratory status. Most importantly, patients should be educated on discontinuing e-cigarette use indefinitely.

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