Cannabis Vaping-induced Lung Injury

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KEY WORDS: cannabis, lung disease, lung injury, marijuana, vaping

IMAJ 2023; 25: 360–361

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PATIENT DESCRIPTION

In September 2020, a 37-year-old man without significant medical history or medication use presented to the emergency department with shortness of breath. The patient denied any history of shortness of breath, travel history, recent sick contacts, or history of lung disease. On arrival, the patient was afebrile with a respiratory rate of 26 breaths per minute (b/m), oxygen saturation 82% on ambient air, blood pressure 130\80 mmHg, and heart rate 130 beats per minute (bpm). He was started on three liters per minute oxygen therapy, which improved his saturation to 90%. Physical examination was remarkable for tachypnea and diffuse bilateral inspiratory lung crackles. Electrocardiogram revealed sinus tachycardia. Routine laboratory investigations (complete blood count and biochemical profile) did not reveal any abnormal findings except an elevated C-reactive protein (CRP) of 6.47 (normal range 0.02–0.50 mg/dl). Arterial blood gas analysis found a pH of 7.44 (normal range 7.35–7.45), pCO_2 of 30.2 (normal range 35–45 mmHg), pO_2 37.3 (normal range 69–116 mmHg), and HCO_3 of 20.1 (normal range 22–26 mmol/L). Total immunoglobulin E (IgE) levels were elevated at 2144 (normal range 0–100 U/ml). Additional lab tests, including urine and blood cultures, human immunodeficiency virus, and a respiratory viral panel were all negative.

The patient underwent chest X-ray (CXR) that showed diffuse bilateral ground-glass opacities (GGO) [Figure 1A]. Computed tomography (CT) also showed diffuse GGO with superimposed interlobular and intralobular septal thickening (crazy paving pattern) in both lungs [Figure 1B–1D].

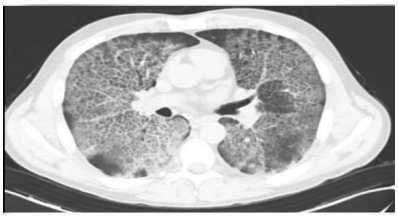
When the patient was interviewed, he denied inhaling any hazardous substances or using any new substances in general. He was subsequently hospitalized for further investigation. Due to suspected hypersensitivity pneumonitis, the patient was treated with intravenous (IV) systemic corticosteroids (prednisone 1 mg/kg) and IV ceftriaxone (2 grams daily) with no visible improvement on CXR or oxygen saturation level (SpO_2 remained at 84% on ambient air). Three days later, the patient underwent a total body CT that did not show any additional extrapulmonary pathology but did show a worsening in the GGO and acute respiratory distress syndrome. Transbronchial biopsies from the right upper and middle lobe were obtained, which revealed lung parenchyma with areas of focal lymphocytic infiltrate, accumulation of macrophages, and focal mild interstitial fibrosis. In addition, we found poorly formed noncaseating granulomas with no signs of fungi-like organisms. An immunostain for *Pneumocystis jirovecii* was negative, as was a Ziehl-Neelsen stain for acid fast organisms. Bronchoalveolar lavage (BAL) fluid analysis showed mixed inflammatory cells. On further questioning, the patient confessed to regularly smoking marijuana and reported two consecu-

Figure 1. Patient chest X-ray and computed tomography at admission

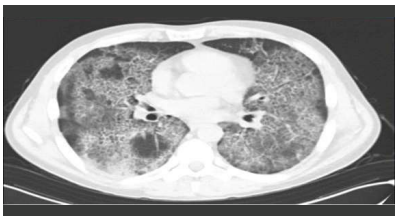
[A] Patient's chest X-ray at admission, which shows diffuse bilateral opacities (red circles).
[B-D] Patient's chest computed tomography at admission showing diffuse bilateral pulmonary infiltrates, crazy paving pattern and diffuse ground glass opacities in both lungs

[A]

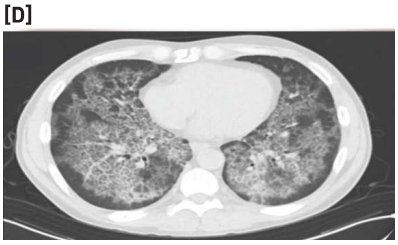
[B]



[C]



[D]



tive years of marijuana smoking (5 times per week for 2 years) as well as recently starting to vape cannabis oil. As a result of this discovery, the most probable diagnosis became lung injury due to marijuana inhalation. The patient's elevated IgE levels could partly be explained by these smoking habits, and the result from the radiology and pathology reports helped to support a diagnosis of inhaled cannabis-induced pneumonitis. The patient was discharged with home oxygen therapy and steroids, as well as follow-up with a pulmonologist. As of November 2021, he was still experiencing chest discomfort and continued oxygen desaturation.

COMMENT

In this patient with no significant medical history, the presenting condition had several potential differential diagnoses in addition to marijuana induced injury, including hypersensitivity pneumonitis, idiopathic pulmonary fibrosis, sarcoidosis, or infection. However, the lack of evidence for an infectious cause as well the patient's background and recent history of starting vaping indicated that cannabis-induced pneumonitis was the most likely diagnosis. Although medical cannabis can be used therapeutically for certain patients, recreational overuse of cannabis could cause many potential unfavorable effects. Data are still limited on the effects of vaping in particular; however, lung complications such as pneumonitis, bronchoconstriction, and irritation of the airways have been previously reported [5].

The current paucity of evidence on outcomes of marijuana use may be partially explained by a few contributing factors, including differing methods of consumption and different strains making comparison difficult. Marijuana smokers have a higher tendency for concurrent tobacco and other drug use. Most studies are retrospective or case reports [5].

CONCLUSIONS

We described a young patient who presented with severe pulmonary symptoms as a complication of inappropriate (vaping) and excessive use of cannabis. Radiographic findings include a crazy paving pattern, interlobular septal thickening, and ground glass opacities. In addition to the adverse effects, marijuana use and misuse has many negative pulmonary side effects. After the patient's first episode of severe pneumonitis, he was followed by a pulmonologist. He continued to exhibit chest discomfort and oxygen desaturation and had not completely improved.

In recent years, the use of cannabis has risen in both teenagers and adults. Various reasons are suspected, most notably the positive and euphoric feelings gained, but increasingly due to pain control and symptom management. The route of delivery is variable and includes vaping, liquid drops, and smoking. People of any age must be aware of the serious damage and complications that may result from the use and misuse of marijuana, as in our case. In some cases, misuse could lead to death. Proper education may lead to a reduction in such incidents.

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Capsule

Fibrogenic macrophages

Macrophages are key players in the response to tissue injury, and specific macrophage subsets can drive fibrosis progression or resolution. **Fabre** and colleagues showed that *CD9+TREM2+*macrophages, which are further defined by the expression of a key set of proteins, can mediate fibrosis. Analysis of human and murine hepatic and pulmonary fibrosis samples revealed accumulation of these macrophages near scar sites and proximal to activated mesenchymal cells. In vitro analysis revealed that type 3 cytokines can promote the differentiation of these macrophages, which are unable to degrade collagen I and contribute to collagen I-associated fibrosis. Blockade of these cytokines in mice was sufficient to limit fibrosis. These findings highlight a critical role for fibrogenic macrophages in this pathological process.

Sci Immunol 2023; 8: add8945

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