

Pot smokers puffing away lung health

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ARTICLE INFO

Article history:

Received 22 March 2019

Received in revised form 9 May 2019

Accepted 14 May 2019

Available online 23 May 2019

Keywords:

Marijuana
Lung injury
Steroids
Smoking

ABSTRACT

Background: Marijuana is the second most commonly used inhalational agent after tobacco. It has been used for therapeutic benefits in cancer, epilepsy, inflammation and pain. Inhalation of marijuana causes reversible and irreversible lung injury.

Case: We present a 26-year-old female with cough, chest pain, epistaxis, hemoptysis, night sweats and breathlessness few hours after smoking marijuana. Physical exam was positive for tachycardia, tachypnea, and diminished coarse breath sounds. Further investigation revealed elevated white blood cell count, chest X-ray, computed tomography of the chest showed bilateral patchy infiltrates. The patient was managed with short term steroid, as antibiotics alone did not work. Radiological improvement of lung injury was noted within 36–48 h.

Conclusion: There is a paucity of treatment guidelines for acute lung injury secondary to marijuana inhalation. We advocate early use of short-term steroids and also more awareness on quitting marijuana smoking to prevent life-threatening complications like myocardial infarction, diffuse alveolar hemorrhage and acute respiratory distress syndrome.

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Introduction

Cannabis more commonly known as Marijuana has been used in multiple civilizations for medicinal purposes for generations and is one of the most commonly used illicit substance globally. According to a report by WHO about 147 million people, 2.5% of the world population, consume cannabis annually.¹ Amongst the American population the prevalence of marijuana use has been steadily increasing with 8.9% of the population being current users. A substantial increase in the number of users was seen in 2016 alone when compared to the last decade with the highest increase seen in those over 26 years of age.²

The properties of marijuana that make it of interest in medicinal use are contributed by two cannabinoids in particular- tetrahydrocannabinol (THC) and cannabidiol (CBD). THC is known to decrease pain, inflammation, nausea and increase appetite. It is also responsible for the psychoactive effects like euphoria and heightened sensory experiences which make it a choice for drug abuse. Unlike THC, CBD does not produce the psychoactive effects but is used in medicine for

reducing pain and inflammation, possibly even treating mental illness and addictions and epileptic seizures.^{3,4} Although limited, there is experimental data regarding its use in palliative care, obstructive sleep apnea and cancer.^{5–7}

It is available in the form inhalants, candies, brownies, capsules and beverages of which the use of pipes, bongs and paper-wrapped joints are the most popular. The effect of marijuana is dictated in part by its method of consumption. As the inhalational form is the most common, several reviews and case studies in the past have tried to assess and summarize the effect of marijuana use on the lungs.^{8–10}

Case

A 26-year-old female patient presented with symptoms of sudden onset cough, chest pain, epistaxis, hemoptysis, night sweats and shortness of breath 7–9 h after smoking marijuana. The patient had a central dull achy type chest pain aggravated by inspiration and relieved on expiration. The patient also mentioned an unintentional weight loss of 10 pounds over 6 months. The patient denied prior history of smoking tobacco or other inhalational agents, oral contraceptive pills use, travel and sick contacts. Examination was significant for tachycardia with a heart rate 109 beats per minute, blood pressure 157/100 mm of Hg, respiratory rate of 26 per minute, SpO₂ 98% at 4 liters of oxygen, dried blood in external nares, bilaterally

Abbreviations: THC, Tetrahydrocannabinol; CBD, Cannabidiol

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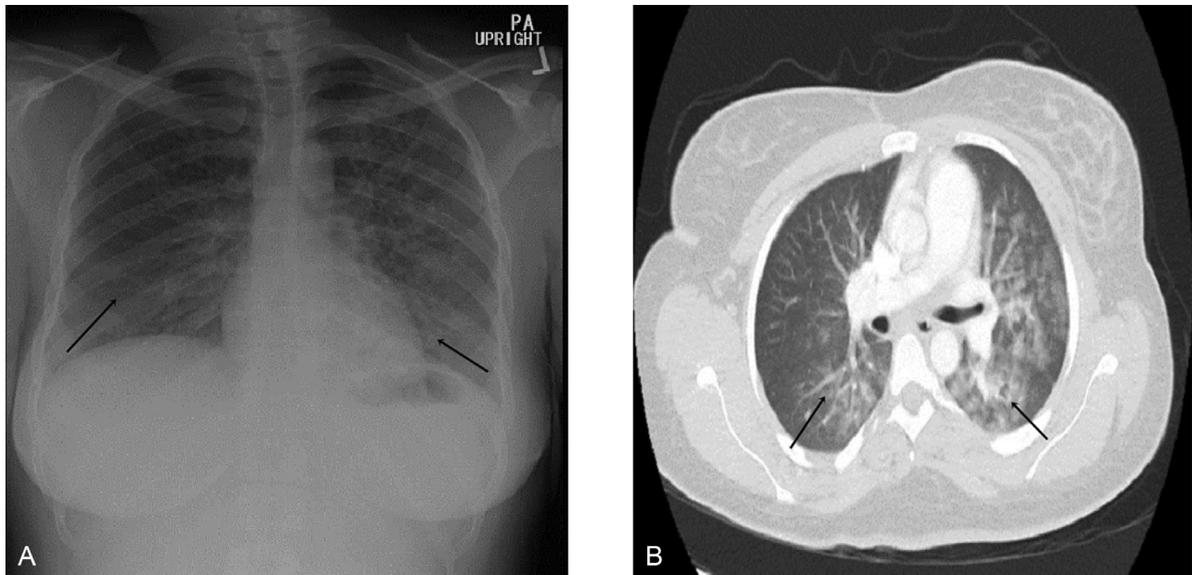


Fig. 1. (A) Chest X-Ray showing multifocal airspace opacities throughout the left lung and in the right upper lung concerning for multifocal pneumonia. (B) Computed Tomography scan of the chest in axial plane showing lesions concerning for multifocal pneumonia but no pulmonary embolus.

diminished and coarse breath sounds. Electrocardiogram did not show any acute changes and troponin were negative. Further workup showed elevated white blood cell count of 16.10 K/uL with neutrophil count 87.9%, platelet count of 154,000/cu mm, INR 1.2, and a urine drug screen was positive for marijuana. Basic metabolic profile and liver function tests were within normal limits, and the urine pregnancy test was negative. Serum inflammatory markers revealed an erythrocyte sedimentation rate of 8 mm/h, C - reactive protein at 1.08 mg/dl. Antinuclear antibodies and purified protein derivative tests were negative. Chest X-ray revealed bilateral patchy infiltrates which were confirmed on computed tomography of the chest. (Fig. 1A and B).

The patient was initially managed with supportive oxygen therapy, bronchodilator as needed and antibiotics, ceftriaxone and azithromycin, based on imaging studies but failed to show any drastic clinical improvement over the next 24 h. On day two she was started on steroids, 60 mg two times a day for the initial 3 days later switched to 60 mg once a day for 2 additional days. The five-day use of steroids resulted in significant clinical improvement.(Fig. 2). Pulmonology was consulted for possible bronchoscopy on day two of her hospital stay. Since there was a drastic clinical improvement after initiation of steroid therapy and rapid clearance of her lung infiltrates, the procedure was deferred at that point.

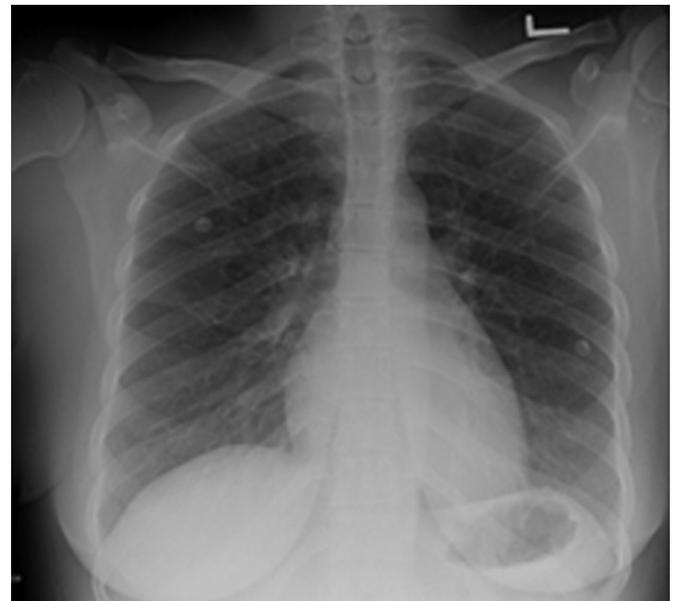


Fig. 2. Chest X-ray showed clearing of opacities within 36–48 h after initiation of treatment.

Discussion

The effects of smoking marijuana are said to be due to complex chemical substances similar to that of tobacco with the exception of nicotine.⁸ A dose-related lung dysfunction is noted which is equivalent to the effect of smoking 2.5–5 cigarettes.⁹

The effects of marijuana are a result of histopathological changes due to bronchial inflammation. Short-term use of marijuana produces symptoms of chronic cough and increased sputum production mediated by bronchodilation which is dependent on the Δ^9 tetrahydrocannabinol (THC) concentration.¹¹ Its long-term effect is a result of the epithelial cell injury and damage to alveolar macrophages predisposing to pulmonary infections due to the reduced inflammatory response.

The presentation of symptoms in patients is dependent on the method, amount and duration of inhalation making each case unique

in terms of establishing a cause. However, there are reported cases of similar presentation of acute shortness of breath and hemoptysis in patients within a few hours of cannabis use.^{12–14} As reported by Hashmi et al.¹³ the patient developed shortness of breath, cough, and hemoptysis immediately after smoking marijuana and evaluation revealed diffuse ground glass opacities. He was managed with diuretics, and a course of antibiotics as seen with the clearing of infiltrates on a repeat computed tomography (CT) of the chest. Similarly, He et al.¹² reported the development of similar symptoms in their patient after vaping cannabis oil. The patient had regularly been vaping once a week for several years and his computed tomography angiography chest revealed diffuse air space opacification. He was successfully managed with high flow oxygen and antibiotics with a dramatic improvement in the first 24-h of treatment. A repeat CT

chest after 2 weeks showed complete resolution of the lung opacities. Corticosteroids were not a part of the management in either of case reports due to a prompt response to the treatment. In our case, we initiated corticosteroid therapy early in the course of treatment as our patient did not have symptomatic relief with antibiotics alone. There was both, symptomatic and radiological improvement as early as 36–48 h. In a recent report published in *Annals*, early initiation of steroids combined with antibiotic use in a patient with documented lung injury from smoking marijuana resulted in a swift resolution, clinically as well as radiologically.¹⁴

Inhalational lung injuries could be a result of marijuana smoking, vaping, and bong pipes alone or in combination with other recreational drugs like phencyclidine, crack cocaine, formaldehyde which further increase the risk of injury to lung mucosa. The possibility of marijuana being laced with other agents adds to the challenge of diagnosis. It is imperative to initiate early treatment in these cases to prevent life-threatening complications like myocardial infarction, diffuse alveolar hemorrhage, and acute respiratory distress syndrome. There is a lack of standard management protocols for marijuana-induced respiratory trauma. The legalization of marijuana along with its increasing use for medicinal purposes will lead to increased prevalence of similar cases requiring physicians to be better equipped in managing them in the future.

In conclusion, there is limited literature regarding the medical management of lung injury secondary to marijuana use in general and the role of steroid in specific. The prompt response to treatment and shortened hospital course with the use of steroids, as in this case, further stresses the importance of exploring the role of steroid initiation early in treatment plans.

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