



Allergic Reactions to Legalized Marijuana Use in Colorado

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Abstract The state of Colorado decriminalized marijuana use in 1975, legalized its use for medical purposes in 2000, and voters approved recreational use in 2016. During this period, there has been a dearth of information about respiratory and skin reactions to marijuana use and/or exposure to the substance from working in the industry or home growing conditions. This brief report, based on patient complaints, evidences Cannabis sativa as a mild allergen. However, significant exposure can elicit marked respiratory and dermatologic allergic reactions requiring proper diagnosis and treatment. Additionally, contaminants such as pesticides or mold related to cannabis production, handling, and/or use may cause adverse reactions of a non-allergic nature treatment.

Keywords Marijuana use · Allergic reactions · Medical practice

Cannabis sativa is a weed allergen (Ocampo and Rans 2015). As such, exposure to the substance, including mold contamination, dust mites, and pesticides used for growing purposes, may result in increased allergic reactivity (e.g., IgE-mediated cannabis allergy). This condition may not receive sufficient attention by clinicians given the increase of legalization for medical and/or recreational cannabis use; and, an under-reporting of health problems among cannabis industry workers and users where the substance remains illegal. This paper describes three patient presentations in detail, with different pathophysiology addressing the spectrum of reactions that may occur to marijuana use and exposure.

Patient #1 ML was a 30-year-old white male who complained of allergic rhinitis and asthma symptoms after exposure to cannabis in 2015. There was no previous history of allergies or asthma. He relocated from Kansas to Colorado 5 years prior to work in the cannabis industry. He initially worked directly with the cannabis plants in grow facilities and ultimately was

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promoted to general manager in retail dispensaries. Exposure to cannabis was significantly less after moving from grow facilities to retail stores. However, he continued to grow his own cannabis plants and smoked marijuana on a regular basis. He had minimal exposure to marijuana edibles.

His complaints were of wheezing, runny nose, itchy eyes, dry cough, and itchy hands with an eczematous rash that got progressively worse over time while in the workplace but improved at home. Physical examination revealed mild nasal mucosal edema (an airway inflammation) and mild diffuse rhonchi on lung auscultation (coarse rattling respiratory sounds often caused by secretions in bronchial airways). Albuterol HFA (inhalation aerosol) helped relieve wheezing and cetirizine (an over-the-counter antihistamine for allergies) was effective for controlling rhinitis (an irritation and inflammation of the mucous membrane inside the nose) and conjunctivitis (an inflammation or swelling of the tissue layer that lines the inner surface of the eye often referred to as “pink eye”) (Fadel et al. 2000). Pulmonary function testing demonstrated moderate restrictive ventilatory defect with superimposed early pulmonary impairment. The FEV1 (forced expiratory volume that measures how much air a person can exhale during a forced breath) was 52% predicted with a 38% improvement post-bronchodilator (a test to assess possible reversibility of bronchoconstriction in diseases such as asthma). His FeNO (fractional exhaled nitric oxide that gives a quantitative measure of airway inflammation to help identify, treat, and manage steroid-responsive patients) was significantly elevated at 89 ppb (normal < 35 ppb). Skin testing revealed multiple sensitizations to pollens and dog dander. Skin testing to marijuana using the “puddle technique” (Wave 3 News 2018) was positive.

Based on testing, patient #1 was diagnosed with asthma exacerbated by marijuana exposure with seasonal and perennial allergic rhinitis, allergic conjunctivitis, and suspected contact dermatitis to marijuana. Patch testing to marijuana to assess contact dermatitis was not performed.

Treatment recommendations included minimizing his environmental exposure to marijuana as much as possible. Medications included daily inhaled corticosteroids, rescue albuterol HFA pm (“when necessary”), daily nasal fluticasone (a synthetic glucocorticoid used to treat nasal symptoms), cetirizine pm, and OTC (over the counter) anti-inflammatory eye drops. On return visit, the patient had significantly improved on the prescribed medication program. He was now sleeping through the night and playing hockey again on a regular basis. He had reduced his marijuana use but had not yet implemented avoidance measures of the substance at home.

Follow-up pulmonary function tests showed improvement in his FEV1 that was now 80% predicted; however, he still had significant bronchial hyper-responsiveness. The FeNO was reduced to 30 ppb. This patient was subsequently lost to follow-up.

Patient #2 AS was a 26-year-old white male who was referred by an emergency room (ER) physician with a suspicion of anaphylaxis manifested as shortness of breath and chest pain after exposure to marijuana smoke.

He was evaluated in our office 3 weeks after experiencing shortness of breath and throat tightening after smoking marijuana. He went to the ER a second time after smoking marijuana again with the same reaction and was prescribed prednisone (a steroid that is used to treat allergic disorders and inflammation). He described in depth how he smoked the “concentrator” wax (hash—blueberry flavored) in addition to the marijuana flower (golden goat—sativa) prior to both episodes. For 2 weeks thereafter, he had progressive worsening of symptoms even after only passive exposure to marijuana smoke. Further investigation revealed that the “concentrate” was a CO₂ extracted marijuana wax that contained up to 60–70%

tetrahydrocannabinol (THC) levels which is in contrast to dispensary marijuana that has THC levels in the range of 20–25%. He admitted to heavy marijuana use, smoking multiple times per day for the past 10 years with a history of post-viral induced asthma but no allergic rhinitis or anaphylaxis.

Physical exam was normal, except for increased throat erythema consistent with his recent upper respiratory infection. Pulmonary function testing revealed mild obstructive impairment with no reversibility post-bronchodilator. His FeNO was 16 ppb. Lab results demonstrated an elevated IgE of 131 IU (international unit) per ml and a WBC (white blood cells that are involved in protecting the body against infectious disease and foreign invaders) of 16.7 on prednisone. Immunocap testing (used to identify allergen sensitivities) was positive to cat dander (class 3), dog dander (class 2), and dustmites (*Dermatophagoides farinae* (class 3) and *pteronyssinus* (class 4)). Testing to pollens was negative.

On a subsequent visit, a “puddle” skin test was performed that was negative to two marijuana strains (Dark Dreams and Jilly Bean) with a good histamine response. However, two of three office workers tested as controls and two other admitted marijuana users demonstrated positive responses. Despite several telephone calls, the patient did not return for the scheduled follow-up visit.

Patient #3 This 28-year-old white male had nasal congestion that was worsening since using “pot” in 2010. He had previously been evaluated by an allergist for allergic rhinitis and was found to be poly-sensitized to multiple indoor and outdoor airborne allergens. He began working as a trimmer at a marijuana growth facility and shortly thereafter developed “extreme” nasal congestion, which was initially intermittent but soon became persistent. He tried holistic therapeutic approaches such as vaporized essential oils, eucalyptus, peppermint, and lavender several times per day to manage the congestion without relief.

During his office visit, he reported that during the past 2 months, he had frequent wheezing with a chronic cough. The results of previous spirometry to assess lung function were normal. Although no diagnosis of asthma was given, his primary care physician gave him an albuterol inhaler for the chest tightness. He admitted frequent marijuana smoking. Of note, he had a history of an adverse food reaction from vinegar that caused throat swelling and two cats at home causing no apparent symptoms. He was otherwise healthy and required no other drugs. Physical examination revealed conjunctival eye injection “red eye” and moderate nasal congestion. His chest examination findings were normal. The patient declined skin testing for seasonal and perennial aeroallergens, pulmonary function testing, and chest and sinus imaging.

He was treated with fluticasone nasal spray, two puffs each nostril daily, cetirizine as needed, and albuterol hydrofluoroalkane for shortness of breath or wheezing and was provided an epinephrine self-injector for any future systemic allergic reactions. Follow-up by telephone revealed that the patient’s symptoms had improved and that the albuterol was helping with exercise tolerance. The patient declined any future follow-up.

Discussion

Much attention has recently been directed toward understanding marijuana use and its adverse respiratory effects (Engler et al. 2013; Owen et al. 2014; Salo et al. 2017). However, our understanding remains incomplete.

The first patient demonstrated classic IgE-mediated allergic reactivity documented by positive skin testing and marked allergic symptoms of histamine release (both respiratory and dermatologic). Given exposure to mold, dander, and dust mite reactivity on in vitro testing, these factors may have been responsible for the “allergic” reactions. In addition, other additives or contaminants such as pesticides may have been playing a non-IgE-mediated irritant role causing symptoms over a longer period. However, the second patient presented a totally different and confusing picture with no pollen reactivity. Since the marijuana flower “puddle” skin test was negative for this patient, whether the wax concentrate contained an excipient, additive, or contaminant to which the patient reacted needs to be considered. While regulations on growth facilities are being continuously refined, such as prohibiting mold (Kagen et al. 1983; Pohl 2012; Salo et al. 2017) and pesticides (Migoya and Baca 2015), new extraction procedures using agents like butane are constantly evolving that can potentially elicit reactions. Also, given the patient’s lack of sensitization to marijuana extracts and persistent clinical symptoms after exposure to passive marijuana smoke, psycho-physiologic reactions need to be considered and appropriately addressed.

In addition to these three patients, other vignette patient presentations in our practice included two Drug Enforcement (DEA) agents who also had severe asthma upon exposure to marijuana confiscations brought into their offices (Silvers 2016). One of these agents was on Xolair, an anti-IgE monoclonal antibody used to prevent severe asthma exposures to marijuana (Di Domenico et al. 2011). The other agent felt that her symptoms were definitely “strain-specific,” with “skunky smelling” strains causing more difficulties. She had to stop working in the field given these reactions.

Another patient doing home-growing experienced allergic rhinitis and asthma. The only definitive therapy would be avoidance of this exposure that he declined preferring to have the symptoms and to be treated with medications. Given increasing home-growing operations in residential areas, this exposure may become an even greater problem.

Conclusion

With increasing access to cannabis/marijuana due to the greater legalization for medical and recreational use, there is an increasing potential for allergic sensitization in the atopic population, by both active use and passive exposure (Silvers 2017; Tashkin 2013). In unusual presentations, mechanisms other than classic IgE-mediated allergic reactivity need to be considered.

Compliance with Ethical Standards The article is in compliance with the protection of all human and animal rights.

Conflict of Interest The author declares that there is no conflict of interest.

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