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Marijuana Use and the Development of Status Asthmaticus: A Case Report

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ABSTRACT

Introduction: Status asthmaticus is an acute exacerbation of asthma caused by increased airflow resistance and mucus plugging. Symptoms of dyspnea may lead to respiratory failure and cardiac arrest. There is limited knowledge about the effects of marijuana on pulmonary function. We report a unique case of status asthmaticus related to frequent marijuana use. **Case Presentation:** A 38-year-old African American male with a past medical history of asthma and two prior episodes of status asthmaticus arrived at Saint Barnabas Medical Center with dyspnea and wheezing that was refractory to home albuterol nebulizer therapy. Despite medical treatment his symptoms did not improve and he collapsed to the floor and required intubation and mechanical ventilation. He reported having smoked marijuana numerous times on the day prior to this admission. He was discharged three days later and was strongly advised to stop smoking marijuana. **Conclusion:** Marijuana is a commonly smoked illicit drug. Although habitual marijuana smokers have symptoms of cough, phlegm and wheezing, we found no reports linking marijuana use to the development of status asthmaticus. Given the rampant abuse of marijuana and its likely burden on healthcare, physicians should be vigilant in discussing the harmful effects of marijuana with asthmatic patients who abuse this drug.

Keywords: Marijuana; Status Asthmaticus; Drugs of Abuse

1. Introduction

Status asthmaticus is an acute aggravation of asthma causing marked dyspnea and extreme wheezing. A progressive increase in airflow resistance and mucus plugging leads to hypoxia, hypercapnia, and acidosis which does not respond to first line therapeutic efforts of bronchodilators and corticosteroids. Possible complications include ischemic central nervous system (CNS) injury, respiratory failure, and cardiac arrest. Patients typically present with this condition following viral respiratory illness, exposure to a potent allergens or irritants, or after exercising in a cold environment. Some patients are under-prescribed the necessary anti-inflammatory therapy or are noncompliant with medications.

No case has previously reported the development of status asthmaticus from marijuana abuse. Our search of the literature did not reveal any clear references to marijuana smoking and its relationship to the development of status asthmaticus. An electronic literature search performed on the Ovid (1950 to present) and Pub Med interfaces identified all papers indexed as containing material relevant to the terms “asthma”, “status asthmaticus”, “asthma exacerbation”, “respiratory complications” and “marijuana”, “cannabis”, “illicit drugs”, “drugs of abuse”, “substance use”. On detailed inspection, few studies contained incomplete data and no previous case reports were published. However, an extensive literature review found supporting evidence of pulmonary complications, such as airway inflammation and bronchospasm in marijuana smokers. We report a unique case of marijuana use and development of status asthmaticus.

2. Case Presentation

A 38-year-old African American male with a past medical history of asthma was seen in the emergency department (ED) at Saint Barnabas Medical Center in Livingston, N.J. because of continued dyspnea and wheezing refractory to home albuterol nebulizer treatment for four hours. The patient was unable to provide a complete history as his speech was limited to single words between breaths. He received three albuterol and ipratropium nebulizer treatments, subcutaneous epinephrine and terbutaline, intravenous methylprednisolone and magnesium sulfate, and was placed on bi-level positive airway pressure.

Past medical history was significant for three emergency department visits due to acute asthma attacks in the past 19 months. During his last admission, chest X-ray

had showed no infiltrates and electrocardiogram was noncontributory. Additionally, five years ago the patient had spontaneous right sided pneumothorax for which a chest tube was inserted. The patient was employed as a full-time promoter for a local radio station. He was physically active and was able to run 30 minutes on the treadmill and vigorously lift weights thrice a week. The patient denied use of any supplements or stimulants with his exercise regimen, and also denied ethanol or tobacco use. However, he stated that he smoked marijuana frequently and had used it several times on the day prior to his ED visit.

On admission, his temperature was 98.4˚F (36.8˚C), pulse 139 beats per minute, respirations 36 per minute, blood pressure 153/131 mm Hg and his oxygen saturation was 98 percent while breathing ambient air. Physical exam showed warm and diaphoretic skin. The chest was symmetric but the patient was tripoding with use of accessory muscles of respiration. Breath sounds were diminished bilaterally. Faint wheezing and a prolonged expiratory phase were heard.

Urine toxicology was positive for tetrahydrocannabinol. Cardiac enzyme creatine kinase-MB (CKMB) was slightly elevated (13 ng/ml) and troponin T was less than 0.010 ng/ml. Echocardiography report showed normal atrial, ventricular, and valuvular function with no pericardial effusion. Computed tomography scan showed pulmonary hyperaeration with emphysematous changes in the right upper lobe.

His symptoms did not improve and he subsequently collapsed to the floor. His oxygen saturation was 80 - 90 percent (%) when bag masked. The patient was intubated with etomidate and succinylcholine and transferred to the intensive care unit. Fentanyl drip was started and the patient was ventilated with pressure regulated volume control (PRVC) mode with a tidal volume of 600 ml, respiratory rate of 20/minute, positive end expiratory pressure (PEEP) of 5 cm of water, and a fraction of inspired oxygen (FIO2) of 40%. Initial arterial blood gas on FIO2 of 100% showed a pH of 6.82, partial pressure of carbon dioxide (PCo2) 136 mm of mercury, partial pressure of oxygen (PO2) 467 mm of mercury, and bicarbonate level of 22 meq/liter with an anion gap of 15.8.

The patient was placed on mechanical ventilation for 24 hours and was treated with intravenous methylprednisolone and ipratropium-albuterol metered dose inhalers. The following day sedation was discontinued and the patient was extubated and placed on nasal cannula. He was transferred from the intensive care unit and discharged three days later to be followed up as an outpatient. Discharge medications included fluticasone propionate and albuterol inhaler, tiotropium bromide handihaler inhalation, and oral methylprednisolone. The patient was strongly advised to stop smoking marijuana.

3. Discussion

Marijuana and cocaine are the most commonly smoked illicit drugs in the United States, with 14.6 million people over 12 years reporting current use [1]. In view of such a high prevalence of marijuana use and the association of greater medical service utilization by its users, it is imperative to review marijuana's short and long term consequences.

Marijuana is derived from the cannabis plant and is intended for use as a psychoactive drug. It is smoked in the form of cigarettes or a pipe to filter out particulate matter. Marijuana smoke contains similar levels of tar as tobacco smoke and up to 50% more carcinogens, [2,3] as well as a five-fold greater increment of carboxyhemoglobin. There are several unique features of marijuana smoking that may increase the risk of lung injury. Wu *et al.* [4] compared individuals smoking filtered cigarettes to those smoking marijuana and found that the later had two-thirds larger puff volume and a one-third greater breath holding time. These factors affect the level of tar and other particulate deposition in the lungs. Marijuana smokers often embrace the hazardous practice of "shotgunning", a practice of inhaling smoke and then exhaling it with positive pressure into another person's mouth. Other practices like engaging in the Muller or Valsalva maneuver while smoking marijuana may also result in pulmonary barotraumas [5,6].

A review by Wolff *et al.* [7] assessed the pulmonary effects of illicit drug use and found that marijuana smoking was associated with airway inflammation, acute bronchospasm, airflow obstruction, diffusion impairment, and emphysema. Studies by Sherman *et al.* [8] and Sarafian *et al.* [9] revealed that marijuana smoking may cause ongoing oxidative stress and DNA damage, processes that may be important in malignant transformation. Johnson and coworkers demonstrated large paraseptal bullous lesions in the lungs of four marijuana smokers who smoked little tobacco and concluded that marijuana smoking may result in emphysema that is different in distribution than seen in tobacco smokers, where the location is mainly centrilobular [10]. In immunocompromised patients, smoking marijuana increases the risk for Aspergillus infection.

The most recent review by Tetrault *et al.* reviewed 34 studies that assessed the impact of short term marijuana use on airway response and long-term marijuana smoking on pulmonary function and respiratory complications revealed an overall beneficial effect of marijuana smoking. Of the twelve studies assessing the short term effects of marijuana smoking on airway response, six studies showed an increase in specific airway conductance after marijuana challenge that ranged from 8% to 48%, and three studies showed an increase in forced expiratory

volume in one second (FEV1) after smoking marijuana compared with baseline ranging from 0.15 to 0.25 L [11]. In the same review, Tashkin *et al.* [12] showed a rapid improvement in airway conductance returning to baseline levels after eight subjects with methacholine-induced bronchospasm smoked a single 2-percent Delta-9 THC cigarette. One study showed no difference in FEV1 after marijuana challenge compared with baseline or placebo [13]. In contrast, the study done by Tashkin *et al*. which examined the impact of more prolonged exposure to marijuana on airway response revealed a decrease of both specific airway conductance and FEV1 compared with baseline [14]. The same review failed to report studies showing a consistent association between long-term marijuana smoking and FEV1/Forced vital capacity (FVC), diffusing capacity of the lung for carbon monoxide (DLCo2), or airway hyper-reactivity. These studies failed to show any consistent worsening of the mentioned parameters.

The short-term effects of marijuana smoking have been paradoxically shown to both improve and worsen bronchospasm. Despite the conflicting results from these small series, it is apparent that the lungs are at risk from the use of continuous or prolonged marijuana smoking. The ubiquitous use of tobacco in these patients may also have confounding and additive effects which are potentially harmful to the lungs.

4. Conclusions

To the best of our knowledge, this is the first reported case of status asthmaticus due to frequent marijuana use. Further investigations into the pulmonary effects of marijuana both acutely and chronically will create a protocol for treatment of its complications, as well as provide evidenced-based recommendations and precautions for the use of legalized marijuana. Given the rampant abuse of marijuana and the burden on healthcare it may produce through the development of emphysema, barotrauma, inflammation and infection, physicians should question their patients carefully about marijuana use. Lastly, efforts to prevent and reduce the use of marijuana, such as advising patients to quit and providing referrals for support and assistance may have substantial public health benefits [15].

Competing Interests: The authors declare they have no competing interests.

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