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CASE REPORT

Chronic Marijuana Use and Chronic Obstructive Pulmonary Disease: A Case Report

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Abstract: Marijuana smoking has become increasingly common in the United States and the world as more states and countries have legalized it for medical and recreational use. There are a number of carcinogens in marijuana smoke similar to those in tobacco smoke, and yet it has been difficult in the literature to find a causal relationship between marijuana smoking and chronic obstructive pulmonary disease (COPD). We present a patient whose main risk factor for emphysema is daily marijuana smoking for upwards of 35 years with no personal history of smoking tobacco, and no other obvious risk factors. Current studies have shown association with chronic marijuana smoking and various lung complaints, forced expiratory volume in 1 second (FEV1) to forced vital capacity (FVC) ratio, and inconsistently with chronic bronchitis. With this case, we add to the current body of literature that suggests a possible relationship between long-term, heavy marijuana use and COPD.

Keywords: carcinogens, chronic obstructive pulmonary disease, emphysema, marijuana, smoking

Introduction

The use of marijuana has increased in prevalence in the United States (US), with 24 states making it legal for recreational use and another 12 states making it legal for medical use as of 2024.[1] Marijuana is now the second-most smoked substance in the US, second to tobacco.[2] Worldwide, there are an estimated 192 million users of cannabis as of 2018, with highest use among the US, European countries, and less so across Asia where use remains lower.[3] Additionally, in the United States, between 2008 and 2022 the per capita rate of reported past-year use increased by 120% and the per capita days of use increased by 210%. Between 1992 and 2022 there was a 15-fold increase in the per capita rate of reported daily or near daily use of marijuana.[4] We are encountering a growing population of patients impacted by marijuana and its health effects.

Smoke from marijuana contains many pulmonary carcinogens including phenols, aldehydes, acrolein, benzpyrene, and benzanthracene. These chemicals are also found in tobacco cigarettes and are known to cause lung disease.[2] Histologically, chronic marijuana smokers have been shown to have increased number and size of submucosal blood vessels corresponding with submucosal edema, and hyperplasia of goblet cells, as well as an increased number of alveolar macrophages with altered structure and function indicating response to increased airway inflammation, which is also seen in tobacco smokers.[5]

Despite increasing numbers of marijuana users being exposed on a regular basis to these chemicals, data and opinions are mixed in regard to the link between marijuana and chronic lung disease.[6] We present a case of a heavy marijuana smoker with severe obstructive lung disease to add to the literature and encourage ongoing discussions and research into this important topic.

Case Report

A 64-year-old man who worked as a liquor store clerk presented to pulmonary clinic for evaluation of dyspnea on exertion (DOE). He reported progressive DOE for the prior 10 years. This progressed from dyspnea when carrying heavy

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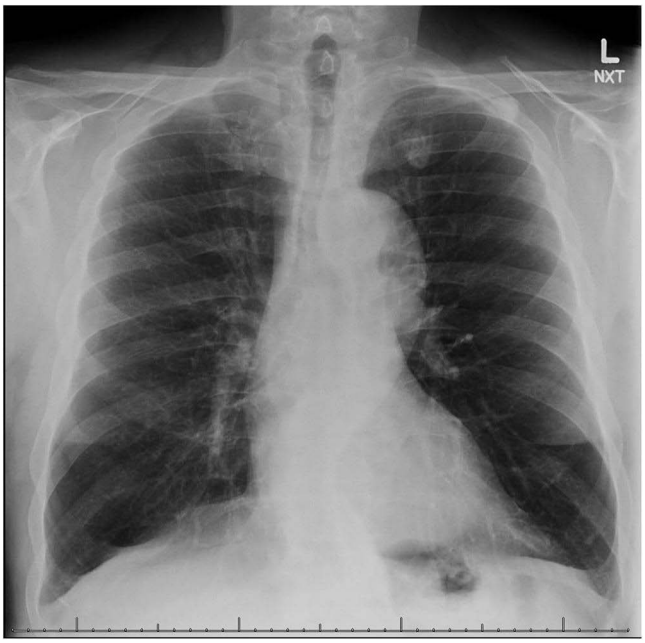

Figure 1 Chest X-ray demonstrating hyperinflation of the lungs.

objects to not being able to walk up a flight of stairs at the time of initial presentation to pulmonary clinic. He reported no significant cough but did hear wheezing at night. Physical exam revealed decreased air movement throughout both lung fields with no wheezes. Chest X-ray revealed hyperinflation of the lungs (Figure 1). Spirometry revealed a forced expiratory volume in 1 second (FEV1) to forced vital capacity (FVC) ratio of 38, an FEV1 of 1.18 L (33%), and an FVC of 3.12 L (65%). He had a normal DLCO on full pulmonary function testing (see Figure 2 for complete details). There was no significant response to inhaled bronchodilators. Computed tomography of the chest revealed mild-moderate emphysema with slight upper lobe predominance (Figure 3). Alpha-1-Antitrypsin level was within normal limits. Past medical history included psoriasis and psoriatic arthritis previously treated with prednisone and methotrexate, which were stopped due to fatigue. Etanercept was started for one month which was also stopped due to fatigue. He had started and continued ustekinumab with good control of his symptoms. Social history revealed a minimal history of secondhand tobacco smoke exposure as a child with his father smoking around him, although not regularly in the home. He moved out of the house as a teenager and the patient himself had no personal history of tobacco smoking. He worked various jobs throughout his life including as a painter, and most recently as a clerk at a liquor store. He never served in the military. He reported 35 years of near-daily marijuana use. A water pipe was used to smoke the marijuana 3–5 times daily during this time. He stopped smoking as his respiratory symptoms worsened.

Discussion

We present this case of a patient with very severe obstruction due to emphysema without significant personal tobacco history, secondhand tobacco exposure, or Alpha-1-Antitrypsin deficiency. There are no case reports of prednisone or Ustekinumab causing obstructive lung disease. There are case reports of both etanercept[7] and methotrexate[8] causing asthma; however, for both medications the obstruction reversed with stopping the medication, making these less likely causes of his chronic obstructive pulmonary disease (COPD). This leaves his chronic marijuana use as the most likely cause of his COPD.

A systemic review and meta-analysis of studies linking secondhand smoke exposure and COPD by Chen et al in 2023 determined an increased risk for COPD with even <5-year duration of secondhand smoke exposure, particularly among women. There was significant heterogeneity amongst these studies in amount of exposure, age of exposure, and length of exposure, making it difficult to know if our patient fits this group. After leaving his home as a teenager, he went on to

		Ref	LLN/ULN	Pre	% Ref	Post	% Ref	%Chg
Spirometry								
FVC	Liters	4.82	(3.9 - 5.8)	3.67	76	3.76	78	3
FEV1	Liters	3.62	(2.8 - 4.4)	1.46	40	1.45	40	-1
FEV1/FVC	%	75	(65.4 - 84.7)	40		38		
FEV1/SVC	%			29				
FEV6	Liters			3.22		3.10		-4
FEF25-75%	L/sec	2.91	(1.3 - 4.5)	0.55	19	0.45	16	-18
FEF50%	L/sec			0.67		0.45		-32
FEF75%	L/sec			0.26		0.26		-2
PEF	L/sec	9.19	(6.8 - 11.6)	3.94	43	4.60	50	17
FET100%	Sec			9.31		10.77		16
FIVC	Liters	4.80	(3.7 - 5.9)	3.18	66	2.14	45	-33
FEF/FIF50				0.24		0.43		82
Lung Volumes								
TLC	Liters	7.18	(5.6 - 8.8)	9.14	127			
VC	Liters	4.80	(3.7 - 5.9)	4.98	104			
IC	Liters	3.17	(2.4 - 3.9)	3.71	117			
FRC N2	Liters	3.77	(2.3 - 5.2)	5.43	144			
ERV	Liters	1.58	(1.2 - 2.0)	1.26	80			
RV	Liters	2.35	(1.6 - 3.1)	4.16	177			
RV/TLC	%	34	(24.7 - 42.3)	46				
LCI				11.27				
Wash Time	Min			3.7				
Diffusing Capacity (Hb 13.3)								
DLCO	mL/min/mmHg	28.2	(20.2 - 36.2)	23.1	82			
DL Adj	mL/min/mmHg	28.2	(20.2 - 36.2)	24.0	85			
DLCO/VA	mL/min/mHg/L	4.09	(2.9 - 5.3)	3.53	86			
VA	Liters	6.28	(5.2 - 7.3)	6.53	104			
IVC	Liters			4.31				

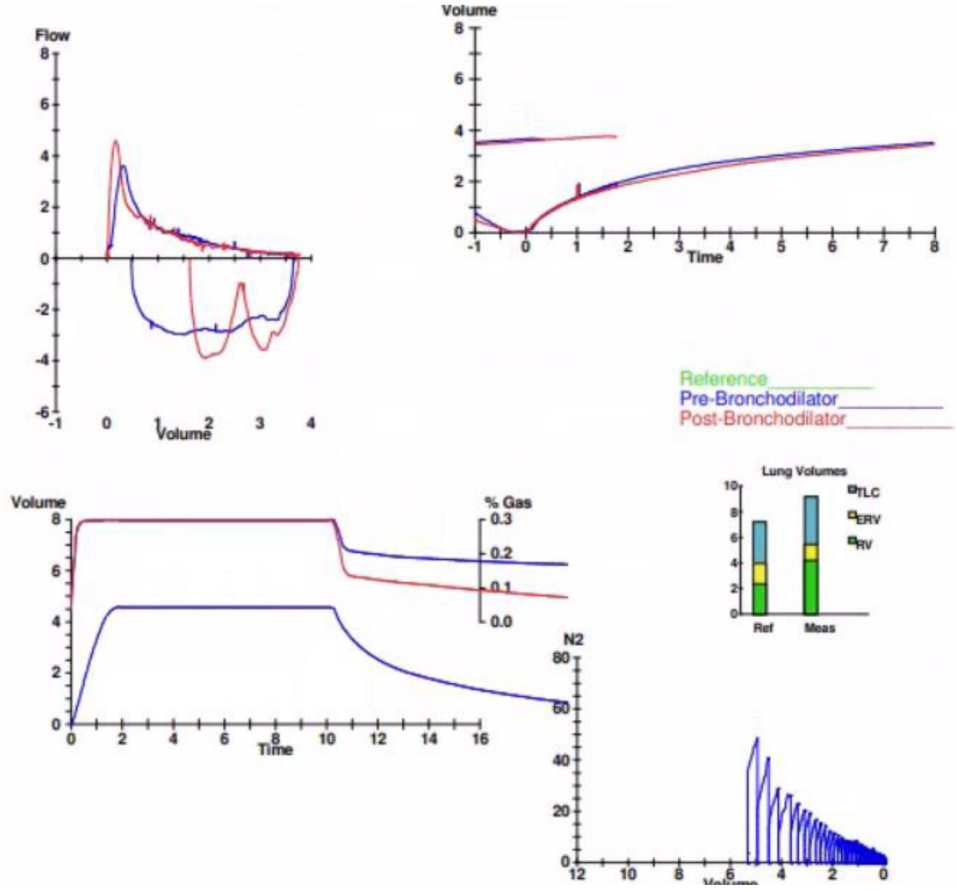

Figure 2 Full pulmonary function testing at time of presentation to pulmonary clinic.

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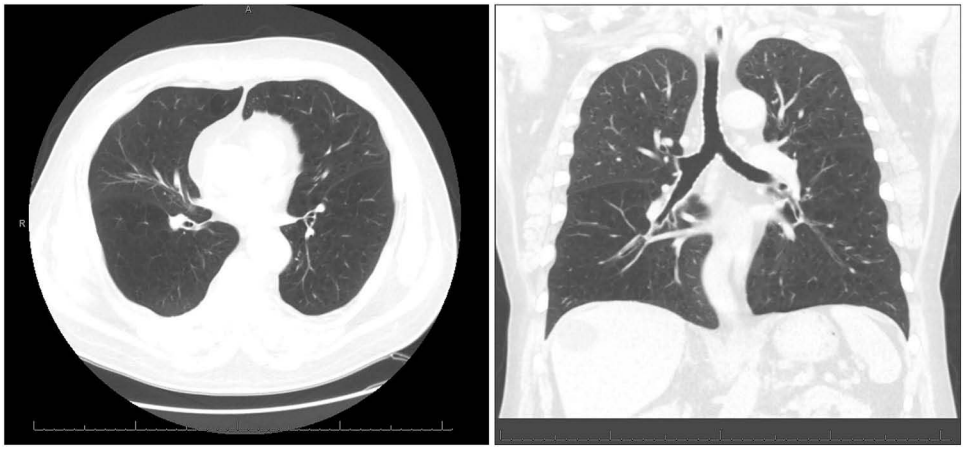

Figure 3 Coronal and axial chest computed tomography imaging demonstrating bullous emphysema.

become a competitive multi-sport athlete, including in running. He had a long and successful athletic career and did not develop respiratory issues until later in life following years of marijuana use and zero secondhand cigarette exposure as an adult, making us believe that secondhand smoke exposure as a child may have put him at greater risk of developing COPD but was not the main cause of his disease.[9]

Due to the many confounding variables in this population, including concomitant tobacco use, differences in methods of use, and differences in frequency and length of use, studies are inconsistent in linking marijuana use and lung disease. Although some studies seem to show symptoms of chronic bronchitis in heavy users, this is not consistent across studies.[10] Polen et al looked at a cohort of 452 marijuana smokers who never smoked tobacco and compared them to 450 demographically similar never smokers of either substance. They noted an increase in outpatient encounters for respiratory complaints among the marijuana users, although did not define these further.[11] Like many other cohorts, these patients tended to be younger (<35 years of age) resulting in far less years of use than our patient.

In their Subpopulations and Intermediate Outcome Measures in COPD Study (SPIROMICS), Morris et al found an increase in FEV1 and FVC among current and former marijuana users; however, they note that their study could not comment on lung function changes among long-term, heavy marijuana only smokers.[12] Bullous lung disease has been reported in several case reports and thought to be related to the large volume breath holds smokers employ.[13] Proving a causal relationship is difficult; however, with our patient's use of a water pipe we postulate his years of marijuana use and breath holds may have contributed to his emphysema.

Conclusion

The current literature is incomplete and inconclusive on the relationship between marijuana and lung disease, though there is more and more evidence to suggest an association. Additionally, there has been an increase in potency of marijuana over the years, which may be contributing to an increase in demonstration of lung disease in heavy use individuals, and even in those with lighter use. A US study reviewed changes in potency of marijuana from 1995 to 2014 and found that overall potency has increased in cannabis plant material from 4% in 1995 to around 12% in 2014.[14] Additional studies should determine if there is a method of use, amount, or number of years that puts our patients at risk for COPD due to marijuana smoking so that we can better counsel them on their use of this common substance.

Abbreviations

COPD, Chronic obstructive pulmonary disease; DOE, Dyspnea on exertion; FEV1, Forced expiratory volume in 1 second; FVC, Forced vital capacity.

Consent for Publication

The patient described in this manuscript has reviewed and consented to publication of its contents. Institutional approval was not required to publish this case.

Acknowledgments

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Disclosure

The authors report no conflicts of interest in this work.

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