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Case report

COPD secondary or associated with cannabis dependence

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1. Introduction

The typical clinical manifestations of Chronic Obstructive Pulmonary Disease (COPD) are explained by the presence of an obstructive process involving the air flow, accompanied by an exaggerated inflammatory response against external agents [1]. The causal factors for COPD are not yet fully understood, but they are related with significant exposure to harmful particles and gasses [2]. The disease is associated to cigarette smoking, in particular, for long stretches of time [3]; but only 20% of smokers develop COPD [4]. There has also been an increase of cases of COPD observed in consumers of other inhaled products or substances [5] and even in non-smokers [6].

Globally, cannabis or marihuana is the most commonly used illegal substance, the prevalence of having used the drug on occasion during life may reach 10% in some populations [7]. Cannabis consumption seems to present some short term beneficial pulmonary effects such as immediate bronchodilation, secondary to reduced airway resistance, which can be compromised in the long term as the cannabis smoke inhaled can have the same harmful effects as those attributed to tobacco [8,9]. The purpose of this article is to preset a case of secondary COPD associated to regular cannabis consumption or dependency.

1.1. The case

A 62-year-old male patient, living in an upper income area of the city, with over six months of clinical symptoms of grade 2 dyspnea (mMRC). Physical examination revealed blood pressure of 120/70 mm HG, a heart rate of 61 beats/minute, breathing rate of 20 breaths/minute, weight of 81 kg, height 184 cm and BMI of 23.92, with no other clinically relevant findings. Significant personal antecedents include an over 40-year history of cannabis consumption with occasional cocaine base use. The patient denied cigarette consumption and biomass exposition. The patient's clinical history also documented esophagus cancer of the adenocarcinoma type and benign prostatic hypertrophy. Both conditions were treated through surgery. Evaluation using the COPD Assessment Test, (CAT): 7 (cough: 2, phlegm: 2, chest tightness: 0, dyspnea: 3, domestic activity: 0, sleep: 0, energy: 0, fear: 0).

A chest x-ray revealed hyperlucency in the upper lobes and ballooning in the left posterior arch of the tenth rib (Fig. 1). There was also evidence of thickening of bronchial walls (Fig. 2). Pre- and post-bronchodilator spirometry informed forced expiratory volume in the first second (VEF1)/forced vital capacity (CVF): 65%, VEF1: 89% of the predicted, CVF: 106% of the predicted, FEF 25–75%: 63 (Fig. 3), carbon monoxide diffusion capacity: reduced, moderate. High resolution chest CT shows radiolucides with focal and central air attenuation located in the upper lobes without an identifiable wall (centrilobullar emphysema). No bullous emphysema are observed (Fig. 4). Category A COPD diagnosis was confirmed (GOLD 2018) and pharmacological management with one 150 μg indacaterol capsule inhaled once daily was initiated with an important remission of symptoms, without clinically significant exacerbations through one year of follow-up.

2. Discussion

In the present case involving an adult male, COPD diagnosis was associated with persistent cannabis use or dependency, without any biomass exposition. Nevertheless, there is controversy or incongruent results with respect to the association of long-term cannabis use and the clinical presentation of COPD, possibly due to biases in the samples, in particular, the relatively young age of consumers, and therefore, not particularly long-term exposure to cannabis smoke [10]. The association between cannabis use and lung function is consistent. But, decrements in FEV1 have never been reported and a reduction in the ratio of FEV1/FVC has been found in some research. However, cannabis smoking can increase FVC [11,12].

Cigarette smoking is often associated with COPD; however, in between 25 and 33% of cases it is impossible to document consumption [6,13]. Exposure to biomass also increases the risk of COPD, and is associated with COPD mainly in women living in rural areas, which is contrary to what was observed in the present case of a man living in a high-income urban area [13].

Cannabis smoke contains chemical toxins in similar or even higher proportions than tobacco smoke [14]. It is clear that Δ 9-tetrahydrocannabinol is only found in cannabis and that nicotine is only

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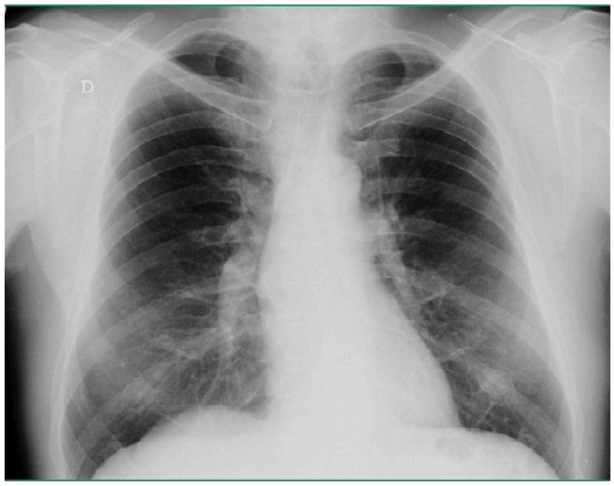

Fig. 1. Posteroanterior radiography.

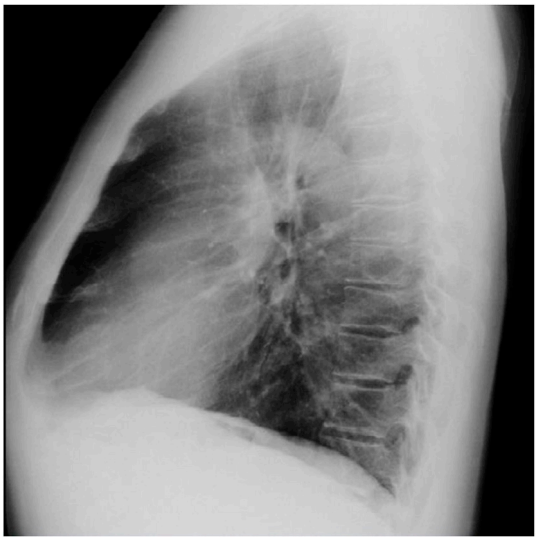

Fig. 2. Lateral radiography.

found in tobacco [15]. Both substances are linked as potential carcinogens affecting the respiratory tract and are a risk factor for COPD [14].

Habitual cannabis consumers often argue that cannabis is less harmful than regular tobacco, and some even consider that it is completely innocuous [8]. Hancox et al. documented -in a cohort of 1037 people-that accumulated cannabis consumption was significantly related to improved forced vital capacity, total lung capacity, functional residual capacity, and residual volume, as well as to reduced airway resistance [16]. In this same line, in a more recent meta-analysis, Martinasek et al. concluded that regular cannabis consumption is related to the clinical presentation of COPD, and to other clinical events such as spontaneous pneumothorax and lung cancer [17]. Cannabis consumers also tend to have carboxyhemoglobin levels that are almost five times higher than tobacco smokers [14]. In contrast to these results, Tan et al. observed –in a sample of 878 adults aged over 40- that cannabis consumption was not associated to the presentation of respiratory symptoms typical of COPD [18].

A study conducted with primates exposed to different doses of cannabis smoke, placebo, or cigarette smoke, showed evidence of hyperplasia of epithelial cells in all groups, but with greater frequency and severity of bronchiolitis, atypical alveolar cell hyperplasia, and fibrosis in those exposed to cannabis smoke. Endocannabinoid was considered a possible cause of such effects [10].

Pletcher et al. assessed the association between exposure to marihuana and pulmonary function with a twenty-year follow-up and observed that consumers with low levels of exposure to cannabis showed improved FEV1 and FVC. However, this effect was the most easily lost with a worsening of FEV1, in contrast to cigarette smokers, in whom the deterioration of pulmonary function by FEV1 and FVC is linearly related with the exposure [19]. It is possible to observe a low FEV1/FVC in some cases because of increased FVC [11,12]. However, in COPD, the FEV1/FVC ratio is decreased and FVC is normal. In other cases, FVC may be decreased in response to air trapping in severe disease and in mixed patterns (restrictive and obstructive at the same time). However, FVC may be high, or higher than 80% of the predicted level as in the present case, which rules out a concomitant restrictive defect. Likewise, the diffusion capacity in this patient is diminished as reported by Macklern et al. [20].

Thus, it is likely that chronic cannabis consumption induces an inflammatory process in the respiratory mucous by means of different pathways or mechanisms [19,21]. And it makes it easier for infections to invade due to the clearly identified effects of cannabinoids in several parts of the immune system; the respiratory system presents a high density of these receptors [22]. It is also considered that the risk of lung injury due to cannabis use is related to the use pattern that includes long and deep smoke inhalation [23].

Cannabis consumption is not as healthy as claimed by regular users or those that meet the criteria to be considered dependent [8]. Cannabis dependence undoubtedly represents a public health problem [24]. On the one hand, because of its negative effects on pulmonary function, and on the other, because it explains a high percentage of years of life spent with disability following the exacerbation of psychotic episodes, fewer academic or work-related achievements, and an increased number of traffic accidents [8,25].

This case invites us to investigate cannabis use as part of the medical history of people suffering from COPD. The limitations are, however, evident given that COPD is a clinical condition that is related to a number of possible precipitating, constitutional, and environmental factors [1,5,6]. For example, the simultaneous consumption of cannabis and cigarettes, whereby studies reveal that the negative effects of cigarette smoking are increased or potentiated when this occurs simultaneously with cannabis consumption [17,25–28]. We also need to think about how to handle cannabis dependence concomitantly with treatment for COPD in order to obtain satisfactory results [29].

We conclude that chronic cannabis consumption may be associated to the presentation of COPD in people that are particularly vulnerable. More research is needed to reveal and better explain the relationship between cannabis use and COPD.

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PARAMETROS	(BTPS)	PRED	PRE-RX BEST	%PRED	POST-RX BEST	%PRED	%CHG
FVC	Liters	5.05	4.93	98	5.33	106	8
FEV1	Liters	3.90	3.37	87	3.47	89	3
FEV1/FVC	%	77	68		65		
FEF25-75%	L/sec	3.51	2.37	68	2.21	63	-7
IsoFEF25-75	L/sec	3.51	2.37	68	2.69	77	13
FEF75-85%	L/sec		0.73		0.61		-16
PEF	L/sec		6.90		6.71		-3
FET100%	Sec		5.74		6.45		12
FIVC	Liters	5.05	4.73	94	4.88	97	3
FEV1	Liters	3.90	3.37	87	3.47	89	3
FIV1	Liters		4.48		4.65		4
FEF/FIF50			0.43		0.38		-12
Vol Extrap	Liters		0.05		0.07		42
FVL ECode			000001		100010		
MVV	L/min	114					
f	BPM						

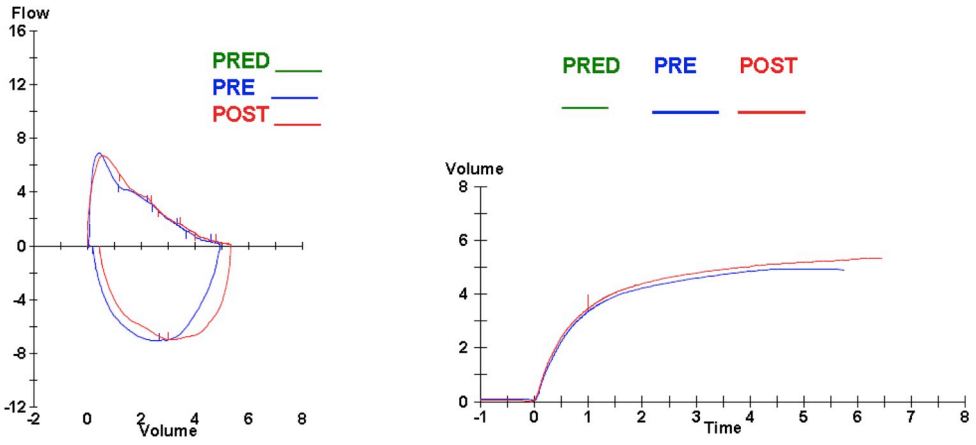

Fig. 3. Spirometry report.

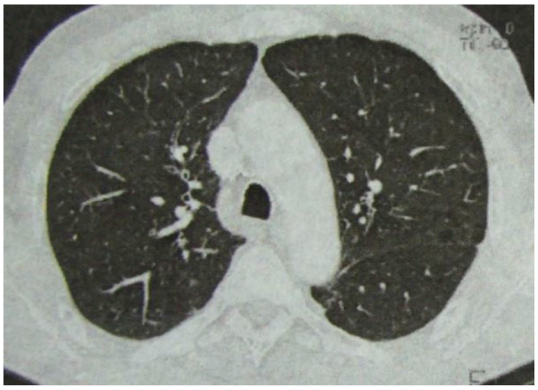

Fig. 4. Computerized tomography.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.rmcr.2019.100902.

References

[1] A. Bourdin, P.R. Burgel, P. Chanez, G. Garcia, T. Perez, N. Roche, Recent advances in COPD: pathophysiology, respiratory physiology and clinical aspects, including comorbidities, Eur. Respir. Rev. 18 (114) (2009) 198–212.

[2] C. Torres, M. García, M. González, [Is chronic obstructive pulmonary disease caused by wood smoke a different phenotype or a different entity?], Arch. Bronconeumol. 52 (8) (2016) 425–431.

[3] B.D. Carter, C.C. Abnet, D. Feskanich, et al., Smoking and mortality—beyond established causes, N. Engl. J. Med. 372 (7) (2015) 631–640.

[4] N. Terzikhan, K. VAerhamme, A. Horman, et al., Prevalence and incidence of COPD in smokers and non-smokers: the Rotterdam Study, Eur. J. Epidemiol. 31 (8) (2016) 785–792.

[5] T. Schikowski, I.C. Mills, H.R. Anderson, et al., Ambient air pollution: a cause of COPD? Eur. Respir. J. 43 (1) (2014) 250–263.

[6] T. Mahmood, R.K. Singh, S. Kant, et al., Prevalence and etiological profile of chronic obstructive pulmonary disease in nonsmokers, Lung. India 34 (2) (2017) 122–126.

[7] W. Hall, L. Degenhardt, Prevalence and correlates of cannabis use in developed and developing countries, Curr. Opin. Psychiatr. 20 (4) (2007) 393–397.

[8] P. Gates, A. Jaffe, J. Copeland, Cannabis smoking and respiratory health: consideration of the literature, Respirology 19 (5) (2014) 655–662.

[9] L.I. Ribeiro, P.W. Ind, Effect of cannabis smoking on lung function and respiratory symptoms: a structured literature review, N. P. J. Prim. Care. Respir. Med. 26 (2016) 16071.

[10] J.M. Chatkin, G. Zabert, I. Zabert I, et al., [Lung disease associated with marijuana use], Arch. Bronconeumol. 53 (9) (2017) 510–515.

[11] M. Ghasemiesfe, D. Ravi, M. Vali M, et al., Marijuana use, respiratory symptoms, and pulmonary function: a systematic review and meta-analysis, Ann. Intern. Med. 169 (2) (2018) 106–115.

[12] D.P. Tashkin, Marijuana and lung disease, Chest 154 (3) (2018) 653–663.

[13] A. Sana, S.M.A. Somda, N. Meda N, et al., Chronic obstructive pulmonary disease associated with biomass fuel use in women: a systematic review and meta-analysis, BMJ Open. Respir. Res. 5 (2018) e000246.

[14] V. Torres, Respiratory commitment in marijuana smokers, Rev. Urug. Med. Intern. 1 (1) (2016) 44–51.

[15] D. Tashkin, Does smoking marijuana increase the risk of chronic obstructive pulmonary disease? CMAJ (Can. Med. Assoc. J.) 180 (8) (2009) 797–798.

[16] R.J. Hancox, R. Poulton, M. Ely, et al., Effects of cannabis on lung function: a population-based cohort study, Eur. Respir. J. 35 (8) (2010) 42–47.

[17] M.P. Martinasek, J.B. McGrogan, A. Maysonet, A systematic review of the respiratory effects of inhalational marijuana, Respir. Care 61 (11) (2016) 1543–1551.

[18] W.C. Tan, C. Lo, A. Jong, et al., Marijuana and chronic obstructive lung disease: a population-based study, Can. Med. Assoc. J. 180 (8) (2009) 814–820.

[19] M.J. Pletcher, E. Vittinghoff, R. Kalhan, et al., Association between marijuana exposure and pulmonary function over 20 years, J. Am. Med. Assoc. 307 (2) (2012) 173–181.

[20] P.T. Macklern, M.R. Becklake, The relationship between the mechanical and diffusing properties of the lung in health and disease, Am. Rev. Respir. Dis. 87 (1963) 47–56.

[21] Z. Helyes, Á. Kemény, K. Csekő, et al., Marijuana smoke induces severe pulmonary hyperresponsiveness, inflammation, and emphysema in a predictive mouse model not via CB1 receptor activation, Am. J. Physiol. Lung Cell Mol. Physiol. 313 (2) (2017) L267–L277.

[22] T.W. Klein, C. Newton, K. Larsen, et al., The cannabinoid system and immune modulation, J. Leukoc. Biol. 74 (4) (2003) 486–496.

[23] A.J. Gordon, J.W. Conley, J.M. Gordon, Medical consequences of marijuana use: a review of current literature, Curr. Psychiatr. Rep. 15 (12) (2013) 419.

[24] L. Karila, P. Roux, B. Rolland, et al., Acute and long-term effects of cannabis use: a review, Curr. Pharmaceut. Des. 20 (25) (2014) 4112–4118.
[25] S. Imtiaz, K.D. Shield, M. Roerecke, et al., The burden of disease attributable to cannabis use in Canada in 2012, Addiction 111 (4) (2016) 653–662.
[26] M. Joshi, A. Joshi, T. Bartter, Marijuana and lung diseases, Curr. Opin. Pulm. Med. 20 (2) (2014) 173–179.
[27] J. Macleod, R. Robertson, L. Copeland, et al., Cannabis, tobacco smoking, and lung function: a cross-sectional observational study in a general practice population, Br. J. Gen. Pract. 65 (631) (2015) e89–e95.
[28] M.H. Lee, R.J. Hancox, Effects of smoking cannabis on lung function, Expert Rev. Respir. Med. 5 (4) (2011) 537–546.
[29] K. Hambleton, M. Bafadhel, R. Russell, Chronic obstructive pulmonary disease: management of chronic disease, Medicine 44 (5) (2016) 310–313.