

component. In addition, during the bronchoscopy extraction there was a tendency to hemorrhage. Said hemorrhagic tendency is due to the bone marrow content of the tumor. We speculate that a relatively small endobronchial osteochondromatous hamartoma could be extracted without any risk with biopsy forceps during flexible bronchoscopy.

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Primary Bullous Disease of the Lung in a Young Male Marijuana Smoker[☆]

Enfermedad primaria bullosa del pulmón en varón joven fumador de cannabis

Dear Editor,

It is estimated that 160 million people smoke cannabis worldwide. The concerns about its pulmonary effects arise from the observation that cannabis is qualitatively similar to tobacco, with the exception of their respective components: tetrahydrocannabinol (THC) and nicotine. Over the long term, smoking cannabis leads to airflow obstruction in the large airways and chronic bronchitis, as well as important deficiencies in pulmonary function.^{1–3}

We present the case of a 27-year-old male with a personal history of tobacco habit of 15 cigarettes/day for the last 12 years, as well as a habitual smoker (3 cigarettes/day) of marijuana, who was admitted to our hospital due to chest trauma related to a traffic accident. Emergency thoracic CT without intravenous contrast revealed bilateral apical and paramediastinal bullae, predominantly on the right, with an especially large bulla (13 cm × 11 cm) in the apical segment of the right upper lobe (RUL) and some areas of parenchymal destruction in the subpleural region of the RUL (Fig. 1).

Analytical studies showed normal arterial blood gases, except for a significant increase in carboxyhemoglobin. Immunoglobulins and alpha-1-antitrypsin were normal.

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Lung function studies showed moderate generalized bronchial obstruction with no response to bronchodilators, moderate affectation of CO diffusion, redistribution of volumes, and air trapping. The toxic effects of tobacco consumption tend to be associated with airway obstruction and centrilobular emphysema.^{1,3} In young smokers of cannabis, there is a documented more common form of bullous disease with a paraseptal distribution that is predominant in the upper lobes. The physiological effects of marijuana in terms of anomalies in lung function are mostly in the large airways, while the effect is less in the smaller-sized airways.^{1–3}

With the exception of the psychoactive composition (THC and other cannabinoids present in marijuana, and nicotine in tobacco), the smoke from the 2 substances is practically identical. However, there are some fundamental differences when comparing the physical dynamics of smoking marijuana with those of smoking tobacco: the depth of the inspiration is 3 times greater when smoking marijuana, which would increase the frequency of barotraumas, especially pneumomediastinum. The physiopathological mechanisms of bullae in susceptible individuals is probably a combination of direct lung toxicity together with changes in pleural pressure and the respiratory tract, associated with the high inspiratory pressures produced when smoking marijuana.^{1,3} On the other hand, the lack of filters in marijuana cigarettes means that 4 times more tar enters the lungs, and the increase in carboxyhemoglobin is up to 5 times more per cigarette.¹

Although it is difficult to completely separate the concomitant role of tobacco, the relatively young age of the patients and low accumulated exposure of tobacco consumption suggests at the very least an additive role of marijuana in the pathogenesis of pulmonary damage.^{1,3}



Fig. 1. Bilateral apical and paramediastinal bullae, predominantly on the right. A large bulla (133 mm × 115 mm) is seen in the right apical segment. Some areas of parenchymal destruction are also identified in the subpleural region of the right upper lobe, with small bronchiectasis in the posterior apical segment of the left upper lobe, and bibasilar subsegmental atelectasis.

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