

A Case of Endobronchial Osteochondromatous Hamartoma Removed Using Flexible Bronchoscopy[☆]

Presentación de un caso de hamartoma osteocondromatoso endobronquial reseado con broncoscopia flexible

Dear Editor,

The common histologic types of endobronchial hamartoma are chondromatous and lipoid hamartoma.¹ Osteochondromatous hamartoma with a bone marrow component has been very infrequently reported in English publications.² We describe an extremely rare case of endobronchial osteochondromatous hamartoma that was centrally located and extracted satisfactorily by flexible bronchoscopy.

A 67-year-old woman came to our hospital complaining of intermittent cough that had been evolving over the previous 2 months. The patient was a non-smoker. No anomalies were found on laboratory testing, including hemogram as well as microbiological sputum tests. Simple chest radiograph revealed no anomalies. For a more detailed evaluation of the cough, thoracic computed tomography was carried out. This demonstrated a 1 cm endobronchial lesion with no specific parenchymatous anomalies (Fig. 1a). The patient underwent bronchoscopy in order to assess the endobronchial lesion. Bronchoscopy demonstrated a polypoid mucosal lesion located in the left lower lobe bronchus (Fig. 1b). During bronchoscopy, its consistency was observed to be as hard as a bone fragment,

and we were then able to completely extract the polypoid lesion with the biopsy forceps (Fig. 1c). Although the lesion had a tendency to hemorrhage, the hemorrhage was controlled with cold saline solution and an adrenalin solution at a ratio of 1:20 000. The anatomic pathology findings demonstrated cartilaginous cells accompanied by bone tissue with myeloid elements, compatible with endobronchial osteochondromatous hamartoma (Fig. 1d and e). After the exeresis of the endobronchial lesion, the cough was resolved. On follow-up, the patient showed no evidence of relapse (Fig. 1f).

The lesion of this patient is an exceptional case of endobronchial osteochondromatous hamartoma. Few cases have been published of these lesions with a bone marrow component that have been surgically resected.² Their treatment should be individualized according to the clinical manifestations of each patient and each hamartoma.^{3,4} Oishi et al.² surgically resected a relatively large osteochondromatous hamartoma that could not be extracted by bronchoscopy. In contrast, our group was able to extract the lesion during flexible bronchoscopy. This case was characterized by a polypoid lesion that was relatively small in diameter. This could be the reason why the lesion was able to be completely extracted by bronchoscopy without significant complications. Endoscopic extraction presents advantages for symptom control and can avoid thoracotomy due to endobronchial lesion.^{5,6}

In the present case, the lesion had a consistency that was as hard as a bone fragment. On biopsy, the hard consistency was different from the spongiform consistency that has generally been described.¹ This seems to be associated with the bone marrow

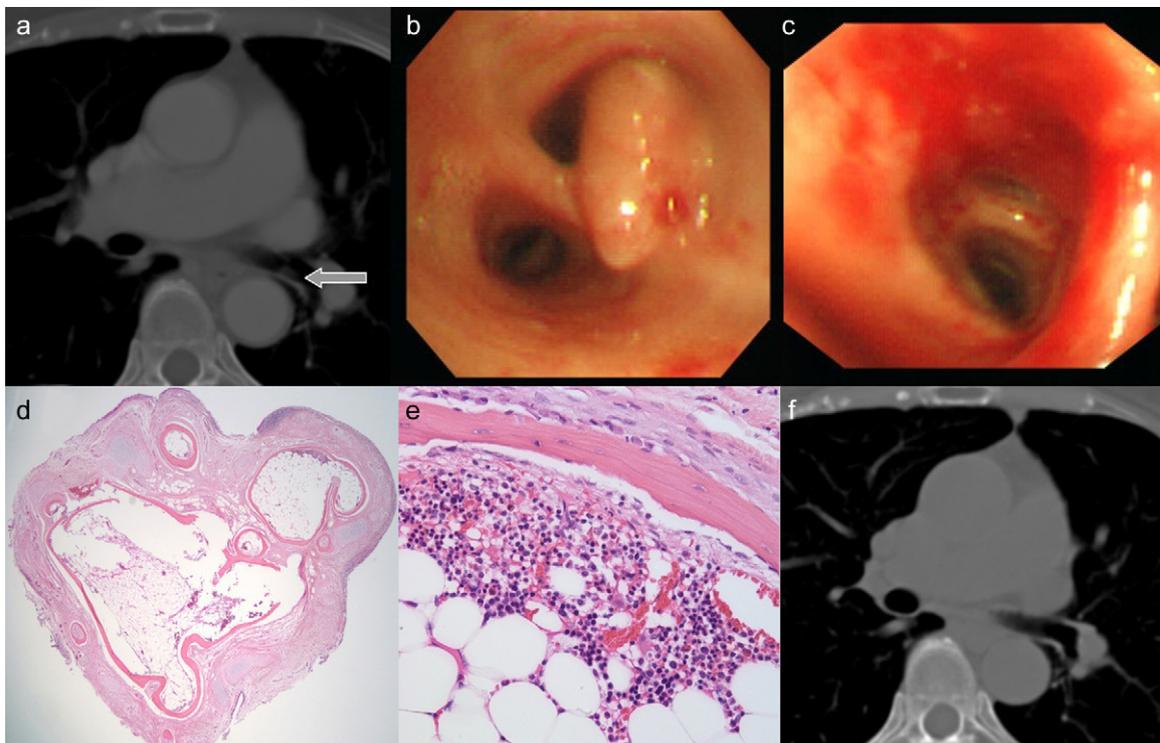


Fig. 1. (a) Computed tomography revealed an endobronchial lesion measuring 1 cm in diameter (arrow). (b) Polypoid mucous lesion located in the left lower lobar bronchus as seen on flexible bronchoscopy. (c) After the total extraction of the polypoid mucous lesion with biopsy forceps. (d) The endobronchial polypoid mass was mainly made up of bone, cartilage and fatty tissue and covered with respiratory epithelium (hematoxylin–eosin stain, $\times 40$). (e) The bone contained myeloid elements, which would suggest true bone marrow (hematoxylin–eosin stain, $\times 200$). (f) Computed tomography showing the elimination of the polypoid lesion 12 months after the bronchoscopic procedure.

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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}