Arch Bronconeumol. 2009;45(7):356-360

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Letters to the Editor

A New Case of Idiopathic Bronchiolocentric Interstitial Pneumonia

Un nuevo caso de neumonía intersticial bronquiolocéntrica idiopática

To the Editor:

We have read with great interest the case report by Muñoz et al[1] on idiopathic bronchiolocentric interstitial pneumonia and we would like to report another case of this uncommon entity, first described by Yousem and Dacic[2] and not yet included in the American Thoracic Society/European Respiratory Society[3] consensus classification. We have found no other cases in MEDLINE since the publication of that case report.[1]

The patient was a 51-year-old housewife, with no relevant family history or known drug allergies. She used to smoke 20 cigarettes a day but had quit 2 years earlier and presented hypothyroidism treated with thyroxine and hypercholesterolemia controlled by diet. For 1 year, she had had 2 exotic birds in her home. The patient reported that her symptoms had begun as catarrh with mucopurulent expectoration and rhinorrhea, and that she then developed a dry cough and gradually worsening exertional dyspnea, which had progressed to dyspnea on minimal effort. Physical examination revealed bibasilar crackles. The chest radiograph showed an interstitial infiltrate, mainly in the lower lobes. The patient had an arterial oxygen saturation (SaO_2) of 83% and was therefore admitted to hospital. Blood tests included complete blood count, urea, glucose, ions, creatinine, liver function, anticytoplasmic and antinuclear neutrophil antibodies, angiotensin converting enzyme, antithyroid antibodies, and immunoglobins G, A, M, and E, and all values were within the normal range. The following parameters were recorded in the arterial blood gas analysis (breathing ambient air): pH of 7.45, PaO_2 of 45 mm Hg, $PaCO_2$ of 34 mm Hg, bicarbonate of 23.6 mmol/, and base excess of 1 mmol/L. High-resolution computed tomography (CT) of the chest revealed patchy ground-glass opacities, a paraseptal bulla, and enlarged left perivascular and right paratracheal lymph nodes. The results of the lung function tests were as follows: forced vital capacity (FVC), 1.68 L (68.4%); forced expiratory volume in 1 second (FEV_1),1.54 L (79.6%); FEV_1/FVC, 91.58%; forced midexpiratory flow rate ($FEF_{25\%-75\%}$), 3.37 L/s (135.1%); carbon monoxide diffusing capacity (DLCO), 3.69 mmol/min/kPa (48.1%), ratio of DLCO to alveolar volume, 1.42 mmol/min/kPa/L (61.2%); total lung capacity, 2.79 (80%), and residual volume, 1.00 L (74.4%). The bronchodilator test was negative. Except for bilateral bronchorrhea, no macroscopic pathologic endobronchial lesions were observed with fiberoptic bronchoscopy. Due to severe desaturation during the bronchoscopic examination, bronchoalveolar lavage could not be performed. Bronchial aspiration (with no significant microbiologic or cytologic findings) and lung biopsy were performed. The microscopic description of the lung biopsy was as follows: fibrocellular proliferation in the lobules and septa with a predominantly cellular infiltrate (lymphocytes, histocytes, and plasma cells around the airways, arterioles, and septa). The parenchyma showed a patchy pattern with areas of septal thickening. The pathologic diagnosis was bronchiolocentric cellular interstitial pneumonia. Treatment was started with prednisone (60 mg/d) and after 6 months of follow-up the patient showed clinical improvement (decrease in exertional dyspnea) and an SaO_2 of 97%. The follow-up CT showed a marked improvement of ground-glass opacities. Lung function had improved slightly, but not significantly: FVC was 1.79 L (72.8%); FEV_1, 1.66 (86.1%); FEV_1/FVC, 93%; and $FEF_{25\%-75\%}$, 3.75 L (150.4%). DLCO was 4.12 mmol/min/kPa (51.6%; ratio of DLCO to alveolar volume, 1.53 mmol/min/kPa/L (63.1%); and total lung capacity, 2.96 L (85.7%). The bronchodilator test was negative. At 9 months, coinciding with a reduction in the dose of prednisone, the patient worsened both clinically (increase in exertional dyspnea) and radiographically (increase in ground-class opacities), and showed a decrease in both DLCO (3.77 mmol/min/kPa: 46%) and SaO_2 (94%), with desaturation during the 6-minute walk test.

Like the patient described by Muñoz et al,[1] our patient showed a functional and histologic pattern similar to that described by Yousem and Dacic,[2] as well as rapid progression to dyspnea on minimal effort. However, our patient also presented respiratory failure, and enlarged mediastinal lymph nodes, not reported by these authors, were observed on the high-resolution CT scan. The laboratory data ruled out the possibility of autoimmune disease, but as the patient had been treated with thyroxine and in contact with 2 exotic birds, and adverse drug reaction and hypersensitivity pneumonitis had to be considered in the differential diagnosis. The literature contains no evidence of an association between thyroxine treatment and interstitial lung or bronchiolocentric disease, although such an association has been described with other drugs.[4] We were unable to perform precipitin testing to rule out hypersensitivity pneumonitis, as the specific reagents for the exotic birds were not available in our laboratory. Bronchoalveolar lavage data, which might have been helpful in the differential diagnosis, were not available either. However, the fact that the patient did not experience significant improvement when the birds were removed from her home and that the histology was not characteristic of this disease allowed us to rule it out. Another entity to be considered in the differential diagnosis, according to Muñoz et al[1] and Yousem and Dacic,[2] is nonspecific interstitial pneumonitis. However, the aggressive onset of symptoms and the bronchiolocentric involvement that our patient experienced are not characteristic of this disease.

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Bullous Emphysema in a Smoker of Marijuana and Tobacco

Enfisema ampolloso en fumadora de marihuana y tabaco

To the Editor:

The relationship between tobacco smoking and chronic obstructive pulmonary disease (COPD) is well known, but the idea that smoking cannabis in its various forms is innocuous has led to habitual consumption and, consequently, to the appearance of associated diseases with the passage of time.

We describe the case of a 30-year-old woman who was a regular smoker of marijuana (34 cigarette-years) and tobacco (17 pack-years). Allergic to penicillin but with no other relevant history, she complained of dyspnea on exertion. Physical examination revealed no abnormalities. A chest radiograph revealed bullae of various sizes in both lung fields. Computed tomography (Figure) showed emphysematous bullae in both pulmonary apices and significant centrilobular and panlobular emphysematous changes with multiple pseudocystic formations ranging in diameter from a few millimeters to 2 cm. The standard workup was normal. The α_1-antitrypsin in blood level was 167 mg/dL. A sweat test showed a value of 40 mmol/L. Spirometry revealed a forced vital capacity (FVC) of 5.08 L (108%), a forced expiratory volume in 1 second (FEV_1) of 3.53 L (95%), and FVC/FEV_1 of 0.69. A bronchodilator test was negative. Lung volume measurements showed a total lung capacity of 6.80 L (113%), a functional residual capacity of 3.79 L (124%), and a residual volume of 1.68 L (97%). Lung diffusion tests showed a carbon monoxide diffusing capacity of 79% and a ratio of carbon monoxide diffusing capacity to alveolar volume of 110%.

The association between marijuana smoking and COPD has been demonstrated.[1] In the short term smoking marijuana leads to bronchodilation,[1] and in the long term it is associated with airflow limitation, chronic cough, bronchitis, and reduced exercise tolerance.[2] Regular smokers of 3 or 4 marijuana cigarettes per day present histologic abnormalities similar to those observed in smokers of approximately 20 tobacco cigarettes per day.[3,4] This difference is partly explained by the distinct ways in which marijuana and tobacco are smoked. Since smokers of marijuana inhale more deeply and retain the smoke for a longer period, the volume is equivalent to 4 times what a smoker of tobacco takes in.[5] Additionally, carboxyhemoglobin and tar levels that result from smoking a marijuana cigarette are from 3 to 5 times greater than the levels produced by a tobacco cigarette. A recent review noted that marijuana smoking is associated with premalignant changes in the lungs, and benzopyrene, an aromatic polycyclic hydrocarbon present in both tobacco and marijuana, has been associated with mutations related with lung cancer.[6]

Furthermore, both marijuana and tobacco seem to be addictive and physicians should not only advise patients against smoking tobacco but also inform them about the harmful effects of marijuana use. This is particularly important when counseling young people.

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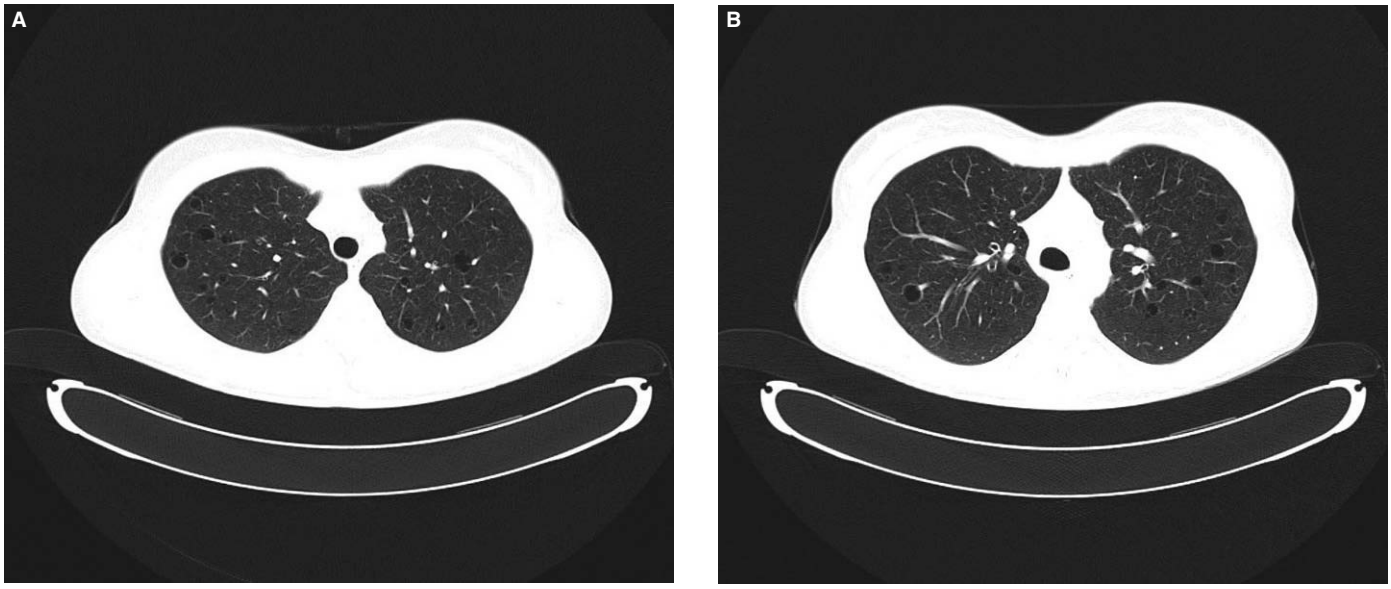

Figure (A and B). Computed tomography showing emphysematous bullae in both lungs and marked centrilobular and panlobular emphysematous changes with multiple pseudocystic formations ranging in diameter from a few millimeters to 2 cm in both lungs.