refers to the existence of lung cancer. In relation to this, we recommend reading an interesting methodological work by Brenner and Grefeller.[6]

Finally, as stated by Campillo-Soto et al, the overlap of the confidence intervals does not preclude a statistically significant difference.[7] In our study, with a confidence interval of 95%, the performance of the transbronchial needle aspiration in the paratracheal and hilar stations was 19.6 to 48.7% for the conventional needle, and 45.4 to 72.9% for the radial endobronchial ultrasound-guided needle. The difference between these two sets of results is statistically significant. As supported by previous studies in other countries, this implies that the second technique probably has a clinically superior efficacy than the first.

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Pulmonary Toxicity due to Methenolone: a Case Report

Toxicidad pulmonar por metenolona: a propósito de un caso

To the Editor:

Drug-induced pulmonary toxicity occurs in between 5 and 30% of patients.[1] We describe a case which meets the diagnostic criteria for pulmonary toxicity due to methenolone.

A male patient, 26 years old, with no pathological history, a professional bodybuilder who was seen for a 24 h history of grade III progressive dyspnoea and dry cough following self-administration of an initial dose of intramuscular methenolone (300mg). In the physical exam he presented no fever, with normal blood pressure, heart rate and breathing. A continuous vesicular murmur was detected with no pathological sounds. The rest proved normal. Analysis showed 18,200 leukocytes/μl (85% neutrophilis and 0.4% eosinophilis), and the results of the arterial blood gas (inhaled oxygen fraction of 0.21) were as follows: pH at 7.45, carbon dioxide blood pressure of 35mmHg, oxygen blood pressure of 60mmHg and HCO_3 of 25mmol/L; the remaining biochemical and coagulation parameters were normal. A simple chest x-ray showed a bilateral alveolointerstitial nodular pattern, Antibiotic treatment was implemented with levofloxacin and the methenolone was discontinued. The CT scan carried out when the patient was admitted showed patches of pneumonitis in ground-glass opacity, predominantly in the upper peripheral regions. The Ziehl-Neelsen stain, Löwenstein-Jensen medium, the *Legionella pneumoniae* and *Streptococcus pneumoniae* antigens, as well as the atypical pneumonia serology (*Mycoplasma pneumoniae*, *L. pneumoniae, Coxiella burnetii*, *Chlamydia pneumoniae* and respiratory syncytial virus), all proved negative. The spirometry showed: forced vital capacity (FVC) 4.76 l (79%), Forced expiratory volume in the first second (FEV_1) 4.45 l (93%) and FEV_1/FVC 93%. The diffusing capacity of the lung for carbon monoxide (DLCO) was 10.17mmol/min/kPa (75%), and the corrected value per alveolar volume (DLCO/VA), 1.81mmol/min/kPa/l (86%). 48 h after admission, antibiotic treatment was suspended. The patient's symptoms improved and an x-ray resolution carried out after seven days, with an oxygen saturation level taken by pulse oximetry (breathing local air) of 97%. After a month, a chest x-ray (fig. 1) continued to show no abnormalities and functional improvement was observed: FVC, 5.45 l (91%), FEV_1 5.06 l (106%), FEV_1/FVC, 92%. The DLCO was 15.41 mmol/min/kPa (114%) and the DLCO/VA, 2.42mmol/min/kPa/l (115%).

Methenolone is an anabolic hormone used by athletes in order to increase their physical performance. A bibliographic search of MEDLINE (1976-2008) and the Pneumotox[2] Web page confirmed that there has been no previous case reported of pulmonary toxicity due to anabolic steroids.

It appears that the drugs produce pulmonary lesions through an immunological or cytotoxic mechanism, which may be presented as acute or subacute. Clinical suspicion was established in the face of indicative symptoms in a patient who had taken a harmful drug, together with the radiological[3] and functional abnormalities of this type of disease. Functional change is often restrictive, with a low carbon monoxide diffusion capability. Diagnosis is reached through exclusion, aetiological and environmental infections must be ruled out. There is a temporary relationship between taking the drug and the start of the symptoms, which improve once the patient stops taking the drug, and recur if they take it again.[4] We believe that this case meets the criteria for considering methenolone as the cause of the lung damage. Although the finding of lymphocytes with an inverse CD4/CD8 ratio in the bronchoalveolar lavage would support the diagnosis,[5,6] in our case we did not consider it necessary given the patient's initial favourable evolution.

In regard to the treatment, administering glucocorticosteroids is not always necessary,[6] as occurred in this case, in which a clinical and radiological improvement took place upon discontinuing the drug.

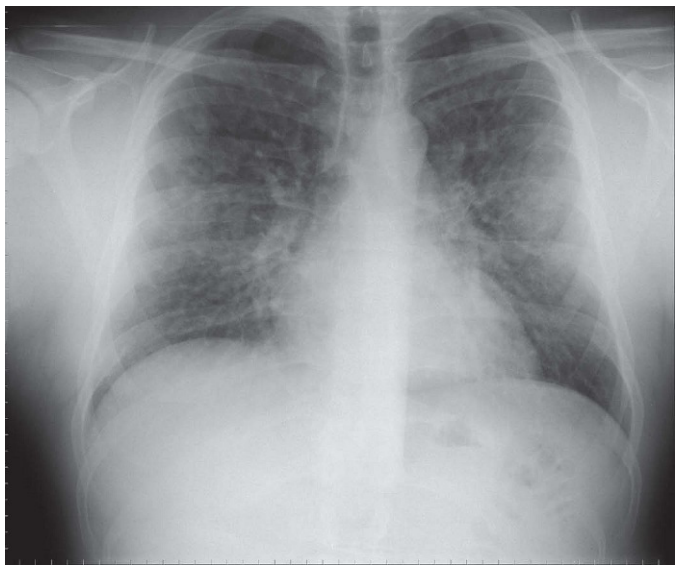

Figure 1. Posteroanterior chest x-ray.

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