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Letters to the Editor

Sarcoidosis with lung and systemic involvement in a 5 year old girl

Sarcoidosis con afectación pulmonar y sistémica en una niña de 5 años

To the Editor:

Sarcoidosis is a granulomatous disease of unknown etiology, and is very rare in children.[1,2] There are two very different clinical forms during childhood: preschool sarcoidosis, which is produced in children under the age of 5, and sarcoidosis in older children. Preschool sarcoidosis includes cases that start between the first few months of life and the age of 4, which is characterized by a triad of cutaneous eruptions, arthritis and uveitis. After the age of 5, juvenile sarcoidosis changes its form of presentation, usually presenting multi-systemic disease. The most frequent form in children over the age of 5 is constitutional syndrome. Together with these general symptoms, fever and pulmonary symptoms are frequent, peripheral adenopathies can be felt and there may be hepatomegaly or splenomegaly.[3] The lung is the most frequently-affected organ. Lung symptoms are normally mild.[4] The parenchymal involvement is usually an interstitial pattern, although nodular, alveolar and fibrotic patterns have been reported.[5] The diagnosis of sarcoidosis is by exclusion. Once the clinical suspicion is established, the most cost-effective test is biopsy.[1] Treatment for sarcoidosis is based on oral steroids and methotrexate.[3,6] Despite good clinical evolution, the long-term prognosis of these cases is uncertain.[6]

We present the case of a five-year-old girl with pulmonary and systemic sarcoidosis. The patient, with no medical history of interest, came to the emergency unit due to fever evolving over 10 days, with no improvement after antibiotic treatment. Her pediatrician informed us that the patient had presented asthenia, periumbilical abdominal pain and intermittent low-grade fever over the previous five months.

Physical examination revealed mild hepatomegaly and splenomegaly. Chest radiography taken at the ER visit revealed a bilateral alveolar-interstitial pattern, predominantly at the bases (fig. 1A). Hemogram was compatible with an acute infectious process. ESR and PCR remained normal throughout the hospital stay. O_2 saturation and capillary gases were normal. The levels of angiotensin converting enzyme (ACE) were high (236 U/L). All cultures, including bronchial brushing, were negative, as well as the rest of the microbiological studies. On later radiography, the appearance of mediastinal and hilar adenopathies were observed, with an improvement in the described pattern. Abdominal ultrasound revealed splenomegaly and multiple perisplenic and retroperitoneal adenopathies, as well as in the iliac fossae, and an important nephrocalcinosis (fig. 1D). Thoracic and abdominal computed tomography (CT) confirmed the findings described, revealing in the pulmonary parenchyma diffuse bilateral alveolar-interstitial pulmonary pattern, with the presence of acinar nodules, thickening of the interlobular septa and areas of ground-glass opacities; hilar and mediastinal adenopathies (fig. 1B-C).

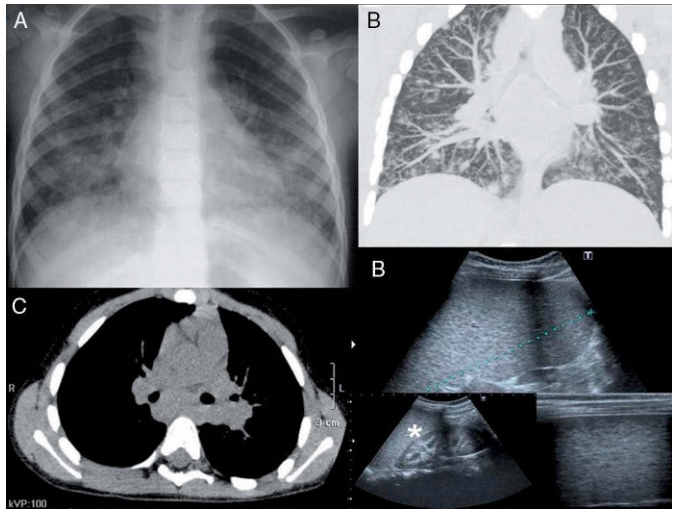

Figure 1. A. Chest x-ray, widening of the paratracheal stripes and increase in hilar size secondary to the presence of adenopathies. Undefined increase of bilateral basal density, with a tendency towards confluence, associated with small nodular images. B. Chest CT scan, ground-glass nodular opacities and well-defined nodules with diffuse distribution. Thickening of the interlobular septa. C. Chest CT scan, hilar and subcarinal adenopathies. D. Abdominal ultrasound, splenomegaly with the presence of a multitude of small focal hypoechogenic lesions due to the presence of granulomas. Increased renal pyramid echogenicity, caused by calcium deposits and nephrocalcinosis

Biopsy was taken from the adenopathy in the right iliac fossa, with a diagnosis of reactive lymphadenitis. Given all these findings, once infectious disease and lymphoproliferative processes were ruled out, the first diagnosis considered was stage II pulmonary sarcoidosis. The following step to arrive at a definitive diagnosis was lung biopsy together with bronchoalveolar lavage, but given the spontaneous regression of the lung lesions and the abdominal lymphadenitis, we decided to adopt a wait-and-see approach and to keep a close watch on the patient.

Six months later, faced with a new pulmonary exacerbation, thoracic CT showed multiple nodules with axillary and mediastinal adenopathies. Treatment with corticosteroids was initiated (15 mg/day), which was increased to 30 mg/day plus methotrexate (7.5 mg/week) after the appearance of focal splenic lesions, suggesting sarcoid granulomas, observed on abdominal ultrasound. The patient currently continues with the established treatment of corticosteroids and methotrexate, and is examined periodically.

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Silicosis in Quartz Conglomerate Workers

Silicosis en trabajadores de conglomerados de cuarzo

To the Editor:

We have read the recent publication entitled *Silicosis: a Disease With an Active Present* by Dr. C. Martínez et al.[1] First of all, we would like to express that we coincide with the reflections of the authors and we would like to provide our experience in this regard.

From July 2009 to February 2010, we have diagnosed six workers with silicosis. They are employed by three companies in the same town and are exposed to the dust generated by handling kitchen and bathroom countertops made of artificial quartz conglomerates.

All the patients are male, and what is remarkable is their young age, with a mean of 29 years (age range: 26-37), and short period of exposure: between 5 and 12 years (mean: 9 years) (table 1). This could be an indication of the high toxicity of these materials,[2] or that the preventative measures are either not sufficient or that they are not being adequately carried out.

In the radiologic studies of the chest, we have found micronodular interstitial patterns predominantly in the upper lobes and bilateral hilar adenopathies quite compatible with typical images of silicosis.[3] These findings, together with a restrictive functional alteration and an occupational history of exposure to silica dust, are sufficient for the diagnosis of silicosis.[4] In our first four cases, in addition, we also performed pulmonary biopsy, confirming a histological diagnosis of interstitial fibrosis compatible with pneumoconiosis.

We believe that the current increase in cases of pneumoconiosis due to exposure to silica in small businesses working with ornamental synthetic stone mandates a rigorous assessment of the preventive measures,[5] as well as active evaluation of the workers in these industries.

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Table 1
Characteristics of the patients, radiological findings and spirometry

	Age/sex	Tobacco habit	Exposure in years	Reason for consultation	Chest CT/radiological pattern	Spirometry
1	30 ♂	Non-smoker	11	Bronchitis	Diffuse micronodular; hilar adenopathies	FVC: 74%. FEV_1: 81%. FEV_1%: 87%
2	37 ♂	Non-smoker	9	Company physical examination	Micronodular in upper fields; hilar adenopathies	FVC: 61%. FEV_1: 68%. FEV_1%: 88%
3	25 ♂	Non-smoker	5	Bronchitis	Reticulum-diffuse nodular; hilar and mediastinal adenopathies	FVC: 66%. FEV_1: 77%. FEV_1%: 92%
4	30 ♂	Ex-smoker	10	Company physical examination	Diffuse micronodular	FVC: 57%. FEV_1: 51%. FEV_1%: 71%
5	28 ♂	Ex-smoker	12	Company physical examination	Diffuse micronodular; hilar adenopathies	FVC: 63%. FEV_1: 56%. FEV_1%: 70%
6	26 ♂	Non-smoker	8	Company physical examination	Micronodular in upper fields	FVC: 66%. FEV_1: 72%. FEV_1%: 85%

CT: computed tomography; FEV_1: forced expiratory volume in 1 second; FVC: forced vital capacity.