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Scientific Letters

Sarcoidosis Associated With Psoriasis: 2 Disease Entities, One Pathogenic Pathway☆

Asociación psoriasis-sarcoidosis: 2 entidades y una vía patogénica común

To the Editor:

Psoriasis is a chronic inflammatory disease mainly affecting the skin. The pathogenic mechanism of this condition is largely due to overstimulation of CD4 Th1 and Th17 lymphocytes, which have a well-established role in the formation of sarcoid granulomas.[1]

We present the case of a 38-year-old woman with a diagnosis of severe psoriasis vulgaris, with a finding of mediastinal lymphadenopathies and ground glass infiltrates, finally diagnosed as sarcoidosis.

A 38-year-old woman with a 9-year history of severe psoriasis vulgaris (Fig. 1) receiving treatment with topical corticosteroids. A chest radiograph and Mantoux testing were performed before starting systemic biological therapy. She had no respiratory symptoms, but the radiograph showed a chance finding of right hilar lymphadenopathies (Fig. 2A). The examination was completed with a chest computed tomography (Fig. 2B), showing pathologically enlarged hilar and mediastinal lymph nodes and bilateral ground glass parenchymal infiltrates. Angiotensin-converting enzyme levels were normal. Fiberoptic bronchoscopy was performed. Bronchial aspirate and bronchoalveolar lavage (BAL) cultures were negative, and cytology was also negative for malignancy. BAL immunophenotyping revealed a lymphocyte population composed predominantly of CD3+ (96%) with a CD4/CD8 ratio of 6.67. Endoscopic ultrasound-guided fine-needle aspiration was performed, but the sample was poorly representative and the result was inconclusive. Mediastinal lymphadenopathy biopsy was performed

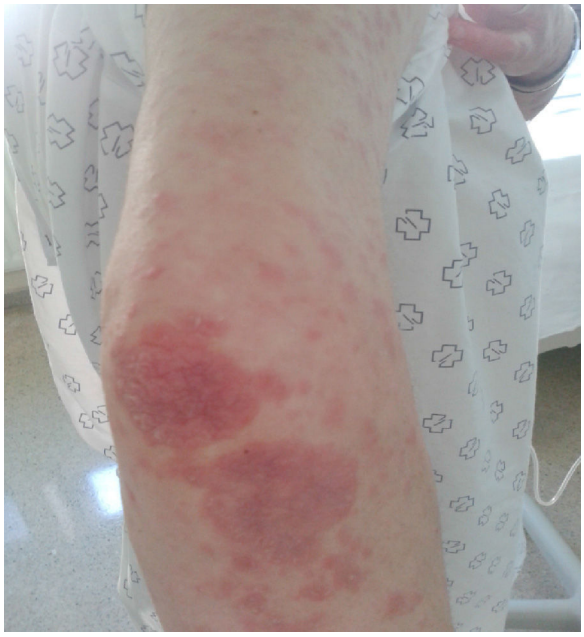

Fig. 1. Severe psoriasis vulgaris on the patient's arm and forearm.

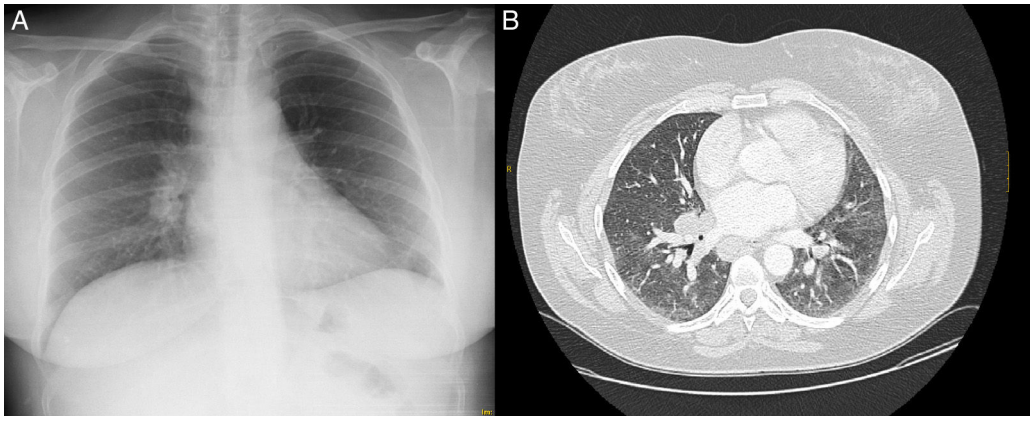

Fig. 2. (A) Chest radiograph showing prominent right lung hilum with nodular morphology. (B) Chest CT showing bilateral hilar lymphadenopathies and ground glass infiltrates.

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using video-assisted mediastinoscopy, revealing blurred lymph node architecture occupied by multiple confluent granulomas of a similar size, formed of epithelioid-like histiocytes, with no necrosis and the presence of multinucleated giant cells. PAS and Ziehl-Neelsen stains were negative. All these findings were consistent with the diagnostic suspicion of stage II sarcoidosis.

Psoriasis is one of the most common systemic inflammatory diseases. It is characterized by increased immune system activity, mainly derived from activation of the Th1 and Th17 lymphocytes.

Sarcoidosis is a chronic disease characterized by the formation of non-caseifying epithelioid granulomas, most commonly in the lungs, skin and eyes. Although the etiology is still partly unknown, we know that the immunological basis of the disease consists of an accumulation of CD4 T cells in the inflammation sites, which interact with macrophages, causing an imbalance in favor of a Th1 cytokine profile, which finally triggers the granulomatous inflammatory process.[1] The role of the Th17 lymphocytes in the formation of sarcoid granulomas has also been established, as has their importance in the alveolitis phase and in progression to the fibrotic stage of the disease.[1]

Very few cases of concomitant psoriasis and sarcoidosis have been reported,[2–4] although recently a greater risk of presenting sarcoidosis has been observed in psoriasis patients compared to the general population, an association that moreover is severity-dependent.[5]

These data all go to support the hypothesis of a shared pathogenic pathway, although studies are needed to elucidate the possible role of other factors, such as the sarcoid disease phenotype, a possible paradoxical effect of treatment of the psoriasis itself (a phenomenon described with anti-TNFα antibodies[6]), the importance of environmental factors such as smoking, and the possible clinical, prognostic and therapeutic implications of the combination of both diseases.

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The Possible Role of Asbestos Exposure in the Pathogenesis of a Thoracic Non-Hodgkin Lymphoma☆

Posible papel de la exposición al asbesto en la patogenia de un linfoma no Hodgkin torácico

To the Editor,

Various studies suggest that a causal relationship might exist between asbestos exposure and the development of lymphoproliferative disorders.[1,2] We report the appearance of a non-Hodgkin lymphoma in a patient with a history and signs of heavy exposure to asbestos.

An 86-year-old man was admitted to a general hospital in January 2011 for dyspnea, fatigue, and weight loss. Chest X-ray showed left pleural effusion. Past disease history included gastric resection for peptic ulcer in 1950, aortic valve replacement in 2008, and colon polypectomy in 2010. The patient had worked for about 14 years as a shipwright with various firms in the shipbuilding industry in Trieste, and as a mechanic for 4 years in an oil refinery. Chest computed tomography showed left pleural effusion, and bilateral thickening of the pleura with calcifications. Thoracoscopy was not performed in view of the patient's age. In June 2012, chest radiography showed an opacity at the apex of the right lung. By July 2012, the thoracic opacity had spread to the right upper mediastinum. The patient died in July 2012 with a diagnosis of acute respiratory failure, due to probable neoplasia of the pleura. Right pleural effusion was observed on autopsy. Right parietal pleura showed large pleural plaques and soft, whitish neoplastic nodules. Similar nodules were visible on the surface of the right lung without infiltration of the parenchyma, and in the pericardium. The left lung showed atelectasis of the lower lobe and marked edema of the upper lobe. Left pleural effusion was no longer visible. Mediastinal lymph nodes appeared enlarged with masses similar to those seen in the right lung. Histological examination found that the nodules in the right pleura, lung and lymph nodes were composed of sheets of small and large lymphoid cells. Immunohistochemistry (CD20, CD3, CD56, CK Ae1-Ae3, synaptophysin) was consistent with a diagnosis of B cell non-Hodgkin lymphoma. The lungs showed asbestosis: high burdens of asbestos bodies (57 000 bodies per gram of dried tissue) were isolated from the lung after chemical digestion of the pulmonary tissue (Smith and Naylor method[3]).

The clinical diagnosis of this patient with a history of severe asbestos exposure and pleural pathology remained undefined. The case is similar in some ways to that described by Parisio et al.[4] Numerous studies have explored the possible relationship between asbestos exposure and non-Hodgkin lymphoma or hematopoietic cancer in general.[1,2,4] The association between non-Hodgkin lymphoma and asbestos-related mesothelioma, in particular, does not seems to be exceptional.[1] In a recent study of a large mesothelioma series, including more than 3600 cases,[2] there were 45 cases of hematopoietic malignancies. While the role of radiation in the genesis of mesothelioma in patients who had received previous radiotherapy is plausible, the majority of non-Hodgkin lymphoma patients were radiotherapy-naïve.

Various elements suggest a relationship between asbestos and non-Hodgkin lymphoma. Firstly, the relatively high prevalence of the association between mesothelioma and lymphoma (both rare in the general population) is difficult to attribute to chance.

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