

this tumor and its natural history, and that treatment options are limited.

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## Delayed Pulmonary Fibrosis (Usual Interstitial Pneumonia) in a Patient With Previous Uncomplicated H1N1-Associated Pneumonia\*



### Fibrosis pulmonar tardía (neumonía intersticial usual) en un paciente con antecedentes de neumonía asociada a H1N1 no complicada

To the Editor,

In February 2011, a 53-year-old man was admitted to our hospital for fever, non-productive cough and dyspnea on effort; he did not report any history of workplace exposure during the medical examination.

Computed tomography (CT) of the chest showed bilateral pulmonary consolidations, mainly involving the lower lobes.

No leukocytosis was seen on the blood tests, and the differential leukocyte count was 72.7% neutrophils and 18.5% lymphocytes. Serum biochemistry results were normal. Tests for anti-neutrophil cytoplasmic antibodies (ANCA), antinuclear antibodies (ANA), anti-DNA antibodies and extractable nuclear antigens (ENA) were negative.

Arterial blood gases (ABG) showed moderate hypoxemia, with partial oxygen pressure ( $\text{PO}_2$ ) 52 mmHg,  $\text{PCO}_2$  34 mmHg, and pH 7.48; arterial oxygen saturation ( $\text{SaO}_2$ ) was 88%.

Pharyngeal swab analysis was positive for H1N1.

The patient was referred to the infectious diseases unit, where he received combined treatment with prednisone 25 mg, orally, twice a day for 10 days, cefotaxime 1 g, intravenously, twice a day for 10 days, inhaled zanamivir (2 mg  $\times$  5 mg) twice a day for 5 days and oseltamivir, 1.75 mg capsule twice a day for 5 days. The patient's clinical situation improved rapidly, as reflected in radiological and ABG results. Twenty days later, he was discharged in good condition.

A chest CT was performed before discharge, showing good recovery of lung function, and ABG results were normal (pH=7.41;  $\text{PCO}_2=37$  mmHg;  $\text{PO}_2=85$  mmHg and  $\text{SaO}_2=96\%$ ).

The patient was then referred for respiratory follow-up, including high-resolution computed tomography (HRCT) scans, ABG analysis and lung function testing with determination of the diffusing capacity of the lungs for carbon monoxide (DLCO).

At the 1, 3 and 6-month follow-up visits, the patient was asymptomatic; serial chest HRCTs showed slight basal consolidation, less than 3 cm in diameter, in the form of reticular and ground glass opacities.

During this time, the radiological signs did not alter either in shape or in size. Lung function test results were normal:  $\text{FVC}=86.6\%$ ;  $\text{FEV}_1=96\%$ ;  $\text{FEV}_1/\text{FVC}$ : 88.47; DLCO: 78% and  $\text{SpO}_2$ : 96%.

In May 2012, the patient became symptomatic again, with non-productive cough and dyspnea on effort. He returned to the clinic with marked reduction in lung function parameters:  $\text{FVC}=66.7\%$ ;  $\text{FEV}_1=76.2\%$ ;  $\text{FEV}_1/\text{FVC}=90.63$ ,  $\text{SpO}_2=91\%$ ; DLCO values were also severely reduced (55% compared to 78%).

Another chest HRCT was then performed, revealing ground glass and peripheral reticular opacity, particularly in the lung bases (Fig. 1A).

Blood and serum test results were normal. A flexible fiberoptic bronchoscopy was performed, and a transbronchial biopsy was obtained, the results of which were indecisive—only inflammatory lymphocyte infiltration and alveolar septal fibrosclerosis were found on histopathology examination. Bronchoalveolar lavage (BAL) results were negative.

A surgical lung biopsy was performed using video-assisted thoracoscopic surgery (VATS) under general anesthesia with one-lung ventilation, and 3-port incision in the right side (one sample per lobe).

Histopathological analysis revealed widened alveolar septa with type II pneumocyte proliferation and mononuclear inflammatory infiltrate with interstitial fibrosis in patchwork pattern, suggesting usual interstitial pneumonia (Fig. 1B). After an incident-free post-operative period, the patient was referred to the pulmonology unit for appropriate medical treatment.

Although the etiology of usual interstitial pneumonia (UIP) is unknown, the following risk factors have been proposed: acute respiratory distress syndrome (ARDS), environmental exposure to metal dust, smoking habit, connective tissue disorders, drug toxicity, chronic viral infections, such as Epstein–Barr virus, cytomegalovirus, hepatitis C virus, human herpesvirus (HHV)-7 and HHV-8.<sup>1</sup>

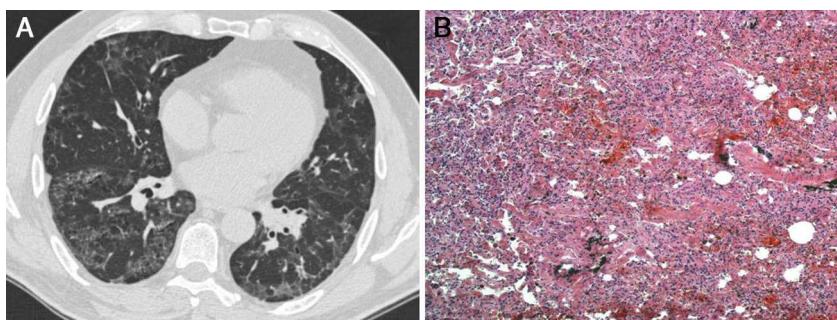
Ground glass opacity on chest imaging studies and reduced DLCO have been reported in H1N1 pneumonia in a study with 3 months' follow-up.<sup>2,3</sup> We report a very uncommon case of late-onset UIP after uncomplicated H1N1 pneumonia in a 53-year-old man, detected in HRCT obtained one year after the disease onset.

These results, along with the histopathology examination, were consistent with the development of pulmonary fibrosis. Pulmonary fibrosis may occur after ARDS or ventilator-associated pneumonia.<sup>4,5</sup>

In our patient, radiological signs of fibrosis were confirmed by histopathological examination of surgical biopsy specimens obtained by standard VATS.<sup>6</sup>

By presenting this case, we wish to draw attention to long-term sequelae in a patient with no prior history of ARDS and who did not require mechanical ventilation. Symptomatic pulmonary

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**Fig. 1.** (A) Chest high-resolution computed tomography showing ground glass and peripheral reticular opacities, particularly evident in the lung bases. (B) Histopathological examination revealed the following: widened alveolar septa with type II pneumocyte proliferation and inflammatory mononuclear infiltrate with interstitial fibrosis in a patchwork pattern, suggesting usual interstitial pneumonia (hematoxylin-eosin staining, original magnification 100×).

fibrosis with a UIP pattern on HRCT and histopathological signs were detected one year after an influenza-like infection, but otherwise, the patient was a healthy adult who did not present any of the comorbidities usually associated with influenza.

Several cases of ARDS subsequent to H1N1 have been described in the literature,<sup>4–6</sup> but this is the first report of a correlation between H1N1 and UIP, and we believe this to be the unique feature of our case.

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## Occult Alveolar Hemorrhage in a Patient With Bronchopulmonary Kaposi Sarcoma<sup>☆</sup>



### Hemorragia alveolar oculta en paciente con sarcoma de Kaposi broncopulmonar

We report the case of 23-year-old man with human immunodeficiency virus (HIV) infection and a history of Kaposi sarcoma of the palate, treated with radiation therapy and antiretrovirals 2 years previously. He presented due to the appearance of violaceous skin lesions on the face, dyspnea and pleuritic pain with a CD4 T-lymphocyte count of 149 cells/mm<sup>3</sup> and viral load of 52 092 copies/ml. High resolution computed tomography of the chest (Fig. 1) revealed bilateral nodules with irregular margins and ground glass opacities, peribronchial cuffing and left pleural effusion. Bronchoscopy showed a raised lesion in the mucous membrane of the apical segment of the right upper lobe. Abundant hemosiderophages were found in the bronchoalveolar lavage (BAL) fluid, confirming alveolar hemorrhage. Immunohistochemistry was positive for human herpesvirus 8 (HHV-8). Treatment began with liposomal doxorubicin and antiretroviral treatment was switched. The patient remains alive at 8 months.

Kaposi sarcoma involving the lung occurs in 6%–32% of patients with acquired immunodeficiency syndrome (AIDS). It presents with skin lesions,<sup>1,2</sup> and in 47%–75% of patients it is diagnosed post-mortem.<sup>3</sup> Lesions can appear in the pulmonary parenchyma, bronchial tree, pleura, chest wall, and mediastinal lymph nodes,<sup>1,3</sup> and CD4 T-lymphocyte count is generally <100 cells/mm<sup>3</sup>.<sup>3</sup> In 80% of cases, death is a result of co-infection, due to cytomegalovirus, *Mycobacterium avium* complex, *Pneumocystis jirovecii*, bacterial pneumonia and herpes simplex infection.<sup>1</sup>

Chest computed tomography reveals poorly defined bilateral nodules, distributed symmetrically around the bronchial vessels (flame-like lesions). Other findings include septal peribronchial and interlobar cuffing, progressive air space consolidation and ground glass opacities.<sup>3</sup>

Lesions on the palate are a strong predictor for bronchopulmonary involvement.<sup>1</sup> Typical lesions observed on bronchoscopy are red or violaceous cherry-like plaques in the bronchial Tree.<sup>1</sup> HHV-8 is detected in BAL, which is highly specific (95%–98.9%) with variable sensitivity (58%–100%).

Occult alveolar hemorrhage has been described in HIV-positive patients with respiratory symptoms and abnormal radiological findings in the absence of hemoptysis. In 35.6% of cases, bronchopulmonary Kaposi sarcoma was detected, and of these 60.5% had occult alveolar hemorrhage.<sup>4</sup>

Vincent et al.<sup>5</sup> subsequently characterized the following risk factors for AIDS-related alveolar hemorrhage: Kaposi sarcoma (OR: 5.3; 95% CI: 1.8–16.7; *P*=.003); cytomegalovirus pneumonia (OR: 9.8; 95% CI: 1–100; *P*=.05); hydrostatic pulmonary

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