typical of Aspergillus and growth of these hyphae on culture. How-
however, histological confirmation is not always possible, so diagnosis
must be based on a combination of clinical, radiological, micro-
biological, and immunological findings.3,4 Azoles are the primary
treatment option in CPA.4 Our patient showed disease progression
during itraconazole treatment, which may have been a conse-
quence of the temporary interruption of treatment or resistance to
that compound. Wild-type resistance to azoles has been reported
in Aspergillus strains in azole-naïve patients.2 Disease progression
may also have been caused by sub-therapeutic serum levels of itra-
conazole during treatment, despite the administration of a higher
than recommended dose. Surgical treatment is controversial, since
it is associated with significant post-operative complications and
disease recurrence, even at distant sites, so should be reserved
for selected patients.2,3 In our case, surgery did not appear to be
the best therapeutic approach, since the patient had bronchiec-
tasis in the contralateral lung, which may have been associated
with fungal colonization.6 Nevertheless, this option could have
been considered if medical intervention had failed. Although
the patient showed no evidence of immunosuppression, still unde-
finite immune deficiencies cannot be ruled out.7 Consequently,
while medical treatment appears to have clear advantages in
cases in which it is well tolerated, the long-term outcome is
unknown.

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Psychogenic Cough: A Rare Cause of Chronic Cough

Tos psicógena: una rara causa de la tos crónica

To the Editor:

Psychogenic cough is a rare cause of chronic cough in adults. It is
typically persistent, disrupts daily activities and causes long-term
morbidty. In contrast with cough of organic etiology, there is no
clinical or laboratory evidence of disease.1,2

Efforts to make an early diagnosis will morbidity, pre-
vent fixation of symptoms and avoid unnecessary procedures and
therapies.2

We report the case of a 45-year-old woman, Caucasian, non-
smoker, no habitual medication and no known previous allergies.
The patient consulted her family doctor due to a 9-week history
of coughing fits.

The cough started and persisted after an upper respiratory
tract infection treated with antibiotics (amoxicillin/clavulanic acid)
and antihistamine (hydroxyzine). It was described as violent,
nonproductive, occurring every few minutes, disrupting speech,
work and daily activities, but not sleep because it disappeared at
night.

Physical examination, including ear, nose and throat evaluation
was normal, and no noticeable motor tic was observed.

No organic etiology was detected on medical examinations,
including chest and sinus X-rays, skin-prick test for allergies,
spirometry with bronchial challenge, thyroid ultrasound and chest
computed tomography.

Pharmacological therapy with antihistaminic/decongestant
(pseudoephedrine+triprolidine), inhaled corticosteroid (budes-
one) and bronchodilator (salbutamol) was ineffective. A trial of
acid suppression (omeprazole), prokinetic (metoclopramide) and
dietary modification also failed.

During investigation, regular consultations were scheduled. In
the course of these consultations, she reported depressive symp-
toms. She related them to work-related distress, which began 2
months before the coughing fits, when she was moved to a new
position. We perceived that cough was always present except
when she spoke about her work-related distress. This observation,
in association with negative findings on diagnostic tests, lack of
response to therapy and the clinical characteristics of the cough,
raised the suspicion of a psychogenic etiology.

A plan of weekly cognitive psychotherapy, in addition to antide-
pressant therapy with sertraline, was initiated. After five weeks,
the cough disappeared and depressive symptoms decreased. No
relapses were reported in the following twelve months.

In adult patients with chronic cough, doctors should always
work toward a clear diagnosis, considering common and rare
illnesses.3

Little has been published on diagnostic approaches.1,4 However,
when extensive evaluation and therapy fail to detect an organic
cause, psychogenic cough should be considered.1,2,3 Upper respira-
tory infections, depressive disorders and work distress have been
described as precipitating factors.1,5 In this case, a psychogenic ori-
gin was first suggested by cough absence during sleep and while
speaking about work distress.1,4

Non-pharmacological therapies have been reported to be more
effective than pharmacological treatments.1 However, there is
a lack of randomized, controlled studies comparing different
strategies.2

This case highlights the role of an empathic and integrated
approach by the family doctor. It made early diagnosis possible,
and non-pharmacological and pharmacological treatments could
be started immediately, thus avoiding specialist referral and iatro-
genic complications.

In conclusion, psychogenic cough is a rare entity, diagnosed after
extensive exclusion of organic causes, positive clinical findings and
response to specific therapy.

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Pulmonary manifestation of lymphomatoid granulomatosis

Granulomatosis infomatoide: afectación pulmonar

To the Editor,

We report the case of a 57-year-old woman, former smoker, with a history of non-pathological cervical and axillary lymphadenopathies, non-necrotizing granulomatous mastitis and acute dorsal myelopathy. She was admitted for bronchopneumonia, and underwent bronchoscopy with transbronchial biopsies, which were inconclusive, and bronchoalveolar lavage (BAL) showing 65% lymphocytes and a normal CD4/CD8 ratio. The patient was readmitted due to right pleural effusion, categorized as lymphocytic exudate with no malignant cellularity, hepatosplenomegaly, and signs of pulmonary hypertension. Low levels of IgG subclasses were reported and treatment was initiated with prednisone. The patient subsequently developed dyspnea, anorexia and asthenia. Chest computed tomography showed peribronchial pulmonary nodules with undefined borders, air bronchogram sign, and tendency to converge into large masses in the lower lobes. These masses were surrounded by ground glass opacities and mediastinal lymphadenopathies, indicative of lymphomatoid granulomatosis. Ground glass opacities and lymphadenopathies, however, are not typical of lymphomatoid granulomatosis, and may have been associated with the patient’s smoking habit (Fig. 1). Lung function tests showed moderate restriction, with 36% diffusion, and laboratory reports revealed leukopenia due to lymphopoenia. A bone marrow biopsy and a second bronchoscopy were performed, from which BAL showed predominant lymphocytes and a normal CD4/CD8 ratio. No additional data could be obtained from biopsy of a paratracheal lymphadenopathy. Culture and cytology were negative. A lung biopsy was obtained, after which the patient showed clinical and radiological worsening.

The result of the bone marrow biopsy suggested a T-cell-rich large-B-cell lymphoma. Chemotherapy was initiated, without improvement. Pathology results from the lung biopsy were inconclusive. The patient continued to worsen rapidly and progressively until she died. Autopsy confirmed diffuse T-cell and histiocytoderich large-B-cell lymphoma, associated with Epstein–Barr virus (EBV), with perivascular involvement and changes indicative of lymphomatoid granulomatosis.

Lymphomatoid granulomatosis was first described in 1972 by Liebow et al. It occurs primarily in patients aged between 40 and 60 years, and mainly in men (2:1). It is an angiocentric and angiodestructive process, affecting extranodal regions that in 90% of cases involves the lung. This disease of the B-cells is thought to be associated with EBV infection, large-B-cell lymphoma, and immunosuppressive states. Typical lung involvement is characterized by nodular lesions, with lymphocytic invasion of the blood vessels, that may converge and cavitate. It is diagnosed from histology findings, including polymorphic lymphoid infiltrates, transmural infiltration of the arteries and veins by lymphoid cells, and focal areas of necrosis.

The treatment of lymphomatoid granulomatosis is controversial, and varies according to the histological grade. In the absence of symptoms and if the histological grade is low, the patient should be monitored. Other cases are treated with prednisone and cyclophosphamide, although standard therapy for non-Hodgkin lymphoma has also been attempted.

Satisfactory therapeutic outcomes have recently been achieved with interferon-alfa-2b and rituximab. The prognosis of lymphomatoid granulomatosis varies: spontaneous remission is observed in 20% of cases, while in others, mean survival is 2 years, with a 5-year mortality of 63%–90%.

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Fig. 1. Chest computed tomography, axial slice. Parenchymal window.