Chronic Lung Infection Caused by *Trichosporon mycotoxinivorans* and *Trichosporon mucoides* in an Immunocompetent Cystic Fibrosis Patient

*Infección pulmonar crónica causada por Trichosporon mycotoxinivorans y mucoides en un paciente inmunocompetente con fibrosis quística*

To the Editor:

Very few studies have been published in the medical literature on systemic infections caused by *Trichosporon* spp in immunocompetent patients, and even fewer in cystic fibrosis (CF) patients. This species is often associated with acute processes with poor prognosis.1-4

We report a review of the literature and a case study of a CF patient with chronic bronchial infection (CBI) caused by *Trichosporon mycotoxinivorans* (T. mycotoxinivorans) and *Trichosporon mucoides*, who progressed well during follow-up.

This is a 37-year-old man, diagnosed at the age of 4.5 months with CF (F508del/G542X), with mild pulmonary and gastrointestinal involvement, and a history of CBI due to methicillin-sensitive *Staphylococcus aureus* and intermittent bronchial infection with *Pseudomonas aeruginosa* resolved in 2005. He continued to receive rapid-action bronchodilators, physiotherapy, pancreatic enzymes, and liposoluble vitamins.

Five years ago, during a routine visit, *T. mucoides* was isolated from a microbiological culture of the sputum. In the complementary examinations, spirometry was normal (FEV1: 2.71 | l65%) with basal oxygen saturation (SO2) 96%. In view of this finding, intravenous conazol 200 mg/24 h was started. In successive sputum cultures, T. mycotoxinivorans was isolated and persisted until the patient’s last visit. No radiological (Bhalla: 16) or functional (FEV1: 3.25 l/95%) and basal SO2: 97%) worsening was observed. Mean exacerbations/year in the 5-year follow-up was 1.2, all of which were mild, treated with oral antibiotic therapy according to the sensitivity profile, similarly to previous years.

The first human infection in CF with *T. mycotoxinivorans* was a case of pneumonia with fatal outcome, published in 2009. Cases published subsequently also had very poor prognosis.1,2 Shah et al.2 reported a series followed up for a maximum of 6 years, of which 4 patients had CBI due to *T. mycotoxinivorans*, and in another, it was isolated once. No correlation was found between this infection and the very high number of subsequent exacerbations, but it can be supposed that *T. mycotoxinivorans* played a part,2 both in the clinical symptoms and the prognosis.

Although it remains to be clarified, workplace exposure, transplantation and treatment, diabetes, inhaled and systemic corticosteroid, malnutrition, severely compromised lung function, intrinsic drug resistance to mycotic infections, or the chronic use of inhaled or systemic antibiotic treatment, may be risk factors for developing *T. mycotoxinivorans* infection in CF.1-4 Our patient did not present any risk factors or clinical, radiological or functional worsening in the 5 years before the appearance of *Trichosporon* spp. We believe that the change of species in our case was related with 2 events. The first was that the *Trichosporon* genus was recently reorganized.1 The second was the method of identification used, phenotyping techniques (API®, VITEK®) and mass spectrometry (MALDI-TOF).3

After close examination of our case, a CBI with no clinical repercussions, and after reviewing the literature, we conclude that *Trichosporon* spp, and in particular *T. mycotoxinivorans*, are associated with widely varying clinical manifestations in CF, although to date most cases have been severe and fast progressing with a fatal outcome. Under the right circumstances,1,3 patients may present chronic infection caused by this fungus.

References


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1 Please cite this article as: de Borja Martínez Muñiz F, Martínez Redondo M, Prados Sánchez C, García Rodríguez J. Infección pulmonar crónica causada por *Trichosporon mycotoxinivorans* y *mucoides* en un paciente inmunocompetente con fibrosis quística. Arch Bronconeumol. 2016;52:400.