Table 1
Clinical Laboratory Tests on Admission and Arterial Blood Gases.

	Hb (g/dl) ER	Hct (%) ER	Urea (mg/dl) ER	Cr (mg/dl) ER	Na (mEq/dl) ER	K (mEq/dl) ER	Urea (mg/dl) Discharge	Cr (mg/dl) Discharge	Na (mEq/dl) Discharge	K (mEq/dl) Discharge	pH	pCO_2 (mmHg)	Bicarbonate (mMol/l)	Base Excess (mMOl/l)
Case 1	17.2	47.5	88	0.91	118	2.81	28	0.65	140	3.1	7.61	46	46.2	24.8
Case 2	16.8	49	127	2.86	128	2	39	1.43	140	5.4	7.47[a]	45.90[a]	32.7[a]	7.7[a]
Case 3	18.5	53.8	94	1.43	131	4.36	56	1.05	134	3.91	7.48	33	24.3	

Cr, creatinine; ER, emergency room; HB, hemoglobin; Hct, hematocrit; K, potassium; Na, sodium.
[a] Venous blood gases.

CF patients sweat excessively, this reabsorption fails to occur, leading to excretion of large amounts of sodium chloride and decreased blood levels of these ions.[3] This induces secondary hyperaldosteronism with metabolic alkalosis due to increased bicarbonate reabsorption and low blood potassium caused by potassium secretion from the collecting tubule.[4] Moreover, the loss of extracellular fluid lowers the glomerular filtration rate and bicarbonate filtration.[1] This condition is described as "pseudo-Bartter" syndrome, and is characterized by metabolic alkalosis, hyponatremia with hypochloremia, with no renal tubule involvement.[1] It is more common in pediatric patients, and occurs only exceptionally in adolescents and adults; it is sometimes the presenting feature of a CF diagnosis.[5] In view of the potential seriousness of these ion alterations, including the risk of arrhythmias with cardiac arrest, muscle paralysis with involvement of the respiratory muscles or laryngospasm, tetany, and metabolic alkalosis convulsions,[1] it is important that certain recommendations are followed. Treatment is based on appropriate fluid replacement and correction of the electrolyte deficit, with high sodium, chloride and potassium supplements to correct alkalosis; appropriate prevention, with the addition of salt to the diet (1–4 g/day, according to patient age); avoidance of situations leading to excess sweating; and the administration of appropriate supplements during strenuous physical activity.[2]

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Recurrent Respiratory Infections in a Patient With Chronic Diarrhea[☆]

Infecciones respiratorias de repetición en paciente con diarrea crónica

To the Editor:

Good's syndrome (GS) is a primary immunodeficiency characterized by thymoma and humoral immunodeficiency. It is the most unusual form of the parathymic syndrome, after far behind *myasthenia gravis* or pure red cell aplasia.[1] The most common forms of clinical presentation are recurrent infections, hematological changes, and chronic diarrhea.[1,2]

We report the case of a 76-year-old man with a history of arterial hypertension, a former smoker of 20 pack-years, with chronic diarrhea which yielded *Campylobacter coli* on culture. He was referred to the respiratory medicine department for repeated respiratory infections. Forced spirometry showed mild obstruction: FEV_1/FVC 0.67, FEV_1 2.1 l (87%), FVC 3.17 l (98%). Skin prick tests for airborne allergens were negative. Clinical laboratory tests showed hemoglobin 11.7 g/dl with normal corpuscular volume, with no impact on platelet levels, and markedly reduced CD19 lymphocytes, with a CD4/CD8 ratio of 1.04. Immunoglobulin levels were low: IgA <5 mg/dl, IgG <74 mg/dl, IgM <5.3 mg/dl. Methicillin-resistant *Staphylococcus aureus* was isolated from repeated sputum cultures. Computed tomography (CT) of the paranasal sinuses showed occupation of the maxillary sinuses, while the chest CT revealed mild bronchiectasis in the middle lobe, lingula and both lower lobes, and a solid multilobulated mass in the anterior mediastinum suggestive of thymoma. VATS was performed, confirming the histological diagnosis of polygonal cell cortical thymoma. With these findings, the patient was determined to have GS, and immunoglobulin replacement therapy was started. He showed good progress and the number of infections fell.

GS mostly appears in patients in their 30s or 40s (unlike our patient), and affects men and women equally.[1] This immunodeficiency is caused by an antibody deficit, and is currently classified as a different entity to common variable immunodeficiency (CVID).[1] It accounts for 2% of cases of primary antibody deficiency treated with immunoglobulin replacement therapy.[3]

The most common clinical manifestation is recurrent respiratory infection, and the major pathogens are *Haemophilus influenzae* and *Pseudomonas* spp. The chronic diarrhea presented by 50% of patients appears to have an autoimmune basis, and the isolation of pathogenic agents is anecdotal.[3]

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Diagnosis is based on the clinical picture and immune response studies. The latter are characterized by reduced B cells and hypogammaglobulinemia with reduced CD4 T cells and an inversed CD4/CD8 ratio.[1]

Treatment of choice is regular intravenous administration of gammaglobulins to treat the humoral immunity, providing clinical benefit in most cases.[2] Resection of the thymoma is also indicated, to prevent the potential risk of locally invasive growth and metastatic dissemination, although the procedure does not seem to improve immunodeficiency.[2]

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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 l/65%) with basal oxygen saturation (SO_2) 96%. In view of this finding, itraconazole 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 SO_2: 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.[1,2,4] 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 spectronomy (MALDI-TOF).[5]

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.

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