

Tabla 1Tabla de pruebas identificativas para los 4 aislamientos de *Candida*

Aislamientos	API 32C®	API 32C® (repetición)	VITEK® MS MALDI-TOF	ITS secuenciación
Primero	<i>C. sake</i>	<i>C. parapsilosis</i>	<i>C. parapsilosis</i>	<i>C. parapsilosis</i>
Segundo	<i>C. sake</i>	<i>C. parapsilosis</i>	<i>C. parapsilosis</i>	<i>C. parapsilosis</i>
Tercero	<i>C. sake</i>	<i>C. sake</i>	<i>Pichia farinosa</i>	<i>C. metapsilosis</i>
Cuarto	<i>C. sake</i>	<i>C. sake</i>	<i>C. haemulonii</i>	<i>C. haemulonii</i>

compararon con aquellas depositadas en la base de datos del GenBank mediante BLAST.

Adicionalmente, se estudió la capacidad del sistema VITEK® MS™ -basado en MALDI-TOF®- (bioMérieux)⁷ para la identificación de los 4 aislamientos, siguiendo la metodología recomendada por el fabricante.

La identificación molecular demostró que los aislamientos correspondían a *Candida parapsilosis* (n=2), *Candida metapsilosis* (n=1) y *Candida haemulonii* (n=1). El sistema VITEK® MS identificó correctamente las 2 cepas de *C. parapsilosis* y la de *C. haemulonii*, pero identificó erróneamente *C. metapsilosis* como *Pichia farinosa*. Este sistema no incluye las especies del grupo «*psilosis*» en la base de datos de MS-ID v1 (bioMérieux).

Nuestros resultados muestran que la identificación de *C. sake* con API 32C® debería confirmarse con un método molecular. La literatura refiere hasta un 86% de eficacia para especies de *Candida* frecuentes y de un 85% en las infrecuentes⁴, pero el auténtico problema de esta prueba es la variabilidad entre observadores y la necesidad de una cierta experiencia a la hora de interpretar las galerías.

Es conveniente evaluar el sistema MALDI-TOF® VITEK® MS para comprobar su posible utilidad como método rápido alternativo, de la misma manera que sería necesario incrementar su base de datos. Algunos trabajos presentan excelentes resultados respecto a la identificación de levaduras por el sistema MALDI-TOF® con rangos de eficacia del 95 al 100%^{1,3,5}.

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Paracoccidioidomycosis and HIV coinfection presenting severe disease even with CD4⁺ cells in the normal range

Paracoccidioidomycosis grave en un paciente coinfectado por el VIH con valores normales de linfocitos CD4⁺

Dear Editors:

Paracoccidioidomycosis is a systemic mycosis caused by *Paracoccidioides brasiliensis*, and it's endemic in many countries of South America.¹⁰ It has been uncommonly reported in AIDS patients until last few years, when it has been more frequently described.^{3,8,9,11} Recently, Morejón et al. showed that paracoccidioidomycosis and HIV-infected patients (n=53) presented CD4⁺ count <200 cells/μL in 83.7%, lymphadenomegaly in 80%, cutaneous lesions in 66.7% and hepatomegaly in 64.2% of studied cases. Moreover, 80% of patients presented a HIV viral load of >30,000 copies/mL of plasma.⁵ These data suggest that, severe paracoccidioidomycosis in HIV/aids patients is an related to pronounced immunosuppression.

However, coinfecting patient can present severe and disseminated disease even with CD4⁺ cell count in the normal range, as observed in this case report.

We report the case of a 21-year-old male patient from an urban area of São Paulo state, Brazil, who was referred with a four-month history of fever, weight loss, lymphadenomegaly and cutaneous lesions. He had been an inmate in a state prison, and had inhaled illicit drugs but denied previous medical problems. His clinical presentation was remarkable due to multiple erythematous, infiltrated and ulcerated skin lesions on his face, trunk and limbs (Figs. 1 and 2). He also presented cervical, axillar and inguinal lymphadenomegaly plus hepato-splenomegaly. Biopsy samples from skin lesions and cervical lymph nodes showed diagnostic structures of *P. brasiliensis* (Fig. 3). Nested-PCR and culture in Mycosel® agar from skin tissue sample were also positive for *P. brasiliensis*. Chest X-ray and computerized tomography (CT) showed normal to discrete interstitial pulmonary infiltration. Abdominal CT revealed diffuse augmentation of liver and spleen and retroperitoneal lymphadenomegaly. A suspected HIV infection was confirmed by

positive serology (ELISA and Western-blot tests). Additional laboratory studies showed $CD4^+ = 875$ cells/ μ L; viral load $>30,000$ copies and double immunodiffusion test for anti-*P. brasiliensis* antigen with positive results (IDD = 1/16). Hematological study was normal with an erythrocyte sedimentation rate of 42 mm/h. Testing of HBV, HCV and HTLV-I was negative.



Fig. 1. Paracoccidioidomycosis: papule, nodule, infiltrated and ulcerative lesion on frontal region.



Fig. 2. Paracoccidioidomycosis: multiple infiltrated and ulcerated lesions with satellite lesions on the back.

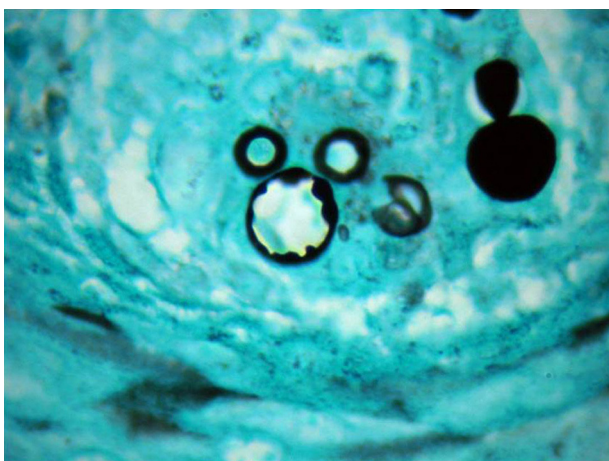


Fig. 3. Paracoccidioidomycosis: typical aspect of *P. brasiliensis* on the dermis (silver methenamine $\times 1000$).

Treatment was performed with amphotericin B, total dose = 1960 mg with clinical cure, followed by trimethoprim-sulfamethoxazole 320 + 1600 mg/daily. HAART therapy was not prescribed at that moment and patient did not return for the follow-up.

This reported case is very useful to demonstrate some aspects related to paracoccidioidomycosis and HIV coinfection: although the expected clinical profile of paracoccidioidomycosis in patients bearing the age of this particular case is to present lymphadenomegaly and skin lesion, the exuberance of this clinical presentation, particularly the magnitude of lymphonodes augmentation and number & aspect of skin lesions draw our attention to possible sub-jacent immunosuppression, in this case an unnoted HIV-infection. It has also been suggested that urban and young patients with severe paracoccidioidomycosis might be routinely screened for HIV infection. Also important is the fact that patients with paracoccidioidomycosis and HIV despite clinical severity can present discrete pulmonary involvement or no detectable involvement at all and, that amphotericin B deoxycholate is still the first choice in severe paracoccidioidomycosis despite its side effects.^{1,2,4,7}

As the HIV infection is spreading to the interior of many countries in South and Central America, the possibility of HIV associated to endemic diseases prevalent in such countries must be considered in our clinical reasoning.⁶

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