Primary cutaneous histoplasmosis in an immunocompromised patient with long-standing rheumatoid arthritis

MICHELLE LARISSA ZINIS 1*, HENRIQUE LUIZ STAUB 2

1PhD, Dermatologist, Preceptor at Pontifícia Universidade Católica do Rio Grande do Sul (PUC-RS), Porto Alegre, RS, Brazil
2PhD, Rheumatologist, Preceptor at PUC-RS, Porto Alegre, RS, Brazil

Study conducted at Hospital São Lucas, Pontifícia Universidade Católica do Rio Grande do Sul (PUC-RS), Porto Alegre, RS, Brazil

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*Correspondence:
Address: Av. Soledade, 569-912, Torre Alfa
Porto Alegre, RS – Brazil
Postal code: 90470-340
dramizini@yahoo.com.br
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INTRODUCTION
Histoplasmosis is the most common endemic fungal infection in Latin America and North America, and an important cause of endemic mycoses worldwide. The fungus is often present in soil contaminated by feces of birds and bats.1

Cutaneous manifestations of histoplasmosis can be divided into primary and secondary lesions.2 Once the fungus undergoes early hematogenous spread, exclusive cutaneous presentation is rare even in immunosuppressed patients, and thus these patients usually present severe systemic disease.3

Primary cutaneous histoplasmosis is a rare form of presentation initially described in 1947,4 with only 23 cases reported in the literature to date.5

We report the case of an immunocompromised patient with rheumatoid arthritis (RA) who developed, unexpectedly, primary cutaneous histoplasmosis (PCH).

CASE REPORT
The patient, a 52-year-old man, presented clinical, laboratory and radiological findings compatible with RA since childhood. As the disease progressed, regardless of a variety of therapies employed, he developed major deformities of the hands, wrists, elbows, hips, knees and ankles. Recently, using low-dose prednisone and methotrexate, he suddenly presented nodular ulcerated lesions on the face as seen in Figures 1 and 2. The diagnostic hypotheses were cutaneous lymphoma, leishmaniasis, sporotrichosis, cryptococcosis, cutaneous tuberculosis, histoplasmosis and tertiary syphilis. Histological examination showed diffuse granulomatous infiltrate with the presence of intracytoplasmic yeast structures (Figure 3). Curettage was performed for culture of material from the necrotic-suppurative areas of ulcerated lesions. Histoplasma capsulatum was the fungus detected by direct mycological examination (Grocott staining). The chest radiograph was normal. Blood cell count was unremarkable. After 6 months of therapy using itraconazole 300 mg daily, the nodules on the skin of the face disappeared. The patient continued to be monitored after completion of treatment without any disease recurrence signals.

DISCUSSION
Histoplasmosis is a disease caused by the dimorphic fungus Histoplasma capsulatum, which is common in tropical and temperate countries. The usual form of presentation is the respiratory one, second to inhalation of airborne conidia.6

In our immunosuppressed RA patient, a disseminated form of histoplasmosis would be the expected presentation, but atypical PCH turned out to be the final diagnosis. Primary cutaneous histoplasmosis has been described in immunocompetent individuals, but reports of this form of presentation in immunocompromised are rare. Today, it is clear that the infection is the result of a complex interaction between pathogen and host.7
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The patient presented lesions with no regional lymphadenopathy and no evidence of systemic involvement. Also, *Histoplasma capsulatum* was isolated and identified. There were only a few descriptions of PCH in patients with autoimmune rheumatic diseases such as the current case. As previously reported, the use of itraconazole as therapy of choice yielded complete response.

In summary, we describe an unusual case of PCH in a patient with long-standing RA treated with steroids and methotrexate. We stress the need for histological and mycological examination of skin lesions suspected in such patients.

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References