

Paracoccidioidomycosis



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South American blastomycosis or paracoccidioidomycosis, ulcers lips. Paracoccidioides brasiliensis. Photo Cochabamba



Paracoccidioidomycosis is caused by infection with Paracoccidioides brasiliensis. The yeasts have a typical steering wheel aspect caused by budding. Copyright ITM

Paracoccidioidomycosis or South American blastomycosis occurs in discrete foci in Latin America. It is thought that the fungus (*Paracoccidioides brasiliensis*) exists in the soil as a mold, with infections happening through the inhalation of conidia (spores). These spores convert into yeasts in the lungs and are then thought to spread to other sites haematogeneously and via the lymphatics. Most primary infections are self-limiting. The organism has the ability to remain dormant in the human host for long periods. It can cause clinical disease at a later time if host defense would become impaired (e.g. depression of cell mediated immune responses). Overt infection results in a progressive mycosis with lesions of the skin, mucous membranes (especially mouth, lips and nose) and internal organs. Long asymptomatic periods enable patients to travel far from endemic areas before developing clinical problems. Lesions in the face, naso- and oropharynx resemble espundia (mucocutaneous leishmaniasis), lupus vulgaris (skin tuberculosis) and syphilis. Papules will ulcerate and enlarge both peripherally and deeper into the subcutaneous tissue. A hard hyperkeratotic border may surround a deep ulcerating crater on the skin. Extensive coalescent ulcerations may eventually result in the



destruction of the uvula, epiglottis and vocal cords. Eating and drinking become difficult and painful. Lymphatic infections lead to painless enlargement of cervical, supraclavicular and/or axillary nodes. Draining sinuses may form. Visceral lesions in liver, spleen and lymph nodes can lead to abdominal pain, hepatosplenomegaly and low-grade fever. Pulmonary involvement occasionally leads to mild symptoms, including cough and sputum production, although the radiograph of the chest can show dramatic involvement of the lungs. Infections tend to be very chronic, but not fatal. Diagnosis relies on demonstration of the yeasts in tissue/secretions. The yeasts are usually abundantly present in the bases and edges of slowly expanding ulcer. In tissue specimens, the yeasts are rather large (15 μ m) with multiple buds, thereby resembling the steering wheel of a boat. Gomori staining of biopsies is useful. Serology by immunodiffusion is positive in \pm 98% of cases. Azoles such as itraconazole are usually very effective as treatment. Amphotericin B can be used if there is no success with oral itraconazole or in case of severe infection. Sulfonamides can be used, but have only moderate activity and should be administered for very long periods.

LAST UPDATED BY ADMIN ON JULY 15TH, 2022