

## On the Purpose of a Case, Simulation Based on Gastric Adenocarcinoma for Opportunistic Infection: Histoplasmosis

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This is a 56-year-old female patient, from the rheumatoid arthritis-bearing area in treatment with Azathioprine plus prednisone; she consulted a gastroenterology department with symptoms of tiredness, weakness, fatigue, weight loss and dyspeptic symptoms.

An upper digestive endoscopy was performed with biopsy taking the presence of infiltrating ulcer lesion in gastric pylorus.

### **Biopsy results**

Negative for dysplasia, granulomatous gastritis. In view of the histopathological findings described, the patient's immunosuppression status and accentuation of dyspeptic symptoms, culture with bacteriology is requested with positive result for histoplasmosis. Immunohistochemistry reports: Presence of *Histoplasma capsulatum*. Negative for dysplasia. Taxial computed tomography with double contrast: reports a slight decrease in the caliber of the light at the pyloric canal level. It was decided to treat itraconazole at a dose of 400 mg/day for 12 weeks. After the treatment, endoscopic control is performed, disappearance of lesions that resembled a neoproliferative process.



**Figure 1**

Histoplasmosis is a granulomatous infection produced by the fungus *Histoplasma capsulatum*. The symptomatic form as classic histoplasmosis is presented as a spectrum that varies from progressive lung infection to acute fulminant infection. Histoplasmosis is acquired by the respiratory route through aerosolized conidia from fecal matter of bats and birds. Macrophage phagocytosis modulated by

T lymphocytes results in a localized granulomatous infection. This initial self-limited process produces minimal symptoms evidenced only by the development of an immune response manifested by a delayed hypersensitivity skin reaction and the production of specific precipitins as well as complement fixing antibodies as well as asymptomatic calcifications in spleen lungs and mediastinal lymph nodes. A small percentage of these episodes progress to progressive pulmonary infection or disseminated infection, usually associated with some state of immunosuppression. Histoplasmosis can be classified according to its topography (pulmonary, extrapulmonary, disseminated), for the duration of the infection (acute, subacute or chronic) and for the pattern of infection (primary versus reactivation). Symptomatic infection can occur as acute primary pulmonary infections, chronic cavitary pulmonary histoplasmosis as well as acute and chronic disseminated infections. Asymptomatic fungemia evidenced by the presence of splenic calcifications occurs during primary infection. This dissemination allows subsequent pulmonary and extrapulmonary reactivation. Reactivation after many years in extrapulmonary tissues, particularly the central nervous system, eyes, adrenal glands, mucocutaneous surfaces and other locations usually occurs in immunosuppressed and elderly. Progressive chronic disseminated histoplasmosis presents with symptoms of tiredness and fatigue with prominent mucous lesions. The mucous involvement is characterized by painful nodules in the tongue and gingiva as well as denuded superficial lesions or crater-shaped ulcers in the oral mucosa or nasal vestibule. In the most subacute forms the symptoms are more prominent, with characteristic involvement of the gastrointestinal tract manifested as abdominal diarrhea pain, masses in terminal and blind ileum, intestinal obstruction or perforation and occasionally massive bleeding. In a study on gastrointestinal histoplasmosis [1-3], 56 specimens from 52 patients were evaluated. Of these 43% presented gastrointestinal symptoms without associated pulmonary symptoms. 31% presented gastrointestinal lesions, 10% presented hepatic lesions and 43% both. The gastrointestinal macroscopic lesions included ulcerations (49% of patients), nodules (21%), bleeding (13%) obstructive masses (6%) and normal mucosa (23%). Microscopic findings included histiocytic lymph infiltration (83%), ulcerations (45%), lymphohistiocytosis nodules (25%) or minimal inflammatory reaction (15%) but only rarely well-defined granulomas (8.5%). It should be considered that the presence of granulomas is rare and if found, the presence of granulomatous gastritis can also be observed in Crohn's disease, tuberculosis, sarcoidosis, syphilis, berylliosis, foreign body reaction and even gastric adenocarcinoma [4].

In summary, we present the case of a patient with histoplasmosis at the gastric level simulating a neoproliferative lesion. The diagnosis of histoplasmosis should be considered in patients with gastrointestinal lesions from endemic areas and/or associated with immunosuppression states. In this case, the patient was a carrier of rheumatoid disease in chronic steroid treatment.

## Bibliography

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