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ACUTE CHOLESTATIC SYNDROME CAUSED BY PARACOCCIDIOIDOMYCOSIS IN AN IMMUNOSUPPRESSED PATIENT – A CASE REPORT

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Abstract: Paracoccidioidomycosis (PCM) is a systemic mycosis endemic in Latin America, caused by fungi of the genus *Paracoccidioides*. Its acute/subacute form can present with disseminated lymph node involvement and, more rarely, with extrinsic compression of the biliary tree, mimicking obstructive hepatobiliary diseases. We describe the case of a young HIV-positive patient with controlled viral load who developed acute cholestatic syndrome of initially unclear etiology. The diagnosis of PCM was confirmed only after surgical investigation and histopathological study. This report reinforces the importance of PCM as a differential diagnosis in immunosuppressed patients with obstructive jaundice, even in the absence of classic signs of mycosis.
Keywords: Paracoccidioidomycosis, cholestatic syndrome, HIV, systemic mycosis, lymphadenopathy.

INTRODUCTION

Acute cholestatic syndrome is characterized by obstruction of bile flow, with accumulation of bile salts, bilirubin, and other components in hepatocytes and blood. The most common causes are cholelithiasis (60%) and periampullary neoplasms (25%), while infections account for less than 5% of cases (SBH, 2021). In the general population, cholestatic syndrome is estimated to be present in up to 30% of patients with jaundice (Fitzgerald et al., 2016).

Paracoccidioidomycosis, in turn, is the main systemic mycosis in Latin America, with about 10 million infected individuals and an incidence of 1-3 cases per 100,000 inhabitants/year. The acute/subacute form, more common in young and immunosuppressed individuals, presents with large visceral lymphadenopathy and can lead to extrinsic compression of the bile duct (Martinez et al., 2021).

In HIV patients, infection with *Paracoccidioides spp.* can manifest more aggressively and atypically, being an important cause of morbidity and mortality in endemic regions (WHO, 2022).

LITERATURE REVIEW

PARACOCCIDIOIDOMYCOSIS (PCM)

PCM is a systemic mycosis caused by dimorphic fungi (Figure 1) of the genera *P. brasiliensis* and *P. lutzii*. Transmission occurs through inhalation of spores present in the soil, especially in rural areas. The infection is often subclinical, but it can progress to acute (juvenile) or chronic forms.

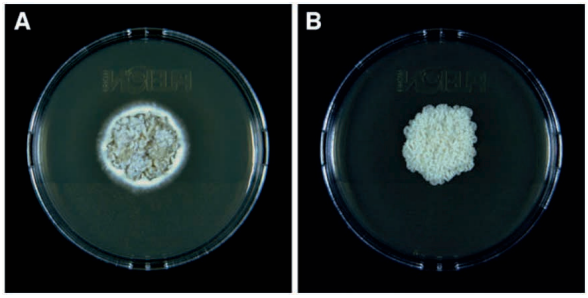


Figure I — Dimorphism of Paracoccidioides spp**.** A - Mycelium (environmental form); B - yeast (tissue form).

Source: Martinez et al., 2021.

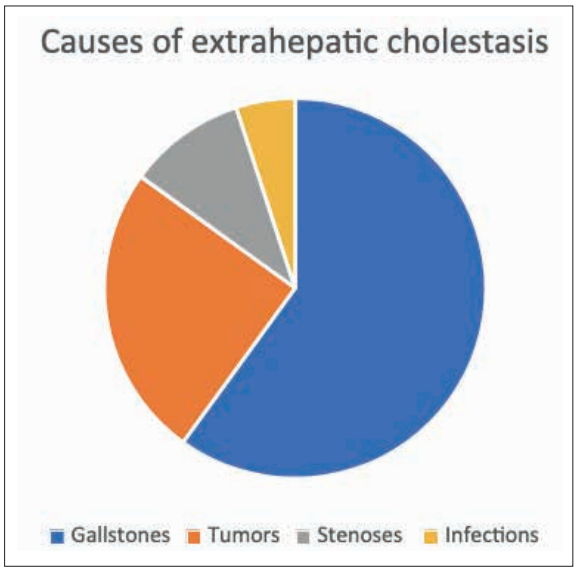
CHOLESTATIC SYNDROME

Cholestatic syndrome can be intrahepatic or extrahepatic (Table 1). Extrahepatic cholestasis, also called obstructive jaundice, is more common and is usually caused by stones or tumors (Graph 1).

Type	Main causes
Intrahepatic	Viral hepatitis, drugs, primary biliary cholangitis
Extrahepatic	Choledocholithiasis, neoplasms, stenosis, extrinsic compression

Table 1 - Classification of causes of cholestasis

Source: Prepared by the authors based on SBH, 2021.



Graph 1 — Causes of extrahepatic cholestasis.
Estimated distribution of the main etiologies: gallstones (60%), periampullary tumors (25%), stenoses (10%), and infections (5%).
Source: Adapted from SBH, 2021.

PCM AND HIV

Patients with HIV and CD4+ <200 cells/mm³ are at higher risk of disseminated PCM, with mixed manifestations and a high mortality rate (32%) (Almeida et al., 2017). Coexistence can alter the natural history of PCM, making it more acute and aggressive.

CASE REPORT

A 29-year-old male patient from Bragança Paulista, smoker (5 years-pack), HIV positive with effective antiretroviral treatment (undetectable viral load, CD4+ 350 cells/mm³), sought care for painless jaundice for 30 days, associated with choluria and fecal acholia. He denied fever, abdominal pain, or weight loss.

On physical examination, he presented with 4+/4+ mucocutaneous jaundice, with no other abnormalities. Laboratory tests revealed a cholestatic pattern (BT 8.7 mg/dL, BD 6.2 mg/dL, FA 480 U/L, GGT 620 U/L, CRP 12 mg/dL). Serology for viral hepatitis was negative. Abdominal ultrasound showed mild hepatic steatosis, cholelithiasis, bile duct dilation, and splenomegaly (15.3 cm). CT scan revealed a hyperdense gallbladder and lymphadenopathy in the intestinal loops (Figure 2; Figure 3; and Figure 4).

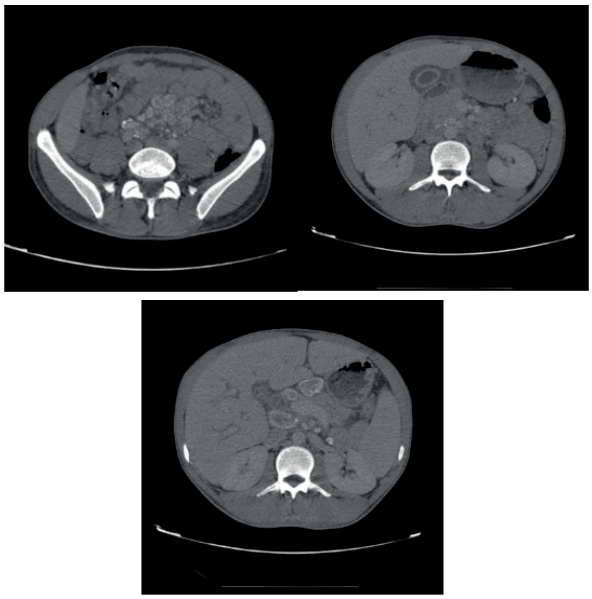


Figure 2 — Abdominal CT scan: large mesenteric lymphadenopathy. Images show hyperdense lymph nodes near the intestinal loops, suggesting an infiltrative/inflammatory process. **Source**: Authors' personal collection. **Figure 3 — Abdominal CT scan: hyperdense gallbladder.** Appearance consistent with cholelithiasis. **Source:** Authors' personal collection. **Figure 4 — Abdominal CT scan: dilation of the intrahepatic bile ducts.** Diffuse biliary dilation without clear evidence of intraluminal obstruction. **Source:** Authors' personal collection.

ERCP was performed, revealing diffuse biliary dilatation without intraluminal obstruction (Figure 5). ERCP allowed papillotomy and extraction of small stones, with an initial diagnostic hypothesis of Mirizzi syndrome. However, the patient remained jaundiced and had a low-grade fever, and laparoscopic cholecystectomy with intraoperative cholangiography, biliary drainage with a Kehr drain, and lymph node and liver biopsies were indicated. After surgery, the patient underwent X-ray and cholangiography via Kehr drain, with good contrast medium progression.

Histopathological examination showed granulomas and fungal structures compatible with Paracoccidioides spp., confirmed by immunohistochemistry.

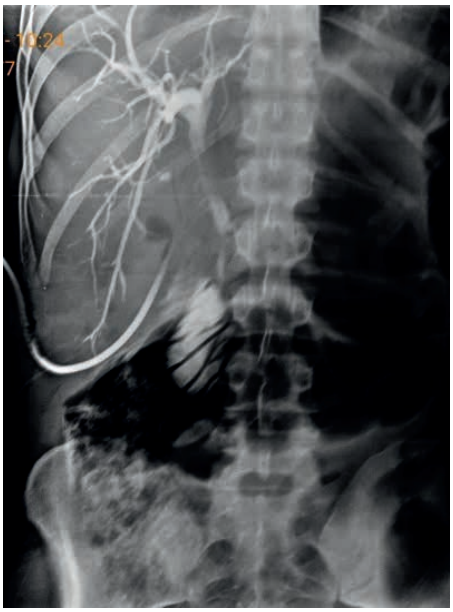


Figure 5 — Postoperative X-ray cholangiography via Kehr drain.

Source: Authors' personal collection.

The patient was discharged with a Kehr drain and showed partial improvement of jaundice, being referred for outpatient follow-up. Two months after discharge, the Kehr drain remained in place, with jaundice persisting, and a follow-up abdominal CT scan showed diffuse abdominal lymphadenopathy.

The patient required readmission due to cholestatic syndrome, and a new ERCP was performed. The examination revealed filling failure near the hepatic hilum due to extrinsic compression near the Kehr drain, with unsuccessful attempts to introduce a prosthesis to dilate the stenosis. Given this finding, the patient was evaluated by the infectious disease team, which indicated escalation of antifungal therapy to amphotericin B, with reevaluation scheduled after adequate treatment of paracoccidioidomycosis.

After two months of treatment with amphotericin and sulfamethoxazole-trimethoprim, the patient returned for a new ERCP attempt. He remained jaundiced and in use of the Kher drain. The procedure revealed distal choledochal stenosis with upstream dilation, spontaneous choledochoduodenal fistula, and non-stenosing extrinsic compression in the duodenal bulb. A plastic biliary stent was successfully placed through the choledochoduodenal fistula, and the Kher drain was subsequently removed. The patient was discharged from the hospital with scheduled outpatient follow-up and a plan to replace the stent in three months.

DISCUSSION

Although underreported, PCM is one of the leading causes of systemic mycosis in Brazil. In HIV-positive patients, the clinical manifestation tends to be more aggressive, with disseminated and lymph node forms (Neves et al., 2015). The case described here illustrates a rare and difficult-to-diagnose presentation: cholestasis caused by extrinsic compression of the bile duct by inflamed lymph nodes infected with *Paracoccidioides spp.*

Characteristic	Immunocompetent	HIV-positive
Clinical form	Chronic	Acute/Subacute
Involvement	Mucous membranes/lungs	Lymph nodes, liver, spleen
Mortality	< 10%	30–35%

Table 1 – Comparison between clinical forms of PCM in immunocompetent and HIV-positive patients

Source: Prepared by the authors based on Almeida et al., 2017; Neves et al., 2015.

The persistence of jaundice even after ERCP with stone extraction raised suspicion of another etiology, leading to the need for surgical intervention and diagnostic confirmation by histopathology. This scenario highlights the importance of MCP as a differential diagnosis of obstructive jaundice in endemic areas.

In addition, the abdominal lymphatic form of PCM can mimic lymphoma, abdominal tuberculosis, or neoplasms, requiring biopsies and specific tests, such as fungal staining and immunohistochemistry. Current literature recommends that, in endemic regions, all cases of atypical cholestasis be investigated for PCM, especially in immunosuppressed patients.

Antifungal treatment should be started as early as possible, preferably with itraconazole in mild/moderate cases or amphotericin B in severe or disseminated cases (MS, 2021). The therapeutic response is usually good when there is early diagnosis and adequate adherence.

CONCLUSION

Paracoccidioidomycosis should be included in the differential diagnosis of obstructive cholestasis, especially in HIV-positive patients in endemic regions. Extrinsic compression by lymphadenopathy is a rare but potentially serious complication. This report reinforces the importance of multidisciplinary approaches and histopathological confirmation in cases of jaundice of unclear etiology.

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