Philip A. Mackowiak, Section Editor

An HIV-Infected Man with an Upset Stomach

(See pages 935–6 for the Photo Quiz)

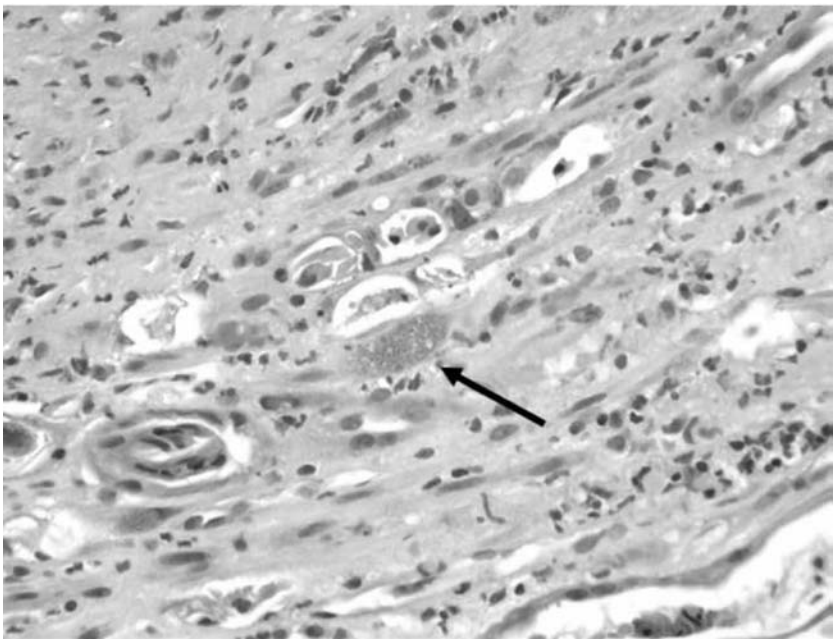

Figure 1. Antral mucosa with chronic active gastritis and *Toxoplasma gondii* cyst *(arrow)*

Diagnosis: gastrointestinal toxoplasmosis.

Immunohistochemical staining confirmed the presence of *Toxoplasma gondii* bradyzoites within tissue cysts with surrounding inflammatory infiltrates in the duodenum, gastric antrum, and colon (figures 1 and 2). The patient initiated treatment with tenofovir-emtricitabine and lopinavir-ritonavir as an inpatient but left the hospital against medical advice before receipt of biopsy results. Pyrimethamine and sulfadiazine were added to the patient's regimen as an outpatient once biopsy results were known, and he missed his appointment for MRI of the brain. Three weeks later, the patient presented with bilateral lower extremity weakness. MRI of the brain performed at that time showed nonenhancing foci of T2 prolongation in the left thalamus and adjacent to the internal capsule, with enhancing foci in the cerebellum and parietal lobe. These findings were considered to be consistent with a combination of treated and untreated toxoplasmosis. The patient continued to receive antiretroviral and antitoxoplasmosis therapy, with clinical and radiographic improvement in gastrointestinal and neurologic symptoms.

To our knowledge, antemortem diagnosis of gastrointestinal toxoplasmosis has only been reported 7 times in the literature. The patients in these cases had presentations similar to that of our patient, with nonspecific symptoms of nausea, abdominal pain, diarrhea, and anorexia. Approximately one-half of these patients had radiographic findings of gastric wall thickening, as did our patient. The diagnosis was confirmed in all cases by endoscopic examination and biopsy findings, which revealed acute or chronic inflammation with tachyzoites and/or bradyzoites. Despite the rarity of antemortem diagnosis, 1 autopsy series found trophozoites by immunohistochemical staining in the gastrointestinal tract of as many as 6.2% of patients with AIDS [1, 2].

As is the case for other forms of extracerebral toxoplasmosis, gastrointestinal toxoplasmosis most likely represents reactivation of *T. gondii* tissue cysts in immunocompromised individ-

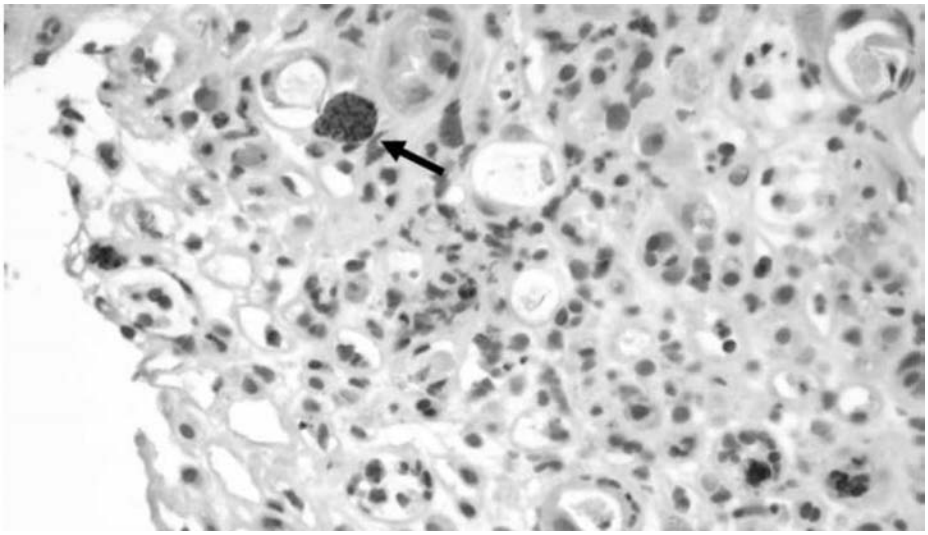

Figure 2. Immunohistochemical staining of a gastric antrum biopsy specimen for *Toxoplasma gondii*

uals. After ingestion of oocyte-contaminated soil or tissue cyst–contaminated meat, bradyzoites are released. These invade the intestinal epithelium and revert to tachyzoites. The tachyzoites subsequently multiply and spread to various tissues of the body, where they again become bradyzoites within tissue cysts [3]. $CD4^+$ cell depletion during HIV infection results in deterioration of cell-mediated immunity and the release of bradyzoites from their containment [4].

The differential diagnosis for gastritis in the HIV-infected population is broad and includes—in addition to the mundane causes of gastric inflammation in the non–HIV-infected population—cytomegalovirus infection, candidiasis, phlegmonous gastritis, cryptosporidiosis, *Mycobacterium avium* infection, and bartonellosis. Definitive diagnosis relies on expeditious endoscopic and pathologic evaluation. Findings of tissue cysts due to *Toxoplasma* species in biopsy specimens are insufficient to establish the diagnosis, because they can represent dormant, contained organisms. The discovery of tachyzoites or of active inflammation around cysts is required to clearly identify an ongoing pathologic process [2, 5, 6].

Data on the treatment of extracerebral toxoplasmosis are limited. The largest studies suggest that standard regimens, similar to those used to treat cerebral toxoplasmosis, should be modestly efficacious, with a response rate of ~64% and a relapse rate of ~19%. Because concomitant CNS involvement has been documented in 41% of all cases of extracerebral toxoplamosis, a high suspicion for occult brain lesions should be maintained, even in the absence of neurologic symptoms [7].

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