possibility of cancer, he was submitted to an upper gastrointestinal endoscopy, which suggested pangastritis. The gastric esrstrongiloidiasis diagnosis was further confirmed in the histopatologic analysis, which revealed the presence of \textit{S. stercoralis} eggs and worms.

\textbf{DISCUSSION:} Even though the stomach involvement is rare, patients whose gastric mucosa is infected are more likely to evolve to the severe forms. Gastroduodenal ulcers and perforations have been reported as possible complications. Stool examination is considered the gold standard diagnostic method, but the histopatologic analysis may be valuable when stool examination fails. The treatment does not depend on the infection’s site and ivermectin is the first line therapy.

\textbf{Key words:} Strongyloides stercoralis; Gastritis; Strongyloidiasis; Infection.
CASE PRESENTATION

A 57-year-old male patient presented to doctor’s appointment with a 6Kg weight loss complaint in the past three months. No other symptom was reported and the physical examination did not present any abnormality. A parasitological stool test was requested, since intestinal parasitic infections are still common in Brazil. Also, an upper gastrointestinal endoscopy (UGE) was performed in order to exclude gastric cancer, which was considered due to patient’s age. Analysis of the parasitological stool test revealed the presence of several *Strongyloides stercoralis*’ larvae, whereas the UGE showed moderate pangastritis. Therefore, a gastric biopsy was conducted (see Figure 1) and its histopathological analysis revealed several forms of *Strongyloides stercoralis* (eggs and larvae). Since this parasite is rarely found in the gastric mucosa of immunocompetent patients, an HIV-serological test was performed and yielded negative results. Similarly, the patient denied being previously submitted to any type of immunosuppressive therapy, such as the use of corticosteroids and immunobiological drugs. No other cause of immunosuppression could be identified through our screening; therefore, establishing the diagnosis of *Strongyloides stercoralis*’ gastritis in an immunocompetent patient (see Figure 2).

![Figure 1](image-url) Gastric mucosal: A & B (HE-100x) – Eggs, larvae and adults of *Strongyloides stercoralis*; C (HE-400x) – Eggs, larvae and adults of *S. stercoralis*.
DISCUSSION

Estrangiloidiasis, as previously mentioned, is an asymptomatic disease in most patients, including the patient described in this report. Some risk factors are associated to severe forms of infections, such as corticosteroids and immunobiological therapies, advanced age, HIV/AIDS and HTLV-1 infections, achlorhydria, H2 blockers use, malnutrition and cancer. The parasite usually infects the small intestine, persisting in it for years. Although it is rarely found in the stomach, patients with gastric involvement present higher risk of developing disseminated disease if they undergo any of the previously mentioned risk factors, especially immunosuppressive therapy[1,7,8]. The action of corticosteroid would be similar to the activating hormones of the ecdysis process (ecdysteroids), worsening autoinfection and leading to a possible fatal outcome[2].

The infectious cycle of Strongyloides sp can be divided in 3 parts – direct cycle, indirect cycle and autoinfection cycle[1,7,8]. The direct cycle is initiated when the rhabditiform larvae from stool matures to filariform larvae and penetrates the skin. Through blood circulation, it reaches the lungs and leads to the development of respiratory symptoms, such as coughing. Coughing, in its turn, allows the worms to reach the pharynx, where they are swallowed and achieve the gastrointestinal tract. In the small intestine, the larvae mature to female adults and produce eggs, which evolve to rhabditiform larvae. The indirect cycle is the development of living adults in the soil by maturation of eggs. Finally, autoinfection cycle occurs when filariform larvae (matured from rhabditiform) penetrates perianal skin and perpetuates the cycle. This last cycle is extremely relevant because allows the infection to perpetuate itself without any another species' participation[1,7,8]. The immune response against the parasite is mainly controlled by T-cells with a predominant TH2 response, that leads to the secretion of important cytokines, such as Interleukin-4 (IL-4), Interleukin-5 (IL-5), and Interleukin-10 (IL-10)[9].

A few patients with gastric Estrangiloidiasis may evolve to gastroduodenal ulcers and gastric perforation[1,10]. Seo et al[1] described a case of gastric involvement associated to a gastric adenocarcinoma, which emphasizes the importance of more accurate diagnostic tools in high risk patients for cancer[10]. Another presentation of gastric Estrangiloidiasis was seen as multiple small gastric nodules[11]. Gastrointestinal symptoms (in the few symptomatic patients) may include nausea, dyspepsia, weight loss, vomiting, constipation and abdominal pain[1,3,8,10].

The gold standard for the diagnosis of Strongyloides sp is based on organism identification on stool examination. Despite this, stool examination has a low predictive negative value – a single stool analysis fails in about 70% of the cases[1,3,6,9]. Another diagnostic tool is the histopathological analysis of the organs in which the infection is suspected, like we performed in this case. Histological findings typically includes gastritis with lymphocytic and eosinophilic infiltration in the lamina propria. Eggs, larvae and adult worms can also be found[1,4,5,9]. Serum eosinophilia can be detected in most of the cases, but may be absent, as it was observed in our case, especially in immunocompromised patients[1,9]. Many serological antibody tests (for example, the ELISA method) are currently available to be used in clinical practice. Regardless of the fact that they are quite sensitive, their specificity is less than adequate, since those antibodies may cross-react with other filarial parasites such as Schistosoma spp and Ascaris spp. Also, they are not capable of distinguishing acute and chronic infections[1,9]. Some alternative diagnostic tools (antigens and molecular diagnosis) have been proposed, but they are not available for clinical use in many hospitals[1,12,13]. The endoscopic findings are usually nonspecific and may show thickened gastric folds, mucosal erosions and ulcers[1,8,10].

Treatment is not altered based on the infection site of Strongyloides sp. and patients with gastric involvement should be treated the same way the ones with others forms of the disease. First line therapy is Ivermectin, 200ug/Kg, for two days[1]. Albendazole Thiabendazole and Mebendazole are considered alternative options[1,3,4,8].

CONCLUSIONS

Even though Strongyloides usually infects the small bowel, the parasite may affect the gastric mucosa in a few patients. Since this presentation presents higher chances of evolving to severe complications, such as gastroduodenal ulcers and gastric perforation, the disease should be considered in the differential diagnosis of any patient who presents inespecific stomachal symptoms, like vomiting and abdominal pain. The diagnosis can be established by stool examination, but some cases might require more sophisticated techniques, such as histopathological analysis.

Figure 2 Gastric mucosal: A e B – Moderate pangastritis at upper gastrointestinal endoscopy (UGE).
REFERENCES


