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Case Report

Coexistence of *Helicobacter pylori* and *Giardia duodenalis* causes severe iron deficiency anaemia in an adult male: a case report

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Abstract

Introduction: Iron deficiency anaemia (IDA), the most prevalent type of anaemia, is recognised as a significant global health concern that affects individuals of all ages.

Case Presentation: Herein, we present a case involving an adult male coinfected with *Helicobacter pylori* and *Giardia duodenalis*, which precipitated severe IDA.

Results: A 24-year-old male presented with symptoms including fatigue, dizziness, headache, abdominal pain, and diarrhoea persisting for four weeks. Thorough blood tests, including complete blood counts, blood film, and iron studies, conclusively established the presence of severe IDA. Furthermore, his faecal sample was collected and subjected to analysis of common bacterial and parasitic gastrointestinal infections. Examination of upper and lower gastrointestinal pathogens indicated that the severe IDA was most likely a result of coinfection with *H. pylori* and *G. duodenalis*. The patient received treatment involving antibiotics and iron replacement therapy, which resulted in an improvement in both his symptoms and laboratory results.

Conclusions: The present report provides crucial insights into the synergistic effect of concurrent *H. pylori* and *G. duodenalis* infections, highlighting their potential to induce severe IDA in infected patients.

Key words: Anaemia; Iron deficiency anaemia; *Giardia duodenalis*; *Helicobacter pylori*.

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Introduction

Iron deficiency anaemia (IDA), the most prevalent type of anaemia, constitutes a global health concern that impacts individuals of all ages, affecting approximately 30% of the world's population [1]. Decreased iron levels in the body perturb haemoglobin synthesis, leading to the ineffective production of red blood cells (RBCs). The diagnostic criteria for IDA can be listed as follows: (a) Haemoglobin levels are less than 12 g/dL for women and less than 13 g/dL for men; (b) serum ferritin is less than 30 ng/mL; (c) transferrin saturation is less than 20%; (d) Mean corpuscular volume (MCV) and mean corpuscular haemoglobin concentration (MCHC) are less than 80 fL and 32 g/dL, respectively; and (e) microcytic hypochromic RBCs evident in the morphology of peripheral blood smear [1]. Common causes of IDA include inadequate iron intake, gastrointestinal bleeding, malabsorption, menstruation in women, heightened iron requirements, or gastrointestinal infections [1,2].

Helicobacter pylori is a gram-negative gastrointestinal bacterium that colonises the gastric mucosa [2]. *H. pylori* is a pervasive bacterial infection in developing nations, afflicting about 50% of the global population [2]. *H. pylori* can give rise to various gastrointestinal complications, such as chronic gastritis, malabsorption, peptic ulcers, and gastric cancer [2]. *H. pylori* infection can facilitate the colonisation of other infections in the gastric mucosa by increasing stomach pH, among them being giardiasis [2,3]. Giardiasis is a parasitic infection caused by *Giardia duodenalis*, also known as *G. lamblia* or *G. intestinalis*, which commonly infects children, travellers, and individuals with impaired immune systems [4,5]. The primary mode of *G. duodenalis* transmission from one person to another is the faecal-oral route [4,5]. *G. duodenalis* stands as one of the most widespread intestinal parasitic infections globally, affecting around 250 million individuals yearly [4,5]. This parasite can cause malnutrition, abdominal pain, vomiting, flatulence, diarrhoea, anorexia, nausea, and weight loss [4,5]. While both *H. pylori* and *G. duodenalis* can individually contribute to IDA by affecting iron absorption in the gastrointestinal tract [2,3], limited

knowledge exists regarding the synergistic impacts of *H. pylori* and *G. lamblia* coinfection on the blood picture of these patients. In this context, we present a case of an adult male suffering from severe IDA due to *H. pylori* and *G. duodenalis* coinfection.

Case Presentation

A 24-year-old man presented to an internal medicine clinic with a chief complaint of headache, fatigue, dizziness, and diarrhoea persisting for four weeks. The physical examination revealed low blood pressure, abdominal pain, and skin pallor. The patient denied any history of gastrointestinal disorders such as Crohn's disease and celiac disease. Table 1 provides a summary of the patient's laboratory results. The analysis showed microcytic hypochromic anaemia with low levels of red blood cell (RBC) count (4.07 × 10^6/μL), haemoglobin (Hb) at 9.4 g/dL, haematocrit at 29.8%, mean cell volume (MCV) of 73.2 fL, and mean cell haemoglobin concentration (MCHC) at 31.1 g/dL. The white blood cell (WBC) count was slightly high at 12.3 × 10^3/μL with remarkable granulocytosis, and the platelet count was high at 554 × 10^3/μL. An elevated RBC distribution width (RDW) was also observed. Results for thalassemia and haemolysis, including Hb electrophoresis, bilirubin, and lactate dehydrogenase (LDH), were unremarkable. Iron studies indicated severe iron deficiency, with serum iron and ferritin levels measuring 23.60 μg/dL and 3.25 ng/mL, respectively. A faecal occult blood test was performed and showed a positive result. Based on the patient's laboratory findings and symptoms, the investigation primarily focused on screening for the most common bacterial and parasitic gastrointestinal infections. No abnormal bacteria or other organisms were found in the stool culture. The patient's faecal sample was subsequently analysed for *Helicobacter pylori, Giardia duodenalis, Entamoeba histolytica,* and *Cryptosporidium* utilising direct wet mount preparation, immune chromatographic rapid test, and enzyme-linked immunosorbent assay (ELISA). The analysis of upper and lower gastrointestinal pathogens confirmed the presence of *H. pylori* and *G. duodenalis*, while *E. histolytica* and *Cryptosporidium* were not detected. Therefore, the most likely cause of the patient's IDA was the coinfection of *H. pylori* and *G. duodenalis*. The treatment regimen included the administration of clarithromycin, metronidazole, omeprazole, and ferrous sulfate, which led to an amelioration of the patient's symptoms and positive changes in his laboratory results. During the four-month follow-up, the patient's haemoglobin and haematocrit levels were measured at 13.9 g/dL and 43.1%, respectively.

Table 1. The patient's laboratory results.

Complete Blood Counts	
RBCs (4.2 - 6.0 × 10^6/μL)	4.07
Haemoglobin (13.5-18.0 g/dL)	9.4
Haematocrit (40-54%)	29.8
MCV (80.0 – 100.0 fL)	73.2
MCH (26.0 – 32.0 pg)	23.1
MCHC (32.0 – 36.0 g/dL)	31.1
RDW (11.5 - 14.5%)	22.1
Platelets (150.0 – 450.0 × 10^3/*μL*)	554
MPV (7.0 – 12.0 fL)	7.6
WBCs (3.5 – 11.0 × 10^3/μL)	12.3
Granulocytes (2.0 - 7.5 × 10^3/μL)	9.4
Lymphocytes (1.0 - 3.0 × 10^3/μL)	2.5
Monocytes (0.1 - 1.0 × 10^3/μL)	0.4
Blood Film	
RBCs	Microcytic hypochromic anaemia with anisocytosis
WBCs	No abnormal or blast cell
Platelets	Thrombocytosis
Haemoglobin Electrophoresis	
Hb A (95 – 100%)	98.1
Hb A_2 (0.0 – 3.5%)	1.9
Hb F (0.0 – 2.0%)	0.0
Chemistry Studies	
Serum iron (60.0 – 180.0 μg/dL)	23.60
Serum ferritin (30.0 – 300.0 ng/mL)	3.25
LDH (140-280 U/L)	142
Total bilirubin (Up to 1.2 mg/dL)	0.5
Stool Analysis	
Occult blood	Positive
Stool culture	Negative
Giardia duodenalis (Wet mount preparation)	Present
Giardia duodenalis (ELISA)	Positive
Helicobacter pylori (Immunochromatography)	Positive
Entamoeba histolytica (ELISA)	Negative
Cryptosporidium (Immunochromatography)	Negative

RBCs: red blood cells; MCV: mean cell volume; MCH: mean cell haemoglobin; MCHC: mean cell haemoglobin concentration; RDW: RBC distribution width; MPV: mean platelet volume; WBCs: white blood cells; Hb: haemoglobin; LDH: lactate dehydrogenase; and ELISA: enzyme-linked immunosorbent assay.

Discussion

The case presented in this study is remarkably unique since the patient suffered from severe iron deficiency anaemia (IDA) due to *H. pylori* and *G. duodenalis* coinfection. The patient showed all the classic symptoms and laboratory findings of IDA, such as headache, fatigue, dizziness, skin pallor, microcytic hypochromic anaemia, anisocytosis, low haemoglobin, low mean corpuscular volume (MCV), high RBC distribution width (RDW), low serum iron, and low ferritin. Considering the potential of occult gastrointestinal bleeding to trigger the onset of IDA accompanied by reactive thrombocytosis [6], the present case prominently featured *H. pylori* infection as the causal agent in the formation of a peptic ulcer,

subsequently resulting in upper gastrointestinal bleeding and the development of IDA. There are several possible mechanisms for *H. pylori*-associated IDA, which are: (a) blood loss from gastroduodenal lesions and mucosal injury caused by *H. pylori* can be a direct cause of IDA; (b) *H. pylori* gastritis leads to impaired absorption of dietary iron as a result of decreased gastric acid secretion and gastric ascorbic acid concentration; (c) it has been proposed that the *H. pylori* bacterium perturbs hepcidin regulation, a central regulator for intestinal iron absorption and macrophages iron release; and (d) it has been suggested that *H. pylori* needs iron for growth and competes human cells for bioavailable iron, resulting in IDA [2,7,8]. However, several studies have concluded that these possible mechanisms for *H. pylori*-associated IDA cannot be extrapolated to all patients [1,3].

As mentioned above, *H. pylori* counteracts stomach acidity by producing urease enzyme, which generates ammonia from stomach lumen urea, leading to decreased levels of stomach acid. This creates an environment in which certain intestinal parasites have a better chance of surviving in the stomach. One of these parasites, which can cross the gastric acidity and colonise the duodenum, jejunum, ileum, and colon, is *G. duodenalis* [3,4]. Several reports from different parts of the world have investigated the presence of *G. duodenalis* infection among patients infected with *H. pylori*. For instance, the frequency of *G. duodenalis* in *H. pylori*-infected patients in Ethiopia, Egypt, Iran, Venezuela, Uganda, Turkey, and Mexico was 26.3%, 52.5%, 41.4%, 70.8%, 30.2%,45.8%, and 50.0%, respectively [4,9-14]. *G. duodenalis* can cause intestinal malabsorption due to impaired epithelial tight junctions, shortening of the epithelial brush border microvilli, and increased enterocyte apoptosis [3,5]. The presence of *H. pylori* and *G. duodenalis* together may exert a synergistic effect on the host by modulating its immune response, resulting in severe epithelial cell damage and malabsorption [3]. It is possible, therefore, that the presence of both pathogens together may suggest another pathogenic mechanism of IDA.

Conclusions

This report provides crucial insights into the synergistic effect of *H. pylori* and *G. duodenalis* when present together, causing severe IDA in infected patients. Thus, IDA patients with upper gastrointestinal symptoms should be investigated for both *H. pylori* and *G. duodenalis* to properly manage their clinical consequences. Further research with a larger sample size should be done to explore the prevalence and precise mechanism of this potential microbial interaction in IDA patients. Good hygiene and sanitation are indispensable for preventing the spread of these infections.

Authors' contributions

Conceptualization, A.A.; methodology, A.A, N.H. and K.J.; validation, A.A. and N.H.; formal analysis, A.A.; investigation, A.A, N.H. and K.J.; data curation, A.A.; writing—original draft preparation, A.A.; writing—review and editing, N.H. and K.J. All authors have read and agreed to the published version of the manuscript.

Data Availability Statement

Data available on request from the corresponding author.

Institutional Review Board Statement

The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of The Hashemite University on the April 10th, 2023 (No. 27/5/2022/2023).

Informed Consent

Written informed consent has been obtained from the patient to publish this report.

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