

Anemia Associated to *Helicobacter pylori* at Teaching Hospital Yalgado Ouédraogo (CHU-YO) of Ouagadougou in Burkina Faso

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Abstract

Introduction: Infection with *Helicobacter pylori* (*H. pylori*) is implicated in iron and vitamin B12 deficiencies without any etiologies. Our prospective study conducted at the Teaching Hospital Yalgado OUEDRAOGO at OUAGADOUGOU (BURKINA FASO) aimed to find a link between *H. pylori* infection and anemia in our context.

Patients and Methods: Were selected, patients with iron deficiency anemia or with resistance to oral iron treatment and vitamin B12 deficiency whose causes were not found. An upper digestive endoscopy was performed in all patients. A complete blood count was performed after *H. pylori* eradication therapy and a correction in iron or in vitamin B12.

Results: We selected 31 anemic patients of whom 29 (93.5%) were infected with *pylori H*. In the 21 patients treated for iron deficiency anemia and *H. pylori* infection, the mean haemoglobin level had risen from 8.72g/dL to 11.48g/dL after the treatment. In the eight patients with vitamin B12 deficiency, seven were infected with *pylori H*. The test of hemoglobin level carried out at the end of the treatment reported a hemoglobin level which varied from 11.3 g/dL to 13.1 g/dL against a mean hemoglobin level of 6.84 g/dL.

Conclusion: There should be a link between *H. pylori* and iron and vitamin B12 deficiencies whose etiologies are not found.

Keywords: Anemia; *Helicobacter pylori*; Iron Deficiency; Vitamin B12 Deficiency; Burkina Faso

Introduction

Helicobacter pylori (*H. pylori*) is a gram-negative bacterium with spiral form, found in the stomach of half of the world's population [1]. Indeed *H. pylori* infection can interfere with many biological processes, both within and outside the stomach, possibly influencing or determining many diseases outside the stomach [2,3]. Since 2012, according to French recommendations, three clinical extra gastroenterological situations justify the research and treatment of *H. pylori* infection: idiopathic thrombocytopenic purpura, iron deficiency anemia without any cause found or by resistance to oral treatment with iron and vitamin B12 deficiencies without any cause found [4]. The combination of iron deficiency anemia and *H. pylori* infection has been found in several studies, suggesting a causal relationship.

Anemia is the reduction in the mass of circulating hemoglobin. In practice this is the decrease in hemoglobin below haemogram reference levels without any change in plasma volume. The normal hemoglobin level in adults vary according to sex, age and gestational age; Indeed, pregnancy causes a hemodilution so that a woman who has 10.5g/dl in the third quarter of pregnancy is not anemic.

Several studies have reported a correction of iron deficiency anemia after the eradication of *H. pylori* [5] Recent studies have shown a link between *H. pylori* infection and vitamin B12 deficiency without any cause found [6]. In BURKINA FASO, Ilboudo D and et al. [7] noticed a frequency of *H. pylori* infection of 75% in children in 1998 and Bougouma A and et al. [8] in 2008 found it at 81.3%.

The studies in Africa on the infection to *H. pylori* especially described his gastroenterologic demonstrations without specifying his hematological demonstrations (the idiopathic thrombocytopenic purpura, iron deficiency anemia without recovered reason or by resistance to an oral treatment by iron and the vitamin B12 deficiency without recovered reason) from where the interest of our survey.

The purpose of this study was to investigate firstly the link between *H. pylori* infection and unexplained iron deficiency anemia or anemia resistance to oral iron treatment, and then secondly, the relationship between *H. pylori* infection and vitamin B12 deficiency anemia without any etiology found in our context.

Patients and Methods

Were selected, patients with iron deficiency anemia or with resistance to oral iron treatment and vitamin B12 deficiency whose causes were not found. An upper digestive endoscopy was performed in all patients. A complete blood count was performed after *H. pylori* eradication therapy and a correction in iron or in vitamin B12.

This was a prospective study which took place from April 1, 2016 to August 31, 2017 at Teaching Hospital Yalgado Ouedraogo (CHU-YO) in Ouagadougou, BURKINA FASO, in the departments of Clinical Hematology, Diagnostic and Interventional Endoscopy, Pathological Anatomy and Cytology.

Were included all anemic patients with iron deficiency, resistance to iron oral treatment, vitamin B12 deficiency whose cause was unknown and who had given their consent for the study. An initial blood count was performed in all the patients and a second blood count was made after *H. pylori* eradication therapy.

The determination of serum ferritin and serum iron were carried out in case of microcytic anemia: mean corpuscular volume (MCV) < 80 femtoliters (fL).

The anemia was classified as iron deficiency when serum iron and ferritin were low (to see below « operational definitions » for the criteria's).

The determination of vitamin B9, B12, of intrinsic antifactor antibody (to see below « operational definitions » for the criterias) (were performed in cases of macrocytic anaemia: MCV > 100 fL).

Each patient underwent an upper gastrointestinal endoscopy (each patient was ordered to take his last meal the night before, at the latest at 8 p.m, permitting to obtain at least 8 hours of gastric emptiness). The endoscopy explored the patient's upper gastrointestinal tract in axial view and in rear-view at the removal of the endoscope. Five (5) biopsies were performed, two (2) in the body, one (1) to the angulus and two (2) in the antrum. The samples were sent with three (3) different tubes (corresponding to the three biopsy sites) in the anatomy and pathological cytology laboratory of the Teaching Hospital Yalgado OUEDRAOGO (CHU-YO).

Research of *H. pylori* in faeces was made at least 30 days after an eradication treatment of the bacterium. The Table 2 precise the sequential treatment that had been prescribed to the patients. Were excluded:

- Patients with a known cause of anemia (fibroids, menorrhagia ...)
- Pregnant and lactating women;
- Those infected with human immune deficiency virus (HIV);
- Sickle cell patients;
- Those who have taken antibiotics in the past four weeks;
- And those who benefited from proton pump inhibitors (PPIs) in the last two weeks before the upper endoscopy.

We proceeded to the collection of data using a survey form indicating the patient's marital status, his socio-demographic characteristics, his history, the reason for consulting, the biological characteristics of anemia, the results of upper gastrointestinal endoscopy, the histological biopsies, the blood counts, the research of *H. pylori* in the faeces after eradication treatment of *Helicobacter pylori*.

- Data were collected and analyzed on a microcomputer using Epi Info 7.
- Statistical comparisons were performed using the chi2 test with a significance level of $p < 0,05$.

Operational definitions:

The diagnostic criteria for anemia were those of WHO, that is to say ahemoglobin level:

- < 13g/dL in men,
- < and 12g/dL in non-pregnant women.

Anemia was considered as moderate when hemoglobin level was ≥ 7 g/dL and severe when it was < 7g/dL.

Serum iron was considered low when it was < 12 μ mol/L.

Serum ferritin was interpreted low in women of childbearing age for a rate < 20 μ g/L, and in postmenopausal women and men for a rate < 30 μ g/L.

The withdrawal for the dosage of the B12 vitamin, serum and erythrocytic B9 and the intrinsic anti-factor antibody had been appropriated in Burkina and had been sent to the CERBA laboratory in France that had done the analyses.

Vitamin B12 deficiency had been defined by a rate of serum vitamin B12 < 140 picomoles/liter (pmol/L).

The value considered as normal for serum folate was between 10.4 and 42.4 nmol/L or between 4 and 18 mg/L; the value considered as normal for erythrocytic folate was set at 340 nmol/L or at 150 mg/L.

The titre of Ac intrinsic anti-factor was found positive for a rate > 1.54 AU/mL (reagents used: Intrinsic Factor Ab, Beckman Coulter).

Results

We selected 31 anemic patients of whom 29 (93.5%) were infected with *H. pylori*. In the 21 patients treated for iron deficiency anemia and pylori H. infection, the mean hemoglobin level had risen from 8,72g/dL to 11.48g/dL after the treatment. In the eight patients with vitamin B12 deficiency, seven were infected with *H. pylori*. The test of hemoglobin level carried out at the end of the treatment reported a hemoglobin level which varied from 11.3 g/dL to 13.1 g/dL against a mean hemoglobin level of 6.84 g/dL.

We collected a total of 39 patients during the study period, that is an annual recruitment rate of 27.5 cases of anemic patients with iron or vitamin B12 deficiencies with unknown etiologies.

Patients with iron deficiency anemia and infected with *H. pylori*. Thirty-one (31) patients had iron deficiency anemia of whom 29/31 (93.50%) were positive for *H. pylori* after the histological study of biopsies parts.

The mean age of patients with anemia due to iron deficiency and infected with *H. pylori* were 34,09 +/- 13,94 years with extremes of 7 and 67 years. The majority of patients 22/29 or 75.86% were found between 20 and 49 years. Women predominated with a number of 23/29 or 79.31% and men's number 6/29 (20.69%). The sex ratio was 0.26. The distribution of patients with iron deficiency anemia and *H. pylori* infection by age and sex were inhomogeneous: from 0 to 19 years and 40 to 49 years we had only female patients, and from 60 to 69 years we found only men; Women predominated in the age group of 20 and 29 years (9 women and 4 men).

Asthenia (19 cases / 29) in anemic patients with iron deficiency and infected with *H. pylori* constituted the main sign encountered with 26.03%. In all, 3 patients had no signs and the discovery was fortuitous. A patient could have more than one sign. (Table 1) shows the characteristics of the distribution of anemic patients with iron deficiency and infected with *H. pylori* according to clinical symptoms.

Signs	Number of cases (n) n = 29	Percentage (%)
Asthenia	19	65,5
Epigastralgia	15	51,7
Dizziness	11	37,9
Dyspnea	10	34,5
Regurgitations	5	17,2
Constipation	3	10,3
Conjunctival pallor	3	10,3
Abdominal pain	2	6,9
Diarrhea	2	6,9
Belching	1	3,4
Precordialgia	1	3,4
Dyspepsia	1	3,4

Table 1: Distribution of anemic patients with iron deficiency and infected with *H. pylori* according to symptoms

Anemia in patients with iron deficiency anemia and infected with *H. pylori* was moderate with 25 cases / 29 (86.2 %) and severe in 4 cases / 29 (13.8 %). The mean hemoglobin level was 8,72 g/dL for both sexes. It was 9.06 g/dL for females and 7.41 g/dL for males. The average MCV in both sexes was 65.37 femtoliters (fL). It was 65.38 fL for females and 65.33 fL for males

The mean serum iron of anemic patients with iron deficiency and infected with *H. pylori* was 9.78 µmol/L with maximum extreme value of 10.57 µmol/L. The mean serum ferritin of anemic patients with iron deficiency and infected with pylori H. was 14.55 µg/L. It averaged 8.29 µg/L for women and 19.56 µg/L for men. Thus 29 patients were infected with *H. pylori* and showed an iron deficiency.

The esophageal endoscopy of anemic patients with iron deficiency and infected with *H. pylori* was abnormal in 15 cases / 29 (51.7 %). Pathologies were: hiatal hernia in 12 cases (41.4 %) and peptic esophagitis grade I in 15 cases (51.7 %). Gastric endoscopy revealed 15 diseases/29: gastropathy with 12 cases / 29, mucosal pallor with one case/29, antral ulcer with one case/29 and one case/29 of duodenal gastric bile reflux. Gastropathy was erythematous in seven cases/29 and purpuric in two cases/29. Atrophic, erosive and nodular gastropathies, were present with one case each. Duodenal endoscopy diagnosed one case of sessile polyp of the bulb and one case of bulbar scar ulcer.

The histology of gastric biopsies in anemic patients with iron deficiency and infected with *H. pylori* according to the Sydney system was characterized by chronic inflammation (100%), atrophy (100%) and the presence of *H. pylori* (100%) in all patients. *H. pylori* was located at the level of the antrum in 29 cases (100%), of the angulus in 25 cases (86.2 %) and the body in 17 cases (58.6 %). In 24 patients/29 or 82.7 % of cases, *H. pylori* was located across the stomach.

Patients with anemia and vitamin B12 deficiency and infected with *H. pylori*.

The mean age of patients with anemia due to vitamin B12 deficiency and infected with *H. pylori* was 39,14 +/-13,77 years with extremes of 20 and 57 years. Women were 4 / 7 (57.1 %) and men were 3, that is a sex- ratio of 0.75.

Fifteen (15) symptoms were recorded in these patients with vitamin B12 deficiency anemia and infected with *H. pylori* (a patient could have more than one symptom), which were : asthenia (6 cases / 7), dizziness (3 cases / 7), dyspnea (1 case / 7), epigastric pains (1 case / 7), regurgitation (1 case / 7), dyspepsia (1 case / 7), paresthesia (1 case / 7) and pyresis (1 case / 7).

It was noted in these patients with anemia and vitamin B12 deficiency and infected with *H. pylori* five cases of moderate anemia and two cases of severe anemia. The mean hemoglobin level was 6.84 g/dL with a mean MCV of 97.64 femtoliters.

The levels of vitamin B12 in these patients with vitamin B12 deficiency anemia and infected with *H. pylori* was lower than normal (< 74 pmol/L) in all the patients and the level of vitamin B9 was normal in all the seven patients (that varied 25 to 2372 nmol/L).

The intrinsic anti- factor antibodies in these patients with vitamin B12 deficiency anemia and infected with *H. pylori* were found positive with a titre that ranged from 114,14 to 263,44 AU/ml (positive if >1,54 AU/mL).

Esophageal endoscopy in these patients with vitamin B12 deficiency anemia and infected with *H. pylori* was normal in two patients/7. Hiatal hernia was present with two cases/7 and peptic esophagitis in four cases/7.

Gastric endoscopy in these patients with vitamin B12 deficiency anemia and infected with *H. pylori* was normal in five cases/7. The pathology found was gastropathy (two cases/7), one atrophic gastritis/ 7cases and one erythematous gastropathy /7 cases.

Duodenal endoscopy in these patients with vitamin B12 deficiency anemia and infected with *H. pylori* was normal in all our seven patients.

The histological appearance of the stomach in these patients with vitamin B12 deficiency anemia and infected with *H. pylori* according to the Sydney system parameters was characterized by: the inflammation was found in all patients (slight in three cases/7 and moderate in four cases/7), activity in six (slight in four cases/7 and moderate in 2 cases/7), atrophy six/7 (slight in 5 cases/7 and moderate in one case/7), metaplasia in four/7 (slight in three cases/7 and moderate in one case/7), dysplasia in one patient/7 (moderate dysplasia); no lymphoid follicle was found. *H. pylori* was found at the antrum in all seven patients, at the angulus in four/7 patients and in the body in four/7 patients.

Therapeutic aspect

Treatment	Frequency (n)	Percentage (%)
IPP+amo+clari/10 days	02	6,45
IPP+amo+clari/7 days	01	3,22
IPP+amo+metro/10 days	13	41,93
IPP+amo+metro/7 days	04	12,90
Sequential Treatment	11	35,50
Total	31	100,00

IPP = lansoprazole; amo = amoxicillin; metro = metronidazole; clari = clarithromycin. Sequential treatment = bismuth Pylera® : capsules all in one (bismuth salt + tetracyclin + metronidazole) (3 × 4 capsules / day) + omeprazole 20 mg × 2 / day during 10 days.

Table 2: Distribution of anemic patients infected with *H. pylori* according to the treatment schedule

Results obtained after treatment

In total 17 patients / 31 or 54,8 % (of which 15 cases of iron deficiency anemia and 2 cases of vitamin B12 deficiency) could to benefit from a search of *H. pylori* in the faeces after treatment (check carried out at least one month after the end of the treatment). The search was negative in 14 cases / 17 and positive in three cases. For iron deficiency, the search of the antigen of the bacterium in the faeces after the treatment appeared negative in 12 cases and positive in three cases. As to vitamin B12 deficiency, the research of the bacteria antigen performed in two patients was negative. Treatment schedules and research of *H. pylori* in the faeces after treatment are outlined in (Table 3).

<i>H. pylori</i> in the faeces after treatment	Sequential (n)	Combination therapy(n)	Total
Negative	06	08	14
Positive	00	03	03
TOTAL	06	11	17

P=0.15H=*helicobacter*

Table 3: Treatment schedules and *H. pylori* research in faeces after treatment

The three patients positive for *H. pylori* in faeces, were all under combination therapy (lansoprazole, amoxicillin and metronidazole for 7 days in two patients, and lansoprazole, amoxicillin and metronidazole for 10 days for the 3rd patient).

We recovered 19 patients/31 or 61.3 % (16 iron deficiency anemia and three vitamin B12 deficiency) could achieve a control blood count at least 2 months after *H. pylori* eradication treatment. In 13 patients it was observed an increase in hemoglobin level. The mean hemoglobin level rose from 8,72 g/dL to 11.48 g/dL, and MCV average from 65.37 femtoliters to 72.60 femtoliters. In 3 patients hemoglobin level remained stable. Note that the majority of patients were under iron deficiency treatment.

The seven patients with vitamin B12 deficiency anemia and infected with *H. pylori* received all intramuscular injection of vitamin B12. Five had their *H. pylori* eradicated (three under sequential treatment and two under combination therapy). The hemogram performed in three patients deficient in vitamin B12, which were supplemented with vitamin showed hemoglobin levels which ranged from 11.3 g/dL to 13.1 g/dL against a mean initial hemoglobin level at 6.84 g/dL ; MCV ranged from 78.5 to 81.2 femtoliters with an mean initial MCV at 97.64 femtoliters.

Discussion

The small size of our sample (39 cases), the financial destitution of patients associated with the non functioning of universal health insurance and the fact that some were living outside Ouagadougou (in province) constituted an impediment to our study. Seven patients could not come to Ouagadougou when we called them to perform free endoscopy for example.

Patients with iron deficiency anemia and infected with *H. pylori*

The mean age of patients with iron deficiency anemia and infected with *H. pylori* was 34.1 ± 14.1 years with extremes of 7 and 67 years. Patients were found mainly between 20 and 29 years (44.8 %) and between 40 and 49 years (31.0 %). Annibale and et al. [10], Attaf and et al. [11] reported results similar to ours. Our results could be explained by the youthfulness of our population that lives mostly in unsanitary conditions exposing it to serious risks of infection with *H. pylori* 23 women with iron deficiency anemia and infected with *H. pylori* were 79.3 % and six men (20.7 %). The sex- ratio was 0.3. This result is similar to most studies [10-14]. This predominance of female infection could be justified by the fact that more women attend health centers than men, being perhaps more concerned about their health. In addition, women because of their status of mothers are prone to anemia during pregnancies and breast-feeding periods. This is also explained by the fact that young women (of childbearing age) represent a group particularly at risk because of their low stock in iron [15].

Iron deficiency anemia associated with *H. pylori* infection was moderate in 25 cases/29 (86.2 %) and severe in four cases (13.8 %). This could be explained by the treatment with iron and folic acid undergone by patients before their specialized consultation in hematology. The mean hemoglobin levels of anemic patients with iron deficiency and infected with *H. pylori* in our series was 9.1 g/dL for females and 7.4 g/dL for males. Our rate of middle hemoglobin of the patient with deficiency martial anemia and infected by *H. pylori* was lower at the men; it would explain itself in part by the systematic treatment made of iron and folate of the women in pregnancy in the BURKINA FASO. These rates were lower than those reported by Annibale and et al. [10] who had a mean hemoglobin level of 10.2 g/dL and Darvishi and et al. [14] who found 10 ± 0.68 g/dL in children. They are, however, higher than those reported by Gheibi and et al. [16] who found a mean hemoglobin level of 6.2 g/dL in children with severe anemia, and Kechida and et al. [17] who recorded a mean hemoglobin level of 7 g/dL. This variability in mean hemoglobin levels could be justified by the diversity of the nutritional status of the various regions and their food habits. The average of the mean corpuscular volume (MCV) of anemic patients with iron deficiency and infected with *H. pylori* was 65.38 femtoliters in women and 65.33 femtoliters in men. For Kibru and et al. [12] the mean MCV was significantly lower in patients infected with *H. pylori* compared to those who were not infected.

The mean rate of ferritin was 14.55 µg/L. It was 8.29 µg/L in women and 19.56 µg/L in men in our series. Most authors had found normal ferritin [14,18-20]. The difference with our results could be justified by our inclusion criteria; indeed, we have included only patients suffering from iron deficiency, that is to say, with a low serum ferritin. In summary, the decrease in the concentration of serum ferritin in patients infected with *H. pylori* could be induced by the absorption of ferritin in the stomach by *H. pylori* [20].

Gastric pathology of anemic patients with iron deficiency and infected with *H. pylori* was dominated by gastritis with 12 cases in our series. Erythematous gastropathy with seven cases, purpuric gastritis with two cases and nodular, erosive and atrophic gastritis with one case each. This predominance of erythematous gastropathy was found by many authors [7,8]. *H. pylori* infection in our patients could explain the predominance of erythematous gastritis resulting from inflammation. The histological aspects

of the stomach of patients with iron deficiency anemia and infected with *H. pylori* were characterized in our study by: chronic inflammation, atrophy and *H. pylori* infection were present in all the patients (100 %). Activity was present in 82.8 % of cases, metaplasia in 10.3 % of cases, dysplasia in 24.1 % of cases and lymphoid follicles in 24.1 % of cases. Inflammation was slight in 6.9 %, moderate in 55.2 % and severe in 37.9 %. Inflammation seemed to be more important in case of iron deficiency anemia associated with *H. pylori* and the atrophy was constant in African and non-Caucasian series. [7,11,13,21-24]. In our study chronic gastritis in anemic patients with iron deficiency and infected with *H. pylori* was located in 100% of cases at the level of the antrum, in 86.2 % at the level of the angulus and 58.6 % of cases at the level of the body. In 81.6 % it was located across the stomach. These results are similar to most studies [10,13]. Patients with pangastritis (rather inflammatory than atrophic) related to *H. pylori* and to unexplained iron deficiency anemia (without endoscopic lesions) have a higher gastric pH and a lower ascorbic acid level (plasma and gastric) compared to patients with unexplained iron deficiency anemia without *H. pylori* gastritis, or to patients with *H. pylori* antral gastritis without anemia. This could be explained by the fact that this inflammatory pangastritis is responsible for a decrease in gastric acid and a concentration of ascorbic acid resulting in a decrease in iron absorption. Ascorbic acid appears to be an important cofactor in the absorption of iron through two mechanisms: [25]

- firstly, by initiating the reduction of ferric iron into ferrous iron;
- and secondly by forming an absorbable molecular complex with ferric iron.

Anemic patients with vitamin B12 deficiency and infected with *H. pylori*

The mean age of anemic patients with vitamin B12 deficiency and infected with *H. pylori* in our series was 39.14 +/- 13.77 years with extremes of 20 and 57 years. In France, Loukili and et al. [6] found a mean age of 69 years in 49 patients suffering from Biermeranemia. Biermeranemia is a disease of the elderly, this could explain the difference with our results. In our series, anemic women with vitamin B12 deficiency and infected with *H. pylori* were four and men were three. In accordance with the literature female's predominance was found [6].

The mean hemoglobin level in anemic patients with vitamin B12 deficiency and infected with *H. pylori* in our series was 6,8 g/dL and the mean MCV was 97.6 femtoliters. This same observation was made by other African authors [24,26].

Our patients with vitamin B12 deficiency and infected with *H. pylori* had a serology positive for intrinsic anti-factor antibodies as found by some authors in France and in Africa [6,24,26].

Therapeutic aspects

Endoscopy of our anemic patients with vitamin B12 deficiency and infected with *H. pylori* was normal in five cases. The pathology encountered was gastropathy (two cases /7) including one atrophic gastritis and one erythematous gastropathy. A macroscopically normal endoscopy does not exclude a vitamin B12 deficiency, the atrophy of the fundus folds is not routinely found in case of cobalamin deficiency [27]. The destruction of parietal cells is not the only cause of gastric malabsorption of vitamin B12; a functional alteration of gastric parietal cells related to *H. pylori* brings about such high deficiencies; the nature of this alteration remains to be determined [28]. According to Maastricht V, achlorhydria (favored by chronic inflammation and gastric fundal atrophy) would be responsible for the malabsorption of vitamin B12. [29]

In 13 patients treated for iron deficiency anemia and *H. pylori* infection, the mean hemoglobin level rose from 8,7 g/dL to 11.5 g/dL and the mean MCV shifted from 65.97 femtoliters to 72 60 femtoliters. In three patients hemoglobin level remained stable. The search for the antigen of the bacterium in the faeces after treatment was negative in 12 cases and positive in 3 cases. The increase in hemoglobin level (and even its correction) after *H. pylori* eradication was observed by several authors [4,25,30-32]. Therefore, iron deficiency whose cause is not found may be related to the infection with *H. pylori* and its eradication can correct anemia [30-32].

Anemic patients with vitamin B12 deficiency and infected with *H. pylori* had all received vitamin B12 injection. Five patients had received eradication treatment three through sequential treatment and two through combination therapy). The hemogram performed at the end of vitamin B12 therapy reported hemoglobin levels which ranged from 11.3 g/dL to 13.1 g/dL against a mean hemoglobin level of 6.8 g/dL; the MCV ranged from 78.5 to 81.2 femtoliters; the search for the antigen of the bacterium was negative. We can deduce that vitamin B12 deficiency may be related to infection with *H. pylori* and its eradication can correct anemia.

Conclusion

There should be a link between *H. pylori* and iron and vitamin B12 deficiencies whose etiologies are not found.

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Competing Interests

The authors declare that they have no financial or personal relationship(s) which may have inappropriately influenced them in writing this paper.

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