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Case report

Recurrent anaemia in a patient with lymphocytic gastritis and vitamin B_{12} deficiency

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ABSTRACT

Lymphocytic gastritis is an idiopathic disease, characterized by intraepithelial infiltration of large numbers of T lymphocytes and often described in association with coeliac disease and *Helicobacter pylori* infection. Although usually associated with iron deficiency anaemia, there is no description on the association between lymphocytic gastritis and secondary vitamin B_{12} deficiency anaemia.

We describe a rare case of recurrent anaemia in a patient with lymphocytic gastritis reversed with vitamin B_{12} replacement.

Introduction

Lymphocytic gastritis is an idiopathic and rare form of chronic gastritis initially described by Haot in 1985 [1]. The clinical picture for this disease varies from moderate to severe dyspepsia, in addition to nausea, vomiting, anorexia, and weight loss [1,2]. Between 1% and 8% of patients with dyspeptic symptoms have lymphocytic gastritis [3,4]. Despite the classic description showing nodules, erosions, and large gastric folds, the appearance of the mucosa on endoscopy may be normal [2]. Studies have reported associations between lymphocytic gastritis and Crohn's disease, Ménétrier's disease, syphilis, *Helicobacter pylori* infection, and coeliac disease [2]. The ultimate diagnosis is anatomopathological and depends on the presence of 25 or more lymphocytes per 100 epithelial cells [1,2].

Vitamin B_{12} deficiency causes megaloblastic anaemia and neuropsychiatric symptoms [5]. Although pernicious anaemia due to atrophic gastritis is the main cause of vitamin B_{12} deficiency, it may also result from other disabsorptive changes in the gastrointestinal tract besides insufficient food intake [6]. For instance, vitamin B_{12} deficiency may be associated with several conditions, including pernicious anaemia, gastrectomy, chronic pancreatitis, coeliac disease, and even with the chronic use of proton pump inhibitors [7]. To our knowledge, anaemia caused by vitamin B_{12} deficiency has not been described in association with lymphocytic gastritis. The current study reports a case of recurrent anaemia in an individual with lymphocytic gastritis reversed with parenteral vitamin B_{12} replenishment.

Case report

A 69-year-old man was hospitalized with complaints of recurrent anaemia and dark stools. Eighteen months earlier, he complained of upper abdominal pain, weight loss, and dark stools. Since then, his medical condition was extensively investigated and he was admitted to the hospital numerous times due to anaemia, black stools, and marked asthenia. He was diabetic and had motor sequelae from two cerebral vascular accidents (CVA). He was taking metformin, enalapril, acetylsalicylic acid, and ferrous sulfate. He was a former smoker (40 pack-years) and an ex-alcoholic (100 g/day). At the time of his first hospitalization, the patient was in good general condition; however, he was very thin, had pallor ++/4+, and was well hydrated. Cardiopulmonary evaluation was normal; he showed no pain to palpation, no palpable masses or visceromegaly, and no signs of peritoneal irritation on abdominal examination. He showed decreased strength in the left

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upper and lower limbs and lower sensitivity and positive Babinski signs on the left side of the body.

Laboratory tests performed at his first hospitalization revealed the following results: haemoglobin: 6.9 g/dL; haematocrit: 21.4%; mean corpuscular volume (MCV): 83.0 μm^3; and mean corpuscular haemoglobin (MCH): 26.7 pg. At the time of his last hospitalization, 18 months later, laboratory tests revealed the following statistics: haemoglobin: 8.8 g/dL; haematocrit: 25.0%; MCV: 86.9 μm^3; MCH: 29.5 pg; reticulocytes: 0.5%; serum iron: 254 mg/dL; ferritin: 230 ng/mL; transferrin: 218 g/dL; transferrin saturation: 98 mg/dL; homocysteine: 7.10 μmol/L [5–12]; and vitamin B_{12}: 167 pg/mL (193–982). The temporal evolution hemoglobin concentrations and MCV, which was assessed every time the patient was treated at the emergency room as an in- or outpatient, is shown in Fig. 1A and B. Endomysial and transglutaminase antibodies were negative.

The consumptive syndrome associated with anaemia in elderly patients suggests possible neoplasia [8]. At the time of his first hospitalization, at the emergency ward, he received blood transfusion, but iron stores and vitamin B_{12} levels were not measured. An abdominal CT scan revealed a peripancreatic epigastric lesion and subcapsular collection. Thus, the patient underwent exploratory laparoscopy with drainage of the collection and biopsy of the lesion with subsequent diagnosis of *hemosuccus pancreaticus*; the hypothesis of intra-abdominal solid tumor was discarded [9]. The presence of structural changes and pancreatic pseudocysts closed the diagnosis of chronic pancreatitis that explained the pain and weight loss [10]. The patient had no clinical, laboratory, tomographic, or transoperative evidence of liver disease or portal hypertension. He was discharged with an iron supplement.

In a subsequent hospitalization, due to asthenia and dark stools, he was already taking ferrous sulfate. His colonoscopy results showed diverticular disease of the colon; mesenteric arteriography results were normal. Colonic diverticula are the main cause of rectal bleeding in elderly individuals [11]. Sometimes, when there is no active bleeding in the colon during colonoscopy, the diverticula are considered a probable cause of lower gastrointestinal bleeding [12]. However, after hospital discharge, despite the patient's adherence to ferrous sulfate, he continued to show a reduction in hemoglobin levels and symptomatic anaemia with no clinical evidence of gastrointestinal bleeding, which led the patient to seek emergency care repeatedly.

Due to recurrent hospital admissions for clinically manifested anaemia with no evidence of intestinal bleeding, the patient underwent upper digestive endoscopy (UDE) on several occasions (Fig. 2) for investigation of the possible cause of anaemia and to exclude the diagnosis of neoplasia. The thickened folds in the fundus and body of the stomach and the presence of elevated gastric lesions friable to the touch evidenced by UDE were suggestive of lymphocytic gastritis. In our hospital, *H. pylori* is investigated only through anatomopathological examination. The first one revealed chronic gastritis, foveolar hyperplasia compatible with lymphocytic gastritis, and negative *H. pylori* test. Immunohistochemistry was positive for Bcl-2, CD20, CD3, CD5, and CD45, and negative for Cyclin D1, thus ruling out the hypothesis of MALT lymphoma and confirming the diagnosis of lymphocytic gastritis.

Following blood transfusion before hospital discharge and chronic oral iron replacement, the patient returned to the hospital 5 times with anaemia symptoms at a gap of 1–3 months. During the last hospitalization, which was 18 months after the onset of the clinical condition, results of gastric biopsies were reviewed by gastroenterologists and pathologists, and the presence of *H. pylori* was identified. The patient was then treated with parenteral vitamin B_{12} replenishment and showed progressive improvement in hemoglobin levels during his hospital stay, which remain normal at the time of writing this report. One month after hospital discharge, *H. pylori* eradication treatment was carried out in the outpatient care unit with amoxicillin, clarithromycin, and omepra-

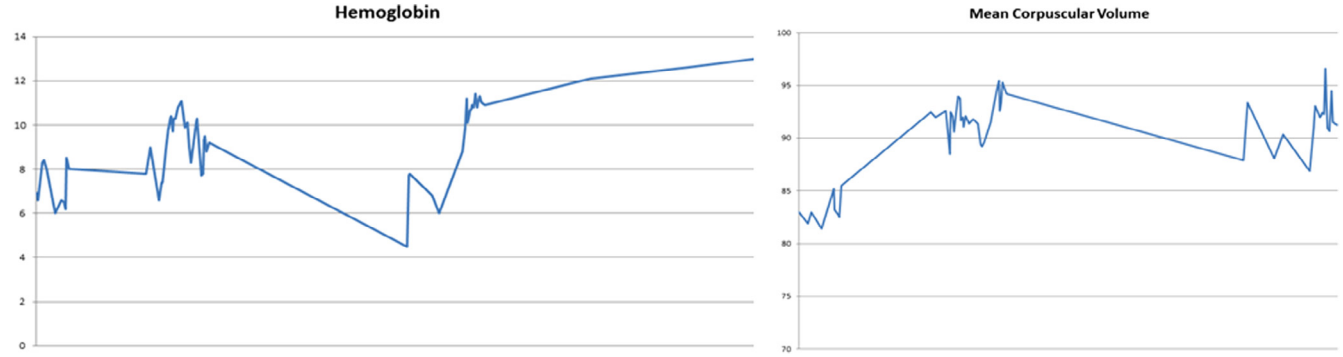

Fig. 1. Haemoglobin concentrations and MCV.

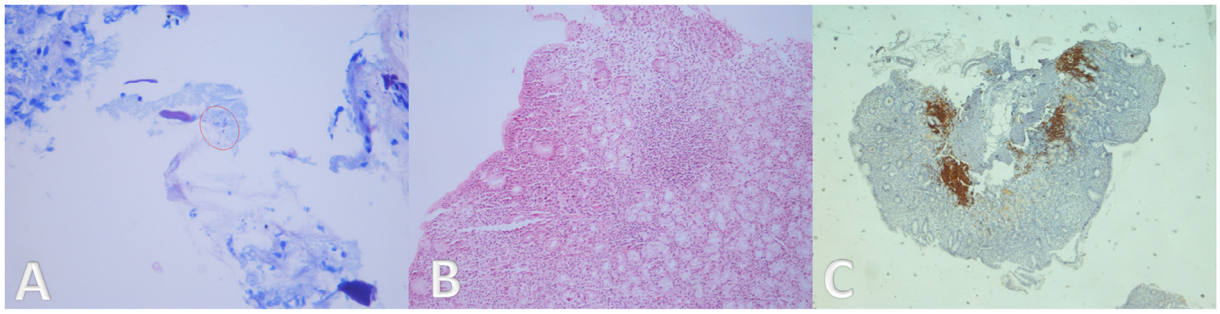

Fig. 2. (A) *Helicobacter pylori* in detail: curved rods stained with the Giemsa method (×400). (B) gastric mucosa permeation and gastric pits (H&E, ×100). (C) Immunohistochemistry revealing B cells (CD20+) forming prominent lymphoid follicles in the *lamina propria*.

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