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GASTRIC CANCER IN COMMON VARIABLE IMMUNODEFICIENCY

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A 52 years-old female, non-smoker, diagnosed with common variable immunodeficiency (CVID) since 2009, presented to the clinic for her periodic intravenous immunoglobulin replacement therapy. Her medical history was remarkable for recurrent Giardiasis and infected bronchiectasis, for which she received several antibiotic regimens.

Nutritional assessment revealed low BMI (15.2) and marked deficiencies due to malabsorption (low calcium, iron, albumin, folic acid, vitamins D and B12). One year previously, because of persistent chronic diarrhea (even after successful Giardia eradication), the patient underwent an upper gastrointestinal endoscopy with systematic biopsies which revealed chronic atrophic gastritis (non-metaplastic, non-dysplastic, absent Helicobacter pylori) and celiac-like enteropathy (with negative transglutaminase 2-targeted subepithelial deposits in the duodenal biopsy samples); ileo-colonoscopy was unremarkable.

In the setting of persistent malabsorption, we performed another upper gastrointestinal endoscopy, which showed the celiac-mimicking nodularity of the duodenal mucosa and a triangular-shaped ulceration in the gastric antrum, 7 mm in diameter, with irregular margins, rigid on biopsy sampling (Figure 1). Histopathology assessment of gastric biopsy specimens showed tumoral infiltrates suggestive of adenocarcinoma (Figure 2). The patient underwent bulbantrectomy with regional lymphadenectomy, with curative intent.

CVID is associated with an increased risk of malignancy, particularly lymphoma and gastric cancer[(1)]. Gastric cancer frequently occurs via the Correa pathway, with progressive histopathological changes in the normal mucosa, usually triggered by Helicobacter pylori infection, to chronic atrophic gastritis, intestinal metaplasia, dysplasia and cancer[(2)].

Particular features of CVID-associated CG are occurrence in younger ages, accelerated carcinogenesis and poorly differentiated histology[(1,3)].

Internal Medicine

Images in Medicine

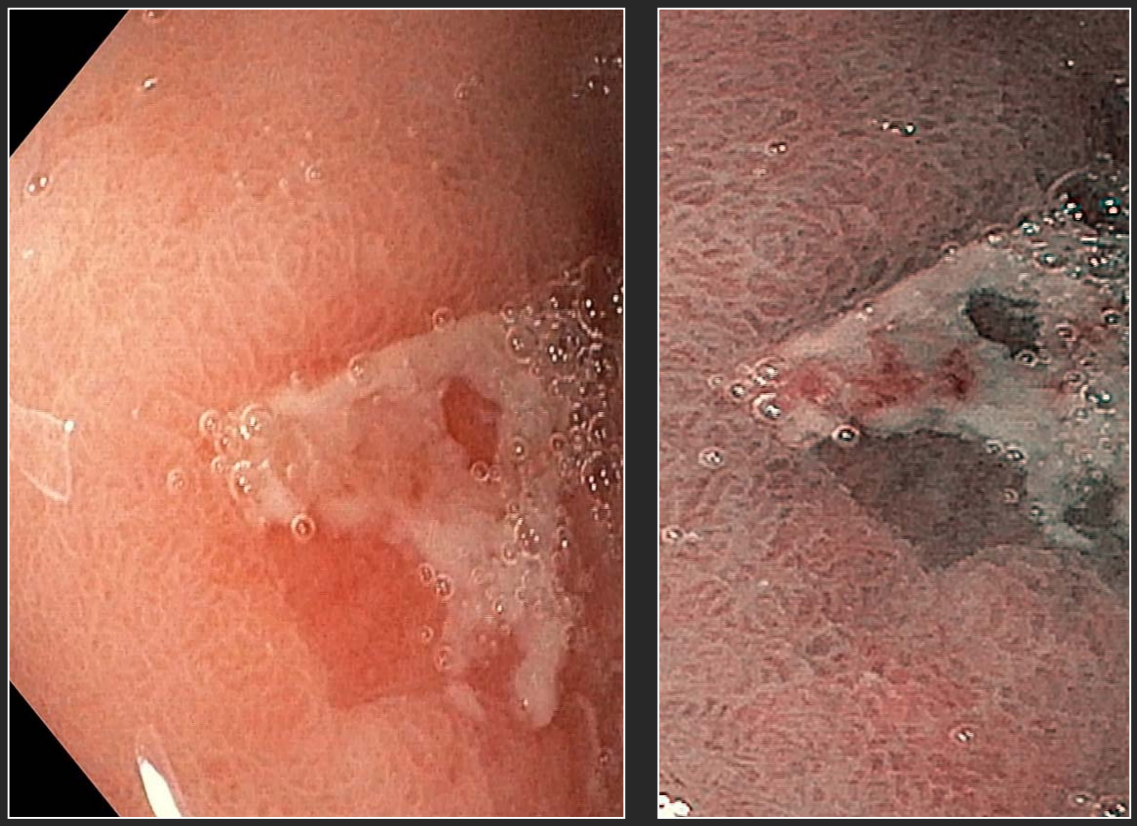

Figure 1. *Endoscopic image of gastric ulceration (left - white light/WL, right - narrow band imaging/NBI)*

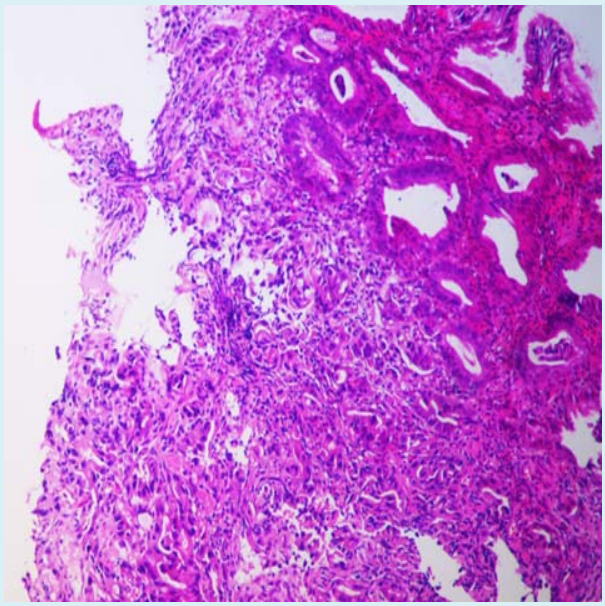

Figure 2. *Hematoxylin/eosin stain of gastric biopsies showing tumoral infiltrates of adenocarcinoma, moderately differentiated*

The mechanisms behind the GC excess in CVID patients include immune dysregulation, genetic factors, pernicious anemia and chronic *Helicobacter pylori infection*[4,5]. Our case report adds up to the current evidence that malignancy in CVID patients can rapidly occur even in non-dysplastic mucosa on recent previous endoscopic examination[3]. A more intensive surveillance than that recommended in MAPS II guideline is warranted in CVID patients[6].

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