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Successful treatment of proton pump inhibitor induced sporadic fundic gland polyps with an argon plasma coagulator in a patient with polycythaemia vera

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ABSTRACT

INTRODUCTION: Proton pump inhibitor (PPI) use is associated with the development of fundic gland polyps (FGPs); discontinuing PPIs is associated with regression of FGPs. Here, we report a rare case of non-respondent FGPs after discontinuation of PPI that were successfully treated using an argon plasma coagulator (APC).

PRESENTATION OF CASE: We present the case of a 68-year-old woman with a history of polycytheamia vera. She also had gastroesophageal reflux disease (GERD) and had been taking 10 mg of omeprazole daily for the past three years. Esophagogastroduedenoscopy (GF) revealed over 100 pedunculated polyps in the gastric body and fundus. Histological examination of the specimens showed dilated oxyntic glands with flattened parietal and mucous cells. Based on these findings and the clinical history, a diagnosis of FGPs was made. Omeprazole use was then discontinued. Repeat GF performed 6 months and 1 year later showed a significant increase in the number and size of the polyps. APC treatment was performed every 6 months for 3 years. Further GF showed a significant decrease in the number and size of the FGPs 4 years after discontinuing PPI.

DISCUSSION: We conclude that PPI use is a strong risk factor for the development of FGPs and discontinuing PPI is associated with regression of FGPs, but not in patients with polycythaemia vera. However, the mechanism involved in the interaction between FGP and polycytheamia vera remains unknown.

CONCLUSION: Non-respondent FGPs after discontinuation of PPI use may be successfully treated using APC.

1. Introduction

Fundic gland polyps (FGPs) are generally considered to be benign and the prevalence in patients undergoing upper endoscopy has been reported to be between 0.5% and 14% [1]. A recent study reported that long-term proton-pump inhibitor use increases the risk of FGPs [2]. There are two different types of FGPs: sporadic FGPs and FGPs in patients with familiar adenomatous polyposis (FAP) [3]. Although proton pump inhibitor (PPI) use is frequent, only a subset of patients develops multiple gastric polyps. While PPIs use is associated with the development of FGPs, discontinuation of PPIs is associated with regression of FGPs. Here, we report an extremely rare case of FGPs treated with an argon plasma coagulator (APC) due to continuous FGP development for almost 4 years after cessation of PPI use in a patient with polycythaemia vera (PV). The related literature is also reviewed. This work has been reported in line with the SCARE criteria [4].

2. Presentation of case

The patient was a 68-year-old woman with chronic epigastric pain who also had polycythaemia vera. She had been taking 10 mg of omeprazole daily for the past three years for gastroesophageal reflux disease (GERD). An esophagogastroduedenoscopy (GF) and

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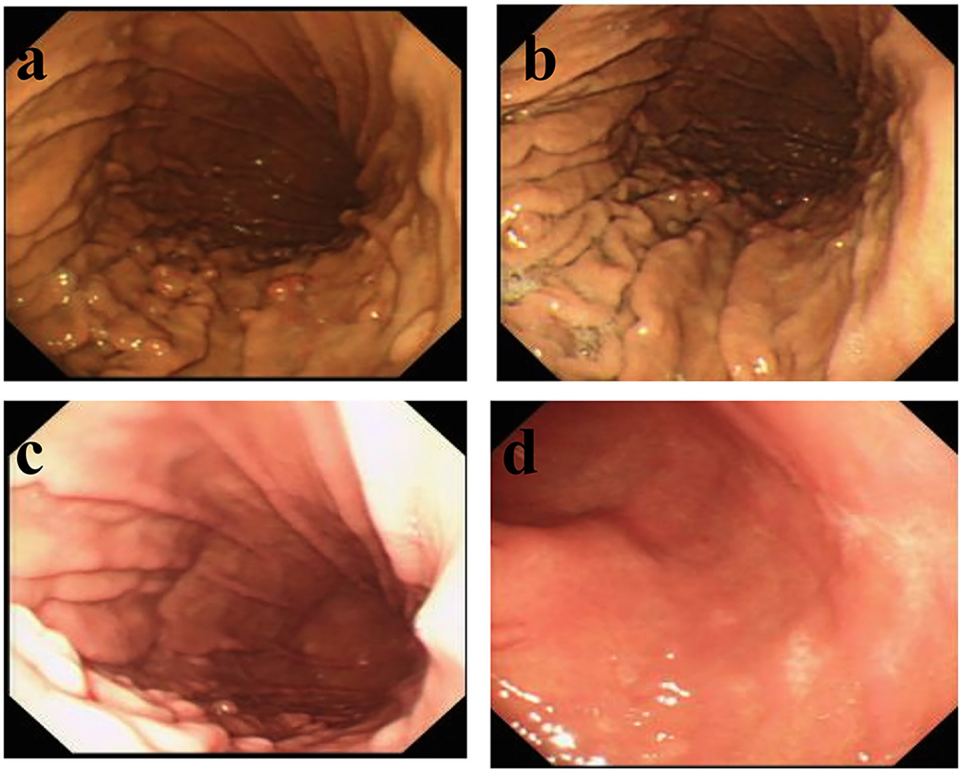

Fig. 1. Gasroendoscopic study image of fundic gland polyps (FGPs).
a: FGPs while the patient was on PPI. **b:** FGPs increased 1 year after discontinuation of PPI. **c:** Two years after discontinuation of PPI with APC treatment. **d:** Four years after discontinuation of PPI with APC treatment.

colonoscopy revealed over 100 pedunculated polyps in the gastric body and fundus (Fig. 1a). The antrum was normal. The size of the polyps was between 0.5 and 2.0 cm. Multiple biopsies of the polyps were taken. Histological examination of the specimens showed multiple fragments of fundic gland mucosa with dilated glands. The dilated gastric glands were lined by mucous neck and ballooned parietal cells. No mucosal inflammation, dysplasia, or Helicobacter pylori infection were observed (Fig. 2a, b). The immunohistochemical analysis showed that caudal-type homeobox 2 (CDX2)-negative cells were present in FGPs (Fig. 2c). The colonoscopy revealed no significant findings. The patient's past medical history included the use of rosuvastatin (2.5 mg, once daily) to treat hypercholestelemia (serum cholesterol level: 272 mg/dl) for almost 4 years. Laboratory findings revealed leukocytosis of 15,400/mm3; red blood cell count (RBC) 5,780,000/mm3; platelet count 276,000/μl; aspartate aminotransferase (AST) 30 U/L; alanine aminotransferase (ALT) 38 U/L; lactate dehydrogenase (LDH) 316 U/L; alkaline phosphates (ALP) 390 U/L; triglycerides 150 mg/dl; total cholesterol 288 mg/dl; creatine 0.7 mg/dl; creatinine phosphokinase (CPK) 393 U/L; blood urea nitrogen (BUN) 14 mg/dl; gastrin 92 pg/mL; prothrombin time 10.8 s; active partial prothrombin time 26 s; and fibrinogen degradation product 3.4 μg/mL. An abdominal computed tomography (CT) scan was negative for hemorrhage or infarction. During treatment for FGPs, the patient was diagnosed with polythetimia vera (PV) with a Janus kinase-2 (JAK2) V617F mutation and treated with ruxolitinib phosphate by the attending hematologist. Based on these findings and the clinical history, a diagnosis of FGPs with polycythaemia vera was made.

Omeprazole use was subsequently discontinued. A repeat GF was performed 6 months and 1 year later and revealed a significant increase in the number and size of the polyps. We applied APC (ERBE USA Inc., Marietta, GA, USA) at 1l/min and 40W over the FGP surface (Fig. 3a and b). During each GF, 50 ~ 100 FGPs were treated. APC treatment was performed every 6 months for almost 3 years, with 6 APC treatments in total. APC treatments resulted in a significant decrease in the size of the FGPs 4 years after discontinuing PPI (Fig. 1b–d). Pathology of biopsy specimens confirmed the presence of FGPs with no malignancy.

3. Discussion

FGP is defined as multiple fundic gland polyps in patients without familiar adenomatous polyposis syndrome (FAP). They are commonly reported in patients in their 60 s and predominantly in females. Jalving et al. reported that PPI therapy longer than 1 year is associated with a 4 fold increased risk of FGPs [3]. The mechanisms remain unknown. Genetic studies have shown that sporadic FGPs are linked to somatic mutations in the β-catenin gene and FAP-associated FGPs to germ-line mutations in the APC tumor suppressor gene [5]. Sporadic FRPs depend on activation of the β-catenin gene phosphorylation sites at exon 3 of the β-catenin gene [6]. Some groups have reported that morules associated with low grade dysplasia in FGPs might belong to the field of morules-associated tumors with wnt/β-catenin pathway disruption [7]. The β-catenin mutations in FGPs indicate a neoplastic nature, but it is known that FGPs have very limited malignant potential. CDX2 positivity in FGPs morules has been correlated with β-catenin expression. However, CDX 2 was negative in this case.

Several authors have reported that parietal cell enlargement has been observed following long-term PPI treatment in both animals and humans [8]. Long-term treatment is associated with both a large cystic area and partial cell hyperplasia and protrusion. The increase in gastrin production secondary to acid suppression could cause the enlargement of parietal cells and decrease the number of chief cells without affecting A-like cells [9]. Otherwise evidence of gastrin acting as a growth factor is lacking. PPI induced FGPs