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## Recommended Citation

Yakoob, J., Abbas, Z., Jafri, W., Ahmad, Z. (2008). Gastric corpus polyps associated with Proton Pump Inhibitors therapy. *Journal of the College of Physicians and Surgeons Pakistan*, 18(3), 172-3.

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# Gastric Corpus Polyps Associated with Proton Pump Inhibitors Therapy

Javed Yakoob, Zaigham Abbas, Wasim Jafri and Zubair Ahmad

## ABSTRACT

The prevalence of Gastroesophageal Reflux Disease (GERD) is rapidly rising in Asia. We describe here a case of 51 years old man who had surgery for esophageal leiomyoma and received long-term therapy with Proton Pump Inhibitors (PPIs) for persisting reflux symptoms. On Esophago-Gastroduodenoscopy (EGD) several sessile polyps were seen in the gastric corpus. Earlier EGD done 15 years back had not demonstrated those polyps. Sections revealed polypoid fragments of glandular epithelium with dilated glands and negative histology for *H. pylori*. Polymerase chain reaction for 16S ribosomal RNA gene (16S rRNA PCR) of *H. pylori* was also negative. This is the first report originating from an Asian country describing Fundal Gland Polyps (FGPs) in the corpus of stomach rather than fundus in a patient on long-term PPI therapy.

**Key words:** Gastroesophageal reflux disease. Proton pump inhibitors. Gastric corpus polyps.

## INTRODUCTION

Gastric Fundic Gland Polyps (FGPs) are sporadic gastric polyps. They are found in upto 84% of patients with Familial Adenomatous Polyposis (FAP). These FGPs occur almost exclusively in patients without *H. pylori* infection.<sup>1</sup> They have always been regarded as benign lesions, with low-grade dysplasia (intraepithelial neoplasia) at the most. There have, however, been case reports of FGPs harbouring severe dysplasia or even gastric adenocarcinoma, particularly when associated with FAP, but also in one sporadic gastric polyp.

Proton pump inhibitors are used widely in the management of acid-related disorders. They are effective for the majority of patients with gastroesophageal reflux disease, peptic ulcer disease and non-steroidal anti-inflammatory drug-induced gastropathy. They are considered safe but there are concerns about the possibility of an association with cancer, infection and gastric atrophy. Long-term usage is difficult to define and most patients take PPIs non-continuously. Self-prescription of medications is common in developing countries and a substantial proportion of long-term users appear not to have a clear indication for their therapy. Also, medications are freely available on the counters of pharmacies without prescription. We report here a case of long-term PPIs use, associated with Gastric Corpus Polyps (GCPs), in a patient with a history of dysmotility symptoms.

## CASE REPORT

A 51-year-old man presented with regurgitation, belching and retrosternal heaviness. He was on PPI (Losec) 40 mg OD, off and on, for the last 17 years. An Esophago-gastroduodenoscopy (EGD) revealed mild pangastric erythema and several whitish pink sessile polyps (< 1 cms) in the gastric corpus (Figure 1). Histology of the biopsies taken from the antrum and corpus revealed mild non-specific inflammatory cell infiltrate (Figure 2-3). *Helicobacter pylori* were not seen. Sections from sessile polyp revealed polypoid fragments of glandular epithelium with dilated glands. No evidence of metaplasia, dysplasia or malignancy was noted in specimens. PCR for 16S rRNA gene of *H. pylori* was negative in biopsy specimens removed from antrum, corpus and fundus of the stomach. His fasting serum gastrin was 180 pg/ml (normal range 13-115 pg/ml). In March 1992, he had a barium meal examination for similar complaints that showed a large lobulated mass in the mid esophagus—a leiomyoma. This was confirmed on an EGD examination demonstrating a bi-lobed extrinsic hemispherical defect mostly on the anterior wall extending from 26 cms from incisors to 34 cms. Esophageal mucosal covering was intact with increased vascularity. MRI demonstrated a mass involving 10 cms segment of esophagus below the aortic arch. He had surgical removal of leiomyoma of esophagus in July 1992. However, after surgery symptoms of reflex became worse. He took PPIs, which gave him some symptomatic relief but epigastric discomfort persisted. A repeat barium swallow examination demonstrated motility disorder involving distal half and relative dilatation of mid portion of esophagus, which did not demonstrate contractility. Subsequent EGDs did not demonstrate any pathology dilatation of the mid portion of esophagus.

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Received September 12, 2007; accepted January 12, 2008.



Figure 1: Gastric corpus polyps.

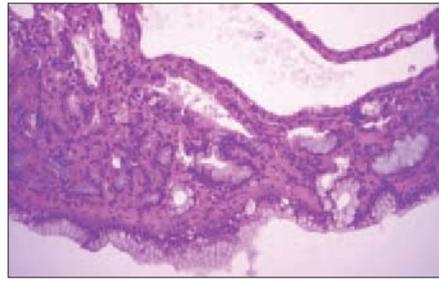


Figure 2: Microcysts lined by fundic epithelium overlying foveolae are shortened H and E (x 20).

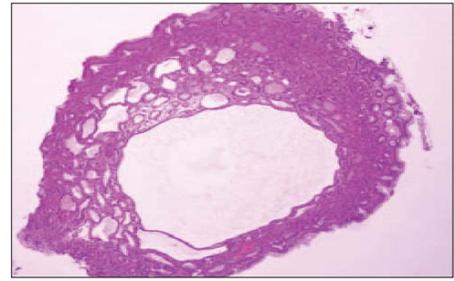


Figure 3: Polypoid fragment of gastric mucosa showing microcysts H and E (x 20).

## DISCUSSION

In our patient, there was a history of having surgery for esophageal leiomyoma, which is known to predispose to esophageal dysmotility. However, dysmotility tends to improve following surgical removal of leiomyoma.<sup>2</sup> The long-term use of PPI for 16 years predisposed him to develop gastric corpus polyps (GCPs). This is uncommon association described in the western literature. However, earlier reports of PPI induced FGPs are from fundus.<sup>3</sup> This is in contrast to hyperplastic gastric polyps associated with *H. pylori* infection, which are commonly described in corpus but may be distributed widely in the stomach. It is known that FGPs are not associated with *H. pylori* infection.<sup>4</sup> In this case, histology demonstrated mild non-specific inflammatory cell infiltrate and *H. pylori* were not seen on histology. Proton pump inhibitors have some bacteriostatic activity against *H. pylori* both *in vitro* and *in vivo* and also inhibit urease activity *in vitro*. It is also known that treatment with PPI is detrimental to *H. pylori* at both the antrum and corpus. However, PCR for 16S rRNA of *H. pylori* was conducted on the gastric biopsies obtained from antrum, corpus and fundus<sup>5</sup> to rule out concomitant *H. pylori* infection predisposing to GCPs. In a previous study, diagnostic yield of PCR for 16S rRNA (16S PCR) gene of *H. pylori* on gastric tissue was higher than Rapid Urease Test (RUT) and histology in patients on PPI and H2RB with *H. pylori* infection.<sup>6</sup> The 16S PCR for *H. pylori* was negative from all sites.

Proton pump inhibitors are a benzimidazole substituted anti-secretory agents. They decrease parietal cell acid secretion by inhibiting hydrogen/potassium-exchanging adenosine triphosphatase. Potent PPIs are preferred for GERD. The main concern with the long-term proton pump inhibitor use includes profound hypoacidity and hypergastrinemia.<sup>7</sup> Proton pump inhibitors induced hypergastrinemia has been associated with the development of FGPs.<sup>8</sup> These FGPs are most commonly reported in the fundus or acid secreting mucosa. Histologically, FGPs are characterized by distorted glandular architecture consisting of microcysts, mostly lined with chief and parietal cells.<sup>9</sup> Also, in humans, diffuse, linear, or micronodular hyperplasia of

enterochromaffin-like cells is observed in 10–30% of chronic PPI users, in particular in *H. pylori* positive patients with more markedly increased gastrin levels.<sup>10</sup> The elevated serum gastrin level in our case was consistent with the chronic PPI use.<sup>10</sup> Most of these patients have moderate to severe inflammation of the body mucosa, often with atrophic changes. However, dysplasia or invasive carcinoid formation has never been described in long-term PPI users. The patients developing such polyps should have a lower gastrointestinal endoscopy to exclude the possibility of familial polyposis when harbouring severe dysplasia or gastric adenocarcinoma.

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