Commentary

iMedPub Journals
www.imedpub.com

Journal of Clinical Gastroenterology and Hepatology
ISSN 2575-7733

2021
Vol.5 No.3:2

Long Term Outcomes of Sporadic Large Fundic Gland Polyps: A Commentary

Abdul Mohammed[1*] and Madhusudhan R Sanaka[2]

[1]Department of Internal Medicine, Cleveland Clinic Foundation, Cleveland, USA

[2]Department of Gastroenterology and Hepatology, Cleveland Clinic Foundation, Cleveland, USA

[*]**Corresponding author:** Abdul Mohammed, Department of Internal Medicine, Cleveland Clinic Foundation, Cleveland, USA, E-mail: mas.shariq@gmail.com

Received date: November 05, 2021; **Accepted date:** November 19, 2021**; Published date:** November 26, 2021

Citation: Mohammed A, Sanaka MR (2021) Long Term Outcomes of Sporadic Large Fundic Gland Polyps: A Commentary. J Clin Gastroenterol Hepatol. Vol.5 No.3:2.

Description

Fundic Gland Polyps (FGPs) were first described by Elster in 1976 [1]. FGPs histologically appear as dilated glandular cysts with superficial foveolar and deep oxyntic mucosa which arises from progressive formation and unfolding of secondary glandular buds [2]. Although they were initially associated with Familial Adenomatous Polyposis (FAP), the sporadic variant was later recognized as the more common entity [3]. Since sporadic FGPs are commonly small in size, with most between 2 mm and 7 mm, data on large (>10 mm) FGPs is lacking [4]. Except for FGPs in familial adenomatous polyposis, the clinical course of sporadic FGPs is generally benign. In a comparative study of 112 hyperplastic polyps, a majority of the identified malignant polyps had a size >10 mm. Dysplasia in FAP-associated FGPs is related to polyp size [5]. However, the malignant potential of large FGPs >10 mm is unknown. We conducted a large retrospective study that evaluates the risk factors, pathogenesis, and risk of gastric cancer in large (>10 mm) sporadic FGPs.

A total of 4000 consecutive patients undergoing EGD from April 2014 and December 2019 in a tertiary care hospital were retrospectively reviewed. Patients were included if they were found to have gastric polyps ≥ 10 mm on EGD and a histopathologic diagnosis of FGP. We only included patients who had sporadic FGP and excluded patients with any hereditary polyposis syndromes like familial adenomatous polyposis (FAP), dysplasia, adenoma, or carcinoma. Data on patient demographics like age, race, sex, body mass index (BMI), past medical history, PPI use, alcohol use, and tobacco use were collected. In addition to indications for EGD, data on polyp characteristics like the location of the polyp, size of the polyp, and histopathology were also collected. Follow-up data were recorded for patients who underwent repeat upper endoscopic evaluation after index or first EGD.

A total of 4000 patients with a diagnosis of FGP were screened and patients with dysplasia, adenoma, carcinoma, inherited polyposis syndromes, less than 10 mm size, and less than 18 years of age were excluded. A total of 132 patients met the study criteria and were included. The most common indication for the EGD was acid reflux (60.6%). Subgroup analysis based on polyp size on follow-up EGD data was performed. At every follow-up EGD large polyps ≥ 10 mm were resected. The polyps were divided into two groups based on the size of polyps, 10-19 mm and ≥ 20 mm. A total of 74 patients underwent at least one repeat EGD at a mean duration of 3.2 ± 2.9 years. The second follow-up EGD was performed in 18 patients at a mean duration of 4.8 ± 3.4 years. Seven patients underwent a third follow-up endoscopy at a mean duration of 7.2 ± 3.2 years. Three patients with dysplasia were found on the first follow-up EGD. Resection of these polyps was curative (R0). There were no dysplastic, adenomatous, or carcinomatous changes on further follow-up. The total follow-up including first, second, and third EGD was 371.3-person-years. The rate of dysplastic changes was 2.6 cases per 1000-person years of follow-up.

Our study suggests that sporadic large FGPs (≥ 10 mm) do not have significant malignant potential. The rate of dysplastic changes was only 2.6 cases per 1000-person years of follow-up. Hence, large sporadic FGPs do not require regular cancer surveillance. The rate of malignant transformation of sporadic FGPs to gastric adenocarcinoma is low. It is estimated to be approximately 1% by experts, and likely confined to polyps 10 mm and more in size, however sufficient data to support this hypothesis is lacking. Only 3 patient's developed dysplasia on follow-up but and no patients developed gastric adenoma or gastric adenocarcinoma. This is within the range of normal variation and is likely due to chance. Although, FGPs more than 20mm in size underwent follow-up endoscopy earlier when compared with polyps 10-19mm in size (3.4 years vs. 2.4 years), they did not differ in the rate of dysplasia. The relative low prevalence of dysplasia in FGPs is likely related to the absence of mutations in the APC gene that are responsible for dysplastic changes observed in FAP [6]. Further H. pylori, a primary etiologic agent of gastric cancer has a negative correlation with the formation of FGPs [7].

Gastroesophageal Reflux Disease (GERD) with and without esophagitis and gastritis were among the most common comorbidities associated with a diagnosis of large sporadic FGPs. A causal relationship between them is unclear, but a majority of these underlying conditions require therapy with PPIs. Sixty-one percent of our study group complained of reflux-like symptoms at the time of endoscopy and diagnosis of FGPs. Long-term PPI therapy is associated with an increased risk of developing gastric polyps [8]. The prevalence of FGPs increases with the duration of PPI usage but is not related to PPI dosing [9]. Tran-Duy et al. analyzed data from 12 studies comprising more than 87,234 patients. Based on systematic review and meta-analysis, they

concluded that long-term use of PPIs of more than 12 months was associated with an increased risk of FGPs [10]. Similarly, Martin et al. pooled data from multiple studies and demonstrated a four-fold or five-fold risk increased risk of developing FGPs with PPI usage of at least 6 months or 12 months [11]. We observed an overwhelming prevalence of PPI use (98.5%) in patients with sporadic large FGPs. It is postulated that FGPs develop as a result of reduced flow of fundic gland secretions due to blocking of mucus production by PPIs [12].

Although PPIs are used in the treatment of H. pylori infection, infection with H. pylori has been shown to promote glandular outflow and prevent cystic dilation and FGP formation [13]. FGPs are reported to regress following the acquisition of H. pylori infection and later increase in occurrence following eradication treatment [14,15]. Only 1 patient with large FGPs in our study was positive for H. pylori infection supporting this hypothesis.

Conclusion

In conclusion, large sporadic FGPs have a significant positive association with PPI use but a negative association with H. pylori infection. GERD is the most common indication for EGD in these patients. Although the rate of follow-up is low, large FGPs do not appear to have significant neoplastic potential and do not require regular surveillance endoscopy.

References

1. Elster K (1976) Histologic classification of gastric polyps. Curr Top Pathol 63:77-93.
2. Lee RG, Burt RW (1986) The histopathology of fundic gland polyps of the stomach. Am J Clin Pathol 86:498-503.
3. Watanabe H, Enjoji M, Yao T, Ohsato K (1978) Gastric lesions in familial adenomatosis coli: their incidence and histologic analysis. Hum Pathol 9:269-283.
4. Torbenson M, Lee JH, Cruz-Correa M, Ravich W, Rastgar K, et al. (2002) Sporadic fundic gland polyposis: A clinical, histological, and molecular analysis. Mod Pathol 15:718-723.
5. Offerhaus GJ, Giardiello FM, Krush AJ, Booker SV, Tersmette AC, et al. (1992) The risk of upper gastrointestinal cancer in familial adenomatous polyposis. Gastroenterol 102:1980-1982.
6. Abraham SC, Park SJ, Mugartegui L, Hamilton SR, Wu TT (2002) Sporadic fundic gland polyps with epithelial dysplasia : evidence for preferential targeting for mutations in the adenomatous polyposis coli gene. Am J Pathol 161:1735-1742.
7. Hsu PI, Lai KH, Hsu PN, Lo GH, Yu HC, et al. (2007) Helicobacter pylori infection and the risk of gastric malignancy. Am J Gastroenterol 102:725-730.
8. Choudhry U, Boyce HW, Coppola D (1998) Proton pump inhibitor-associated gastric polyps: a retrospective analysis of their frequency, and endoscopic, histologic, and ultrastructural characteristics. Am J Clin Pathol 110:615-621.
9. Ally MR, Veerappan GR, Maydonovitch CL, Duncan TJ, Perry JL, et al. (2009) Chronic proton pump inhibitor therapy associated with increased development of fundic gland polyps. Dig Dis Sci 54: 2617-2622.
10. Tran-Duy A, Spaetgens B, Hoes AW, de Wit NJ, Stehouwer CDA (2016) Use of Proton Pump Inhibitors and Risks of Fundic Gland Polyps and Gastric Cancer: Systematic Review and Meta-analysis. Clin Gastroenterol Hepatol 14:1706-1719.e5.
11. Martin FC, Chenevix-Trench G, Yeomans ND (2016) Systematic review with meta-analysis: Fundic gland polyps and proton pump inhibitors. Aliment Pharmacol Ther 44:915-925.
12. Cats A, Schenk BE, Bloemena E, Roosedaal R, Lindeman J, et al. (2000) Parietal cell protrusions and fundic gland cysts during omeprazole maintenance treatment. Hum Pathol 31: 684-690.
13. Sakai N, Tatsuta M, Hirasawa R, Iishi H, Baba M, et al. (1998) Low prevalence of Helicobacter pylori infection in patients with hamartomatous fundic polyps. Dig Dis Sci 43:766-772.
14. Watanabe N, Seno H, Nakajima T, Yazumi S, Miyamoto S, et al. (2002) Regression of fundic gland polyps following acquisition of Helicobacter pylori. Gut 2002; 51: 742-745.
15. Notsu T, Adachi K, Mishiro T, Ishimura N, Ishihara S (2020) Fundic gland polyp prevalence according to Helicobacter pylori infection status. J Gastroenterol Hepatol 35: 1158-1162.