BMC Gastroenterology

RESEARCH Open Access

Clinicopathological characteristics of 30 cases of pyloric gland adenoma: a single center case series

Junzhen Hou[1], Fandong Meng[2], Bing Yue[3], Peng Li[2*] and Ningning Dong[2*]

Abstract

Objective To investigate the clinicopathological characteristics of patients with pyloric gland adenoma (PGA), as well as their prognosis.

Methods Data of 30 cases from 24 patients, who had been histologically diagnosed as PGA, was retrospectively analyzed. Clinical characteristics, histopathological features, treatment and prognosis were assessed and analyzed.

Results Among the 24 patients, there were 15 females and 9 males, with an average age of 59 years old. Ten cases were located in the stomach, 4 in the duodenal bulb and 16 in the gallbladder. The average maximum diameter was 13.5 mm and morphologically the vast majority were type 0-I. The assessment of the background mucosa identified autoimmune gastritis (AIG), *Helicobacter Pylori*-associated gastritis and familial adenomatous polyposis (FAP) in the stomach, ectopic gastric mucosa in the duodenum and chronic cholecystitis in the gallbladder. Endoscopic resection was performed on the vast majority of gastric and duodenal cases, while all gallbladder PGAs underwent cholecystectomy. Histologically, 33.3% of PGAs showed high-grade dysplasia.

Conclusions Due to malignant potential, it is recommended that PGAs should be completely resected, especially if they are large or show high-grade features. For gastric and duodenal PGAs, endoscopic resection has been proven to be safe and effective. Whether PGAs occurring in the gallbladder and stomach are different tumors still requires more cases for further study.

Keywords Pyloric gland adenoma, Clinicopathological features, High-grade dysplasia, Endoscopic resection

*Correspondence:
Peng Li
lipeng@ccmu.edu.cn
Ningning Dong
dongnn83@163.com
[1]Department of Gastroenterology, Shijingshan Teaching Hospital of Capital Medical University, Beijing Shijingshan Hospital, Beijing 100040, China
[2]Department of Gastroenterology, Beijing Friendship Hospital, Capital Medical University, State Key Laboratory of Digestive Health, National Clinical Research Center for Digestive Disease, Beijing Key Laboratory of Early Gastrointestinal Cancer Medicine and Medical Devices, 95 Yong'an Road, Xi-cheng District, Beijing 100050, China
[3]Department of Pathology, Beijing Friendship Hospital, Capital Medical University, Beijing 100050, China

Introduction

Pyloric gland adenomas (PGAs) are rare neoplasms, dominated by the differentiation of gastric pyloric glands/mucous neck cells. They were first reported by Elster in 1976 [1]. PGAs most commonly occur in the stomach, but extra-gastric sites of PGAs include the duodenum, gallbladder, esophagus, bile duct, and rectum [2–6]. Gastric PGAs may occur sporadically or in patients with polyposis syndrome, such as familial adenomatous polyposis (FAP). A clinical study from Germany showed that PGAs accounted for 2.7% of all gastric polyps and 9% of neoplastic gastric polyps [2], suggesting that PGAs may be

under-recognized. PGA is clinically important, because 12–51.2% of PGAs show sustained progression to well-differentiated adenocarcinoma at the time of initial diagnosis [2, 3, 7], indicating the malignant potential of PGAs and the need for complete resection.

Due to their rarity, PGAs are not well characterized and remain difficult to diagnose without histopathology. Furthermore, the pathogenesis of PGAs is still unclear, and their association with various forms of mucosal injury has not been well documented in the literature. Herein we retrospectively reviewed the clinical and histological data of 24 patients with PGAs, who were diagnosed and treated at Beijing Friendship Hospital, Capital Medical University from January 2020 to December 2023, aiming to assist endoscopists and pathologists in improving their ability to diagnose and treat of this tumor.

Materials and methods

We retrospectively analyzed the clinicopathological features of 30 cases of PGA from 24 patients, who underwent endoscopic or surgical resection at Beijing Friendship Hospital of Capital Medical University from January 2020 to December 2023. The study was approved by the Ethics Committee of Beijing Friendship Hospital, Capital Medical University (Beijing, China), and all patients signed an informed consent for treatment.

The demographics, clinical manifestations and histopathological features were obtained from the patients' medical records. We evaluated the following characteristics: gender, age, clinical symptoms, lesion location and size, morphology, associated comorbidities, background mucosa (inflammation and atrophy), histological and immunological features, treatment and follow-up. In stomach and duodenum, *Helicobacter pylori* (*H. pylori*) infection status, endoscopic characteristics (such as shape, color and boundary) were also evaluated, and the microvessel (MV) and microsurface (MS) pattern were observed on magnifying endoscopy (ME) with narrow-band imaging (NBI), using the vessel plus surface classification system as diagnostic criteria.

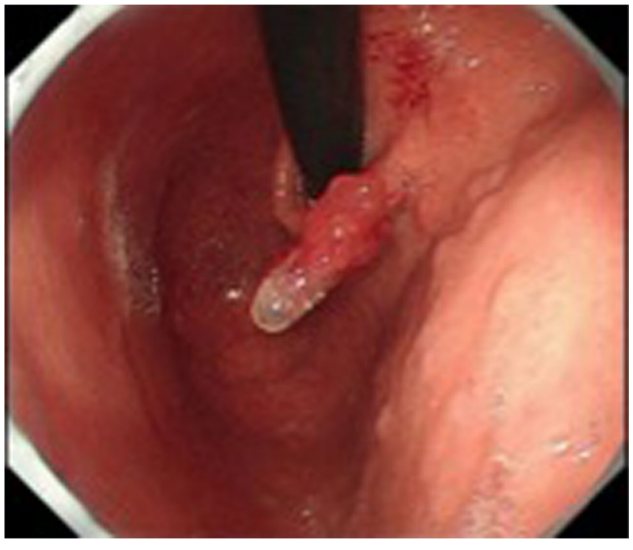

Fig. 1 Image of endoscopy of a gastric PGA presented as type 0-I with hyperemia on the surface and the surrounding mucosa showed marked atrophic gastritis

H. pylori infection status was determined by various methods, including ^{13}C-urea breath test (UBT) (Shenzhen China National Nuclear Corporation Heidewe Biotechnology, China), serum *H. pylori* antibody test, microscopic observations of biopsied/resected specimens, and endoscopic manifestations [8]. Additionally, the past history of prior successful *H. pylori* eradication therapy was taken into consideration. The ^{13}C-UBT test was conducted on patients who had not taken proton pump inhibitors within the past two weeks, and antibiotics or bismuth within the past 4 weeks. The cut-off value to distinguish whether the ^{13}C-UBT is positive or negative was defined as 4‰. Based on the results of these tests, the patients were classified as current *H. pylori* infection, past *H. pylori* infection or *H. pylori*-uninfected.

Autoimmune gastritis (AIG) is diagnosed according to histological criteria, which has been considered as the gold standard. The typical histological characteristics include marked reduction of oxyntic glands, pseudopyloric and intestinal metaplasia, and the proliferation of mucus-neck and foveolar cells.

The histopathological features were analyzed by hematoxylin and eosin (HE) staining and immunohistochemical staining (for Mucin 6, Mucin 5AC, Mucin 2, CD 10, Pepsinogen-I, Ki67, and P53). The degree of epithelial dysplasia and background mucosa were also analyzed. An En-Vision two-step method was used for immunohistochemical labelling. The P53 antibody was purchased from Ventana Company in the United States. The pepsinogen-I antibody and H+/K+-ATPase were both purchased from Abcam Company in the United States. Other antibodies were purchased from Fuzhou Maixin Medical Technology Co., Ltd. Negative and positive controls were established for the above markers.

Results

Clinical features

The study included 30 cases of PGA from 24 patients, comprising 15 females (15/24, 62.5%) and 9 males (9/24, 37.5%). The average age at diagnosis was 59 years old (range, 24 to 81 years). Most patients were asymptomatic, except for 6 patients (6/24, 25%) presenting with non-specific symptoms, such as epigastric pain/distension, dyspepsia, heartburn, and acid regurgitation. Out of the 30 cases of PGA, 10 were located in the stomach (Fig. 1, Case 3) (10/30, 33.3%) (including 6 in the gastric body, 3 in the fundus and 1 in the antrum), 4 in the duodenal

bulb (Fig. 2, case 13) (4/30, 13.3%), and 16 in the gallbladder (16/30, 53.3%). The average maximum diameter was 13.5 mm (range, 2 ~ 45 mm).

Of the 14 gastric and duodenal PGAs, their endoscopic manifestations were diverse on conventional endoscopy. According to Paris classification [9], ten PGAs were classified as type 0-I (Fig. 1, Case 3), three as type 0-IIa (Fig. 3, case 5), and one as type 0-IIb. Furthermore, five lesions (5/14, 35.7%) were accompanied by hyperemia and redness, while the remaining nine lesions (9/14, 64.3%) were generally the same color as the surrounding mucosa. On ME with NBI, irregular MV/MS pattern was observed in five PGAs (Fig. 4, Case 8), which were histologically confirmed as high-grade dysplasia. In terms of gastric comorbidities, one case was complicated with multiple gastric neuroendocrine tumors (g-NETs) (Fig. 5, Case 2), another case with multiple early gastric cancer, and a third case with advanced gastric cancer outside the PGA.

Among the 16 cases of gallbladder PGA from 10 patients, who were all asymptomatic, 14 cases were resected due to gallbladder polyps, while the remaining 2 cases were tiny tumors of 2 mm, which were incidentally discovered after cholecystectomy due to cholecystolithiasis. Macroscopically, among the 16 cases of gallbladder PGA, except for 2 cases with tiny tumors classified as type 0-IIa, others were all classified as type 0-I.

Pathological features

Regarding the background mucosa, of the 10 gastric PGAs, 5 cases (5/10, 50%) were AIG (Fig. 1, Case 3), 4 cases (4/10, 40%) were *H. Pylori* -associated gastritis, and one case (1/10, 10%) was FAP. Meanwhile, ectopic gastric mucosa was found around all the 4 duodenal PGAs (Fig. 2, case 13). All the 16 cases of gallbladder PGA were surrounded with chronic cholecystitis.

According to WHO (2019) classification system for digestive system tumors [10], PGAs with low-grade dysplasia (LGD) are characterized by closely packed pyloric-type glands. These glands are lined by cuboidal/low columnar epithelia, and are partially accompanied by dilated glands. Neoplastic cells exhibit a defined ground-glass appearance, with clear or lightly eosinophilic cytoplasm. The round to ovoid nuclei are basally located, with inconspicuous nucleoli. PGAs with high-grade dysplasia (HGD) are characterized by disturbed architecture, crowded nuclei, and loss of nuclear polarity. In the present study, 20 cases of PGA (20/30, 66.7%) demonstrated LGD, 9 cases (9/30, 30.0%) demonstrated LGD simultaneously with HGD (Fig. 6), and only one case (1/30, 3.3%) demonstrated HGD. Immunohistochemical staining revealed that all PGAs were positive for both MUC6 (Fig. 7) and MUC5AC (Fig. 8), but did not express MUC2 or CD10. Meanwhile, five PGAs showed focal positivity

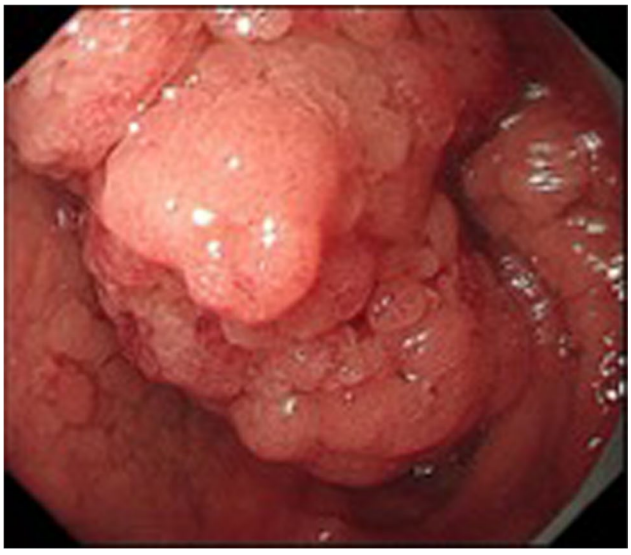

Fig. 2 Image of endoscopy of a PGA in the duodenal bulb with ectopic gastric mucosa surrounding the tumor

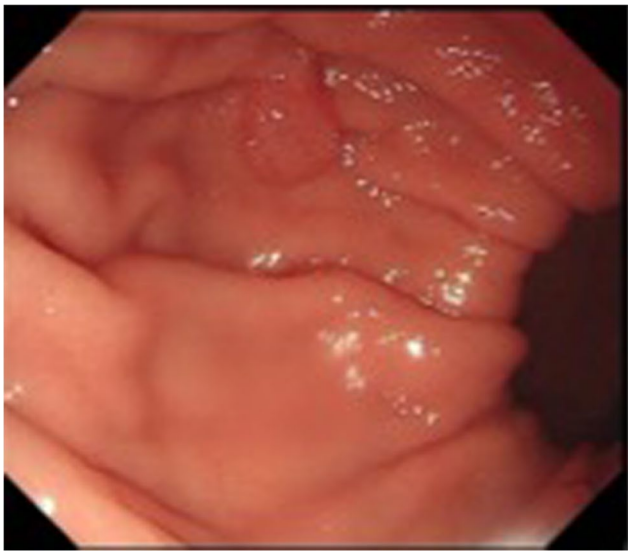

Fig. 3 Image of endoscopy of a gastric PGA presented as type 0-IIa

on Pepsinogen-I staining (including three gastric PGAs, one duodenal PGA and one gallbladder PGA). P53 and Ki67 were predominantly expressed on the surface of PGAs. Wild-type p53 expression was increased mainly in the MUC5AC positive region, and the Ki67 proliferation index was also increased in the same region.

Treatment and prognosis

Among the 14 gastric and duodenal PGAs, 12 patients (12/14, 85.7%) underwent endoscopic resection. including eight cases with endoscopic submucosal dissection (ESD) and four cases with endoscopic mucosal resection (EMR), all of which were completely resected without any serious complications such as perforation,

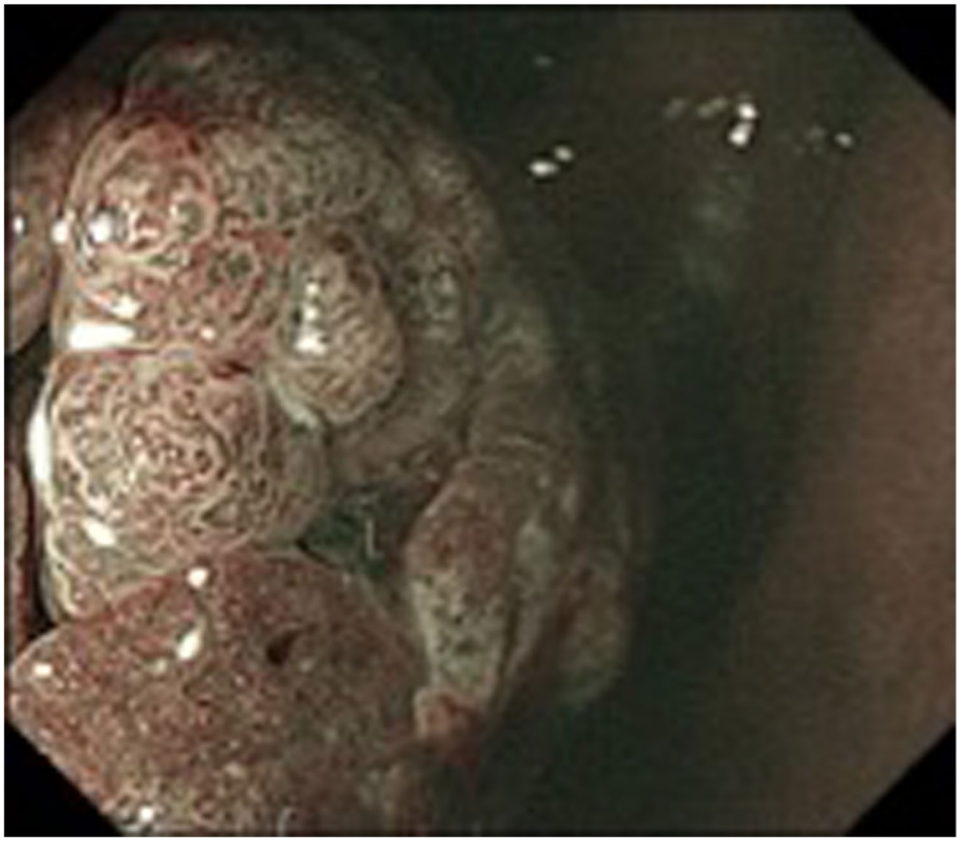

Fig. 4 Image of a gastric PGA demonstrated irregular microvessel and microsurface pattern on magnifying endoscopy with narrow-band imaging

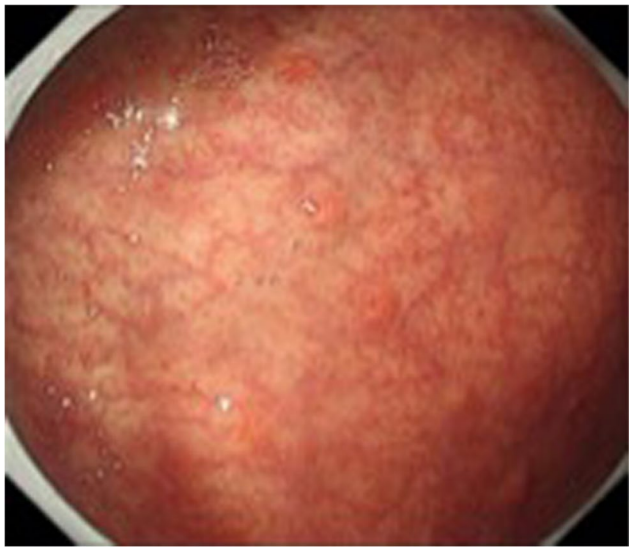

Fig. 5 Image of endoscopy of a gastric PGA complicated with multiple gastric neuroendocrine tumors

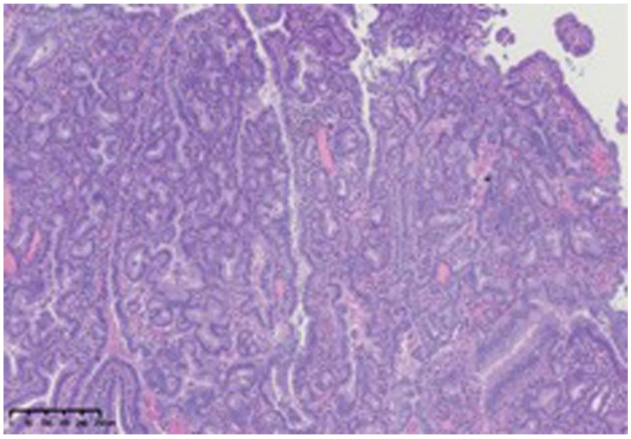

Fig. 6 Medium-power magnification of a PGA on HE staining demonstrated low-grade dysplasia (yellow arrow) simultaneously with high-grade dysplasia (red arrow)

obvious bleeding, or infection. One case of a small lesion of PGA was removed by biopsy forceps, and the patient was followed up with repeated endoscopic examination that showed no lesion. Another patient with a lesion in the gastric antrum underwent total gastrectomy due to concurrent advanced gastric cancer in the corpus. Follow-up gastroscopy (range, 6 to 37 months) was performed for all the 14 patients after resection, and no residual or recurrent lesion was observed.

All the 16 cases of gallbladder PGAs underwent cholecystectomy, due to gallbladder polyps or cholecystolithiasis, all of which were completely resected. Three cases were lost to follow-up, and the remaining seven cases

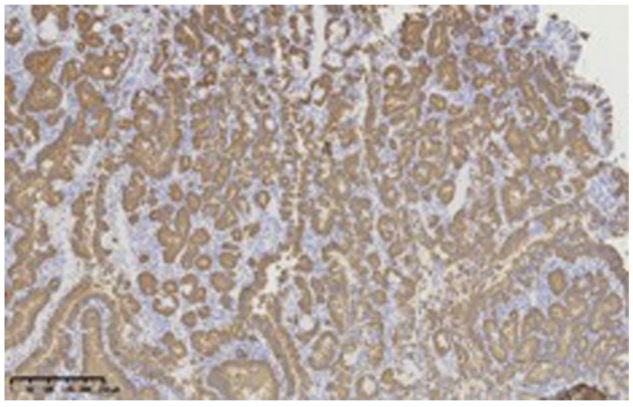

Fig. 7 Immunohistochemical staining for MUC6 showed diffuse and strong expression

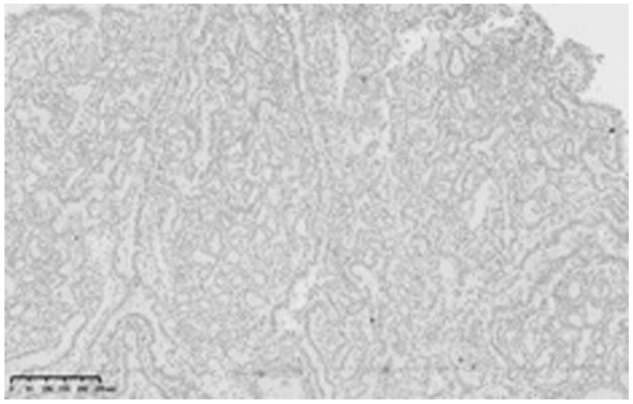

Fig. 8 Immunohistochemical staining for MUC5AC showed co-expression in neoplastic glands

were followed up for 6–61 months. No recurrence or metastasis was found.

A summary of the clinicopathological features of the 30 cases of PGA from 24 patients was shown in Table 1.

Discussion

In the present study, we reported the clinicopathologic features of 30 cases of PGA from 24 patients. PGAs are more common in elderly patients, with a modest female predominance, and the age at diagnosis ranged from 24 to 81 years old, which are consistent with previous literature [2, 3, 6]. PGAs are rare neoplasms of the gastrointestinal tract, mostly asymptomatic and found incidentally. However, a few of patients presented with nonspecific dyspeptic symptoms, including epigastric pain/distension, dyspepsia or heartburn and acid regurgitation in the present study. Furthermore, according to previous literature, a few patients presented with anemia secondary to chronic hemorrhage and gastric outlet obstruction due to PGA [11].

PGAs are primarily found in the stomach, duodenum and gallbladder, but occasionally occur in the esophagus, rectum and bile ducts [2, 4, 5, 12, 13]. Gastric PGAs are most commonly located in the body/fundus, followed by the antrum and cardia. These are consistent with the present study.

According to previous literatures [13–15], on conventional white light endoscopy, PGAs in the stomach and duodenum were non-specific, and presented as polypoid, dome-shaped, nodular protrusions, uneven mucosa, flat protrusions, ulcers, or submucosal tumor-like protrusions. In the present study, the morphology ranged from dome-shaped or nodular protrusions, superficial flat protrusions to uneven mucosa. According to Paris classification, type 0-I and type 0-IIa were the most common. The surface of PGAs could be either hyperemic and reddish or unaltered. On ME with NBI, irregular MS/MV and the observation of papillary/villous surface pattern may suggest high-grade dysplasia or carcinoma [3].

PGAs need to be differentiated from hyperplastic polyps endoscopically and foveolar-type adenomas histologically. According to WHO Classification (2019) [10], gastric adenomas are subdivided into PGAs and foveolar-type adenomas. The main diagnostic challenge in PGAs is to distinguish them from foveolar-type adenomas. Immunohistochemical staining for MUC6 and MUC5AC can aid in the differential diagnosis. The absence of a well-defined apical mucin cap distinguishes PGAs from foveolar-type cell dominated lesions. Foveolar-type adenomas have a well- defined apical mucin cap and immunohistochemical staining for MUC5AC is positive, whereas MUC6 is negative. On the contrary, PGAs do not have a well- defined apical mucin cap and immunohistochemical staining for MUC5AC and MUC6 are both positive.

Choi et al. [3] defined three immunohistochemical phenotypes for PGAs: pure pyloric type with strong MUC-6 expression; predominant foveolar type (the rarest type) with diffuse expression of MUC-5AC but ≤ 10% of MUC-6 expression; and mixed type (the majority type) with variable expression of MUC-5AC and MUC-6. According to this classification, among the 30 cases of PGA in the present study, 23 cases were of mixed type (9 in the stomach, 4 in the duodenum, and 10 in the gallbladder) and 7 cases were of pure pyloric type (1 in the stomach and 6 in the gallbladder). The study did not find any predominant foveolar type, which is generally consistent with previous literature [3].

In the present study, Immunohistochemical staining revealed that all PGAs were positive for both MUC6 and MUC5AC. Furthermore, Pepsinogen-I, secreted by the chief cells and mucous neck cells, showed focal positive expression in five cases (including three gastric PGAs, one duodenal PGA and one gallbladder PGA). Based on these findings, it is inferred that histologically PGAs probably originate from pyloric gland, pyloric gland

Table 1 Clinicopathological characteristics of the 30 cases of PGA from 24 patients

Num-ber	Gen-der	Age (years)	Symptoms	Location	MD (mm)	Morph-ology	Comorbidities	H. Pylori infection	treatment	Dysplasia
1	F	65	-	Gastric fundus	20	0-I	AIG	UN	EMR	LGD + HGD
2	F	40	epigastric distension	Gastric body	10	0-I	FAP	UN	EMR	LGD
3	F	69	dyspepsia	Gastric body	15	0-I	AIG, g-NET	UN	ESD	LGD
4	M	57	-	Gastric body	4	0-IIa		Cur	Biopsy	LGD
5	F	76	-	Gastric body	12	0-IIa	AIG	UN	EMR	LGD
6	M	59	-	Gastric antrum	15	0-I	AGC	Cur	Gastrecto-my	LGD
7	F	67	-	Gastric fundus	45	0-I		Pre	ESD	HGD
8	F	59	acid regurgitation	Gastric fundus	40	0-I	AIG	UN	ESD	LGD + HGD
9	F	62	epigastric pain	Gastric body	7	0-IIb		UN	ESD	LGD
10	F	70	epigastric pain	Gastric body	6	0-IIa	AIG, EGC	UN	ESD	LGD
11	F	68	heartburn, acid regurgitation	Duodenal bulb	40	0-I		Cur	ESD	LGD + HGD
12	M	58	-	Duodenal bulb	20	0-I		Cur	ESD	LGD + HGD
13	M	78	-	Duodenal bulb	25	0-I		NA	ESD	LGD
14	M	58	-	Duodenal bulb	8	0-I		UN	EMR	LGD
15	F	48	-	Gallbladd-er	6	0-I	GS, CC	NA	cholecystec-tomy	LGD
16	F	53	-	Gallbladd-er	20	0-I	CC	NA	cholecystec-tomy	LGD + HGD
				Gallbladd-er	6	0-I				LGD
17	F	52	-	Gallbladd-er	2	0-IIa	GS, CC	NA	cholecystec-tomy	LGD
18	M	81	-	Gallbladd-er	15	0-I	GS, CC	NA	cholecystec-tomy	LGD + HGD
				Gallbladd-er	12	0-I				LGD + HGD
				Gallbladd-er	8	0-I				LGD
19	M	60	-	Gallbladd-er	2	0-IIa	GS, CC	NA	cholecystec-tomy	LGD
20	M	26	-	Gallbladd-er	20	0-I	CC	NA	cholecystec-tomy	LGD + HGD
				Gallbladd-er	10	0-I				LGD + HGD
21	F	47	-	Gallbladd-er	4	0-I	CC	NA	cholecystec-tomy	LGD
				Gallbladd-er	4	0-I				LGD
				Gallbladd-er	4	0-I				LGD
22	F	51	-	Gallbladd-er	15	0-I	CC	NA	cholecystec-tomy	LGD

Table 1 (continued)

Num-ber	Gen-der	Age (years)	Symptoms	Location	MD (mm)	Morph-ology	Comorbidities	H. Pylori infection	treatment	Dysplasia
23	F	32	-	Gallbladd-er	4	0-I	CC	NA	cholecystec-tomy	LGD
24	M	74	-	Gallbladd-er	7	0-I	CC	NA	cholecystec-tomy	LGD

Gender (F = Female; M = Male); *H.pylori* Infection status, *Cur* Currently infected, *Pre* Previously infected, *UN* Uninfected with *H. pylori*, *NA* Not available, *MD* Maximum diameter of the maximum lesion, *AGC* Advanced gastric carcinoma, *EGC* Early gastric cancer, *ESD* Endoscopic submucosal dissection, *EMR* Endoscopic mucosal resection, *GS* Gallbladder stone, *CC* Chronic cholecystitis

metaplasia, ectopic gastric mucosa, or mucous neck epithelium, which is consistent with previous literature [16]. Moreover, by analyzing the present cases, it was found that PGAs of the gallbladder, stomach, and duodenum cannot be completely separated in terms of immunophenotype, and gastric pyloric gland/mucous neck cell differentiation, which is not consistent with previous literature [17]. Currently, there are few molecular studies on PGA. Through next-generation sequencing technology, Setia et al. [18] discovered that all of the PGAs with low-grade dysplasia had gene mutations such as APC, KRAS, and GNAS. In high-grade dysplasia/adenocarcinoma, multiple gene mutations were found including APC, CTNNB1, KRAS, GNAS, TP53, CDKN2A, PIK3CA, and EPHA5. However, the gene mutation ratio of APC, KRAS, and GNAS decline in comparison with that in low-grade dysplasia, and the tumor mutation burden is higher. In previous literature [17], gene mutations in KRAS and GNAS in gallbladder PGA are fewer compared with those in gastric and duodenal, and He et al. believed that PGA may be due to that gallbladder PGA can't be detected at an early stage, and the proportion of high-grade cases is relatively high. Whether PGAs occurring in the gallbladder and stomach are different tumors still requires more cases for further study.

Gastric PGAs often occur in the background of AIG and *H.pylori*-associated gastritis, as well as genetic predisposition (such as FAP), chemical gastritis and normal mucosa [2, 3, 19]. In the present study, all the 10 gastric PGAs arose from the background of abnormal mucosa, including AIG, *H.Pylori*-associated gastritis, and FAP. Furthermore, gastric PGAs may coexist with gastric neuroendocrine tumors, early or advanced gastric cancer. Hence it is recommended to make a full assessment of the background mucosa and *H. pylori* infection status for evaluation of other preneoplastic condition of the stomach.

The four duodenal PGAs occurred in the setting of ectopic gastric mucosa. All the 16 cases of gallbladder PGA were surrounded with chronic cholecystitis. Extragastric PGAs are currently believed to be associated with pyloric gland ectopia or metaplasia in the setting of chronic mucosal injury [2, 20–22].

PGAs are clinically significant due to their malignant potential. Kushima R et al. [14] demonstrated a "pyloric gland adenoma-adenocarcinoma sequence" by analyses of mucin immunohistochemistry and CGH for the first time. Chlumska A et al. [6] showed that the nature of PGAs is associated with the accumulation of p53 and the mutation of oncogenes GNAS, KRAS, CTTNB1, and the tumor suppressor genes SMAD4 and TP53 through molecular genetics research. Moreover, it is crucial to acknowledge PGAs as neoplasms that carry a risk of transforming into invasive adenocarcinoma, which is supported by several reports [19, 20, 23–25] that have identified PGA either at diagnosis or progressing to mucosal carcinoma, invasive carcinoma, and even lymph node metastasis during follow-up. These studies indicated the possibility of malignant transformation of PGA, which could further develop into high-grade dysplasia and adenocarcinoma. Immunohistochemical staining for Ki67 and p53 can aid in identifying whether they are combined with adenocarcinoma or not. The risk of HGD and adenocarcinoma increases with the size of PGA, tubule-villous structure, and the presence of AIG as well as mixed immunophenotype [3].

In this study, the vast majority of the gastric and duodenal PGAs were treated with endoscopic resection, including ESD and EMR, all of which had been completely resected without any serious complications. It is suggested that endoscopic resection of PGAs is safe and effective in the stomach and duodenum. Gallbladder PGAs, mostly found incidentally due to gallbladder polyps or cholecystolithiasis, need to be completely resected by cholecystectomy.

There are two major limitations in this study that could be addressed in future research. First, this study has limitations inherent to its single-center and retrospective design. The limited number of cases may limit the universality of the results. Multi-center collaborations are warranted to validate these results across multiple clinical settings. However, because of the rarity of PGA, prospective study is hard to implement. Second, molecular study cannot be performed in our center so far, hence we are unable to compare the molecular characteristics of our cases of PGA with previous literature.

Conclusions

Due to malignant potential, it is recommended that PGAs should be completely resected, especially if they are large or show high-grade features. For gastric and duodenal PGAs, endoscopic resection has been proven to be safe and effective. Whether PGAs occurring in the gallbladder and stomach are different tumors still requires more cases for further study.

Acknowledgements
None.

Authors' contributions
Junzhen Hou conceived of and performed the study, wrote the manuscript, and gave final approval of the version to be published. Fandong Meng participated in the conception of the study and data collection. Bing Yue was responsible for the pathological analysis. Peng Li conceived of the study and participated in critically revising the manuscript. Ningning Dong conceived of and performed the study, participated in critically revising the manuscript. All authors read and approved the final manuscript.

Funding
The study was funded by National Key Research and Development Program of China (2023YFC2507400, 2023YFC2507404), and National Major Project of Science and Technology for the Prevention and Control of the "Four Major Chronic Diseases" (2024ZD0520904).

Data availability
The reader can access the research data by contacting the corresponding author via mail dongnn83@163.com.

Declarations

Ethics approval and consent to participate
This study was approved by the Ethic Committee of Beijing Friendship Hospital, Capital Medical University (Beijing, China). All the 24 patients provided informed consent for treatment and participation.

Consent for publication
Not applicable. All details of participants' images or information have been de-identified so that the identities of the patients may not be ascertained in any way.

Competing interests
The authors declare no competing interests.

Received: 3 May 2024 / Accepted: 23 June 2025
Published online: 07 July 2025

References

1. Elster K. Histologic classification of gastric polyps[J]. Curr Top Pathol. 1976;63:77–93. https://doi.org/10.1007/978-3-642-66481-6_3.
2. Vieth M, Kushima R, Borchard F, et al. Pyloric gland adenoma: a clinico-pathological analysis of 90 cases[J]. Virchows Arch. 2003;442(4):317–21. https://doi.org/10.1007/s00428-002-0750-6.
3. Choi WT, Brown I, Ushiku T, et al. Gastric pyloric gland adenoma: a multicentre clinicopathological study of 67 cases[J]. Histopathology. 2018;72(6):1007–14. https://doi.org/10.1111/his.13460.
4. Park K, Kim DH, Lee SD, et al. Pyloric gland adenoma of the esophagus treated by endoscopic submucosal dissection: a case report[J]. Gut Liver. 2022;16(3):483–6. https://doi.org/10.5009/gnl210357.
5. Vieth M, Kushima R, de Jonge JP, et al. Adenoma with gastric differentiation (so-called pyloric gland adenoma) in a heterotopic gastric corpus mucosa in the rectum[J]. Virchows Arch. 2005;446(5):542–5. https://doi.org/10.1007/s00428-005-1242-2.
6. Chlumska A, Waloschek T, Mukensnabl P, et al. Pyloric gland adenoma: a histologic, immunohistochemical and molecular genetic study of 23 cases[J]. Cesk Patol. 2015;51(3):137–43 PMID:26421956.
7. Vieth M, Kushima R, Mukaisho K, et al. Immunohistochemical analysis of pyloric gland adenomas using a series of Mucin 2, Mucin 5AC, Mucin 6, CD10, Ki67 and p53[J]. Virchows Arch. 2010;457(5):529–36. https://doi.org/10.1007/s00428-010-0968-7.
8. Kamada T, et al. Helicobacter pylori infection and endoscopic gastritis -Kyoto classification of gastritis. Nihon Shokakibyo Gakkai Zasshi. 2015;112(6):982–93. https://doi.org/10.11405/nisshoshi.112.982.
9. Update on the paris classification of superficial neoplastic lesions in the digestive tract[J]. Endoscopy, 2005,37(6):570-8. https://doi.org/10.1055/s-2005-861352
10. 10. WHO Classification of Tumours Editorial Board. WHO Classification of Tumours. Digestive System Tumours [M]. 5th ed. Lyon: IARC Press, 2019.
11. Pimentel R, Gravito-Soares E, Gravito-Soares M, et al. Dead-end stomach: a giant and pedunculated gastric pyloric gland adenoma conditioning gastric outlet obstruction and anemia[J]. Rev Esp Enferm Dig. 2021;113(9):678–9. https://doi.org/10.17235/reed.2021.7820/2021.
12. Kushima R, Vieth M, Mukaisho K, et al. Pyloric gland adenoma arising in Barrett's esophagus with mucin immunohistochemical and molecular cytogenetic evaluation[J]. Virchows Arch. 2005;446(5):537–41. https://doi.org/10.1007/s00428-004-1185-z.
13. Pezhouh MK, Park JY. Gastric pyloric gland adenoma[J]. Arch Pathol Lab Med. 2015;139(6):823–6. https://doi.org/10.5858/arpa.2013-0613-RS.
14. Kushima R, Vieth M, Borchard F, et al. Gastric-type well-differentiated adenocarcinoma and pyloric gland adenoma of the stomach[J]. Gastric Cancer. 2006;9(3):177–84. https://doi.org/10.1007/s10120-006-0381-8.
15. Cakar E, Bayrak S, Pasaoglu E, et al. Pyloric gland adenoma: a case report[J]. Case Rep Gastroenterol. 2013;7(3):392–5. https://doi.org/10.1159/000355342.
16. Nakajo K, Oono Y, Kuwata T. Case of pyloric gland adenoma accompanied by a component of foveolar epithelial-type adenoma within the lesion[J]. Dig Endosc. 2018;30(5):673. https://doi.org/10.1111/den.13180.
17. He C, Fukumura Y, Toriyama A, et al. Pyloric Gland Adenoma (PGA) of the gallbladder: a unique and distinct tumor from PGAs of the stomach, duodenum, and pancreas[J]. Am J Surg Pathol. 2018;42(9):1237–45. https://doi.org/10.1097/PAS.0000000000001117.
18. Setia N, Wanjari P, Yassan L, et al. Next-generation sequencing identifies 2 genomically distinct groups among pyloric gland adenomas[J]. Hum Pathol. 2020;97:103–11. https://doi.org/10.1016/j.humpath.2019.11.004.
19. Chen ZM, Scudiere JR, Abraham SC, et al. Pyloric gland adenoma: an entity distinct from gastric foveolar type adenoma[J]. Am J Surg Pathol. 2009;33(2):186–93. https://doi.org/10.1097/PAS.0b013e31817d7ff4.
20. Miller GC, Kumarasinghe MP, Borowsky J, et al. Clinicopathological features of pyloric gland adenomas of the duodenum: a multicentre study of 57 cases[J]. Histopathology. 2020;76(3):404–10. https://doi.org/10.1111/his.13996.
21. Kitagawa D, Yamasaki T, Ikeda T, et al. A case of pyloric gland adenoma with high-grade dysplasia in the duodenum arising from heterotopic gastric mucosa observed over 5 years[J]. Clin J Gastroenterol. 2023;16(1):26–31. https://doi.org/10.1007/s12328-022-01721-6.
22. Kushima R, Ruthlein HJ, Stolte M, et al. 'Pyloric gland-type adenoma' arising in heterotopic gastric mucosa of the duodenum, with dysplastic progression of the gastric type[J]. Virchows Arch. 1999;435(4):452–7. https://doi.org/10.1007/s004280050425.
23. Vieth M, Montgomery EA. Some observations on pyloric gland adenoma: an uncommon and long ignored entity! [J]. J Clin Pathol. 2014;67(10):883–90. https://doi.org/10.1136/jclinpath-2014-202553.
24. Schaefer IM, Cameron S, Middel P, et al. Pyloric gland adenoma of the cystic duct with malignant transformation: report of a case with a review of the literature[J]. BMC Cancer. 2012;12:570. https://doi.org/10.1186/1471-2407-12-570.
25. Khor TS, Brown I, Kattampallil J, et al. Duodenal adenocarcinoma arising from a pyloric gland adenoma with a brief review of the literature[J]. BMJ Case Rep. 2010;2010:bcr1020103385. https://doi.org/10.1136/bcr.10.2010.3385.

Publisher's Note