

## Original Article

# Endoscopic and clinicopathological features of gastric adenocarcinoma of fundic gland mucosa type: a case report and literature review

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**Abstract:** Background: Gastric adenocarcinoma of fundic gland mucosa type (GA-FGM) is a low-grade atypia and well-differentiated adenocarcinoma. Thus far, ten cases of GA-FGM have been reported in the literature. We report a case and discuss the clinicopathological features of GA-FGM to improve the understanding and diagnosis of GA-FGM. Methods: We retrospectively reviewed the clinical, endoscopic, histological, and immunophenotypic features and treatment of 1 case of GA-FGM, and combined this with literature discussion. Results: This is a 52-year-old male patient who underwent upper gastrointestinal endoscopy and was found to have a type IIc lesion on the upper part of the stomach. Most of the surface of the cancer tissue was covered with normal epithelium as observed under low magnification. The surface of the focal mucosa was covered with tumorous gastric pit epithelial cells. Deep tumors are mainly composed of a large number of eosinophils and a small number of gastric pit cells, which are mottled. Tumor cell nucleus showed mildly atypical characteristics with loss of nuclear polarity and visible nuclear fission. The glandular structure had significant atypia. Tumor invaded the submucosa. Immunohistochemistry showed that MUC5AC was positive on the surface and deep pit-epithelial-like tumor cells. The deep cancer tissues were widely positive for pepsinogen I (pepsin A) and H+/K+-ATPase, and some cancer cells expressed MUC6. The Ki-67 proliferation index was approximately 3%. Conclusions: GA-FGM is a rare new histological type of gastric cancer. The possibility of GA-FGM should be considered when the gastric intrinsic mucosal epithelial cells are mildly atypical and the fundic gland structure is markedly atypical and the distribution pattern of the relative normal mucosal immune phenotype was observed, especially in biopsy specimens.

**Keywords:** Gastric adenocarcinoma of fundic gland mucosa type, gastric adenocarcinoma of fundic gland type, endoscopic submucosal dissection

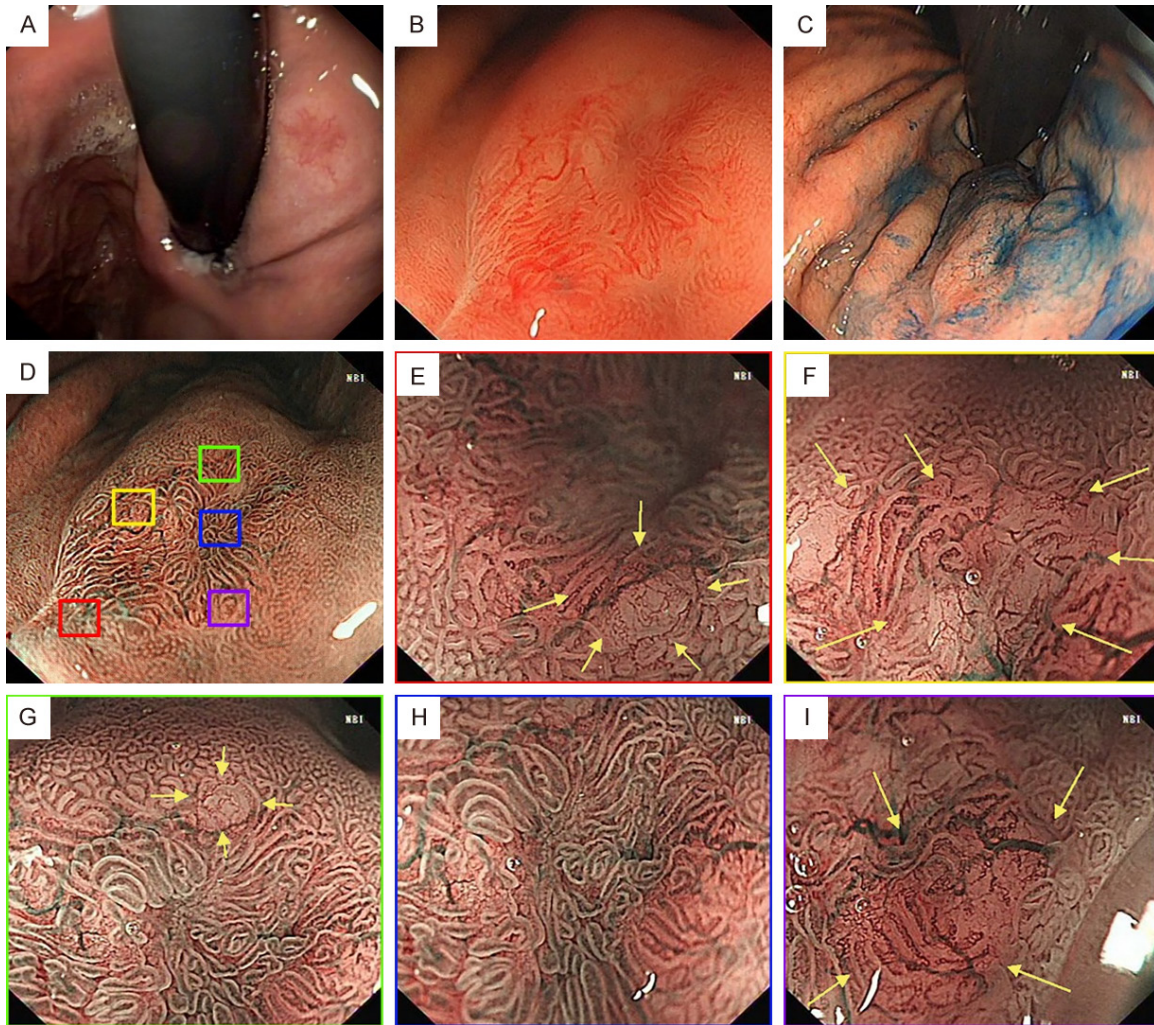
## Introduction

Gastric adenocarcinoma of the fundic gland type (GA-FG) is a rare disease entity first reported by Tsukamoto et al [1] in 2007. GA-FG is a well-differentiated adenocarcinoma with mild nuclear atypia. In 2010, Ueyama et al [2] further confirmed that "GA-FG" is a low-grade malignancy, and the tumor mainly differentiates into mucous neck cells and chief cells; it also differentiates into parietal-type cells. They proposed GA-FG as a new entity of gastric ade-

nocarcinoma. According to previous reports, GA-FG accounted for 0.98%-1.6% [3, 4] of gastric cancer cases, and the reported number of cases has kept increasing [5].

Recently, a "gastric adenocarcinoma of fundic gland mucosa type (GA-FGM)" [6] similar to "GA-FG" but with a different nomenclature has appeared. GA-FG mainly proliferates in the deeper layers of the mucosa, while the epithelium of the surface layer is a non-neoplastic region [2-5, 7-10]. In addition to fundic gland

## Gastric adenocarcinoma of fundic gland mucosa type



**Figure 1.** GA-FGM under white light endoscope (A-C) and observed by magnified NBI (D-I). (A) A superficial depression type lesion is observed on the small curved side of the upper part of the stomach. (B) The marginal crypt epithelium (MCE) of close observation is elongated and disordered. Dendritic vessels are observed. (C) Indigocarmine staining. The boundary of the lesion is not clear. (D) Clockwise enlargement of different color box areas. (E) Blood vessel tortuosity. (F) The MCE is long-lined and partially disappears. The upper glandular opening is pinhole-like. (G) Microvascular hyperplasia. (H) The elongated MCE has different lengths and directions. (I) The MCE is not clear and is partly in granular shape.

cell-like differentiation, a lesion showing MUC-5AC-positive foveolar epithelium-like differentiation is defined as “GA-FGM” [6]. Thus far, only ten cases of GA-FGM have been reported [6, 11-13]. In the present study, we report a case and discuss the clinicopathological features of GA-FGM with literature to improve the understanding and diagnosis of GA-FGM.

### Case presentation

#### *Clinical manifestation*

A 52-year-old man who underwent upper gastrointestinal endoscopy (GIF-Q150, Olympus,

Tokyo, Japan) for intermittent upper abdominal pain at a local hospital, and was found to have type IIc lesion (**Figure 1A**) on the small curved side of the upper part of the stomach. A biopsy was performed, and the pathological diagnosis was chronic inflammation of the gastric mucosa. The patient was admitted to our hospital in May, 2018. Physical examination revealed no abnormal finding in the chest and abdomen.

#### *Auxiliary examination*

Ordinary white light endoscopic (GIF-H260Z+CV-290/CLV-290SL, Olympus, Tokyo, Japan) examination revealed that the lesion was clas-

## Gastric adenocarcinoma of fundic gland mucosa type

**Table 1.** Antibodies used, their pretreatments, dilutions, and manufacturers

Antibody	Clone	Dilution	Pretreatment*	Manufacturer
MUC2	Ccp58	Ready to use	Citric acid	Celnovte Biotech, China
MUC5AC	MRQ-19	Ready to use	EDTA	Celnovte Biotech
MUC6	MRQ-20	Ready to use	EDTA	Celnovte Biotech
CD10	56C6	Ready to use	EDTA	Celnovte Biotech
Pepsinogen I	7G3	1:1600	EDTA	Abcam, Boston, USA
H+/K+-ATPase	2G11	1:400	Citric acid	Enzo, Farmingdale, NY
CD31	JC/70A	Ready to use	EDTA	Maixin Biotech, China
D2-40	D2-40	Ready to use	Citric acid	Maixin Biotech
CD56	123C3.D5	Ready to use	Citric acid	Maixin Biotech
Syn	27G12	Ready to use	EDTA	Celnovte Biotech
CgA	5H7	Ready to use	EDTA	Celnovte Biotech
Hp	MX014	Ready to use	Citric acid	Maixin Biotech
CDX2	SP21	Ready to use	EDTA	Maixin Biotech
Villin	CWWB1	Ready to use	EDTA	Celnovte Biotech
Desmin	D33	Ready to use	No pretreatment	Maixin Biotech
C-erb-2	EP3	Ready to use	EDTA	Celnovte Biotech
β-catenin	CAT-5H10	Ready to use	Citric acid	Maixin Biotech
Cyclin D1	EP12	Ready to use	EDTA	Celnovte Biotech
p53	DO-7	Ready to use	EDTA	Celnovte Biotech
Ki-67	MIB-1	Ready to use	EDTA	Celnovte Biotech

\*Antigen repair was performed at high temperature and high pressure using potassium EDTA (1 mM pH 9.0)/citric acid (0.01 M, pH 6.0) antigen repair fluid.

sified as type IIc with a diameter of <10 mm. The lesion color was faded, and dendritic vessels were readily visible. The marginal crypt epithelium (MCE) was elongated with varying lengths and directions (**Figure 1B**). The boundary was not clear after spraying indicarmine (concentration: 0.2%) (**Figure 1C**). Magnifying narrow-band imaging (NBI) observation: The lesion boundary was clear, abnormal vascular morphology; some MCE disappeared, and some MCEs were significantly elongated (**Figure 1D-I**). The background mucosal glandular opening was pinhole-like, with no atrophy and intestinal metaplasia.

The C-13 breath test was negative. Other blood test results were normal. Abdominal computed tomography examination showed no abnormalities.

### Treatment and follow-up

The lesion was judged to be an indication for endoscopic submucosal dissection (ESD). After the patient's informed consent and signature, ESD was performed for one-time complete resection. Endoscopic examination at 12 months

after ESD showed good healing results of the original lesion areas.

### Materials and methods

The tumor specimens were fixed in 10% neutral formalin, then conventional paraffin sectioning and hematoxylin and eosin (H&E) staining were performed. The morphology of tumor tissue was observed under a light microscope. The EliVision-2 kit of immunohistochemistry were provided by Maixin Biotech (catalog No. KIT-5930, Fuzhou, China). The experimental procedures were carried out according to the instructions. The primary antibodies and their pretreatment methods are listed in **Table 1**.

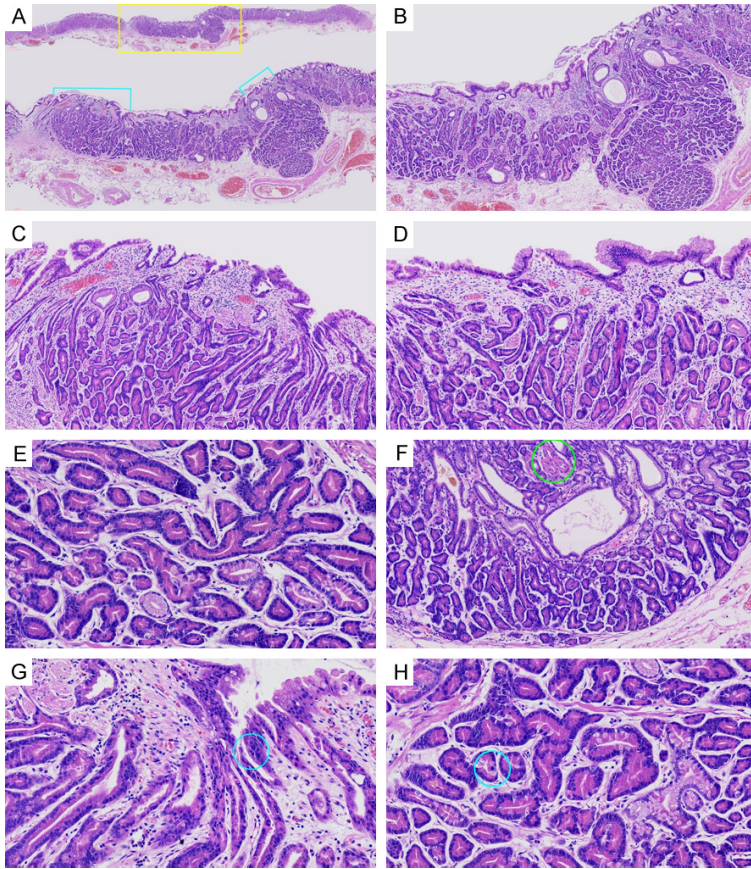
### Results

#### Pathological examination

Visual observation: A mucosal tissue, 28 mm × 22 mm in size, with a type IIc lesion at 7 mm from the proximal margin and with a size of 9 mm × 6 mm.

Histological morphology: The boundary of cancer tissue was clear, and some cancer tissue

## Gastric adenocarcinoma of fundic gland mucosa type



**Figure 2.** GA-FGM. (A) The deeply stained area at the center is GA-FGM tissue (in the yellow box). The cancer tissue infiltrates the submucosa. The surface of the focal area is covered by cancer tissue (below the cyan line). (B) Dense hyperplasia of tumor glands. (C) The focal surface layer is covered with the tumorous epithelial cells, which are connected to the underlying cancer tissue. (D) The tumorous glands are inconsistent in direction, and the surface is covered with normal epithelial tissue. (E) The branched tubular gland is complex in structure and resembles an “antler” or “ginger”. (F) Thick-walled blood vessels (in the green circle) are visible in the cancer tissue. The front edge of the tumor grew in an expansive manner. (G) High magnification, the direction of the cancer cells are disordered, and loss of nuclear polarity, showing nuclear division (within the cyan circle). (H) The same gland is composed of eosinophilic epithelial cells and cytoplasmic translucent pit-epithelial-like cells, and showing mottled shape. The cancer cells are columnar, and nuclear division is observed (within the cyan circle). HE staining (A-H).

were located in the submucosa (**Figure 2A** and **2B**). Most of the surface of the cancer tissue was covered with normal epithelium as observed under low magnification. The surface of the focal mucosa was covered with atypical gastric pit epithelial cells, and there was a band area with sparse regional glands under the epithelium (**Figure 2C** and **2D**).

The morphology of the cancer tissue was relatively consistent. Branched tubular glands densely proliferated with disordered arrangement.

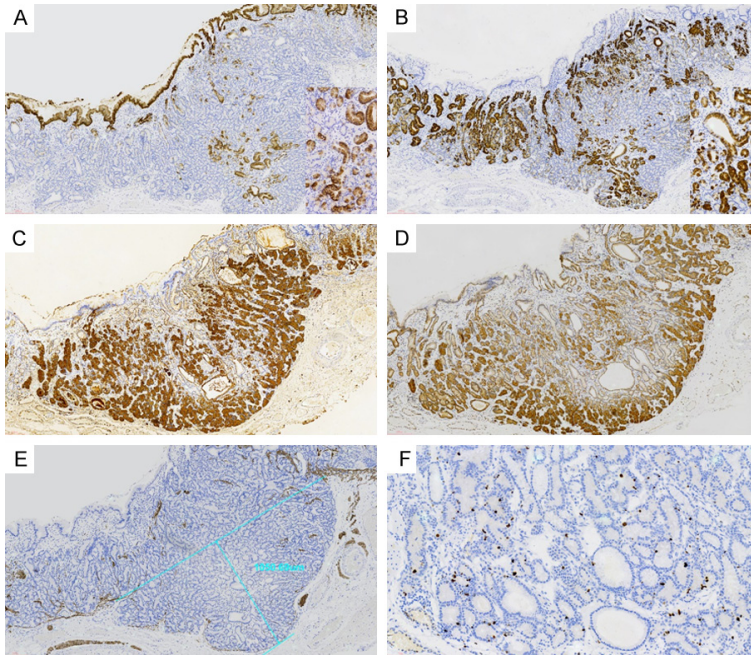
Some gland structures were complex, and resembled “antler” or “ginger” (**Figure 2E**). The leading edge of the cancer tissue was infiltrated in an expanding growth pattern (**Figure 2F**).

The cancer cells were arranged in a columnar or high columnar shape. The most common was eosinophilic epithelial cells, which had a slightly enlarged nucleus in comparison to normal epithelium with a round to rod-like or irregularly shaped nucleus, with deep nuclear staining, fine and dense chromatin, and centralized distribution of the chromatin at the edge. These cancer cells had median amphoteric nucleoli, and nuclear division could be observed. Atypical nuclear division could also be observed. The polarity of the nucleus was disordered or lost. The nuclei were arranged in a crowded or even overlapping manner and located on the basal side. The cytoplasm was abundantly and obviously eosinophilic. In some areas, the polarity of cells was disordered. Another type of cells were pit-epithelial-like cells with a round or oval nucleus located on the basal side. The nucleus was slightly vacuolated. The chromatin was slender, sparse, with clear nucleoli; the cytoplasm was transparent or lightly stained; and the polarity of nucleus was

disordered. The two types of cells appeared together in the same gland, or they were scattered and distributed in the same gland in a mottled condition (**Figure 2G** and **2H**).

Proliferative mucosal muscles were observed between the tumor glands. There was no obvious fibrous connective tissue hyperplasia. A small number of lymphocytes, plasma cells, and eosinophils was observed. No necrosis, ulcers, or ulcer scars were observed. The mucosa around the lesion was close to the normal

## Gastric adenocarcinoma of fundic gland mucosa type



**Figure 3.** GA-FGM immunohistochemical staining. MUC5AC-positive in pit epithelium and deep pit-epithelial-like tumor cells (A), partially cancerous MUC6-positive (B), and most cancer cells express pepsinogen I (C) and H<sup>+</sup>/K<sup>+</sup>-ATPase (D). Desmin staining showed a small amount of mucosal muscle remaining in the cancer tissue with an infiltration depth greater than 1000  $\mu$ m (E). The Ki-67 proliferation index is approximately 3% (F).

status without atrophy and obvious inflammation. No *Helicobacter pylori* (Hp) was observed.

### Immunophenotype findings

MUC5AC was positive on the surface and deep pit-epithelial-like tumor cells. The deep cancer tissues were widely positive for pepsinogen I (pepsin A) and H<sup>+</sup>/K<sup>+</sup>-ATPase, and some cancer cells expressed MUC6. Desmin was significantly reduced in the mucosal muscle tissue within the lesion. Ki-67-positive cells were scattered and unevenly distributed with 3% in the highest area (Figures 3 and 4A-F). The proportion of Cyclin D1-positive cells was approximately 75%. The  $\beta$ -catenin membrane was positive, and no staining was observed in the nucleus. Synaptophysin, CD56, Chromogranin A, MUC2, CD10, CDX2, and Villin were negative; p53 was scattered with different strengths in a few positive cells, and tumor cells were not stained for c-erbB-2. CD31 and D2-40 showed no cancer infiltration in the lacunae of vessels and lymphatics, respectively, and Hp staining was negative.

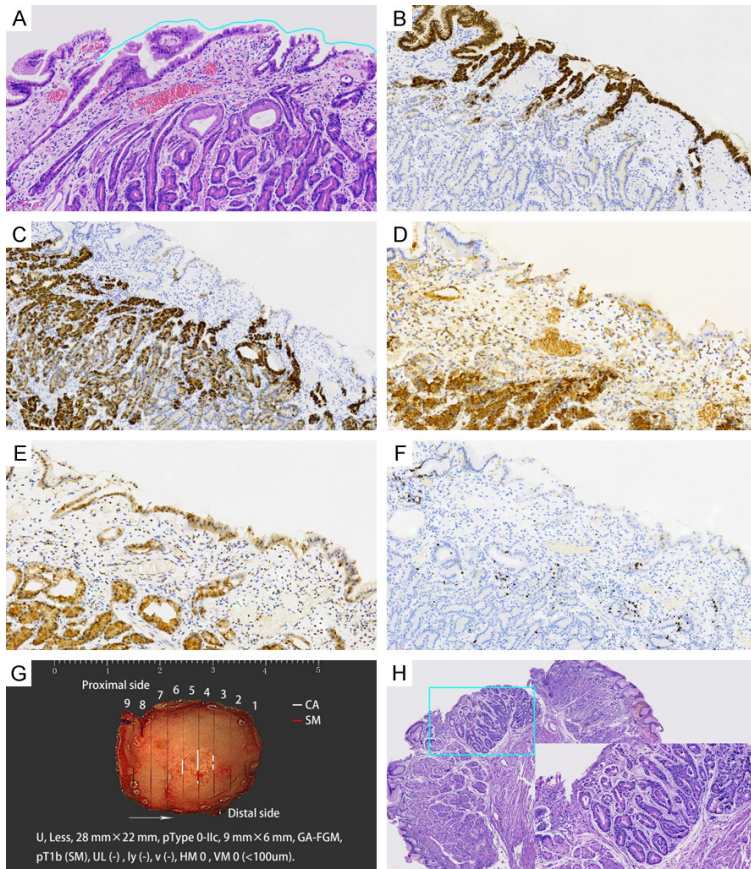
Final pathological diagnosis was GA-FGM (Figure 4G). The pathological sections of preoperative biopsy conducted in another hospital were retrospectively reviewed (Figure 4H), and the histological morphology was the same as that of the ESD specimen.

### Discussion

GA-FGM is a low-grade atypia and well-differentiated gastric adenocarcinoma, and is bidirectionally differentiated into the fundic gland and the pit epithelial cells. In this case report of GA-FGM, we found that both histomorphology and immunohistochemical staining showed that the very small area of the mucosal surface covered the tumorous epithelium and differentiated into the gastric pit epithelial cells. The cancer tissue located deep in the mucosa was dominated by gastric fundus adenocarcinoma cells, and two types of cancer cells, namely pit epithelium type and gastric fundic glandular type, were observed in the same tumorous gland.

Since its first discovery in 2015 [6] along with the cases reported by us, 11 more have been reported in the literature (Table 2) [6, 11-13]. GA-FGM is more common in older men with an average age of 67.2 years and a median age of 67 years (range 52-87 years). The male-to-female ratio is 2.7:1 (8:3). Most GA-FGM cases were diagnosed in the upper part of the stomach, followed by the middle part of the stomach. The average tumor diameter is 7.7 mm, and the median diameter is 6 mm (range 3-23 mm). According to the Paris classification, the most common type is 0-IIa type (6 cases), followed by 0-IIc type (2 cases) or 0-IIa+IIc type (2 cases); 0-I type is rare (1 case). In approximately 90% of cases, the tumor infiltrated into the submucosa layer, indicating that the tumor has a high tendency to infiltrate into the submucosa, which is similar to that of GA-FG [2-5, 7-9]. GA-FGM is one of the representative tumors of

## Gastric adenocarcinoma of fundic gland mucosa type



**Figure 4.** GA-FGM. The cyan line indicates gastric pit epithelial cells covered with a neoplastic atypical nucleus (A), and Immunohistochemical staining of the tumor epithelial cell MUC5AC of the corresponding area strong positive (B), MUC6 negative (C), pepsinogen I negative (D), and H<sup>+</sup>/K<sup>+</sup>-ATPase positive (E), Ki-67 proliferative activity is extremely low (F). The patient's information on disease diagnosis and prognosis are included in the reconstructed images of ESD specimens (G). A deeply stained area (in the cyan box) and a slight depression in the preoperative gastric biopsy section are shown, with further magnified GA-FGM tissue, including the surface covering the epithelium (illustration) (H). HE staining (A, H).

Hp-negative gastric cancer, and it also occurs in Hp-positive gastric fundic mucosa (Table 2). GA-FGM is likely to be accidentally found during endoscopy examinations [6, 11, 12].

In this case, endoscopy revealed a clear tumor boundary, and the characteristic of the irregular microvascular and microsurface pattern [14], suggesting that it was a differentiated type of gastric cancer. Because most of the mucosal surface of the lesion was covered with normal epithelium, the lesion exhibited a fading color, which was similar to the submucosal tumor-like appearance of a carcinoid [15]. When the tumor was considered as lymphoma or undifferentiated carcinoma, it tended to infiltrate and destroy the epithelium of the glandu-

lar fossa. The white area was not distinguishable. In contrast, in the present case report, the glandular structure of GA-FGM remained intact. However, normal pit epithelial cells were replaced by tumor cells, and the white area was identifiable. In extremely rare cases, GA-FGM could occur in the submucosal ectopic glandular gland tissue, and the intrinsic mucosal tissue above the tumor was free of tumors [13]. Occasionally, GA-FGM could also occur with regular adenocarcinomas [12].

Cancer cells usually have mildly polymorphic or atypical nuclei with loss of nuclear polarity. The nucleus shows division and even atypical mitosis with obvious atypia in the glandular structure. Tumor infiltration destroyed mucosal muscles and invaded the submucosa, which could help to diagnose GA-FGM and distinguish it from benign gastric glandular polyps. Gastric pit epithelial cells were observed in the fundic glandular polyps, and sometimes, ectopic hyperplasia of the gastric pit epithelial cells was also observed. Currently, there are reports of gastric pit epithelial-type differentiated adenocarci-

noma in sporadic fundic glandular polyps [16]. It is worth noting that there were no abnormalities in the glandular cells of the stomach. In addition to differentiation into gastric fundic cells, GA-FGM differentiated into pit-epithelial-like cells and covered the mucosal surface. Immunohistochemical staining could help to differentiate GA-FGM from GA-FG [2-5, 7-10].

In this case, eosinophilic tumors expressed MUC6, pepsinogen I, and H<sup>+</sup>/K<sup>+</sup>-ATPase, suggesting that these tumors differentiated into mucous neck cells/chief cells and parietal cells (differentiation of fundic gland). Tumor cells covering the mucosal surface and deep pit-epithelial-like cells expressed MUC5AC, indicating that these were gastric pit epithelial type tumor

## Gastric adenocarcinoma of fundic gland mucosa type

**Table 2.** Reported cases of GA-FGM

Study (y)	Patient	Age (y)/ Sex	Location	Tumor size (mm)	Macroscopic type	Invasive depth	<i>H. pylori</i> infection	Treatment	IHC	Ki-67 LI
Tanabe (2015) [6]	1	54/male	U	6	0-IIa+IIc	pT1b	NA	ESD	Pep>MUC6	5%
	2	60/male	U	9	0-IIa+IIc	pT1b	Negative	ESD	Pep=MUC6	<1%
	3	66/male	U	6	0-IIc	pT1b	Negative	ESD	Pep=MUC6	5%
	4	67/female	U	6	0-IIa	pT1b	NA	ESD	Pep=MUC6	2%
	5	67/female	U	7	0-IIa	pT1b	Negative	ESD	Pep<MUC6	10%
	6	74/male	U	5	0-IIa	pT1b	NA	ESD	Pep<MUC6	2%
Fujiwara (2015) [11]	7	68/female	U	4	0-IIa	pT1b	Negative	ESD	NA	NA
	8	74/male	U	7	0-IIa	pT1b	Negative	ESD	NA	NA
Takahashi (2017) [12]	9	87/male	M	3	0-I	pT1a	Positive	ESD	Pep=MUC6	NA
Uchida (2018) [13]	10	70/male	M	23	0-IIa	pT1b	NA	ESD	Pep<MUC6	14%
Our case (2018)	11	52/male	U	9	0-IIc	pT1b	Negative	ESD	Pep>MUC6	3%

U upper stomach, M middle stomach, NA not available, Pep pepsinogen I, Ki-67 LI Ki-67 labeling index, ESD endoscopic submucosal dissection, IHC Immunohistochemical, Pep pepsinogen I.

cells. GA-FG lacks this immunophenotypic characteristic [2-5, 7-9]. The labeling index of Ki-67 was mostly less than 5%, occasionally up to 14% (Table 2).

Because of the small atypia of tumor cells and the similarity of histomorphology and immunophenotype to normal gastric mucosa, GA-FGM is easily misdiagnosed as non-neoplastic lesions, especially, in the diagnosis of biopsy specimens. Therefore, glands with mild atypical cells should be observed for architectural atypia and invasive behavior. This is the main point of GA-FGM diagnosis. Because only a few tumors are exposed to the mucosal surface and differentiate into pit epithelium, so experienced pathologists may misdiagnose GA-FG. In order to prevent misdiagnosis of this kind of lesion, it is necessary to master its endoscopic and clinicopathological features. We provide a good learning case. The pathogenesis of GA-FGM is still unclear. Further study is needed to investigate whether the overexpression of cyclin D1 protein in our case could be related to the occurrence and development of tumors in the further time period.

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### Disclosure of conflict of interest

None.

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