

# Case Report the Neoplastic Transformation of Gastric Hyperplastic Polyp in a Context of Autoimmune Gastritis: Improving the Diagnosis

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## Case Report

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## Abstract

**Background:** Gastric hyperplastic polyp commonly arises in the abnormal background mucosa, including autoimmune gastritis, also known as type A gastritis. There are no obvious clinical symptoms in the early stages of the disease, which makes it easy to be misdiagnosed and missed, and has a potential risk of malignant transformation over time.

**Case presentation:** In 2020, a 67-year-old woman was admitted for review 6 years after gastric adenoma resection. Upper endoscopy revealed multiple lobulated polyps in the gastric body, which was histologically diagnosed as gastric adenomatous polyp as before. Interestingly, we discovered that the patient had undergone multiple polypectomies at the same part, which was inconsistent with the pathogenesis of adenomatous polyp. Then we did histological examination and revealed that partial epithelial hyperplasia and dysplasia, and the neoplastic areas were interlaced with normal mucosa. We further found that the background diagnosis was autoimmune gastritis. These results supported the diagnosis of neoplastic transformation of gastric hyperplastic polyp, not gastric adenomatous polyp. With the doubt of possible be misdiagnosed, we reviewed the medical history. Retrospective analysis in 2014, 2015 and 2016 confirmed the presence of autoimmune gastritis. Unfortunately, serological tests were not performed and no special treatment was done. Fortunately, the correct diagnosis was eventually confirmed as neoplastic transformation of gastric hyperplastic polyp in a context of autoimmune gastritis in 2020, which enables patients to receive normal treatment and monitoring, and avoids further deterioration of the disease.

**Conclusions:** The purpose of this case report is to increase clinical awareness of neoplastic transformation of gastric hyperplastic polyp in a context of autoimmune gastritis, and hope promise for early diagnosis and treatment.

## Introduction

Gastric hyperplastic polyp (GHP) is one of the most common polyps encountered in the stomach, especially in the antrum, and detected in 1.9% of 110,000 patients subjected to gastroscop [1]. GHP occurs in all ages, but the high incidence age is 65–70 years old. Of which, 58%-70.5% of patients with GHP are female [2], strongly indicating a gender difference in the occurrence and development of GHP. Morphologically, GHP ranges in size from a few millimeters to a few centimeters [3], leading to be misdiagnosed as tumor or cancer under endoscopy. Some of patients with GHP may present with dyspepsia, heartburn, abdominal pain, or anemia, but most of them are usually asymptomatic [4], which makes it difficult to be diagnosed in advance. GHP was considered to be a non-tumor lesion previously [5]. However, an increasing number of studies [6,7] have shown that GHP could be complicated with dysplasia and canceration, especially for polyps larger than 2 cm in diameter have a higher risk of neoplastic transformation.

In general, GHP occurs in the abnormal background mucosa, particularly in autoimmune gastritis (AIG), also known as type A gastritis [8]. In the context of AIG, GHP is often multiple, large, pedunculate or subpedunculate, and easy to produce neoplastic transformation [3]. Zhang et al. [9] reported that the incidence of neoplastic transformation of GHP in AIG is approximately 2.8%. In this paper, we report a case of gastric hyperplastic polyp in a context of autoimmune gastritis misdiagnosed as adenomatous polyp. Fortunately, the correct diagnosis was eventually confirmed by comprehensive analysis of gastroscopy, pathology and serology. Therefore, when patients recurrent GHP in the body of gastric with a history of anemia, we should be alert to the possibility of AIG, especially repeated recurrence in the same position after polyp resection, formulate a reasonable gastroscopy and follow-up program to achieve early diagnosis and treatment, to avoid complications and neoplastic transformation.

## Case Presentation

In August 2020, a 67-year-old Chinese woman was admitted to our hospital for review 6 years after gastric adenoma resection. Her symptoms included fatigue, poor appetite and lost 4kg in a year, and she was accompanied by a six-year history of iron deficiency anemia, which has been taking orally with iron and folic acid. The results from blood tests performed showed an iron deficiency anemia and decreased hemoglobin. No abnormalities were found in the Female Tumor Markers or Gastrointestinal Tumor Markers (Table 1). Upper endoscopy revealed multiple lobulated polyps with a size of 0.3-2.0cm in the gastric body (Fig. 1A). Subsequently, electrotomy, electrocautery and endoscopic hemostasis were performed (Fig. 1B), and the diameter of the removed polyp specimen was 0.3-1.2cm, which was gray and soft. The biopsy specimen obtained from the polyp was histologically diagnosed as gastric adenomatous polyp as before.

Interestingly, we discovered that the patient had undergone multiple polypectomies in the same part of the corpus, and was accompanied with anemia all the time. Therefore, adenomatous polyp did not seem to be able to explain the disease completely. Then we did histological examination and revealed that partial epithelial hyperplasia and dysplasia, and the neoplastic areas were interlaced with normal mucosa, as indicated by positive Ki-67 staining in the whole layer of tumor area and a decrease in mucus-secreting goblet cells in the surface epithelium. (Fig. 1C-1F). These results supported the diagnosis of neoplastic transformation of gastric hyperplastic polyp, not gastric adenomatous polyp.

Through further clinical and pathological collaboration, we further found that the normal mucosa around polyps showed atrophy of inherent glands in gastric mucosa with intestinal metaplasia and pyloric gland metaplasia (Fig. 1G, 1H), and linear and nodular hyperplasia of ECL cells based on chromogranin A and synaptophysin staining (Fig. 1I, 1J). Meanwhile, the anti-parietal cell antibody was positive (Table 1). Collectively, these data demonstrated that the background diagnosis was AIG.

The patient had multiple surgeries with a long history of anemia, the onset of AIG, so what was the relationship between neoplastic polyps and AIG? Is there any possibility of misdiagnosis in this patient before? To address these questions, we reviewed the medical history.

Table 1  
Laboratory data in August 2020

Laboratory findings	Value	Unit
White blood cells	4.07	$\times 10^9/\mu\text{l}$
Red blood cells	4.2	$\times 10^6/\mu\text{l}$
Mean corpuscular volume	83.3	fL
Hemoglobin	112 ↓	g/dL
Hematocrit	35	%
Platelets	135	$\times 10^4/\mu\text{l}$
Serum iron	7.23 ↓	$\mu\text{mol/L}$
Total iron binding capacity	68	$\mu\text{mol/L}$
Ferritin	13.3	ng/ml
CA724	< 1.500	U/ml
CEA	2.22	ng/ml
AFP	3.630	ng/ml
Anti-parietal cell antibody	(+)	
↓ The patient's value was below normal.		

In November 2014, the first visit of this patient was due to a physical examination. The results from positron emission tomography-computed tomography revealed gastric space occupation, as demonstrated by the vegetable pattern mass of the gastric wall on the greater curvature of the gastric body protrudes into the gastric cavity, and metabolism increases. Therefore, neoplastic lesions were considered. Upper endoscopy showed that there were  $1.5 \times 2.0\text{cm}$  polyps in lobulated shape on the greater curvature of the gastric body, with scattered erosion on the surface (Fig. 2A, 2B). The pathological results showed that partial epithelial hyperplasia, and dysplasia, the tumor lesions were strongly positive for Ki-67 and PAS staining showed mucus secretion reduced, (gastric body) tubular adenomatous polyps were considered (Fig. 2C-2F). The results of blood tests listed in Table 2 showed no obvious abnormality. Collectively, these results seemed to point to the diagnosis of adenomatous polyp.

However, what is the truth? Retrospective analysis of the sections of the original fundus glandular gastric mucosa showed fewer inherent glands and pyloric gland metaplasia (Fig. 2G, 2H). Supplementary immunohistochemical staining confirmed the presence of specific linear and nodular hyperplasia of ECL cells in the gastric mucosa around adenomatous polyps, which was typical morphological evidence for AIG. (Fig. 2I, 2J). Unfortunately, serological tests were not performed at that moment. Therefore, no special treatment was done except that the follow-up was informed six months later.

Table 2  
Laboratory data in November 2014

Laboratory findings	Value	Unit
White blood cells	3.11	$\times 10^9/\mu\text{l}$
Red blood cells	4.2	$\times 10^6/\mu\text{l}$
Mean corpuscular volume	89.3	fL
Hemoglobin	118	g/dL
Hematocrit	37.5	%
Platelets	169	$\times 10^4/\mu\text{l}$
Serum iron	8.50	$\mu\text{mol/L}$
Total iron binding capacity	48	$\mu\text{mol/L}$
Ferritin	17.1	ng/ml
CA724	1.340	U/ml
CEA	4.01	ng/ml
AFP	3.860	ng/ml

In July 2015, upper endoscopy was repeated, and the results revealed that there were several titanium clips fixed on the mucosa in 2014, and an arty polypoid ridge with a size of about 0.4\*0.4cm in the same place. The mucosa around the surface of the hyperplasia was rough and less smooth, and polyp electrotomy was performed (Fig. 3A, 3B). Retrospective analysis of the biopsy pathology showed gastric hyperplastic polyp with neoplastic change in part of epithelium and nodular hyperplasia of a few ECL cells in surrounding background mucosa (Fig. 3C-3E). It suggested that the patient may have autoimmune gastritis, despite the absence of serological tests and the recurrent polyp may have been misdiagnosed as an adenomatous polyp.

In April 2016, the patient underwent upper endoscopy again and discovered the original titanium clip of the gastric body remained with small polyps around it, as shown in Fig. 4. Later, removed together with the titanium clip. After that, the patient felt that his symptoms were well and did not undergo electronic gastroscopy review. which is a very regrettable for the health of patient.

Therefore, the patient developed recurrent hyperplastic polyps with neoplastic changes on the same gastric mucosa, but which was misdiagnosed as adenomatous polyp due to the neglect of the background of autoimmune gastritis. The whole clinical process and timeline of this patient is exhibited in Fig. 5. Fortunately, the correct diagnosis was eventually confirmed by comprehensive analysis of gastroscopy, pathology and serology. Of course, many important information has also been ignored. For example, the patient was elderly female, the onset site was gastric corpus and polyps recurred in a short

period after repeated resection, which were consistent with the epidemiological characteristics of AIG. Therefore, when patients recurrent GHP in the body of gastric with a history of anemia, we should be alert to the possibility of AIG, especially repeated recurrence in the same position after polyp resection.

## Discussion

GHP was considered to be a non-tumor lesion previously. An increasing number of studies have shown that GHP can be complicated with dysplasia and canceration, especially for polyps larger than 2 cm in diameter, the higher risk of neoplastic transformation. The reported average probability of neoplastic change of GHP ranges from 0.6 to 4.5% [10]. Therefore, early diagnosis of GHP is of great clinical significance. At the present, pathologic diagnostic criteria of Nakamura for GHP with malignant transformation are as follows [11]: 1) the coexistence of benign and malignant lesions in the same polyp, 2) existence of sufficient evidence that malignant lesion was previously a benign polyp, 3) sufficient cellular and structural dysplasia in the malignant area. Moreover, Ki-67 immunostaining and PAS staining also play an important role in the early diagnosis of neoplastic transformation of GHP. The expression of Ki-67 is able to assist in judging the development of tumor cells which can be used as a common indicator of cell proliferation activity. Shibahara et al. [12] suggested that examinations of Ki67 were important markers of malignant transformation of GHP, they conducted a study of GHP with malignant transformation and Ki-67 immunostaining showed positive in the carcinoma and dysplastic cells. PAS staining showed that mucin was diffusely positive in the epithelial surface cell nuclear and perinuclear membrane of normal gastric mucosa, some were homogeneous, some were diffusely stained. In contrast, the decrease or disappearance of PAS staining indicates the injury of normal mucosal epithelial cells / neoplastic lesions. Therefore, GHP has the possibility of malignant transformation and is easy to be misdiagnosed. Various methods need to be adopted for comprehensive diagnosis.

An increasing number of studies have shown that AIG can promote the neoplastic transformation of GHP [7, 11]. In general, GHP occurs in the abnormal background mucosa, particularly in AIG. Stimulated by high levels of gastrin, the association between GHP and AIG is well verified [13] and the data demonstrated that 12% GHP related to AIG [14]. The development of cancer is deemed to a multi-step process through hyperplasia-dysplasia-carcinoma sequence [15], and AIG confers an increased risk of intestinal-type gastric cancer [16]. AIG is a chronic progressive inflammatory mediated by CD4 + T lymphocytes caused by autoantibodies attacking parietal cell, which can lead to atrophy and metaplasia of secretory acid glands in the corpus-fundus gastric mucosa, and involve digestive, blood, nerve and other systems. The high incidence of AIG occurs in older people, female and those with a history of autoimmune diseases. Moreover, Hershko et al. [17] reported that microcytic anemia which was present in 50 % of patients with AIG. As the elapse of time, vitamin B12 is absorbed obstacle, and lead to megaloblastic anemia, even the effect of oral medicaments cannot be controlled and produce pernicious anemia. In AIG patients, gastric acid is reduced or even deficient due to atrophy of acid-secreting glands, resulting in reduced secretion of somatostatin, which is not sufficient to inhibit the secretion of gastrin by G cells, arising hypergastrinemia in response, causing hyperplasia of ECL in acid-secreting mucosa, occurring dysplasia as well as carcinoma.

Nevertheless, GHP is still easy to be missed or misdiagnosed as adenomatous polyp. Gastric adenomatous polyps are defined by the World Health Organization as localized polypoid lesions consisting of tubular and/or villous structures lined with dysplastic epithelium [18]. The majority of adenomatous polyps are found in the antrum and angle, followed by gastric fundus, which are solitary, exophytic sessile or pedunculated mass in endoscopic finding [5]. It is difficult to distinguish the pattern pathologically with malignant transformation of GHP in AIG. The largest difference between the two situations is prognosis: unlike GHP, which is difficult to resect and recurrent [19], adenomatous polyp resection has a benign prognosis, low recurrence rate and long follow-up period after complete resection. Due to the limitation of the operation, there is no need to assist any medical treatment after the operation.

In our patient, the patient developed recurrent hyperplastic polyps with neoplastic changes on the same gastric mucosa. The histological findings showed linear or nodular hyperplasia of enterochromaffin-like cell in the glandular of gastric mucosa, simultaneously, and the polyp morphology still maintained the morphological basis of GHP, gastric pits hyperplasia, elongation and expansion. However, some epithelial cells showed dysplasia, spreading from the proliferation zone to the surface. Ki-67 and PAS staining also supported the morphological interpretation results. All these results showed neoplastic transformation of GHP in a context of autoimmune gastritis. Unfortunately, we were not aware of possibility of AIG in our earlier work and draw the wrong conclusion.

From this case, we summarize some certain experiences. First of all, gastric polyp lesions ought to be considered according to different sites of distinct disease spectrum. Secondly, staying on highly vigilant when gastric antrum mucosa is normal, on the contrary existing abnormal or atrophic in gastric corpus mucosa, especially pay attention to track the basic information from patients, such as, age, gender and history of autoimmune diseases. Finally, during the endoscopy, the endoscopist ought to notice the mucosal status around polyps and gastric antrum mucosa and take samples not only from the biopsy site recommended by the Sydney system (five biopsy samples should be obtained: two from the corpus, two from the antrum and one from the incisura angularis) [20], but also take samples from the mucosa adjacent to the polyp. It may reduce missed diagnosis and misdiagnosis, particularly when the workload is heavy. Simultaneously, pathologists are supposed to focus on clinical information, cooperate closely with clinicians, discover diagnostic clues actively and follow up serological tests to assist diagnosis. Endoscopic monitoring should be considered in all patients with AIG, especially elderly patients [21]. Once polyps are found in patients, in order to avoid the risk of cancer, endoscopic polypectomy should be considered in these GHP actively. To sum up, in order to make patients benefit more, clinicians, pathologists and endoscopist can study together and cooperate closely.

## Abbreviations

GHP

Gastric hyperplastic polyp

AIG

Autoimmune gastritis

# Declarations

## Acknowledgements

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## Authors' contributions

YX collected patient information, analyzed results and wrote the first draft of the manuscript. HH collected information and performed the research. YW had pathological staining. ZS, LW, DP, XG, NY, WZ, YY did the upper gastrointestinal examination. TM supervised and interpreted the experimental data, critically revised the manuscript. YL designed the research, analyzed the data, and revised the manuscript critically. All authors read and approved the final manuscript.

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## Availability of data and materials

The original contributions presented in the study are included in the article, further inquiries can be directed to the corresponding authors.

## Ethics approval and consent to participate

Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

## Consent for publication

*Written informed consent was obtained from the patient for publication of this case report and accompanying images.*

## Competing interests

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

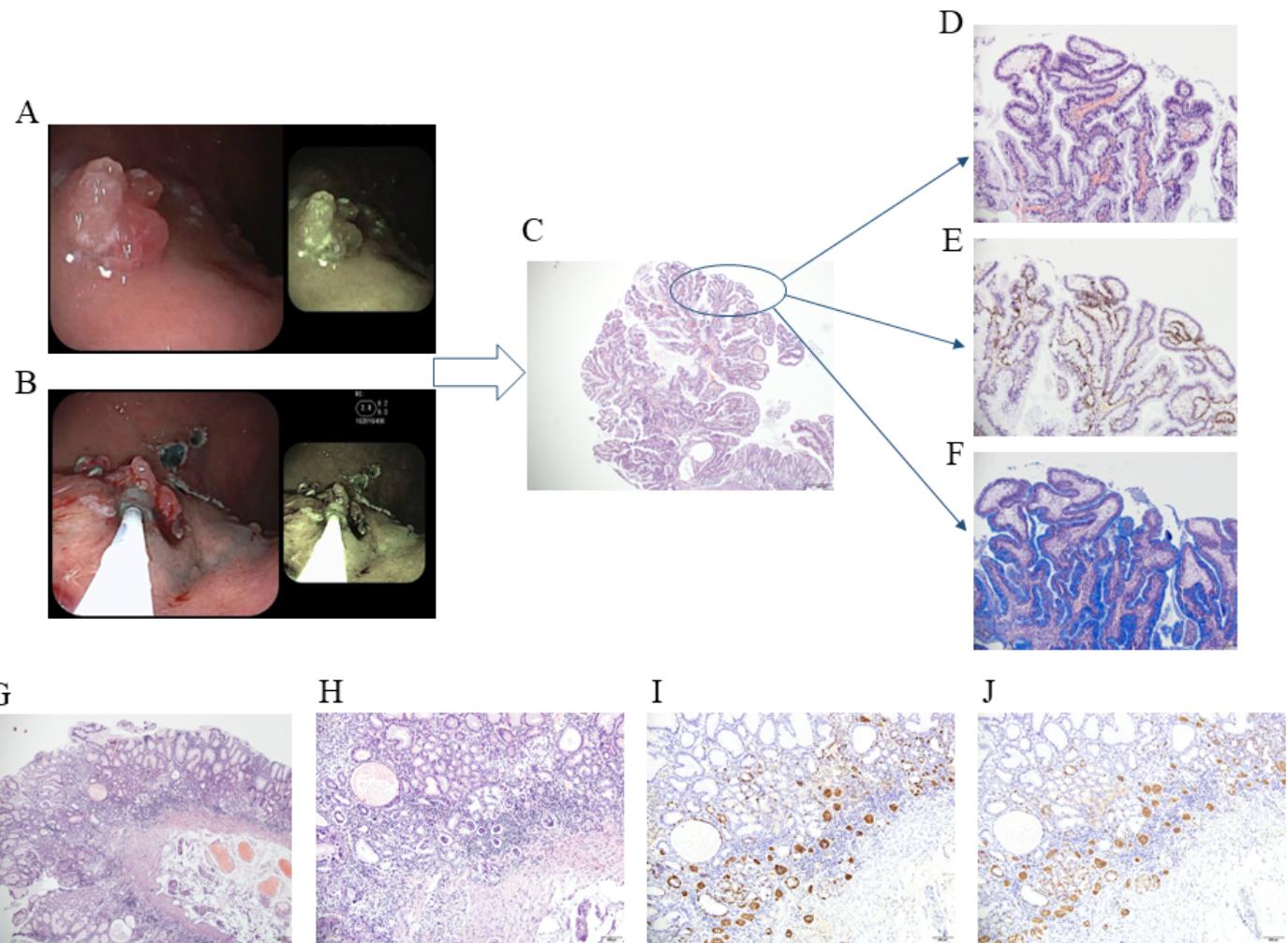
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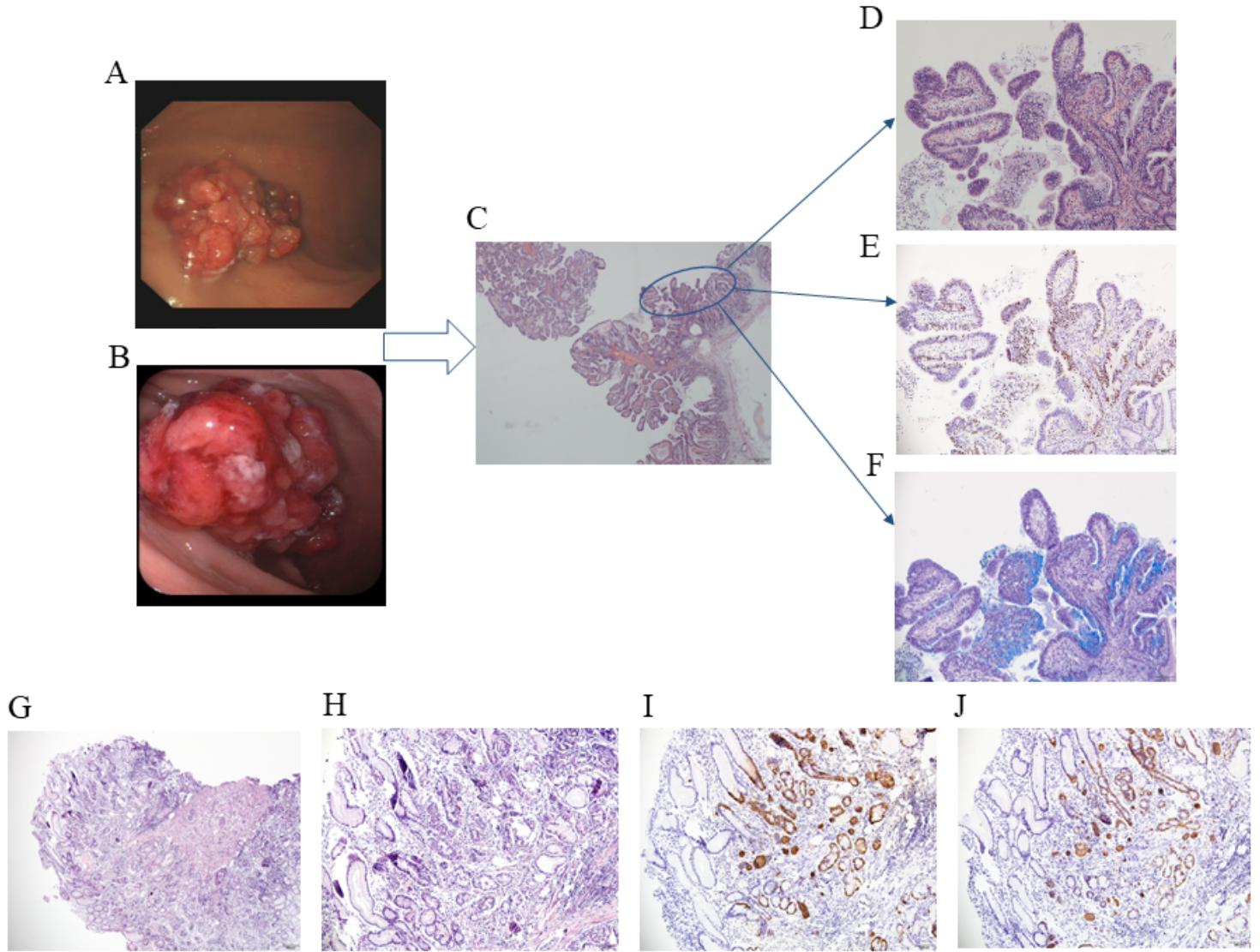
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## Figures



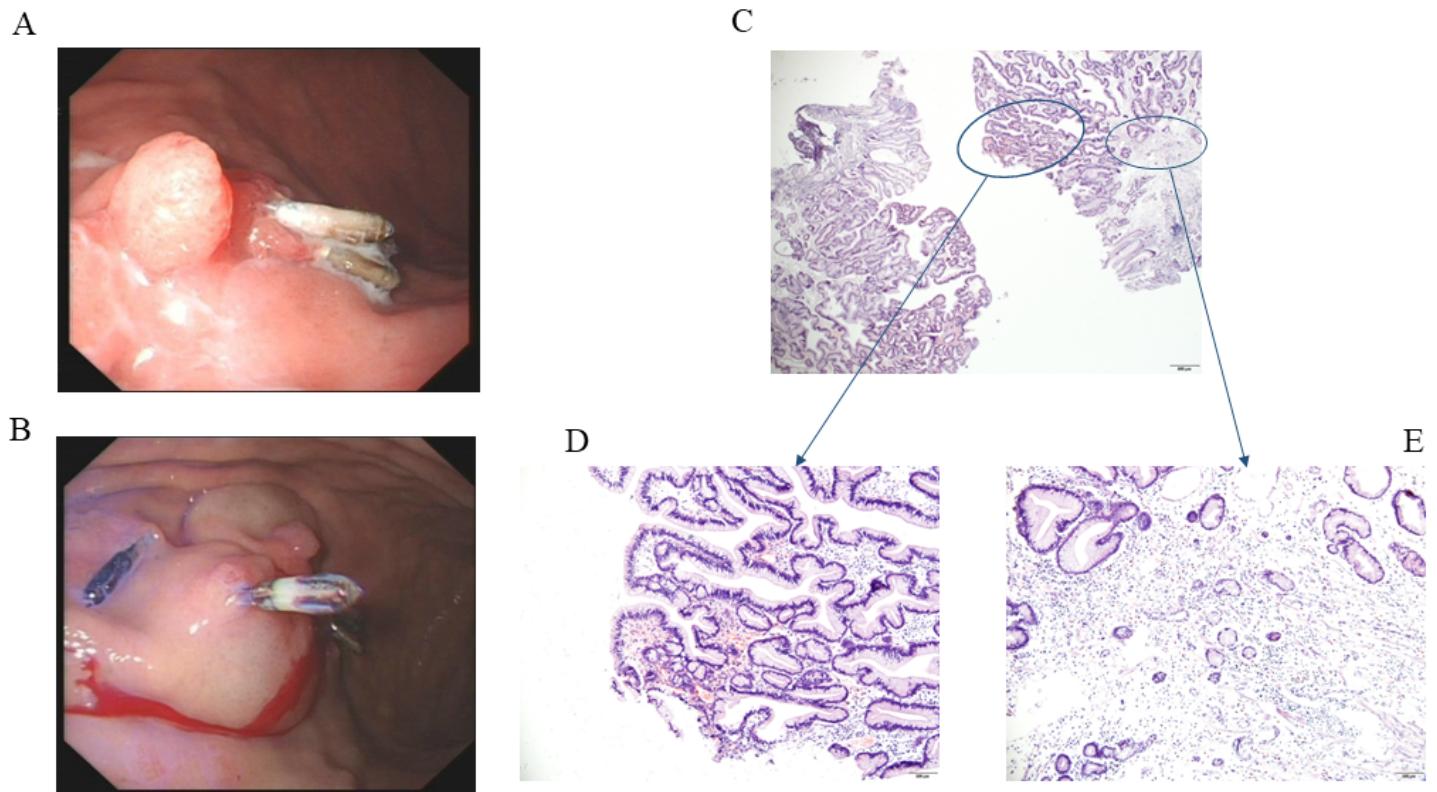
**Figure 1**

Endoscopic, histological images and examination of the background mucosa in 2020. (A) Multiple lobulated polyps with a size of 0.3-2.0cm in the gastric body. (B) Endoscopic submucosal dissection. electrotomy + electrocautery and endoscopic hemostasis were performed for the pedunculated polyp on the gastric body. (C) Low magnification image of the boxed area by hematoxylin and eosin (HE) staining (20X). (D) Partial epithelial hyperplasia and atypical hyperplasia (100X). (E) The neoplastic areas were interlaced with normal mucosa, and Ki-67 was positive in the whole layer of tumor area (100X). (F) The area of tumor change is characterized by a decrease in mucus-secreting goblet cells in the surface epithelium (100X). (G) Low magnification image of the boxed area by HE staining (40X). (H) Atrophy of inherent glands in gastric mucosa with intestinal metaplasia and pyloric gland metaplasia(100X). (I and J) Linear and nodular hyperplasia of ECL cells was revealed by chromogranin A and synaptophysin staining(100X).



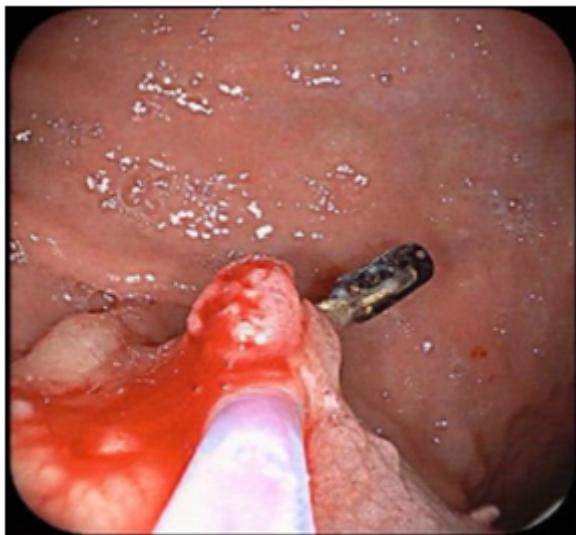
**Figure 2**

Endoscopic, histological findings and examination of the background mucosa in 2014. (A and B) 1.5×2.0cm polyps in lobulated shape on the greater curvature of the gastric body, with scattered erosion on the surface. (C) Low magnification image of the boxed area by HE staining (20X). (D) Gastric hyperplastic polyp with neoplastic change in part of epithelium (100X). (E) Ki-67 staining of the border between the hyperplastic glands and the tumor glands. The tumor lesions were strongly positive for Ki-67 ( $+>15$ ) (100X). (F) PAS staining showed a reduced mucus secretion in the tumor area (100X). (G) Low magnification image of the boxed area by HE staining (40X). (H) The original fundus glandular gastric mucosa by HE staining showed fewer inherent glands and pyloric gland metaplasia (100X). (I and J) Linear and nodular hyperplasia in ECL cells (100X).



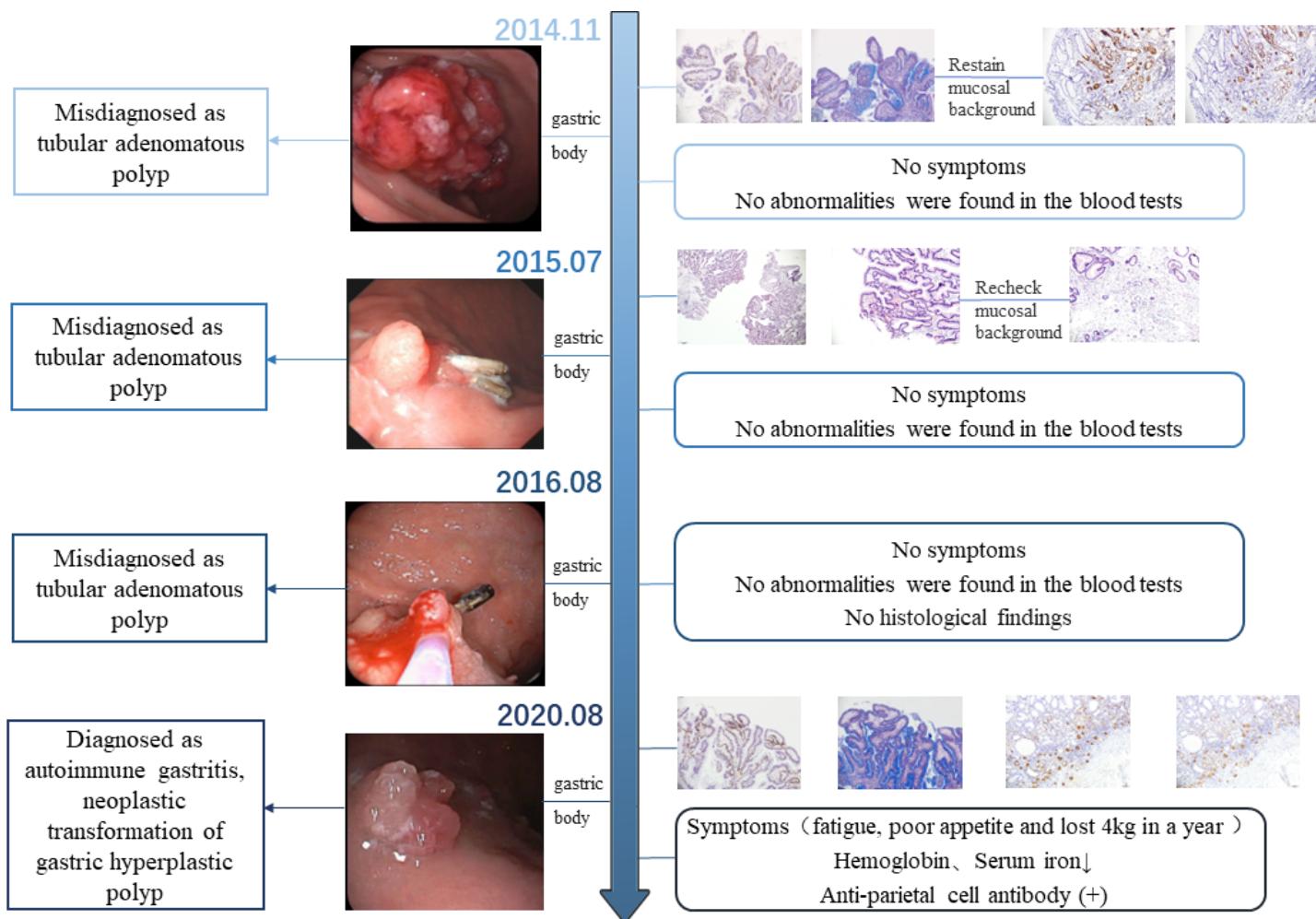
**Figure 3**

Endoscopic and histological findings in 2015. (A and B) an arty polypoid ridge with a size of about  $0.4 \times 0.4$  cm. (C) Low magnification image of the boxed area (20X). (D) Gastric hyperplastic polyp with neoplastic change in part of epithelium (100 X). (E) Nodular hyperplasia of a few ECL cells in surrounding background mucosa (100X).



**Figure 4**

Endoscopic findings. The original titanium clip of the gastric body remained with small polyps around it.



**Figure 5**

Case report timeline and process.