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CASE REPORT

Asymptomatic gastric adenomyoma and heterotopic pancreas in a patient with pancreatic cancer: A case report and review of the literature

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Author contributions: Shi BM contributed to the conception and design of the study and performed the operation on the patient; Li K collected the data, performed the data analysis, and wrote the initial draft of the manuscript; Yan X and Liu NB participated in the clinical management and follow-up of the patient and analyzed and interpretated the data; all authors contributed to drafting and revising the manuscript and approved the submitted version.

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Abstract

BACKGROUND

Gastric adenomyoma (GA) is a rare submucosal benign neoplasm that occurs mostly in the gastric antrum and is often misdiagnosed. No standard treatment has been established for this disease in cases of malignancy.

CASE SUMMARY

A 75-year-old woman with a 10-year history of hypertension was admitted to the Emergency Department of our hospital complaining of paroxysmal exacerbation of acute abdominal pain for 1 d with no apparent cause. Enhanced computed tomography and magnetic resonance imaging indicated a mass in the caudal pancreas, cholecystitis, and cholecystic polypus. Gastrointestinal endoscopy showed a mass arising from the gastric antrum. Due to the imaging findings, pancreatic cancer (PC), gastric lesion, cholecystitis, and cholecystic polypus were our primary consideration. Radical pancreatectomy, splenectomy, and cholecystectomy were performed successfully, and the gastric tumor was locally resected. Postoperative paraffin specimens confirmed the diagnosis of caudal PC, GA, and heterotopic pancreas (HP). Unfortunately, the patient died 13 mo later due to PC metastases to the liver, lung, and adrenal glands.

CONCLUSION

GA is a rare benign disease, especially when occurring with HP. It may stem from the same origin as HP. This is the first case report to date of a patient suffering from the simultaneous occurrence of GA, HP, and PC. GA is a lesion that can mimic other benign or malignant gastrointestinal diseases; thus, a definitive diagnosis depends on postoperative pathological biopsy. Although GA and HP are both benign lesions, they should be resected because there is a chance of malignancy. Additional research should be conducted to better understand these submucosal lesions.

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Core Tip: Gastric adenomyoma (GA) is a rare gastric submucosal disease that is even rarer when combined with heterotopic pancreas (HP) or pancreatic cancer (PC). HP and GA may have the same origin. This is the first case report of the simultaneous occurrence of GA, HP, and PC. In addition, 24 patients with HP and GA were investigated to identify variations among the cases and clarify the importance of surgical resection. GA can mimic other benign or malignant gastrointestinal diseases; thus, a definitive diagnosis depends on postoperative paraffin biopsy. Surgery is a vital procedure to cure GA and HP in cases of malignant transformation.

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INTRODUCTION

Heterotopic pancreas (HP), also known as aberrant pancreas and accessory pancreas, is any isolated pancreatic tissue that grows outside the pancreas itself and has no anatomical connection with the normal pancreas[1]. Gastric adenomyoma (GA), also known as myoepithelial hamartoma, myoglandular hamartoma, adenomyomatous hamartoma, and adenomyosis in the stomach[2], is a rare benign tumor occurring in the submucosal layer of the gastric wall. Over 100 years after the discovery of HP in 1727 by Jean-Schultz[1], GA was first reported by Magnus-Alsleben[3] in 1903, who described it as a lesion that consists of smooth muscle fibers and circular crescent-shaped lumina that are lined with a single-layer of cylindrical epithelium. In 1909, a physician in Germany named Heinrich differentiated gastric HP into three types: Type I, with pancreatic acini and islets; type II, with acini; and type III, with only undifferentiated ducts[4]. He then named type III HP GA, with type III containing proliferated smooth muscle cells and a mucus-excreting epithelium.

GA has a low occurrence rate; only 52 cases have been reported as of 2016, according to Bhardwaj *et al*[5]. From 2016 on, only eight cases of GA confirmed by pathological biopsy have been reported. These cases were examined by tissue biopsy, which should contain both heterotopic pancreatic acini in the gastric wall and several undifferentiated mucus-secreting ducts lined with columnar or cubic epithelial cells surrounded by overgrown bundles of smooth muscle cells in the outermost layer. There are only 24 cases (including ours) of simultaneous HP and GA confirmed by pathological results reported in the literature. There have been no pathological biopsy reports revealing a combination of HP, GA, and pancreatic cancer (PC).

CASE PRESENTATION

Chief complaints

A 75-year-old woman with a diagnosis of pancreatic mass was admitted for surgery.

History of present illness

A 75-year-old Chinese woman was admitted because of paroxysmal exacerbation of acute abdominal pain for 1 d with no apparent cause. The pain was not responsive to changes in body position and did not radiate to the other side. The patient did not complain of nausea, sour regurgitation, or abdominal fullness but did note slight fatigue.

History of past illness

The patient had hypertension for the previous 10 years, which was well controlled by drug therapy.

Personal and family history

The patient had a more than 10-year history of hypertension that was under fairly satisfactory control *via* medication.

Physical examination

Abdominal physical examination showed a flattened belly, normal borborygmus, and epigastric tenderness and rebound pain, with a negative Murphy's sign.

Laboratory examinations

Blood biochemistry tests revealed the following: Elevated blood amylase (742 U/L; chromatometry reference range: 35-135 U/L), undetectable blood lipase (chromatometry reference range: less than 79 U/L), low albumin (32.2 g/L; reference range: 40-55 g/L), low calcium (2.06 mmol/L; reference range: 2.25-2.75 mmol/L), low platelets (98 × 10^9/L; reference range: 100-300 × 10^9/L), and highly elevated CA242 (over 150 IU/mL; reference range: 0-20 IU/mL), CA50 (289.6 IU/mL; reference range: 0-20 IU/mL), and CA199 (627.4 IU/mL; reference range: 0-37 IU/mL).

Imaging examinations

A plain CT scan showed a mass in the body and tail of the pancreas, with little exudation (Figure 1A). The morphology of the pancreas was disrupted, indicating edema, hyperemia, and fat liquefaction. The stomach appeared normal (Figure 1B). Two days later, contrast-enhanced CT showed a cystic solid mass surrounding splenic vessels in the caudal pancreas, indicating a pancreatic malignant tumor (Figure 2A). Multiple hepatic cysts, cholecystitis, and cholecystic polypus were also detected. The gastric antral wall was found to be thickened (Figure 2B). Gastric endoscopy was performed 3 d later. The images showed chronic superficial and atrophic gastritis, a protruding lesion in the submucosal area of the antrum (Figure 3), and no *Helicobacter pylori* infection. Contrast MRI was performed the following day and indicated PC (Figure 4A) and uniform thickening of the antral wall (Figure 4B). Due to the aforementioned imaging findings, we decided to perform operations to relieve PC and tried to figure out the nature of the gastric mass intraoperatively.

Pathological result postoperatively

The gastric mass under pathological examination post operation was a gray, solid, and tough nodule measuring 2 cm × 2.2 cm × 1 cm in the pylorus. Immunohistochemical staining showed that the tumor cells were positive for CK8, partially positive for Ki-67, and negative for CD147. Micrography showed multiple multifocal heterotopic pancreatic acini (Figure 5A), mucus-excreting glands such as Brunner's glands, and undifferentiated ducts surrounded by proliferative smooth muscles in the submucosal layer, which typically indicate GA with HP. The tissue in the other slice showed undifferentiated epithelial cells covered with thickened smooth muscle, indicating GA alone (Figure 5B), with no signs of malignancy. Regarding the 3.5 cm × 2 cm × 3 cm pancreatic mass, distinct dysplasia of the acini and cells arranged in a mass without polarity indicated cancer cells. The nuclei were deeply basophilic, with common karyokinesis, enlarged in size, and increased in number compared to those in normal tissue (Figure 6).

FINAL DIAGNOSIS

The patient was diagnosed with caudal PC, gastric HP, and GA, accompanied by acute pancreatitis, cholecystic polypus, and hypertension.

TREATMENT

The patient underwent radical pancreatectomy, splenectomy, and cholecystectomy, and the gastric tumor was locally resected. During the operation, a mass measuring 4 cm × 4 cm × 3 cm was excised from the tail of the pancreas, and the antral mass was

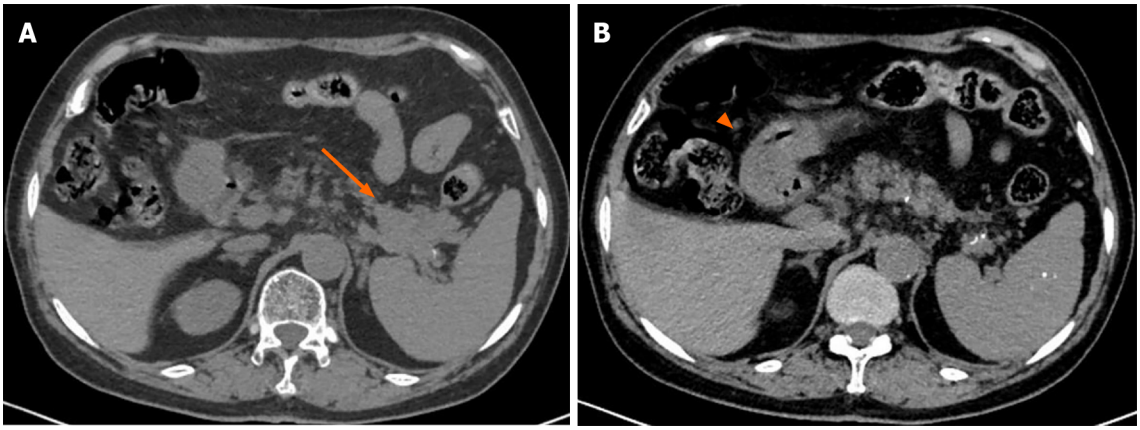

Figure 1 Upper abdominal computed tomography images. A: A lobulated solid mass (orange arrow) in the cauda pancreas invading the splenic pedicle; B: The gastric antrum is full of chyme, and the accurate condition of the gastric wall is not clear (orange arrowhead).

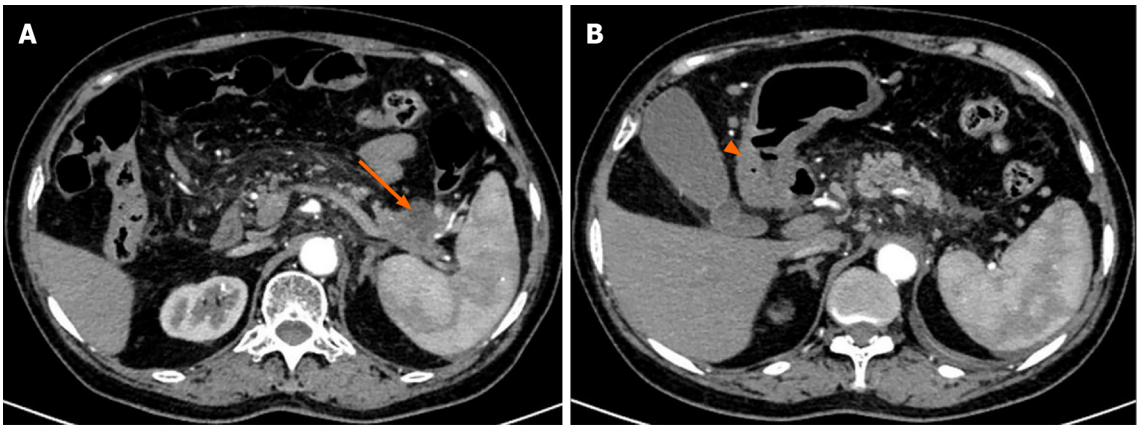

Figure 2 Enhanced upper abdominal computed tomography images. A: A cystic solid mass measuring 3.5 cm × 3 cm × 2 cm in the cauda pancreas (orange arrow) surrounding the splenic vessels; B: Thickening of the antral wall and slight obstruction of the pylorus (orange arrowhead).

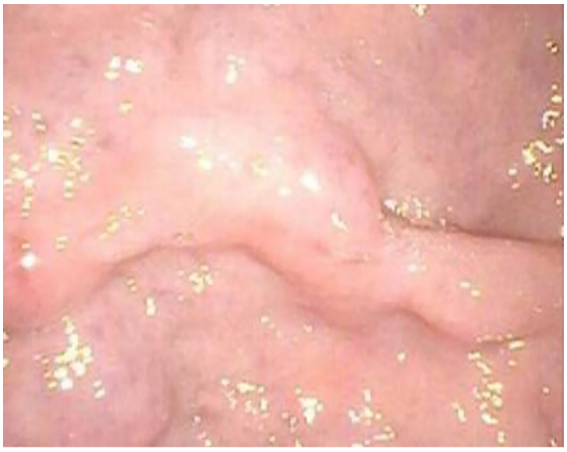

Figure 3 Gastric endoscopy revealed a submucosal lesion that arose from the surface of the pylorus.

excavated 1 cm away from its rim. Microscopy analysis revealed that the frozen biopsy sample was a benign lesion.

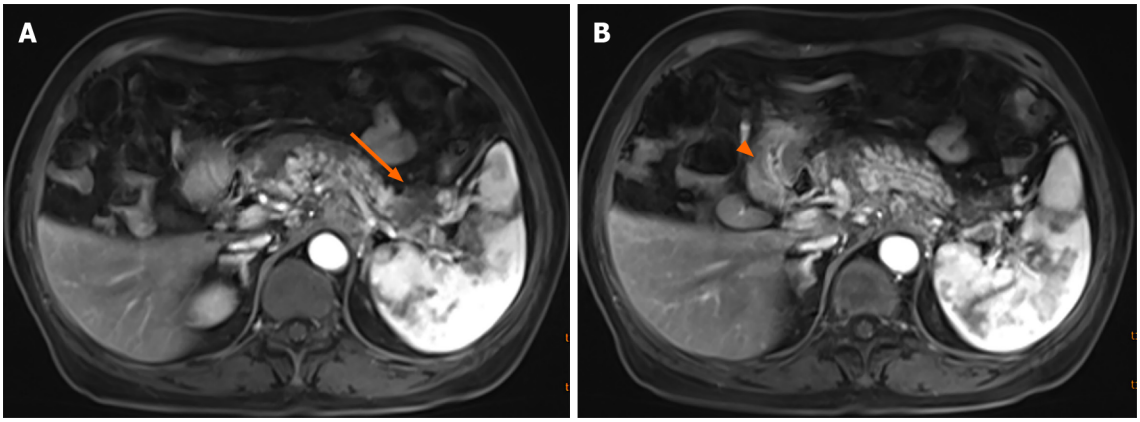

Figure 4 Enhanced upper abdominal magnetic resonance imaging. A: An intensified mass (orange arrow); B: Thickening of the pyloric wall (orange arrowhead).

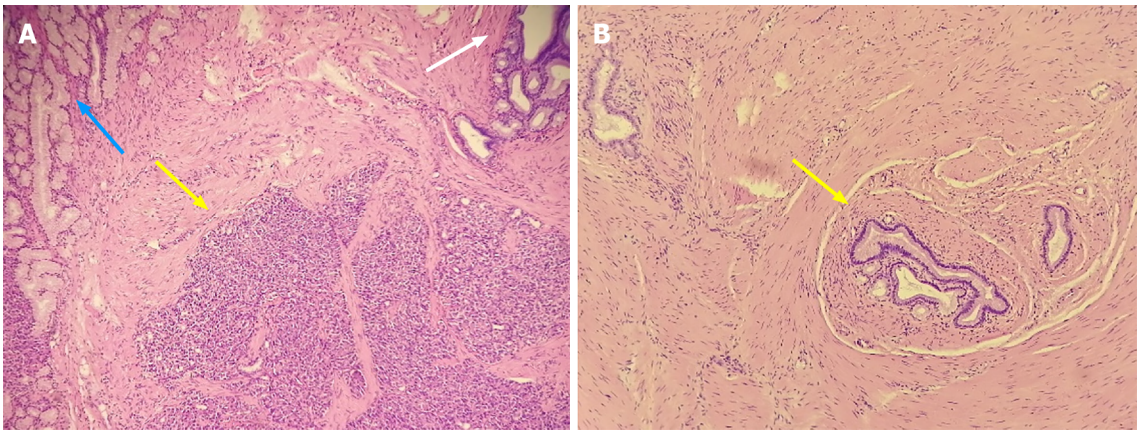

Figure 5 Histology (HE, 40 × magnification). A: Disorganized pancreatic acini joining together and forming rough structures without islets and separated by bundles of smooth muscle (yellow arrow); the concomitant Brunner's glands (blue arrow); and the undifferentiated mucus-secreting ducts, somewhat similar to gastric glands (white arrow); B: Typical gastric adenomyoma in another section of the gastric mass (yellow arrow) showing a mucus-secreting duct lined with columnar or cubic epithelial cells and surrounded by proliferating smooth muscle cells without heterotopic pancreas nearby.

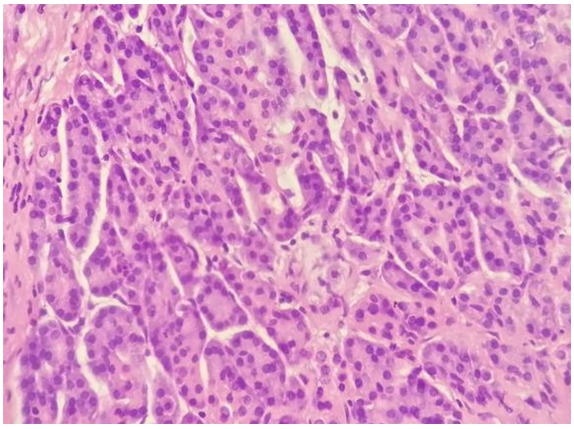

Figure 6 Histology (HE, 200 × magnification).

OUTCOME AND FOLLOW-UP

Postoperatively, the patient went through sequential chemotherapy and regular follow-ups. However, she unfortunately died 13 mo postoperatively due to PC metastases to the liver, lung, and adrenal glands (April 2017).

DISCUSSION

As described by Choi *et al*[6] in 2000, gastric submucosal tumors are uncommon lesions (< 2% of surgically resected gastric masses). The most common types are gastrointestinal stromal tumor (GIST), leiomyoma, and gastric lipoma[7]. GA is one of the rarest diseases among gastric submucosal tumors, and its definition is based on pathological classifications. As mentioned before, there have been only 60 cases of GA up to now. In 2017, Emerson *et al*[7] have reported that 17 of the 571 cases that could not be fully resected by endoscopic submucosal dissection were GA, while these data contained no pathological images. Even if these lesions are indeed GA, as well as those who did not undergo operations and thus had no definitive diagnosis, a consensus has been reached that GA is an uncommon disease[8-10]. GA has occurred in all age groups, from 7 d[8] to 84 years[9]. To some extent, its low occurrence rate is because the majority of these cases are asymptomatic[10-12]; however, some GA patients experience epigastric pain and discomfort, with/without nausea[13], vomiting[14], dyspepsia[15], and melena[16]. Concerning small children or infants, the first symptom may be nonbilious vomiting[8,13-14,17-19] or esophageal reflux[20]. Some GA cases are detected by autopsy[20]. Meaningful diagnosis methods include radiographical methods such as barium meal X-ray[21], in which a filling defect in the gastric antrum or pylorus region provides an imaging diagnosis[22,23], CT[4], ultrasound (US)[13], gastrointestinal endoscopy ultrasound[24], and MRI[17]. However, the only confirmatory test for GA is pathological biopsy.

Because of its rare occurrence, GA is typically not considered and can be easily misdiagnosed since it is similar to other benign or malignant gastrointestinal lesions, such as GIST[15,21,25-27], leiomyoma[5,28], gastric carcinoma[29], gastric adenocarcinoma[30], and pylorus stenosis[13-14,17], making the differential diagnosis challenging.

Although GA is indeed a rare submucosal disease, other diseases can accompany GA, such as HP. As previously mentioned, there have been only 20 known cases of the simultaneous occurrence of HP and GA. Among these cases (Table 1), no reports have identified any differences in the occurrence rate by sex, age, or district. Most of these patients experienced pain or discomfort, and only a few cases were detected by physical examination. Although most of the patients had benign lesions, two experienced malignant transformation. All of the biopsy tissues were collected from the pyloric antrum (either the lesser or greater curvature). There are several discussions about the source of both lesions, apart from Heinrich's 1909 definition.

As proposed by Erberich *et al*[2] in 2000, HP in the stomach is thought to be derived from the dorsal anlage. As shown in the pathological images, HP appears to be located on the surface of the polyp, accompanied by Brunner's glands and GA in the lower layers, as if the deep-seated lesion is triggered by exocrine pancreatic acini. However, even if HP secretes hormones and fluid, these substances can only be released into the gastric lumen, without affecting smooth muscle cells and undifferentiated mucus-secreting cells in the submucosa. In another position described by Takeyama *et al*[18] in 2007, GA was found to be a component of hamartoma without well-differentiated pancreatic tissue and an independent lesion rather than a part of HP. However, we found that GA can accompany HP, but it can also develop alone, as depicted in Figure 5. In summary, GA can be described as an independent lesion (*i.e.*, without HP) or with local inflammatory changes caused by HP.

Thus, we approve Heinrich's definition. GA and HP are, in fact, the same lesion. Differences in cytology, histology, and biological behavior exist because different parts of the primary gut remain *in situ* (Figure 7). In the fourth week of embryonic development, the pancreas derives from both the ventral and dorsal antrum[31], and some parts of the primary pancreatic cells that remain are affected by the gastric local microenvironment and develop into different tissues, including the original pancreatic glands, gastric glands, Brunner's glands, undifferentiated glands, and proliferating smooth muscle cells. Different tissues exist because of the different phases at which these cells leave from the original site and the different microenvironments. The proliferating smooth muscle and undifferentiated mucus-secreting epithelial cells converge, perhaps because they are in the same differentiation state. In a word, HP and GA stem from the same origin.

A standard treatment for GA and HP has not been established. In the 20 patients with GA and HP, except for those with cancerous lesions, the operative methods included endoscopic or laparoscopic mass resection, partial gastrectomy, or subtotal and total gastrectomy depending on the severity of the disease and the general condition of the patient. However, all lesions were successfully removed, and symptoms were relieved.

Table 1 Characteristics of patients with heterotopic pancreas and gastric adenomyoma

Ref.	Year	Age	Sex	Main complaint	Diagnostic method[1]	Clinical diagnosis[2]	Surgery
Bedir *et al*[10]	2018	26 yr	F	Occasionally detected[3]	Radiography	GA	Mini-gastric bypass; subtotal gastrectomy
Campbell[35]	1949	37 yr	F	Indigestion and discomfort	Radiography	Gastric benign tumor	Partial gastrectomy
Eisenberger *et al*[21]	2001	21 yr	F	Emesis, heartburn, mid/epigastric pain, and weight loss	Radiography endoscopy	Benign GIST	Mini-laparotomy; mass dissection
Emerson *et al*[7]	2004	52 yr	M	Epigastric and left upper quadrant pain, postprandial emesis, bloating, abdominal distention	Endoscopy	N/A[4]	50% gastrectomy with vagotomy
Faigel *et al*[36]	2001	34 yr	F	Dyspepsia and nausea	Radiography endoscopy	N/A	Endoscopic mucosal resection
Floros *et al*[37]	1982	29 yr	M	Intermittent epigastric pain	Radiography	N/A	Endoscopic wedge resection
Haubrich[16]	1955	60 yr	F	Melena	Radiography	Early malignant neoplasm	Subtotal gastrectomy
Kagawa *et al*[38]	2007	26 yr	F	Intermittent severe abdominal pain, high fever	Radiography endoscopy	Benign submucosal tumor	Laparoscopic wedge resection; pyloroplasty
Kamrani *et al*[39]	2019	15 yr	F	Nausea and vomiting	Radiography endoscopy	N/A	Distal gastrectomy with a gastroduodeno-stomy
Keshgegian *et al*[28]	1978	30 yr	F	Left upper quadrant pain	Radiography	Leiomyoma	Mass dissection
Kerkez *et al*[15]	2011	5 yr	F	Intermittent epigastric pain	Radiography	N/A	Distal gastrectomy, cyst excision with gastroduodenostomy
Nabi *et al*[26]	2012	35 yr	M	Intermittent epigastric pain and vomiting	Radiography endoscopy	GIST	Hemigastrectomy followed by gastrojejunal anastomosis
Portale *et al*[29]	2007	71 yr	M	Recurrent duodenal ulcer	Radiography endoscopy	Gastric carcinoma	Subtotal gastrectomy with BII reconstruction
Reardon *et al*[40]	1999	31 yr	F	Acute epigastric pain	Radiography endoscopy	Leiomyoma or a lipoma	Laparoscopic resection with an end-to-end gastroduodenostomy
Rhim *et al*[8]	2013	1 wk	M	Persistent vomiting	Radiography	N/A	Antrectomy with Billroth I anastomosis
Song *et al*[27]	2004	35 yr	M	Occasionally detected	Radiography endoscopy	GIST	Wedge resection
Zarling[41]	1981	25 yr	F	Epigastric discomfort and early satiety	Radiography endoscopy	N/A	Distal antrectomy
Vandelli *et al*[42]	1993	42 yr	F	Intermittent postprandial epigastric pain, nausea, and vomiting	Endoscopy	N/A	Polypectomy and Billroth II gastrectomy
Erberich *et al*[2]	2000	48 yr	M	Epigastric pain and vomiting	Radiography endoscopy	Gastric polyps	Local excision of both lesions
Janota *et al*[23]	1966	27 yr	M	Epigastric pain	N/A	Peptic ulcer	Partial gastrectomy; closure of the duodenal stump
Lasser *et al*[22]	1977	35 yr	M	Discomfort	Radiography	N/A	Subtotal gastrectomy
Stewart *et al*[20]	1984	81 yr	M	Renal failure, anemia, and melena	N/A	N/A	N/A
		53 yr	M	Discomfort	Radiography	N/A	Endoscopic resection

[1]Diagnostic methods include radiographic methods such as ultrasound, X-ray, computed tomography, and magnetic resonance imaging, as well as endoscopic methods such as gastroenteroscopy.
[2]Clinical diagnosis made before operation.
[3]Detected by regular body examination or other abdominal surgeries.
[4]No data. GA: Gastric adenomyoma; GIST: Gastrointestinal stromal tumor; N/A: Not applicable.

Currently, more precise methods aided by the innovation of surgical instruments,

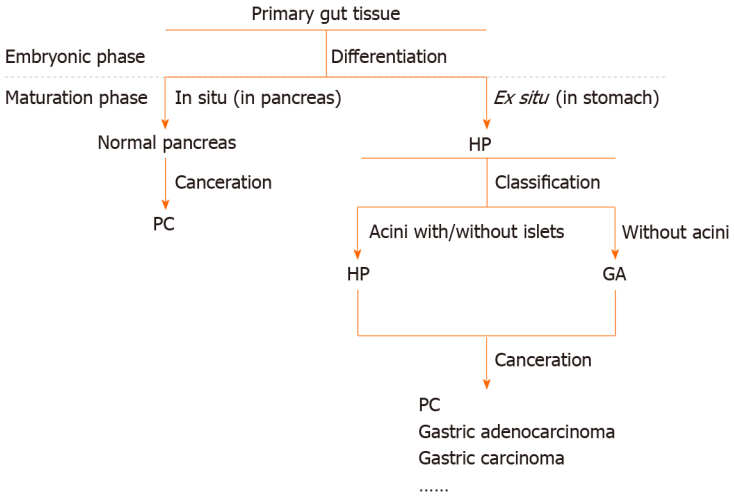

Figure 7 Flow chart of differentiation from primary gut tissue into normal pancreas, heterotopic pancreas, and gastric adenomyoma. PC: Pancreatic cancer; HP: Heterotopic pancreas; GA: Gastric adenomyoma.

Nevertheless, the prognosis depends on the malignant tumor on the caudal pancreas. If it spreads to other organs and reoccurs, the patient's chances of survival could be extremely low.

CONCLUSION

GA is a rare submucosal benign lesion that typically occurs in the gastric antrum and seldom occurs with HP. This report describes a patient diagnosed with PC along with asymptomatic GA and HP. HP and GA are actually the same lesion, but differences between the two exist because of the various differentiation statuses and local microenvironments. We investigated a previous report of 20 patients with simultaneous HP and GA and found that the surgical method used is important in the diagnosis and treatment of GA.

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