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(CASE REPORT)

Gastritis cystica profunda: Case report of an uncommon condition and a brief review of the literature

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Abstract

Gastritis cystica profunda (GCP) is a relatively uncommon benign disease characterized by cystic dilatation and submucosal herniation of the gastric glands. Its clinical and radiological features tend to simulate gastric malignancy, rendering a serious diagnostic dilemma. We describe a GCP case that highlights the diagnostic challenges and underscores the importance of multidisciplinary approach to establish a definitive diagnosis.

A 51-year-old man with a history of gastric polypectomy reported with chronic abdominal pain, early satiety, and unintentional loss of weight. His family history was positive for gastric adenocarcinoma. Endoscopy showed that there is an umbilicated nodular lesion with an umbilication in the center of the site of previous polypectomy. Endoscopic ultrasound revealed a submucosal mass of cystic spaces, hypoechoic, and without ruling out malignancy. A multidisciplinary tumor board suggested surgical resection. The patient underwent a laparoscopic wedge resection. The resected specimen, pathologically examined, revealed a diagnosis of GCP, characterized by hyperplastic glands with cystic dilatation extending into the submucosa, but without evidence of dysplasia or malignancy. The patient's postoperative course was uneventful, with full recovery of symptoms and no recurrence during the three-year follow-up.

The differential diagnosis of gastric submucosal tumors should include GCP when gastric surgery or instrumentation is present. The characteristic of malignancy mimicry makes it challenging to rule out infections requiring full-thickness surgical excision completely, necessitating a definitive diagnosis in many instances. Total resection is diagnostic and curative, and the prognosis is excellent.

Keywords: Gastritis Cystica Profunda; Gastric Submucosal Tumor; Gastric Polypectomy; Gastric Cancer Mimic

1. Introduction

Gastritis cystica profunda (GCP) is a rare, benign hypertrophic gastric lesion characterized by hyperplastic and cystically distended gastric glands that have gone through the muscularis mucosae to reach the submucosal layer. [1] Although the etiology is not completely understood, it is generally considered a reactive process that occurs due to chronic injury of the mucosa. Prior gastric surgery, including gastroenterostomy, is the most frequent predisposing factor but it is

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increasingly identified in unoperated stomachs, typically in conjunction with chronic gastritis, peptic ulcer disease, or, as in our case, past endoscopy, such as polypectomy. [2] [3]

The clinical implications of GCP are that it can replicate more malignant pathologies, especially gastric adenocarcinoma. Patients can be without symptoms or have generic symptoms, including chronic abdominal pain, bleeding, anemia, or loss of weight. The overlap of this symptomatology with endoscopic and radiological observations of submucosal mass, thickened folds, or an ulcerative lesion frequently makes it quite impossible to distinguish between it and malignancy by preoperative investigations. [4] [5] Even complex applications such as endoscopic ultrasound (EUS) can fail to deliver a definitive diagnosis, with the appearances being like other submucosal tumors, including gastrointestinal stromal tumors (GISTs). This leaves clinicians in a diagnostic dilemma, where they may have to resort to surgical intervention to obtain a definitive diagnosis using a histopathological specimen. [5] [6]

We describe a case of a 51-year-old man who presented with disturbing symptoms and was diagnosed with GCP at the location of a prior polypectomy. This Case demonstrates the prototypical diagnostic dilemma of GCP, the importance of a multidisciplinary approach to management, and the importance of surgical resection to exclude malignancy decisively and offer a curative treatment. Based on this case and a brief literature review, we aim to raise awareness of this rare entity and comment on the key characteristics of its diagnosis and treatment.

2. Case presentation

A 51-year-old male reported to the gastroenterology clinic with chronic intermittent abdominal pain and early satiety. The patient also stated that he feels full after eating small portions of food, leading to unintended weight loss of about 8 pounds over the last three months. He explained these symptoms as work stress and had been self-treating with extra vitamins and antacids rather than visiting his primary doctor. Eighteen months ago, he underwent a 2 cm hyperplastic gastric polyp removal. He denied a history of a peptic ulcer, gastroesophageal reflux, or inflammatory bowel disease. He smokes (20 years, one pack/day), and he is moderate in alcohol consumption (4-6 drinks/week). His diet typically consists of a high intake of processed foods and a low intake of fresh fruits and vegetables. His history showed that his father had gastric adenocarcinoma, and his mother had been consistently affected by peptic ulcer disease.

The physical examination revealed no significant epigastric tenderness with rebound or guarding. Laboratory work was unremarkable, with no palpable masses or hepatosplenomegaly noted. The remainder of the examination was unremarkable. Considering the alarming symptom complex and family history, upper endoscopy was performed, which showed a 1.5 cm elevated, nodular lesion with central umbilication of the gastric antrum at the previous polypectomy site. The neighboring mucosa was thickened in a cobblestone pattern, and the lesion was firm with patches of surface erosion. The differential clinical diagnosis included: recurrent hyperplastic polyps (given prior history and similar presentation); gastric adenocarcinoma (family history, smoking, weight loss, and anemia); peptic ulcer disease (despite negative H. pylori, NSAIDs or other causes possible); gastric lymphoma (B symptoms absent but epigastric pain and weight loss present); functional dyspepsia (though weight loss makes this less likely); and gastritis cystica profunda (rare but possible, especially with prior polypectomy history). Endoscopic features did not rule out malignancy. Cross-sectional imaging revealed thickening of the gastric wall in a focal pattern, without lymphadenopathy or evidence of distant disease. Endoscopic ultrasound revealed hypoechoic polypoid thickening that consisted of mucosa and submucosa with numerous small cystic areas in the submucosal layer without invasion of the muscularis propria.

Because of the uncertainty in the diagnosis regarding the endoscopic findings and the patient's family history of gastric cancer, the case was presented at a multidisciplinary tumor board. The team recommended wedge resection to obtain a full-thickness specimen for definitive diagnosis. The patient was subjected to a laparoscopic wedge resection of the gastric antrum, and the entire lesion was removed. Surgery included a 4.5 x 3.2 cm wedge of the gastric wall. The cut surface revealed a 1.8 cm well-demarcated lesion, with multiple small cystic spaces visible throughout the wall. There was no gross evidence of invasion beyond the submucosa. Pathological microscopic examination revealed the typical features of GCP, which included hyperplastic gastric mucosa with long, tortuous glands and significant cystic dilatation that penetrates the deep submucosa. Benign gastric epithelium containing surface, neck, and parietal cells lined the cystic spaces. The gastric mucosa around the polypoid mass showed chronic inflammation and mild fibrosis. Notably, there was no H. pylori infection, no dysplasia, atypia, or malignancy, and all surgical margins were negative. (Figure 1 A, B)

The patient had an uneventful postoperative recovery and was discharged on postoperative day 2, and resumed a normal diet after one week. Two weeks after surgery, he was symptom-free and back to normal activity. Lifestyle changes (dietary modifications and smoking cessation), as well as surveillance endoscopy, were recommended for long-

term follow-up in the presence of a family history and previous gastric pathology. The patient followed all recommended lifestyle changes, and at the three-year follow-up, there were no signs of symptom recurrence or complications.

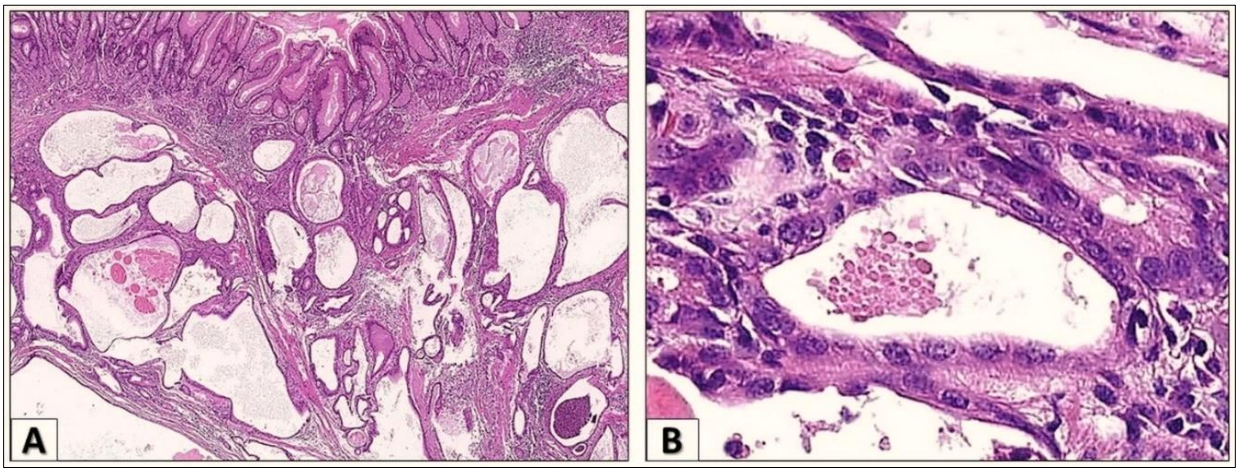

Figure 1 Microscopic features of gastritis cystica profunda (GCP)

- **1A**: Low power view showing hyperplastic gastric mucosa, showing long, tortuous glands with severe cystic dilatation that penetrates the deep submucosa (HandE stain X20)
- **1B**: High power view showing benign gastric epithelium lining the cystic spaces (HandE stain X60)

3. Discussion

3.1. History, Epidemiology, and Pathogenesis

GCP is a rare, benign stomach disorder characterized by hyperplasia and cystic atrophy of gastric glands that permeate through the muscularis mucosae and into the submucosa. Gastritis cystica profunda was the term formally coined by Franzin and Novelli in 1981 to denote the similarity in appearance to colitis cystica profunda, despite its histological nature being originally described in the mid-20th century. [7] [8] [9] The mechanism of pathogenesis of GCP is incompletely understood but is commonly believed to be a reactive mechanism secondary to mucosal injury. The most common theory suggests that disruption within the muscularis mucosa, often due to previous gastric surgery or endoscopic procedures such as polypectomy, allows for the caudal migration of gastric epithelial cells, which in turn undergo cystic alterations. [2] It is also associated with factors such as chronic ischemia, inflammation, and reactions to foreign materials like sutures, Helicobacter pylori infection (H. pylori), or Epstein-Barr virus (EBV) infection, as well as chronic inflammation. [1] It should be noted that cases of GCP have been reported in patients without a history of gastric surgery, leaving the etiology still unclear. [2] GCP is also reported to be associated with chronic active and atrophic gastritis. Chronic atrophic gastritis is a considerable risk factor for cancer development and warrants careful consideration. [10]

The incidence of GCP may be lower because of its insidious manifestation. It is more common in older adults, and most reported cases are found in patients older than 50 and a predisposition towards males with a male/female ratio of about 2.5:1. [1] Although historically associated with patients who had a history of gastric surgery (e.g., gastroenterostomy), current literature suggests an almost equal prevalence in unoperated stomachs, indicating that other etiologies are also highly important. [11] This is specifically applicable to our case because the GCP of the patient developed at the location of a prior polypectomy, which is a known risk factor of the mucosal disruption in the site of the initial development of this condition.

Ki-67 is a proliferation marker applied to cell division in GCP. Some reports indicate that the K-67 labelling index in GCP is typically higher than that in normal stomach tissue, which is reflected in the fact that cell proliferation activity is more active in GCP. [1] Consequently, GCP is considered a potential precursor to cancerous lesions. However, GCP is characterized, in contrast to cancer, by a wide variety of benign cellular morphology, the absence of dysplasia, and a pattern of wild-type p53 immunostaining, which distinguishes it from malignancy. [4]

3.2. The evolving terminology: Gastritis cystica polyposa VS GCP

These two terms are related and have been a source of confusion. The core of the issue lies in historical naming conventions and the subsequent refinement of pathological definitions. Over the years, various names have been used to describe cystic glandular changes in the stomach, including "gastritis cystica polyposa," "multiple polypoid cystic gastritis," and "heterotopic submucosal cysts". [12]

Gastritis Cystica Polyposa is the older, broader term. In the original description, the term "gastritis cystica polyposa" was notably used by Littler in 1972. They described it as a condition typically found at a gastroenterostomy site, characterized by the endoscopic appearance of polyps ("polyposa") and the microscopic finding of cystic glands ("cystica"). [8] [12] Gastritis cystica polyposa was essentially a broader, more descriptive term that encompassed both the macroscopic (polypoid) appearance and the microscopic (cystic) features. It did not strictly define the depth of the cystic glands.

Gastritis cystica profunda" (GCP) is the modern, more precise term. In 1981, Franzin and Novelli proposed the term "gastritis cystica profunda" to more accurately describe the key pathological feature: the deep ("profunda") location of the cystic glands within the submucosa. They drew a parallel to the well-established entity "colitis cystica profunda" in the colon. [9] The defining feature of GCP was the herniation of gastric glands through the muscularis mucosae into the submucosa (and sometimes even the muscularis propria). This is the crucial distinction. [13]

Modern pathology views GCP as a specific subtype within the broader spectrum of what was once referred to as "gastritis cystica polyposa." In a study conducted by Huang et al., they proposed the most accepted way to classify these lesions today. They divided the subtypes into: First, gastritis cystica polyposa (as an overarching concept), which can be seen as a general term for inflammatory gastric conditions with both polypoid features and cystic glands. It can be divided based on the depth of the cysts. [12] Second: Gastritis Cystica Superficialis, in which the cystic changes are confined to the mucosa (above the muscularis mucosae). This is a less clinically significant finding. Moreover, the third is GCP, in which the cystic glands breach the muscularis mucosae and extend into the submucosa or deeper. This is the entity that mimics malignancy and is the subject of our case report. [13]

While a lesion can be both polypoid (the "polyposa" part) and have deep cysts (the "profunda" part), the term GCP has become the preferred and more precise diagnostic term because the submucosal invasion is the most clinically and pathologically important feature. Our current understanding of these terms is that "gastritis cystica polyposa" is considered an older, descriptive umbrella term. "Gastritis cystica profunda" is the modern, specific diagnosis for the clinically relevant form of this condition, where the glands have gone deep.

3.3. Clinical and Imaging Presentation of GCP and Challenges in Diagnosis

GCP clinical presentation is usually nonspecific, and most of it is identified by chance and during endoscopy to investigate other indicators. [1] Chronic epigastric pains are most frequently reported by patients, followed by gastrointestinal bleeding (melena or hematemesis), anemia, and weight loss when symptomatic. There is also an early satiety and gastric outlet obstruction. [4] Our patient was a 51-year-old male with a typical symptomatic picture, such as persistent intermittent pain in the abdomen, early satiety, and a massive loss of weight in an inadvertent manner. Together with the risk factors of his history, such as a history of smoking, a low fresh produce diet, and a concerning family history of gastric adenocarcinoma, these symptoms, combined with a clinical picture that is concerning and indicative of malignancy, necessitated a detailed diagnostic workup.

GCP has a challenging preoperative diagnosis because of nonspecific clinical manifestations, and the imaging features it presents overlap considerably with other gastric diseases, most prominently with malignancy. A diagnostic process typically begins with upper endoscopy, which is often triggered by symptoms such as those in our patient's case. GCP may appear endoscopically in various forms, including a submucosal tumor with either normal or erythematous overlying mucosa, a polypoid or elevated lesion, or thickened gastric folds. [3] The endoscopy appearance of a 1.5 cm elevated, nodular lesion with a central umbilication and its surrounding cobblestone mucosa at a prior polypectomy site in our case was highly suggestive of a neoplastic process, either recurrent adenoma or emerging adenocarcinoma, rather than a benign lesion. Surface erosion added to this suspicion, and ulceration may be a sign of both GCP and gastric cancer. [1] [2]

The extent of gastric lesions is usually assessed by cross-sectional imaging, including computed tomography (CT). CT in GCP often shows focal or diffuse thickening of the gastric wall and can include cystic alterations of the submucosa. These findings, however, are not distinct and may be indistinguishable from infiltrative gastric adenocarcinoma or GISTs. [14] The CT scan of our patient revealed that he had focal thickening of the gastric wall without any obvious signs of

lymphadenopathy or metastasis, something that did not negate malignancy but pointed to the ineffectiveness of traditional imaging in this regard.

Endoscopic ultrasound (EUS) provides a higher level of assessment of the layers of the gastric wall and has become an essential instrument in the diagnostic process. GCP presents as a hypoechoic, heterogeneous mass that arises in the mucosa or submucosa, with or without a few anechoic or cystic spaces, which is the characteristic EUS appearance of the lesion. [Machicado et al 2014] The EUS of our patient revealed these features, characterized by a hypoechoic polypoid thickening of the mucosa and submucosa, with numerous small cystic areas. More importantly, the EUS did not exhibit invasion into the muscularis propria, which is another significant finding that may help distinguish GCP from more aggressive tumors. [2] [6] Nevertheless, the presence of other submucosal tumors, including GISTs, that can also manifest as hypoechoic masses with cystic alterations, indicates that EUS cannot be a conclusive test. [13]

Moreover, fine-needle aspiration (FNA) with EUS may also be non-diagnostic, as the aspirate might only include superficial mucosal cells or be unable to produce a definite cytological diagnosis, thereby missing the vital submucosal cystic glands. Such a diagnostic uncertainty, particularly in a patient with a worrisome presentation and a history of gastric cancer in his family, makes definitive histopathological examination of a resected specimen the sole dependable approach to diagnosis. [15]

3.4. Pathological Diagnosis: Gross, Microscopic, and Immunohistochemical Features

Due to the diagnostic uncertainty posed by clinical and radiographic evidence, gastritis cystica GCP can be diagnosed only definitively with the help of a histopathological examination of a resected specimen. Endoscopic biopsies are not always adequate, as they typically only target the surface mucosa and do not accurately reflect the usual submucosal alterations of GCP. [15] So, as the multidisciplinary tumor board decided in the case of our patient, a full-tumor surgical excision (e.g., a wedge resection) is often necessary to give the pathologist sufficient tissue so that he can make a definite diagnosis. GCP lesions are generally well-delimited and may present as a polypoid lesion, submucosal tumor, or a portion of thickened gastric wall on gross examination. The characteristic appearance of the disease is frequently observed on the cut surface, where numerous small cystic spaces of varying sizes containing mucoid secretions can be found in the submucosa. [1] [4] This was exactly the result of our patient, whose resected specimen demonstrated a 1.8 cm well-characterized lesion with recognizable cystic spaces, with no apparent invasion into the deeper muscular layers, which is in line with the literature

Microscopically, GCP is characterized by hyperplasia of the gastric foveolar epithelium and disruption of the muscularis mucosae. [3] A benign, single-layer columnar epithelium is usually found to line these ectopic glands and may also contain cells of the surface mucosa and neck cells and even parietal cells, as these are a result of the overlying gastric pits. [4] An overlying submucosal stroma is typically fibrotic and characterized by a chronic inflammatory infiltrate comprising lymphocytes and plasma cells. [2] [4] These typical features were confirmed by the microscopic study in our case, which revealed hyperplastic mucosa with tortuous and cystically enlarged glands, extending deep into the submucosa and lined by benign gastric epithelium. [2] Notably, no high-grade dysplasia, atypia, or malignancy was present, and all margins of the surgery were clear in our case.

IHC studies are important in distinguishing GCP from the most significant mimic of this disease, well-differentiated adenocarcinoma. Although some studies have reported higher levels of Ki-67 and p53 in the deeper layers of GCP lesions, especially in those related to dysplasia or carcinoma, the lack of these markers in our specimen provided a good indication of a benign diagnosis. [16]

Currently, there are no clear molecular markers of GCP; however, studies on pathways that include variables such as the KCNE2 potassium channel subunit are in progress. Therefore, a combination of gross, microscopic, and IHC results provides the gold standard for diagnosing GCP and preemptively excluding malignancy.

3.5. Differential Diagnosis of GCP

The clinical and radiological manifestations of GCP establish a wide differentiating diagnosis, encompassing a spectrum of benign and malignant lesions of the gastric submucosa. The most severe and difficult difference is when compared to invasive gastric adenocarcinoma. This is what we were most worried about in our case because the patient had alarming weight loss, early-satiety symptoms, a family history of gastric cancer, and endoscopic findings of an ulcerated, nodular lesion. Even though it has been reported that weight loss is less prevalent in GCP than in adenocarcinoma, its occurrence entails a high index of suspicion of malignancy. [2]

The other malignant diseases in the differential are GISTs and gastric lymphoma. GISTs are hypoechoic submucosal masses that may contain cystic alterations on EUS, like those seen in GCP, making differentiation difficult without tissue examination. Gastric lymphoma may also present with a thickened gastric wall and ulcerative lesions, but it is commonly associated with B symptoms systemically, which were not present in our patient. [2] [5]

There are also several benign entities in the differential diagnosis. Inverted hyperplastic polyps can mimic GCP, and in our patient, a recurrent hyperplastic polyp was considered as an option due to his history of polypectomy at the same location. [2] Another condition to consider is Menetrier disease, characterized by giant hypertrophic gastric folds and foveolar hyperplasia, which has been reported to be associated with GCP. Nevertheless, there were several reasons why Menetrier disease could be less likely in this situation. The first lesion was localized and in the gastric antrum, and Menetrier disease is usually characterized by diffuse thickening of the gastric body and fundus, sparing the antrum. Second, protein-losing gastropathy, resulting in hypoalbuminemia and peripheral edema, which were absent in our patient but whose laboratory work was not remarkable, is a hallmark of Menetrier disease. [17] Although both conditions are characterized by foveolar hyperplasia, the topography of the lesion and the lack of systemic protein loss predisposed the diagnosis of Menetrier disease. There are also less frequent submucosal tumors, including leiomyoma, lipoma, and schwannoma, which are included in the differential. [17]

In our case, the diagnostic measures undertaken were procedural and in line with best practices in assessing such unclear gastric lesions. The first upper endoscopy and cross-sectional imaging were essential in characterizing the lesion, but they turned out to be non-diagnostic and could not exclude malignancy. A critical step that followed the use of EUS gave a detailed imaging of the gastric wall layers. EUS results of a hypoechoic mass with cystic spaces limited to the submucosa were very suggestive of GCP, yet could not rule out GIST.

As this was a diagnostic uncertainty case and the clinical issue of cancer is relevant, the multidisciplinary tumor board made the right decision to administer a complete-thickness wedge resection. This was in response to the recognized inefficiency of preoperative diagnostic methods, such as the low yield of endoscopic biopsies in detecting submucosal pathologies, and the emphasis was placed on a conclusive histopathological diagnosis. The surgical resection was used both as a diagnostic measure to exclude malignancy and as a treatment measure that completely abated the symptomatic lesion.

3.6. Management, Outcome, and Malignant Potential of GCP

Management usually depends on the size, location, symptoms, and suspicion of dysplasia or malignancy of the lesion. Subsequently, the first-line treatment approach is often endoscopic resection. Both endoscopic mucosal resection (EMR) and endoscopic submucosal dissection (ESD) have been successfully used in GCP to achieve complete removal and histologic diagnosis. [3] [5] [18] This was the course of action taken for our patient, whose symptomatic presentation and inconclusive preoperative workup made surgical intervention necessary. A laparoscopic wedge resection was ideal as it provided a full-thickness specimen for definitive diagnosis while being a minimally invasive procedure, leading to an uneventful postoperative recovery. Growing evidence supports this treatment as safe, minimally invasive, and cost-effective, especially for lesions without malignancy. [1] In the event of recurrence, a repeat endoscopic resection often suffices, although surgical resection can be employed, usually less commonly. [18] When endoscopic access is limited, partial or total gastrectomy is the next option. It is the mainstay, especially when there is malignancy. [1] When gastric adenocarcinoma and GCP are both present, surgery is the choice. [2]

Surgery is also the primary option when symptoms such as severe bleeding, obstruction, or mass effect develop. [3]. In very rare cases, such as those which are asymptomatic, conservative management is employed, usually consisting of surveillance or acid suppression therapy (e.g., proton-pump inhibitors, PPIs) [1]. There is a lack of evidence to support using this approach as initial management, which is why it is exceedingly rare. Management post-resection should include initiation of acid suppression therapy (PPIs) and regularly timed surveillance via endoscopy. Though rare, evidence indicates an association between GCP and gastric cancer. [3] [19]

The prognosis of GCP is generally favorable following complete resection. Most patients experience a complete resolution of their symptoms, as was seen in our patient who became symptom-free shortly after surgery and remained so at three years of follow-up. Recurrence of GCP after complete excision is rare. Although it is considered a benign lesion, its complete resection is prudent given the potential of misdiagnosis with gastric adenocarcinoma. [1] [5] This issue is due to difficulty in distinguishing between the two because of their endoscopic appearance and location within the submucosa. Increasing studies and case reports suggest that GCP may act as a precursor or coexist with malignant neoplasm, particularly in patients with a history of chronic gastritis, prolonged gastric mucosal insult, or a history of gastric surgeries. [4] [19] [20] Although the outcomes of endoscopic resection in patients with a non-dysplastic GCP are

quite good, it is still advised to follow up closely with occasional EGDs, particularly in elderly patients and patients with a gastric surgical history. [3] [20] Although there are no universally standardized surveillance guidelines specifically for post-resection GCP, a consensus is emerging that periodic endoscopic follow-up is prudent.

The frequency and duration of surveillance should be tailored to the individual patient, considering risk factors such as the presence of dysplasia in the original lesion, a family history of gastric cancer, or other gastric pathologies. [1] For our patient, the recommendation for lifestyle changes (smoking cessation, dietary improvement) and ongoing surveillance endoscopy was a critical component of his long-term management plan. This strategy appropriately addresses both the excellent prognosis of his resected GCP and the heightened baseline risk conferred by his family history and previous gastric pathology, ensuring early detection of any future abnormalities.

3.7. What have we Learned from this Case?

The given case of gastritis cystica profunda (GCP) can be taken as a useful clinical reminder of several main concepts in gastroenterology and surgical pathology. It highlights the idea that, although not widespread, GCP must be part of the differential diagnosis for any patient with a gastric submucosal lesion, especially those who have had prior gastric instrumentation, such as polypectomy. The fact that our patient underwent a polypectomy at the lesion site provides strong support for the current theory, which states that mucosal injury can be considered a precursor to GCP development.

The difficulty of diagnosis presented by GCP is extremely high, which is demonstrated in this case. The presence of nonspecific, yet alarming, symptoms in the patient (chronic pain, early satiety, and weight loss) along with a family history of gastric cancer caused a clinical scenario in which the primary consideration was malignancy. The endoscopic and imaging results that followed were uncertain and could not conclusively exclude adenocarcinoma. This is one of the fundamental lessons that the diagnosis of GCP at the preoperative stage is a diagnosis of exclusion. Despite the high-quality diagnostic images, such as EUS, a high index of suspicion of malignancy should be maintained. It supports the idea that clinical and radiological observations are not always adequate to distinguish benign and malignant submucosal lesions in the stomach.

The case highlights the irreplaceable value of a multidisciplinary approach in treating complex gastric cases. The choice of proceeding with a surgical wedge resection was a judgment of a tumor board that considered the clinical risk factors of the patient in comparison with the inconclusive diagnostic evidence. This shared practice was used to ensure that the most suitable decision was made, as it balanced the need to diagnose the patient definitively with the application of the least invasive surgical procedure. The resection was effective in both diagnostic and therapeutic terms, providing a solid diagnosis of a benign lesion and resulting in full resolution of symptoms.

This case supports the importance of long-term patient management overall, despite a benign diagnosis. This advice of onward endoscopic observation and lifestyle change was not based solely on the resected GCP, but on the patient's risk profile and family history. This enables us to see beyond the current pathology and assess the overall future risk to the patient, allowing for a comprehensive and accountable plan of follow-up. Ultimately, our examination of this patient confirms that GCP is a reliable simulator of malignancy, and managing it requires a comprehensive workup, a low surgical excision threshold to ascertain the diagnosis, and a long-term follow-up plan.

Abbreviations

- Gastritis cystica profunda (GCP);
- Endoscopic ultrasound (EUS);
- Gastrointestinal stromal tumors (GISTs)

4. Conclusion

This case of gastritis cystica profunda (GCP) illustrates several key gastroenterological principles. First, GCP should be considered in the differential diagnosis of gastric submucosal lesions, particularly in patients with a history of prior gastric procedures, as mucosal injury may predispose to the development of GCP. Second, the case demonstrates the diagnostic challenges inherent to GCP. GCP remains a diagnosis of exclusion since clinical and radiological findings cannot reliably differentiate benign from malignant submucosal lesions. Third, the case underscores the value of multidisciplinary management. The tumor board recommended surgical wedge resection, balancing the need for definitive diagnosis with minimal invasiveness.

Finally, the case highlights the importance of comprehensive long-term management. This holistic approach considers not only the immediate pathology but the patient's overall risk profile, ensuring appropriate long-term follow-up planning.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations

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- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Statement of informed consent

Consent for the publication of this Case was obtained from the patient.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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