



# Bouveret Syndrome simultaneous with an incidental solitary Gastric Neuroendocrine Tumor in a 44-year-old female - A Case Report

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**Introduction and importance:** Bouveret Syndrome (BS) is a rare type of gallstone ileus, where a gallstone passes via a cholecystoduodenal fistula and causes gastric outlet obstruction. It comprises 0.3–0.5% of cholelithiasis complications. It primarily affects females with an average incidence age of 74 years. Gastric Neuroendocrine Tumors (G-NETs) make up merely 2% of all forms of gastric neoplasia and are known to be an extremely rare occurrence. Their annual incidence is estimated to be one to two per 1 million individuals, and they comprise 8.7% of all known neuroendocrine neoplasia of the gastrointestinal system.

**Case presentation:** We present the case of a 44-year-old Middle Eastern female who presented to the clinic due to multiple episodes of food containing biliary nonprojectile emesis accompanied by epigastric pain. Preoperative radiological workup revealed a Bezoar blocking the gastric outlet and a G-NET in the mucosal layer of the stomach.

**Clinical discussion:** Surgical intervention consisted of excising the impacted calculus to relieve the existing gastric outlet obstruction simultaneous to performing an uncut Roux-en-Y to treat the G-NET condition. The patient underwent complete recovery.

**Conclusion:** BS is an extremely rare form of gallstone ileus and gastric outlet obstruction. Its clinical presentation is nonspecific and results in misdiagnosis. Additionally, it is rare to occur in our patient's age group. NETs are also profoundly rare forms of neoplasia. To the best of our knowledge, no previous cases of simultaneous BS and G-NET have been documented. Therefore, clinical awareness should be raised to timely implement the necessary therapeutic interventions.

**Keywords:** Case Report, Abdominal Surgery, Gastric Neuroendocrine Tumor, Gastric Outlet Obstruction, Bouveret Syndrome, Cholecystoduodenal Fistula

## Introduction

Leon Bouveret was the French physician who first documented cases of Bouveret Syndrome (BS) in 1896 and that is the reason it was named after him. BS is an extremely rare class of gallstone ileus, where a gallstone passes via a cholecystoduodenal fistula and blocks the gastric outlet which leads to gastric outlet obstruction<sup>[1,2]</sup>. This syndrome is primarily encountered in the

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## HIGHLIGHTS

- Bouveret Syndrome is a rare type of gallstone ileus that causes gastric outlet obstruction. It comprises 0.3–0.5% of cholelithiasis complications.
- Bouveret syndrome occurs primarily in the elderly population and mainly in females, where the average age of incidence is 74 years.
- Gastric Neuroendocrine Tumors make up merely 2% of all gastric neoplasia. They are extremely rare, and their annual incidence is one to two per 1 million individuals.
- Gastric Neuroendocrine Tumors comprise 0.6–2% of all incidences of gastric polyps, and they are discovered incidentally during esophagogastroduodenoscopy

elderly population and mainly in females, where the average age of incidence is 74 years. Moreover, the male-to-female gender-specific prevalence ratio is 1 : 1.86<sup>[3]</sup>. Multiple etiologies have been proposed for BS, such as patient age above 60 years, previous history of cholelithiasis, a stone sized greater than 2 cm, and the patient being of the female gender<sup>[4]</sup>. Diagnosing this syndrome could be greatly challenging because of its unusual and nonspecific patient presentation which largely imitates presentations of gastric outlet obstruction, such as loss of appetite, nausea, emesis, weight changes, and hematemesis. This results in a misdiagnosis of the case and a delay in treatment<sup>[3]</sup>. The other disease which we are

shedding light on in this case report is Gastric Neuroendocrine Tumors (G-NETs). G-NETs are a direct product of the stomach mucosa's enterochromaffin-like cells. They make up merely 2% of all forms of gastric neoplasia<sup>[5]</sup>. G-NETs are known to be an extremely rare occurrence. Their annual incidence is estimated to be one to two per 1 million individuals, and they comprise 8.7% of all known neuroendocrine neoplasia of the gastrointestinal system<sup>[6]</sup>. They have various pathological forms and possess a wide spectrum of clinical, anatomical, and functional features<sup>[7-9]</sup>.

The work has been reported in line with the SCARE criteria and the revised 2020 SCARE guidelines<sup>[10]</sup>.

## Presentation of case

### Patient information

We hereby present the case of a 44-year-old Middle Eastern female patient who is of a known medical history of Iron Deficiency Anemia (IDA) for 2 years before admission. Her chief complaint was multiple episodes of mixed biliary and food containing nonprojectile emesis which began 10 days prior to presentation. The initial onset of symptoms was sudden 1–2 h following a meal. Its frequency ranged from two to three times per day. Contents included food and biliary fluid, color was yellowish and did not include blood or mucous. Symptoms were accompanied by localized epigastric abdominal pain. It was sudden, intermittent, pressing in nature, did not radiate to any other region, stands by 05/10 on the patient's pain scale, did not have a specific trigger, and was partially relieved by paracetamol tablets. Furthermore, unintentional loss of weight since the start of symptoms was reported. It was not accurately documented on a scale, but the patient noticed herself becoming small for her clothes. Symptoms were also accompanied by general fatigue and loss of appetite. On the other hand, no change of her bowel habits' frequency, consistency, or texture was reported. Furthermore, no change in the color of urine or stool was experienced. Additionally, no jaundice, no cold sweats, night sweats, or fever

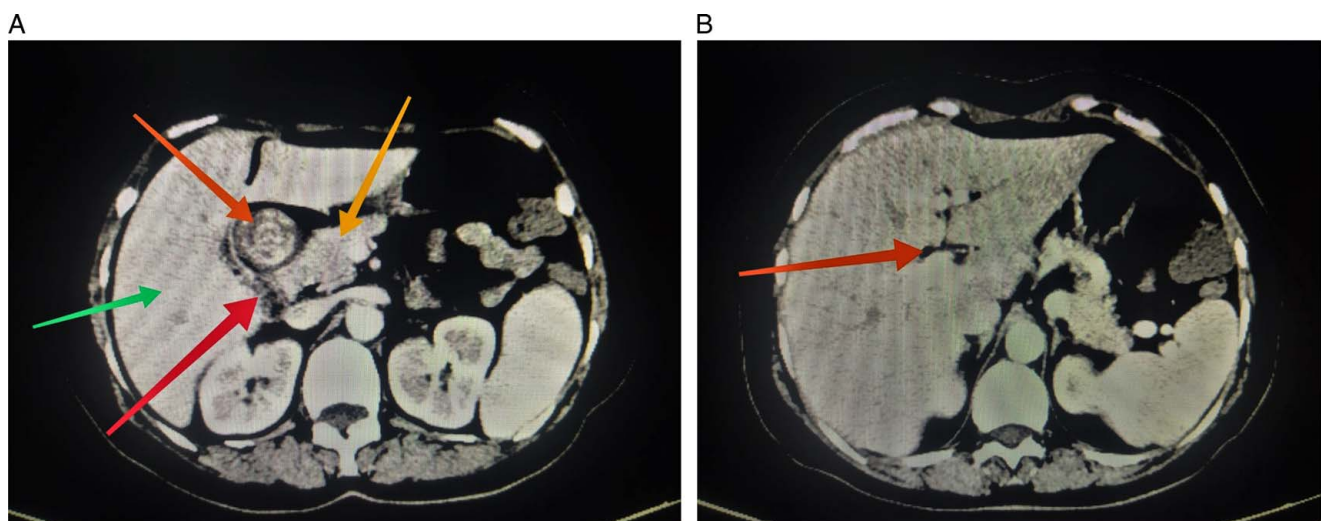
were experienced. Her medical history solely included an IDA for 2 years. Her drug history included ferrous sulphate and omeprazole. Her surgical, family, allergic, and psychosocial histories were negative. She also negated any exposure to arsenic, chemotherapy, or irradiation. A previous similar occurrence of symptoms was denied. Her BMI was 23 kg/m<sup>2</sup>.

### Clinical findings

Physical examination included facial pallor on inspection and delayed capillary refill to 4 s on palpation. The remaining findings on palpation, percussion, and auscultation were uneventful. Laboratory panel results upon patient admission revealed values consistent with a microcytic hypochromic IDA (Hemoglobin: 10 g/dl, Hematocrit: 36%, Mean Corpuscular Volume: 70.5 fl, Mean Corpuscular Hemoglobin: 20 pg, Mean Corpuscular Hemoglobin Concentration: 28.5%, and Red Cell Distribution Width: 21.7%). Other abnormal lab investigations included the increased values of Alanine Transaminase: 103 µl, Aspartate Transaminase: 58 µl, Direct Bilirubin: 0.38 mg/dl, Alkaline Phosphatase: 327 µl, Pancreatic Amylase: 189 µl, and tumor marker CA 19-9: 56.94 µ/ml. Other tumor markers, such as Alpha Fetoprotein and Carcinoembryonic Antigen were within normal. Further lab values were normal as well.

### Diagnostic assessment

Preoperative abdominal ultrasound revealed a severely shrunken gallbladder with no secretions within. A prominent hypoechoic stone formation measuring (25 mm) was also demonstrated. The remaining findings were insignificant, and no regional lymphadenopathy was reported. A multislice computed tomography scan with contrast was then carried out. It revealed the presence of pneumobilia within the intrahepatic/extrahepatic biliary tracts and confirmed the formation of Cholelithiasis. A pyramidal-shaped solid stone-like formation was seen at the level of the lumen of the first segment of the duodenum. It has no contrast material reuptake. A cholecystoduodenal fistula formation is



**Figure 1.** (A) Preoperative Multi-Slice Computed Tomography with contrast image in cross-sectional view where the orange arrow points to stone impacted in the duodenum, the yellow arrow points to the pancreas, the red arrow points to the second and third segments of the duodenum, and the green arrow points to the liver. (B) Preoperative multislice computed tomography with contrast image in cross-sectional view where the orange arrow identifies the pneumobilia found in the intrahepatic/extrahepatic biliary ducts.

suspected as well (Fig. 1A–B). The remaining regions including the chest, abdomen, and pelvis were radiologically free of any lesions. Furthermore, no lymphadenopathy was demonstrated. An esophagogastroduodenoscopy was achieved. It revealed generalized atrophic gastritis in addition to a sessile polyp measuring (1 cm) was visualized in the gastric antrum (Fig. 2A). The duodenal bulb included partially digested food materials (Bezoar) obstructing the pyloric opening which was later chemically analyzed after surgery to be a calcium-based gallbladder stone (Fig. 2B). Bypassing the said segment and taking biopsies from it was not possible due to the obstruction. Furthermore, a diverticular-like structure was seen which was revealed intraoperatively to be the cholecystoduodenal fistula (Fig. 2C). An endoscopic ultrasound was also performed. Endoscopically, it showed a sessile polyp sized (1 cm) in the gastric antrum. Radiologically, it revealed a hypochoic formation measuring (13 × 5.4 mm) at the extent of the mucosa with no apparent extension to the submucosa or the other layers (Fig. 3). Said polyp was endoscopically excised and immediately sent for histopathological analysis. Final histopathological analysis of the specimens revealed a (15 mm) polypoid that conforms with a Grade I well-differentiated NET. Tumor cells minimally invaded through the mucosa into the submucosa. Analysis was completed via Hematoxylin and Eosin staining (Fig. 4A–B) and immunohistochemical analysis staining positive for Chromogranin A and Synaptophysin, while revealing a low proliferative index Ki-67 less than 3% (Fig. 5A–C) and had less than two mitoses/2 mm<sup>2</sup>. However, it stained negative for Neuron-Specific Enolase (NSE). To prepare our patient for surgical intervention, necessary steps were taken. These included setting up intravenous access, administering prophylactic antibiotics, and blood sampling for crossmatching. No obstacles were encountered during this phase.

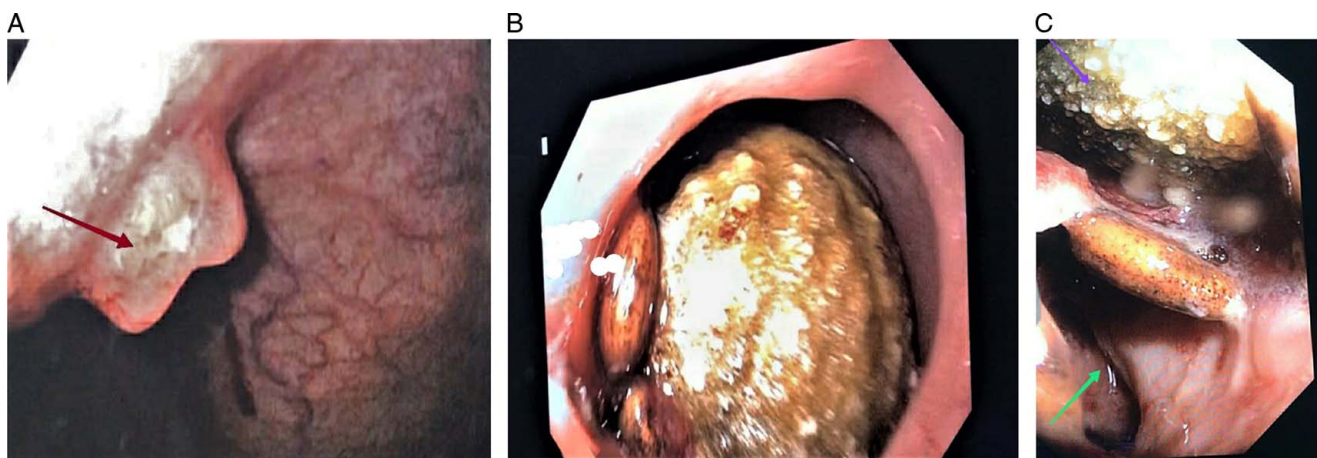
### Therapeutic intervention

Since bypassing the pyloric opening and taking biopsies from the affected section was not possible during the preoperative diagnostic period when it was attempted during esophagogastroduodenoscopy, open surgical intervention was deemed

necessary. Surgery was successfully performed at our specialized university hospital. It was accomplished by a General Surgery specialist with 12 years of experience. The operation was done under general anesthesia with no perioperative complications. Via a supraumbilical midline incision, a cholecystoduodenal fistula was seen. It was resected along with the gallbladder. Furthermore, a large stone was found impacted in the duodenal bulb. Therefore, gastric antrectomy plus the first segment of the duodenum along with a segment of the greater omentum was excised. Then, reconstruction in the form of an uncut Roux-en-Y was achieved. The resected stone which was causing the gastric outlet obstruction was sent for chemical analysis that later revealed that it is mainly composed of calcium. Resected specimens were immediately sent for histopathological analysis via Hematoxylin and Eosin. Results revealed no residual tumor cells present in any specimen (Fig. 6A–D). The patient underwent complete postoperative recovery and was therefore discharged to the outpatient setting within 7 days of surgery. Sterile wound dressings were regularly applied, and postoperative antibiotics were administered. She was afterward referred and followed up by our oncology department. She has been followed up for 4 years now. The patient's symptoms had completely resolved and her follow-up laboratory investigations all yielded normal results. Additionally, her IDA status has also significantly improved because of the return of healthy food intake and adherence to iron supplement therapy. Regular visitations at our clinic were scheduled to ensure her successful recovery. Clinical physical and radiological examinations were carried out and revealed normal results.

### Discussion

Bouveret Syndrome is the nomenclature given when a gallstone escapes the gallbladder via a cholecystoduodenal fistula and impacts the gastric outlet to result in the occurrence of gastric outlet obstruction. Leon Bouveret pioneered the documentation of this pathology in 1896<sup>[11]</sup>. BS is an extremely rare manifestation of gallstone ileus and comprises merely 1–3% of all cases of



**Figure 2.** (A) Esophagogastroduodenoscopy image revealing the sessile gastric polyp located in the antrum and marked by the red arrow. (B) Esophagogastroduodenoscopy image revealing what was believed to be a Bezoar blocking the gastric outlet. It was later confirmed by chemical analysis to be a calcium-based gallbladder stone. (C) Esophagogastroduodenoscopy image revealing what was believed to be a diverticulum marked by the green arrow. It was intraoperatively revealed to be a cholecystoduodenal fistula. It was situated across from the impacted gallbladder stone which is marked by the purple arrow.

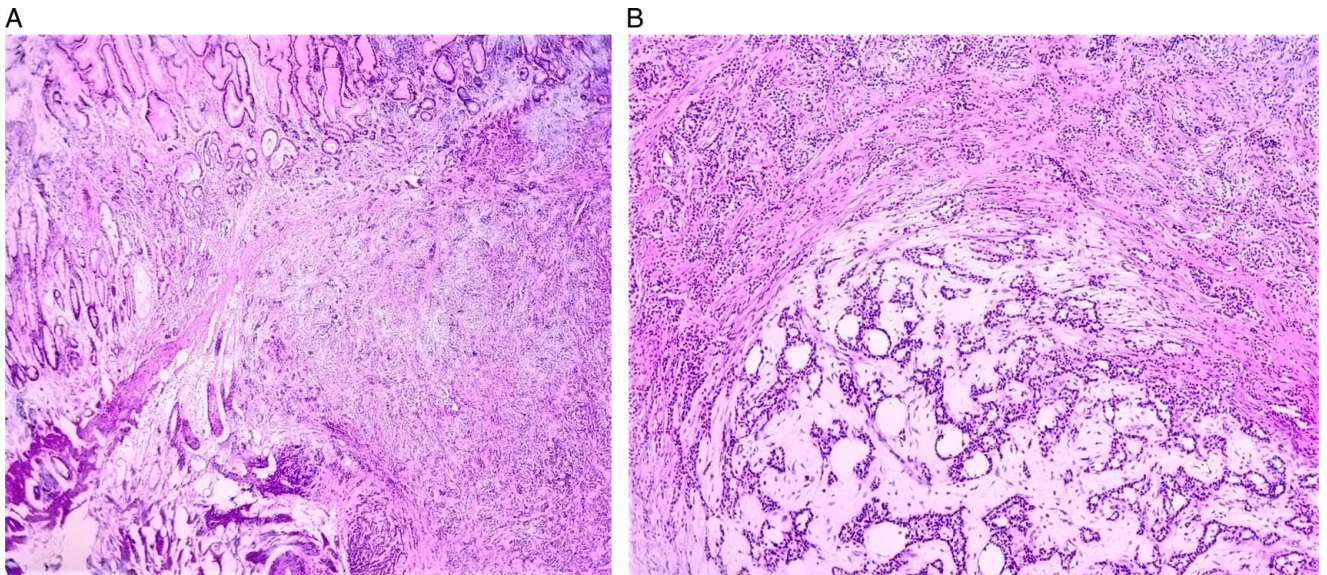




**Figure 3.** Preoperative endoscopic ultrasound where the orange arrow points at the hypoechoic formation measuring (13×5.4 mm) at the extent of the mucosa with no extension to the submucosa or the other layers.

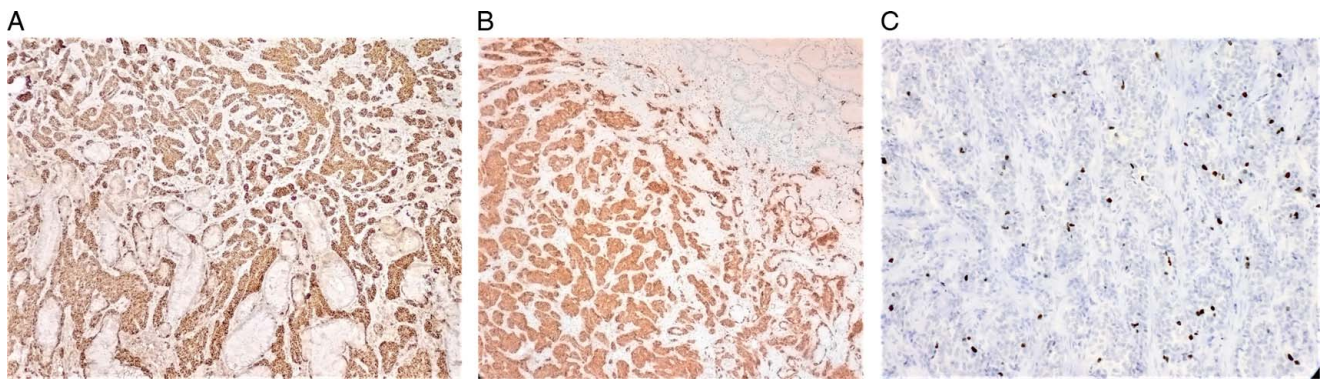
gallstone ileus<sup>[12]</sup>. This rare form of gallstone ileus accounts for 0.3–0.5% of all cases of cholelithiasis. It is a direct consequence of the formation of tight adhesions and subsequent necrosis between the gallbladder and the digestive alimentary tract which results in fistula development<sup>[13]</sup>. Overall, 85% of cases are spontaneously resolved when the stone passes along with feces and/or with emesis, whereas 15% of stones that are larger than 25 mm get lodged in the following anatomical locations in descending order; terminal ileum, proximal ileum, jejunum, colon, and rarest in the stomach and duodenum<sup>[14–16]</sup>. Regarding patient clinical presentation, patients mostly present with nonspecific symptoms that resemble those of upper gastrointestinal tract obstruction. Those symptoms could include abdominal pain, episodes of emesis, and decreased appetite. On rare occasions, patients could present with hematemesis. On the other hand, physical

examination sometimes marks the presence of dehydration symptoms, abdominal tenderness on palpation, and distention<sup>[3]</sup>. Diagnosing BS preoperatively could be a genuine challenge for physicians. Moreover, radiological modalities, such as contrast-enhanced computed tomography scans, plain abdominal radiography, and abdominal ultrasound can only result in a diagnosis in merely 21% of situations. It is worth noting that the probability to diagnose BS is significantly raised with the use of computed tomography due to its high sensitivity. Traditionally, Riggler’s Triad is the known radiological sign to look for in radiological imaging. It includes an ectopic gallstone, signs of bowel obstruction like a distended stomach for example, and pneumobilia<sup>[17–19]</sup>. In our case, we could not identify Riggler’s Triad because no distention or signs of bowel obstruction were witnessed. Only pneumobilia and an ectopic gallstone were clear to be seen. Esophagogastroduodenoscopy enables physicians to clearly demarcate the presence and status of the occlusive object – the stone – where it looks like a hard mass with the corresponding prior segment of the gastrointestinal tract dilated. In a few cases, we can also visualize the cholecystoduodenal fistula<sup>[13,20]</sup>. This condition warrants immediate intervention to treat it. Otherwise, there is a variety of dangerous complications which could arise due to a delay or inadequate treatment (i.e. repeated episodes of ileus, gallbladder malignancy, ascending cholangitis, recurrent bouts of cholecystitis, and gastrointestinal hemorrhage)<sup>[21]</sup>. Treatment includes many conventional and novel modalities. Surgical intervention is varied. One of those varieties is an all-in-all one-phase surgical procedure where the surgeon repairs the present cholecystoduodenal fistula, and simultaneously performs cholecystectomy and enterolithotomy to remove the impacted stone<sup>[1]</sup>. The reason why this is favored over laparoscopic interventions is that around half of laparoscopic modalities fail in accomplishing the task and result in conversion to open surgery<sup>[22]</sup>. Our other equally important illness which aided our patient was a form of G-NETs. G-NETs are direct derivatives of



**Figure 4.** (A) Histopathological analysis via Hematoxylin and Eosin (H&E) staining of the resected gastric polyp revealing invasive proliferation of small monotonous cells invading through muscularis mucosa into the submucosa (H&E, × 40). (B) Histopathological analysis via H&E staining of the resected gastric polyp revealing tumor cells are small monotonous with round nuclei, arranged in cords, small nests, and cribriform-like pattern (H&E, × 100).

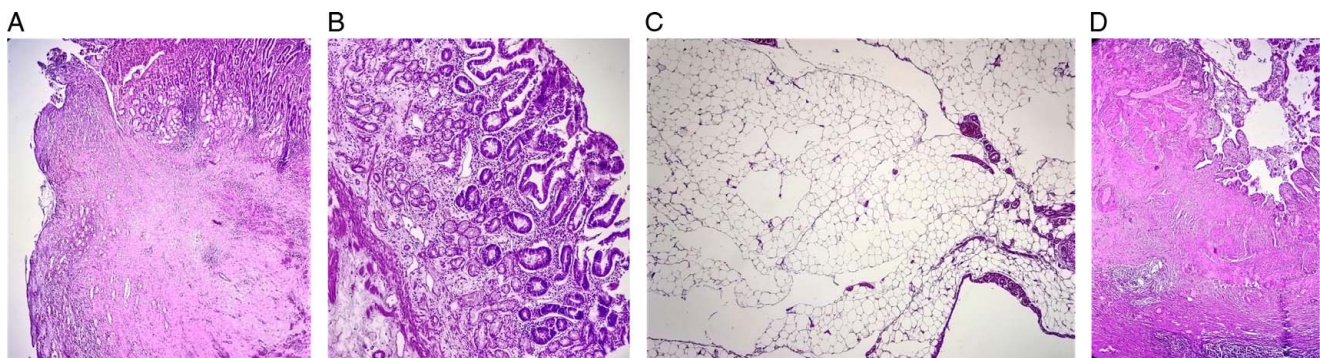




**Figure 5.** (A) Immunohistochemical (IHC) staining of the resected gastric specimens revealing tumor cells are positive for Chromogranin A (IHC,  $\times 100$ ). (B) IHC staining of the resected gastric specimens revealing tumor cells are positive for Synaptophysin (IHC,  $\times 100$ ). (C) IHC staining of the resected gastric specimens revealing Ki-67 proliferation index is low ( $< 3\%$ ) (IHC,  $\times 200$ ).

the stomach mucosa's enterochromaffin-like cells. They represent 2% of all classes of gastric neoplasia<sup>[5]</sup>. G-NETs are extremely rare where their annual incidence is ranged between one and two cases per 1 million individuals, and they comprise 8.7% of all known neuroendocrine neoplasia of the gastrointestinal tract<sup>[6]</sup>. G-NETs comprise 0.6–2% of all incidences of gastric polyps, and they are mainly discovered incidental findings during esophagogastroduodenoscopy<sup>[23–25]</sup>. A significant amount of data was analyzed according to the Surveillance, Epidemiology, and End Results in the United States during the timeframe 2000–2015 for the purpose of discovering the race-dependent incidence rate of G-NETs. The discovered incidence rate for local G-NETs was 1.5/100 000, 3.4/100 000, and 1.54/100 000 in Caucasians, Africans, and Hispanics, respectively<sup>[26]</sup>. Regarding the average age of incidence, it was 59.8 years in Japan<sup>[27]</sup> and 63 years in the USA according to the Surveillance, Epidemiology, and End Results database<sup>[28]</sup>. The WHO redefined the classification of NETs in 2017 whereby tumor Grade I is assigned to a NET when it's well-differentiated with a Ki-67 proliferation index less than 3% and less than two mitoses/2 mm<sup>2</sup><sup>[29]</sup>. Accordingly, Grade I was the one assigned to our patient's G-NET. Regarding types of G-NETs, type 1 is the most prevalent and accounts for 70–80% of all G-NETs<sup>[30,31]</sup>. It is classically

prevalent in individuals aged 60–70 years old with a clear female-to-male dominance<sup>[32,33]</sup>. The etiology behind this type is chronic atrophic gastritis<sup>[34]</sup> where the annual development risk for G-NETs in a patient background of chronic atrophic gastritis is merely 0.4–0.7%<sup>[35,36]</sup>. Patient clinical presentation is variable and nonspecific. Potential symptoms include vague abdominal pain, anemia, and nausea<sup>[31,37]</sup>. To diagnose G-NETs, multiple methods are available. However, endoscopic ultrasound is vital in the preoperative diagnostic phase. It provides proper visualization of the lesion, its location, its invasion of the local anatomical structures, and possible regional lymphadenopathy<sup>[38,39]</sup>. The final diagnosis can only be established after proper histopathological analysis of the lesion<sup>[40]</sup>. Furthermore, immunohistochemical staining is crucial for making a diagnosis where chromogranin A and synaptophysin are the main stains used for G-NETs<sup>[38]</sup>. Treatment modalities are numerous, and all revolve around the core concept of surgical excision of the G-NET. Antrectomy is performed in type 1 after the endoscopic resection of the lesion. This yields in excellent results in decreasing neoplastic recurrence<sup>[41,42]</sup>. Finally, the prognosis of type 1 G-NETs is adequate with over 95% 5-year patient survival rate<sup>[37,43]</sup>.



**Figure 6.** (A) Histopathological analysis via Hematoxylin and Eosin (H&E) staining of the resected stomach segment at the site of polypectomy revealing an ulcer bed with abundant granulation tissue and fibrosis. No residual tumor cells noted (H&E,  $\times 40$ ). (B) Histopathological analysis via H&E staining of the resected stomach segment at the site of polypectomy revealing reactive gastropathy-like appearance with mild atrophy and intestinal metaplasia (H&E,  $\times 100$ ). (C) Histopathological analysis via H&E staining of the resected segment of the greater omentum is free of tumor cells (H&E,  $\times 40$ ). (D) Histopathological analysis via H&E staining of the resected gallbladder revealing moderate lymphocytic inflammatory infiltrate, muscular hyperplasia, and adventitial fibrosis (H&E,  $\times 100$ ).

## Conclusion

BS is an extremely rare form of gallstone ileus and gastric outlet obstruction. It is rare regarding all epidemiological parameters. Moreover, its clinical presentation is nonspecific and results in misdiagnosis. Furthermore, it is widely uncommon to occur in our patient's age group. On the other hand, NETs are highly rare forms of neoplasia which rarely occur in our patient's age group population. To the best of our knowledge, no previous similar cases of simultaneous BS and G-NET have been documented, especially ones from our country. For this reason, documenting these cases if they were separate is a valid endeavor and is now more valuable than they were simultaneously occurring. Therefore, clinical awareness should be raised to timely deploy the required therapeutic interventions.

## Ethics approval

N/A.

## Consent of patient

Written informed consent was obtained from the patient for the publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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## Author contributions

O.A.: Conceptualization, resources, who wrote, original drafted, edited, visualized, validated, literature reviewed the manuscript, and the corresponding author who submitted the paper for publication.

J.S.: Supervision, visualization, validation, resources, and review of the manuscript.

Z.I.: Histopathologist and cytologist who aided in histopathological analysis of the microscopic specimens, in addition to validation and review of the manuscript.

F.A. and R.S.: Visualization, validation, and review of the manuscript. M.I.: surgical assistant in the operation, supervision, project administration, validation, resources, and review of the manuscript.

A.A.: General Surgery specialist who performed and supervised the operation, in addition to supervision, project administration, and review of the manuscript. All authors read and approved the final manuscript.

## Conflicts of interest disclosure

None.

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