Case report

Gastric outlet obstruction secondary to heterotopic pancreas being mistaken for gastric cancer: “Case report”

Gosa Bejiga
Adama Hospital Medical College, Adama, P.O. Box:84, Ethiopia

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Abstract

Introduction: Heterotopic pancreas (HP) is an uncommon and often incidental finding in clinical practice. It is the presence of pancreatic tissue distinct from the normal pancreas and with its own ductal and vascular supply. Distal stomach is the most common location of heterotopic pancreas followed by duodenum and jejunum. Most patients with heterotopic pancreas are asymptomatic. Gastric outlet obstruction is a rare presentation of heterotopic pancreas that can follow chronic inflammation and fibrosis of the pylorus from pancreatic secretion, pancreatitis, or malignant transformation. Heterotopic pancreas can be confused for gastric carcinomas on CECT and endoscopy. The aim of this paper is to present a rare case of gastric outlet obstruction due to heterotopic pancreas, mistaken for gastric cancer on endoscopy and CT scan, and its management.

Presentation of the case: A 45 years old male from Adama, Ethiopia presented with vomiting of 8 months, which worsened since the last one month. He has no history of smoking and diabetes. He occasionally drinks alcohol. Physical examination was normal. CECT scan and endoscopy suggested distal gastric cancer. Radical subtotal gastrectomy done as gastric cancer couldn’t be ruled out with excellent outcome. Histopathology revealed obstructing prepyloric chronic fibrosis and heterotopic pancreas.

Discussion: Heterotopic pancreas is a rare pathological entity, clinical diagnosis is difficult preoperatively and frequently an incidental finding at laparotomy.

Conclusion: Though rare, heterotopic pancreas can present with gastric outlet obstruction and cause diagnostic confusion with gastric cancer. Definitive diagnosis is by histology that can also guide limited resection intraoperatively.

1. Introduction

Gastric outlet obstruction (GOO) is a clinical syndrome characterized by epigastric abdominal pain and postprandial vomiting due to partial or complete mechanical obstruction. Currently, the most common cause of GOO is malignancy with distal gastric cancer in 35% [1] and pancreatic cancer in 15 to 25% of patients [2]. In the past, peptic ulcer disease was the leading cause of GOO, but now with the use of proton pump inhibitors and H. pylori eradication therapies, less than 5% of complicated duodenal ulcers and less than 1–2% of complicated gastric ulcers lead to GOO [3]. Heterotopic pancreas is a rare cause of GOO with limited number of case reports [4].

Heterotopic pancreas is defined as the presence of pancreatic tissue outside its normal location in the pancreas and having neither anatomical nor vascular continuity with the pancreas [5]. It is also known as aberrant pancreas, pancreatic rest, or accessory pancreas [6]. Heterotopic pancreas most commonly occurs in the upper gastrointestinal tract, with occasional cases reported in the ileum, bile ducts, mediastinum, gall bladder, fallopian tubes, splenic hilum, omentum and lungs [7].

Most patients with heterotopic pancreas are asymptomatic and frequently incidental finding at laparotomy; however, some can develop gastrointestinal bleeding, obstruction, pancreatitis or malignant transformation [6,8,9]. For a clinical purpose, it is important to keep in mind that all diseases arising in the true pancreas can also develop in heterotopic pancreatic tissue [10]. As symptomatic patients are rare and no specific set of clinical features, it is difficult to diagnose heterotopic pancreas before operation [11]. Despite advances in diagnostic modalities, differentiating heterotopic pancreas from neoplasm still remains a challenge to clinicians [12]. Here, I present a case of gastric outlet obstruction secondary to heterotopic pancreas that was uncovered after surgery. This work is reported in line with the SCARE 2020 criteria [13].

Abbreviations: GOO, gastric outlet obstruction; HP, heterotopic pancreas.
E-mail address: info@adamahmc.edu.et.

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2. Presentation of the case

A 45 years old male employee from Adama town, East Shoa, Ethiopia presented with complaint of vomiting of ingested matter sometimes mixed with blood of 8 months duration. He had history of burning epigastric pain, which relieved by taking meal and oral antacid for the past 20 years. However, since the last one month the pain worsened and failed to improve with proton pump inhibitor and associated with vomiting of 1 to 2 episodes per day. He has no significant weight loss, which was 2 kg within the last one year. He has no history of smoking, diabetes or hypertension. He occasionally drinks alcohol. Physical examination was normal.

Complete blood count (CBC), serum electrolytes, liver function tests and renal function tests were normal. Serum albumin was slightly low 3.623 g/dl (normal range 3.8 to 5.1 g/dl). Abdominal CECT scan showed 5.4 cm long antral wall thickening with luminal narrowing and significant gastric dilatation (Fig. 1). No lymphadenopathies, pleural effusion, peritoneal fluid or secondaries seen, and the fat plane between the thickened distal gastric wall and pancreas is obscured but no clear mass infiltration of the pancreas seen (Fig. 2); concluding Gastric outlet obstruction secondary to antral carcinoma, T3N0M0. Chest X-ray and ECG are normal. Upper GI endoscopy showed lower esophageal inflammatory change with ulcer; swollen erythematous and ulcerated mass lesion around the pylorus obstructing the pyloric orifice preventing further progress of the scope to duodenum concluding gastric outlet obstruction secondary to prepyloric cancer (Fig. 3). Endoscopic biopsy showed bland non-atrophic focally eroded pyloric mucosa with overlying fibrino-purulent exudates and lymphocyte predominant inflammation in lamina propria; \textit{Helicobacter pylori} seen in glandular lumen, but no dysplasia or malignancy seen; concluding prepyloric chronic active gastritis with superimposed acute erosive changes.

Exploratory laparotomy showed dilated stomach with soft to firm mass at prepyloric area, few lymph node enlargements along left gastric artery, sub pyloric and transverse mesocolic area. Radical subtotal gastrectomy done for suspected gastric cancer, removing 70% of the stomach enblock with lymph nodes and omentum. Continuity maintained with gastro-jejunostomy and Brawn’s anastomosis.

Gross tissue examination showed erythematous circumferential anthal thickening, pyloric obstruction and normal duodenal mucosa (Fig. 4). Intraluminal inspection of the specimen showed necrotic tissue, normal pale gastric body mucosa, erythematous antrum with circumferential thickening, mucosal ulcer, raised lesion with central depression (looks like umbilicus) on greater curvature side, and obstructed pyloric canal (Fig. 5). The mass described on endoscopy was not seen, probably necrotized. Histopathology revealed the diagnosis of prepyloric chronic fibrosing inflammation causing obstruction plus heterotopic pancreatic tissue. Histopathology did not show malignant process. Patient discharged on the seventh postoperative day, triple therapy (amoxicillin 1 g, clarithromycin 500 mg, and omeprazole 20 mg, twice daily for ten days) was given on follow up. Postoperative recovery was uneventful. Now it has been 5 months, the patient gained 8Kg weight and fully functional with no new complaint.

3. Discussion

Heterotopic pancreas is defined as the presence of pancreatic tissue outside its normal location in the pancreas and having neither anatomical nor vascular continuity with the pancreas. The true incidence of heterotopic pancreas in the general population is unknown. It is reported in 0.2% of abdominal surgeries, 0.55% to 13.7% of autopsy series and commonly seen in 30 to 50 years of age with male preponderance [14].

Heterotopic pancreas is most commonly seen in stomach, duodenum and jejunum, with other areas like esophagus, ileum, omentum, gall bladder, Meckel’s diverticulum, and mediastinum are less common locations of heterotopic pancreas reported in literatures [6,15].
extremely rare, with a reported incidence of <1% [16], and with the better prognosis as compared to true pancreas [17]. For a clinical purpose, it is important to keep in mind that all diseases arising in the true pancreas can also develop in heterotopic pancreatic tissue [19].

The main differential diagnoses of HP include gastrointestinal stromal tumors, gastrointestinal autonomic nerve tumors, gastric carcinoids, lymphomas and gastric carcinomas [18]. In the case presented here, HP was mistaken for gastric antral carcinoma that was diagnosed after surgery.

Despite advances in diagnostic modalities, differentiating heterotopic pancreas from neoplasm still remains a challenge to clinicians [12]. In the case presented here, endoscopy and CECT put diagnosis of pyloric and antral carcinoma with GOO respectively that was proven heterotopic pancreas after surgery.
There is no efficient preoperative diagnostic test for HP. The only definitive diagnostic test is histology, but CECT, endoscopy, and EUS are of some help when the lesion is small and typical presenting as submucosal mass with central depression [14]. However, typical endoscopic findings of HP include well-circumscribed submucosal lesion with central umbilication is observed in less than half of the cases, causing difficulty of differentiating HP from other diseases like GIST, leiomyoma, or sub mucosal gastric carcinoma. EUS-guided fine needle aspiration (FNA) or fine needle biopsies (FNB) are helpful methods for histologic diagnosis of submucosal lesions like GISTs and heterotopic pancreas [19].

Treatment of histologically proven asymptomatic HP is debatable [20]. As medical treatment is not effective for symptomatic patients, localized surgical excision is adequate and the treatment of choice, unless malignant transformation is present [14]. Frozen section is useful to decide extent of resection, and avoiding unnecessary radical surgery after excluding malignancy intra-operatively [14]. Excision results in excellent prognosis.

If the patient presents with GOO due to HP as the case presented here, antrectomy with gastro-jejunostomy or gastro-duodenostomy are options. Lymph node dissection is not indicated. My patient was treated with radical subtotal gastrectomy, gastrojejunostomy with Brawn's anastomosis as gastric cancer could not be ruled out by the investigations.

4. Conclusion

Even though rare, diagnosis of HP should be considered in patients with GOO. Antrectomy and gastrojejunostomy is the best treatment with
excellent out comes in cases of obstructing, large distal gastric HP. It is safe to do distal gastrectomy for GOO if malignancy is suggested by imaging, especially in resource-limited areas like ours where there is no frozen section pathology and EUS services. Gastric cancer must be ruled out in patients with GOO, as it is the most frequent cause.

Provenance and peer review

Not commissioned, externally peer reviewed.

Consent

Written informed consent was obtained from the patient’s next of kin for 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.

Ethical approval

The study is exempt from ethical approval in our setup.

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Declaration of competing interest

None declared.

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