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Case Report

From chronic diarrhea to diagnosis: unmasking rare giant gastrinoma

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ABSTRACT

Gastrinomas are rare neuroendocrine tumors, most commonly arising in the pancreas or duodenum and typically present with peptic ulcer disease and gastric acid hypersecretion. Atypical symptoms such as chronic diarrhoea and abdominal pain, along with partial response to proton pump inhibitors, may delay diagnosis and timely treatment. We report the case of a 35 years old male who presented with a one-year history of persistent watery diarrhoea and intermittent abdominal pain. Clinical examination revealed an abdominal mass and imaging demonstrated a giant pancreatic head lesion and biochemical analysis showed markedly elevated serum gastrin levels. Histopathology confirmed the diagnosis of gastrinoma. The unusually large tumor size, combined with an atypical presentation as chronic diarrhoea, highlights the diagnostic challenges associated with gastrinomas. This case underscores the importance of considering neuroendocrine tumors, including Zollinger–Ellison Syndrome (ZES), in the differential diagnosis of unexplained chronic diarrhoea and abdominal pain. Timely recognition and management are crucial to improving outcomes. Giant gastrinomas (>12 cm) with multifocality in the context of ZES have not been reported in the literature, making this case a unique addition.

Keywords: Chronic diarrhoea, Giant gastrinoma, Neuroendocrine tumor, Zollinger ellison syndrome

INTRODUCTION

Gastrinoma is an uncommon type of neuroendocrine tumor (NET), with a reported global incidence ranging from 0.1 to 15 cases per million population. Although rare, it is implicated in approximately 0.1% of all peptic ulcers and in 2–5% of recurrent ulcer cases.[1] Gastrinomas lead to excessive gastric acid secretion, marked hyperacidity and atypical ulcer formation a clinical entity first documented by Zollinger and Ellison in 1955.[2] Because of their scarcity and the absence of pathognomonic symptoms, these tumours are often overlooked or misdiagnosed. The clinical spectrum may include abdominal pain, persistent secretory diarrhea, esophagitis and, in some cases, hypercalcemia. Majority of patient presents with abdominal pain and diarrhea due to peptic ulcer disease but only 10% of cases may present with diarrhea as the sole symptom which may result in delayed diagnosis.[3] While elevated fasting serum gastrin is a crucial diagnostic clue, with sensitivity of >99%, it's a screening test for gastrinoma which can reduces diagnostic delays. But confirmation ultimately depends on histopathological evaluation supported by immunohistochemistry as some Gastrinomas might have normal gastrin levels. In a typical patient, the fasting gastrin levels are less than 100 pg/ml, however, a suspicion for gastrinoma is higher if the fasting gastrin levels are greater than 300 pg/ml, Gastrin levels greater than 1000 pg/ml with a gastric pH of less than 2 are considered diagnostic of gastrinoma.[4] At present, complete surgical excision remains the only established curative option for gastrinoma.

CASE REPORT

A 35 years old male presented to our department with a one-year history of loose stools, occurring approximately three times daily, described as frothy, watery and non-bloody. He also reported intermittent, colicky abdominal pain of one-year duration, occurring roughly once every ten days. The symptoms were getting temporarily relieved by symptomatic treatment with proton pump

inhibitors. He also had chest pain, felt once a week associated with breathing difficulty and a persistent bloating sensation. His symptoms were responding to proton pump inhibitors. No nausea, vomiting, melena or tenesmus were reported. He was on treatment for Psychiatric illness somatoform disorder and was on treatment for hypertension. Patient denied smoking, alcohol or drug abuse.

On physical examination there was a palpable well-defined mass in the right hypochondrium 8×8 cm firm in consistency well defined surface and margins, non-tender with slight internal mobility. No abdominal tenderness was present. His heart and lungs were normal to auscultation. Vitals were normal. blood investigations including complete blood count, renal and liver function tests, metabolic panel, serum calcium, amylase, lipase was normal. ECG and ECHO done were normal. Stool examinations were also normal. Upper GI scopy was done which showed multiple nodular lesions with ulcerations noted in fundus, body of stomach (Figure 1A) and ulcerations in the first part of duodenum (Figure 1B). Serum fasting gastrin level was more than 1 lakh pg/ml. CECT Abdomen revealed a large well defined solid lesion 12×12×10 cm with central necrotic areas and post contrast enhancement seen on bowel wall predominantly in second part of duodenum and head of pancreas, with two adjacent satellite lesions of 4×4 cm (Figure 1C, D).

Histopathological examination from nodular lesions of stomach showed findings of chronic gastritis with enterochromaffin cell hyperplasia (Figure 2A). USG guided FNAC of lesion arising from pancreas showed findings suggestive of Neuroendocrine tumor (Figure 2B). Considering the patient persistent symptoms with failed medical management, enlarging mass per abdomen and suspicion of malignant lesion, patient was planned for surgical intervention. During Surgery, there was a smooth walled mass lesion of size 12×12×7.5 cm seen to be arising from head of the pancreas and root of mesentery with close contact with Superior mesenteric vein and middle colic vessels (Figure 3A). Three satellite lesions each measuring approximately 4×4 cm, were found in three locations: one posterior to D2, another along the lateral wall of D2 and the third inferior to D3. En-block resection of the tumor done (Figure 3B). Cut section of the specimen showed intact pseudo capsule with areas of congestion. (Figure 3C). The tumor was histopathologically diagnosed using hematoxylin and eosin which showed a well encapsulated lesion. The tumor cells were uniformly round with eosinophilic cytoplasm and oval nuclei arranged in zellballen pattern with smooth chromatin and inconspicuous small nucleolus (Figure 3D). Pseudo rosettes are also seen with areas of hemorrhage and Immunohistochemical analysis performed showed strong positivity for synaptophysin (Figure 3E) and positive for Cytokeratin 20. The cellular proliferative index assessed by Ki-67 was <1%. The histopathological features were consistent with grade 1 well differentiated neuroendocrine tumor.

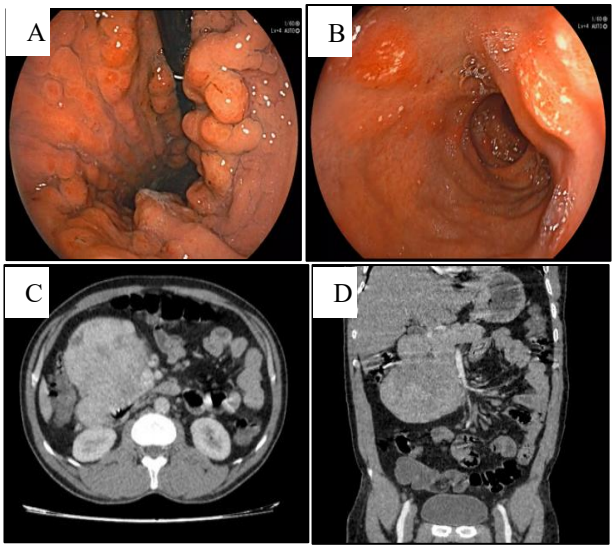

Figure 1: (A) Upper GI endoscopy image suggestive of nodular ulcerated lesions in stomach; (B) upper GI endoscopy showing duodenal ulcers; (C) axial cut of CECT abdomen showing giant tumor of size 12×12×10 cm arising from head of pancreas; (D) coronal cut of CECT abdomen showing tumoral extension to root of mesentery.

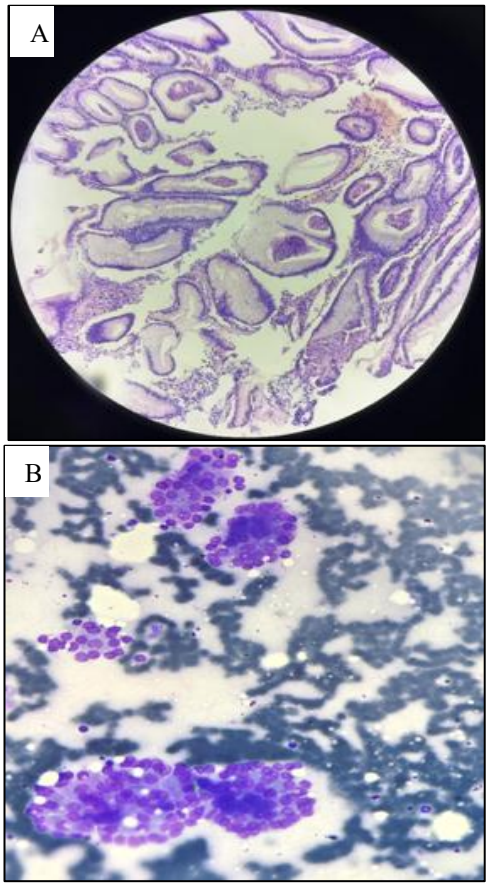

Figure 2: (A) HPE from gastric nodular tissue – Enterochromaffin cell hyperplasia; (B) USG guided FNAC from giant lesion showing uniform small round blue cell suggesting neuroendocrine tumor.

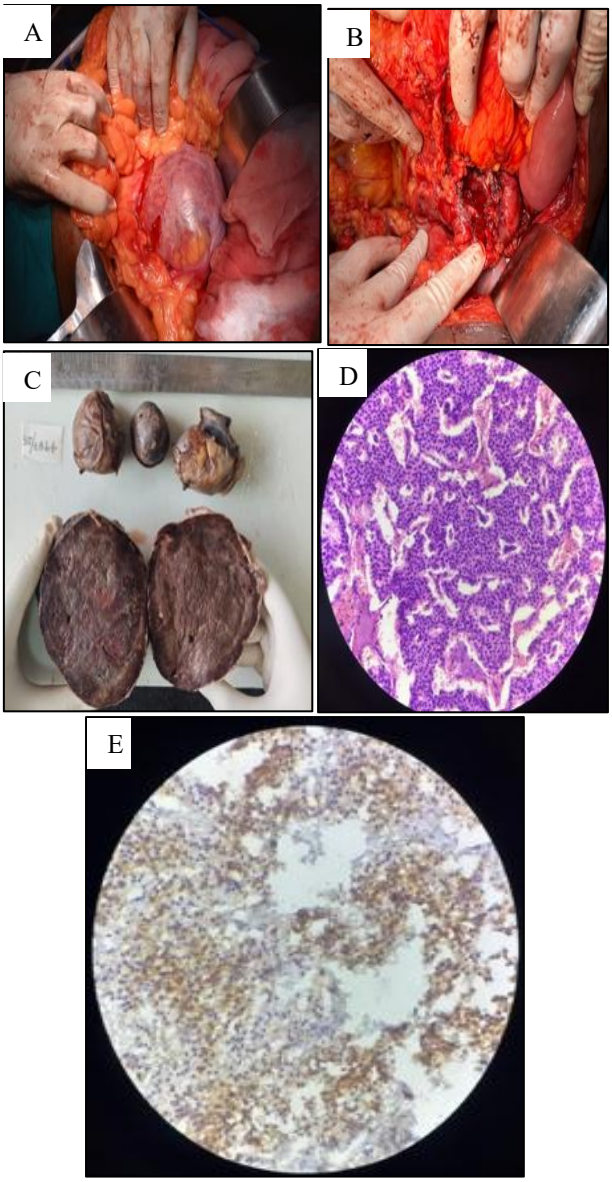

Figure 3: (A) Intraop image giant lesion; (B) intraop image post resection; (C) specimen consists of four discrete globular masses, largest measuring 12×12×7.5 cm. Externally capsulated with congested vessels and solid consistency. Cut surface shows grey brow to blackish colouration with firm and homogenous appearance with areas of congestion; (D) microscopic 20X image showing small round tumor cells with eosinophilic cytoplasm and oval nuclei arranged in zellballen pattern with smooth chromatin and inconspicuous small nucleolus suggesting a neuroendocrine tumor; (E) IHC showing strong positivity for Synaptophysin with Ki-67 <1%.

The clinical symptoms, associated with high gastrin levels and neuroendocrine tumor in our patient confirms the diagnosis as Zollinger Ellison syndrome. Following the Enbloc resection of lesions, patient symptoms subsided and recovered well. Gastrin level dropped to 484 pg/ml. Patient was under follow-up for next 1 year monitoring his symptoms and gastrin levels and no recurrence were observed.

DISCUSSION

As the study by Zhang et al explains that gastrinoma is a rare neuroendocrine tumor.[1] Second most common type of NET tumor after Insulinoma. Atypical presentation with non-specific complaints such as chronic diarrhea, weight loss and fatigue and its rarity makes delay in preoperative diagnosis.[3,5] Our patient presented with chronic diarrhea and Abdominal pain due to gastritis which was transiently responding to proton pump inhibitors. Always consider neuroendocrine tumours as an initial differential diagnosis for patient with non-specific or unexplained gastrointestinal symptoms. Gastrinoma is of two types. Sporadic (75%) or could be associated with MEN1 syndrome (25%) which is characterised by parathyroid, pancreatic and pituitary tumors manifesting as hyperparathyroidism, pancreatic NETs and adenoma respectively.[1]

The patient had no association with MEN1 syndrome. In our case due to the chronicity of symptoms and non-resolution of symptoms with antibiotics and proton pump inhibitors and a palpable mass per abdomen. OGD scopy was done which revealed extensive gastric nodules with ulcers at atypical locations and prominent mucosal folds. Biopsy from the ulcerative lesions and folds showed enterochromaffin cell hyperplasia with chronic gastritis which was pointing towards Gastrinoma even though a mass per abdomen was contradicting the same. Serum fasting gastrin levels were unusually high was suggesting and CECT revealed three major lesions within the Gastrinoma triangle.

Gastrinoma triangle, is bounder medially by pancreatic head and body, superiorly by bile ducts, inferiorly by the duodenal second and third portions.[3,6] For sporadic gastrinomas, Surgery should include laparotomy, complete enucleation/ resection of the primary, routine regional lymphadenectomy (at least in the gastrinoma triangle) and intraoperative liver exploration as the only curative treatment, as lymph node and liver metastasis are frequent and surgery is shown to reduce the rate of development of liver metastasis which is considered to be the most important prognostic factor for long term survival.[4,7-9]

Conservative treatment with proton pump inhibitors, somatostatin analogues like octreotide, lanreotide and chemotherapy with streptozotocin and 5-Fluorouracil or doxorubicin and molecular targeted agents like sunitinib and everolimus is only recommended for patients who are unsuitable for surgery or for patients with widespread metastasis.[1,10] Pancreatic tumor distant from the pancreatic duct can be enucleated. Resections are required when tumor is close to pancreatic duct (<3 mm). Distal pancreatic resection should be performed for caudally located tumors and duodenectomy performed to

detect small duodenal gastrinomas. For sporadic left sided pancreatic Gastrinoma, central or distal pancreatectomy (with or without splenectomy) can be.[7] In selected patients with pancreatic head Gastrinoma and those with local recurrence or persisting tumor after previous surgery, pancreaticoduodenectomy may be an alternative. In patient with MEN 1/ZES, surgery without Whipple resection is associated with > 90% recurrence.[7]

CONCLUSION

This case highlights a rare clinical scenario of a giant (>12 cm) multifocal gastrinoma associated with Zollinger–Ellison syndrome, an entity not reported in literature. Surgical resection remains the cornerstone and only curative treatment, while conservative approaches are reserved for patients unfit for surgery or with disseminated metastatic disease.

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