



Case Report

Type 3 gastric neuroendocrine tumor presenting as a solitary gastric fundus polyp: A case report

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ABSTRACT

Gastric neuroendocrine tumors are uncommon neoplasms arising from neuroendocrine cells of the gastric mucosa. They are classified into three main clinical types based on underlying etiology. We present the case of a 45-year-old woman with a 6-month history of epigastric discomfort and postprandial fullness. Upper gastrointestinal endoscopy revealed a solitary 1.5 cm polypoid lesion in the gastric fundus. Histopathological evaluation confirmed a well-differentiated Grade 1 gastric neuroendocrine tumor. The patient had no history of autoimmune atrophic gastritis, and serum gastrin was only mildly elevated, supporting a diagnosis of type 3 (sporadic) gastric neuroendocrine tumor. Endoscopic snare polypectomy was performed successfully, and follow-up revealed no recurrence. This case highlights the importance of recognizing type 3 gastric neuroendocrine tumors and reinforces the role of endoscopic resection in selected small, well-differentiated lesions.

Keywords: Endoscopic resection, Gastric neuroendocrine tumor, Polypectomy, Type 3 gastric NET

INTRODUCTION

Neuroendocrine tumors are neoplasms that arise from cells exhibiting both endocrine and neuronal characteristics. These cells are present throughout the gastrointestinal tract and form the diffuse neuroendocrine system. Gastric neuroendocrine tumors account for a small percentage of gastrointestinal neuroendocrine tumors and are classified into three types based on their pathogenesis. Type 1 tumors are associated with autoimmune atrophic gastritis; type 2 tumors occur in the setting of Zollinger-Ellison syndrome or multiple endocrine neoplasia type 1; and type 3 tumors are sporadic, typically solitary, and have greater malignant potential. We report a case of type 3 gastric neuroendocrine tumor in a middle-aged woman, focusing on the diagnostic pathway, management, and follow-up recommendations.

CASE REPORT

A 45-year-old woman presented with a 6-month history of epigastric discomfort, postprandial fullness, bloating, and intermittent nausea. There was no significant medical or family history. Upper gastrointestinal endoscopy on 14 December 2024 revealed a solitary polypoid lesion measuring ~1.5 cm, arising from the gastric fundus just below the gastroesophageal junction [Figure 1]. The mucosal surface was smooth and intact. Biopsy samples were obtained.



Figure 1: Upper gastrointestinal endoscopy showing a solitary, smooth, pedunculated polypoid lesion in the gastric body/fundus region. The lesion appears ~1.5-2 cm in size with intact overlying mucosa.

Histopathological examination demonstrated features of a well-differentiated neuroendocrine tumor, Grade 1, with a Ki-67 proliferation index of 2% and <2 mitoses per 10 high-power fields. Immunohistochemistry showed chromogranin A positivity, synaptophysin positivity, and a Ki-67 labeling index of 2%.

Endoscopic ultrasound (EUS) was considered; however, it could not be performed due to financial constraints and the unavailability of EUS at our center. Further staging investigations to assess involvement of other sites or distant metastasis, including computed tomography and functional imaging, were not performed due to the patient's financial limitations.

Laboratory evaluation revealed serum gastrin of 170 pg/mL (reference <100 pg/mL) and vitamin B12 level of 240 pmol/L (reference 133-675 pmol/L). There was no endoscopic or serological evidence of autoimmune atrophic gastritis.

The patient underwent endoscopic snare polypectomy on 19 December 2024 [Figures 2-4]. The procedure was completed without complications, and the patient was discharged on the same day. Repeat histopathology confirmed complete excision of a well-differentiated Grade 1 gastric neuroendocrine tumor with negative margins.

Representative histopathology microphotographs are provided in the revised manuscript. Follow-up at 3 and 6 months showed no recurrence, and the patient remains asymptomatic. Regular endoscopic surveillance every 6-12 months was advised.

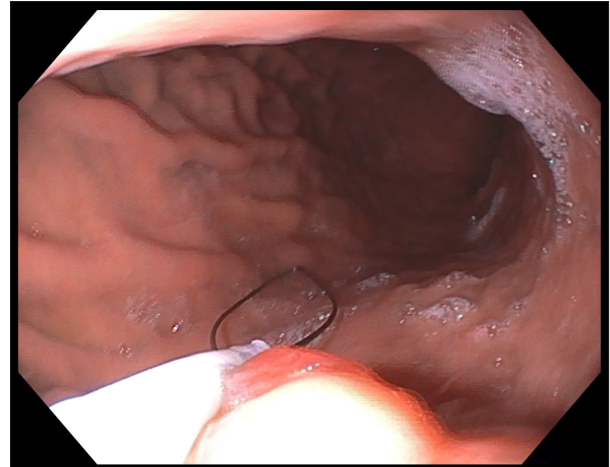


Figure 2: Snare placement around the base of the gastric polyp during endoscopic polypectomy. The pedunculated morphology of the lesion is clearly visualized.



Figure 3: Post-polypectomy mucosal bed showing a clean base with minimal oozing and no evidence of perforation or active bleeding following successful en bloc excision.

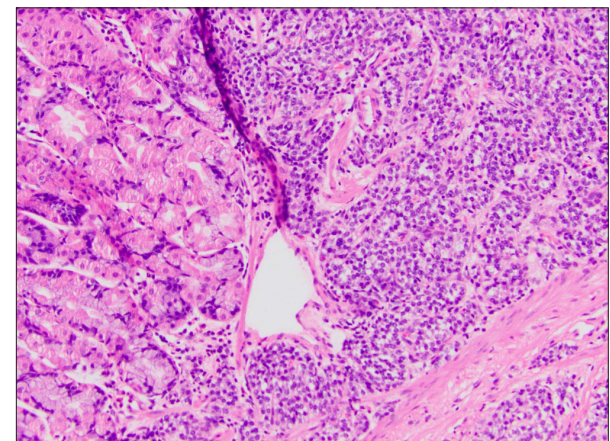


Figure 4: Type 3 gastric neuroendocrine tumors arise in the absence of a known etiology (atrophic gastritis, Zollinger-Ellison, etc.). Hematoxylin and eosin, ×400 magnification.

DISCUSSION

Neuroendocrine cells are specialized cells that receive neuronal signals and release hormones into the bloodstream. These hormones regulate a wide range of physiological functions, resulting in close coordination between the nervous and endocrine systems, a process known as neuroendocrine integration.¹ Neuroendocrine cells are found in multiple organs, including the pituitary gland, parathyroid glands, and the adrenal medulla, which are predominantly composed of these cells. In addition, neuroendocrine cells are scattered throughout the gastrointestinal, respiratory, and pancreatic tracts, collectively forming the diffuse neuroendocrine system.²

Gastric neuroendocrine tumors are classified into three major types based on their underlying cause, biological behavior, and clinical features.³ Type 1 tumors are associated with autoimmune atrophic gastritis and resultant hypergastrinemia, type 2 tumors occur in patients with Zollinger-Ellison syndrome and multiple endocrine neoplasia type 1, and type 3 tumors are sporadic, typically solitary, and not associated with hypergastrinemia or autoimmune gastritis.⁴ They tend to be more aggressive than type 1 and 2 tumors.

Histological grading

Gastric neuroendocrine tumors are graded based on the Ki-67 proliferation index and mitotic activity.

Diagnosis

Diagnosis of gastric neuroendocrine tumors involves multiple steps: Upper gastrointestinal endoscopy with biopsy to evaluate the size, number, and morphology of lesions. Assessment for atrophic gastritis or other mucosal abnormalities. Laboratory tests, including fasting serum gastrin and vitamin B12 levels. Staging investigations, when indicated, such as computed tomography (CT), octreotide scan, gallium-68 DOTATATE positron emission tomography/computed tomography (PET/CT), or EUS.

Treatment

Type 1 gastric neuroendocrine tumors

Type 1 gastric neuroendocrine tumors are usually indolent, and lesions smaller than 1–1.5 cm with Grade 1 histology are typically managed with endoscopic surveillance, as most remain stable over time. Endoscopic resection is recommended for selected lesions larger than 1.5 cm or when only a few polyps are present. Antrectomy, and rarely subtotal or total gastrectomy, may be considered for larger lesions or those with Grade 2 histology. Long-acting

somatostatin analogues are rarely indicated, while gastrin or cholecystokinin-2 receptor antagonists are promising therapeutic options that are still under clinical evaluation. Management of associated pernicious anemia is also an important component of treatment.

Type 2 gastric neuroendocrine tumors

Type 2 gastric neuroendocrine tumors are commonly associated with Zollinger–Ellison syndrome and are managed by controlling acid hypersecretion with high-dose proton pump inhibitors. Surgical removal of the gastrinoma is advised when feasible, including pancreaticoduodenectomy in selected cases. Endoscopic or surgical resection of gastric neuroendocrine tumors is performed depending on the size and number of lesions, and somatostatin analogues are used for unresectable tumors. Evaluation and management of associated multiple endocrine neoplasia type 1 features, including genetic screening of family members, are essential in these patients.

Type 3 gastric neuroendocrine tumors

Type 3 gastric neuroendocrine tumors are usually more aggressive and require complete removal of the tumor, most often by surgical resection unless metastatic disease is present. Endoscopic resection may be considered in selected cases with solitary tumors smaller than 2 cm and with Grade 1 or Grade 2 histology. In patients with metastatic disease, somatostatin analogues are used for Grade 1 or Grade 2 tumors, peptide receptor radionuclide therapy is considered for progressive disease, and chemotherapy is indicated for Grade 3 tumors.

CONCLUSION

Type 3 gastric neuroendocrine tumors are rare and require accurate diagnosis for appropriate management. This case illustrates that small, well-differentiated type 3 tumors can be effectively treated with endoscopic resection. Regular surveillance is essential to detect recurrence early.

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