Case Report

A Man with Diarrhea

Kayalvizhi Rajini, T. Rajkumar Solomon, Aravind Arumugham, Balamurali Rangachari

Department of Digestive
Health and Disease, Kilpauk
Medical College, Chennai,
Tamil Nadu, India

ABSTRACT

Diarrhea, a common presenting complaint which we come across in our day-to-day practice, is often self-limited but can be challenging at times. Here, we report one such patient. A 54-year-old male presented with diarrhea, abdominal pain, anorexia, and weight loss. Routine blood and stool examination was not helpful. A colonoscopy done elsewhere showed a fistulous opening in the anal canal for which he underwent a fistulectomy. Esophagogastroduodenoscopy revealed severe esophagitis, multiple duodenal ulcers, and a fundic gland polyp from which biopsies were taken. The fundic gland polyp showed oxyntic gland hyperplasia. Basal acid output and serum fasting gastrin subsequently measured were also high. A diagnosis of Zollinger–Ellison syndrome was made, and the patient was started on high-dose proton-pump inhibitors, to which he had excellent response. Repeat endoscopy showed resolutions of all lesions. Endoscopic ultrasound (EUS) and DOTA (Gallium (68Ga) DOTA-TATE) scan revealed a tumor in the duodenum and pylorus, respectively. On surgical exploration, the tumor was identified in the second part of the duodenum and was removed. The patient has remained asymptomatic since then.

KEYWORDS: Basal acid output, chronic diarrhea, Zollinger–Ellison syndrome

INTRODUCTION

Chronic diarrhea can sometimes be difficult to diagnose, especially for the primary care physician. Of the varied causes of diarrhea, Zollinger–Ellison syndrome (ZES) is a frequently underdiagnosed condition.^[1,2] Although many advances have been made in diagnosing ZES, measurement of basal acid output (BAO) still holds an important place, especially in a resource-limited setting.

CASE REPORT

A 54-year-old man working in cooperatives came to our hospital in May 2016 with 5 months of watery diarrhea moving his bowels 15–20 times/day with nocturnal episodes and not associated with tenesmus, blood, or mucus in stool. He experienced severe, colicky constant upper abdominal pain that prevented him from eating. Despite fasting, he continued to pass watery stools and had lost 15 kg over 5 months. He did not report to have fever, skin rashes, tremors, or joint pain.

Three months before this admission, he received a diagnosis of fistula-in-ano by a primary care physician,

for which he underwent fistulectomy and was treated with antibiotics. A colonoscopy done at that time showed a normal study up to the cecum except for a fistulous opening in the anal canal. He had no other medical or surgical comorbidities in the past.

On admission, he was visibly distressed due to pain and was severely dehydrated. His physical examination was remarkable for an intensely tender upper abdomen and a healed perianal scar.

INVESTIGATIONS

Blood work showed leukocytosis, predominant neutrophils, and prerenal azotemia. His stool examination and culture were negative. Esophagogastroduodenoscopy (EGD) revealed severe esophagitis and multiple ulcers with necrotic base in the duodenum and upper jejunum from which biopsies revealed nonspecific inflammation [Figure 1]. A small fundic polyp was noted, biopsy of which showed oxyntic

Address for correspondence: Dr. Kayalvizhi Rajini,
Q 101, 3rd Avenue, Anna Nagar, Chennai - 600 040,
Tamil Nadu, India.
E-mail: kayalamma@gmail.com

Access this article online

Quick Response Code:

Website: www.jdeonline.in

DOI: 10.4103/jde.JDE_27_17

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How to cite this article: Rajini K, Solomon TR, Arumugham A, Rangachari B. A man with diarrhea. J Dig Endosc 2018;9:85-7.

Figure 1: Endoscopy showing multiple duodenal ulcers

Figure 3: Complete healing of ulcers in the duodenum after proton-pump inhibitor therapy

gland hyperplasia. Considering a possibility of ZES, we took the patient off proton-pump inhibitor (PPI) and and H₂ receptor antagonists and measured basal acid output.^[3] BAO was elevated (11.4 mmol/h). The gastric pH was <2. Subsequent serum fasting gastrin levels measured were also high (404 pg/ml). A contrast-enhanced computed tomography of the abdomen was unremarkable except for prominent rugal folds. EUS showed a submucosal nodule in the first part of duodenum with an inconclusive fine-needle aspiration cytology. ⁶⁸Ga-DOTA-NOC scan revealed avid uptake in the medial wall of pylorus of the stomach, highly suggestive of neuroendocrine tumor (NET) [Figure 2].

Treatment

Initially, he was hydrated intravenously, after which renal parameters normalized. The patient was started on high-dose PPIs (omeprazole 120 mg/day) with which he had immediate resolution of abdominal pain and diarrhea. Ten days after PPI therapy, we repeated EGD which showed complete resolution of the esophagitis and ulcers in the duodenum and jejunum [Figure 3]. The patient later underwent pyloroduodenotomy, and a thorough examination of the pylorus and duodenum

Figure 2: DOTA scan showing prominent uptake in the region of the pylorus

Figure 4: Resected tumor

was undertaken. The pylorus was normal, and the first part of the duodenum had a submucosal nodule which was excised [Figure 4]. Pathologists reported it as NET showing insular and trabecular architecture. Unfortunately, due to various reasons, we could not do immunohistochemistry staining.

Follow-up

Postsurgery, the patient recovered well and is on regular follow-up. He remains asymptomatic till date.

DISCUSSION

ZES is caused by ectopic secretion of gastrin by a NET (gastrinoma), which causes excessive gastric acid secretion, characteristically causing peptic disease (often severe) and/or gastroesophageal reflux disease with an incidence of 0.1–3 persons/million/year. About 60% to 90% are malignant. More than half of them are in the duodenum. In our case, the DOTA scan revealed uptake in the medial wall of the pylorus, but it was

actually located in the first part of the duodenum which was confirmed on surgery.

Serum fasting gastrin levels are typically >1000 pg/dl. Sometimes, one cannot solely rely on serum fasting gastrin alone.^[3] If levels are only modestly increased, as in our case, a combination of tests can be used to establish the diagnosis. In a country like India, where secretin is not widely available, the measurement of BAO is a useful and easy test to do.^[4]

Histologically, most gastrinomas are well differentiated and show a trabecular and pseudoglandular pattern. Their proliferative activity (i.e., the Ki67 index) varies between 2% and 10% but is mostly close to 2%. Immunohistochemically, almost all gastrinomas stain for gastrin.

Tumor-localizing methods are many, of which somatostatin receptor scintigraphy, EUS, and intraoperative ultrasound are more yielding. Pharmacological control of acid secretion can be achieved with twice-daily PPI (starting from 60 mg/day for omeprazole) or H₂-receptor antagonists. Most authorities recommend surgical exploration of ZES patients for a possible curative resection as long as diffuse metastatic disease to the liver is absent and the patient does not have Multiple Endocrine Neoplasia –Type I. In advanced cases, chemotherapy and somatostatin analogs/peptide receptor radionuclide therapy have been used.^[5]

Learning points

1. ZES, though uncommon, should be considered in all patients with painful watery diarrhea

2. BAO measurement and pH measurement are simple, cost-effective tests that can be collected at bedside and can guide higher investigations.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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