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Neuroendocrine tumor in the cecum: An unusual cause of chronic diarrhea in a young adult

Tumor neuroendocrino en el ciego; una causa inusual de diarrea crónica en un adulto joven

Neuroendocrine tumors (NETs) are epithelial tumors with neuroendocrine differentiation that synthetize or secrete bioactive hormones and monoamines.[1] They are an infrequent cause of chronic diarrhea and their diagnosis in young patients is rare.

NETs account for 0.4% of all tumors and 2.5% of gastrointestinal tumors. Sixty-five percent of NETS arise in the gastrointestinal tract and pancreas. A total of 12.9% are found in the colon, of which 70% affect the right-sided colon. Even though they are responsible for less than 1% of cases of chronic diarrhea, it is essential to consider them in the differential diagnosis, after ruling out other more frequent causes.

A 28-year-old man had a past medical history of obsessive-compulsive disorder treated with lamotrigine, vilazodone, and clonazepam, which he decided to suspend 4 months before his initial consultation. He sought medical attention due to 12 months of presenting with Bristol 6–7 stools, abundant and explosive, with a frequency of 4–5 bowel movements per day, many of which were nocturnal, that did not decrease with fasting. He stated having steatorrhea, and no creatorrhea or lientery. He reported the presence of occasional mucus and blood in his stools, tenesmus, and colicky, nonradiating abdominal pain in the mesogastrium of 4/10 intensity, associated with food intake, with relief after bowel movements. Physical examination revealed pain upon superficial and deep palpation of the flank and right iliac fossa and a body mass index of 18. He had previously been seen for those symptoms, undergoing upper gastrointestinal endoscopy, resulting in a diagnosis of irritable bowel syndrome and treatment with antispasmodics, with no improvement.

The patient was now studied for chronic diarrhea, ordering blood and stool tests (Table 1). Esophagogastroduodenoscopy was within normal parameters. The histopathologic study reported non-atrophic congestive gastropathy, with no villous atrophy in the duodenum. Colonoscopy revealed a 30 mm exophytic lesion in the cecum, with an ulcerated surface and friable to manipulation (Fig. 1A). Contrast-enhanced abdominopelvic tomography identified a

Table 1 Results of initial tests carried out in the approach to the patient.

Initial testing	Results	Normal range
Leukocytes	7.87 10^3 / ul	4.4–10
Neutrophils	5.63 10^3 / ul	2–8
Lymphocytes	1.51 10^3 / ul	1–4.4
Hemoglobin	14.9 g/dl	13.5–17.3
MCV	83.8 fl	76–96
MCH	29.1 pg	28–33
Glucose	88 mg/dl	50–100 mg/dl
TSH	2.56 uIU/mL	0.27–4.2
Free T3	2.84 pg/mL	1.71–3.71
Free T4	1.16 ng/dl	0.8–2
Sodium	141.4 mEq/l	135–145 mEq/l
Potassium	4.21 mEq/l	3.5–4.5 mEq/l
Calcium	9.8 mg/dl	8.2–10.5 mg/dl
Magnesium	1.93 mg/dl	1.7–2.2 mg/dl
Creatinine	1.08 mg/dl	0.70–1.20 mg/dl
Urea	29 mg/dl	16.6–48.5 mg/dl

Table 1 Results of initial tests carried out in the approach to the patient.

Initial testing	Results	Normal range
Total bilirubin	0.40 mg/dl	0.1–1.2
Direct bilirubin	0.17	0.0–0.14
Indirect bilirubin	0.23	
AST	14 IU/l	0–40 IU/l
TGP	16 IU/l	0–45 IU/l
Alkaline phosphatase	146.3 IU/l	0–400 IU/l
GGT	12 IU/l	12–64 IU/l
Albumin	4.72 g/dl	3.5–5.2 g/dl
ESR	2 mm/h	0–10
CRP	0.3 mg/l	0.0–5.00
Immunoglobulin A (IgA)	275 mg/dl	83–453 mg/dl
IgA anti-tissue transglutaminase antibodies (IgA anti-tTg)	4.40 U/mL	0–25 U/mL
Giardia lamblia antigen	Negative	
Fecal elastase	220 mcg/g	>200
Fecal calprotectin	342 mcg/g	<50 mcg/g
Stool culture	Pathogen not isolated	

AST: aspartate transferase; ALT: alanine transaminase; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; GGT: gamma-glutamyl transferase; MCH: mean corpuscular hemoglobin; MCV: mean corpuscular volume; TSH: thyroid stimulating hormone; T3: triiodothyronine; T4: thyroxine.

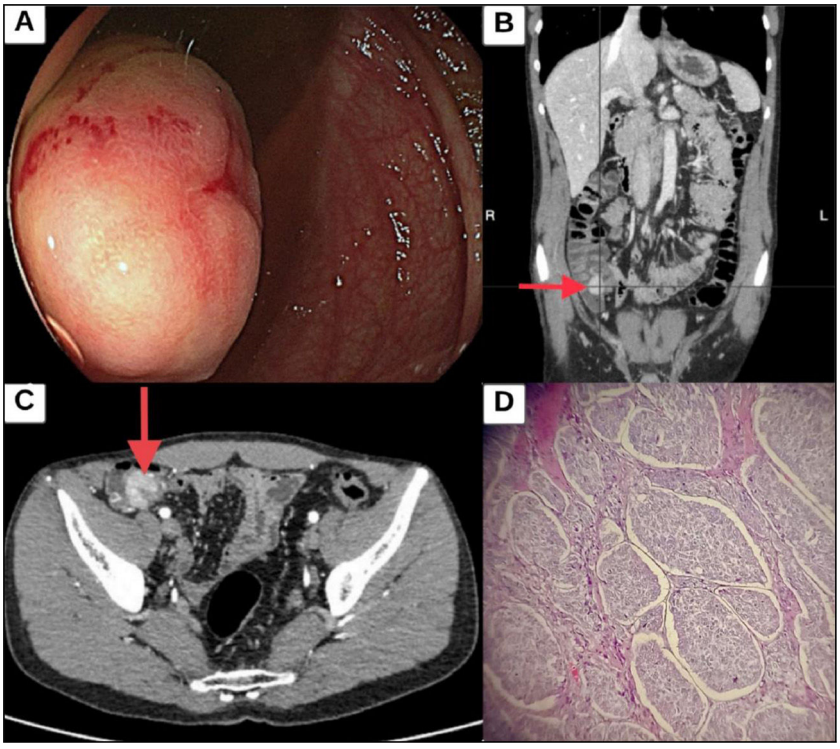

Figure 1 (A) Colonoscopy showing a 3 cm lesion in the cecum. (B and C) Coronal and axial views of contrast-enhanced CAT scans, showing a lesion at the medial and inferior edge of the cecum, characterized by an 18 mm focal thickening of the wall, with endoluminal extension. (D) Histopathologic section of a grade 2, well differentiated neuroendocrine tumor, showing an epithelial tumor with nest-shaped growth, cells with moderately pleomorphic nuclei, and lax or dense chromatin, as well as mitosis and scant cytoplasm. The tumor is developed in a vascularized stroma and shows infiltration through all the layers of the colon, extending beyond the serosa.
Source: Clinical history of the patient.

lesion on the medial and inferior edge of the cecum, with focal thickening measuring 18 mm, necrosis, and paracecal lymph nodes (Fig. 1B and C).

The patient then underwent right hemicolectomy with lymph node resection. His preoperative chromogranin A level was 117 ng/mL (NV < 85 ng/mL), and his somatostatin level was 6 ng/mL (NV < 10 ng/mL), which were within normal parameter ranges. The histopathologic study reported a well-differentiated grade 2 neuroendocrine tumor (NET) (pT2, pN1, pM0) with invasion in all layers, extending beyond the serosa, and exhibiting lymphovascular permeation. The tumor was positive for chromogranin +++, synaptophysin +++, and Ki-67 (3–20%), and metastasized to 2/28 lymph nodes (Fig. 1D).

Postoperative controls at 2, 4, and 6 months showed improved bowel movement pattern and mild abdominal pain. Serologic studies 12 months later: chromogranin A 58 ng/mL (NV 85 ng/mL), 5-hydroxyindoleacetic acid (5-HIAA) in urine at 24 h of 2 mg (NV < 2—9 mg/24 h), and Tc-99 m octreotide scintigraphy negative for somatostatin receptors, indicative of remission. Colonoscopy and biopsies of the anastomosis showed no tumor activity. Chronic diarrhea, defined as three or more bowel movements per day with soft stools (Bristol 6–7) for more than 4 weeks, is a frequent cause of gastroenterology consultation and is more common in adults >65 years of age. Its approach requires a detailed anamnesis and the identification of alarm symptoms, to differentiate organic disease and functional disease, and select the patients who warrant an early invasive study. It includes a wide spectrum of conditions, such as tumors, inflammation of the colon or small bowel, malabsorption, and motility disorders, which leads to performing an excessive number of studies. Of the clinical forms, secretory diarrhea is produced by an imbalance between the intestinal absorption and secretion processes, characterized by an increase in fluid and electrolyte secretion from the plasma into the intestinal lumen. It is clinically manifested as abundant liquid stools (>1 liter per day) that persist even during fasting and are predominantly nocturnal.[2]

In our patient, the presence of alarm symptoms of large volume, continuous, nocturnal stools associated with weight loss, together with elevated fecal calprotectin, guided the approach toward an underlying organic cause.[3]

NETs of the colon are differentiated from neuroendocrine carcinomas by their degree of aggressiveness. They have an estimated prevalence of 5.2/100,000 persons/year,[4] and a mean patient age at diagnosis of 63 years.[5] Diagnosis can be delayed 5–7 years, underlining the importance of timely evaluation.[6] NETs account for 25% of all gastroenteropancreatic neuroendocrine carcinomas. They are aggressive in the colon, unlike those located in the appendix and rectum, which are more indolent. Their detection is mainly incidental but they are also associated with abdominal pain, rectal bleeding, chronic secretory diarrhea, weight loss and/or anorexia.[7] Gastroenteropancreatic NETs have a high metastatic capacity, affecting the liver, lymph nodes, peritoneum, bone, and lung.[8] Diagnosis is based on the combination of morphology through endoscopic, imaging, and immunohistochemical studies, due to the presence of serotonin-producing enterochromaffin cells. They are positive for synaptophysin, chromogranin A, and CD56 and should be classified as well-differentiated G1/G2/G3 tumors; when they share non-neuroendocrine histology, they should be classified as poorly differentiated.[9] Surgical resection is the treatment of choice whenever feasible.[10] Unfortunately, clinical and pathologic characteristics do not always enable patients at a high risk for recurrence or metastasis to be selected. Most of these tumors are detected at advanced stages and have a 5-year survival rate of 71% in stage 1 and 10% in stage IV.[11]

Ethical considerations

The authors declare that no experiments were conducted on humans or animals in this research. The information utilized was obtained from the patient's clinical records after receiving a written statement of informed consent from the patient. All the necessary means were carried out to guarantee data confidentiality, omitting all information that could identify the patient through images or data in the article. Thus, review or approval by the institutional ethics committee was not required.

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Declaration of competing interest

The authors declare that there is no conflict of interest.

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Endoscopic treatment of baroliths: A case report

Tratamiento endoscópico de bariolito: a propósito de un caso

In the early 1900s, contrast media began to be utilized in radiologic studies to evaluate soft tissues. Bismuth, iron, lead, and strontium were media that were not adequately regulated and so were discontinued.[1] In 1902, Walter Cannon pioneered the use of barium as a contrast medium, after which it replaced bismuth, which was needed by the war industry.[2] Barium sulfate is the most widely utilized agent in radiologic studies of the digestive tract and has become an essential tool because it is exact, efficient, cost-effective, and very low-risk.[1] However, adverse reactions associated with its use have been described, most of which are self-limited, although there are reports of case fatalities.[3] Among the complications are anaphylaxis, at 1 in 2.5 million cases,[3] intoxication with neurologic symptoms,[4] granuloma (first described in 1954),[5] barium extravasation, venous intravasation, accidental aspiration, and barium impaction.[1] Therefore, in addition to barium contrast, water-soluble contrast may be used for radiologic visualization of the gastrointestinal tract. The water-soluble medium does not cover the mucosa, as barium does, but passively fills the intestinal lumen and is indicated when there is suspected perforation or communication with the peritoneal cavity. Nevertheless, certain small perforations can only be visualized with barium, depending on their location. Water-soluble contrast use is also suggested in postoperative patients, in whom the peristalsis produced by barium is to be avoided.[6] A barolith is defined as the concretion of barium sulphate and feces in the intestine after the performance of a radiologic procedure with a barium contrast agent.[7] First described by Valladares in 1950,[8] there are only isolated cases appearing in the literature, along with one systematic review on articles describing a total of 31 cases published within the time frame of 1950 and 2006. Obstruction due to a barolith is reported as a late presentation, associated with a low-residue diet, dehydration, colonic dysmotility, fluid and electrolyte imbalance, diabetic enteropathy, and Parkinson's disease.[8] In 1978, the use of the water-soluble contrast medium, Gastrografin® (diatrizoate), was described as a therapeutic method. Four cases had successful results, given that the medium stimulates peristalsis and its high osmolarity dissolves the impaction, its transit is adequate, despite the presence of ileus, and it does not aggravate bowel obstruction. Nevertheless, no medical associations have made any recommendations, and there have been no subsequent studies.[9]

The symptoms associated with baroliths are abdominal pain, nausea, vomiting, constipation, obstruction, peritonitis due to barium, and intestinal perforation. The severe complications of megarectum and abdominal compartment syndrome may present. In 2019, Pathan et al. recommended that patients with risk factors for impaction, who underwent a barium study, have x-rays taken 2–3 days after the study and receive laxatives as a preventive measure.[10]

We present herein a case of complete bowel occlusion secondary to baroliths. The patient was initially treated conservatively, after which he underwent successful endoscopic disimpaction. This article aims to inform its readers about the potential complications associated with barium studies, which are infrequent and rarely described in the scientific literature.

An 80-year-old man had a medical history of cognitive decline, under treatment with memantine, donepezil, and quetiapine; hyperuricemia; squamous cell cancer of the left