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CASE REPORT

# Eosinophilic Enteritis Presenting with Recurrent Abdominal Pain and Bowel Obstruction in the Absence of Peripheral Eosinophilia: A Rare Case Report

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**Background:** Eosinophilic enteritis is a rare subtype of eosinophilic gastrointestinal disorders characterized by eosinophilic infiltration of the small intestine. Clinical presentation varies according to the depth of bowel wall involvement. Muscularis-predominant disease is exceptionally uncommon and frequently lacks peripheral eosinophilia, which poses considerable preoperative diagnostic difficulty.

**Case Presentation:** A 23-year-old woman presented with acute severe upper-quadrant abdominal pain, nausea, and vomiting, on a background of three similar hospitalizations over two years without a definitive diagnosis. Imaging showed segmental ileal narrowing with proximal small-bowel dilatation and ascites. Laboratory testing revealed leukocytosis without peripheral eosinophilia. Because of persistent obstruction, a limited right hemicolectomy with ileal resection was performed. Histopathology demonstrated dense eosinophilic infiltration (markedly increased, exceeding 50 eosinophils per high-power field) predominantly involving the muscularis propria and subserosa, with preserved mucosa. Peritoneal biopsies showed eosinophilic inflammation. Malignancy, tuberculosis, vasculitis, and parasitic infection were excluded. Postoperative recovery was uneventful.

**Conclusion:** Eosinophilic enteritis should be considered in the differential diagnosis of recurrent abdominal pain and small-bowel obstruction, even when peripheral eosinophilia is absent. Histopathological examination is essential for definitive diagnosis, and awareness is particularly important in resource-limited settings where advanced diagnostic modalities are unavailable.

**Keywords:** eosinophilic enteritis, eosinophilic gastrointestinal disorders, small-bowel obstruction, peripheral eosinophilia, muscularis propria, resource-limited settings, case report

## Background

Eosinophilic enteritis (EE) is a rare subtype of eosinophilic gastrointestinal disorders (EGIDs), a family of conditions that also includes eosinophilic esophagitis (EoE) and eosinophilic gastritis (EoG).[1] It was first described by Kaijser in 1937[2] and is characterized by eosinophilic infiltration within the bowel wall, most often affecting the stomach and small intestine.[3]

According to the Klein classification,[4] EE is divided into mucosal, muscular, and serosal forms based on the predominant intestinal layer involved, with clinical manifestations depending on the depth of involvement.[5] The muscularis-predominant subtype is extremely rare, representing only a small proportion of reported cases, and typically presents with bowel wall thickening and features of mechanical obstruction.[6,7] Because imaging and laboratory findings are nonspecific and peripheral eosinophilia may be absent, preoperative diagnosis is often not possible, and many patients are diagnosed only after surgical exploration and histopathological examination.

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We report a rare case of muscularis-predominant eosinophilic enteritis presenting with recurrent abdominal pain and small-bowel obstruction in the absence of peripheral eosinophilia, to emphasize its inclusion in the differential diagnosis of unexplained recurrent abdominal pain and to support timely management, particularly in resource-limited settings.

## Case Presentation

A 23-year-old woman presented to the emergency department with acute-onset severe upper-quadrant abdominal pain of two days' duration. The pain was intermittent and associated with nausea and vomiting. She denied fever, chills, constipation, or hematochezia. She had no history of inflammatory bowel disease, chronic constipation, diarrhea, tumors, hernia, or prior abdominal surgery. She specifically denied any use of nonsteroidal anti-inflammatory drugs (NSAIDs) or other medications known to cause eosinophilic infiltration, and she reported no food allergies or recent dietary changes. To protect patient confidentiality, all identifying information has been anonymized and written informed consent was obtained from the patient for publication of this case report and any accompanying images.

The patient had experienced three previous episodes of abdominal pain over the preceding two years, each requiring hospitalization, with no definitive etiology identified despite prior evaluation.

On physical examination, the abdomen was distended with upper-quadrant tenderness, guarding, and rebound tenderness. Although these signs may suggest peritoneal inflammation, there was no clinical or subsequent intraoperative evidence of generalized peritonitis or bowel perforation; the findings were attributed to marked bowel distension and localized eosinophilic inflammation of the peritoneum.

Initial laboratory evaluation revealed leukocytosis with a white-blood-cell count of $15.28 \times 10^9$/L, predominantly neutrophilic (88.4%), with a normal peripheral eosinophil count (0.3%). Serum electrolytes showed mild hypernatremia (147.2 mmol/L) with a normal serum potassium (3.9 mmol/L) (Table 1). Serum β-human chorionic gonadotropin (β-hCG) obtained at admission was negative (2.25 mIU/mL), excluding pregnancy. Human immunodeficiency virus (HIV) serology was negative. Total serum immunoglobulin E (IgE) was elevated, although the quantitative value was not retained in the laboratory record. Serum tryptase and antinuclear antibody (ANA) testing were not performed because these assays are not routinely available in our setting; this is acknowledged as a limitation.

A plain abdominal radiograph demonstrated dilated central bowel loops with multiple air–fluid levels (Figure 1). Contrast-enhanced computed tomography (CT) of the abdomen and pelvis, performed with intravenous and oral contrast, revealed long-segment bowel wall narrowing involving the terminal ileum and colon, with proximal small-bowel dilatation up to 4 cm and multiple air–fluid levels (Figure 2).

**Table 1** Initial Laboratory Findings on Admission

| Parameter | Patient Value | Reference Range |
|---|---|---|
| White-blood-cell count | $15.28 \times 10^9$/L | $4.0–11.0 \times 10^9$/L |
| Neutrophils | 88.4% | 40–75% |
| Eosinophils | 0.3% | 1–6% |
| Sodium | 147.2 mmol/L | 135–145 mmol/L |
| Potassium | 3.9 mmol/L | 3.5–5.1 mmol/L |
| Serum β-hCG | 2.25 mIU/mL | <5 mIU/mL (negative) |
| CA-125 | 13.05 U/mL | <35 U/mL |
| Alpha-fetoprotein (AFP) | 1.66 ng/mL | <10 ng/mL |
| Carcinoembryonic antigen (CEA) | 1.54 ng/mL | <5 ng/mL |
| HIV serology | Negative | Negative |

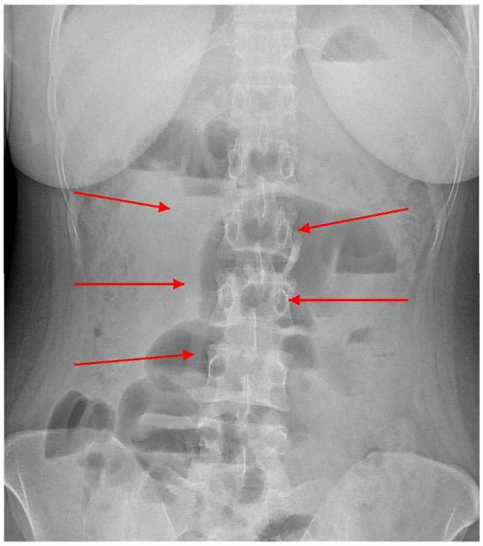

**Figure 1** Plain abdominal radiograph demonstrating dilated central small-bowel loops with multiple air–fluid levels (arrows).

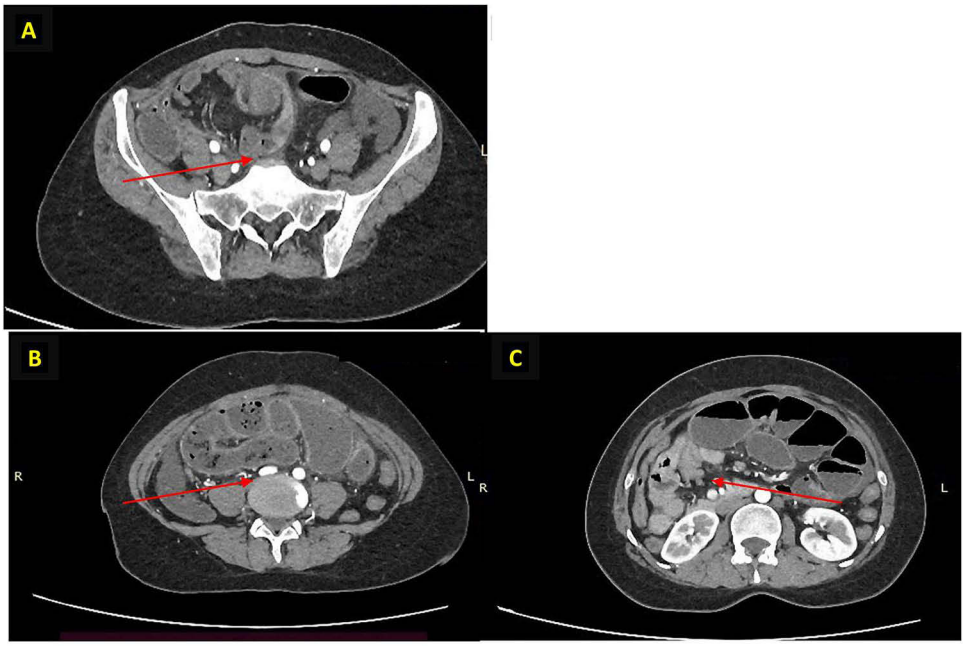


**Figure 2** Contrast-enhanced computed tomography (CT) of the abdomen and pelvis showing long-segment bowel wall narrowing involving the terminal ileum and caecum (arrows), with upstream small-bowel dilatation up to 4 cm and multiple air–fluid levels. (**A**) Axial pelvic view. (**B**) Axial mid-abdominal view. (**C**) Axial upper-abdominal view.

A repeat CT scan performed with intravenous, oral, and rectal contrast identified two discrete strictures, each approximately 3 cm in length, located in the mid-ileum and terminal ileum, respectively (Figure 3). There was interval improvement of the previously observed colonic involvement. Passage of stool during hospitalization excluded complete intestinal obstruction, and no discrete obstructing mass was identified.

Diagnostic paracentesis revealed exudative ascitic fluid, with a total protein level of 5.01 g/dL, albumin 2.94 g/dL, and lactate dehydrogenase (LDH) 330.10 U/L. Cytological examination was negative for malignant cells and demonstrated reactive mesothelial cells. Microbiological cultures showed no growth, and GeneXpert testing for *Mycobacterium tuberculosis* was negative (Table 2). Serum-ascites albumin gradient (SAAG), ascitic fluid total and differential cell counts, and adenosine deaminase (ADA) activity were not available because these tests are not routinely performed in

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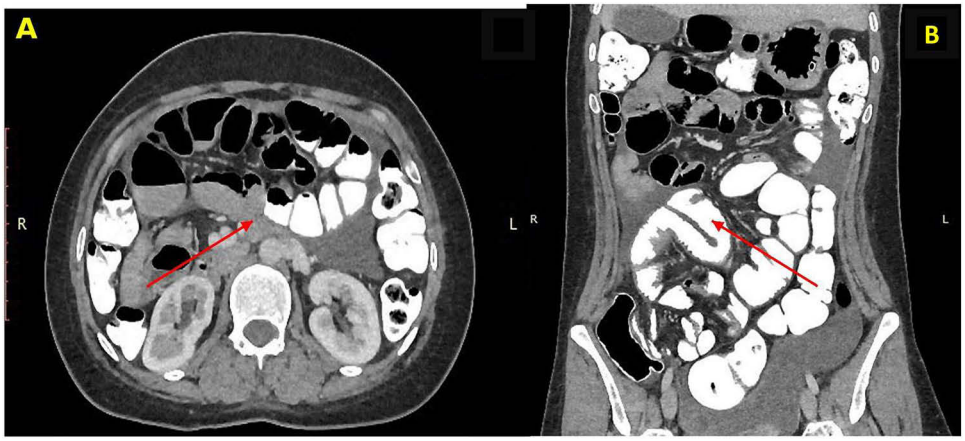


**Figure 3** Repeat CT scan performed with oral, intravenous, and rectal contrast, identifying two discrete strictures (arrows) of approximately 3 cm each, located in the mid-ileum and terminal ileum. (**A**) Axial view. (**B**) Coronal reconstruction.

our laboratory; this is acknowledged as a limitation. Tumor markers, including CA-125 (13.05 U/mL), alpha-fetoprotein (AFP, 1.66 ng/mL), and carcinoembryonic antigen (CEA, 1.54 ng/mL), were within normal limits.

Because of persistent small-bowel obstruction of unclear etiology, the patient underwent diagnostic laparoscopy, which revealed hemorrhagic ascites and centrally conglomerated dilated bowel loops with a thick fibrinous peritoneal covering and a stricture of the ileum near the ileocecal valve. The procedure was converted to open surgery. A limited adhesiolysis was performed to mobilize the bowel; the lead point of obstruction corresponded to the terminal ileal stricture rather than to the adhesions themselves, and the adhesions were judged to be secondary to the underlying transmural inflammation. A limited right hemicolectomy was then performed, incorporating the terminal ileal stricture, together with ileocolic anastomosis. Biopsies were obtained from associated peritoneal and mesenteric deposits. The mid-ileal stricture identified on preoperative imaging was not resected at the index operation, in order to preserve small-bowel length; the plan was to address this stricture medically with corticosteroids, with surgical intervention reserved for failure of medical therapy.

Gross examination of the resected bowel segment (15 × 3 cm) demonstrated an edematous bowel wall with focal hemorrhagic areas (Figure 4). Microscopic examination revealed dense eosinophilic inflammatory infiltration, markedly increased and exceeding 50 eosinophils per high-power field, predominantly involving the muscularis propria and subserosal layers (Figure 5). The mucosa was preserved and architecturally intact, and only scattered eosinophils were identified within the submucosa. Peritoneal biopsy specimens demonstrated prominent eosinophilic inflammatory infiltrates, also qualitatively estimated at more than 50 eosinophils per high-power field. There was no evidence of malignancy, granulomatous inflammation, vasculitis, or tuberculosis.

**Table 2** Ascitic Fluid Analysis

| **Parameter** | **Result** |
|---|---|
| Appearance | Hemorrhagic, exudative |
| Total protein | 5.01 g/dL |
| Albumin | 2.94 g/dL |
| Lactate dehydrogenase (LDH) | 330.10 U/L |
| Cytology | Negative for malignancy; reactive mesothelial cells |
| Microbiological culture | No growth |
| GeneXpert (*Mycobacterium tuberculosis*) | Negative |

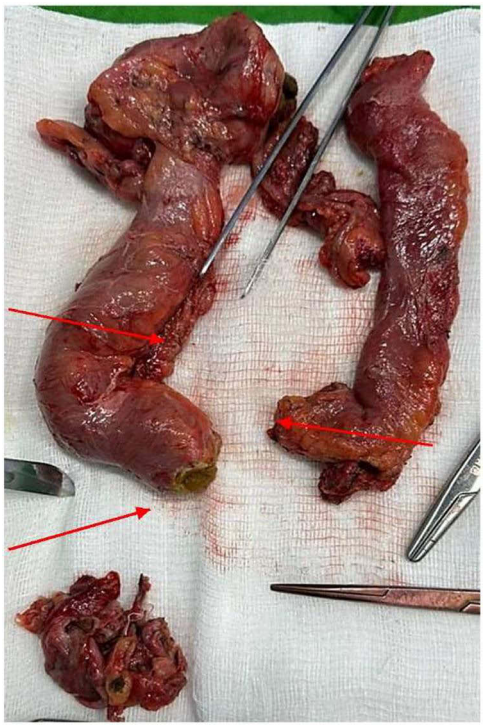

**Figure 4** Gross specimen of the resected ileocolic segment (15 × 3 cm) demonstrating an edematous bowel wall with focal hemorrhagic areas (arrows).

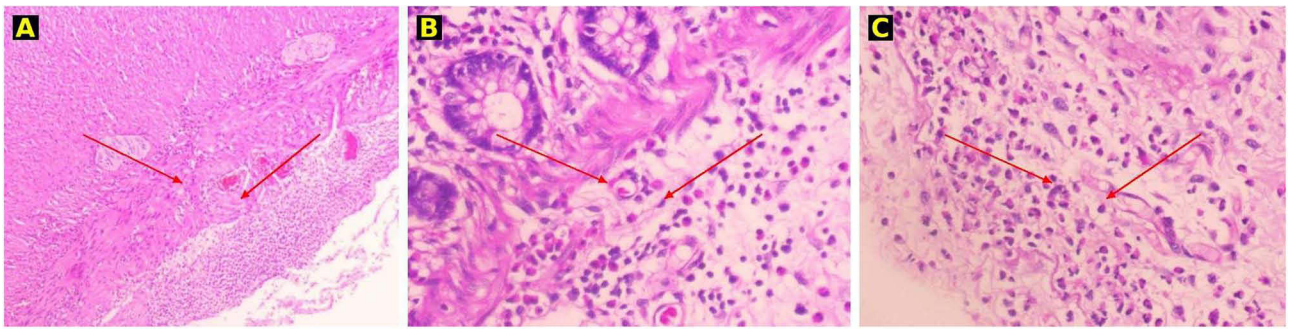


**Figure 5** Photomicrograph of the resected bowel (hematoxylin and eosin stain) showing dense eosinophilic inflammatory infiltration (arrows) predominantly involving the muscularis propria and subserosa, with preserved mucosa. (**A**) Low-power examination showing dense subserosal eosinophilic infiltrates. (**B**) High-power examination showing submucosal eosinophilic infiltrates. (**C**) High-power examination of peritoneal tissue showing eosinophilic infiltrates.

The postoperative course was uneventful. Follow-up imaging showed no anastomotic leak, with only minimal reactive pelvic collections and mesenteric stranding. The patient remained clinically stable and was discharged on postoperative day 7. After discharge, she was started on oral prednisolone at 0.5 mg/kg/day as maintenance therapy, with a planned slow taper over 8–12 weeks guided by clinical response. Continued outpatient follow-up with clinical and radiological review was arranged to monitor for recurrence and to assess the residual mid-ileal stricture.

## Discussion

Eosinophilic enteritis is an uncommon inflammatory disorder that is frequently underrecognized in patients with nonspecific gastrointestinal symptoms. It is rare globally, with fewer than 400 cases described since Kaijser's original report and an estimated prevalence of approximately 1 per 100,000 population.[4,6] Its true prevalence is likely underestimated[8] because of its nonspecific gastrointestinal manifestations, and patients frequently undergo repeated imaging and empirical treatment before a definitive diagnosis is established. Regional or national epidemiological data from the Horn of Africa, and from Somalia


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specifically, are not available; to the best of our knowledge, this is among the first detailed reports of muscularis-predominant EE from Somalia. The condition can occur across all age groups,[7] and its pathogenesis remain incompletely understood, although available evidence supports a role for immunoglobulin E (IgE)–mediated hypersensitivity and delayed T-helper type 2 (Th2) immune responses.[9,10]

In the present case, the patient presented with recurrent abdominal pain progressing to mechanical bowel obstruction, with radiologic evidence of intestinal wall thickening and histopathologic confirmation of dense eosinophilic infiltration. Diagnosis requires compatible gastrointestinal symptoms together with histopathological evidence of eosinophilic infiltration, in the absence of secondary causes of intestinal eosinophilia such as parasitic infection, drug reactions, inflammatory bowel disease, or malignancy. Endoscopic findings are variable and non-specific, and definitive diagnosis therefore relies on histopathological evaluation of intestinal biopsies, generally requiring dense eosinophilic infiltration exceeding 15–50 eosinophils per high-power field depending on the anatomical location.[11,12] In our patient, eosinophil density markedly exceeded this threshold within the muscularis propria and subserosa.

Eosinophilic enteritis is often mistaken for similar conditions, and in this patient, each important differential was systematically excluded. Intestinal tuberculosis was considered highly relevant given regional endemicity, but ascitic fluid GeneXpert testing was negative and histopathology showed no granulomas, caseation, or acid-fast bacilli. Crohn's disease was excluded by the absence of transmural lymphoid aggregates, non-caseating granulomas, fissuring ulcers, and cobblestoning. Intestinal malignancy, including lymphoma and adenocarcinoma, was excluded by negative tumor markers, negative ascitic fluid cytology, and a histopathological pattern of pure eosinophilic inflammation without atypia. Drug-induced eosinophilic enteropathy was considered unlikely given the absence of any NSAID or relevant drug exposure. Parasitic infection was excluded by the histopathological absence of parasites or characteristic granulomatous reactions and by negative clinical screening. Vasculitis, including eosinophilic granulomatosis with polyangiitis, was excluded by the absence of extra-intestinal manifestations, the negative clinical picture, and the histopathological absence of vasculitic changes; ANA could not be performed locally, and this is acknowledged as a limitation.

Although several reports suggest a potential association with food allergies and hypersensitivity to environmental allergens,[4,13,14] other studies have not demonstrated a direct role of allergy in pathogenesis.[15–17] In the present case, the patient had no personal or family history of food allergy, asthma, atopic dermatitis, or other hypersensitivity conditions, although total serum IgE was elevated. Eosinophilic enteritis is classically described as a triad of gastrointestinal symptoms, eosinophilic infiltration, and peripheral eosinophilia; however, peripheral eosinophilia is not consistently present and is absent in a subset of patients,[6,15] as in this case. The absence of elevated peripheral eosinophil counts therefore does not exclude the diagnosis, and histopathological examination remains central.

Systemic corticosteroids remain the first-line therapy for EE and typically produce rapid clinical and radiological improvement. Prednisone or equivalent regimens are commonly used, followed by gradual tapering according to clinical response. In resource-limited settings, corticosteroids represent a practical and cost-effective treatment option because of their availability and affordability. Alternative therapeutic approaches include dietary modification, elimination of potential food allergens, and, in refractory cases, immunomodulators or biologic therapies targeting interleukin-5 (IL-5) or IgE pathways. The six-food elimination diet (SFED) achieves higher histological remission rates (approximately 70–75%) in EoE than the four-food elimination diet (FFED, approximately 50–60%), although adherence is often more challenging.[18,19] A step-up dietary strategy (FFED first, then SFED if needed) may optimise both efficacy and feasibility. Biologic agents, while effective in EGIDs, are impractical in low-income settings because of cost and limited availability, and structured elimination diets require nutritional counselling and follow-up that are not consistently available.

Despite medical management, surgical intervention may become unavoidable in the presence of complications such as complete bowel obstruction, perforation, or diagnostic uncertainty.[11,20] In the present case, obstruction necessitated surgical evaluation both to relieve the mechanical complication and to obtain diagnostic tissue. The diagnostic overlap with intestinal tuberculosis, Crohn's disease, drug-induced enteropathy, malignancy, and parasitic infection can lead to delay, especially in regions where infectious diseases are endemic and tend to dominate clinical suspicion.

This case report has several limitations. First, as a single-patient report, it does not permit generalisation of findings. Second, several specialised investigations that would have strengthened the diagnostic evaluation were not available in our setting, including quantitative serum IgE beyond qualitative elevation, serum tryptase, ANA, SAAG, ascitic fluid

total and differential cell counts, and ascitic fluid ADA activity. Third, long-term follow-up data are limited to the early postoperative period, and further follow-up will be required to assess recurrence and the outcome of medical therapy for the residual mid-ileal stricture. Finally, eosinophil counts per high-power field are reported as a qualitative estimate rather than a formally quantified mean count; however, infiltration markedly exceeded standard diagnostic thresholds.

## Conclusion

Eosinophilic enteritis is a rare but important differential diagnosis in patients presenting with recurrent abdominal pain and unexplained bowel obstruction. The disease may occur in the absence of peripheral eosinophilia or a history of atopy, and its clinical and radiological features often overlap with more common inflammatory, infectious, and malignant conditions. Histopathological confirmation remains essential. In resource-limited settings, diagnostic challenges delay recognition and increase the risk of complications; however, once identified, EE typically responds well to systemic corticosteroid therapy. Maintaining a high index of suspicion, ensuring timely tissue biopsy, and strengthening multidisciplinary collaboration are critical to improving diagnostic accuracy and patient outcomes. Enhanced awareness and improved diagnostic infrastructure are particularly important in low-resource environments to facilitate early detection and appropriate management of this treatable condition.

## Data Sharing Statement

The data supporting this case report's findings are available from the corresponding author upon reasonable request.

## Ethical Approval and Consent

Ethical approval for this case report was obtained from the Institutional Review Board of Dr. Sumait Hospital, SIMAD University, Mogadishu, Somalia. Written informed consent was obtained from the patient for publication of this case report and any accompanying images. All identifying patient information has been anonymized. All procedures were conducted in accordance with the ethical standards of the institutional and national research committees and with the Declaration of Helsinki.

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## Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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## Disclosure

The authors declare no financial or non-financial competing interests in this case report.

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