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Case Report

Postpartum serosal eosinophilic gastroenteritis with eosinophilic ascites mimicking an acute surgical abdomen

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ABSTRACT

Serosal eosinophilic gastroenteritis is a rare form of eosinophilic gastrointestinal disease, typically associated with peripheral eosinophilia and eosinophilic ascites. Its non-specific presentation may mimic acute surgical conditions and delay diagnosis. We report a case occurring in the postpartum period that presented as an acute surgical abdomen. A woman in her 30s presented with acute abdominal pain, vomiting, and diarrhoea two months after undergoing a caesarean section. Clinical, laboratory, radiological, surgical, cytological, histopathological and molecular investigations were performed to establish the diagnosis and exclude secondary causes of eosinophilia. Computed tomography demonstrated moderate-volume ascites and diffuse bowel wall thickening, predominantly involving the jejunum and gastric antrum. Exploratory laparoscopy excluded surgical acute abdomen and postoperative complications from caesarean section. Further investigation revealed peripheral hypereosinophilia, eosinophil-rich ascitic fluid and dense eosinophilic infiltration of the gastric mucosa. Parasitic, haematological and neoplastic causes of eosinophilia were excluded and serosal eosinophilic gastroenteritis was diagnosed. Systemic corticosteroid therapy resulted in rapid clinical improvement, resolution of ascites, and progressive normalisation of peripheral eosinophilia, with no recurrence following treatment discontinuation. Serosal eosinophilic gastroenteritis should be considered in patients presenting with unexplained ascites, gastrointestinal symptoms and peripheral eosinophilia after exclusion of secondary causes. Awareness of this rare presentation may facilitate earlier diagnosis and appropriate treatment, while the possible influence of postpartum immunological changes remains uncertain.

Keywords: Eosinophilic gastroenteritis, Eosinophilic ascites, Postpartum period, Hypereosinophilia, Acute abdomen, Atopy

INTRODUCTION

Eosinophilic gastrointestinal diseases (EGIDs) comprise a heterogeneous group of disorders characterised by gastrointestinal symptoms and pathological eosinophilic inflammation of the gastrointestinal tract in the absence of identifiable secondary causes.[1] Recent international consensus recommends the use of EGID as an umbrella term, with individual disorders named according to the gastrointestinal segment involved.[2] Nevertheless, the term eosinophilic gastroenteritis remains widely used in the literature, particularly in descriptions based on the traditional classification according to the depth of eosinophilic infiltration into mucosal, muscular and serosal forms.[1,3]

Serosal involvement is the least common presentation and is characteristically associated with eosinophilic ascites and peripheral eosinophilia.[3,4] As symptoms are non-specific and may resemble infectious, inflammatory,

malignant or acute surgical conditions, diagnosis can be challenging and is dependent on the integration of clinical, laboratory, radiological and histological findings together with the exclusion of secondary causes of eosinophilia.[1,3]

Association with allergic disease is frequently reported, although the underlying pathophysiology remains incompletely understood.[1,3] Rare cases of serosal eosinophilic gastrointestinal disease have also been reported in the postpartum period.[5]

We report a case of serosal eosinophilic gastroenteritis presenting 2 months postpartum with acute abdominal pain and peripheral hypereosinophilia, initially mimicking an acute surgical abdomen.

CASE REPORT

A woman in her 30s presented to the emergency department with a 1-day history of diffuse abdominal pain, vomiting, and diarrhoea, accompanied by a 2-3-day history of subjective abdominal distension. She reported no fever. She was 2 months postpartum following caesarean delivery of a twin pregnancy complicated by severe pre-eclampsia, intrahepatic cholestasis of pregnancy, wound infection and postoperative wound dehiscence.

Her medical history included allergic rhinitis, cutaneous allergies under immunoallergology follow-up and irritable bowel syndrome under Gastroenterology follow-up. Her regular medication consisted of nifedipine and inhaled budesonide, and she reported an allergy to fluconazole.

On admission to the emergency department, the abdomen was diffusely tender, with severe pain that was difficult to control, but there was no guarding or other evidence of peritoneal irritation. Laboratory investigations demonstrated marked leukocytosis of 40.4×10^9/L, with neutrophilia of 18.5×10^9/L and marked peripheral eosinophilia of 14.5×10^9/L (35.9%). The platelet count was elevated at 491×10^9/L. Haemoglobin, renal function, liver enzymes and electrolytes were within normal limits. C-reactive protein was minimally elevated at 3.1 mg/L, and arterial blood gas analysis, including lactate, was unremarkable.

Contrast-enhanced computed tomography of the abdomen and pelvis demonstrated moderate-volume ascites associated with diffuse bowel wall thickening and oedema, predominantly involving the jejunum and gastric antrum, with associated vascular engorgement consistent with an extensive inflammatory process (Figure 1). Diffuse bladder wall thickening was also observed. The uterus and adnexa were unremarkable, and there was no evidence of bowel obstruction or abscess.

The patient was evaluated by the Obstetrics and Gynaecology team, and no gynaecological cause for her clinical presentation was identified.

Given the severity of the abdominal pain, recent caesarean delivery and imaging findings, an acute intra-abdominal surgical complication was initially suspected. Urgent exploratory laparoscopy was therefore performed to exclude hollow viscus perforation or a postoperative complication from caesarean section.

A large volume of turbid, citrine-yellow peritoneal fluid was identified in all four abdominal quadrants. Gastric antrum was thickened and exhibited inflammatory changes, with a predominantly irregular, granular appearance of the posterior wall. Small bowel loops showed oedema and inflammatory changes (Figure 2). Systematic exploration of the abdominal cavity revealed no identifiable cause of peritonitis, with no evidence of visceral perforation, diverticulitis, appendicitis or cholecystitis. Pelvic organs were macroscopically normal. Ascitic fluid was collected for cytological and microbiological analysis.

Initial cytological examination of the ascitic fluid showed numerous neutrophils without evidence of malignancy. However, because ascites subsequently reaccumulated and peripheral hypereosinophilia persisted, further investigation was undertaken, and review of the cytological slides demonstrated an ascitic fluid markedly eosinophil-rich, with no malignant cells identified. Immunophenotypic analysis showed mature eosinophils accounting for 86.9% of the total cellularity, with no evidence of B-cell or T-cell lymphoproliferative disease or immature myeloid cells, confirming eosinophilic ascites.

Upper gastrointestinal endoscopy revealed erythematous mucosal changes affecting the gastric body, most prominently the antrum, and the duodenal bulb. Biopsies were obtained from the oesophagus, stomach and duodenum, with histopathological examination demonstrating dense eosinophilic infiltration of the gastric antrum and body and of the duodenal lamina propria, with preserved glandular and villous architecture (Figure 3). No evidence of *Helicobacter pylori* infection, dysplasia or malignancy was identified.

Magnetic resonance enterography performed during hospitalisation demonstrated mild diffuse small bowel wall thickening and mucosal hyperenhancement, without fixed stenosis or mechanical obstruction. The previously documented ascites had completely resolved.

An extensive investigation was undertaken to exclude secondary causes of eosinophilia.

Stool parasitology and *Strongyloides stercoralis* serology were negative. Bone marrow aspiration was performed for morphological examination, genetic analysis, and

immunophenotyping. The bone marrow aspirate revealed 2.5% blasts and significant eosinophilia (15%), including eosinophil precursors. Molecular analysis showed no evidence of the BCR: ABL1 fusion gene or rearrangements involving the FIP1L1, CHIC2, or PDGFRA genes, making a myeloid or lymphoid neoplasm with eosinophilia unlikely. Immunophenotypic analysis revealed no abnormalities. Additionally, a nasal turbinate biopsy was performed to exclude eosinophilic granulomatosis with polyangiitis (EGPA), which showed no evidence of vasculitis or granulomatous inflammation.

Computed tomography of the chest and transthoracic echocardiography showed no evidence of systemic organ involvement.

Diagnosis of serosal eosinophilic gastroenteritis was supported by the combination of marked peripheral hypereosinophilia, eosinophilic ascites, gastric eosinophilic infiltration, and the exclusion of secondary causes of eosinophilia.

Following exclusion of secondary causes, a 60 mg daily dose of intravenous methylprednisolone was initiated. The patient showed immediate clinical improvement, with progressive resolution of abdominal pain, vomiting and diarrhoea and marked improvement of the ascites. No additional immunosuppressive therapy or dietary intervention was required during hospitalisation.

Before discharge, intravenous methylprednisolone was switched to oral prednisolone with a gradual tapering regimen. Corticosteroid therapy was successfully discontinued after 2 months without recurrence of gastrointestinal symptoms or ascites.

Peripheral eosinophil counts progressively normalised during follow-up, paralleling the sustained clinical improvement. Follow-up abdominal ultrasonography demonstrated complete resolution of the ascites and previously observed bladder wall thickening. At the most recent follow-up, the patient remained asymptomatic, with no clinical, laboratory or radiological evidence of disease recurrence.



Figure 1 (A-C): CT scan demonstrating moderate-volume ascites, diffuse wall thickening and edema of the jejunum and gastric antrum, as well as marked thickening and edema of the urinary bladder wall. (A) Coronal plane; (B) Axial plane; (C) Sagittal plane.



Figure 2 (A and B): Intraoperative images showing thickening and erythema of the small bowel loops with turbid, citrine-yellow peritoneal fluid (A), and thickening of the posterior wall of the gastric antrum with an irregular surface appearance (B).

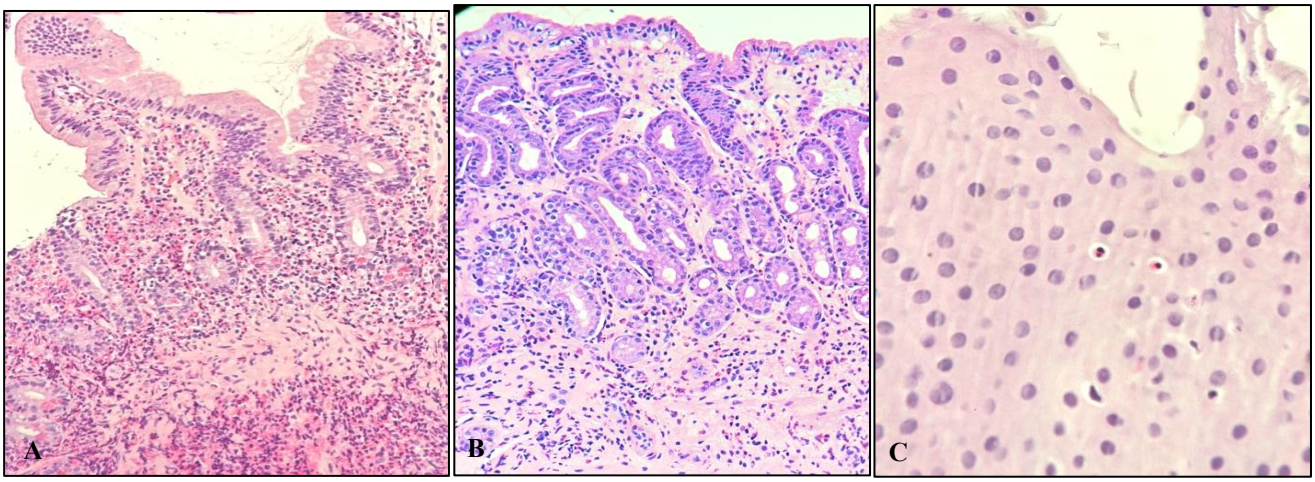


Figure 3 (A-C): On histological examination, duodenal mucosa had preserved villous architecture; the *lamina propria* had an inflammatory infiltrate composed of numerous eosinophils with focal aggregates. (A) (H and E, ×200). Frequent eosinophils (>30 eosinophils/ high power field [HPF]) were also noted within the *lamina propria* of gastric mucosa. (B) (H and E, ×200). Mild nonspecific esophagitis was observed in the esophageal biopsies, with rare intraepithelial eosinophils found within the esophageal mucosa (<15 eosinophils / HPF) (C) (H and E, ×400).

DISCUSSION

Serosal eosinophilic gastroenteritis is the least common form of eosinophilic gastrointestinal disease according to the traditional layer-based classification.[3] It is characterised by abdominal distension, eosinophilic ascites and marked peripheral eosinophilia, often accompanied by a favourable response to corticosteroid therapy, as described by Talley et al and supported by subsequent reviews.[3,4]

The presentation of our patient illustrates the diagnostic difficulty associated with this phenotype. Acute severe abdominal pain, diffuse bowel wall thickening and ascites occurring shortly after caesarean delivery raised concern for a postoperative intra-abdominal complication and prompted exploratory laparoscopy. Serosal eosinophilic disease can resemble other inflammatory or surgical abdominal conditions, and diagnosis requires exclusion of alternative causes of gastrointestinal and peripheral eosinophilia.[1,3] The combination of gastrointestinal symptoms, peripheral hypereosinophilia, eosinophil-rich ascitic fluid, gastric eosinophilic infiltration and the absence of an identifiable secondary cause ultimately supported the diagnosis in our patient.

A notable diagnostic challenge was the initial misinterpretation of the ascitic fluid. Despite its turbid, citrine-yellow appearance and initially reported neutrophilic predominance, subsequent cytological review revealed marked eosinophilia. A similar finding was reported by Ferreira et al who highlighted that automated cell counting may misclassify eosinophils as neutrophils, potentially delaying diagnosis.[6] Therefore, unexplained peripheral eosinophilia associated with apparent neutrophilic ascites should prompt cytological reassessment, particularly in the absence of an identifiable infectious source.

Histological diagnosis may be challenging due to the patchy distribution of eosinophilic infiltration and the limited ability of superficial endoscopic biopsies to detect deeper-layer involvement, particularly in serosal disease.[4,7] In our patient, however, gastric biopsies revealed dense eosinophilic infiltration, supporting the diagnosis alongside the presence of eosinophilic ascites.

Patient's atopic background is also noteworthy. Allergic conditions, including asthma, allergic rhinitis, food or drug allergy and eczema, have been reported in a substantial proportion of patients with eosinophilic gastroenteritis.[3] A recent review cited data from a United States administrative database in which 45.6% of patients reported allergic symptoms such as rhinitis and asthma.[1] However, atopy is not universal and should be regarded as a supportive clinical feature rather than a diagnostic requirement.

The onset of eosinophilic gastrointestinal disease in the postpartum period raises the possibility of an association with pregnancy-related immunological changes. Although similar cases have been reported in the literature, the available evidence remains limited to isolated case reports, and a causal relationship has not been established.[5] In our patient, the postpartum period may therefore represent a potential contributing factor, although its role in triggering the disease remains uncertain.

Systemic corticosteroids remain a mainstay of treatment for symptomatic eosinophilic gastroenteritis, with particularly favourable responses reported in patients with serosal involvement.[1,4] The rapid clinical

improvement and sustained remission observed in our patient following corticosteroid therapy are consistent with previous reports.

The longer-term course of eosinophilic gastrointestinal disease is variable. Some patients experience a single episode, whereas others develop recurrent or chronic disease requiring prolonged treatment.[1] In a retrospective cohort of 35 patients, Havlichek et al found that serosal involvement, together with weight loss, hypoalbuminaemia or anaemia at diagnosis, was associated with a chronic disease course requiring long-term medication.[8] Despite marked eosinophilia, ascites and serosal involvement, our patient achieved sustained remission after a single course of corticosteroid therapy. Continued clinical follow-up remains appropriate given the heterogeneous natural history of the disease.

CONCLUSION

Serosal eosinophilic gastroenteritis should be considered in patients presenting with gastrointestinal symptoms, unexplained ascites and peripheral eosinophilia, particularly when infectious, malignant and other secondary causes have been excluded. Eosinophilic ascites may mimic an acute surgical or infectious abdomen and can lead to unnecessary invasive procedures if not recognised. Early diagnosis allows prompt corticosteroid treatment, which may result in rapid clinical improvement. Although this case occurred in the postpartum period, the available evidence is insufficient to establish a causal relationship between postpartum immunological changes and disease onset.

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