Eosinophilic Gastrointestinal Disease Masquerading Radiographically as Peritoneal Carcinomatosis

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ABSTRACT

Introduction: Eosinophilic gastrointestinal disease (EGID) is a rare disorder characterized by gastrointestinal symptoms and eosinophilic infiltration of the gastrointestinal tract in the absence of secondary causes of eosinophilia.

Case Presentation: A 76-year-old man presented with abdominal pain, early satiety, and ascites. Imaging and laparoscopy were concerning for peritoneal carcinomatosis. Laboratory evaluation revealed peripheral eosinophilia. Extensive infectious, autoimmune, and neoplastic evaluations were negative. Biopsies and cytology revealed no evidence of malignancy but demonstrated eosinophilic tissue infiltration supporting a diagnosis of EGID. Corticosteroid treatment led to rapid resolution of eosinophilia. The patient was diagnosed with serosal EGID and subsequently transitioned to mepolizumab, with sustained disease control.

Discussion: Hypereosinophilia often requires extensive evaluation. This case highlights the diagnostic complexity of EGID, particularly when it mimics advanced malignancy.

Conclusions: EGID should be considered in patients with unexplained eosinophilia and radiographic findings suggestive of malignancy, particularly within the abdomen or gastrointestinal tract. Early recognition may prevent unnecessary interventions and allow timely initiation of effective therapy.

INTRODUCTION

Eosinophilia is defined as an elevated level of eosinophils in the serum or tissues.[1] It may arise from a broad spectrum of underlying conditions, including allergic, infectious, neoplastic, genetic, and immune-mediated disorders.[2]

Hypereosinophilic syndromes (HES) comprise a group of rare disorders characterized by an absolute eosinophil count greater than 1500/ μL and eosinophil-associated organ damage.[3] Idiopathic HES, a subset of HES with no identifiable underlying cause, is a rare clinical entity that can present with diverse manifestations affecting multiple organ systems. Eosinophilic gastrointestinal diseases (EGID) are rare disorders characterized by gastrointestinal symptoms and eosinophilic infiltration of the gastrointestinal tract in the absence of secondary causes of eosinophilia.[4] EGID and idiopathic HES share significant clinical overlap. Interleukin-5 (IL-5) inhibitors, including mepolizumab, reslizumab, and benralizumab, have shown promising efficacy in the treatment of eosinophil-mediated diseases.[5]

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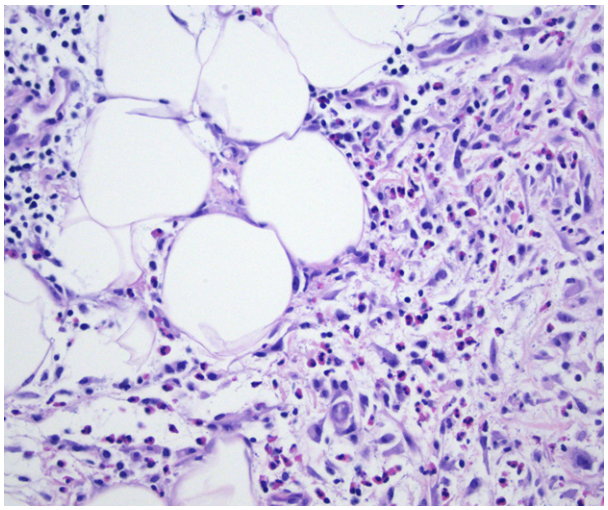
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CASE PRESENTATION

A 76-year-old man with a medical history of hypertension, hyperlipidemia, gastroesophageal reflux disease, Raynaud's disease, eczema, transient ischemic attack, and recurrent *Clostridioides difficile* infection presented with a 2-month history of progressive right upper quadrant abdominal pain.

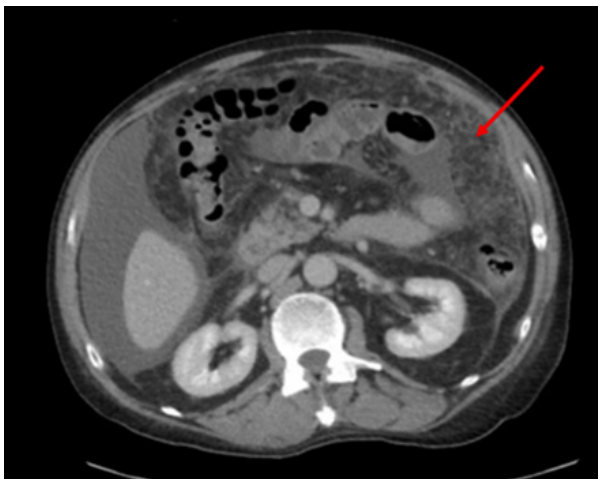
The patient initially presented to the emergency department reporting a progressive sensation of abdominal fullness, early satiety, and worsening right upper quadrant pain over the preceding week. Physical examination revealed significant distension concerning for ascites. Notably, no cutaneous rash was present. Laboratory examination on admission showed hypoalbuminemia (2.8 g/dL), low total protein (6.2 g/dL), normal liver function test results, elevated C-reactive protein (13.6 mg/dL), elevated erythrocyte sedimentation rate (38 mm/h), a white blood cell count of 10.7 K/μL), normal hemoglobin, elevated

Figure 1. Histologic Image of Omentum Biopsy



Hematoxylin and eosin-stained section at 400X magnification showing eosinophil-rich chronic inflammation in a background of abundant reactive myofibroblasts.

Figure 2. Representative Slice From Computed Tomography Abdomen/Pelvis With Intravenous Contrast



CT was notable for diffuse nodular infiltration of the omentum along with interloop mesenteric fluid. There was also medium volume ascites and small bilateral (right greater than left) pleural effusions.

platelet count (427 K/μL), a normal absolute neutrophil count (7270/μL), lymphopenia (970/μL), monocytosis (1480/μL), eosinophilia (800/μL), a normal absolute basophil count (20/μL), and an elevated absolute immature granulocyte count (120/μL).

Computed tomography (CT) of the abdomen and pelvis revealed a new moderate volume ascites and mesenteric edema. Chest CT showed a small right pleural effusion without evidence of malignancy. Right upper quadrant ultrasonography demonstrated omental infiltration and stranding, as well as moderate ascites. Liver morphology and Doppler studies were normal. Magnetic resonance imaging (MRI) with magnetic resonance cholangiopancreatography showed gallbladder wall lesions, periportal lymphadenopathy, and peritoneal enhancement and nodularity concerning for carcinomatosis, although findings were nonspecific. Tissue sampling was recommended. Electrocardiography revealed a new left bundle branch block. Transthoracic echocardiogram demonstrated a normal left ventricular ejection fraction (55%) and normal right ventricular function.

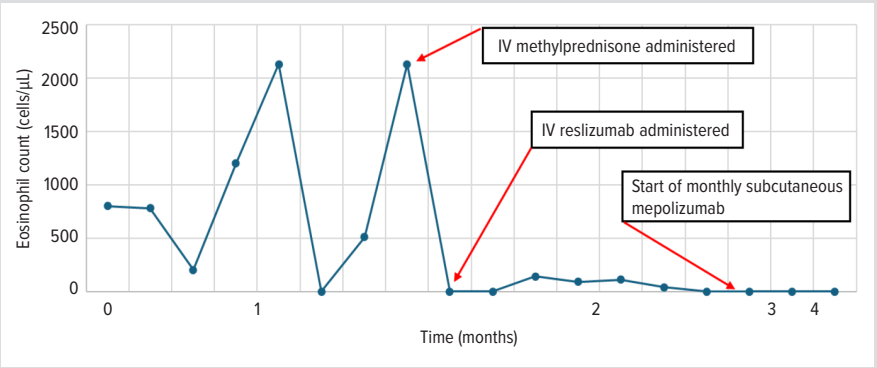
The patient underwent paracentesis, with removal of 2.5 L of ascitic fluid. Acid-fast bacilli smear results were negative, and fluid was sent for culture. Surgical oncology was consulted, and the patient underwent exploratory laparoscopy, which demonstrated diffuse omental caking with abundant brown ascitic fluids. Omental biopsy specimens and additional ascitic fluid samples were obtained. Frozen sections demonstrated abundant inflammation and atypical cells without definitive evidence of carcinoma. Further classification was deferred pending permanent sections, and portions of the specimen were sent for flow cytometry and chromosome analysis. The patient was discharged while pathology results were pending. Ascitic fluid cytology was negative for malignancy but showed eosinophilic and neutrophilic leukocytes. Flow cytometry did not identify an abnormal lymphocyte population, and chromosome analysis was not completed because of insufficient metaphase cells. Permanent histology of the biopsy specimens revealed eosinophil-rich chronic inflammation (Figure 1).

One month later, the patient was readmitted with worsening shortness of breath, wheezing, and abdominal pain. Laboratory evaluation was notable for a normal white blood cell count (9.3 K/μL), elevated platelet count (551 K/μL), normal hemoglobin, normal troponin, monocytosis (1310/μL), and marked eosinophilia (2130/μL), with normal neutrophil and basophil counts.

Repeat CT of the abdomen and pelvis showed persistent infiltration concerning for peritoneal carcinomatosis, with increased ascites compared with prior imaging (Figure 2). Small bilateral pleural effusions also were present. CT pulmonary angiography showed no pulmonary emboli or pulmonary infiltrates. Hematology was consulted for evaluation of eosinophilia. Peripheral smear demonstrated only nonspecific thrombocytosis and mild monocytosis. The patient underwent a paracentesis, with removal of 700 mL of peritoneal fluid, and thoracentesis, with removal of 2200 mL of pleural fluid. Pleural fluid analysis demonstrated 2068 nucleated cells/μL and was mildly exudative according to Light criteria, with pleural fluid lactate dehydrogenase (LDH) of 131 U/L (serum LDH, 116 U/L) and pleural fluid protein of 2.8 g/dL (serum protein, 5.5 g/dL). Differential counts showed 14% neutrophils, 25% lymphocytes, 43% macrophages, and 17% eosinophils. Pleural fluid cytology was negative for malignancy, and concurrent flow cytometry showed no evidence of a lymphoproliferative disorder.

Additional evaluation of eosinophilia revealed negative *Strongyloides* IgG antibody, *Toxocara* IgG antibody, and QuantiFERON-TB Gold test results. Serum IgE levels were within normal limits. Tryptase and vitamin B12 levels were normal. Autoimmune serologic testing, including RNA polymerase III antibody, polymyositis panel, Scl-70 antibody, cyclic citrullinated peptide IgG antibody, rheumatoid factor, antinuclear antibody, extractable nuclear antigen panel, myeloperoxidase IgG antibody, and proteinase 3 IgG antibody, was negative. Stool examination for parasites, including direct preparation and trichrome staining, was unremarkable.

Figure 3. Serial Absolute Eosinophil Counts Over the Course of Hospitalization and Follow-up, Demonstrating Rapid Normalization Following Corticosteroid Initiation and Sustained Suppression With Mepolizumab



Following discharge, the patient underwent bone marrow biopsy, which demonstrated essentially normal morphologic findings, with no lymphoid abnormalities detected by concurrent flow cytometry. Molecular testing identified a mutation with a low variant allele frequency of uncertain clinical significance in the setting of otherwise unremarkable marrow findings. Platelet-derived growth factor receptor alpha rearrangement and Janus kinase 2 mutation testing were negative. Therefore, there was no evidence of a myeloid variant to explain the patient's eosinophilia. Positron emission tomography (PET) showed no evidence of malignancy. Subsequent outpatient colonoscopy likewise revealed no evidence of malignancy. Ongoing monitoring of blood counts was recommended.

Allergy consultation initially determined that the patient met diagnostic criteria for idiopathic hypereosinophilic syndrome because testing did not identify a hematologic malignancy or other apparent cause. Subsequently, serosal EGID with peripheral hypereosinophilia was considered a more specific diagnosis. The patient was treated initially with intravenous methylprednisone 60 mg. The following day, complete resolution of eosinophilia was observed (eosinophil count, 0/μL), and he received reslizumab, an IL-5 antagonist. He was discharged on a prednisone taper.

At follow-up in the outpatient allergy clinic, the patient was transitioned to mepolizumab 300 mg subcutaneously administered every 4 weeks. Serial absolute eosinophil counts were monitored every 2 weeks and remained suppressed. These trends are shown in Figure 3. Despite IL-5 inhibitor therapy and resolution of peripheral eosinophilia, the patient experienced recurrent ascites. Repeat peritoneal fluid flow cytometry obtained after paracentesis approximately 6 months later remained negative for malignancy.

Although the ascites subsequently resolved, recurrent pleural effusions persisted, necessitating placement of an indwelling pleural catheter. Pleural fluid analysis from subsequent thoracentesis remained marginally exudative, based on mild protein elevation, but persistent eosinophilia was not observed in the context of treatment with mepolizumab.

The patient was referred to a quaternary referral center for further evaluation. No underlying malignancy or alternative etiology was identified, and he was advised to continue mepolizumab indefinitely given the favorable clinical response and good tolerability. Follow-up with repeat PET imaging, pulmonary function testing, and troponin monitoring was recommended, with cardiac MRI advised if troponin levels became elevated to assess for myocardial involvement. To date, the patient has had no evidence of malignancy or another etiology for hypereosinophilia and remains on outpatient mepolizumab therapy.

DISCUSSION

Eosinophilia refers to an increased number of eosinophils in the tissues and/or blood. Absolute eosinophil counts greater than 450 to 550/μL are commonly considered elevated, although thresholds may vary depending on laboratory standards.[1] Hypereosinophilia refers to an absolute eosinophil count >1500/μL.[2] The differential diagnosis of eosinophilia is broad and encompasses allergic, infectious, neoplastic, genetic, and immune-mediated disorders.[2] Given the broad possible etiologies, an extensive diagnostic workup is often necessary. A detailed clinical history is frequently the most important clue to identifying the underlying etiology of eosinophilia.[1]

Hypereosinophilic syndromes are a rare group of disorders characterized by an absolute eosinophil count of 1500/μL or greater, evidence of end-organ involvement attributable to eosinophilia, and the absence of an alternative explanation.[2] Idiopathic hypereosinophilic syndrome (HES) is one potential cause of eosinophilia. Idiopathic HES has been reported to present with a wide variety of manifestations, including persistent diarrhea and other gastrointestinal symptoms, as well as cutaneous, pulmonary, and cardiac involvement, as summarized in the Table.[6-9]

EGID is a rare disorder characterized by gastrointestinal (GI) symptoms and eosinophilic infiltration of specific areas of the GI

Table. Representative Published Cases of Eosinophilic Gastrointestinal Disease (EGID) or Hypereosinophilic Syndrome (HES) With Gastrointestinal Involvement

Author, Year	Patient Presentation	Treatment	Outcome
Foreman et al, 2024[6]	70-year-old male presenting with generalized erythematous and pruritic cutaneous lesions and peripheral hypereosinophilia	Corticosteroids	Complete resolution of skin lesions
Wang et al, 2025[7]	73-year-old male with pulmonary hypertension and asthma presenting with persistent eosinophilia	Corticosteroids, mepolizumab	Significant response to corticosteroids and mepolizumab
Gruber et al, 2024[8]	50-year-old male presenting with diarrhea, nausea, emesis, abdominal pain, and tissue biopsies revealing mucosal eosinophilia	Corticosteroids	Significant improvement in symptoms and eosinophilia
Khalid et al, 2019[9]	83-year-old female presenting with worsening dyspnea, found to have eosinophilic myocarditis	Corticosteroids	Significant clinical improvement

tract in the absence of secondary causes of eosinophilia.[4] EGID can involve the mucosal, muscular, or serosal layers of the GI tract and is often classified accordingly. The serosal subtype is uncommon and may present with eosinophilic ascites.[10] In this case, the distinction between idiopathic HES and EGID, particularly serosal EGID, warrants consideration.

The radiographic appearance of omental infiltration often presents a diagnostic challenge because it may result from either benign or malignant processes. Distinguishing among these etiologies on imaging alone can be particularly difficult.[11] Omental biopsy is often required for definitive evaluation and has been shown to be more sensitive than peritoneal cytology for detecting malignancy.[12]

Mepolizumab and reslizumab are humanized monoclonal antibodies that target IL-5.[13] Mepolizumab is approved in the United States for treatment of severe eosinophilic asthma, eosinophilic granulomatosis with polyangiitis, and HES.[14]

We present a case of a 76-year-old man who presented with progressive abdominal swelling, early satiety, and right upper quadrant pain. Laboratory studies demonstrated persistent eosinophilia, and imaging revealed omental infiltration concerning for peritoneal carcinomatosis. An extensive evaluation for eosinophilia, including infectious, autoimmune, hematologic, and oncologic causes, was unrevealing. The patient ultimately was diagnosed with serosal EGID and treated with methylprednisone and reslizumab during hospitalization before transitioning to mepolizumab as an outpatient. Subsequent evaluation at a quaternary referral center likewise failed to identify an alternate cause of eosinophilia.

Given the negative malignancy workup, eosinophil-predominant histopathologic findings, and GI serosal involvement, serosal EGID was considered the most likely diagnosis. Although idiopathic HES remains a diagnosis of exclusion, distinguishing it from EGID may have limited clinical significance in some cases, as both conditions can present with overlapping manifestations and may respond to IL-5 targeted therapies. The patient continues to demonstrate sustained suppression of eosinophilia control and symptomatic improvement while receiving mepolizumab.

CONCLUSIONS

Our case describes a unique presentation of eosinophilic disease mimicking peritoneal carcinomatosis. Eosinophilia encompasses a broad differential diagnosis, including allergic, infectious, neoplastic, and immune-mediated disorders, and often requires a comprehensive evaluation. Although serosal EGID was diagnosed in this case, idiopathic HES remained a relevant consideration in the differential diagnosis.

This case underscores the importance of careful interpretation of the complete blood cell count and differential and highlights the need to maintain a broad differential diagnosis in patients with persistent eosinophilia and radiographic evidence of omental infiltration. Eosinophilic disorders may mimic malignancy, and although distinguishing between overlapping syndromes such as idiopathic HES and EGID may be diagnostically important, management strategies often overlap. IL-5 inhibitors, including mepolizumab, may offer an effective targeted treatment option for patients with serosal EGID and related eosinophilic disorders.

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