CLINICAL VIGNETTE

Peripheral Eosinophilia

Evangelia Kirimis, MD

A 36-year-old male was admitted to the hospital with abdominal pain and diarrhea. He had a longstanding history of inflammatory bowel disease (IBD) diagnosed in his twenties and idiopathic pancreatitis, but had most recently been under very good control for several years with the use of vedolizumab. Three weeks prior to admission, he had a significant upper respiratory infection leading to sinusitis and persistent cough, and was treated with amoxicillin. His viral symptoms improved, but about a week later he noted mild abdominal pain and diarrhea. The symptoms initially waxed and waned, steadily increased leading to a urgent care visit. Evaluation was unremarkable except for a noted leukocytosis to 19 10^3/mcL with prominent eosinophilia with absolute count of 12 10^3/mcL. He was sent home as his symptoms did improve but a few days later his abdominal pain and diarrhea increased and he was unable to tolerate oral intake, and presented to the emergency room. White blood cell increased to 34 10^3/mcL with predominantly eosinophils of 26 10^3/mcL. There was a mild anemia with a hemoglobin of 12 g/dL, but otherwise no other notable lab abnormalities. Computed tomography of the abdomen and pelvis noted no diverticulosis, diverticulitis, appendicitis, other acute findings. He was admitted for observation and hydration. The patient did not feel that his symptoms were similar to his prior IBD flares, and his gastroenterologist also did not feel the findings were consistent with his IBD. His symptoms slowly improved in the hospital and his white blood cell count slowly decreased to 17 10^3/mcL by discharge three days later. The eosinophilia was slower to fall but was improving by discharge. The patient was seen in outpatient hematology one week after discharge. His abdominal symptoms had almost resolved, his white blood cell count was 13 g/dL with an eosinophil count improved to 7.7 10^3/mcL. His hemoglobin was also now in normal range. Another follow-up two weeks later showed resolution of the prior blood count abnormalities.

The literature has noted similar incidences of drug-induced eosinophilic colitis.[1] A prior case was described related to antibiotic use.[1] Like in the above patient, amoxicillin and cephalexin were used leading to diarrhea and profound peripheral eosinophilia.[1] Subsequent colonoscopy in this case report noted colitis and biopsies of these areas noted eosinophilic colitis.[1] This report also indicated symptoms and eosinophilia resolved with no intervention.[1] Eosinophilic colitis is felt to be rare[1-3] estimated at about 2.3/100,000 adults.[2] Besides medications, it has been associated with helminthic infections.[1-3] There may be an association with ulcerative colitis as seen in our patient but the mechanism is unknown.[1,3] Other medications associated with this disorder include tacrolimus, clozapine, NSAIDs, gold, and carbamazepine.[1-3] Colonic eosinophilia has also been noted in autoimmune diseases, celiac disease, and hypereosinophilic syndrome.[3] There is some thought that there may be a genetic predisposition or some relation to environmental exposures as it is more common in people with known atopic diseases such as asthma, dermatitis, food allergies, eczema, etc.[1-3] Similarly, despite the rarity of the disease, it is not unusual for multiple family members to have the same diagnosis.[1,3] Given the low incidence, there are no clear treatment guidelines.[1] Removal of any potential offending medications and treating any possible helminthic infections is critical.[1] Furthermore, given the noted association with food allergies, elimination diets have been advised but are generally poorly tolerated.[3] Otherwise, in idiopathic cases, a short course of steroids has been reported to be highly effective.[1,2] Steroids help mitigate growth factors released by eosinophils.[3] Immunomodulators like azathioprine have been used in refractory cases or where steroids cannot be weaned.[3]

Interestingly, eosinophils are commonly seen throughout the GI tract.[2] They are rare in the stomach and increase in the small and large intestines, with highest numbers in the cecum.[2] Eosinophils are crucial for protecting the gut from pathogens, food allergens, and the microbiome.[2] They can produce anti-inflammatory mechanisms to help protect the GI tract.[2] However, as expected, in elevated numbers, tissue damage can result when these normal mechanisms are overstimulated for secondary or idiopathic reasons as in eosinophilic colitis.[2]

In the patient above, hematology was consulted regarding the concern for significant peripheral eosinophilia. However, given the patient's acute symptoms and no other blood test abnormalities, the history suggested a primary GI tract process and not a hypereosinophilic syndrome which was the immediate consult concern. This was consistent with his steady improvement with no intervention. Gastroenterology did not feel colonoscopy was necessary to confirm the diagnosis given the quick and steady improvement. It was recommended that he avoid amoxicillin in the future as it seemed like the likely instigator of the disorder.

REFERENCES

1. **Mogilevski T, Nickless D, Hume S**. β-lactam-associated eosinophilic colitis. *BMJ Case Rep.* 2015 Jun 23;2015:

bcr2014206964. doi: 10.1136/bcr-2014-206964. PMID: 26106168; PMCID: PMC4480096.
2. **Walker MM, Potter MD, Talley NJ**. Eosinophilic colitis and colonic eosinophilia. *Curr Opin Gastroenterol*. 2019 Jan;35(1):42-50. doi: 10.1097/MOG.0000000000000492. PMID: 30480590.
3. **Alfadda AA, Storr MA, Shaffer EA**. Eosinophilic colitis: epidemiology, clinical features, and current management. *Therap Adv Gastroenterol*. 2011 Sep;4(5):301-9. doi: 10.1177/1756283X10392443. PMID: 21922029; PMCID: PMC3165205.