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CLINICAL VIGNETTE

The Need to Screen – Strongyloides Colitis in a Patient on Apremilast

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Introduction

Peripheral eosinophilia is well known to include among its potential causes both parasitic disease and autoimmune disease. Not all parasitic infections are overtly symptomatic, with many being occult aside from the finding of peripheral eosinophilia. When an autoimmune disease is identified, anchoring on the diagnosis as the sole cause of eosinophilia could lead to missing a clinically relevant occult infectious process. Immunosuppressive therapy to treat the autoimmune disease could then lead to potentially serious complications from activation of occult parasitic disease. Here is a case of Strongyloides colitis that developed after starting apremilast in a patient with chronic peripheral eosinophilia.

Case

A 37-year-old woman raised in Guatemala with a history of chronic peripheral eosinophilia attributed to known psoriasis was referred to ID after colon biopsy revealed colitis with mucosal eosinophilic infiltration. She reported a 16-year history of psoriasis as well as peripheral eosinophilia with absolute eosinophil counts greater than 1,000 for at least three years that had been attributed to the psoriasis without further evaluation. The psoriasis had been treated with topical clobetasol, however, she had developed psoriatic arthritis prompting addition of apremilast. After several months on apremilast her psoriatic arthritis was well controlled but she developed what started as pain in her rectal area that subsequently spread to her upper abdomen and became associated with intermittent diarrhea. Her symptoms worsened and prompted a colonoscopy with biopsy revealing eosinophilic cryptitis and significantly elevated eosinophils in the lamina propria from the cecum to the sigmoid colon though sparing the rectum. It was suspected that apremilast may have been the cause so it was discontinued. However, repeat colonoscopy four months later continued to show colitis with mucosal eosinophilic infiltration. She was temporarily transitioned to adalimumab and parasitic serologic screening obtained detecting Strongyloides stercoralis IgG antibodies. The peripheral eosinophilia was ongoing with an absolute eosinophil count of 1,008. She also had mild anemia with a hemoglobin of 12.7 but otherwise unremarkable blood counts and chemistry panels. Stool samples were submitted for Strongyloides screening by the Baermann method and the initial sample returned positive for Strongyloides rhabditiform larvae, confirming the diagnosis of active Strongyloides infection. She did not exhibit any respiratory or skin manifestations

or other features concerning for hyperinfection or disseminated infection. She was treated with ivermectin 200mcg/kg PO BID for two days followed by a second course two weeks later. Follow up stool studies one month later were negative for Strongyloides and she reported clinical improvement in the diarrhea and abdominal pain. Repeat colonoscopy approximately four months after therapy showed resolution of the colitis with normal colonic mucosa and no increase in intra-epithelial eosinophils. Repeat Strongyloides IgG antibody levels approximately five months after therapy had decreased by nearly half, the peripheral eosinophilia had resolved, and she reported feeling back at baseline without no gastrointestinal symptoms.

Discussion

This case highlights the risks of anchoring on a diagnosis, particularly when a patient has risk factors for alternative diagnoses. This woman with epidemiologic risk for Strongyloides was known to have chronic eosinophilia that was attributed to psoriasis without further investigation. The augmentation of immunosuppression to treat her psoriasis appears to have led to worsening of her chronic Strongyloides infection such that she had clinically significant and symptomatic colitis. While it is fortunate her disease appears to have been limited to the colon, one can wonder of the potential morbidity of disseminated disease, particularly if she received additional immunosuppression with glucocorticoids.

Many patients infected with Strongyloides are asymptomatic, especially in its chronic form, and peripheral eosinophilia or elevated IgE levels may be the only identifiable abnormalities.¹ Therefore, one must remember to consider the diagnosis when screening before immunosuppression, particularly in the setting of peripheral eosinophilia in patients with epidemiologic risk for the disease. Serologic testing is the most sensitive test commonly available, however, a positive test cannot differentiate between current or prior disease, nor can it exclude a positive result due to cross-reactivity with other helminths, and thus does not confirm the presence of active infection.² Stool screening is notoriously low in sensitivity, with multiple stool samples often needed to make the diagnosis. In general, no less than three should be sent, and sending more is reasonable if clinical suspicion is high. In order to increase sensitivity of stool testing, the stool should be sent for evaluation using the conven-

tional Baermann technique or one of its modifications which are more sensitive for detecting *Strongyloides*.³ PCR testing of stool or tissue can be performed when feasible, but is not widely available and so does not have utility for routine care.^{4,5} Colonoscopy may identify the organism by means of findings on gross examination and by biopsy, but is not typically used as a primary test strategy given it is more invasive and less practical for routine screening.⁶

Oral ivermectin is used to treat *Strongyloides*. Uncomplicated acute or chronic infection is generally treated with ivermectin 200mcg/day for two days. Some recommend administration of a second 2-day course fourteen days after the initial course to treat any remaining parasites from autoinfection.⁷ In cases of hyperinfection or disseminated disease, ivermectin 200mcg/kg/day is administered daily for at least two weeks, with some recommending continuing therapy until stool studies remain negative for two weeks. If ivermectin is contraindicated due to allergies or in patients with high grade Loa loa infection, albendazole is an alternative, although slightly less effective treatment for *Strongyloides*.⁸ Although there is no gold standard test of cure, as in this patient, response to treatment can be assessed by the patient demonstrating clinical improvement in symptoms, with negative stool parasite screening if previously positive, fall in the *Strongyloides* antibody titer, and resolution of the peripheral eosinophilia.

Conclusions

Peripheral eosinophilia is a non-specific finding that can be associated with parasitic infection, allergic conditions, malignancy, and autoimmune disease. More than one underlying process may contribute to peripheral eosinophilia at a given time. It is therefore important not to anchor on the suspected diagnosis, particularly when eosinophilia is attributed to autoimmune disease, as the concomitant presence of a parasitic infection could have significant management and prognostic implications in the setting of immunosuppression to treat the autoimmune disease. When eosinophilia is identified, and especially prior to starting immunosuppressive therapy, infectious diseases screening should be performed as guided by epidemiologic risk factors.

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