Although olfaction is impaired early in the vast majority of people with idiopathic Parkinson’s disease (PD), we have been struck by how infrequently patients complain about loss of taste, an unavoidable result of olfactory disturbance. Taste and dietary intake may be altered in PD for a number of reasons, including, but not limited to chewing and swallowing dysfunction, slowness, decreased saliva production, and flexed posture that interfere with eating. In addition, patients with PD frequently lose weight, most likely also for many different physical and emotional reasons. We observed that when PD patients complained of weight loss and were counseled to eat more ice cream, often they reported already eating large amounts of it. We therefore performed a pilot study to determine if ice cream preference was actually increased in PD.

METHODS

A six-question survey was distributed at meetings of PD support associations, one in RI and the other in CT. The surveys were given to all attendees over the age of 50. Non-PD subjects served as normal controls (NC). Attendees were asked to volunteer to fill out the anonymous questionnaires. The questionnaires asked the same six questions of patients and controls, as to whether there had been changes in desire for, or consumption of, sweets or ice cream since the patient had developed PD. The data were analyzed with T-tests or chi-square analyses, utilizing p=0.05 as the significance threshold, and comparisons were made across gender and disease duration.

RESULTS

Twenty-nine female PD patients and 42 female NC, along with 30 male PD patients and 15 male NC filled out questionnaires anonymously. Female PD and NC did not differ in age (PD=68.9, controls 65.4; p=.23). Male NC were too few to use for comparative analysis. Female PD patients had a significant increase in their desire for ice cream compared to the NC group (60.7% vs 19.0%, p=.001, respectively) although the increase in ice cream consumption was not different in the two groups. In the female PD group, there were positive correlations between use of dopaminergic drug use and sweets consumption (p<.01), illness duration and ice cream preference (p=.05), and illness duration and ice cream intake (p=.02). When groups were collapsed across gender, individuals with PD compared to the NC group reported greater increase in sweets preference (41.5% in the PD vs 23.8%, respectively, p<.03), increased sweets consumption (43.1% vs 26.2%, respectively, p<.05), and increased ice cream preference with (52.4% vs 22.2%, respectively, p<.001), although the reported increase in ice cream consumption was not statistically different.

DISCUSSION

Our results confirmed our observation that people with PD develop an increased desire for ice cream. While the cause was not evaluated in this pilot study, it could be due to the ease of swallowing, a change in taste or an innate attraction to a high calorie, sweet food.

Increased consumption of animal fat and saturated fat has been associated with an increased risk of developing PD as has an increase in consumption of dairy products. PD patients losing weight have been found to increase their intake of fat.

Our observations are limited to those who already have PD. However, the increase in ice cream preference and consumption could conceivably begin early in the disease course, and increase with disease progression. This could explain the observed association between developing PD and increased fat and dairy consumption. Published studies did not distinguish ice cream from other food groups, limiting our ability to speculate on how supportive our findings are of these other reports. We also found that there was a general increase in sweets preference, within the PD group.

Our results are very preliminary. Limitations to our findings include small sample size, particularly with respect to healthy male control participants. Nevertheless our results did support our clinical impression of an increased desire to eat ice cream among PD patients compared to a healthy comparison group.

Ice cream is an easy to swallow, high energy food that PD patients seem to gravitate to. We see no reason, without further data, to discourage this tendency.

REFERENCES


This work was performed when Clifford Meyers was a Brown medical student, and Melissa A. Amick, PhD, was in the Department of Human Behavior and Psychiatry, Memorial Hospital of Rhode Island.
Eosinophilia Secondary To Strongyloides
In Rhode Island
Samir Dalia, MD, and Gerald A. Colvin, DO

With an increase in travel and an influx of immigrants and refugees from the tropics over the last few decades, clinicians in Rhode Island are more commonly encountering tropical diseases. The Federation for American Immigration Reform estimated that the average annual rate of increase in the foreign-born population in Rhode Island to be 2400 persons, with the Dominican Republic and Guatemala two of the largest countries from which people emigrate.1 As a result, hematologic abnormalities such as eosinophilia can arise without any other symptoms, perplexing clinicians as to the proper workup. Hematologists at The Rhode Island Hospital have noticed a significant increase in referrals of eosinophilia with mild leukocytosis or anemia, making it important to discuss major causes in immigrant populations. Infections such as hookworm and Strongyloides stercoralis (Strongyloides) are the most common parasitic nematodes to cause eosinophilia in tropical and subtropical areas.2 We present a case to illustrate the workup of eosinophilia and to describe the diagnosis and treatment of Strongyloides.

CASE
A 42 year-old man with a history of diabetes and hypercholesterolemia who emigrated four years prior from Columbia was referred to the hematology clinic after his primary care physician found a leukocytosis with increased eosinophils. The patient denied any diarrhea, rashes, itching, allergies, nausea, vomiting, cough, fevers, chills, night sweats, weight changes, or any other symptoms.

The patient’s family medical history was relevant for an unknown anemia disorder and diabetes. The patient did not smoke, drink or use any illicit drugs or herbal medications. His medications included metformin, fenofibrate, olmesartan, pravastatin and aspirin. He denied any new medication changes or allergies to any medications.

On exam he was afebrile, with a blood pressure of 143/80 and a pulse of 75 beats per minute. He was well nourished and comfortable. Cardiopulmonary exam showed no abnormalities. Abdominal exam showed a soft abdomen without any masses or organomegaly. Extremity, lymph node exam and skin exam were all negative.

Laboratory data showed a BUN of 8 mg/dL (normal [nl] 7-25 mg/dL) and creatinine of 0.7g/dl (nl 0·4-1·3 mg/dL). Liver function tests and electrolytes were normal. White blood cell count was 12 x 10^3/mm^3 (nl 3.5-11 x 10^3/mm^3) with 10% (nl 1-3%) eosinophils present. Peripheral blood flow cytometry was negative for any lymphocyte abnormality. Hemoglobin and platelet count was within normal limits.

The patient had further workup with stool ova and parasites, Strongyloides IgG antibody, hemoglobin electrophoresis, IgE level, and serum protein electrophoresis. Results showed an IgE level of 1381 mIU/ML (nl 3-209) and a Strongyloides IgG antibody of 8.37 (nl <1) indicating an underlying infection of Strongyloides. The patient was treated with two courses of albendazole therapy with improvement of his eosinophilia.

DISCUSSION
Peripheral blood eosinophilia can occur from a variety of causes including parasitic infections. From primary blood disorders to systematic diseases to infectious processes, eosinophilia is sometimes...