Case of the Month

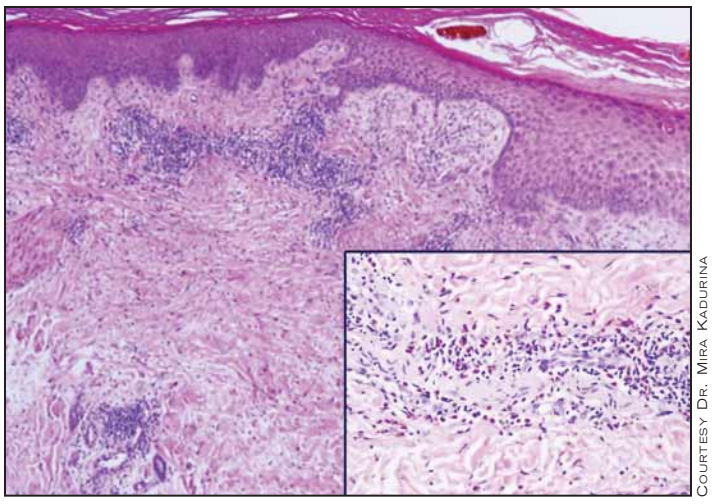
Diagnosis: Idiopathic Hypereosinophilia

LONDON — Laboratory evaluation also revealed anemia and an elevated erythrocyte sedimentation rate. Histology findings included hyperkeratosis, irregular acanthosis, and a mixed inflammatory infiltrate of lymphocytes, plasmocytes, and numerous eosinophils. Thrombosis of small vessels in the dermis and edema of the vessel walls also was noted.

No cause for her eosinophilia could be identified despite a meticulous search. Reactive eosinophilia, such as can occur with parasitic infections, and clonal disorders of the bone marrow associated with eosinophilia, such as various types of leukemia, were ruled out.

The diagnosis, therefore, was idiopathic hypereosinophilia syndrome, Dr. Mira Kadurina said in a poster presentation at the 14th Congress of the European Academy of Dermatology and Venereology.

Some investigators have proposed that idiopathic hypereosinophilia syndrome is a Th2-mediated disease characterized by clonal expansion of a T-cell population able to produce interleukin (IL)-5 and IL-4. Pathogenic T cells—usually CD3 negative, CD4 positive—display an aberrant surface phenotype.



COURTESY DR. MIRA KADURINA

Histology findings included a mixed inflammatory infiltrate of lymphocytes, plasmocytes, and numerous eosinophils.

The clinical presentation is heterogeneous and includes myeloproliferative and lymphocytic variants. In the more aggressive myeloid variant, patients can have chromosomal abnormalities, hepatosplenomegaly, cardiac complications, and myeloid malignancies. The prognosis is poor, said Dr. Kadurina of the department of dermatology and venereology at the Military Medical Academy in Sofia, Bulgaria.

The lymphocytic variant may be a primitive lymphoid disorder characterized by nonmalignant expansion of an IL–5-producing T-cell population. Cutaneous manifestations can include pruritus, eczema, erythroderma, and urticaria.

Immunohistochemical staining of tissue specimens from this patient revealed the presence of CD43-positive and CD4-positive cells, as well as CD8-negative and CD20-negative lymphocytes.

She was treated with prednisone, 60 mg/day, which was gradually tapered to 15 mg/day over a month's time. After treatment was withdrawn she once again developed disseminated, erythematous, pruritic lesions, this time involving the hands and feet. The fingers became painful, cyanotic, and swollen, initially after exposure to cold. A painful ulcer appeared on the third finger of the right hand.

Raynaud's phenomenon, identified by capillaroscopy, was an unusual cutaneous complication of the idiopathic hypereosinophilia syndrome in this patient, Dr. Kadurina wrote.

The administration of methylprednisolone, 60 mg/day, and pentoxifylline, 800 mg/day, led to a remission; the corticosteroid dosage was tapered to 5 mg/day over 45 days.

During 3 months of follow-up no new lesions appeared, the vasoconstriction of the patient's hands disappeared, and the finger ulcer healed.

A proposed explanation for the development of Raynaud's phenomenon and digital gangrene in association with hypereosinophilia, as occurred in this patient, is that major basic protein and eosinophil cationic proteins located in the eosinophil granule matrix contributed to the formation of microthrombi.

Efforts continue to further explicate the pathogenesis. "Future progress in unveiling variants of the syndrome is likely to consign to history the term idiopathic, replacing it with an array of well-defined hematologic disorders," Dr. Kadurina wrote.

—Nancy Walsh

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