On admission, the leukocyte count was 12.57 × 10⁹ cells/L, erythrocyte sedimentation rate was 68 mm/h, and C-reactive protein level was 73 mg/L; cultures of blood and sputum were negative. Azathioprine and colchicine therapies were stopped, prednisone therapy (5 mg/d) was continued, and subcutaneous anakinra therapy (100 mg/d) was started. Within 7 to 10 days, there was a dramatic remission in fever, marked improvement in oral and genital ulcers, no evidence of thrombophlebitis, and decreases in erythrocyte sedimentation rate (28%) and C-reactive protein level (86%) (Figure). When anakinra dosing was reduced to alternate days, oral ulcers and fever reappeared but remitted on return to daily dosing. After 20 months of daily anakinra (100 mg) and prednisone (5 mg) treatment, the patient remains disease-free.

Discussion: The mechanisms underlying the pathogenesis of Behçet disease remain unknown, and the presence of clinical clusters indicates that there are several pathways (1). The amelioration of disease with anakinra in this patient is consistent with the concept that Behçet disease is an IL-1–mediated autoinflammatory disease (2). The ethnic component of Behçet disease links it to other autoinflammatory diseases, such as familial Mediterranean fever and the tumor necrosis factor receptor–associated periodic syndrome, which occur with increased frequency in patients with Behçet disease (3) and are also responsive to anakinra. The pathologic abnormality in autoinflammatory diseases is increased production of IL-1β. Blood monocytes from patients with these diseases release more active IL-1β than do cells from unaffected individuals. Reducing IL-1β activity reduces disease activity (4). Although anakinra is presently approved for reducing IL-1 activity, monoclonal anti–IL-1β antibodies and IL-1 Trap have also been used to successfully treat other autoinflammatory diseases (5). The near-complete resolution of clinical, biochemical, and hematologic abnormalities in our patient suggests that IL-1β is a primary mediator of inflammation in Behçet disease. Similar resolution of disease severity has been observed with anakinra in patients with adult-onset Still disease or neonatal-onset multisystem inflammatory disease that was resistant to conventional therapy as well as anti–tumor necrosis factor-α therapy (4). The half-life of anakinra is less than 6 hours, which explains disease flare-ups with dose reduction.

Conclusion: Anakinra may be a treatment option for patients with Behçet disease that is resistant to conventional therapy.

Costantino Botsios, MD, PhD
Paulo Sfriso, MD, PhD
Antonio Farlan, MD
Leonardo Punzi, MD, PhD
University of Padova
Padova 35122, Italy

Charles A. Dinarello, MD
University of Colorado Health Sciences Center
Denver, Colorado 80262

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References
enzyme and creatine phosphokinase levels and had a low-density lipoprotein cholesterol level of 3.94 mmol/L (152 mg/dL).

Discussion: The improved tolerance to simvastatin after the patient stopped drinking green tea and the results of the kinetic studies suggest a clinically relevant green tea–statin interaction. The underlying mechanism of the interaction needs to be investigated. In healthy volunteers, green tea extracts had only minor effects on the activity of CYP450 3A4, the main simvastatin-metabolizing enzyme (2, 3). Thus other target molecules of green tea may be involved. We cannot say whether the interaction relates to an individual genetic susceptibility to metabolic modulation by green tea or whether it is a common effect of this infusion.

Conclusion: To our knowledge, this is the first report of a case of statin intolerance associated with green tea consumption. Popularized data indicating green tea consumption as a "natural" strategy to prevent diseases (4) may increase the chances of this infusion in patients who are taking statins. Thus, pending corroboration, it may be time to consider green tea to be among unexpected triggers of statin toxicity.

Jose Pablo Werba, MD
Monica Giroli, PhD
Viviana Cavalca, PhD
Maria Cristina Nava, MD, PhD
Elena Tremoli, PhD
Centro Cardiologico Monzino, Istituto di Ricovero e Cura a Carattere Scientifico
Milan 20138, Italy
Lorenzo Dal Bo, PhD
Institute for Pharmacokinetic and Analytical Studies
Ligornetto 6853, Switzerland

Potential Financial Conflicts of Interest: None disclosed.

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