Lipid Profiles in Patients With Ulcerative Colitis Receiving Tofacitinib—Implications for Cardiovascular Risk and Patient Management

To the editors,

With great interest we read the article of Sands, Colombel, et al, which reflects on the increases of lipid levels in patients with ulcerative colitis treated with tofacitinib after the OCTAVE registration trials. Tofacitinib treatment was associated with modest, reversible, dose-dependent increases in serum total cholesterol, high-density cholesterol, and low-density cholesterol after the 8-week induction period, and the levels remained elevated during maintenance therapy.

As a support for clinicians, the study presents a well-defined algorithm for the management of lipid changes in patients with ulcerative colitis who start tofacitinib treatment. However, the question arises as to whether the application of this algorithm should be limited to tofacitinib.

As the authors explain, chronic inflammation is associated with lower lipid levels. Therefore, inflammation that resolves after treatment is a likely explanation for the observed lipid increases. This concept is illustrated by the small but significant inverse correlation between C-reactive protein and lipid levels, not only in tofacitinib users but also in the placebo arm. Dietary cholesterol is absorbed in the intestinal wall through Niemann-Pick C1 Like 1 protein receptors. In patients with inflammatory bowel disease (IBD), both systemic inflammation and local inflammation of the intestine could play a role in lower lipid levels during active disease.

Previous studies described increasing lipid levels in patients with IBD who were treated with other immune-modulating drugs including corticosteroids, cyclosporine, and tumor necrosis factor alpha antibodies. It is unknown whether the pathways leading to lipid changes are different between drug classes. Therefore, more in-depth studies are urgently needed in this field.

In anticipation of studies addressing these important questions, we underline the importance of lipid evaluation in the IBD population and suggest that Sands, Colombel, et al replace tofacitinib by “immune-modulating therapy” in the proposed algorithm.

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