Dear Sir,

We appreciate the Letter to the Editor by Drs. Park and Shin regarding our recent manuscript describing a case of reversible Henoch–Schönlein purpura (HSP) complicating adalimumab (ADA) therapy. We are thankful for their attention to our work and for the interesting arguments raised for our consideration.

In the face of such uncertainty in the pathogenesis of HSP related to anti-TNFα therapy (particularly ADA therapy) we would stress the importance of finding underlying mechanisms responsible for such adverse events.

As outlined by Drs. Park and Shin and many other authors, TNF inhibitors induce an immune deviation from Th1 phenotype to the Th2 phenotype, which can lead to eosinophilia and elevated IgE levels.1 Mostly, isolated eosinophilia while on anti-TNFα therapy is not clinically significant, but the release of toxic granule proteins (such as eosinophil-derived neurotoxin, eosinophil cationic protein (ECP), eosinophil peroxidase and eosinophil major basic protein) might potentially lead to adverse phenomena, namely asthma,2 eosinophilic cellulitis (Wells' syndrome),3 HSP and HSP nephritis.4 This should be reminded mainly when persistent eosinophilia is identified during TNFα inhibition.

Although in our case report we did not assay serum levels of ECP, as blood eosinophil count was slightly increased, that could have been an enlightening detail of the pathogenic mechanism underlying ADA related HSP and HSP nephritis.

As the use of anti-TNFα continues to increase, the diagnosis and management of side effects will become an important challenge. We too hope that our paper, as well as the points raised in the Letter to the Editor, will help understand and avoid deleterious reactions to anti-TNFα therapy.

Conflict of interest statement

The author declares no conflict of interest.

References


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