Reply to “Managing T2-high severe asthma in HIV-infected patients”

To the Editor:
First of all, we thank Terl and Jesenak for their interest in our article and for taking time to let us know about their case. When we decided to publish our case report, it was with the interest of showing our results to someone in the situation of having to make a complicated decision in a patient like this. The authors’ answer, seeing a similar case, reinforces our decision.

On a clinical level, we have to say that our patients have certain differences. Although the case Terl and Jesenak present has childhood onset asthma associated with allergy and atopic march, our patient has a very clear onset in adulthood and did not have associated allergy or atopy. Thanks to the close analytical follow-up, we have been able to see how the onset of asthmatic symptoms was associated with eosinophilia. Before 2016, the highest reported value of eosinophils, with at least 4 analytical tests per year since 2011, was 280 cells/mm³ and, as we reported in the article, once the symptoms started, it reached much higher values. Otolaryngologist evaluation revealed grade I polyps only on the left side. To date, he is adequately tolerant of nonsteroidal anti-inflammatory drugs, so from our point of view, he is a case of severe eosinophilic asthma associated with nasosinusal polyps, but he does not meet the classic Fernand-Widal triad.

As far as the choice of biologics is concerned, the first thing we must take into account is that our patient started treatment a year earlier, and, as of the end of 2018, the supply of biologics was limited. At the time, our case raised few doubts, and probably the choice would have been the same regardless of the HIV. But for the patient Terl and Jesenak present, if hypothetically evaluated in October 2018, the choice would have been more complicated. The doubt would have been probably between omalizumab and dupilumab, as Terl and Jesenak decided. On one hand, there is evidence showing the improvement that occurs in patients with atopic dermatitis and asthma treated with omalizumab, even with IgE above the limits. On the other hand, the relationship between IgE, eosinophils, and HIV is controversial and from our point of view requires further study and elaboration. Most of the available data are in children, and more than eosinophilia, they relate elevated IgE to a worse prognosis. Regarding dupilumab, the first phase 3 study had just been published at that time, and it was not routinely used. It would have been difficult to apply not only in a standard patient but also in a patient with HIV.

Cases like this, and other special comorbidities, will appear in the coming years. The more information available at the immunological level on the effects caused by biological drugs, and the better we know the inflammatory pathways associated with asthma, the better we will be able to make rational and personalized selections in our patients.

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