Commentary: Human Immunodeficiency Virus and Allergic Bronchopulmonary Aspergillosis

To the Editor—We read with great interest the brief report of a case of allergic bronchopulmonary aspergillosis (ABPA) in a human immunodeficiency virus (HIV)/acquired immune deficiency syndrome (AIDS)-infected patient on combined antiretroviral therapy (cART) by Galiatsatos et al [1]. Allergic bronchopulmonary aspergillosis usually complicates lung disease in patients affected by cystic fibrosis (CF) or asthma. The association of ABPA with other lung disease such as chronic obstructive pulmonary disease, bronchiectasis, Kartagener's syndrome, chronic granulomatous disease, and hyper-immunoglobulin (Ig)E syndrome has been suggested but not confirmed [2, 3]. The diagnosis of ABPA in Galiatsatos's [1] patient satisfied recent criteria proposed by Agarwal [2]: presence of predisposing condition (asthma), 2 of 2 obligatory criteria (total IgE > 1000 IU/mL, elevate IgE levels against *Aspergillus fumigatus*) and 2 of 3 additional criteria (radiological findings, eosinophilia) [4].

It is interesting to note that in the discussion, the authors share important insights on the still poorly explored field of ABPA development in patients with HIV infection. More specifically, they suggest the possible existence of a hyperactive immune response contributing to the development of ABPA in AIDS patient on cART and with controlled HIV infection [1]. Although such a possibility is highly suggestive, we think the presence of some genetic mutations should also be investigated, which might explain per se the development of ABPA in this patient. Indeed, the reported clinical picture of ABPA with bronchiectasis, a major phenotypic feature of CF, impose an appropriate investigation of milder forms of CF (with normal pancreatic function) [5]. Moreover, the presence of other Cystic Fibrosis Transmembrane conductance Regulator (CFTR)-related disorders should also be ruled out. In this regard, the frequency of ABPA was much higher in patients with these disorders rather than in the general population (67% vs 4%, respectively) [6].

In conclusion, we thank the authors for sharing this very peculiar case. Further studies are needed to enrich our understanding of any possible association between ABPA and HIV infection and to better define and weigh the role of CFTR mutations against this backdrop.

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