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LETTER TO THE EDITOR

Anti-dsDNA antibodies and their association with paradoxical psoriasis induced by TNF alfa inhibitors

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Paradoxical psoriasis or psoriasiform reaction is an adverse effect, represented by occurrence of a disease caused by the therapeutic class of drugs normally used to cure or improve symptoms of psoriasis. Anti-TNF agents have a dominant place in the management of inflammatory diseases, including psoriasis (1, 2). We present ten patients in whom psoriasiform skin lesions developed after the initiation of anti-TNF alfa therapy (adalimumab 6 patients, infliximab 4 patients) for Crohn disease (4), ulcerative colitis (3), psoriasis (2), and hidradenitis suppurativa (1). The aim of this study was to describe clinical features, analyze antinuclear antibodies ANA and anti-dsDNA and identify by genotyping HLA class I a II alleles associated with psoriasis. Five patients developed primary plaque type psoriasis, five palmoplantar pustular psoriasis (PPP). Four of five patients with PPP did not have positive HLA alleles associated with psoriasis. Our study confirms the results of multiple studies describing higher incidence of PPP (46.2 %) than in psoriatic population (1.7 %) (2, 3). Antinuclear antibodies (ANA) and double-stranded DNA antibodies were detected by ELISA. ANA serum samples were positive in 10 % of patients and anti-dsDNA in 70 % of patients. The mechanism driving the formation of ANA and anti-dsDNA antibodies is poorly understood and their clinical significance is unknown (4). Frequency of ANA and anti-dsDNA in the patients on anti-TNF alfa varies extremely in literature, possibly due to different methods of detection used. Pink et al. in their study suggest that the development of ANA and anti-dsDNA antibodies on anti-TNF alfa treatment may act as a marker of forthcoming treatment failure. In anti-TNF alfa induced lupus erythematosus, ANA and anti-dsDNA antibodies are well established and quite common (ANA 90 %, anti dsDNA 70–90 %). Nevertheless, we have limited data about other immunologically mediated side reactions (5). Our data suggest that paradoxical psoriasis induced by anti-TNF inhibitors is associated with the production of anti-dsDNA autoantibodies, but not with that of ANA antibodies. Further prospective research is necessary before assessing general recommendations for daily practice. Genetic predisposition, clinical manifestation, and production of anti-dsDNA seem to be fundamental in differentiating between adverse effect of anti-TNF alfa and associated disease comorbidity.

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